SWAPE Technical Consultation, Data Analysis and Litigation Support for the Environment

2656 29th Street, Suite 201
Santa Monica, CA 90405

Matt Hagemann, P.G, C.Hg.
(949) 887-9013
mhagemann@swape.com

September 7, 2017

Mitchell Tsai
Mitchell M. Tsai, Esq.
1055 E. Colorado Boulevard, 5th Fl.
Pasadena, CA 91106

Subject: Comments on the Devil's Gate Reservoir Sediment Removal and Management Project

Dear Mr. Tsai:

We have reviewed the Recirculated Final Environmental Impact Report (RFEIR) for the Devil's Gate Reservoir Sediment Removal and Management Project ("Project") prepared by the Los Angeles County Flood Control District (LACFCD), as the lead agency under the California Environmental Quality Act (CEQA). The Project is located in the City of Pasadena, in Los Angeles County approximately 14 miles north of downtown Los Angeles. The sediment removal phase of the Project will haul approximately 7,650 CY of sediment per day using double dump trucks which have an estimated capacity of 16 to 20 CY. Removal of the sediment, vegetation, trees, and organic debris is expected to require an average of 50 round truck trips per hour, with an estimated maximum of 425 round trips per day during excavation activities. The trucks will drive through local neighborhoods, and the 134 and 210 Freeways. The diesel trucks will operate for an estimated 9 months per year, 6 days per week, for 5 years.

Our analysis of the RFEIR demonstrates that the RFEIR's finding that the Project's significant NOx-related air quality impacts from on-road truck emissions would be mitigated to less than significant levels with implementation of Mitigation Measure (MM) AQ-1 is inaccurate and unsupported. As revised, MM AQ-1 would require all sediment removal dump trucks used for the Project to be exclusively Environmental Protection Agency's (EPA) Model Year 2010 (MY2010) trucks.[1] However, the table in the RFEIR which estimates the Project's mitigated NOx emissions assuming the use of MY2010 trucks relies on outdated emissions estimates that have been replaced by recent in-use emissions studies which demonstrate that MY2010 and later engines, which contain selective catalytic reduction emission control systems (SCR) to reduce NOx emissions pursuant to the EPA 2007 rules, emit between 5 to 18 times higher NOx emissions

[1] RFEIR, p. 431.

than the EPA certification assumed.[2] The RFEIR fails to disclose this critical evidence, and fails to quantify mitigated emissions using current on-road emissions estimates.

The in-use emissions studies include a series of studies conducted by the University of California Riverside's Center for Environmental Research and Technology (CE-CERT) and prepared for SCAQMD. [3] These studies provide substantial evidence demonstrating that the use of MY2010 trucks will result in lower NOx reductions – and consequently higher NOx emissions – than previously assumed by the District. An analysis of the CE-CERT studies is therefore critical to the District's evaluation of the efficacy of MM AQ-1.[4]

By failing to disclose and quantify the reduced in-use emissions reductions offered by MY2010 trucks, the RFEIR fails to accurately estimate the Project's mitigated emissions. Thus, the RFEIR's conclusion that the Project's NOx emissions will be reduced below SCAQMD levels of significance using MM AQ-1 alone is unsupported. The District must prepare an updated air quality analysis is prepared that adequately evaluates the effectiveness of the Project's proposed mitigation in reducing the significant NOx and other criteria air pollutant emissions generated during Project sediment removal activities. The RFEIR may not be certified until the District implements all feasible mitigation measures to reduce these significant impacts to less than significant levels.

Air Quality

As part of the proposed Project's goal to restore and maintain the flood capacity at the Devil's Gate Reservoir, the EIR developed an extensive sediment removal plan to provide flood protection for communities located downstream (FEIR, p. ES-1). According to information disclosed in the EIR, the Project proposes to remove approximately 2.4 million cubic yards (mcy) of excess sediment in the Reservoir (RFEIR, p. ii). Using hauling trucks with a 16 to 20 cy capacity, the Project proposes to haul approximately 7,650 cy of sediment per day from the Project site to several predetermined facilities (p. ES-5). The facilities that will be used for the majority of the proposed Project's sediment placement will include the Waste Management Facility in Azusa, the Vulcan Materials Reliance Facility in Irwindale, and the Manning Pit Sediment Placement Site (SPS) in Irwindale, while the Scholl Canyon Landfill will be used to dispose of vegetation and organic debris. Sediment may also be placed at the following disposal facilities in the Sunland area of Los Angeles: Sheldon Pit, Sun Valley Fill Site, Bradley Landfill, and Boulevard Pit (FEIR, p. 7). Furthermore, the EIR concludes that the Project's sediment removal and maintenance activities are "expected to require an average of approximately 50 truck trips per hour,

[2] Durbin, Thomas D, et al. (February 2017). Final Report: Heavy-Duty Chassis Dynamometer Test Program, p. 60; Miller, Wayne, et. al. (September 2013). In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines, available at http://www.cert.ucr.edu/research/efr/2013_AQMD_in-use_retrofit_Miller.pdf.

[3] Durbin, Thomas D, et al. (February 2017). Final Report: Heavy-Duty Chassis Dynamometer Test Program, p. 60; Miller, Wayne, et. al. (September 2013). In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines, available at http://www.cert.ucr.edu/research/efr/2013_AQMD_in-use_retrofit_Miller.pdf.

[4] Id.; see also http://www.cert.ucr.edu/research/efr/2016%20CWI%20LowNOx%20NG_Finalv06.pdf;

with an estimated 425 round hauling truck trips per day during excavation activities" for an estimated 9 months per year, 6 days per week, over a 5-year duration (FEIR, p. 22, p. 85).

As a result of the substantial number of hauling truck trips expected to occur during Project construction, the October 2014 FEIR determined that that the proposed Project would emit approximately 378 pounds per day (lbs/day) of NOx emissions, which significantly exceeds the South Coast Air Quality Management District's (SCAMQD) daily threshold of 100 lbs/day (FEIR, p. 86). The FEIR concluded that with implementation of mitigation, the Project's NOx emissions would be reduced to a less than significant level (p. 88). Specifically, the FEIR stated,

> "As shown below in the impact discussions in AIR QUALITY-2 and AIR QUALITY-4, sediment removal activities have the potential to violate an air quality standard or contribute substantially to an existing or projected air quality violation. This is due to emissions of NOX exceeding the Daily Regional Thresholds during sediment removal, resulting in a potentially significant impact. Use of sediment removal dump trucks that meet EPA's emission standards for Model Year 2007 and use of off-road equipment that meets, at a minimum, EPA's emission standards for Tier 3 equipment, would result in a reduction of NOX emissions to less than the SCAQMD Regional Threshold for NOX. Implementation of Mitigation Measures MM AQ-1 and MM AQ-2 will result in a reduction of NOX emissions to less than the SCAQMD Regional Threshold for NOX" (p. 83).

The original FEIR incorrectly assumed that trucks required to meet the EPA's 2007 or later emissions standards under MM AQ-1 would result in compliance with EPA Model Year 2010 levels (FEIR p. 84, p. 88). However, EPA regulations clearly states that EPA's 2007 standards were phased in over a 3-year period for diesel truck engines in Model Years 2007-2010.[5] Furthermore, EPA regulations state that only 50 percent of Model Year 2007 trucks are required to comply with EPA 2007 emissions standards, whereas 100 percent of Model Year 2010 trucks are required to comply with the standards.[6] In response, a judgment from the Superior Court of the County of Los Angeles invalidating MM AQ-1 and requiring that portions of the FEIR be revised and recirculated. In response to the judgment, the RFEIR revised MM AQ-1 to state the following:

> "LACFCD shall require all construction contractors during the sediment removal phase of the Proposed Project to use only sediment removal dump trucks that meet the EPA's emission standards for Model Year 2010 or later" (RFEIR, ES-12).

Although the revisions made to MM AQ-1 in the recirculated portions of the FEIR now require that 100 percent of the Project's hauling trucks must meet the EPA's 2007 emissions standards, new evidence demonstrates that the use of Model Year 2010 hauling trucks will actually result in less effective NOx

[5] "U.S. EPA 2007/2010 Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements", *available at:* http://www.meca.org/regulation/us-epa-20072010-heavyduty-engine-and-vehicle-standards-and-highway-diesel-fuel-sulfur-control-requirements

[6] *Ibid.*

reductions than previously assumed. The District appears to have overlooked this critical information in preparing the RFEIR.

The CE-CERT studies, prepared between 2013 and 2017, conducted real-time in-use studies of truck emissions using heavy-duty chassis dynamometers to measure actual NOx and other air pollutant emissions. The studies concluded that Model Year 2010 (and later) trucks emit NOx at levels that are 5 to 18 times higher than the levels assumed in the original 2007 EPA certification standard. Because MM AQ-1 requires the use of MY2010 trucks, MM AQ-1 will similarly result in higher, unmitigated NOx emissions that are 5 to 18 times higher than the levels assumed in the RFEIR. An updated air quality analysis must be prepared which quantifies the Project's NOx emissions using the actual MY2010 performance emissions quantified in the CE-CERT studies. If the results of that study demonstrate that mitigated NOx emissions remain above applicable levels of significance (i.e. over 100 lbs/day), the District must implement additional mitigation measures to reduce NOx emissions from on-road trucks to less than significant levels.

New Evidence Provided in University of California Riverside's Center for Environmental Research and Technology Studies

CE-CERT conducted three recent studies on the in-use emissions generated by EPA Model Year 2007 and Model Year 2010 heavy-duty engines.[7] The studies were prepared in response to concerns over the lack of data on the in-use emissions generated by diesel engines traveling under typical driving conditions. Prior to the CE-CERT studies, the only substantial test cycle research available on the ability of diesel engines equipped with DPFs and SCR systems to meet emissions certification levels had been done in a laboratory setting.[8] Specifically, one study explains that, "although measurements in laboratories were showing NOx and PM emissions meeting the stringent certification standards, some values from in-use conditions were showing increased emissions of ammonia from LNG trucks and of NOx from diesel trucks".[9] The CE-CERT studies were prepared for SCAQMD[10] to provide a better understanding of actual emissions inventories on MY 2007 and MY 2010 trucks, and to provide further information for use in the development of regulations for in-use vehicles.

One of the CE-CERT's studies, *In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines,* evaluated sixteen heavy-duty vehicles used in transportation,

[7] As the study explains, the most technologically advanced diesel engines available today are equipped with diesel particulate filters (DPFs) to meet PM standards for 2007 and newer engines and SCR systems to meet NOx standards for 2010 and newer engines*Ibid*, pp. 15

[8] Durbin, Thomas D, et al. (February 2017). Final Report: Heavy-Duty Chassis Dynamometer Test Program; Maldonado, Hector, et. al. (April 2007). Final Report: Evaluation of On-road Results from a Test of Heavy-Duty Trucks, p. ii; Miller, Wayne, et. al. (September 2013). In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines, p. xvi

[9] *Ibid*, p. 1

[10] See e.g. Miller, In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines, September 2013, prepared for Mr. Adewale Oshinuga, South Coast Air Quality Management District.

refuse, and goods movement applications to test the efficiency of emissions reductions resulting from retrofit technology.[11] The study concluded that, while NOx emissions were dependent upon multiple variables, shorter trip lengths and/or frequent stops during use resulted in significantly higher NOx emissions that exceeded the emissions certification level.[12] The study states,

> "In summary, the data from this study suggests that 2010 compliant SCR-equipped HDD vehicles are exhibiting high in-use NOx emissions that can be as high as 2 g/hp-h under low load conditions represented by short trips or frequent stops… looking ahead, the overall results suggest NOx emissions are still a concern for selected activities, and SCR performance needs to be investigated during wide in-use, on-road operation to characterize its impact on local inventories."[13]

The CE-CERT study concludes that heavy-duty diesel engine trucks emit in-use emissions that are up to an entire order of magnitude higher than their certification levels.[14] Similarly, CE-CERT's *Heavy-Duty Chassis Dynamometer Test Program* report tested five vehicles in four phases of the California Air Resource Board's (CARB) Heavy Heavy-Duty Diesel Truck test cycles (i.e., idle, creep, transient, and cruise). The study concluded that creep cycle had the "highest NOx emissions on a g/mile or g/bhp basis since it is comprised of short, low-speed accelerations between periods of idle that yield lower loads and exhaust temperatures, and that cover a very short distance (0.124 miles)".[15] The studies note that emission levels vary depending on the type of vehicle, driving conditions, and the specific mode of engine operation. However, the studies provide clear and substantial evidence demonstrating that, under typical driving conditions, EPA-certified MY2007 and MY2010 heavy-duty trucks emit NOx emissions that exceed their emission certification levels, which therefore results in NOx emissions that may exceed applicable air quality thresholds.

Failure to Adequately Mitigate Significant Criteria Air Pollutant Emissions at Congested Intersections

As discussed above, the excess in-use NOx emissions documented in the CE-CERT studies undermines the RFEIR's conclusion that the use of MY2010 trucks alone will be adequate mitigation to reduce the Project's NOx emissions to less than significant levels. The RFEIR's conclusion that NOx emissions will be "less than significant after mitigation" is therefore unsupported. Additionally, substantial evidence contained in the CE-CERT studies demonstrates that the Project trucks' NOx emissions will remain significant and inadequately mitigated during periods in which the trucks are idling or creeping during heavy traffic conditions.

---

[11] Miller, Wayne, et. al. (September 2013). In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines, p. xvi

[12] *Ibid,* p. 117

[13] Miller, Wayne, et. al. (September 2013). In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines.

[14] For reference: emissions certification levels are 0.2 grams per break horsepower-hour (g/bhp-h).

[15] *Ibid*, p. 9

The Project will require 425 round trip hauling truck trips per day to export sediment from the Project site. The traffic analysis in the FEIR demonstrates that those trucks will be reduced to minimal speeds or will be stuck in stop-and-go traffic at specific intersections near the Project site. The FEIR relies upon the Highway Capacity Manual (HCM) method to estimate the Project's impact to the level of service (LOS) at various intersections along the Project's proposed haul truck routes (FEIR, p. 245). According to the FEIR, HCM methodology determines LOS at a particular intersection based on the delay of vehicles at the intersection (FEIR, p. 245). The FEIR explains that, "the HCM bases its delay on an adjusted flow using a mean control delay for the highest 15 minute period within the hour. The LOS for signalized intersections ranges from LOS A, which indicates excellent operating conditions, to LOS F, which represents congested conditions with excessive vehicle delay" (see excerpt below) (TIA, p. 32).

TABLE 2-6
Caltrans Freeway Ramp HCM LOS Criteria

LOS	Freeway Ramp Density (pc/mi/ln)
A	≤ 10.0
B	> 10.0 and ≤ 20.0
C	> 20.0 and ≤ 28.0
D	> 28.0 and ≤ 35.0
E	> 35.0
F	Exceeds Capacity

Source: **HCM 2000, Exhibit 25-4**

According to the FEIR's analysis, "the Berkshire Place and I-210 eastbound ramps intersection is anticipated to operate at an unacceptable LOS during the AM peak hour, resulting in a temporary significant impact" (see excerpt below) (FEIR, p. 249).

Table 3.16-3: LOS for Devil's Gate Reservoir to/from I-210 (eastern disposal sites), Year 2014 with Project Traffic

Intersection		AM				MID-DAY (12-2 PM)				MID-DAY (2-4 PM)				PM			
Intersection # / Name		LOS	HCM Delay	HCM V/C	ICU LOS	LOS	HCM Delay	HCM V/C	ICU LOS	LOS	HCM Delay	HCM V/C	ICU LOS	LOS	HCM Delay	HCM V/C	ICU LOS
1	Berkshire Place and I-210 eastbound ramps	F	51.4	-	-	B	10.8	-	-	C	23.7	-	-	D	31.6	-	-
2	Berkshire Place and I-210 westbound ramps	A	9.5	-	-	A	4.9	-	-	A	7.2	-	-	A	3.8	-	-
3	Oak Grove Drive and Berkshire Place	B	19.2	0.93	B	A	5.6	0.29	A	A	8.4	0.57	B	A	8	0.57	B
4	Oak Grove Drive and Foothill Freeway Overpass	C	19.2	-	-	A	9.6	-	-	A	9.5	-	-	B	11.3	-	-
5	Windsor Avenue and Oak Grove Drive/Woodbury Road	C	34.7	0.94	D	B	14.3	0.54	A	B	17.1	0.58	A	C	24.9	0.76	C
6	Windsor Avenue/Arroyo Boulevard and I-210 westbound ramps	B	10.4	0.66	B	A	6	0.31	A	A	7.3	0.41	A	A	8.2	0.45	B

The FEIR asserts that the impact to the Berkshire Place and I-210 eastbound ramps would be reduced by use of an alternative truck route (FEIR, p. 249). However, the FEIR admits that, while conditions under the alternative route are expected to result in a LOS C or better at the intersection, the use of the proposed alternative route relies upon the uncertain subsequent approval of new traffic control measures by the City of Pasadena that the District has not obtained:

> "Proposed Project haul trucks would avoid using the Berkshire Place and I-210 eastbound ramps intersection during the AM peak period by instead using the Windsor/Arroyo and I-210 ramps. This would require the median on Oak Grove Drive to be restriped to a Two Way Left Turn Lane (TWLTL). Trucks exiting the Devil's Gate Reservoir driveway will cross the two lanes of oncoming westbound traffic on Oak Grove Drive and utilize the TWLTL if necessary to merge into the eastbound traffic. The changes to Oak Grove Drive would require the approval of the City of Pasadena" (FEIR, p. 250) (emphasis added).

The FEIR subsequently notes that the alternative route's requirements "cannot be legally imposed by the LACFCD since the location is under the jurisdiction of the City of Pasadena" and that although "every reasonable effort will be made to coordinate with and receive approval to implement this impact reduction measure" the Lead Agency cannot guarantee that this measure will actually be implemented (FEIR, p. 250). The FEIR ultimately concedes that "these temporary impacts could remain potentially significant" (FEIR, p. 271). Therefore, the Project's haul truck route is likely to include substantial traffic delays at one or more intersections with LOS F conditions.

As stated above, according to the FEIR, a LOS F intersection is characterized as having "congested conditions" and "excessive vehicle delay" (FEIR, p. 246), which describes the conditions under which in-use NOx emissions were found to be the highest in the studies conducted by CE-CERT (*see* section titled New Evidence Provided in University of California Riverside's Center for Environmental Research and Technology Studies). Therefore, there is substantial evidence showing that the FEIR's previous conclusion that the Project's NOx-related air quality impact would be mitigated by implementation of MM AQ-1 is incorrect and unsupported, as this traffic impact is likely to generate substantially higher NOx emissions due to potential queuing and stop-and-go traffic resulting from the Berkshire Place and I-210 eastbound intersection. For example, according to the *Heavy-Duty Chassis Dynamometer Test Program* report, in-use NOx emissions were found to be the highest during the creep cycle (i.e., short, low-speed accelerations between periods of idle), where the highest emissions were found to be 3.613 g/bh-hr.[16] This emissions estimate is approximately 18 times higher than the 0.2 g/bh-hr NOx certification level. Therefore, the RFEIR's exclusive reliance on the use of MY2010 trucks to reduce the Project's NOx emissions is inadequate, as the potential in-use emissions resulting from use of MY2010 diesel trucks that are queuing and idling at the Berkshire Place and I-210 eastbound ramp intersection could be up to 18 times higher than what is anticipated by the RFEIR. The fact that the Berkshire Place and I-210 eastbound intersection will operate at an LOS level F demonstrates that trucks passing through this intersection will be forced into the slow moving and short, low-speed acceleration conditions that have been shown by the CE-CERT studies to emit significantly higher levels of NOx emissions. Thus, slow traffic conditions may cause the MY2010 trucks to emit up to 18 times more NOx than the RFEIR assumes, potentially resulting in severe and unmitigated NOx emissions.

Based on our review of the CE-CERT studies, the data contained in them, and the Project's air quality and traffic analyses, we conclude that this traffic-related impact, in combination with the Project's existing significant NOx and other criteria air pollutant emissions, has the potential to cause the Project's NOx emissions to exceed SCAQMD thresholds using MY2010 trucks, thus resulting in significant and unmitigated air quality impacts.

Moreover, the RFEIR's assertion that the Project's construction-related NOx emissions would be reduced from approximately 378 lbs/day to just 82 lbs/day emissions through the use of Model Year 2010 hauling trucks is unsupported. Therefore, we find that the RFEIR lacks substantial evidence to support the conclusion that the Project would not result in a significant air quality impact, and specifically that the RFEIR lacks evidence on which to conclude that MM AQ-1 will mitigate NOx emissions to less than significant levels. In contrast, there is substantial evidence suggesting that the air quality impacts from the Project's 425 daily hauling truck trips will not be adequately mitigated. The RFEIR cannot be certified until an updated air quality analysis is prepared that adequately evaluates the effectiveness of mitigating the Project's NOx emissions generated during sediment removal activities, and mitigates those emissions to less than significant levels.

[16] Durbin, Thomas D, et al. (February 2017). Final Report: Heavy-Duty Chassis Dynamometer Test Program, p. 60

Health Risks from Diesel Particulate Matter Underestimated

In an effort to determine the potential excess cancer risk posed to nearby sensitive receptors from diesel particulate matter (DPM) emitted by sediment transportation activities and operational maintenance, the October 2014 FEIR prepared a health risk assessment (HRA). However, our review of the HRA demonstrates that there are multiple incorrect assumptions made within the health risk assessment calculation itself. As a result, the excess cancer risk is underestimated, and should not be relied upon to determine Project significance. Our review of estimated Project emissions of DPM from sediment removal activities and operational maintenance determined that significant air quality impacts may be generated through the use of diesel-fueled heavy-duty trucks. Given that the CE-CERT studies NOx emissions in MY2010 trucks remain higher than originally estimated, and the RFEIR has increased the mitigated emissions estimates for off-road construction vehicles,[17] it is apparent that the RFEIR has underestimated mitigated DPM emissions. The use of MY2010 trucks alone, as required under MM AQ-1, is therefore not guaranteed to reduce DPM emissions to less than significant levels. The RFEIR should be revised and recirculated and include an updated health risk assessment that more accurately estimates the potential cancer risk from.

Failure to Account for Tire Wear and Brake Wear Emissions in Air Dispersion Model

Appendix C of the FEIR discusses the assumptions and values utilized to determine the health risk posed to nearby sensitive receptors. According to Appendix C, the emission rates used to determine the concentration of PM10 emitted during sediment transportation activities were derived from emission factors utilized in the FEIR's Air Quality Analysis (Appendix B) (Appendix C, p. 12). The report states, that "the Air Quality Analysis found that the haul trucks on surface streets would have PM10 emissions rates of 0.1461 grams per mile during the sediment removal activities (year 2015) and 0.0493 grams per mile during the operational maintenance activities (year 2020)…this results in emission rates of 4.340E-06 grams per second for the sediment removal phase and 1.465E-06 grams per second for the operational maintenance phase, which was used in AERMOD for all road volume line sources" (Appendix C, p. 12). These emission rates, inputted into the air dispersion model AERMOD, however, only include the PM10 running exhaust emissions emitted by the heavy-duty sediment transport trucks. The emission rate inputted into AERMOD fails to incorporate PM10 emissions from tire wear and from brake wear. As a result, the PM10 concentration at each sensitive receptor location is greatly underestimated.

EMFAC2011 estimates the directly emitted emissions of total PM10 by calculating the emissions from exhaust, tire wear, and brake wear.[18] Particulate matter from brakes and tires is defined as the airborne portion of the "wear" that can be created by abrasion, corrosion, and turbulence.[19] These wear processes result in particles being suspended in the atmosphere. The size, chemical composition, and emission rate of particles arising from such sources contributes to atmospheric particle concentrations. The emissions from tire and brake wear are greater in heavy-duty haul trucks when compared to

[17] RFEIR, p. 692.

[18] http://www.arb.ca.gov/msei/emfac2011-documentation-final.pdf, p. 105

[19] "Brake and Tire Wear Emissions from On-road Vehicles in MOVES2014." U.S. EPA, December 2014, *available at*: file:///C:/Users/Rob1/Downloads/420R14013.PDF, p. 2

passenger cars because the mass of the vehicle that requires slowing down or stopping is far greater.[20] This also results in the use of larger brake pads and an increase in the quantity of brake pads installed due to the number of wheels typically seen on a heavy-duty haul truck; all of these factors contribute to the rate at which particulate matter is being emitted. By not including the PM10 emissions that would occur from braking and general tire abrasion during sediment transportation, the total directly emitted PM10 emissions are vastly underestimated. An updated FEIR should be prepared to correct for this issue, and should conduct a revised health risk assessment to accurately represent the health risks posed to nearby sensitive receptors.

Failure to Incorporate Age Sensitivity Factors

The FEIR fails to take into account age specific factors for children and infants within the health risk calculation. The health risk assessment conducted in the FEIR does not (1) adjust the daily breathing rate to account for children and infants; and (2) does not apply an Age Sensitivity Factor to the risk posed to children and infants. As a result, the potential excess cancer risk posed to children and infants is not accurately represented, and the health risk assessment in the FEIR should not be relied upon to determine Project significance.

Age Sensitivity Factors

The District's failure to apply an Age Sensitivity Factor to its health risk assessment calculations violates basic health risk assessment parameters defined by California's Office of Environmental Health Hazard Assessment (OEHHA).[21] According to OEHHA's 2015 *Guidance Manual for Preparation of Health Risk Assessments*, "cancer risk is calculated by multiplying the daily inhalation or oral dose, by a cancer potency factor, the age sensitivity factor, the frequency of time spent at home, and the exposure duration divided by averaging time, to yield the excess cancer risk."[22] OEHHA explicitly states that the excess cancer risk should be calculated separately for each age grouping. According to OEHHA, "studies have shown that young animals are more sensitive than adult animals to exposure to many carcinogens…therefore, OEHHA developed age sensitivity factors (ASFs) to take into account the increased sensitivity to carcinogens during early-in-life exposure." The guidance document continues on to explain that "in the absence of chemical-specific data, OEHHA recommends a default ASF of 10 for the third trimester to age 2 years, and an ASF of 3 for ages 2 through 15 years to account for potential increased sensitivity to carcinogens during childhood."[23]

OEHHA is tasked by the State with to developing guidelines for conducting health risk assessments under the Air Toxics Hot Spots Program (Health and Safety Code Section 43360(b)(2)). OEHHA initially

[20] http://www.epa.gov/otaq/models/moves/documents/420r14013.pdf, p. 2

[21] The Project's original health risk assessment was prepared prior to the approval of OEHHA's final guidance, which was formally adopted in March of 2015. However, there were many technical support documents (TSDs) available that were published and formally adopted by OEHHA prior to the time the analysis was conducted (October 2014) which recommend the use of ASFs. Moreover, the District had the opportunity to correct this error in the RFEIR, but failed to do so.

[22] http://oehha.ca.gov/air/hot_spots/2015/2015GuidanceManual.pdf

[23] http://oehha.ca.gov/air/hot_spots/2015/2015GuidanceManual.pdf, p. 8-4

developed Technical Support Documents (TSDs) in 1999-2000 in response to this statutory requirement. Since 2000, they have revised and adopted TSDs in an effort to present updated methodologies that reflect scientific knowledge and techniques developed since the previous guidelines were prepared; in particular, to explicitly include consideration of possible differential effects on the health of infants, children and other sensitive subpopulations, in accordance with the mandate of the Children's Environmental Health Protection Act (Senate Bill 25, Escutia, Chapter 731, Statutes of 1999, Health and Safety Code Sections 39669.5 et seq.).[24]

In May 2009, OEHHA formally adopted the *Technical Support Document for Cancer Potency Factors*.[25] This TSD provides the Age Sensitivity Factors (ASF) recommended by OEHHA and the USEPA, stating that "OEHHA is applying the ASF of 10 for exposures during the third trimester of pregnancy to age 2…" and an ASF of 3 for ages 2 to 16.[26] The document continues on to state that "this timetable was also selected by U.S. EPA (2005) in their supplemental guidance for assessing early-life susceptibility to carcinogens."[27]

The FEIR failed to incorporate ASFs into the Project's health risk assessment calculations, and the RFEIR makes the same error by not revising the original HRA. Not only is this inconsistent with OEHHA's 2015 guidance manual, but it is also inconsistent with the numerous TSDs published and formally approved prior to the preparation of the FEIR by OEHHA, which all explicitly state that ASFs should be utilized when conducting health risk assessments. Omission of these factors result in an underestimation of the potential health risk posed to infants and children.

Age Specific Inhalation Rates

The District's failure to apply an age specific inhalation rate to the risk calculation is also contrary to guidelines specified by OEHHA. Exposure through inhalation is a function of the breathing rate, the exposure frequency, and the concentration of a substance in the air. According to OEHHA, "for residential exposure, the breathing rates are determined for specific age groups, so inhalation dose is calculated for each of these age groups…" The document explicitly states that "these age specific groupings are needed in order to properly use the age sensitivity factors for cancer risk assessments."[28] Therefore, the health risk assessment should not only apply age sensitivity factors to the appropriate age group, but it should also apply the appropriate inhalation rate to each age group. The Project's health risk analysis fails to include this information and, therefore, does not provide an accurate assessment of health risks from DPM emissions.

---

[24] http://www.oehha.ca.gov/air/hot_spots/tsd052909.html

[25] "Technical Support Documentation for Cancer Potency Factors." OEHHA, May 2009, *available at:* https://oehha.ca.gov/media/downloads/crnr/tsdcancerpotency.pdf

[26] http://www.oehha.ca.gov/air/hot_spots/2009/TSDCancerPotency.pdf, p. 61

[27] http://www.oehha.ca.gov/air/hot_spots/2009/TSDCancerPotency.pdf, p. 60

[28] http://oehha.ca.gov/air/hot_spots/2015/2015GuidanceManual.pdf, p. 5-23

Breathing rates for children and infants were also adopted by OEHHA prior to the time the analysis was conducted (October 2014). In August of 2012, OEHHA formally adopted the *Technical Support Document for Exposure Assessment and Stochastic Analysis*.[29] Chapter three of this document discusses "age-specific breathing rates for use in health risk assessments for short-term exposure…and for long-term daily average exposures resulting from continuous or repeated 8-hour exposure."[30] OEHHA recommends the long-term daily breathing rates in Table 3.1 of this document (see excerpt below).

Table 3.1. Recommended Point Estimates for Long-Term Daily Breathing Rates

	3rd Trimester	0<2 years	2<9 years	2<16 years	16<30 years	16<70 years
	L/kg-day					
Mean	225	658	535	452	210	185
95th Percentile	361	1090	861	745	335	290
	m^3/day					
Mean	15.3	6.2	10.7	13.3	15.0	13.9
95th Percentile	23.4	11.2	16.4	22.6	23.5	22.9

Therefore, these breathing rates should have been applied at the time the analysis was conducted, and should be applied now in an updated health risk assessment in an effort to determine the potential cancer risk posed to children and infants living near the Project site.

Updated Health Risk Assessment Indicates Cancer Risk above Thresholds

In an effort to determine a more accurate health risk, we conducted our own analysis to correct for these issues.[31] Prior to the approval of the Project, the FEIR originally proposed to remove approximately 2.9 million cubic yards of sediment from an approximately 120-acre site; this option, however, was not implemented. Instead, Alternative 3, Configuration D, Option 2, otherwise known as the "Environmentally Superior Alternative," was approved and implemented. Consistent with the sediment removal activities discussed in the Permit Application, the Environmentally Superior Alternative proposed to remove approximately 2.4 million cubic yards of sediment from an approximately 71-acre site (FEIR Table ES-2, p. ES-21). Even though the quantity of sediment removed is slightly less than the originally proposed Project and less acreage would be affected, the LACDPW indicated that the daily activity level would not be less (FEIR Appendix B, p. 40). The LACDPW still estimates a rate of removal of 7,650 cubic yards per day over a five year period and an average of 425 trucks per day travelling to the same disposal sites. Furthermore, the LACDPW expects the staffing needs and schedules to remain the same under the Environmentally Superior Alternative (FEIR Appendix B, p. 40). Due to the fact that the removal activities between the originally proposed Project and the approved Alternative will remain the same, we estimated the health risk posed to nearby sensitive

[29] http://www.oehha.ca.gov/air/hot_spots/tsd082712.html

[30] "Final Technical Support Document for Exposure Assessment and Stochastic Analysis." OEHHA, August 2012, *available at:* https://oehha.ca.gov/media/downloads/crnr/chapter32012.pdf, pp. 1

[31] Note that we calculated cancer risk using the factors identified in the FEIR / RFEIR. We did not update the FEIR's emission factors to include the tire wear and brake wear emissions, and as a result, did not update the PM10 concentrations modeled in AERMOD. Therefore, the Project's potential cancer risk is likely to be higher than the calculations included in our analysis.

receptors using the various parameters and assumptions specified in the FEIR for the originally proposed Project.

The Project is anticipated to occur in two phases: (1) Sediment Removal Phase, also referred to as the "construction" phase; and (2) Reservoir Management Phase, also referred to as Operational Maintenance (FEIR, p. ES-7). The Sediment Removal Phase "is expected to occur over the course of approximately 5 years" starting in summer of 2015 (FEIR, p. ES-7). The Reservoir Management Phase "is expected to start after the completion of the main Sediment Removal Phase" (FEIR, p. ES-7). According to the FEIR Air Quality Analysis (Appendix B), the Sediment Removal activities will occur 235 days per year for five years, and the Reservoir Management activities will occur 65 days per year (Appendix B, p. 7 of 30). Therefore, in order to accurately calculate the exposure frequency that nearby sensitive receptors will be subject to, we substituted the default exposure frequency of 350 days per year with these values.

OEHHA recommends that an exposure duration (residency time) of 30 years be used to estimate individual cancer risk for the maximally exposed individual resident (MEIR), with the exposure duration starting in the third trimester to accommodate for the increased susceptibility of exposures in early life.[32] Utilizing the information specified in the FEIR and the values recommended by OEHHA, we assumed that the proposed Project would occur over 30 years, with the Sediment Removal Phase beginning during the third trimester of a woman's pregnancy and occurring for five years, and we assumed that the Reservoir Management Phase would begin after the completion of the first phase, and would continue for an additional 25 years.

We conducted an exposure assessment utilizing a residential duration of 30 years, starting at the third trimester of a woman's pregnancy. OEHHA recommends using an exposure duration of 0.25 years for the third trimester, and an age sensitivity factor of 10.[33] From age zero to two, we applied an age sensitivity factor of ten, and from age two to sixteen, we applied an age sensitivity factor of three, per OEHHA guidance.[34] OEHHA also recommends that the 95th percentile breathing rates for each age group be used to determine risk.[35] Appendix C of the FEIR specifies that the maximally exposed individual resident (MEIR) for Haul Routes 1A/1E and for Haul Routes 1B/1F is sensitive receptor eight, with an annual concentration of 0.08 µg/m^3 during the Sediment Removal Phase, and 3.4 x 10^{-4} µg/m^3 during the Reservoir Management Phase (Appendix C, p. 14-16). The results of our calculations are summarized in the table below.

Description	**Parameter**	**Units**	**Excess Health Risk for Maximally Exposed Individual Resident**					**Total**
Age Group	-	-	3rd Trimester	0 - < 2 years	2 - < 5 years	5 - < 16 years	16 - < 30 years	-

[32] http://oehha.ca.gov/air/hot_spots/2015/2015GuidanceManual.pdf, p. 8-6
[33] http://oehha.ca.gov/air/hot_spots/2015/2015GuidanceManual.pdf p. 8-6
[34] http://oehha.ca.gov/air/hot_spots/2015/2015GuidanceManual.pdf, Table 8.3, p. 8-5
[35] http://oehha.ca.gov/air/hot_spots/2015/2015GuidanceManual.pdf p. 5-23, 5-24

Exposure Duration	ED	years	0.25	2	2.75	11	14	30
Age Sensitivity Factor	ASF	-	10	10	3	3	1	-
Daily Breathing Rate	DBR	L/kg-day	361	1090	745	745	335	-
Average Daily Dose	-	mg/kg-day	9.89E-08	2.39E-06	2.25E-06	3.82E-08	1.89E-08	-
Cancer Potency Factor for DPM	CPF	1/(mg/kg-day)	1.1	1.1	1.1	1.1	1.1	-
Exposure Frequency	EF	days/year	235	235	235	65	65	-
Averaging Time	AT	days	25550	25550	25550	25550	25550	-
Concentration	Cair	$\mu g/m^3$	0.08	0.08	0.08	0.00034	0.00034	-
Cancer Risk	-	-	7.30E-07	1.76E-05	4.97E-06	2.34E-08	4.46E-09	**2.34E-05**

The excess cancer risk at sensitive receptor location eight, during both phases of the proposed Project, would result in an estimated excess cancer risk of 23.4 in one million, which far exceeds SCAQMD's significance threshold of 10 in one million.[36] Using the same methods and input parameters as above, we calculated estimated excess cancer risks at the various residential sensitive receptors identified by the FEIR's Health Risk Assessment technical report (Appendix C). The results of these calculations are summarized in the table below.

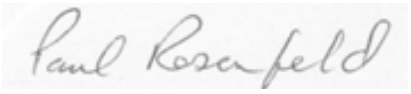
Residential Sensitive Receptor	Haul Route	Annual Concentration ($\mu g/m^3$)		Excess Cancer Risk	Excess Cancer Risk in One Million	SCAQMD Threshold	Exceed?
		Sediment Removal	Operational Maintenance				
1	2A	0.0089	0.00043	2.63E-06	2.63	10	No
2	2A	0.0089	0.00040	2.63E-06	2.63	10	No
3	2A	0.0075	0.00019	2.20E-06	2.20	10	No
4	2A	0.0077	0.00016	2.26E-06	2.26	10	No
5	2A	0.0050	0.00028	1.48E-06	1.48	10	No
6	2A	0.0007	0.00005	2.08E-07	0.21	10	No
3	1A/1E	0.0481	0.00117	1.41E-05	***14.14***	10	***Yes***
6	1A/1E	0.0340	0.00016	9.94E-06	9.94	10	No
7	1A/1E	0.0561	0.00027	1.64E-05	***16.40***	10	***Yes***
8	1A/1E	0.0800	0.00034	2.34E-05	***23.38***	10	***Yes***
9	1A/1E	0.0583	0.00012	1.70E-05	***17.03***	10	***Yes***
10	1A/1E	0.0301	0.00003	8.79E-06	8.79	10	No
11	1A/1E	0.0116	0.00001	3.39E-06	3.39	10	No
3	1B/1F	0.0481	0.00117	1.41E-05	***14.14***	10	***Yes***
6	1B/1F	0.0340	0.00016	9.94E-06	9.94	10	No
7	1B/1F	0.0561	0.00027	1.64E-05	***16.40***	10	***Yes***
8	1B/1F	0.0800	0.00034	2.34E-05	***23.38***	10	***Yes***
9	1B/1F	0.0583	0.00012	1.70E-05	***17.03***	10	***Yes***

[36] http://www.aqmd.gov/docs/default-source/ceqa/handbook/scaqmd-air-quality-significance-thresholds.pdf?sfvrsn=2

10	1B/1F	0.0303	0.00003	8.85E-06	8.85	10	No
11	1B/1F	0.0119	0.00001	3.47E-06	3.47	10	No
1	4A/B	0.0039	0.00022	1.16E-06	1.16	10	No
2	4A/B	0.0020	0.00008	5.90E-07	0.59	10	No
3	4A/B	0.0089	0.00051	2.64E-06	2.64	10	No

As is indicated in the table above, eight of the roughly 23 residential receptors identified in Appendix C of the FEIR would have an estimated cancer risk value greater than SCAQMD's significance threshold. Our analysis demonstrates that when the health risk assessment is conducted using the recommended procedures and values set forth by OEHHA, the excess cancer risk posed to the maximally exposed individual resident (MEIR) far exceeds SCAQMD's significance threshold of 10 in one million. It should be noted that our analysis does not provide the most conservative estimate. As previously stated, the emissions from tire wear and brake wear in heavy-duty trucks were not accounted for in the Health Risk Assessment. As a result, the particulate matter concentration at any given point is most likely much greater than the values calculated in the FEIR's AERMOD. An EIS should be prepared to include a revised modeling effort that accounts for the tire wear and brake wear emissions, and subsequently, an updated health risk assessment should be also prepared to include these new values and recommended OEHHA age specific factors.

Sincerely,



Paul E. Rosenfeld, Ph.D.

Matt Hagemann, P.G., C.Hg.

Hadley Nolan

EXHIBIT 1

SWAPE Technical Consultation, Data Analysis and Litigation Support for the Environment

1640 5th St.., Suite 204 Santa
Santa Monica, California 90401
Tel: (949) 887-9013
Email: mhagemann@swape.com

Matthew F. Hagemann, P.G., C.Hg., QSD, QSP

Geologic and Hydrogeologic Characterization
Industrial Stormwater Compliance
Investigation and Remediation Strategies
Litigation Support and Testifying Expert
CEQA Review

Education:

M.S. Degree, Geology, California State University Los Angeles, Los Angeles, CA, 1984.
B.A. Degree, Geology, Humboldt State University, Arcata, CA, 1982.

Professional Certifications:

California Professional Geologist
California Certified Hydrogeologist
Qualified SWPPP Developer and Practitioner

Professional Experience:

Matt has 25 years of experience in environmental policy, assessment and remediation. He spent nine years with the U.S. EPA in the RCRA and Superfund programs and served as EPA's Senior Science Policy Advisor in the Western Regional Office where he identified emerging threats to groundwater from perchlorate and MTBE. While with EPA, Matt also served as a Senior Hydrogeologist in the oversight of the assessment of seven major military facilities undergoing base closure. He led numerous enforcement actions under provisions of the Resource Conservation and Recovery Act (RCRA) while also working with permit holders to improve hydrogeologic characterization and water quality monitoring.

Matt has worked closely with U.S. EPA legal counsel and the technical staff of several states in the application and enforcement of RCRA, Safe Drinking Water Act and Clean Water Act regulations. Matt has trained the technical staff in the States of California, Hawaii, Nevada, Arizona and the Territory of Guam in the conduct of investigations, groundwater fundamentals, and sampling techniques.

Positions Matt has held include:

- Founding Partner, Soil/Water/Air Protection Enterprise (SWAPE) (2003 – present);
- Geology Instructor, Golden West College, 2010 – 2014;
- Senior Environmental Analyst, Komex H2O Science, Inc. (2000 -- 2003);

- Executive Director, Orange Coast Watch (2001 – 2004);
- Senior Science Policy Advisor and Hydrogeologist, U.S. Environmental Protection Agency (1989–1998);
- Hydrogeologist, National Park Service, Water Resources Division (1998 – 2000);
- Adjunct Faculty Member, San Francisco State University, Department of Geosciences (1993 – 1998);
- Instructor, College of Marin, Department of Science (1990 – 1995);
- Geologist, U.S. Forest Service (1986 – 1998); and
- Geologist, Dames & Moore (1984 – 1986).

Senior Regulatory and Litigation Support Analyst:

With SWAPE, Matt's responsibilities have included:

- Lead analyst and testifying expert in the review of over 100 environmental impact reports since 2003 under CEQA that identify significant issues with regard to hazardous waste, water resources, water quality, air quality, Valley Fever, greenhouse gas emissions, and geologic hazards. Make recommendations for additional mitigation measures to lead agencies at the local and county level to include additional characterization of health risks and implementation of protective measures to reduce worker exposure to hazards from toxins and Valley Fever.
- Stormwater analysis, sampling and best management practice evaluation at industrial facilities.
- Manager of a project to provide technical assistance to a community adjacent to a former Naval shipyard under a grant from the U.S. EPA.
- Technical assistance and litigation support for vapor intrusion concerns.
- Lead analyst and testifying expert in the review of environmental issues in license applications for large solar power plants before the California Energy Commission.
- Manager of a project to evaluate numerous formerly used military sites in the western U.S.
- Manager of a comprehensive evaluation of potential sources of perchlorate contamination in Southern California drinking water wells.
- Manager and designated expert for litigation support under provisions of Proposition 65 in the review of releases of gasoline to sources drinking water at major refineries and hundreds of gas stations throughout California.
- Expert witness on two cases involving MTBE litigation.
- Expert witness and litigation support on the impact of air toxins and hazards at a school.
- Expert witness in litigation at a former plywood plant.

With Komex H2O Science Inc., Matt's duties included the following:

- Senior author of a report on the extent of perchlorate contamination that was used in testimony by the former U.S. EPA Administrator and General Counsel.
- Senior researcher in the development of a comprehensive, electronically interactive chronology of MTBE use, research, and regulation.
- Senior researcher in the development of a comprehensive, electronically interactive chronology of perchlorate use, research, and regulation.
- Senior researcher in a study that estimates nationwide costs for MTBE remediation and drinking water treatment, results of which were published in newspapers nationwide and in testimony against provisions of an energy bill that would limit liability for oil companies.
- Research to support litigation to restore drinking water supplies that have been contaminated by MTBE in California and New York.

- Expert witness testimony in a case of oil production-related contamination in Mississippi.
- Lead author for a multi-volume remedial investigation report for an operating school in Los Angeles that met strict regulatory requirements and rigorous deadlines.

- Development of strategic approaches for cleanup of contaminated sites in consultation with clients and regulators.

Executive Director:

As Executive Director with Orange Coast Watch, Matt led efforts to restore water quality at Orange County beaches from multiple sources of contamination including urban runoff and the discharge of wastewater. In reporting to a Board of Directors that included representatives from leading Orange County universities and businesses, Matt prepared issue papers in the areas of treatment and disinfection of wastewater and control of the discharge of grease to sewer systems. Matt actively participated in the development of countywide water quality permits for the control of urban runoff and permits for the discharge of wastewater. Matt worked with other nonprofits to protect and restore water quality, including Surfrider, Natural Resources Defense Council and Orange County CoastKeeper as well as with business institutions including the Orange County Business Council.

Hydrogeology:

As a Senior Hydrogeologist with the U.S. Environmental Protection Agency, Matt led investigations to characterize and cleanup closing military bases, including Mare Island Naval Shipyard, Hunters Point Naval Shipyard, Treasure Island Naval Station, Alameda Naval Station, Moffett Field, Mather Army Airfield, and Sacramento Army Depot. Specific activities were as follows:

- Led efforts to model groundwater flow and contaminant transport, ensured adequacy of monitoring networks, and assessed cleanup alternatives for contaminated sediment, soil, and groundwater.
- Initiated a regional program for evaluation of groundwater sampling practices and laboratory analysis at military bases.
- Identified emerging issues, wrote technical guidance, and assisted in policy and regulation development through work on four national U.S. EPA workgroups, including the Superfund Groundwater Technical Forum and the Federal Facilities Forum.

At the request of the State of Hawaii, Matt developed a methodology to determine the vulnerability of groundwater to contamination on the islands of Maui and Oahu. He used analytical models and a GIS to show zones of vulnerability, and the results were adopted and published by the State of Hawaii and County of Maui.

As a hydrogeologist with the EPA Groundwater Protection Section, Matt worked with provisions of the Safe Drinking Water Act and NEPA to prevent drinking water contamination. Specific activities included the following:

- Received an EPA Bronze Medal for his contribution to the development of national guidance for the protection of drinking water.
- Managed the Sole Source Aquifer Program and protected the drinking water of two communities through designation under the Safe Drinking Water Act. He prepared geologic reports, conducted public hearings, and responded to public comments from residents who were very concerned about the impact of designation.

- Reviewed a number of Environmental Impact Statements for planned major developments, including large hazardous and solid waste disposal facilities, mine reclamation, and water transfer.

Matt served as a hydrogeologist with the RCRA Hazardous Waste program. Duties were as follows:

- Supervised the hydrogeologic investigation of hazardous waste sites to determine compliance with Subtitle C requirements.
- Reviewed and wrote "part B" permits for the disposal of hazardous waste.
- Conducted RCRA Corrective Action investigations of waste sites and led inspections that formed the basis for significant enforcement actions that were developed in close coordination with U.S. EPA legal counsel.
- Wrote contract specifications and supervised contractor's investigations of waste sites.

With the National Park Service, Matt directed service-wide investigations of contaminant sources to prevent degradation of water quality, including the following tasks:

- Applied pertinent laws and regulations including CERCLA, RCRA, NEPA, NRDA, and the Clean Water Act to control military, mining, and landfill contaminants.
- Conducted watershed-scale investigations of contaminants at parks, including Yellowstone and Olympic National Park.
- Identified high-levels of perchlorate in soil adjacent to a national park in New Mexico and advised park superintendent on appropriate response actions under CERCLA.
- Served as a Park Service representative on the Interagency Perchlorate Steering Committee, a national workgroup.
- Developed a program to conduct environmental compliance audits of all National Parks while serving on a national workgroup.
- Co-authored two papers on the potential for water contamination from the operation of personal watercraft and snowmobiles, these papers serving as the basis for the development of nation-wide policy on the use of these vehicles in National Parks.
- Contributed to the Federal Multi-Agency Source Water Agreement under the Clean Water Action Plan.

Policy:

Served senior management as the Senior Science Policy Advisor with the U.S. Environmental Protection Agency, Region 9. Activities included the following:

- Advised the Regional Administrator and senior management on emerging issues such as the potential for the gasoline additive MTBE and ammonium perchlorate to contaminate drinking water supplies.
- Shaped EPA's national response to these threats by serving on workgroups and by contributing to guidance, including the Office of Research and Development publication, Oxygenates in Water: Critical Information and Research Needs.
- Improved the technical training of EPA's scientific and engineering staff.
- Earned an EPA Bronze Medal for representing the region's 300 scientists and engineers in negotiations with the Administrator and senior management to better integrate scientific principles into the policy-making process.
- Established national protocol for the peer review of scientific documents.

Geology:

With the U.S. Forest Service, Matt led investigations to determine hillslope stability of areas proposed for timber harvest in the central Oregon Coast Range. Specific activities were as follows:

- Mapped geology in the field, and used aerial photographic interpretation and mathematical models to determine slope stability.
- Coordinated his research with community members who were concerned with natural resource protection.
- Characterized the geology of an aquifer that serves as the sole source of drinking water for the city of Medford, Oregon.

As a consultant with Dames and Moore, Matt led geologic investigations of two contaminated sites (later listed on the Superfund NPL) in the Portland, Oregon, area and a large hazardous waste site in eastern Oregon. Duties included the following:

- Supervised year-long effort for soil and groundwater sampling.
- Conducted aquifer tests.
- Investigated active faults beneath sites proposed for hazardous waste disposal.

Teaching:

From 1990 to 1998, Matt taught at least one course per semester at the community college and university levels:

- At San Francisco State University, held an adjunct faculty position and taught courses in environmental geology, oceanography (lab and lecture), hydrogeology, and groundwater contamination.
- Served as a committee member for graduate and undergraduate students.
- Taught courses in environmental geology and oceanography at the College of Marin.

Matt taught physical geology (lecture and lab and introductory geology at Golden West College in Huntington Beach, California from 2010 to 2014.

Invited Testimony, Reports, Papers and Presentations:

Hagemann, M.F., 2008. Disclosure of Hazardous Waste Issues under CEQA. Presentation to the Public Environmental Law Conference, Eugene, Oregon.

Hagemann, M.F., 2008. Disclosure of Hazardous Waste Issues under CEQA. Invited presentation to U.S. EPA Region 9, San Francisco, California.

Hagemann, M.F., 2005. Use of Electronic Databases in Environmental Regulation, Policy Making and Public Participation. Brownfields 2005, Denver, Coloradao.

Hagemann, M.F., 2004. Perchlorate Contamination of the Colorado River and Impacts to Drinking Water in Nevada and the Southwestern U.S. Presentation to a meeting of the American Groundwater Trust, Las Vegas, NV (served on conference organizing committee).

Hagemann, M.F., 2004. Invited testimony to a California Senate committee hearing on air toxins at schools in Southern California, Los Angeles.

Brown, A., Farrow, J., Gray, A. and **Hagemann, M.**, 2004. An Estimate of Costs to Address MTBE Releases from Underground Storage Tanks and the Resulting Impact to Drinking Water Wells. Presentation to the Ground Water and Environmental Law Conference, National Groundwater Association.

Hagemann, M.F., 2004. Perchlorate Contamination of the Colorado River and Impacts to Drinking Water in Arizona and the Southwestern U.S. Presentation to a meeting of the American Groundwater Trust, Phoenix, AZ (served on conference organizing committee).

Hagemann, M.F., 2003. Perchlorate Contamination of the Colorado River and Impacts to Drinking Water in the Southwestern U.S. Invited presentation to a special committee meeting of the National Academy of Sciences, Irvine, CA.

Hagemann, M.F., 2003. Perchlorate Contamination of the Colorado River. Invited presentation to a tribal EPA meeting, Pechanga, CA.

Hagemann, M.F., 2003. Perchlorate Contamination of the Colorado River. Invited presentation to a meeting of tribal repesentatives, Parker, AZ.

Hagemann, M.F., 2003. Impact of Perchlorate on the Colorado River and Associated Drinking Water Supplies. Invited presentation to the Inter-Tribal Meeting, Torres Martinez Tribe.

Hagemann, M.F., 2003. The Emergence of Perchlorate as a Widespread Drinking Water Contaminant. Invited presentation to the U.S. EPA Region 9.

Hagemann, M.F., 2003. A Deductive Approach to the Assessment of Perchlorate Contamination. Invited presentation to the California Assembly Natural Resources Committee.

Hagemann, M.F., 2003. Perchlorate: A Cold War Legacy in Drinking Water. Presentation to a meeting of the National Groundwater Association.

Hagemann, M.F., 2002. From Tank to Tap: A Chronology of MTBE in Groundwater. Presentation to a meeting of the National Groundwater Association.

Hagemann, M.F., 2002. A Chronology of MTBE in Groundwater and an Estimate of Costs to Address Impacts to Groundwater. Presentation to the annual meeting of the Society of Environmental Journalists.

Hagemann, M.F., 2002. An Estimate of the Cost to Address MTBE Contamination in Groundwater (and Who Will Pay). Presentation to a meeting of the National Groundwater Association.

Hagemann, M.F., 2002. An Estimate of Costs to Address MTBE Releases from Underground Storage Tanks and the Resulting Impact to Drinking Water Wells. Presentation to a meeting of the U.S. EPA and State Underground Storage Tank Program managers.

Hagemann, M.F., 2001. From Tank to Tap: A Chronology of MTBE in Groundwater. Unpublished report.

Hagemann, M.F., 2001. Estimated Cleanup Cost for MTBE in Groundwater Used as Drinking Water. Unpublished report.

Hagemann, M.F., 2001. Estimated Costs to Address MTBE Releases from Leaking Underground Storage Tanks. Unpublished report.

Hagemann, M.F., and VanMouwerik, M., 1999. Potential Water Quality Concerns Related to Snowmobile Usage. Water Resources Division, National Park Service, Technical Report.

VanMouwerik, M. and **Hagemann, M.F**. 1999, Water Quality Concerns Related to Personal Watercraft Usage. Water Resources Division, National Park Service, Technical Report.

Hagemann, M.F., 1999, Is Dilution the Solution to Pollution in National Parks? The George Wright Society Biannual Meeting, Asheville, North Carolina.

Hagemann, M.F., 1997, The Potential for MTBE to Contaminate Groundwater. U.S. EPA Superfund Groundwater Technical Forum Annual Meeting, Las Vegas, Nevada.

Hagemann, M.F., and Gill, M., 1996, Impediments to Intrinsic Remediation, Moffett Field Naval Air Station, Conference on Intrinsic Remediation of Chlorinated Hydrocarbons, Salt Lake City.

Hagemann, M.F., Fukunaga, G.L., 1996, The Vulnerability of Groundwater to Anthropogenic Contaminants on the Island of Maui, Hawaii. Hawaii Water Works Association Annual Meeting, Maui, October 1996.

Hagemann, M. F., Fukanaga, G. L., 1996, Ranking Groundwater Vulnerability in Central Oahu, Hawaii. Proceedings, Geographic Information Systems in Environmental Resources Management, Air and Waste Management Association Publication VIP-61.

Hagemann, M.F., 1994. Groundwater Characterization and Cleanup at Closing Military Bases in California. Proceedings, California Groundwater Resources Association Meeting.

Hagemann, M.F. and Sabol, M.A., 1993. Role of the U.S. EPA in the High Plains States Groundwater Recharge Demonstration Program. Proceedings, Sixth Biennial Symposium on the Artificial Recharge of Groundwater.

Hagemann, M.F., 1993. U.S. EPA Policy on the Technical Impracticability of the Cleanup of DNAPL-contaminated Groundwater. California Groundwater Resources Association Meeting.

Hagemann, M.F., 1992. Dense Nonaqueous Phase Liquid Contamination of Groundwater: An Ounce of Prevention... Proceedings, Association of Engineering Geologists Annual Meeting, v. 35.

Other Experience:

Selected as subject matter expert for the California Professional Geologist licensing examination, 2009-2011.

EXHIBIT 2

SWAPE Technical Consultation, Data Analysis and Litigation Support for the Environment

SOIL WATER AIR PROTECTION ENTERPRISE
1640 Fifth Street, Suite 204
Santa Monica, California 90401
Attn: Paul Rosenfeld, Ph.D.
Mobil: (310) 795-2335
Office: (310) 434-0110
Fax: (310) 434-0011
Email: prosenfeld@swape.com

Paul Rosenfeld, Ph.D.

Chemical Fate and Transport & Air Dispersion Modeling

Principal Environmental Chemist

Risk Assessment & Remediation Specialist

Education

Ph.D. Soil Chemistry, University of Washington, 1999. Dissertation on VOC filtration.

M.S. Environmental Science, U.C. Berkeley, 1995. Thesis on organic waste economics.

B.A. Environmental Studies, U.C. Santa Barbara, 1991. Thesis on wastewater treatment.

Professional Experience

Dr. Rosenfeld is the Co-Founder and Principal Environmental Chemist at Soil Water Air Protection Enterprise (SWAPE). His focus is the fate and transport of environmental contaminants, risk assessment, and ecological restoration. His project experience ranges from monitoring and modeling of pollution sources as they relate to human and ecological health. Dr. Rosenfeld has investigated and designed remediation programs and risk assessments for contaminated sites containing, petroleum, MtBE and fuel oxygenates, chlorinated solvents, pesticides, radioactive waste, PCBs, PAHs, dioxins, furans, volatile organics, semi-volatile organics, perchlorate, heavy metals, asbestos, PFOA, unusual polymers, and odor. Significant projects performed by Dr. Rosenfeld include the following:

Litigation Support

Client: Missouri Department of Natural Resources (Jefferson City, Missouri)
Serving as an expert in evaluating air pollution and odor emissions from a Republic Landfill in St. Louis, Missouri. Conducted. Project manager overseeing daily, weekly and comprehensive sampling of odor and chemicals.

Client: Louisiana Department of Transportation and Development (Baton Rouge, Louisiana)
Serving as an expert witness, conducting groundwater modeling of an ethylene dichloride DNAPL and soluble plume resulting from spill caused by Conoco Phillips.

Client: Missouri Department of Natural Resources (St. Louis, Missouri)
Serving as a consulting expert and potential testifying expert regarding a landfill fire directly adjacent to another landfill containing radioactive waste. Implemented an air monitoring program testing for over 100 different compounds using approximately 12 different analytical methods.

Client: Baron & Budd, P.C. (Dallas, Texas) and Weitz & Luxeinberg (New York, New York)
Served as a consulting expert in MTBE Federal Multi District Litigation (MDL) in New York. Consolidated ground water data, created maps for test cases, constructed damage model, evaluated taste and odor threshold levels. Resulted in a settlement of over $440 million.

Client: The Buzbee Law Firm (Houston, Texas)
Served as a as an expert in ongoing litigation involving over 50,000+ plaintiffs who are seeking compensation for chemical exposure and reduction in property value resulting from chemicals released from the BP facility.

Client: Environmental Litigation Group (Birmingham, Alabama)
Serving as an expert on property damage, medical monitoring and toxic tort claims that have been filed on behalf of over 13,000 plaintiffs who were exposed to PCBs and dioxins/furans resulting from emissions from Monsanto and Cerro Copper's operations in Sauget, Illinois. Developed AERMOD models to demonstrate plaintiff's exposure.

Client: Baron & Budd P.C. (Dallas Texas) and Korein Tillery (St. Louis, Missouri)
Served as a consulting expert for a Class Action defective product claim filed in Madison County, Illinois against Syngenta and five other manufacturers for atrazine. Evaluated health issues associated with atrazine and deterimied treatment cost for filtration of public drinking water supplies. Resulted in $105 million dollar settlement.

Client: The Buzbee Law Firm (Houston, Texas)
Served as a consulting expert in catalyst release and refinery emissions cases against the BP Refinery in Texas City. A jury verdict for 10 employees exposed to catalyst via BP's irresponsible behavior.

Client: Baron & Budd, P.C. (Dallas, Texas)
Served as a consulting expert to calculate the Maximum Allowable Dose Level (MADL) and No Significant Risk Level (NSRL), based on Cal EPA and OEHHA guidelines, for Polychlorinated Biphenyls (PCBs) in fish oil dietary supplements.

Client: Girardi Keese (Los Angeles, California)
Served as an expert testifying on hydrocarbon exposure of a woman who worked on a fuel barge operated by Chevron. Demonstrated that the plaintiff was exposed to excessive amounts of benzene.

Client: Mason & Cawood (Annapolis, Maryland) and Girardi & Keese (Los Angeles, California)
Serving as an expert consultant on the Battlefield Golf Club fly ash disposal site in Chesapeake, VA, where arsenic, other metals and radionuclides are leaching into groundwater, and ash is blowing off-site onto the surrounding communities.

Client: California Earth Mineral Corporation (Culver City, California)
Evaluating the montmorillonite clay deposit located near El Centro, California. Working as a Defense Expert representing an individual who owns a 2,500 acre parcel that will potentially be seized by the United States Navy via eminent domain.

Client: Matthews & Associates (Houston, Texas)
Serving as an expert witness, preparing air model demonstrating residential exposure via emissions from fracking in natural gas wells in Duncan, Texas.

Client: Baron & Budd P.C. (Dallas, Texas) and Korein Tillery (St. Louis, Missouri)
Served as a consulting expert for analysis of private wells relating to litigation regarding compensation of private well owners for MTBE testing. Coordinated data acquisition and GIS analysis evaluating private well proximity to leaking underground storage tanks.

Client: Lurie & Park LLP (Los Angeles, California)
Served as an expert witness evaluating a vapor intrusion toxic tort case that resulted in a settlement. The Superfund site is a 4 ½ mile groundwater plume of chlorinated solvents in Whittier, California.

Client: Mason & Cawood (Annapolis, Maryland)
Evaluated data from the Hess Gasoline Station in northern Baltimore, Maryland that had a release resulting in flooding of plaintiff's homes with gasoline-contaminated water, foul odor, and biofilm growth.

Client: The Buzbee Law Firm (Houston, Texas)
Evaluated air quality resulting from grain processing emissions in Muscatine, Iowa.

Client: Anderson Kill & Olick, P.C. (Ventura, California)
Evaluated historical exposure and lateral and vertical extent of contamination resulting from a ~150 million gallon Exxon Mobil tank farm located near Watts, California.

Client: Packard Law Firm (Petaluma, California)
Served as an expert witness, evaluated lead in Proposition 65 Case where various products were found to have elevated lead levels.

Client: The Buzbee Law Firm (Houston, Texas)
Evaluated data resulting from an oil spill in Port Arthur, Texas.

Client: Nexsen Pruet, LLC (Charleston, South Carolina)
Serving as expert in chlorine exposure in a railroad tank car accident where approximately 120,000 pounds of chlorine were released.

Client: Girardi & Keese (Los Angeles, California)
Serving as an expert investigating hydrocarbon exposure and property damage for ~600 individuals and ~280 properties in Carson, California where homes were constructed above a large tank farm formerly owned by Shell.

Client: Brent Coon Law Firm (Cleveland, Ohio)
Served as an expert, calculating an environmental exposure to benzene, PAHs, and VOCs from a Chevron Refinery in Hooven, Ohio. Conducted AERMOD modeling to determine cumulative dose.

Client: Lundy Davis (Lake Charles, Louisiana)
Served as consulting expert on an oil field case representing the lease holder of a contaminated oil field. Conducted field work evaluating oil field contamination in Sulphur, Louisiana. Property is owned by Conoco Phillips, but leased by Yellow Rock, a small oil firm.

Client: Cox Cox Filo (Lake Charles, Louisiana)
Served as testifying expert on a multimillion gallon oil spill in Lake Charles which occurred on June 19, 2006, resulting in hydrocarbon vapor exposure to hundreds of workers and residents. Prepared air model and calculated exposure concentration. Demonstrated that petroleum odor alone can result in significant health harms.

Client: Cotchett Pitre & McCarthy (San Francisco, California)
Served as testifying expert representing homeowners who unknowingly purchased homes built on an old oil field in Santa Maria, California. Properties have high concentrations of petroleum hydrocarbons in subsurface soils resulting in diminished property value.

Client: Law Offices Of Anthony Liberatore P.C. (Los Angeles, California)
Served as testifying expert representing individuals who rented homes on the Inglewood Oil Field in California. Plaintiffs were exposed to hydrocarbon contaminated water and air, and experienced health harms associated with the petroleum exposure.

Client: Orange County District Attorney (Orange County, California)
Coordinated a review of 143 ARCO gas stations in Orange County to assist the District Attorney's prosecution of CCR Title 23 and California Health and Safety Code violators.

Client: Environmental Litigation Group (Birmingham, Alabama)
Served as a testifying expert in a health effects case against ABC Coke/Drummond Company for polluting a community with PAHs, benzene, particulate matter, heavy metals, and coke oven emissions. Created air dispersion models and conducted attic dust sampling, exposure modeling, and risk assessment for plaintiffs.

Client: Masry & Vitatoe (Westlake Village, California), Engstrom Lipscomb Lack (Los Angeles, Califronia) and Baron & Budd P.C. (Dallas, Texas)
Served as a consulting expert in Proposition 65 lawsuit filed against major oil companies for benzene and toluene releases from gas stations and refineries resulting in contaminated groundwater. Settlement included over $110 million dollars in injunctive relief.

Client: Tommy Franks Law Firm (Austin, Texas)
Served as expert evaluating groundwater contamination which resulted from the hazardous waste injection program and negligent actions of Morton Thiokol and Rohm Hass. Evaluated drinking water contamination and community exposure.

Client: Baron & Budd P.C. (Dallas, Texas) and Sher Leff (San Francisco, California)
Served as consulting expert for several California cities that filed defective product cases against Dow Chemical and Shell for 1,2,3-trichloropropane groundwater contamination. Generated maps showing capture zones of impacted wells for various municipalities.

Client: Weitz & Luxenberg (New York, New York)
Served as expert on Property Damage and Nuisance claims resulting from emissions from the Countywide Landfill in Ohio. The landfill had an exothermic reaction or fire resulting from aluminum dross dumping, and the EPA fined the landfill $10,000,000 dollars.

Client: Baron & Budd P.C. (Dallas, Texas)
Served as a consulting expert for a groundwater contamination case in Pensacola, Florida where fluorinated compounds contaminated wells operated by Escambia County.

Client: Environmental Litigation Group (Birmingham, Alabama)
Served as an expert on groundwater case where Exxon Mobil and Helena Chemical released ethylene dichloride into groundwater resulting in a large plume. Prepared report on the appropriate treatment technology and cost, and flaws with the proposed on-site remediation.

Client: Environmental Litigation Group (Birmingham, Alabama)
Served as an expert on air emissions released when a Bartlo Packaging Incorporated facility in West Helena, Arkansas exploded resulting in community exposure to pesticides and smoke from combustion of pesticides.

Client: Omara & Padilla (San Diego, California)
Served as a testifying expert on nuisance case against Nutro Dogfood Company that constructed a large dog food processing facility in the middle of a residential community in Victorville, California with no odor control devices. The facility has undergone significant modifications, including installation of a regenerative thermal oxidizer.

Client: Environmental Litigation Group (Birmingham, Alabama)
Serving as an expert on property damage and medical monitoring claims that have been filed against International Paper resulting from chemical emissions from facilities located in Bastrop, Louisiana; Prattville, Alabama; and Georgetown, South Carolina.

Client: Estep and Shafer L.C. (Kingwood, West Virginia)
Served as expert calculating acid emissions doses to residents resulting from coal-fired power plant emissions in West V
irginia using various air models.

Client: Watts Law Firm (Austin, Texas), Woodfill & Pressler (Houston, Texas) and Woska & Associates (Oklahoma City, Oklahoma)
Served as testifying expert on community and worker exposure to CCA, creosote, PAHs, and dioxins/furans from a BNSF and Koppers Facility in Somerville, Texas. Conducted field sampling, risk assessment, dose assessment and air modeling to quantify exposure to workers and community members.

Client: Environmental Litigation Group (Birmingham, Alabama)
Served as expert regarding community exposure to CCA, creosote, PAHs, and dioxins/furans from a Louisiana Pacific wood treatment facility in Florala, Alabama. Conducted blood sampling and environmental sampling to determine environmental exposure to dioxins/furans and PAHs.

Client: Sanders Law Firm (Colorado Springs, Colorado) and Vamvoras & Schwartzberg (Lake Charles, Louisiana)
Served as an expert calculating chemical exposure to over 500 workers from large ethylene dichloride spill in Lake Charles, Louisiana at the Conoco Phillips Refinery.

Client: Baron & Budd P.C. (Dallas, Texas)
Served as consulting expert in a defective product lawsuit against Dow Agroscience focusing on Clopyralid, a recalcitrant herbicide that damaged numerous compost facilities across the United States.

Client: Sullivan Papain Block McGrath & Cannavo (New York, New York) and The Cochran Firm (Dothan, Mississippi)

Served as an expert regarding community exposure to metals, PAHs PCBs, and dioxins/furans from the burning of Ford paint sludge and municipal solid waste in Ringwood, New Jersey.

Client: Rose, Klein & Marias LLP (Los Angeles, California)
Served as an expert in 55 Proposition 65 cases against individual facilities in the Port of Los Angeles and Port of Long Beach. Prepared air dispersion and risk models to demonstrate that each facility emits diesel particulate matter that results in risks exceeding 1/100,000, hence violating the Proposition 65 Statute.

Client: Rose, Klein & Marias LLP (Los Angeles, California) and Environmental Law Foundation (San Francisco, California)
Served as an expert in a Proposition 65 case against potato chip manufacturers. Conducted an analysis of several brands of potato chips for acrylamide concentrations and found that all samples exceeded Proposition 65 No Significant Risk Levels.

Client: Gonzales & Robinson (Westlake Village, California)
Served as a testifying expert in a toxic tort case against Chevron (Ortho) for allowing a community to be contaminated with lead arsenate pesticide. Created air dispersion and soil vadose zone transport models, and evaluated bioaccumulation of lead arsenate in food.

Client: Environment Now (Santa Monica, California)
Served as expert for Environment Now to convince the State of California to file a nuisance claim against automobile manufactures to recover MediCal damages from expenditures on asthma-related health care costs.

Client: Trutanich Michell (Long Beach, California)
Served as expert representing San Pedro Boat Works in the Port of Los Angeles. Prepared air dispersion, particulate air dispersion, and storm water discharge models to demonstrate that Kaiser Bulk Loading is responsible for copper concentrate accumulating in the bay sediment.

Client: Azurix of North America (Fort Myers, Florida)
Provided expert opinions, reports and research pertaining to a proposed County Ordinance requiring biosolids applicators to measure VOC and odor concentrations at application sites' boundaries.

Client: MCP Polyurethane (Pittsburg, Kansas)
Provided expert opinions and reports regarding metal-laden landfill runoff that damaged a running track by causing the reversion of the polyurethane due to its catalytic properties.

Risk Assessment And Air Modeling

Client: Hager, Dewick & Zuengler, S.C. (Green Bay, Wisconsin)
Conducted odor audit of rendering facility in Green Bay, Wisconsin.

Client: ABT-Haskell (San Bernardino, California)
Prepared air dispersion model for a proposed state-of-the-art enclosed compost facility. Prepared a traffic analysis and developed odor detection limits to predict 1, 8, and 24-hour off-site concentrations of sulfur, ammonia, and amine.

Client: Jefferson PRP Group (Los Angeles, California)
Evaluated exposure pathways for chlorinated solvents and hexavalent chromium for human health risk assessment of Los Angeles Academy (formerly Jefferson New Middle School) operated by Los Angeles Unified School District.

Client: Covanta (Susanville, California)
Prepared human health risk assessment for Covanta Energy focusing on agricultural worker exposure to caustic fertilizer.

Client: CIWMB (Sacramento, California)
Used dispersion models to estimate traveling distance and VOC concentrations downwind from a composting facility for the California Integrated Waste Management Board.

Client: Carboquimeca (Bogotá, Columbia)
Evaluated exposure pathways for human health risk assessment for a confidential client focusing on significant concentrations of arsenic and chlorinated solvents present in groundwater used for drinking water.

Client: Navy Base Realignment and Closure Team (Treasure Island, California)
Used Johnson-Ettinger model to estimate indoor air PCB concentrations and compared estimated values with empirical data collected in homes.

Client: San Diego State University (San Diego, California)
Measured CO_2 flux from soils amended with different quantities of biosolids compost at Camp Pendleton to determine CO_2 credit values for coastal sage under fertilized and non-fertilized conditions.

Client: Navy Base Realignment and Closure Team (MCAS Tustin, California)
Evaluated cumulative risk of a multiple pathway scenario for a child resident and a construction worker. Evaluated exposure to air and soil via particulate and vapor inhalation, incidental soil ingestion, and dermal contact with soil.

Client: MCAS Miramar (San Diego, California)
Evaluated exposure pathways of metals in soil by comparing site data to background data. Risk assessment incorporated multiple pathway scenarios assuming child resident and construction worker particulate and vapor inhalation, soil ingestion, and dermal soil contact.

Client: Naval Weapons Station (Seal Beach, California)
Used a multiple pathway model to generate dust emission factors from automobiles driving on dirt roads. Calculated bioaccumulation of metals, PCBs, dioxin congeners and pesticides to estimate human and ecological risk.

Client: King County, Douglas County (Washington State)
Measured PM_{10} and $PM_{2.5}$ emissions from windblown soil treated with biosolids and a polyacrylamide polymer in Douglas County, Washington. Used Pilat Mark V impactor for measurement and compared data to EPA particulate regulations.

Client: King County (Seattle, Washington)
Created emission inventory for several compost and wastewater facilities comparing VOC, particulate, and fungi concentrations to NIOSH values estimating risk to workers and individuals at neighboring facilities.

Air Pollution Investigation and Remediation

Client: Republic Landfill (Santa Clarita, California)
Managed a field investigation of odor around a landfill during 30+ events. Used hedonic tone, butanol scale, dilution-to-threshold values, and odor character to evaluate odor sources and character and intensity.

Client: California Biomass (Victorville, California)
Managed a field investigation of odor around landfill during 9+ events. Used hedonic tone, butanol scale, dilution-to-threshold values, and odor character to evaluate odor sources, character and intensity.

Client: ABT-Haskell (Redlands, California)
Assisted in permitting a compost facility that will be completely enclosed with a complex scrubbing system using acid scrubbers, base scrubbers, biofilters, heat exchangers and chlorine to reduce VOC emissions by 99 percent.

Client: Synagro (Corona, California)
Designed and monitored 30-foot by 20-foot by 6-foot biofilter for VOC control at an industrial composting facility in Corona, California to reduce VOC emissions by 99 percent.

Client: Jeff Gage (Tacoma, Washington)
Conducted emission inventory at industrial compost facility using GC/MS analyses for VOCs. Evaluated effectiveness of VOC and odor control systems and estimated human health risk.

Client: Daishowa America (Port Angeles Mill, Washington)
Analyzed industrial paper sludge and ash for VOCs, heavy metals and nutrients to develop a land application program. Metals were compared to federal guidelines to determine maximum allowable land application rates.

Client: Jeff Gage (Puyallup, Washington)
Measured effectiveness of biofilters at composting facility and conducted EPA dispersion models to estimate traveling distance of odor and human health risk from exposure to volatile organics.

Surface Water, Groundwater, and Wastewater Investigation/Remediation

Client: Confidential (Downey, California)
Managed groundwater investigation to determine horizontal extent of 1,000 foot TCE plume associated with a metal finishing shop.

Client: Confidential (West Hollywood, California)
Designing soil vapor extraction system that is currently being installed for confidential client. Managing groundwater investigation to determine horizontal extent of TCE plume associated with dry cleaning.

Client: Synagro Technologies (Sacramento, California)
Managed groundwater investigation to determine if biosolids application impacted salinity and nutrient concentrations in groundwater.

Client: Navy Base Realignment and Closure Team (Treasure Island, California)
Assisted in the design and remediation of PCB, chlorinated solvent, hydrocarbon and lead contaminated groundwater and soil on Treasure Island. Negotiated screening levels with DTSC and Water Board. Assisted in the preparation of FSP/QAPP, RI/FS, and RAP documents and assisted in CEQA document preparation.

Client: Navy Base Realignment and Closure Team (MCAS Tustin, California)
Assisted in the design of groundwater monitoring systems for chlorinated solvents at Tustin MCAS. Contributed to the preparation of FS for groundwater treatment.

Client: Mission Cleaning Facility (Salinas, California)
Prepared a RAP and cost estimate for using an oxygen releasing compound (ORC) and molasses to oxidize diesel fuel in soil and groundwater at Mission Cleaning in Salinas.

Client: King County (Washington)
Established and monitored experimental plots at a US EPA Superfund Site in wetland and upland mine tailings contaminated with zinc and lead in Smelterville, Idaho. Used organic matter and pH adjustment for wetland remediation and erosion control.

Client: City of Redmond (Richmond, Washington)
Collected storm water from compost-amended and fertilized turf to measure nutrients in urban runoff. Evaluated effectiveness of organic matter-lined detention ponds on reduction of peak flow during storm events. Drafted compost amended landscape installation guidelines to promote storm water detention and nutrient runoff reduction.

Client: City of Seattle (Seattle, Washington)
Measured VOC emissions from Renton wastewater treatment plant in Washington. Ran GC/MS, dispersion models, and sensory panels to characterize, quantify, control and estimate risk from VOCs.

Client: Plumas County (Quincy, California)

Installed wetland to treat contaminated water containing 1% copper in an EPA Superfund site. Revegetated 10 acres of acidic and metal laden sand dunes resulting from hydraulic mining. Installed and monitored piezometers in wetland estimating metal loading.

Client: Adams Egg Farm (St. Kitts, West Indies)
Designed, constructed, and maintained 3 anaerobic digesters at Springfield Egg Farm, St. Kitts. Digesters treated chicken excrement before effluent discharged into sea. Chicken waste was converted into methane cooking gas.

Client: BLM (Kremmling, Colorado)
Collected water samples for monitoring program along upper stretch of the Colorado River. Rafted along river and protected water quality by digging and repairing latrines.

Soil Science and Restoration Projects

Client: Hefner, Stark & Marois, LLP (Sacramento, California)
Facilitated in assisting Hefner, Stark & Marois, LLP in working with the Regional Water Quality board to determine how to utilize Calcium Participate as a by-product of processing sugar beets.

Client: Kinder Morgan (San Diego County, California)
Designed and monitored the restoration of a 110-acre project on Camp Pendleton along a 26-mile pipeline. Managed crew of 20, planting coastal sage, riparian, wetland, native grassland, and marsh ecosystems. Negotiated with the CDFW concerning species planting list and success standards.

Client: NAVY BRAC (Orote Landfill, Guam)
Designed and monitored pilot landfill cap mimicking limestone forest. Measured different species' root-penetration into landfill cap. Plants were used to evapotranspirate water, reducing water leaching through soil profile.

Client: LA Sanitation District Puente Hills Landfill (Whittier, California)
Monitored success of upland and wetland mitigation at Puente Hills Landfill operated by Sanitation Districts of Los Angeles. Negotiated with the Army Corps of Engineers and CDFG to obtain an early sign-off.

Client: City of Escondido (Escondido, California)
Designed, managed, installed, and monitored a 20-acre coastal sage scrub restoration project at Kit Carson Park, Escondido, California.

Client: Home Depot (Encinitas, California)
Designed, managed, installed and monitored a 15-acre coastal sage scrub and wetland restoration project at Home Depot in Encinitas, California.

Client: Alvarado Water Filtration Plant (San Diego, California)
Planned, installed and monitored 2-acre riparian and coastal sage scrub mitigation in San Diego California.

Client: Monsanto and James River Corporation (Clatskanie, Oregon)
Served as a soil scientist on a 50,000-acre hybrid poplar farm. Worked on genetically engineering study of Poplar trees to see if glyphosate resistant poplar clones were economically viable.

Client: World Wildlife Fund (St. Kitts, West Indies)
Managed 2-year biodiversity study, quantifying and qualifying the various flora and fauna in St. Kitts' expanding volcanic rainforest. Collaborated with skilled botanists, ornithologists and herpetologists.

Publications

Chen, J. A., Zapata, A R., Sutherland, A. J., Molmen, D. R,. Chow, B. S., Wu, L. E., **Rosenfeld, P. E.,** Hesse, R. C., (2012) Sulfur Dioxide and Volatile Organic Compound Exposure To A Community In Texas City Texas Evaluated Using Aermod and Empirical Data. American Journal of Environmental Science, 2012, 8 (6), 622-632

Rosenfeld, P.E. & Feng, L. (2011). *The Risks of Hazardous Waste*, Amsterdam: Elsevier Publishing.

Cheremisinoff, N.P., & **Rosenfeld, P.E.** (2011). *Handbook of Pollution Prevention and Cleaner Production: Best Practices in the Agrochemical Industry*, Amsterdam: Elsevier Publishing.

Gonzalez, J., Feng, L., Sutherland, A., Waller, C., Sok, H., Hesse, R., **Rosenfeld, P.** (2011). PCBs and Dioxins/Furans in Attic Dust Collected Near Former PCB Production and Secondary Copper Facilities in Sauget, IL. *Procedia Environmental Sciences* 4(2011):113-125.

Feng, L., Wu, C., Tam, L., Sutherland, A.J., Clark, J.J., **Rosenfeld, P.E.**, (2010). Dioxin and Furan Blood Lipid and Attic Dust Concentrations in Populations Living Near Four Wood Treatment Facilities in the United States. *Journal of Environmental Health* 73(6):34-46.

Cheremisinoff, N.P., & **Rosenfeld, P.E.** (2010). *Handbook of Pollution Prevention and Cleaner Production: Best Practices in the Wood and Paper Industries*, Amsterdam: Elsevier Publishing.

Cheremisinoff, N.P., & **Rosenfeld, P.E.** (2009). *Handbook of Pollution Prevention and Cleaner Production: Best Practices in the Petroleum Industry*, Amsterdam: Elsevier Publishing.

Wu, C., Tam, L., Clark, J., **Rosenfeld, P**. (2009). 'Dioxin and furan blood lipid concentrations in populations living near four wood treatment facilities in the United States', in Brebbia, C.A. and Popov, V., eds., *Air Pollution XVII: Proceedings of the Seventeenth International Conference on Modelling, Monitoring and Management of Air Pollution*, Tallinn, Estonia. 20-22 July, 2009, Southampton, Boston. WIT Press.

Tam L. K.., Wu C. D., Clark J. J. and **Rosenfeld, P.E.** (2008) A Statistical Analysis Of Attic Dust And Blood Lipid Concentrations Of Tetrachloro-p-Dibenzodioxin (TCDD) Toxicity Equivalency Quotients (TEQ) In Two Populations Near Wood Treatment Facilities. Organohalogen Compounds, Volume 70 (2008) page 002254.

Tam L. K.., Wu C. D., Clark J. J. and **Rosenfeld, P.E.** (2008) Methods For Collect Samples For Assessing Dioxins And Other Environmental Contaminants In Attic Dust: A Review. Organohalogen Compounds, Volume 70 (2008) page 000527.

Hensley, A.R. A. Scott, J. J. J. Clark, **P. E. Rosenfeld** (2007) "Attic Dust and Human Blood Samples Collected near a Former Wood Treatment Facility" Environmental Research. 105, pp 194-197.
Rosenfeld, P.E., J. J. J. Clark, A. R. Hensley, M. Suffet. (2007) "The Use of an Odor Wheel Classification for Evaluation of Human Health Risk Criteria for Compost Facilities" –Water Science & Technology 55(5): 345-357.

Rosenfeld, P. E., M. Suffet. (2007) **"**The Anatomy Of Odour Wheels For Odours Of Drinking Water, Wastewater, Compost And The Urban Environment **"** Water Science & Technology 55(5): 335-344.

Sullivan, P. J. Clark, J.J.J., Agardy, F. J., **Rosenfeld, P.E.,** (2007) "Toxic Legacy, Synthetic Toxins in the Food, Water, and Air in American Cities," Elsevier Publishing, Boston Massachusetts.

Rosenfeld P.E., and Suffet, I.H. (Mel) (2007) "Anatomy Of An Odor Wheel" Water Science and Technology, In Press.

Rosenfeld, P.E., Clark, J.J.J., Hensley A.R., Suffet, I.H. (Mel) (2007) "The use of an odor wheel classification for evaluation of human health risk criteria for compost facilities." Water Science And Technology, In Press.

Hensley A.R., Scott, A., **Rosenfeld P.E.,** Clark, J.J.J. (2006) "Dioxin Containing Attic Dust And Human Blood Samples Collected Near A Former Wood Treatment Facility." The 26th International Symposium on Halogenated Persistent Organic Pollutants – DIOXIN2006, August 21 – 25, 2006. Radisson SAS Scandinavia Hotel in Oslo Norway.

Rosenfeld, P.E., and Suffet I.H. (2004) "Control of Compost Odor Using High Carbon Wood Ash", Water Science and Technology, Vol. 49, No. 9. pp. 171-178.

Rosenfeld, P.E., Clark J. J. and Suffet, I.H. (2004) "Value of and Urban Odor Wheel." (2004). WEFTEC 2004. New Orleans, October 2 - 6, 2004.

Rosenfeld, P.E., and Suffet, I.H. (2004) "Understanding Odorants Associated With Compost, Biomass Facilities, and the Land Application of Biosolids" Water Science and Technology. Vol. 49, No. 9. pp 193-199.

Rosenfeld, P.E., and Suffet I.H. (2004) "Control of Compost Odor Using High Carbon Wood Ash", Water Science and Technology, Vol. 49, No. 9. pp. 171-178.

Rosenfeld, P. E., Grey, M. A., Sellew, P. (2004) Measurement of Biosolids Odor and Odorant Emissions from Windrows, Static Pile and Biofilter. Water Environment Research. 76 (4): 310-315 JUL-AUG 2004.

Rosenfeld, P. E., Grey, M., (2003) Two stage biofilter for biosolids composting odor control. Seventh International In Situ And On Site Bioremediation Symposium. Batelle Conference Orlando Florida. June 2 and June 6, 2003.

Rosenfeld, P.E., Grey, M and Suffet, M. 2002. "Controlling Odors Using High Carbon Wood Ash." Biocycle, March 2002, Page 42.

Rosenfeld, P.E., Grey, M and Suffet, M. (2002). "Compost Demonstration Project, Sacramento, California Using High-Carbon Wood Ash to Control Odor at a Green Materials Composting Facility Integrated Waste Management Board Public Affairs Office, Publications Clearinghouse (MS–6), Sacramento, CA Publication #442-02-008. April 2002.

Rosenfeld, P.E., and C.L. Henry. 2001. Characterization of odor emissions from three different biosolids. Water Soil and Air pollution. Vol. 127 Nos. 1-4, pp. 173-191.

Rosenfeld, P.E., and Henry C. L., 2000. Wood ash control of odor emissions from biosolids application. Journal of Environmental Quality. 29:1662-1668.

Rosenfeld, P.E., C.L. Henry and D. Bennett. 2001. Wastewater dewatering polymer affect on biosolids odor emissions and microbial activity. Water Environment Research. 73: 363-367.

Rosenfeld, P.E., and C.L. Henry. 2001. Activated Carbon and Wood Ash Sorption of Wastewater, Compost, and Biosolids Odorants Water Environment Research, 73: 388-392.

Rosenfeld, P.E., and Henry C. L., 2001. High carbon wood ash effect on biosolids microbial activity and odor. Water Environment Research. Volume 131 No. 1-4, pp. 247-262.

Rosenfeld, P.E, C.L. Henry, R. Harrison. 1998. Oat and Grass Seed Germination and Nitrogen and Sulfur Emissions Following Biosolids Incorporation With High-Carbon Wood-Ash. Water Environment Federation 12th Annual Residuals and Biosolids Management Conference Proceedings. Bellevue Washington.

Chollack, T. and **P. Rosenfeld.** 1998. Compost Amendment Handbook For Landscaping. Prepared for and distributed by the City of Redmond, Washington State.

P. Rosenfeld. 1992. The Mount Liamuiga Crater Trail. Heritage Magazine of St. Kitts, Vol. 3 No. 2.

P. Rosenfeld. 1993. High School Biogas Project to Prevent Deforestation On St. Kitts. Biomass Users Network, Vol. 7, No. 1, 1993.

P. Rosenfeld. 1992. British West Indies, St. Kitts. Surf Report, April issue.

P. Rosenfeld. 1998. Characterization, Quantification, and Control of Odor Emissions From Biosolids Application To Forest Soil. Doctoral Thesis. University of Washington College of Forest Resources.

P. Rosenfeld. 1994. Potential Utilization of Small Diameter Trees On Sierra County Public Land. Masters thesis reprinted by the Sierra County Economic Council. Sierra County, California.

P. Rosenfeld. 1991. How to Build a Small Rural Anaerobic Digester & Uses Of Biogas In The First And Third World. Bachelors Thesis. University of California.

England Environmental Agency, 2002. Landfill Gas Control Technologies. Publishing Organization Environment Agency, Rio House, Waterside Drive, Aztec West, Almondsbury BRISTOL, BS32 4UD.

Presentations

Sok, H.L.; Waller, C.C.; Feng, L.; Gonzalez, J.; Sutherland, A.J.; Wisdom-Stack, T.; Sahai, R.K.; Hesse, R.C.; **Rosenfeld, P.E.** "Atrazine: A Persistent Pesticide in Urban Drinking Water." Urban Environmental Pollution, Boston, MA, June 20-23, 2010.

Feng, L.; Gonzalez, J.; Sok, H.L.; Sutherland, A.J.; Waller, C.C.; Wisdom-Stack, T.; Sahai, R.K.; La, M.; Hesse, R.C.; **Rosenfeld, P.E.** "Bringing Environmental Justice to East St. Louis, Illinois." Urban Environmental Pollution, Boston, MA, June 20-23, 2010.

Rosenfeld, P.E. (2009) "Perfluoroctanoic Acid (PFOA) and Perfluoroactane Sulfonate (PFOS) Contamination in Drinking Water From the Use of Aqueous Film Forming Foams (AFFF) at Airports in the United States" Presentation at the 2009 Ground Water Summit and 2009 Ground Water Protection Council Spring Meeting, April 19-23, 2009. Tuscon, AZ.

Rosenfeld, P.E. (2009) "Cost to Filter Atrazine Contamination from Drinking Water in the United States" Contamination in Drinking Water From the Use of Aqueous Film Forming Foams (AFFF) at Airports in the United States" Presentation at the 2009 Ground Water Summit and 2009 Ground Water Protection Council Spring Meeting, April 19-23, 2009. Tuscon, AZ.

Rosenfeld, P. E. (2007) "Moss Point Community Exposure To Contaminants From A Releasing Facility" Platform Presentation at the 23rd Annual International Conferences on Soils Sediment and Water, October 15-18, 2007. University of Massachusetts, Amherst MA.

Rosenfeld, P. E. (2007) "The Repeated Trespass of Tritium-Contaminated Water Into A Surrounding Community Form Repeated Waste Spills From A Nuclear Power Plant" Platform Presentation at the 23rd Annual International Conferences on Soils Sediment and Water, October 15-18, 2007. University of Massachusetts, Amherst MA.

Rosenfeld, P. E. (2007) "Somerville Community Exposure To Contaminants From Wood Treatment Facility Emissions" Poster Presentation at the 23rd Annual International Conferences on Soils Sediment and Water, October 15-18, 2007. University of Massachusetts, Amherst MA.

Rosenfeld P. E. "Production, Chemical Properties, Toxicology, & Treatment Case Studies of 1,2,3-Trichloropropane (TCP)" – Platform Presentation at the Association for Environmental Health and Sciences (AEHS) Annual Meeting, San Diego, CA, 3/2007.

Rosenfeld P. E. "Blood and Attic Sampling for Dioxin/Furan, PAH, and Metal Exposure in Florala, Alabama" – Platform Presentation at the AEHS Annual Meeting, San Diego, CA, 3/2007.

Hensley A.R., Scott, A., **Rosenfeld P.E.,** Clark, J.J.J. (2006) "Dioxin Containing Attic Dust And Human Blood Samples Collected Near A Former Wood Treatment Facility." APHA 134 Annual Meeting & Exposition, Boston Massachusetts. November 4 to 8th, 2006.

Paul Rosenfeld Ph.D. "Fate, Transport and Persistence of PFOA and Related Chemicals." Mealey's C8/PFOA Science, Risk & Litigation Conference" October 24, 25. The Rittenhouse Hotel, Philadelphia.

Paul Rosenfeld Ph.D. "Brominated Flame Retardants in Groundwater: Pathways to Human Ingestion, Toxicology and Remediation PEMA Emerging Contaminant Conference. September 19. Hilton Hotel, Irvine California.

Paul Rosenfeld Ph.D. "Fate, Transport, Toxicity, And Persistence of 1,2,3-TCP." PEMA Emerging Contaminant Conference. September 19. Hilton Hotel in Irvine, California.

Paul Rosenfeld Ph.D. "Fate, Transport and Persistence of PDBEs." Mealey's Groundwater Conference. September 26, 27. Ritz Carlton Hotel, Marina Del Ray, California.

Paul Rosenfeld Ph.D. "Fate, Transport and Persistence of PFOA and Related Chemicals." International Society of Environmental Forensics: Focus On Emerging Contaminants. June 7,8. Sheraton Oceanfront Hotel, Virginia Beach, Virginia.

Paul Rosenfeld Ph.D. "Rate Transport, Persistence and Toxicology of PFOA and Related Perfluorochemicals". 2005 National Groundwater Association Ground Water And Environmental Law Conference. July 21-22, 2005. Wyndham Baltimore Inner Harbor, Baltimore Maryland.

Paul Rosenfeld Ph.D. "Brominated Flame Retardants in Groundwater: Pathways to Human Ingestion, Toxicology and Remediation." 2005 National Groundwater Association Ground Water And Environmental Law Conference. July 21-22, 2005. Wyndham Baltimore Inner Harbor, Baltimore Maryland.

Paul Rosenfeld, Ph.D. and James Clark Ph.D. and Rob Hesse R.G. Tert-butyl Alcohol Liability and Toxicology, A National Problem and Unquantified Liability. National Groundwater Association. Environmental Law Conference. May 5-6, 2004. Congress Plaza Hotel, Chicago Illinois.

Paul Rosenfeld, Ph.D., 2004. Perchlorate Toxicology. Presentation to a meeting of the American Groundwater Trust. March 7th, 2004. Pheonix Arizona.

Hagemann, M.F., **Paul Rosenfeld, Ph.D.** and Rob Hesse, 2004. Perchlorate Contamination of the Colorado River. Invited presentation to a meeting of tribal representatives, Parker, AZ.

Paul Rosenfeld, Ph.D. A National Damage Assessment Model For PCE and Dry Cleaners. Drycleaner Symposium. California Ground Water Association. Radison Hotel, Sacramento, California. April 7, 2004.

Paul Rosenfeld, Ph.D. and James Clark Ph.D. Understanding Historical Use, Chemical Properties, Toxicity and Regulatory Guidance of 1,4 Dioxane. National Groundwater Association. Southwest Focus Conference. Water Supply and Emerging Contaminants. February 20-21, 2003. Hyatt Regency Phoenix Arizona.

Paul Rosenfeld, Ph.D. Underground Storage Tank Litigation and Remediation. California CUPA Forum. Marriott Hotel. Anaheim California. February 6-7, 2003.

Paul Rosenfeld, Ph.D. Underground Storage Tank Litigation and Remediation. EPA Underground Storage Tank Roundtable. Sacramento California. October 23, 2002.

Rosenfeld, P.E. and Suffet, M. 2002. Understanding Odor from Compost, Wastewater and Industrial Processes. Sixth Annual Symposium On Off Flavors in the Aquatic Environment. International Water Association. Barcelona Spain. October 7- 10.

Rosenfeld, P.E. and Suffet, M. 2002. Using High Carbon Wood Ash to Control Compost Odor. Sixth Annual Symposium On Off Flavors in the Aquatic Environment. International Water Association. Barcelona Spain. October 7- 10.

Rosenfeld, P.E. and Grey, M. A. 2002. Biocycle Composting For Coastal Sage Restoration. Northwest Biosolids Management Association. Vancouver Washington. September 22-24.

Rosenfeld, P.E. and Grey, M. A. 2002. Soil Science Society Annual Conference. Indianapolis, Maryland. November 11-14.

Rosenfeld. P.E. 2000. Two stage biofilter for biosolids composting odor control. Water Environment Federation. Anaheim California. September 16, 2000.

Rosenfeld. P. E. 2000. Wood ash and biofilter control of compost odor. Biofest. October 16, 2000.Ocean Shores, California.

Rosenfeld, P. E. 2000. Bioremediation Using Organic Soil Amendments. California Resource Recovery Association. Sacramento California.

Rosenfeld, P.E., C.L. Henry, R. Harrison. 1998. Oat and Grass Seed Germination and Nitrogen and Sulfur Emissions Following Biosolids Incorporation With High-Carbon Wood-Ash. Water Environment Federation 12th Annual Residuals and Biosolids Management Conference Proceedings. Bellevue Washington.

Rosenfeld, P.E., and C.L. Henry. 1999. An evaluation of ash incorporation with biosolids for odor reduction. Soil Science Society of America. Salt Lake City Utah.

Rosenfeld, P.E., C.L. Henry, R. Harrison. 1998. Comparison of Microbial Activity and Odor Emissions from Three Different Biosolids Applied to Forest Soil. Brown and Caldwell, Seattle Washington.

Rosenfeld, P.E., C.L. Henry. 1998. Characterization, Quantification, and Control of Odor Emissions from Biosolids Application To Forest Soil. Biofest Lake Chelan, Washington.

Rosenfeld, P.E., C.L. Henry, R. B. Harrison, and R. Dills. 1997. Comparison of Odor Emissions From Three Different Biosolids Applied to Forest Soil. Soil Science Society of America, Anaheim California.

Professional History

Soil Water Air Protection Enterprise (SWAPE); 2003 to present; Founding And Managing Partner
UCLA School of Public Health; 2007 to 2010; Lecturer (Asst Res)
UCLA School of Public Health; 2003 to 2006; Adjunct Professor
UCLA Environmental Science and Engineering Program; 2002-2004; Doctoral Intern Coordinator
UCLA Institute of the Environment, 2001-2002; Research Associate
Komex H_2O Science, 2001 to 2003; Senior Remediation Scientist
National Groundwater Association, 2002-2004; Lecturer
San Diego State University, 1999-2001; Adjunct Professor
Anteon Corp., San Diego, 2000-2001; Remediation Project Manager
Ogden (now Amec), San Diego, 2000-2000; Remediation Project Manager
Bechtel, San Diego, California, 1999 – 2000; Risk Assessor
King County, Seattle, 1996 – 1999; Scientist
James River Corp., Washington, 1995-96; Scientist
Big Creek Lumber, Davenport, California, 1995; Scientist
Plumas Corp., California and USFS, Tahoe 1993-1995; Scientist
Peace Corps and World Wildlife Fund, St. Kitts, West Indies, 1991-1993; Scientist
Bureau of Land Management, Kremmling Colorado 1990; Scientist

Teaching Experience

UCLA Department of Environmental Health (Summer 2003 through 2010) Taught Environmental Health Science 100 to students, including undergrad, medical doctors, public health professionals and nurses. Course focuses on the health effects of environmental contaminants.

National Ground Water Association, Successful Remediation Technologies. Custom Course In Sante Fe, New Mexico. May 21, 2002. Focused on fate and transport of fuel contaminants associated with underground storage tanks.

National Ground Water Association; Successful Remediation Technologies Course in Chicago Illinois. April 1, 2002. Focused on fate and transport of contaminants associated with Superfund and RCRA sites.

California Integrated Waste Management Board, April and May, 2001. Alternative Landfill Caps Seminar in San Diego, Ventura, and San Francisco. Focused on both prescriptive and innovative landfill cover design.

UCLA Department of Environmental Engineering, February 5 2002 Seminar on Successful Remediation Technologies focusing on Groundwater Remediation.

University Of Washington, Soil Science Program, Teaching Assistant for several courses including: Soil Chemistry, Organic Soil Amendments, and Soil Stability.

U.C. Berkeley, Environmental Science Program Teaching Assistant for Environmental Science 10.

Academic Grants Awarded

California Integrated Waste Management Board. $41,000 grant awarded to UCLA Institute of the Environment. Goal: To investigate effect of high carbon wood ash on volatile organic emissions from compost. 2001.

Synagro Technologies, Corona California: $10,000 grant awarded to San Diego State University. Goal: investigate effect of biosolids for restoration and remediation of degraded coastal sage soils. 2000.

King County, Department of Research and Technology, Washington State. $100,000 grant awarded to University of Washington: Goal: To investigate odor emissions from biosolids application and the effect of polymers and ash on VOC emissions. 1998.

Northwest Biosolids Management Association, Washington State. $20,000 grant awarded to investigate effect of polymers and ash on VOC emissions from biosolids. 1997.

James River Corporation, Oregon: $10,000 grant was awarded to investigate the success of genetically engineered Poplar trees with resistance to round-up. 1996.

United State Forest Service, Tahoe National Forest: $15,000 grant was awarded to investigating fire ecology of the Tahoe National Forest. 1995.

Kellogg Foundation, Washington D.C. $500 grant was awarded to construct a large anaerobic digester on St. Kitts in West Indies. 1993.

Cases that Dr. Rosenfeld Provided Deposition or Trial Testimony

In the Court of Common Pleas of Tuscarawas County Ohio
John Michael Abicht, et al., *Plaintiffs,* vs. Republic Services, Inc., et al., *Defendants*
Case Number: 2008 CT 10 0741 (Cons. w/ 2009 CV 10 0987)

In the Court of Common Pleas for the Second Judicial Circuit, State of South Carolina, County of Aiken
David Anderson, et al., *Plaintiffs,* vs. Norfolk Southern Corporation, et al., *Defendants.*
Case Number: 2007-CP-02-1584

In the Circuit Court of Jefferson County Alabama
Jaeanette Moss Anthony, et al., *Plaintiffs*, vs. Drummond Company Inc., et al., *Defendants*
Civil action No. CV 2008-2076

In the Ninth Judicial District Court, Parish of Rapides, State of Louisiana
Roger Price, et al., *Plaintiffs*, vs. Roy O. Martin, L.P., et al., *Defendants*.
Civil Suit Number 224,041 Division G

In the United States District Court, Western District Lafayette Division
Ackle et al., *Plaintiffs*, vs. Citgo Petroleum Corporation, et al., *Defendants*.
Case Number 2:07CV1052

In the United States District Court for the Southern District of Ohio
Carolyn Baker, et al., *Plaintiffs*, vs. Chevron Oil Company, et al., *Defendants*.
Case Number 1:05 CV 227

In the Fourth Judicial District Court, Parish of Calcasieu, State of Louisiana
Craig Steven Arabie, et al., *Plaintiffs*, vs. Citgo Petroleum Corporation, et al., *Defendants*.
Case Number 07-2738 G

In the Fourteenth Judicial District Court, Parish of Calcasieu, State of Louisiana
Leon B. Brydels, *Plaintiffs*, vs. Conoco, Inc., et al., *Defendants.*
Case Number 2004-6941 Division A

In the District Court of Tarrant County, Texas, 153rd Judicial District
Linda Faust, *Plaintiff*, vs. Burlington Northern Santa Fe Rail Way Company, Witco Chemical Corporation A/K/A Witco Corporation, Solvents and Chemicals, Inc. and Koppers Industries, Inc., *Defendants*.
Case Number 153-212928-05

In the Superior Court of the State of California in and for the County of San Bernardino
Leroy Allen, et al., *Plaintiffs*, vs. Nutro Products, Inc., a California Corporation and DOES 1 to 100, inclusive, *Defendants*.
John Loney, Plaintiff, vs. James H. Didion, Sr.; Nutro Products, Inc.; DOES 1 through 20, inclusive, *Defendants*.
Case Number VCVVS044671

In the United States District Court for the Middle District of Alabama, Northern Division
James K. Benefield, et al., *Plaintiffs*, vs. International Paper Company, *Defendant*.
Civil Action Number 2:09-cv-232-WHA-TFM

In the Superior Court of the State of California in and for the County of Los Angeles
Leslie Hensley and Rick Hensley, *Plaintiffs*, vs. Peter T. Hoss, as trustee on behalf of the Cone Fee Trust; Plains Exploration & Production Company, a Delaware corporation; Rayne Water Conditioning, Inc., a California corporation; and DOES 1 through 100, *Defendants.*
Case Number SC094173

In the Superior Court of the State of California in and for the County of Santa Barbara, Santa Maria Branch
Clifford and Shirley Adelhelm, et al., all individually, *Plaintiffs*, vs. Unocal Corporation, a Delaware Corporation; Union Oil Company of California, a California corporation; Chevron Corporation, a California corporation; ConocoPhillips, a Texas corporation; Kerr-McGee Corporation, an Oklahoma corporation; and DOES 1 though 100, *Defendants.*
Case Number 1229251 (Consolidated with case number 1231299)

In the United States District Court for Eastern District of Arkansas, Eastern District of Arkansas
Harry Stephens Farms, Inc, and Harry Stephens, individual and as managing partner of Stephens Partnership, *Plaintiffs*, vs. Helena Chemical Company, and Exxon Mobil Corp., successor to Mobil Chemical Co., *Defendants.*
Case Number 2:06-CV-00166 JMM (Consolidated with case number 4:07CV00278 JMM)

In the United States District Court for the Western District of Arkansas, Texarkana Division
Rhonda Brasel, et al., *Plaintiffs*, vs. Weyerhaeuser Company and DOES 1 through 100, *Defendants.*
Civil Action Number 07-4037

In The Superior Court of the State of California County of Santa Cruz
Constance Acevedo, et al. *Plaintiffs* Vs. California Spray Company, et al. *Defendants*
Case No CV 146344

In the District Court of Texas 21st Judicial District of Burleson County
Dennis Davis, *Plaintiff*, vs. Burlington Northern Santa Fe Rail Way Company, *Defendant.*
Case Number 25,151

In the United States District Court of Southern District of Texas Galveston Division
Kyle Cannon, Eugene Donovan, Genaro Ramirez, Carol Sassler, and Harvey Walton, each Individually and on behalf of those similarly situated, *Plaintiffs*, vs. BP Products North America, Inc., *Defendant.*
Case 3:10-cv-00622

EXHIBIT 3

HADLEY KATHRYN NOLAN

SOIL WATER AIR PROTECTION ENTERPRISE
2656 29th Street, Suite 201
Santa Monica, California 90405
Mobile: (678) 551-0836
Office: (310) 452-5555
Fax: (310) 452-5550
Email: hadley@swape.com

SWAPE Technical Consultation, Data Analysis and Litigation Support for the Environment

EDUCATION

UNIVERSITY OF CALIFORNIA, LOS ANGELES **B.S. ENVIRONMENTAL SCIENCES & ENVIRONMENTAL SYSTEMS AND SOCIETY** *JUNE 2016*

PROJECT EXPERIENCE

SOIL WATER AIR PROTECTION ENTERPRISE ***SANTA MONICA, CA***

AIR QUALITY SPECIALIST

SENIOR PROJECT ANALYST: CEQA ANALYSIS & MODELING

- Modeled construction and operational activities for proposed land use projects using CalEEMod to quantify criteria air pollutant and greenhouse gas (GHG) emissions.
- Organized presentations containing figures and tables that compare results of criteria air pollutant analyses to thresholds.
- Quantified ambient air concentrations at sensitive receptor locations using AERSCREEN, a U.S. EPA recommended screening level dispersion model.
- Conducted construction and operational health risk assessments for residential, worker, and school children sensitive receptors.
- Prepared reports that discuss adequacy of air quality and health risk analyses conducted for proposed land use developments subject to CEQA review by verifying compliance with local, state, and regional regulations.

SENIOR PROJECT ANALYST: GREENHOUSE GAS MODELING AND DETERMINATION OF SIGNIFICANCE

- Evaluated environmental impact reports for proposed projects to identify discrepancies with the methods used to quantify and assess GHG impacts.
- Quantified GHG emissions for proposed projects using CalEEMod to produce reports, tables, and figures that compare emissions to applicable CEQA thresholds and reduction targets.
- Determined compliance of proposed land use developments with AB 32 GHG reduction targets, with GHG significance thresholds recommended by Air Quality Management Districts in California, and with guidelines set forth by CEQA.

PROJECT ANALYST: ASSESSMENT OF AIR QUALITY IMPACTS FROM PROPOSED DIRECT TRANSFER FACILITY

- Assessed air quality impacts resulting from implementation of a proposed Collection Service Agreement for Exclusive Residential and Commercial Garbage, Recyclable Materials, and Organic Waste Collection Services for a community.
- Organized tables and maps to demonstrate potential air quality impacts resulting from proposed hauling trip routes.
- Conducted air quality analyses that compared quantified criteria air pollutant emissions released during construction of direct transfer facility to the Bay Area Air Quality Management District's (BAAQMD) significance thresholds.
- Prepared final analytical report to demonstrate local and regional air quality impacts, as well as GHG impacts.

PROJECT ANALYST: EXPOSURE ASSESSMENT OF LEAD PRODUCTS FOR PROPOSITION 65 COMPLIANCE DETERMINATION

- Calculated human exposure and lifetime health risk for over 300 lead products undergoing Proposition 65 compliance review.
- Compiled and analyzed laboratory testing data and produced tables, charts, and graphs to exhibit emission levels.
- Compared finalized testing data to Proposition 65 Maximum Allowable Dose Levels (MADLs) to determine level of compliance.
- Prepared final analytical lead exposure Certificate of Merit (COM) reports and organized supporting data for use in environmental enforcement statute Proposition 65 cases.

ACCOMPLISHMENTS

- **Academic Honoree,** Dean's List, University of California, Los Angeles ***MAR 2013, MAR 2014, JAN 2015, JAN 2016***

EXHIBIT 4

Air Toxics Hot Spots Program Risk Assessment Guidelines

Technical Support Document for Exposure Assessment and Stochastic Analysis

August 2012

Secretary for Environmental Protection
California Environmental Protection Agency
Matthew Rodriquez

Director
Office of Environmental Health Hazard Assessment
George V. Alexeeff, Ph.D., D.A.B.T.

Final

August 2012

Air Toxics Hot Spots Program
Risk Assessment Guidelines

Technical Support Document

Exposure Assessment and Stochastic Analysis

Office of Environmental Health Hazard Assessment
California Environmental Protection Agency
1515 Clay Street, 16th Floor
Oakland, California 94612 (510) 622-3200

Project Lead: Robert J. Blaisdell, Ph.D.

Reviewed by:

Melanie A. Marty, Ph.D.

Chief, Air Toxicology and Epidemiology Branch, OEHHA

George V. Alexeeff, Ph.D.

Director, OEHHA

OEHHA acknowledges the following contributors:

Amy Arcus-Arth, D.V.M., M.P.V.M

James F. Collins, Ph.D., D.A.B.T.

Daryn E. Dodge, Ph.D.

Richard Lam, Ph.D.

Brian Malig, M.P.H.

Andrew G. Salmon, M.A., D.Phil.

Kathleen Vork, Ph.D. M.P.H

Aijun Albert Wang, Ph.D.

Roberta Welling, M.S., M.P.H.

Gregory Harris [a]

Anthony Servin, P.E. [a]

Steven Yee [a]

Yan-Ping Zuo [a]

Air Toxicology and Epidemiology Branch
Office of Environmental Health Hazard Assessment

[a] California Air Resources Board

Air Toxics “Hot Spots” Program Risk Assessment Guidelines

Technical Support Document for

Exposure Assessment and Stochastic Analysis

TABLE OF CONTENTS

1 Introduction

The Air Toxics "Hot Spots" Information and Assessment Act (AB 2588, Connelly, stat. 1987; Health and Safety Code Section 44300 et seq.) is designed to provide information on the extent of airborne emissions from stationary sources and the potential public health impacts of those emissions. Facilities provide emissions inventories of chemicals specifically listed under the "Hot Spots" Act to the local Air Pollution Control and Air Quality Management Districts (Districts) and ultimately to the state Air Resources Board. Following prioritization of facilities by the Districts, facilities may be required to conduct a health risk assessment.

Health risk assessment involves a comprehensive analysis of the dispersion of the specific facility's air emissions, and the extent of human exposure via all relevant pathways (exposure assessment), the toxicology of those chemicals (dose-response assessment), and the estimation of cancer risk and noncancer health impacts to the exposed community (risk characterization). Most "Hot Spots" risk assessments are conducted by contractors for the facility; some are conducted in-house and some by the local air districts. AB-2588 mandates the Office of Environmental Health Hazard Assessment (OEHHA) to review Hot Spots risk assessments and the findings are conveyed to the District by letter. The District may require the facility to notify the impacted public if the risk assessment shows risks above a level deemed acceptable by the District.

The Air Toxics "Hot Spots" Act was amended to require that the Office of Environmental Health Hazard Assessment (OEHHA) develop risk assessment guidelines for the Air Toxics "Hot Spots" program (SB 1731, Calderon, stat. 1992; Health and Safety Code Section 44360(b)(2)). The amendment specifically requires OEHHA to develop a "likelihood of risks" approach to health risk assessment. Therefore, the OEHHA developed a stochastic, or probabilistic, approach to exposure assessment to fulfill this requirement. The previous version of this document, the *Technical Support Document for Exposure Assessment and Stochastic Analysis,* was final in September 2000 (OEHHA, 2000a). This revision of the document updates OEHHA (2000a) by incorporating scientific advances in the field of exposure assessment, and newer data on exposure variates. Exposure variates are consumption estimates for various media and values for fate and transport modeling such as fish bioaccumulation factors.

All facilities are required to conduct a point estimate risk assessment using OEHHA's recommended exposure variates. Facilities may choose to also conduct a stochastic assessment of exposure (and risk) to provide more information to the risk managers and the public. The stochastic approach described in this document provides guidance to the facility operators who want to conduct a stochastic risk assessment, and facilitates use of supplemental information to be considered in the health risk assessment. It provides a method for quantification of the portion of exposure variability for which sufficient data exist to permit estimation. This document does not present an approach for quantification of uncertainty in exposure assessment.

OEHHA has developed a series of documents describing the information supporting the dose-response assessment for "Hot Spots" chemicals and the exposure assessment methodologies. The Children's Environmental Health Protection Act (SB-25) was passed in 1999 and mandated that OEHHA ensure that our risk assessment procedures were protective of children's health. OEHHA developed the methodology presented in the *Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document for the Derivation of Non-cancer Reference Exposure Levels* (RELs) (OEHHA, 2008) to ensure that our procedures for REL development were protective of children. The 2008 document supersedes the earlier documents for acute RELS, (OEHHA 1999a) and chronic RELS (OEHHA, 2000b). However, RELs developed under the previous OEHHA Guidance (1999a and 2000b) that have not undergone re-evaluation under the OEHHA (2008) updated methodology remain in effect for the Hot Spots program. New and revised RELs are being developed using the 2008 Guidelines and periodically released for public comment and review by the State's Scientific Review Panel on Toxic Air Contaminants (SRP).

OEHHA also developed the *Technical Support Document for Cancer Potency Factors: Methodologies for Derivation, Listing of Available Values, and Adjustments to Allow for Early Life Stage Exposures* (OEHHA, 2009) after the passage of SB-25 to ensure that cancer dose-response takes into account the vulnerability of children. The 2009 document supersedes the *Technical Support Document for Determining Cancer Potency Factors* (OEHHA, 1999b).

This revision of the *Technical Support Document for Exposure Assessment and Stochastic Analysis* describes the exposure algorithms, and point estimates and distributions of key exposure variates that can be used for the exposure analysis component of Air Toxics "Hot Spots" risk assessments. OEHHA reassessed exposure variates for children to ensure they would not underestimate exposure under our SB-25 mandate. We also incorporated advances in the field of exposure assessment since the previous version of the document. The document includes a description of the point estimate and stochastic multipathway exposure assessment approaches and a brief summary of the information supporting the selection of default assumptions. OEHHA developed this document in consultation with the Air Resources Board (ARB) and the California Air Pollution Control Officers Association (CAPCOA). The ARB provided Chapter 2 and associated appendices describing the air dispersion and deposition modeling.

A tiered approach to risk assessment, which allows for both consistency and flexibility, is described in Section 1.4. OEHHA's proposed algorithms, default point estimates and distributions of variates for each major exposure pathway are described in Chapters 3 through 10. The algorithms, with one exception, are identical to the previous version of this document (OEHHA, 2000). We condensed portions of the algorithm for dermal absorption, simplifying the equation and calculation. The algorithms used in our exposure model are largely consistent with the U.S. EPA (1991) Risk Assessment Guidance for Superfund Sites, with some modifications. The point estimates and distributions were updated based on newer data.

Finally, we are updating the *Air Toxics 'Hot Spots' Risk Assessment Guidance Manual* (OEHHA, 2003). This updated document, which will be available soon for public comment and peer review by the SRP, contains the essential information to conduct a health risk assessment based on the three technical support documents described above.

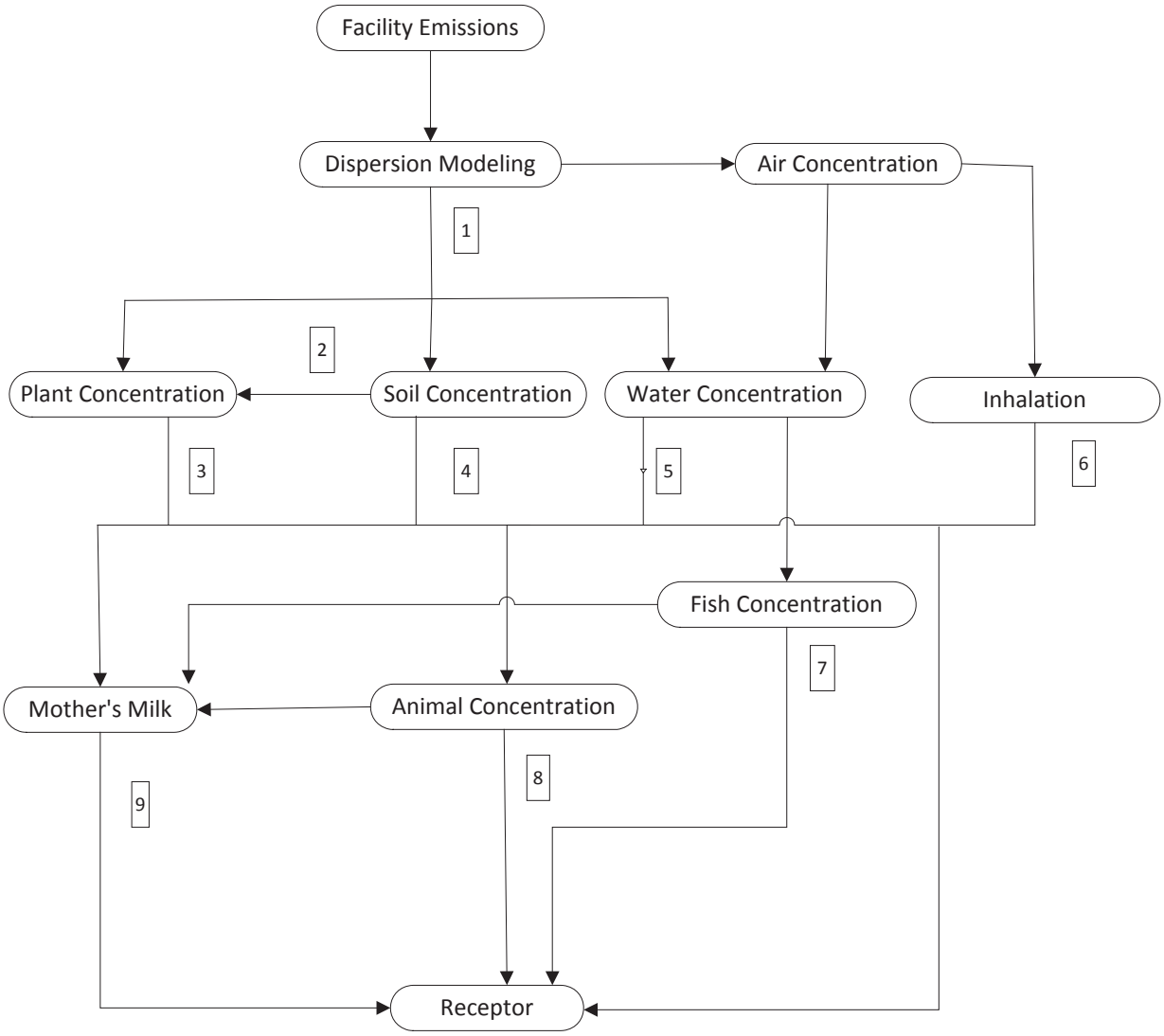
1.1 Multipathway Nature of Exposure Assessment

Exposure assessment of airborne emissions includes not only an analysis of exposure via the inhalation pathway, but also noninhalation pathways of indirect exposure to airborne toxicants. There are data in the literature demonstrating that for some compounds, significant exposure occurs following deposition of airborne material onto surface water, soils, edible plants (both food, pasture and animal feed), and through ingestion of breast milk. Examining both direct inhalation and indirect noninhalation exposure pathways reveals the full extent of exposure to airborne emissions (see Figure 1.1).

However, only certain chemicals are evaluated via the multipathway approach in the Air Toxics "Hot Spots" risk assessments. In general, there is a higher potential for indirect exposure to chemicals which tend to bioconcentrate or bioaccumulate (e.g., lipophilic semi-volatile organics), or otherwise accumulate in the environment (e.g., metals). Semi-volatile and non-volatile organic and metal toxicants can be directly deposited onto surface waters, soil, leaves, fruits and vegetables, grazing forage, and so forth. This is particularly important when these chemicals are associated with particulate matter. Cows, chickens, and other food animals can become contaminated through inhalation, and ingestion of contaminated surface water, pasture, feed and soil. Fish can become contaminated via bioconcentration from water and bioaccumulation from their food. Produce can become contaminated via root uptake from soils and direct deposition. Thus, humans can be exposed through ingestion of contaminated meat, fish, produce, water and soil, as well as from breathing contaminated air, and via dermal exposure. In addition, nursing infants can be exposed via breast milk.

The exposure variates are presented by chapter in this Document roughly in order of importance to an Air Toxics Hot Spots facility risk assessment. The breathing rate (Chapter 3) is the most important pathway; all chemicals must include an inhalation assessment. The breathing rate chapter is followed by chapters discussing the pathways that are automatically included if a risk assessment finds semi- or non-volatile Hot Spots chemicals: the soil ingestion pathway (Chapter 4), the mother's milk pathway (Chapter 5), and the dermal exposure pathway (Chapter 6). The remaining chapters contain the pathways that are only presented in a risk assessment in cases where it has been shown that these exposure pathways exist: the home-produced food pathway (Chapter 7), the water intake pathway (Chapter 8), and the fish consumption pathway (Chapter 9).

Figure 1.1 Exposure Routes



1. Deposition
2. Root Uptake by plants.
3. Human Consumption of Leafy, Protected, Exposed and Root Produce. Animal consumption of pasture and feed.
4. Soil Ingestion by humans and animals, and dermal exposure to soil.
5. Water consumption from surface water sources
6. Inhalation by humans and animals
7. Fish consumption
8. Consumption of beef, chicken and pork.
9. Mother's milk consumption.

Inhalation exposure is assessed for all “Hot Spots”-listed chemicals which have either Cancer Potency Factors and/or Reference Exposure Levels (see the Technical Support Documents mentioned in paragraph 2 for information on these values (OEHHA,2008, 2009), available at http://www.oehha.ca.gov/air/hot_spots/index.html). The noninhalation exposures are assessed only for semivolatile organics and metals listed in Appendix E, Table E.2. These chemicals have oral RELs and/or oral cancer potency factors. Appendix E contains a description of the process used to decide which chemicals should be evaluated by multipathway exposure assessment.

Only the exposure pathways which exist at a particular site need to be assessed in the Air Toxics Hot Spots program. For example, if a fishable body of water is impacted by facility emissions, then exposure through consumption of angler-caught fish is assessed. Otherwise, that pathway may be omitted from the risk assessment. Likewise if no backyard or local commercial produce or animals are raised in the impacted area, then the risk assessment need not consider dose through the ingestion of animal food products or produce. The “Hot Spots” program does not currently assess run off into surface drinking water sources because of the complex site-specific information required. The water consumption of surface waters pathway is rarely invoked in the “Hot Spots” program.

All risk assessments of facilities emitting chemicals listed in Table E.2 need to include an evaluation of exposure from breast milk consumption, soil ingestion, and dermal absorption from soil, since these exposure pathways are likely to exist at all sites. Table E.3 lists the chemicals that should be evaluated by the breast milk exposure pathway. The determination of the appropriate exposure pathways for consideration in the risk assessment should be made in conjunction with the local Air Pollution Control or Air Quality Management District. Justification for excluding an exposure pathway should be clearly presented.

1.2 The Point Estimate Approach

The point estimate approach (sometimes referred to as deterministic) is the traditional approach for site-specific risk assessments in the Hot Spots program. In the point estimate approach, a single value is assigned to each variate in the model (e.g., a breathing rate in L/kg BW-day). The point estimates chosen sometimes represent upper-end values for the variate and sometimes reflect a mean or central tendency estimate. The outcomes of a point estimate model are single estimates of either cancer risk or of the hazard index for noncancer effects. The point estimates of risk are generally considered near the high-end of the range of estimated risks, based on variability in exposure; quantitative information on population variability is generally lacking. However, the older point estimate approach to exposure assessment left open the question of variability in exposures of the general population. For example, it was unclear what percentage of the population would breathe more or less than a 20 m^3/day inhalation rate. The research stimulated by the desire to incorporate population variability in stochastic approaches has allowed informed selection of point estimates

that cover a defined percentage of the population, within the limitations and uncertainties of the available scientific data.

1.2.1 Need for Exposure Variates for Specific Age Groupings

In the previous exposure guideline, we presented distributions and point estimates for use in exposure assessment for children less than 12 years of age and for adolescents and adults up to age 70 years. Risk assessments were conducted for different durations of exposure based on estimates of how long people live at a single location (9 years for the average, 30 years for a high end estimate, and 70 years for a lifetime).

This update retains the evaluation of the 9, 30 and 70 year exposure durations, which represent approximately the mean, 90th percentile and lifetime of residence time. However, *The Technical Support Document for Cancer Potency Factors: Methodologies for Derivation, Listing of Available Values, and Adjustments to Allow for Early Life Stage Exposures* (OEHHA, 2009) concludes that the potency of carcinogens, and thus cancer risk, varies based on the lifestage at exposure. To address this concern, OEHHA applies a weighting factor to early life exposures, termed the Age Sensitivity Factor (ASF) (see OEHHA, 2009 for details). Cancer risk is multiplied by an ASF of ten to weight lifetime risk from exposures occurring from the third trimester of pregnancy to age less than 2. Likewise, for exposure from age 2 to less than 16 years, an ASF of three is applied.

Using these Age Sensitivity Factors (ASFs) requires a different approach to calculation of cancer risk from the traditional methods. Accounting for effects of early-in life exposure requires accounting for both the increased potency of early in life exposure to carcinogens and the greater exposure on a per kg body weight that occurs early in life due to behavioral and physiological differences between infants and children, and adults.

The lifetime risk is a summation of risks from the third trimester to age 2 yrs, 2 to age 16 and 16 to age 70 years. Similarly, when estimating cancer risk for a 9 year (average duration living at given residence) exposure to facility emissions or a 30 year (high-end duration living at a given residence) exposure to facility emissions, the cancer risks are similarly summed, starting with early-in-life exposures. These calculations are as follows:

9-year exposure duration - Calculation of Cancer Risk from the Third Trimester to Age Nine:

$$\text{Cancer Risk} = [(ADD_{\text{third trimester}} \times CPF \times 10) \times 0.3\ \text{yrs}/70\ \text{yrs}] + [(ADD_{0\ \text{to}\ <2\text{yrs}} \times CPF \times 10) \times 2\ \text{yrs}/70\ \text{yrs}] + [(ADD_{2 < 9\text{yrs}} \times CPF \times 3) \times 7\ \text{yrs}/70\ \text{yrs}]$$

30-year exposure duration - Calculation of Cancer Risk from Third Trimester to Age 30:

$$\text{Cancer Risk} = [(ADD_{\text{third trimester}} \times CPF \times 10) \times 0.3 \text{ yrs}/70 \text{ yrs}] + [(ADD_{0 \text{ to } <2\text{yrs}} \times CPF \times 10) \times 2 \text{ yrs}/70 \text{ yrs}] + [(ADD_{2 < 16\text{yrs}} \times CPF \times 3) \times 14 \text{ yrs}/70 \text{ yrs}] + [(ADD_{16 < 30\text{yrs}} \times CPF \times 1) \times 14\text{yrs}/70 \text{ yrs}$$

Lifetime (70 year) exposure duration - Calculation of Cancer Risk from Third Trimester to Age 70:

$$\text{Cancer Risk} = [(ADD_{\text{third trimester}} \times CPF \times 10) \times 0.3 \text{ yrs}/70 \text{ yrs}] + [(ADD_{0 \text{ to } <2\text{yrs}} \times CPF \times 10) \times 2 \text{ yrs}/70 \text{ yrs}] + [(ADD_{2 < 16\text{yrs}} \times CPF \times 3) \times 14 \text{ yrs}/70 \text{ yrs}] + [(ADD_{16 < 70\text{yrs}} \times CPF \times 1) \times 54 \text{ yrs}/70 \text{ yrs}$$

where:

ADD = Average Daily Dose, mg/kg-d, for the specified time period (estimated using the exposure variates presented in the TSD)
CPF = Cancer Potency Factor $(\text{mg/kg-d})^{-1}$
Age Sensitivity Factor third trimester to less than 2 years = 10
Age Sensitivity Factor age 2 to less than 16 years = 3
Age Sensitivity Factor age 16 to less than 70 years = 1

Exposure from all pathways evaluated by the Hot Spots program tends to be greater for children per kilogram body weight, particularly for the third trimester to less than age 2 years. Therefore exposure variates are needed for the third trimester (mother's exposure), ages 0 to <2 years, 2 to <9 years, 2 < 16 years,16 to <30 years, and 16 to 70 years in order to properly estimate cancer risk for the age ranges specified in OEHHA (2009) as well as the residential exposure duration periods (9, 30, and 70 years). This document presents intake rates for the necessary age groupings for inhalation, food consumption, drinking water consumption, breast milk consumption, inadvertent soil ingestion, and dermal exposure useful to estimate exposure and thus cancer risk.

Estimating dose for the fetus during the third trimester of pregnancy is not easy because it will vary from chemical to chemical depending on the toxicokinetics. An approximation of the dose during the third trimester can be made by assuming the dose (mg/kg body weight) is the same as the mother's dose (mg/kg-body weight). The mother is assumed to fall into the age range sixteen to less than thirty. This approximation is uncertain and will over or underestimate dose in some instances. The dose during the third trimester tends to be considerably less than the dose during ages zero to less than two, so separate calculations of dose during the third trimester and ages zero to two years are needed.

The point estimate approach has the advantages of simplicity and consistency, and in the Air Toxics "Hot Spots" program consistent application across the state is critical to comparing risks across facilities for the notification and risk reduction provisions of the statute. Risk communication is relatively straightforward with a point estimate approach. However, a single point estimate approach does not provide information on

the variability in the dose or risk estimates. Some Information about the potential range of risks in the population can be presented as average or high-end point estimates of risk.

1.3 The Stochastic Approach ("Likelihood of Risks" Approach)

As noted earlier, the amended Act specifically requires OEHHA to develop a "likelihood of risks" approach to health risk assessment. Therefore, the OEHHA developed a stochastic, or probabilistic, approach to exposure assessment to fulfill this requirement. The stochastic approach to Hot Spots risk assessment developed by OEHHA estimates the population variability in cancer risk resulting from variability in intake rates such as breathing rate, infant breast milk ingestion, and meat and produce ingestion. The data on variability in risk assessment variates are largely limited to intake rates of contaminated media. Data are particularly sparse on the variability in fate and transport variates (e.g., soil half life). Therefore only a portion of the overall variability in exposure can be characterized in our model. However, for the less complicated pathways such as the inhalation pathway, the variability in breathing rate probably represents a major portion of the overall variability in exposure.

As noted in U.S. EPA (1995), true uncertainty represents lack of knowledge about a variate or factor that impacts risk which may be reduced by further study. There are uncertainties associated with measurement, with models of environmental fate (e.g., air dispersion models), and with dose-response models. Uncertainty may stem from data gaps that are filled by the use of assumptions. Although methods such as expert elicitation have been occasionally used to try to quantify true uncertainty in individual risk assessments, the cost of such methods is outside the scope of what would be reasonable for the Hot Spots program.

Variability can be measured empirically in data describing an exposure variate. Variability arises from true heterogeneity in characteristics of a population such as differences in rate of intake of various media (air, water, food, soil). The stochastic analysis approach presented in this document attempts to quantify some of the variability in exposure in the risk estimates by using measured variability in data describing key exposure variates. A parametric model (e.g., lognormal) can be fit to measures of, for example, food consumption in a representative sample of a population in order to characterize the variability of that variate for a population. The stochastic approach uses a distribution of values, or a parametric model for the distribution, as input for one or more variates in the model. Risk estimates can be expressed as a distribution by propagating the variance of exposure variates through the model using Monte Carlo simulation. This allows estimation of some of the variability in exposure in the risk estimate.

The primary benefits of stochastic analysis are the quantitative treatment of some of the variability in risk estimates and the increase in information on which to base decisions. The risk manager can determine what percentage of the population would be protected if emissions were reduced by a certain amount. However, it can be difficult to

communicate the results of a stochastic risk assessment to the public and risk managers.

Better characterization of total variability in exposure would require more research. Typical intake rates for various age ranges and longitudinal data on the same individuals over time are not usually available. Short term survey data on representative samples of populations of interest are all that are available for many variates. Such data can overestimate exposure particularly in the upper percentiles when considerable intraindividual variability occurs. Some important exposure variates such as soil ingestion lack sufficient data to characterize variability.

Neither the stochastic approach nor the point estimate approach to exposure assessment presented in this document deals with uncertainty or variability in the dose-response assessment. While human variability in response to toxicants is an increasingly active area of research, more data are needed to better account for human interindividual variability in risk assessments. We have evaluated the impact of age-at-exposure on carcinogenic potency (OEHHA, 2009). As noted above, that analysis resulted in application of ASFs to account semi-quantitatively for variability in response to carcinogens due to age. OEHHA also modified the methodology for developing Reference Exposure Levels (OEHHA, 2008) to more explicitly account for potential sensitivity of infants and children.

OEHHA carefully evaluated the available literature characterizing variability for important exposure variates. Even though in some cases there were studies presenting valid parametric models for exposure variates in the literature, the age ranges did not correspond to our current needs. In other cases, we obtained unpublished raw data from published studies or performed our own analyses on publically available databases such as the Continuing Survey of Food Intake for Individuals (CSFII) or the National Health and Nutrition Examination Survey (NHANES). The methodology is described in the individual chapters in this document as well as in the peer reviewed scientific literature for some variates. If the data or studies were not adequate to characterize variability in a variate (e.g., soil ingestion) point estimates are recommended.

We have taken the approach that enough data must be available to adequately characterize a distribution. While some papers in the risk assessment literature make speculative assumptions about the shape of an input distribution in the absence of data, this cannot be readily justified in most cases. Additional assumptions regarding a distribution in the absence of data may increase uncertainty and may not improve the knowledge about the range of risks in a population.

Distributions of exposure variates are presented in this document for the age ranges needed to assess cancer risk using the age sensitivity factors for specific age groups.

Thus, estimation of dose using the stochastic approach for the various age groupings is similar to the point estimate approach. The intake distributions for ages 16 to 30 years are generally used for women in their third trimester of pregnancy if intake data specific

for this group is lacking. Distributions for the ages specified In Section 1.2.1 above should be used to determine the dose ranges.

1.4 Tiered Approach to Risk Assessment

During the development of risk assessment guidelines for the Hot Spots program, a number of stakeholders wanted the option of using non-default site-specific point estimates and distributions for assessing exposure where more appropriate. Thus OEHHA developed a tiered approach to accommodate this concern (Table 1). The first Tier is the simplest point estimate approach to estimating exposure to facility emissions. In Tier 1, the risk assessor must use the point estimates developed by OEHHA for all exposure variates, other than obvious site-specific parameters such as the volume of a body of impacted water. Tier 2 allows use of site-specific point estimates of exposure variates as long as these estimates can be justified. The risk assessor must supply the data and methods used for the site-specific estimates, and the site-specific estimates must be reproducible and justified, and approved by OEHHA. Tier 3 allows use of OEHHA-derived distributions of a number of exposure variates so that a “likelihood of risks” approach can be utilized, as called for in the statutory language. This allows one to estimate risk based on a distribution of exposures, rather than a single point estimate. Tier 4 allows use of site-specific distributions of exposure parameters as long as they can be justified and are approved by OEHHA. The risk assessor must supply the data and methods used for the site-specific distributions for exposure variates, and the site-specific estimates must be reproducible and justified.

Most facilities in the Air Toxics “Hot Spots” program may not require a complicated stochastic analysis for sufficient characterization of risks from emissions. In order to allow the level of effort in a risk assessment to be commensurate with the importance of the risk management decision, a tiered approach to risk assessment is recommended. The tiers are meant to be applied sequentially to retain consistency across the state in implementing the Air Toxics “Hot Spots” program while allowing flexibility.

The benefits of a tiered approach to site-specific risk assessment include consistency across the state, comparability across facilities, and flexibility in the approach to assessing risks. A simple health-protective point estimate risk assessment will indicate whether a more complex approach is warranted, and will help prioritize limited resources. The tiered risk assessment approach facilitates use of site-specific supplemental information in the risk assessment to better characterize the risks. Finally, more information is available to risk managers and the public when a tiered approach is fully utilized.

TABLE 1 – THE TIERED APPROACH TO RISK ASSESSMENT

Tier	Description	When Applied
Tier 1	Utilizes OEHHA default point estimates of exposure variates	All risk assessments must include a Tier 1 assessment
Tier 2	Utilizes site-specific point estimates for exposure variates (justified, and approved by OEHHA)	If desired by risk assessor, a Tier 2 approach may be presented in addition to Tier 1
Tier 3	Utilizes OEHHA distributions of exposure variates	A Tier 3 approach may be presented in addition to Tier 1
Tier 4	Utilizes site-specific justified distributions of exposure variates (justified, and approved by OEHHA)	A Tier 4 approach may be presented in addition to Tier 1

1.4.1 Tier 1

Tier 1 is the first step in conducting a comprehensive risk assessment with a point estimate approach, using algorithms and point estimates of input values presented in the following chapters. Each facility conducts a Tier 1 risk assessment to promote consistency across the state for all facility risk assessments and allow comparisons across facilities.

Condensed guidance, including tables of the point estimate values recommended by OEHHA in the following chapters, is given in the companion document *Air Toxics Hot Spots Risk Assessment Guidance Manual*, which we are in the process of updating. Site-specific values (e.g. the volume of water in an impacted lake) have to be provided by the risk assessor.

Mean and high-end point estimates for key exposure variates were estimated by OEHHA from available data. To be health-protective, high-end estimates for the key intake exposure variates are used for the dominant pathways in Tier 1.

If a risk assessment involves multipathway exposures, then the risk assessor needs to evaluate which pathways are dominant by conducting an initial assessment using the high-end point estimates for those key intake variates, that have been evaluated by OEHHA. Dominant pathways are defined for these purposes as the two pathways that contribute the most to the total cancer risk estimate when using high-end estimates of key intake variates. High-end estimates for key intake variates for the two dominant pathways and mean values for key variates in the exposure pathways that are not dominant are then used to estimate risks. If the food pathway is the dominant pathway, then the highest single produce or meat type (e.g., exposed produce) using the high

end estimates should be determined. The risk for the other food pathways then should be estimated using the average intake values.

This approach will lessen the problem of compounding high-end exposure estimates while still retaining a health-protective approach for the more important exposure pathway(s). It is unlikely that any one person would be on the high-end for all the intake variates. It is our experience that inhalation is generally a dominant pathway posing the most risk in the Air Toxics "Hot Spots" program; occasionally risks from other pathways may also be dominant for lipophilic compounds or metals. Therefore, for many facilities emitting volatile chemicals, the inhalation pathway will be the only pathway whose risks are assessed using a high-end intake estimate. For the Air Toxics "Hot Spots" program, the point of maximum impact for cancer risks is the location with the highest risks using this method.

OEHHA is recommending the hazard index (HI) approach to assess the potential for noncancer health impacts (OEHHA, 2008). The hazard index is calculated by dividing the concentration in air by the Reference Exposure Level for the substance in question and summing the ratios for all chemicals impacting the same target organ (OEHHA, 2008).

There may be instances where a noninhalation pathway of exposure contributes substantially to a noncancer chronic hazard index. In these cases, the high-end estimate of dose is appropriate to use for the two dominant pathways' noninhalation hazard indices. The point of maximum impact for noncancer chronic health effects is the modeled point having the highest non cancer chronic hazard index (adding noninhalation and inhalation hazard indices when appropriate for systemic effects). The inhalation chronic HI calculation does not involve a high end and average inhalation rate, as the airborne concentration is divided by the REL to calculate an HI (OEHHA, 2008).

There are 8-hour RELs for a number of chemicals. These RELs can be used in different exposure scenarios, such as, to evaluate noncancer risk to offsite workers (and other offsite receptors impacted routinely by facility emissions) who are repeatedly exposed for approximately eight hours at the workplace. The 8 hr RELs may also be useful for assessing impacts to residents when assessing the emissions from a non-continuously operating facility (see Chapter 2). In cases where there are only chronic RELs for a chemical, the Hazard Index for offsite workers can be calculated by adding the Hazard Quotient for a chemical with an 8-hour REL to a chemical where only a chronic REL is available. Eventually 8-hour and chronic RELs will be developed for all Hot Spots chemicals as OEHHA completes its evaluation of RELs under SB-25. There are no noninhalation pathways to consider in calculation of acute hazard indices.

The relatively health-protective assumptions incorporated into the Tier 1 risk assessment (e.g., high-end values for key variates in the driving pathways) make it unlikely that the risks are underestimated for the general population. If the results indicate that a facility's estimated cancer risk and noncancer hazard are below the level

of regulatory concern, further analysis may not be warranted. If the results are above a regulatory level of concern, the risk assessor may want to proceed with further analysis as described in Tier 2 or a more resource-intensive stochastic modeling effort described in Tiers 3 and 4 to provide the risk manager with more information on which to base decisions. While further evaluation may provide more information to the risk manager, the Tier 1 evaluation is useful in comparing risks among a large number of facilities.

1.4.2 Tier 2

The risk assessor may want to analyze the risks using point estimates more appropriate for the site being evaluated. This second tier approach would replace some of the defaults recommended in this document with values more appropriate to the site. A Tier 2 risk assessment would use the point estimate approach with justifiable point estimates for important site-specific variates. Use of this supplemental site-specific information may help to better characterize the risks.

Certain exposure variates such as breast milk consumption or inhalation rate would not be expected to vary much from site to site. Other variates for which OEHHA has provided point estimates may vary significantly from site-to-site. If the facility has data indicating that an OEHHA point estimate value is not appropriate in their circumstance, they may provide an alternative point estimate value. For example, if there are data indicating that consumption of fish from an impacted fishable body of water is lower than the OEHHA-recommended fish consumption rate, then the facility can use those data to generate a point estimate for fisher-caught fish consumption from that body of water.

If site-specific values are substituted, the values need to be justified. All data and procedures used to derive them should be clearly documented, and reasonable justification should be provided for using the alternative value. The Districts and OEHHA should be able to reproduce the point estimate from the data presented in the risk assessment. As noted above, OEHHA must approve the site-specific point estimates.

In a Tier 2 approach, the risk assessor may want to present multiple alternative point estimate scenarios with several different assumptions encompassing reasonable “average” and “high-end” exposures for important pathways. This may be an issue in the case where data on a key exposure variate for that particular site are lacking. For example, in a case where soil ingestion is a dominant pathway, if a key variate in the model is the number of days children spend outdoors in contact with soil, it may be most appropriate to run the model more than once using several different assumptions about the exposure frequency. Such scenario development is easily communicated to the risk manager and the public, and serves as a semi-quantitative analysis of the exposure variability using a point estimate approach to risk assessment. In any risk assessment where alternative point estimates representing different exposure scenarios are presented, all information used to develop the point estimates needs to be presented clearly in the risk assessment. Also, a justification for the exposure scenarios needs to be included.

If the risk is below a level of regulatory concern, further analysis may not be warranted. If the risk estimate is still above a level of concern, then the risk assessor may want to proceed with a more complex stochastic analysis as described in Tier 3 to get a fuller characterization of the variability in the exposure estimate.

1.4.3 Tier 3

The third tier risk assessment involves stochastic analysis of exposure using algorithms and distributions for the key exposure variates specified in this document. Point estimates specified in this document for those exposure variates without distributions should be used. Since a stochastic approach to risk assessment provides more information about the range and probability of risk estimates, Tier 3 can serve as a useful supplement to the Tier 1 and 2 approach. In the third tier, variance propagation methods (e.g., Monte Carlo analysis) are used to derive a range of risk estimates reflecting the known variability in the inputs as described in the distributions characterized in this document. Recommended distributions for use in a stochastic analysis and the scientific bases for these distributions are provided in Chapters 3 through 9 of this document.

OEHHA is recommending that a stochastic analysis be performed for cancer risk assessment only. OEHHA has not currently identified a stochastic approach to the exposure part of noncancer risk assessment that would provide value. OEHHA is recommending a point estimate approach only for assessing the impact of AB-2588 facilities on workers employed at nearby work sites (i.e., the offsite worker). We have not developed a breathing rate distribution that would be appropriate for a stochastic offsite worker risk assessment.

Commercial software is available that can be used to conduct a stochastic analysis. The Air Resources Board has developed the Hot Spots Analysis and Reporting Program (HARP) that can perform Tier 3 stochastic analyses as well as Tier 1 risk assessments. The HARP software includes an air modeling module and emissions reporting modules.

1.4.4 Tier 4

OEHHA's stochastic model is based on the best available scientific data that have undergone public comment and peer review. However, a fourth tier risk assessment could also be conducted if site-specific conditions suggest that alternative or additional distributions (and point estimates) for variates may be more appropriate than those provided by OEHHA. In a Tier 4 risk assessment, the risk assessor could characterize the distribution of variates that are important to the overall calculation of risk for which OEHHA provides only a point estimate. Or, the risk assessor may wish to use distributions other than those supplied by OEHHA for important variates that impact the risk. The scientific basis and documentation for alternative and additional distributions should be presented clearly in the risk assessment. Clear, reasonable justification

would need to be provided in the risk assessment for using alternative distributions or point estimates, and OEHHA must approve the site-specific distributions. Such distributions would be based on data from the literature or site-specific data gathered by the facility.

The quality of data would need to be sufficient to reasonably justify the selection of the parametric model (e.g., normal, lognormal, etc.) used to characterize the empirical distribution. It is not necessary, however, that the data fit a given parametric model as defined by conservative statistical criteria such as the Kolmogrov-Smirnoff test. If a distribution is nonparametric, it may be used as a custom distribution in a variance propagation model such as a Monte Carlo simulation.

In each case where alternate distributions or point estimates are used, it is important that the results be compared with the results obtained using any point estimates and/or distributions recommended in this document by OEHHA (e.g., the Tier 1 and 3 risk assessments). This is necessary to identify the contribution of the new information to the risk assessment. The District and OEHHA staff and any interested parties should be able to easily verify the assumptions, and duplicate the results.

1.5 Exposure Assessment Pathways

Chapters 3 through 10 are organized by exposure pathway, and present the algorithms used for both the point estimate and stochastic approach to exposure assessment. The scientific basis for each recommended point estimate and distribution for key variates is presented. In the instances where the variate is site-specific (e.g., volume of a body of water), default point estimates or distributions are not provided. In general, key studies used in evaluating a point estimate value or distribution are briefly discussed along with procedures used to characterize the distribution. OEHHA procedure for significant figures is to round at the end of any calculation. Thus the exposure variates are generally rounded to 2 or 3 significant figures. The risk estimates are generally rounded to 1or 2 significant figures in the risk assessments conducted by facilities.

1.6 Individual Risk, versus Population Risk, and Duration of Exposure to Facility Emissions

In past practice, the risk managers generally made decisions on the lifetime cancer risk to the “Maximally Exposed Individual” at the site of highest modeled concentration(s) of carcinogen(s). However, relying on estimated cancer risk to the maximally exposed individual is problematic for scenarios where there may be a risk of cancer that falls below the typical risk management threshold of 10^{-5}, but a large number of people are exposed at that level. Facilities with cancer risks estimated above 10^{-5} but that expose few people may face risk management actions, but a facility that exposed thousands of people just below the risk management threshold would not. Both the concept of population risk and individual risk are important for public health protection (discussed in Chapter 11).

In trying to resolve this dilemma, OEHHA reconsidered the issues of individual risk, population risk, duration of time at a single residence and activity patterns. The previous recommendation for risk managers was to rely on the 70 year risk estimate without consideration of whether or not people resided at the same address for 70 years, or were away from home parts of the day. The previous guidelines also suggested estimating cancer risk for shorter residence times (9 and 30 years, based on EPA analyses of duration of residence at a single address). Thirty years is approximately the 90^{th} percentile of residency in California, according to newer data and is consistent with estimates of thirty years for the 90^{th} percentile of residency duration nationally, and is thus a more realistic portrayal of the maximum reasonable length of exposure that would occur at the residential point of maximal impact. The previous recommendation of relying on the cancer risk estimate to the maximally exposed individual for a 70 year exposure duration contained an element of protection for the population since individual exposure was defined as an entire lifetime, although the risk was likely spread over different individuals living at the maximally exposed location since very few people live at the same address longer than 30 years. Presenting individual cancer risk as a thirty year risk rather than a seventy year risk is easier from a risk communication standpoint because it is a more realistic exposure scenario. OEHHA is thus suggesting that the risk manager when making a decision based on cancer risk to the MEIR use the risk estimated for a 30 year exposure scenario. However, this lessens the element of protection for the population – someone is always living around a given facility. Thus, OEHHA makes a recommendation to consider population risk separately in assessing public health impacts (Chapter 11).

In the example above, there will be more theoretical cancer cases when a larger facility with estimated cancer risk just under the 10^{-5} threshold has a large populated zone of impact, than for the small facility impacting a few people with a cancer risk estimate just over the 10^{-5} threshold. The public health impacts may not be adequately addressed if the cancer risks at the residential or worker point of maximum impact are below the level of significant risk determined by the District. It is important to look at improved methods of assessing the public health impact of facilities with more diffuse emissions impacting larger areas with large impacted populations. Therefore, OEHHA recommends that the number of people residing within the 1×10^{-6} and greater cancer risk isopleths be determined using census data and that the risk managers use this information to decide on appropriate risk management. This is in addition to simply basing a risk management decision on the cancer risk to the maximally exposed individual without regard to the size of the zone of impact and the population exposed. Strengthening population protection will help protect public health.

1.7 SB-352

SB-352 was passed in 2003 and requires California school districts to perform a risk assessment for proposed school sites located within 500 feet, or 150 m, of a freeway or busy roadway. SB-352 specifies that OEHHA's Hot Spots risk assessment guidance procedures be used for the assessment. School children and staff are present at the school site for less than 24 hours so hourly breathing rates that reflect playground activities and classroom activities are appropriate for such assessments. We have included recommended breathing rates in Chapter 3 of this document for appropriate age ranges for elementary, junior high and high school and staff at such schools for such assessments. The age ranges provided also allow for early-in-life exposure age ranges. The South Coast Air Quality Management District has a document that discusses air quality concerns when selecting school sites (SCAMD, 2005).

1.8 Summary

This revision of the Exposure Assessment and Stochastic Analysis Document allows estimation of exposure for age ranges of children. In addition we have incorporated advances in the field of exposure assessment since the last revision and new point estimates and distributions of exposure variates, based on new data. The Exposure Assessment and Stochastic Analysis document retains the option of tiered risk assessment so that site-specific factors can be taken into account.

OEHHA has reviewed and incorporated the extensive body of exposure assessment literature that has been published since the 2000 Exposure and Stochastic Analysis Technical Support Document in order to refine our exposure assessment model.

1.9 References

OEHHA (1999a). *Air Toxics Hot Spots Risk Assessment Guidelines Part I: Technical Support Document for the Determination of Acute Reference Exposure Levels for Airborne Toxicants*. Office of Environmental Health Hazard Assessment, Cal/EPA. March 1999.

OEHHA (1999b). *Air Toxics Hot Spots Risk Assessment Guidelines Part II: Technical Support Document for Describing Available Cancer Potency Factors*. Office of Environmental Health Hazard Assessment, Cal/EPA. April 1999.

OEHHA (2000a). *Air Toxics Hot Spots Risk Assessment Guidelines Part IV: Technical Support Document for Exposure Assessment and Stochastic Analysis*, Office of Environmental Health Hazard Assessment, Cal/EPA.

OEHHA (2000b). *Air Toxics Hot Spots Risk Assessment Guidelines Part III: Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels for Airborne Toxicants*. Office of Environmental Health Hazard Assessment, Cal/EPA. February 2000.

OEHHA (2008). *Air Toxics Hot Spots Risk Assessment Guidelines. Technical Support Document for the Derivation of Non-cancer Reference Exposure Levels.* Office of Environmental Health Hazard Assessment, Cal/EPA.

OEHHA (2009). *Air Toxics Hot Spots Risk Assessment Guidelines.. Technical Support Document for Cancer Potency Factors: Methodologies for Derivation, Listing of Available Values, and Adjustments to Allow for Early Life Stage Exposures* Office of Environmental Health Hazard Assessment, Cal/EPA.

SCAQMD (2005) South Coast Air Quality Management District (SCAQMD) Air Quality Issues in School Site Selection Guidance Document June 2005 www.aqmd.gov/prdas/aqguide/doc/School_Guidance.pdf

U.S. EPA (1991) Risk Assessment Guidance for Superfund: Volume I –Human Health Evaluation Manual. Office of Emergency and Remedial Response, U.S. Environmental Protection Agency, Washington, DC 20460 EPA/540/R-92/003 Publication 9285.7-01 B December 1991

U.S. EPA (1995). Policy for Risk Characterization at the U.S. Environmental Protection Agency. Memorandum from Carol Browner to Administrators, U.S. Environmental Protection Agency, Washington, D.C., March 21, 1995.

2 Air Dispersion Modeling

2.1 Air Dispersion Modeling in Risk Assessment: Overview

Estimates of air concentrations of emitted toxicants in the surrounding community from a facility's air emissions are needed in order to determine cancer and noncancer risks. One approach to determining the concentration of air pollutants emitted from the facility is to do air monitoring in the surrounding community. However, there are a number of disadvantages to this approach. Ambient air monitoring is costly because good estimates of an annual average concentration typically require monitoring at least one day in six over a year. Because it is costly, monitoring is usually limited to a select number of pollutants, and a limited number of sites. There can be significant risks from some chemicals at or even below the monitoring detection limit, which can add considerable uncertainty to risk estimates if many of the measurements are below or near the detection limit. Monitoring measures not only facility emissions but also general ambient background as well. It can be difficult and expensive to distinguish between the two using monitoring, particularly if general ambient background levels are high relative to the contribution of facility emissions. These limitations often make it impractical to use monitoring in a program such as the Air Toxics Hot Spots program with hundreds of facilities.

Air dispersion models have several advantages over monitoring. Modeling can provide greater spatial detail and the costs are relatively cheap by comparison. For example, dispersion models can estimate the pollutant concentration in air at many receptor locations (hundreds to thousands) and for a multitude of averaging periods. Air dispersion models have been validated using air monitoring.

There are, however, uncertainties associated with the typical usage of air dispersion modeling. The use of meteorological data from the nearest airport may not ideally be the best representation of localized conditions. Gaussian plume air dispersion models ignore calm hours. This can bias model predictions towards underestimation. Some dispersion models offer limited chemical reactions within the algorithms; however, we generally assume the pollutant is inert for the near-field atmospheric travel time. This may bias estimated concentrations towards over-prediction for those pollutants that are highly reactive in the atmosphere. Air dispersion model results are only as good as the emissions estimates and emissions estimates can be uncertain. However, on the whole, the advantages of air dispersion modeling for a program like the Air Toxics Hot Spots far outweigh the disadvantages.

Professional judgment is required throughout the dispersion modeling process. The local air quality district has final authority on modeling protocols. The following guidance is intended to assist in the understanding of dispersion modeling for risk assessments.

Air dispersion modeling includes the following steps (see Figure 1):

(1) Create an emission inventory of the toxic releases (Section 2.2)

(2) Identify the source types (Section 2.3)

(3) Identify the terrain type (Section 2.4)

(4) Determine the detail needed for the analysis: screening or refined (Section 2.5)

(5) Identify the population exposure (Section 2.6)

(6) Identify the receptor network (Section 2.7)

(7) Obtain meteorological data (for refined air dispersion modeling only) (Section 2.8)

(8) Select an air dispersion model (Section 2.9)

(9) Prepare a modeling protocol and submit to the local Air District (hereafter referred to as “the District”) (Section 2.14)

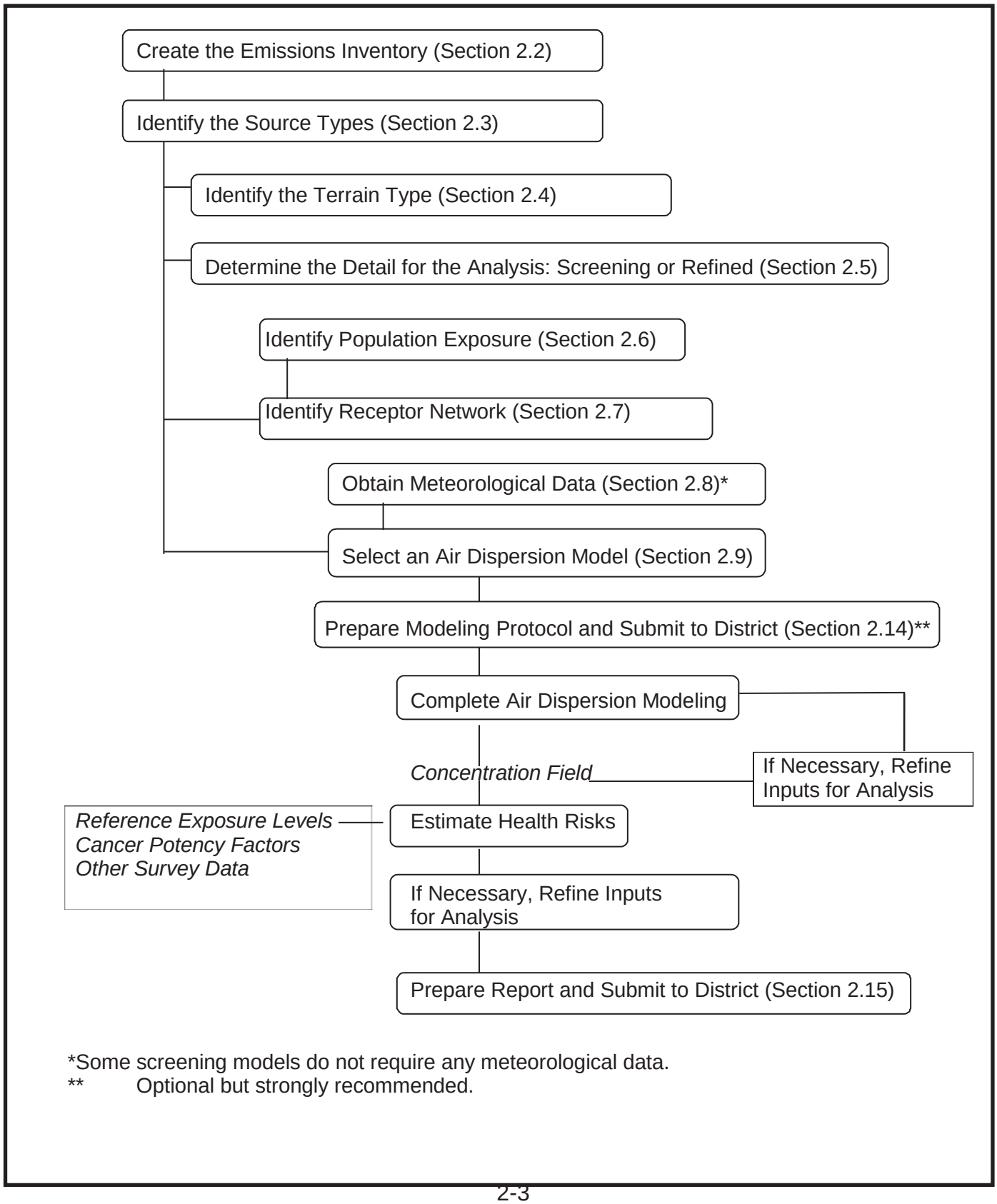
(10) Complete the air dispersion analysis

(11) If necessary, redefine the receptor network and return to Step 10

(12) Complete the risk assessment

(13) If necessary, refine the inputs and/or the model selection and return to Step 8

FIGURE 1. OVERVIEW OF THE AIR DISPERSION MODELING PROCESS.



*Some screening models do not require any meteorological data.
** Optional but strongly recommended.

The output of the air dispersion modeling analysis includes a receptor field of ground level concentrations of the pollutant in ambient air. These concentrations can be used to estimate an inhaled dose for estimation of inhalation cancer risk, or used to determine a hazard index for acute, and chronic noncancer risks. It should be noted that in the Air Toxics "Hot Spots" program, facilities simulate the dispersion of the chemical emitted as an inert compound, and do not model any atmospheric transformations or dispersion of products from such reactions. The U.S. EPA Guideline on Air Quality Models (U.S. EPA, 2005) should be consulted when evaluating reactive pollutants for other regulatory purposes.

2.2 Emission Inventories

The Emission Inventory Reports ("Inventory Reports"), developed under the Air Toxics "Hot Spots" Information and Assessment Act (AB2588), contain data that are used in air dispersion modeling and risk assessment evaluations. The Inventory Reports include emission sources, emitted substances, emission rates, emission factors, process rates, and release parameters (area and volume sources may require additional release data generally available in Emissions Inventory Reports). This information is developed according to the California Air Resources Board (CARB) Emission Inventory Criteria and Guidelines ("Inventory Guidelines") Regulation[1] and the Emission Inventory Criteria and Guidelines Report ("Inventory Guidelines Report"), which is incorporated by reference into the Regulation.

Updated emission data for process changes, emission factor changes, material/fuel changes, or shutdown must be approved by the District prior to the submittal of the health risk assessment (HRA). Ideally, the District review of updated emissions could be completed within the modeling protocol. In addition, it must be stated clearly in the risk assessment if the emission estimates are based on updated or revised emissions (e.g., emission reductions). This section summarizes the requirements that apply to the emission data which are used for Air Toxics "Hot Spots" Act risk assessments.

2.2.1 Air Toxics "Hot Spots" Emissions

2.2.1.1 Substances Emitted

The risk assessment should identify all substances emitted by the facility which are on the Air Toxics "Hot Spots" Act list of substances (Appendix A I-III, Inventory Guideline Report). The list of substances is compiled by the CARB for the Air Toxics "Hot Spots" Program.

[1] Title 17, California Code of Regulations, Sections 93300-93300.5

The Inventory Guidelines specify that Inventory Reports must identify and account for all listed substances used, manufactured, formulated, or released during the routine and predictable operations of the facility (e.g., including, but not limited to, continuous and intermittent releases and predictable process upsets or leaks). Under the regulations, the list is divided into three groups for reporting purposes[2]. The first group (listed in Appendix A-I of the Inventory Guidelines Report) has all pollutants whose emissions must be quantified. The second group (listed in Appendix A-II of the Inventory Guidelines Report) includes substances where emissions do not need to be quantified; however, facilities must report whether the substance is used, produced, or otherwise present on-site. The third group (listed in Appendix A-III of the Emissions Inventory Guidelines Report) includes substances whose emissions need not be reported unless the substance is manufactured by the facility. Chemicals or substances in the second and third groups should be listed in a table in the risk assessment.

Facilities that must comply with the Resource Conservation and Recovery Act and Comprehensive Environmental Response, Compensation and Liability Act (RCRA/CERCLA) requirements for risk assessment need to consult the Department of Toxic Substances Control (DTSC) Remedial Project Manager to determine which substances must be evaluated in their risk assessment in addition to the list of "Hot Spots" chemicals. Some RCRA/CERCLA facilities may emit chemicals that are not currently listed under the "Hot Spots" Program.

2.2.1.2 Emission Estimates Used in the Risk Assessment

The risk assessment must include emission estimates for all substances that are required to be quantified in the facility's emission inventory report. Specifically, risk assessments should include both the annual average emissions and maximum 1-hour emissions for each pollutant. Emissions for each substance must be reported for the individual emitting processes and devices within a facility. Total facility emissions for an individual air contaminant will be the sum of emissions reported, by process, for that facility. Information on daily and annual hours of operation and relative monthly activity must be reported for each emitting process. Devices and emitting processes must be clearly identified and described and must be consistent with those reported in the emissions inventory report.

The HRA should include tables that present the emission information (i.e., emission rates for each substance released from each process) in a clear and concise manner. The District may allow the facility operator to base the HRA on more current emission estimates than those presented in the previously submitted emission inventory report (i.e., actual enforceable emission reductions realized by the time the HRA is submitted to the District). If the District allows the use of more current emission estimates, the

[2] The most recent amendments became effective September 26, 2007.

District must review and approve the new emissions estimates prior to use in the risk assessment. The risk assessment report must clearly state what emissions are being used and when any reductions became effective. Specifically, a table identifying both the previous and current emission estimates should be included. The District should be consulted concerning the specific format for presenting the emission information.

Facilities that must also comply with RCRA/CERCLA requirements for risk assessments need to consult the DTSC Remedial Project Manager to determine what constitutes appropriate emissions data for use in the risk assessment. Source testing may be required for such facilities even if it is not required under the “Hot Spots” Program. Additional requirements for statistical treatment of source test results may also be imposed by the DTSC on RCRA/CERCLA facilities.

2.2.1.3 Emission Release Parameters

Emission release parameters (e.g., stack height and inside diameter, stack gas exit velocity, release temperature and emission source location in UTM coordinates) are needed as inputs to the air dispersion model. The Inventory Guidelines specify the release parameters that must be reported for each stack, vent, ducted building, exhaust site, or other site of exhaust release. Additional information may be required to characterize releases from non-stack (volume and area) sources; see U.S. EPA dispersion modeling guidelines or specific user's manuals. This information should also be included in the air dispersion section of the risk assessment. This information must be presented in tables included in the risk assessment. Note that some dimensional units needed for the dispersion model may require conversion from the units reported in the Inventory Report (e.g., Kelvin (K) vs. degrees Fahrenheit (°F)).

2.2.1.4 Operation Schedule

The risk assessment should include a discussion of the facility operation schedule and daily emission patterns. Weekly or seasonal emission patterns may vary and should be discussed. This is especially important in a refined risk assessment. Diurnal emission patterns should be simulated in the air dispersion model because of diurnal nature of meteorological observations. A table should be included with emission schedule on an hourly and yearly basis. In addition, for the purposes of exposure adjustment, the emission schedule and exposure schedule should corroborate any exposure adjustment factors. For more information about exposure adjustment factors, see Section 2.8(a). Alternatively, exposure adjustment can be made through refining the air dispersion analysis. See Section 2.11.1.2(h) for special case modeling.

2.2.1.5 Emission Controls

The risk assessment should include a description of control equipment, the emitting processes it serves, and its efficiency in reducing emissions of substances on the Air Toxics “Hot Spots” list. The Inventory Guidelines require that this information be included in the Inventory Reports, along with the emission data for each emitting

process. If the control equipment did not operate full-time, the reported overall control efficiency must be adjusted to account for downtime of control equipment. Any entrainment of toxic substances to the atmosphere from control equipment should be accounted for; this includes fugitive releases during maintenance and cleaning of control devices (e.g., baghouses and cyclones).

2.2.2 Landfill Emissions

Emission estimates for landfill sites should be based on testing required under Health and Safety Code Section 41805.5 (AB 3374, Calderon) and any supplemental AB 2588 source tests performed to characterize air toxics emissions from landfill surfaces or through off-site migration. The District should be consulted to determine the specific Calderon data to be used in the risk assessment. The Air Toxics "Hot Spots" Program risk assessment for landfills should also include emissions of listed substances for all applicable power generation and maintenance equipment at the landfill site. Processes that need to be addressed include stationary IC engines, flares, evaporation ponds, composting operations, boilers, and gasoline dispensing systems.

2.3 Source Characterization

Pollutants are released into the atmosphere in many different ways. The release conditions need to be properly identified and characterized to appropriately use the air dispersion models.

2.3.1 Source Type

Source types can be identified as point, line, area, or volume sources for input to the air dispersion model. Several air dispersion models have the capability to simulate more than one source type.

2.3.1.1 Point Sources

Point sources are probably the most common type of source and most air dispersion models have the capability to simulate them. Typical examples of point sources include: isolated vents and stacks.

2.3.1.2 Line Sources

In terms of modeling, line sources are treated as a special case of either an area or a volume source. Consequently, they are normally modeled using either an area or volume source model as described below. Examples of line sources include: conveyor belts and rail lines, freeways, and busy roadways. Mobile sources and rail lines do not come under the purview of the Hot Spots program, but they are required to be evaluated under SB-352. SB-352 requires a risk assessment performed under the Hot Spots risk assessment guidance for proposed school sites within 500 feet of a busy roadway. Dedicated air dispersion models are available for motor vehicle emissions

from roadways which are a special type of line source. These models (i.e., CALINE3, CAL3QHCR, and CALINE4) are designed to simulate the mechanical turbulence and thermal plume rise due to the motor vehicle activity on the roadway. However, these dedicated models use the Pasquill-Gifford dispersion stability classes for dispersion; the AERMOD dispersion model uses a more advanced continuous stability estimation method based on observations. The limitation with AERMOD is that the user needs to estimate initial mixing (Szo, and Syo) for mechanical turbulence and thermal plume rise is not available. Consult with the District prior to conducting roadway modeling to determine model use.

For practical information on how to simulate roadway emission dispersion using these models, see the California Air Pollution Control Officer's Association (CAPCOA) website at http://www.capcoa.org or the Sacramento Metropolitan AQMD (SMAQMD) website at http://www.airquality.org/ceqa/RoadwayProtocol.shtml. The SMAQMD has a document titled, "Recommended Protocol for Evaluating the Location of Sensitive Land Uses Adjacent to Major Roadways"(January, 2010). The ARB recommends this document for SB-352 risk assessments.

2.3.1.3 Area Sources

Emissions that are to be modeled as area sources include fugitive sources characterized by non-buoyant emissions containing negligible vertical extent of release (e.g., no plume rise or distributed over a fixed level).

Fugitive particulate ($PM_{2.5}$, PM_{10}, TSP) emission sources include areas of disturbed ground (open pits, unpaved roads, parking lots) which may be present during operational phases of a facility's life. Also included are areas of exposed material (e.g., storage piles and slag dumps) and segments of material transport where potential fugitive emissions may occur (uncovered haul trucks or rail cars, emissions from unpaved roads). Fugitive emissions may also occur during stages of material handling where particulate material is exposed to the atmosphere (uncovered conveyors, hoppers, and crushers).

Other fugitive emissions emanating from many points of release may be modeled as area sources. Examples include fugitive emissions from valves, flanges, venting, and other connections that occur at ground level, or at an elevated level or deck if on a building or structure. Modern dispersion models include an option for an initial vertical extent (Szo) where needed.

2.3.1.4 Volume Sources

Non-point sources where emissions include an initial vertical extent should be modeled as volume sources. The initial vertical extent may be due to plume rise or a vertical distribution of numerous smaller sources over a given area. Examples of volume sources include buildings with natural fugitive ventilation, building roof monitors, and line sources such as conveyor belts and rail lines.

2.3.2 Quantity of Sources

The number of sources at a facility may influence the selection of the air dispersion model. Some dispersion models are capable of simulating only one source at a time, and are therefore referred to as single-source models (e.g., AERSCREEN).

In some cases, for screening purposes, single-source models may be used in situations involving more than one source using one of the following approaches:

- combining all sources into one single “representative” source

In order to be able to combine all sources into one single source, the individual sources must have similar release parameters. For example, when modeling more than one stack as a single “representative” stack, the stack gas exit velocities and temperatures must be similar. In order to obtain a conservative estimate, the values leading to the higher concentration estimates should typically be used (e.g., the lowest stack gas exit velocity and temperature, the height of the shortest stack, and a receptor distance and spacing that will provide maximum concentrations, etc.).

- running the model for each individual source and superimposing results

Superimposition of results of single sources of emissions is the actual approach followed by all the Gaussian models capable of simulating more than one source. Simulating sources in this manner may lead to conservative estimates if worst-case meteorological data are used or if the approach is used with a model that automatically selects worst-case meteorological conditions, especially wind direction. The approach will typically be more conservative the farther apart the sources are because each run would use a different worst-case wind direction.

Additional guidance regarding source merging is provided by the U.S. EPA (1995a). It should be noted that depending upon the population distribution, the total burden can actually increase when pollutants are more widely dispersed. If the total burden from the facility or zone of impact (see Section 2.6.1) could increase for the simplifying modeling assumptions described above, the District should be consulted.

2.4 Terrain Type

Two types of terrain characterizations are needed for input to the appropriate model. One classification is made according to land use and another one according to topography.

2.4.1 Terrain Type – Land Use

Some air dispersion models (e.g., CALINE) use different dispersion coefficients (sigmas) depending on the land use over which the pollutants are being transported. The land use type is also used by some models to select appropriate wind profile

exponents. Traditionally, the land type has been categorized into two broad divisions for the purposes of dispersion modeling: urban and rural. Accepted procedures for determining the appropriate category are those suggested by Irwin (1978): one based on land use classification and the other based on population.

The land use procedure is generally considered more definitive. Population density should be used with caution and should not be applied to highly industrialized areas where the population density may be low. For example, in low population density areas a rural classification would be indicated, but if the area is sufficiently industrialized the classification should already be "urban" and urban dispersion parameters should be used.

If the facility is located in an area where land use or terrain changes abruptly, for example, on the coast, the District should be consulted concerning the classification. If need be, the model should be run in both urban and rural modes and the District may require a classification that biases estimated concentrations towards overprediction. As an alternative, the District may require that receptors be grouped according to the terrain between source and receptor.

AERMOD is the recommended model for a wide range of applications in rural or urban conditions. AERMOD uses a planetary boundary layer scaling parameter to characterize stability. This approach is a departure from stability categories estimated with the land use procedures. Rather AERMOD preprocessors, AERMET and AERMAP, are used to characterize land type as they process meteorological data and terrain receptors, respectively.

As it applies to plume models other than AERMOD, the Land Use Procedure is described as follows.

2.4.1.1 Land Use Procedure

(1) Classify the land use within the total area A, circumscribed by a 3 km radius circle centered at the source using the meteorological land use typing scheme proposed by Auer (1978) and shown in Table 2.1.

(2) If land use types I1, I2, C1, R2 and R3 account for 50 percent or more of the total area *A* described in (1), use urban dispersion coefficients. Otherwise, use appropriate rural dispersion coefficients.

2.4.1.2 Population Density Procedure

(1) Compute the average population density (*p*) per square kilometer with *A* as defined in the Land Use procedure described above. (Population estimates are also required to determine the exposed population; for more information see Section 2.6.3.)

(2) If p is greater than 750 people/km^2 use urban dispersion coefficients, otherwise, use appropriate rural dispersion coefficients.

TABLE 2.1 IDENTIFICATION AND CLASSIFICATION OF LAND USE TYPES (AUER, 1978)

Used to define rural and urban dispersion coefficients in certain models.

Type	Use and Structures	Vegetation
I1	*Heavy Industrial* Major chemical, steel and fabrication industries; generally 3-5 story buildings, flat roofs	Grass and tree growth extremely rare; <5% vegetation
I2	*Light-moderate industrial* Rail yards, truck depots, warehouses, industrial parks, minor fabrications; generally 1-3 story buildings, flat roofs	Very limited grass, trees almost totally absent; <5% vegetation
C1	*Commercial* Office and apartment buildings, hotels; >10 story heights, flat roofs	Limited grass and trees; <15% vegetation
R1	*Common residential* Single family dwelling with normal easements; generally one story, pitched roof structures; frequent driveways	Abundant grass lawns and light-moderately wooded; >70% vegetation
R2	*Compact residential* Single, some multiple, family dwelling with close spacing; generally <2 story, pitched roof structures; garages (via alley), no driveways	Limited lawn sizes and shade trees; <30% vegetation
R3	*Compact residential* Old multi-family dwellings with close (<2 m) lateral separation; generally 2 story, flat roof structures; garages (via alley) and ashpits, no driveways	Limited lawn sizes, old established shade trees; <35% vegetation
R4	*Estate residential* Expansive family dwelling on multi-acre tracts	Abundant grass lawns and lightly wooded; >80% vegetation
A1	*Metropolitan natural* Major municipal, state, or federal parks, golf courses, cemeteries, campuses; occasional single story structures	Nearly total grass and lightly wooded; >95% vegetation
A2	Agricultural rural	Local crops (e.g., corn, soybean); >95% vegetation
A3	*Undeveloped* Uncultivated; wasteland	Mostly wild grasses and weeds, lightly wooded; >90% vegetation
A4	Undeveloped rural	Heavily wooded; >95% vegetation
A5	*Water surfaces* Rivers, lakes	

2.4.2 Terrain Type - Topography

Surface conditions and topographic features generate turbulence, modify vertical and horizontal winds, and change the temperature and humidity distributions in the boundary layer of the atmosphere. These in turn affect pollutant dispersion and models differ in their need to take these factors into account.

The classification according to terrain topography should ultimately be based on the topography at the receptor location with careful consideration of the topographical features between the receptor and the source. Differentiation of simple versus complex terrain is unnecessary with AERMOD. In complex terrain, AERMOD employs the well-known dividing-streamline concept in a simplified simulation of the effects of plume-terrain interactions. For other plume models, such as SCREEN3, topography can be classified as follows:

2.4.2.1 Simple Terrain (also referred to as “Rolling Terrain”)

Simple terrain is all terrain located below stack height including gradually rising terrain (i.e., rolling terrain). Note that *Flat Terrain* also falls in the category of simple terrain.

2.4.2.2 Intermediate Terrain

Intermediate terrain is terrain located above stack height and below plume height. The recommended procedure to estimate concentrations for receptors in intermediate terrain is to perform an hour-by-hour comparison of concentrations predicted by simple and complex terrain models. The higher of the two concentrations should be reported and used in the risk assessment.

2.4.2.3 Complex Terrain

Complex terrain is terrain located above plume height. Complex terrain models are necessarily more complicated than simple terrain models. There may be situations in which a facility is “overall” located in complex terrain but in which the nearby surroundings of the facility can be considered simple terrain. In such cases, receptors close to the facility in this area of simple terrain will “dominate” the risk analysis and there may be no need to use a complex terrain model. It is unnecessary to determine which terrain dominates the risk analysis for users of AERMOD.

2.5 Level of Detail: Screening vs. Refined Analysis

Air dispersion models can be classified according to the level of detail which is used in the assessment of the concentration estimates as “screening” or “refined”. Refined air dispersion models use more robust algorithms capable of using representative meteorological data to predict more representative and usually less conservative estimates. Refined air dispersion models are, however, more resource intensive than their screening counterparts. It is advisable to first use a screening model to obtain conservative concentration estimates and calculate health risks. If the health risks are

estimated to be above the threshold of concern, then use of a refined model to calculate more representative concentration and health risk estimates would be warranted. There are situations when screening models represent the only viable alternative (e.g., when representative meteorological data are not available).

It is acceptable to use a refined air dispersion model in a "screening" mode for this program's health risk assessments. In this case, a refined air dispersion model is used:

- with worst-case meteorology instead of representative meteorology
- with a conservative averaging period conversion factor to calculate longer term concentration estimates

Note that use of worst case meteorology in a refined model is not the normal practice in New Source Review or Ambient Air Quality Standard evaluation modeling.

2.6 Population Exposure

The level of detail required for the analysis (e.g., screening or refined), and the procedures to be used in determining geographic resolution and exposed population require case-by-case analysis and professional judgment. The District should be consulted before beginning the population exposure estimates and as results are generated, further consultation may be necessary. Some suggested approaches and methods for handling the breakdown of population and performance of a screening or detailed risk analysis are provided in this section.

In addition to estimating individual cancer risk at specific points such as the MEI (maximally exposed individual), OEHHA recommends determining the number of people who reside with the 1×10^{-6}, 1×10^{-5}, $1x\ 10^{-4}$, and higher cancer risk isopleths. The information can be used to assess the population risk.

2.6.1 Zone of Impact

As part of the estimation of the population exposure for the cancer risk analysis, it is necessary to determine the geographic area affected by the facility's emissions. An initial approach to define a "zone of impact" surrounding the source is to generate an isopleth where the total excess lifetime cancer risk from inhalation exposure to all emitted carcinogens is greater than 10^{-6} (one in 1,000,000). For noncarcinogens, a second and third isopleth (to represent both the chronic and acute impacts) should be created to define the zone of impact for the hazard index from both inhalation and noninhalation pathways greater than or equal to 1.0. For clarity these isopleths may need to be presented on separate maps in the HRA.

The initial "zone of impact" can be determined as follows:

- Use a screening dispersion model (e.g., AERSCREEN) to obtain concentration estimates for each emitted pollutant at varying receptor distances from the source. Several screening models feature the generation of an automatic array of receptors which is particularly useful for determining the zone of impact. In order for the model to generate the array of receptors the user needs to provide some information normally consisting of starting distance, increment and number of intervals.

- Calculate total cancer risk and hazard index (HI) for each receptor location by using the methods provided in the risk characterization sections of the Air Toxics Hot Spots Risk Assessment Guidance Manual.

- Find the distance where the total inhalation cancer risk is equal to 10^{-6}; this may require redefining the receptor array in order to have two receptor locations that bound a total cancer risk of 10^{-6}. Secondly and thirdly, find the distance where the chronic and acute health hazard indices are declared significant by the District (e.g., acute or chronic HI = 1.0).

Some Districts may prefer to use a cancer risk of 10^{-7} as the zone of impact. Therefore, the District should be consulted before modeling efforts are initiated. If the zone of impact is greater than 25 km from the facility at any point, then the District should be consulted. The District may specify limits on the area of the zone of impact. Ideally, these preferences would be presented in the modeling protocol (see Section 2.14).

Note that when depicting the risk assessment results, risk isopleths must present the total cancer and noncancer risk from both inhalation and noninhalation pathways. The zone of impact should be clearly shown on a map with geographic markers of adequate resolution (see Section 2.6.3.1).

2.6.2 Population Estimates for Screening Risk Assessments

A screening risk assessment should include an estimate of the maximum exposed population. For screening risk assessments, a detailed description of the exposed population is not required. The impact area to be considered should be selected to be health protective (i.e., will not underestimate the number of exposed individuals). A health-protective assumption is to assume that all individuals within a large radius of the facility are exposed to the maximum concentration. If a facility must also comply with the RCRA/CERCLA risk assessment requirements, health effects to on-site workers may also need to be addressed. The DTSC's Remedial Project Manager should be consulted on this issue. The District should be consulted to determine the population estimate that should be used for screening purposes.

2.6.3 Population Estimates for Refined Risk Assessments

The refined risk assessment requires a detailed analysis of the population that is exposed to emissions from the facility. Where possible, a detailed population exposure analysis provides estimates of the number of individuals in residences and off-site

workplaces, as well as at sensitive receptor sites such as schools, daycare centers and hospitals. The District may require that locations with high densities of sensitive individuals be identified (e.g., schools, daycare centers, hospitals). The overall exposed residential and worker populations should be apportioned into smaller geographic subareas. The information needed for each subarea is:

(1) the number of exposed persons, and

(2) the receptor location where the calculated ambient air concentration is assumed to be representative of the exposure to the entire population in the subarea.

A multi-tiered approach is suggested for the population analysis. First, the census tracts impacted by the facility should be identified (see Section 2.6.3.1). A census tract may need to be divided into smaller subareas if it is close to the facility where ambient concentrations vary widely. The District may determine that census tracts provide sufficient resolution near the facility to adequately characterize population exposure. The HARP software will provide population estimates that are consistent with the methodology discussed in this document.

Further downwind where ambient concentrations are less variable, the census tract level may be acceptable to the District. The District may determine that the aggregation of census tracts (e.g., the census tracts making up a city are combined) is appropriate for receptors which are considerable distances from the facility. If a facility must also comply with the RCRA/CERCLA risk assessment requirements, health effects to on-site workers may also need to be addressed. The DTSC's Remedial Project Manager should be consulted on this issue. In addition, the district should be consulted about special cases where evaluation of on-site receptors is appropriate, such as facilities frequented by the public or where people may reside (e.g., military facilities).

2.6.3.1 Census Tracts

For a refined risk assessment, the boundaries of census tracts can be used to define the geographic area to be included in the population exposure analysis. Digital maps showing the census tract boundaries in California can be obtained from "The Thomas Guide"® on the World Wide Web. Statistics for each census tract can be obtained from the U.S. Census Bureau. The website address for the U.S. Census Bureau is http://www.census.gov. Numerous additional publicly accessible or commercially available sources of census data can be found on the World Wide Web. A specific example of a census tract is given in Appendix J. The HARP software includes U.S. census data and is a recommended tool for performing population exposure estimates.

The two basic steps in defining the area under analysis are:

(1) Identify the "zone of impact" (as defined previously in Section 2.6.1) on a map detailed enough to provide for resolution of the population to the subcensus tract level. (The U.S. Geological Survey (USGS) 7.5-minute series maps and the maps

within the HARP software provide sufficient detail.) This is necessary to clearly identify the zone of impact, location of the facility, and sensitive receptors within the zone of impact. If significant development has occurred since the USGS survey, this should be indicated. A specific example of a 7.5-minute series map is given in Appendix J.

(2) Identify all census tracts within the zone of impact using a U.S. Bureau of Census or equivalent map (e.g., Thomas Brothers, HARP Software). If only a portion of the census tract lies within the zone of impact, then only the population that falls within the isopleth should be used in the population estimate or burden calculation. To determine this level of detail, local planning and zoning information may need to be collected. When this more detailed information is not available, then a less refined approach is to include the census data if the centroid of the census block falls within the isopleths of interest. The census tract boundaries should be transferred to a map, such as a USGS map (referred to hereafter as the "base map".)

An alternative approach for estimating population exposure in heavily populated urban areas is to apportion census tracts to a Cartesian grid cell coordinate system. This method allows a Cartesian coordinate receptor concentration field to be merged with the population grid cells. This process can be computerized and minimizes manual mapping of centroids and census tracts. The HARP software includes this function and will provide population estimates that are consistent with the methodology discussed here.

The District may determine that aggregation of census tracts (e.g., which census tracts making up a city can be combined) is appropriate for receptors that are located at considerable distances from the facility. If the District permits such an approach, it is suggested that the census tract used to represent the aggregate be selected in a manner to ensure that the approach is health protective. For example, the census tract included in the aggregate that is nearest (downwind) to the facility should be used to represent the aggregate.

2.6.3.2 Subcensus Tract

Within each census tract are smaller population units. These units [urban block groups (BG) and rural enumeration districts (ED)] contain about 1,100 persons. BGs are further broken down into statistical units called blocks. Blocks are generally bounded by four streets and contain an average of 70 to 100 persons. However, the populations presented above are average figures and population units may vary significantly. In some cases, the EDs are very large and identical to a census tract.

The area requiring detailed (subcensus tract) resolution of the exposed residential and worker population will need to be determined on a case-by-case basis through consultation with the District. The District may determine that census tracts provide sufficient resolution near the facility to adequately characterize population exposure.

Employment population data can be obtained at the census tract level from the U.S. Census Bureau or from local planning agencies. This degree of resolution will generally not be sufficient for most risk assessments. For the area requiring detailed analysis, zoning maps, general plans, and other planning documents should be consulted to identify subareas with worker populations.

The boundaries of each residential and employment population area should be transferred to the base map.

2.6.4 Sensitive Receptor Locations

Individuals who may be more sensitive to toxic exposures than the general population are distributed throughout the total population. Sensitive populations may include young children and chronically ill individuals. The District may require that locations with high densities of sensitive individuals be identified (e.g., schools, daycare centers, hospitals). The risk assessment should state what the District requirements were regarding identification of sensitive receptor locations.

Although protection of sensitive individuals is incorporated into OEHHA's risk assessment methodology in both cancer risk and noncancer risk assessment, the assessment of risk at the specific location of such sensitive individuals (e.g., schools, hospitals, or nursing homes) may be useful to assure the public that such individuals are being considered in the analysis. For some chemicals (e.g., mercury and manganese) children have been specifically identified as the sensitive subpopulation for noncancer health impacts, so it can be particularly appropriate to assess school sites.

2.7 Receptor Siting

2.7.1 Receptor Points

The modeling analysis should contain a network of receptor points with sufficient detail (in number and density) to permit the estimation of the maximum concentrations. Locations that must be identified include the maximum estimated off-site risk or point of maximum impact (PMI), the maximum exposed individual at an existing residential receptor (MEIR) and the maximum exposed individual at an existing occupational receptor (worker) (MEIW). All of these locations (i.e., PMI, MEIR, and MEIW) must be identified for assessing cancer and noncancer risks. It is possible that the estimated PMI, MEIR, and MEIW risk for cancer, chronic noncancer, and acute noncarcinogenic risks occur at different locations. The results from a screening model (if available) can be used to identify the area(s) where the maximum concentrations are likely to occur. Receptor points should also be located at the population centroids (see Section 2.7.2) and sensitive receptor locations (see Section 2.6.4). The exact configuration of the receptor array used in an analysis will depend on the topography, population distribution patterns, and other site-specific factors. All receptor locations should be identified in the risk assessment using UTM (Universal Transverse Mercator) coordinates and receptor

number. The receptor numbers in the summary tables should match receptor numbers in the computer output. In addition to UTM coordinates, the street address(es), where possible and as required by the local district, should be provided for the PMI, MEIR and MEIW for carcinogenic and noncarcinogenic health impacts.

2.7.1.1 Receptor Height

To evaluate localized impacts, receptor height should be taken into account at the point of maximum impact on a case-by-case basis. For example, receptor heights may have to be included to account for receptors significantly above ground level. Flagpole receptors at the height of the breathing zone of a person may need to be considered when the source receptor distance is less than a few hundred meters. Consideration must also be given to the noninhalation pathway analysis which requires modeling of chemical deposition onto soil or water at ground level as a first step. A health protective approach is to select a receptor height from 0 meters to 1.8 meters that will result in the highest predicted downwind concentration. Final approval of this part of the modeling protocol should be with the District, or reviewing authority.

2.7.2 Centroid Locations

For each subarea analyzed, a centroid location (the location at which a calculated ambient concentration is assumed to represent the entire subarea) should be determined. When population is uniformly distributed within a population unit, a geographic centroid based on the shape of the population unit can be used. If only a portion of the census tract lies within the isopleth or area of interest, then only the population that falls within the isopleth should be used in the calculation for population exposure. To determine this level of detail, local planning and zoning information may need to be collected. Where populations are not uniformly distributed, a population-weighted centroid may be used. Another alternative uses the concentration at the point of maximum impact within that census tract as the concentration to which the entire population of that census tract is exposed. While this less refined approach is commonly accepted, Districts should be contacted to approve this method prior to its use in a risk assessment.

The centroids represent locations that should be included as receptor points in the dispersion modeling analysis. Annual average concentrations should be calculated at each centroid using the modeling procedures presented in this chapter.

For census tracts and BG/EDs, judgments can be made using U.S. census data, census tracts maps, and street maps to determine the centroid location. At the block level, a geographic centroid is sufficient.

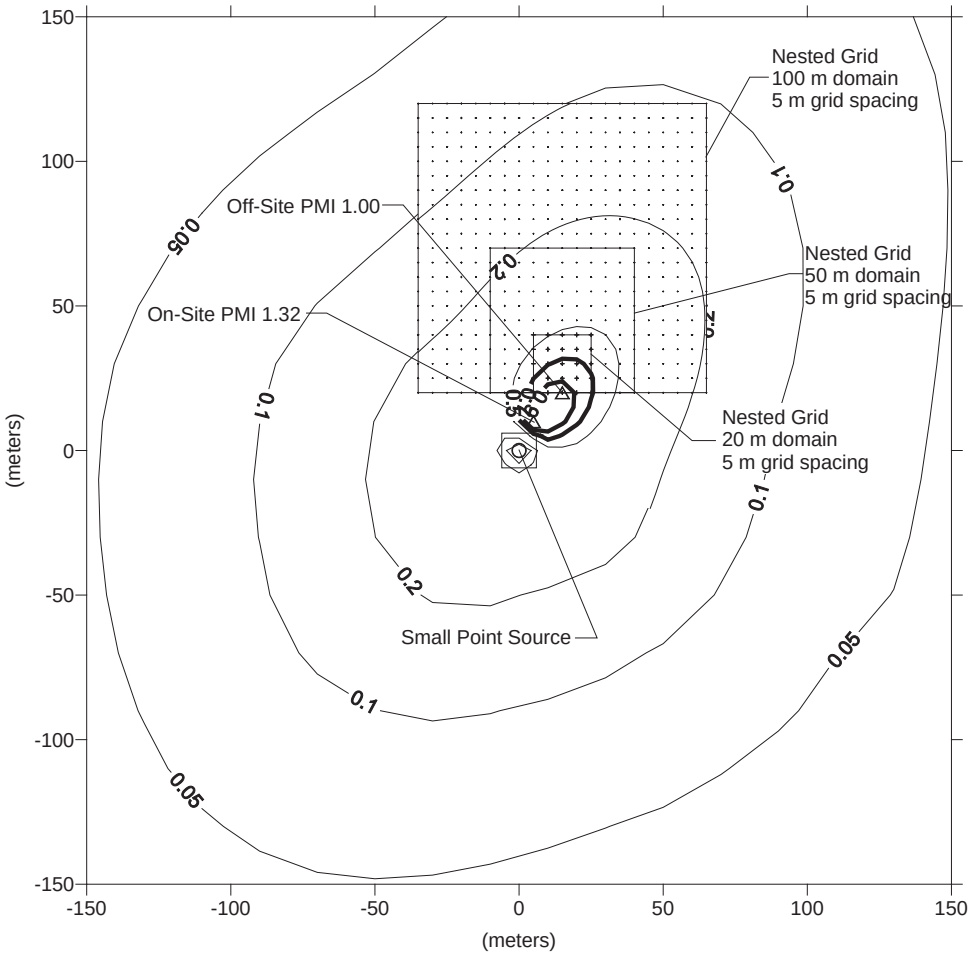
2.7.3 Spatial Averaging of Modeling Results

Since the inception of the “Hot Spots” and the air toxics programs in California, health risk assessment (HRA) results for an individual have typically been based on air

dispersion modeling results at a single point or location. With a few exceptions, this method has been traditionally used for all types of receptors (e.g., PMI, MEIR, MEIW, pathway receptors, etc.). The assumptions used in risk assessment are designed to prevent underestimation of health impacts to the public – a health protective approach.

To identify the individual receptor (e.g., PMI, MEIR, etc), air dispersion modeling of pollutant emissions estimate ground level concentrations (GLC) at downwind receptors, which are distributed in a grid pattern of sufficient size and density to capture the maximum concentration. Figure 2 shows an example of the PMI and concentration isopleths. Under some conditions, the PMI may be significantly higher than receptors only a few meters away. In these cases, it may be unrealistic for the PMI to represent the 70-year exposure for long-term risk calculations.

FIGURE 2 – FIGURE 2CONCENTRATION ISOPLETHS



It is prudent public health practice to err on the side of public health protection in face of uncertainty; however, when exposure models can be refined, better scientific estimates of exposure and risk can be obtained. Basing risk estimates on a single highest point (PMI, MEIR, or MEIW) does not take into account that a person does not remain at one

location on their property, or often in one location at the workplace over an extended period of time. Thus, using a single point with the highest air concentration that is not representative of the average concentration at a residence will tend to overestimate exposure and risk. One to five years of meteorological data do not necessarily fully characterize the variability in meteorological conditions over longer periods (e.g., 30 to 70 years) and thus the concentrations at a single point are likely to be more diffuse than the modeling estimates based on one year of meteorological data. U.S.EPA modeling guidance suggests that five years of consecutive meteorological data strongly represent a longer average such as 70 years. The average air concentration over a small area is likely to be more representative than the determination the air concentration at a single point, particularly in those situations where the concentrations falls off rapidly around the single point.

In order to understand how spatial averaging would impact air dispersion modeling results with various types of facilities, the ARB, in conjunction with the OEHHA, performed sensitivity analyses to evaluate the impacts of spatially averaging air dispersion modeling results. That information is presented in detail in Appendix C. Based on these sensitivity analyses, we feel it is reasonable and appropriate to include spatial averaging techniques in air toxic risk assessments as supplemental information to Tier 1 information (i.e., modeling results that are based on the air concentration from a single point or location). While all risk assessments must include results based on Tier 1 methodology, the spatially-averaged concentrations around the point of interest (e.g., PMI, MEIR, MEIW, multipathway exposure evaluations, etc.) could also be included as an option in risk assessments and for risk management decisions subject to approval by the District or reviewing agency.

A few reasons that support the inclusion of spatially-averaged modeled concentrations in risk assessment include the following.

- Averaging results over a small domain will give a more representative picture of individual exposure and risk than an estimate based on one single location within their property.

- Spatial averaging will allow air dispersion modeling and risk assessment results to be characterized as the estimated concentration and risk in a discrete area of interest, rather than an exact value for a single location.

- From a risk communication standpoint, the ARB and OEHHA feel it is more appropriate to present the modeling output and the calculated health impacts as the potential impacts within a small or discrete area, rather than an exact value at a specific point on a grid or map.

- Spatial averaging is the recommended procedure in ARB's Lead Risk Management Guidelines (2001) and has been used in several complex source HRAs [e.g., Roseville Railyard (2004), Ports of LA/LB (2006), Port of Oakland (2008)].

- Spatially averaging the deposition concentrations over pasture land or a water body for multipathway exposure scenarios is a planned upgrade for the HARP Software. This will provide an option that will appropriately refine multipathway exposure assessments. Average deposition on a water body is not necessarily well represented by the single highest point of deposition, or deposition at the geographic center of the water body. Likewise, since produce is grown over the entire surface of the garden and cows graze the entire pasture, deposition is better estimated by evaluating the entire area rather than using a single point.

2.7.4 Spatial Averaging Method

The spatial averaging sensitivity study in Appendix C is based on simulating emissions from a point, volume, area, and line sources. Each source type (e.g., point) is simulated as a small, medium or large source. Line sources are only simulated as small and large. In addition, meteorological data collected at five different locations in California were used. Nested spatial average grids of various domains were used to study the differences on the spatial average concentration. In the case of the 20 meter by 20 meter spatial average nested grid, the spatial average concentration showed little change over the PMI for medium and large sources. In the case for small sources, the spatial average concentration is 45% to 80% of the PMI concentration. Individual source type and meteorological conditions will cause variations in these results.

The results of the spatial averaging sensitivity study in Appendix C shows that sources with low plume rise that result in a PMI, MEIW, or MEIR located at or near the property fence line are most sensitive to spatial averaging. Source types with high plume rise (e.g., tall stacks) show a PMI far downwind where the concentration gradient is more gradual and therefore spatial averaging has a lesser effect. While spatial averaging can be used regardless of source size or the location of the PMI, the following conditions generally apply when a source is a good candidate for spatial averaging

- The MEIR, MEIW, or PMI is located at the fence line or close to the emission source.

- The concentration gradient is high near the PMI. This is more associated with low level plumes such as fugitive, volume, area, or short stacks.

- A long term average is being calculated to represent a multi-year risk analysis based on one to five years of meteorological data. Note that spatial averaging should **not** be used for short term (acute) calculations.

2.7.4.1 Residential Receptors

To remain health protective when evaluating a residential receptor, spatial averaging should not take place using large nested domains. The domain used for spatial averaging should be no larger than 20 meters by 20 meters with a maximum grid

spacing resolution of five meters. This domain represents and area that is approximately the size of a small urban lot.

In general, the method for calculating the spatial average in air toxic risk assessments includes the following steps.

1. Locate the off-site PMI, MEIW, or MEIR with a grid resolution spacing of no greater than five meters. Two or more model runs with successively finer nested grid resolutions centered on the new PMI may be required to locate the final PMI.

2. Center the spatial average nested grid on the off-site receptors about the PMI, MEIW, or MEIR. Limit the nested grid to no larger than 20 meters by 20 meters. The grid resolution spacing should be no greater than five meters. With a five meter grid resolution, the 20 meter by 20 meter nest will result in 25 receptors.

3. Some configurations of source activity and meteorological conditions result in a predominant downwind plume center line that is significantly askew from one of the four ordinate directions. In this case, a tilted nested grid is necessary to coincide with the dominant plume centerline. Polar receptors are easier to implement than a tilted rectangular grid. The domain of the polar receptor field should be limited to a 15 meter radius. See Appendix C for detailed instructions on tilted polar receptors.

4. Calculate the arithmetic mean of the long term period average concentration (e.g., annual average) of the nested grid of receptors to represent the spatial average.

Appendix C shows explicit details for selecting, placing, and tilting a nested grid for rectangular or polar receptor grids. In addition, the sensitivity study is also available.

2.7.4.2 Worker Receptors

Offsite worker locations (e.g. MEIW) may also be a candidate for spatial averaging. However, workers can be at the same location during almost their entire work shift (e.g., desk/office workers). When this is the situation, then a single location and corresponding modeled concentration are appropriate to use. If spatial averaging is used, care should be taken to determine the proper domain size and grid resolution that should be used. To be consistent with the residential receptor assumptions and remain health protective, a maximum domain size should be no larger than 20 meters by 20 meters with a maximum grid spacing resolution of five meters. However, if workers routinely and continuously move throughout the worksite over a space greater than 20 meters by 20 meters, then a larger domain may be considered. The HRA or modeling protocol shall support all assumptions used, including, but not limited to, documentation for all workers showing the area where each worker routinely performs their duties. The

final domain size should not be greater than the smallest area of worker movement. Other considerations for determining domain size and grid spacing resolution may include an evaluation of the concentration gradients across the worker area. The grid spacing used within the domain should be sufficient in number and detail to obtain a representative concentration across the area of interest. The size of the domain and resolution of points shall be subject to approval by the District, ARB, or other reviewing authority.

2.7.4.3 Pastures or Water Bodies

The simplified approach of using the deposition rate at the centroid, a specific point of interest, or the PM location for an area being evaluated for noninhalation exposures(e.g. a body of water used for fishing, a pasture used for grazing, etc) is still acceptable for use in HRA. However, evaluating deposition concentrations over pasture land or a water body for multipathway exposure scenarios using spatial averaging could give more representative estimates of the overall deposition rate. Use of spatial averaging in this application is subject to approval by the District, ARB, or other reviewing authority.

When using spatial averaging over the deposition area, care should be taken to determine the proper domain size to make sure it includes all reasonable areas of potential deposition. The size and shape of the pasture or water body of interest should be identified and used for the modeling domain. The grid spacing or resolution used within the domain should be sufficient in detail to obtain a representative deposition concentration across the area of interest. One way to determine the grid resolution is to include an evaluation of the concentration gradients across the deposition area. The HRA or modeling protocol shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the pasture or water body, maps, representative coordinates, grid resolution, concentration gradients, etc.). The size of the domain and grid resolution are subject to approval by the reviewing authority.

In lieu of the details required in the above description, the approach used for the other receptors (e.g., MEIR, MEIW) that uses a domain size not greater than 20 meters by 20 meters, centered on the PMI or point of interest, with a maximum grid spacing resolution of five meters can be used. This default refined approach would apply to deposition areas greater than 20 meters by 20 meters. For smaller deposition areas, the simplified approach of using the PMI or the actual smaller domain can be used.

The HRA or modeling protocol shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the lake or water body, maps, representative coordinates, etc.). Other considerations for determining domain size and grid spacing resolution should include an evaluation of the concentration gradients across the deposition area. The grid spacing used within the domain should be sufficient in number and detail to obtain a representative deposition concentration across the area of interest. This information should also be included in the HRA and modeling protocols

2.8 Meteorological Data

Refined air dispersion models require hourly meteorological data. The first step in obtaining meteorological data should be to check with the District for data availability. Other sources of data include the National Weather Service (NWS), National Climatic Data Center (NCDC), Asheville, North Carolina, military stations and private networks. Meteorological data for a subset of NWS stations are available from the U.S. EPA Support Center for Regulatory Air Models (SCRAM). The SCRAM can be accessed at www.epa.gov/scram001/main.htm. All meteorological data sources should be approved by the District. Data not obtained directly from the District should be checked for quality, representativeness and completeness. U.S. EPA provides guidance (U.S. EPA, 1995e) for these data. The risk assessment should indicate if the District required the use of a specified meteorological data set. All memos indicating District approval of meteorological data should be attached in an appendix. If no representative meteorological data are available, screening procedures should be used.

The analyst should acquire enough meteorological data to ensure that the worst-case meteorological conditions are represented in the model results. The US-EPA Guideline on Air Quality Models (U.S. EPA 2005) prefers that the latest five years of consecutive meteorological data be used to represent long term averages (i.e., cancer and chronic). Previous OEHHA guidance allowed the use of the worst-case year to save computer time. The processing speed of modern computers has increased to the point where processing five years of data over one year is no longer burdensome. However, the District may determine that one year of representative meteorological data is sufficient to adequately characterize the facility's impact. This may especially be the case when five years of quality consecutive data are not available.

During the transitional period from night to day (i.e., the first one to three hours of daylight) the meteorological processor may interpolate some very low mixing heights. This is a period of time in which the mixing height may be growing rapidly. When predicted concentrations are high and the mixing height is very low for the corresponding averaging period, the modeling results deserve additional consideration. For receptors in the near field, it is within the model formulation to accept a very low mixing height for short durations. However, it would be unlikely that the very low mixing height would persist long enough for the pollutants to travel into the far field. In the event that the analyst identifies any of these time periods, they should be discussed with the District on a case-by-case basis.

2.8.1 Modeling to Obtain Concentrations used for Various Health Impacts

The following section outlines how air dispersion modeling results are used or adjusted for a receptor that is exposed to either a non-continuous or continuously emitting source.

2.8.1.1 Modeling and Adjustments for Inhalation Cancer Risk at a Worksite

Modeled long-term averages are typically used for cancer risk assessments. In an inhalation cancer risk assessment for an offsite worker, the long-term average should represent what the worker breathes during their work shift. However, the long-term averages calculated from AERMOD typically represent exposures for receptors that were present 24 hours a day and seven days per week (i.e., residential receptors). To estimate the offsite worker's concentration, there are two approaches. The more refined, complex, and time consuming approach is to post-process the hourly raw dispersion model output and examine the hourly concentrations that fall within the offsite worker's shift. See Appendix M for information on how to simulate the long-term concentration for the offsite worker that can be used to estimate inhalation cancer risk.

In lieu of post-processing the hourly dispersion model output, the more typical approach is to obtain the long-term average concentration as you would for modeling a residential receptor and approximate the worker's inhalation exposure using an adjustment factor. The actual adjustment factor that is used to adjust the concentration may differ from the example below based on the specifics of the source and worker receptor (e.g., work-shift overlap). Once the worker's inhalation concentration is determined, the inhalation dose is calculated using additional exposure frequency and duration adjustments. See Chapter 3 for more information on the inhalation dose equation.

2.8.1.1.1 Non-Continuous Sources

When modeling a non-continuously emitting source (e.g., operating for eight hours per day and five days per week), the modeled long-term average concentrations are based on 24 hours a day and seven days per week for the period of the meteorological data set. Even though the emitting source is modeled using a non-continuous emissions schedule, the long-term concentration is still based on 24 hours a day and seven days per week. Thus, this concentration includes the zero hours when the source was not operating. For the offsite worker inhalation risk, we want to determine the long-term concentration the worker is breathing during their work shift. Therefore, the long-term concentration needs to be adjusted so it is based only on the hours when the worker is present. For example, assuming the emitting source and worker's schedules are the same, the adjustment factor is 4.2 = (24 hours per day/8 hours per shift)x(7 days in a week/5 days in a work week). In this example, the long term residential exposure is adjusted upward to represent the exposure to a worker. Additional concentration adjustments may be appropriate depending on the work shift overlap. These adjustments are discussed below.

The calculation of the adjustment factor from a non-continuous emitting source is summarized in the following steps.

a. Obtain the long-term concentrations from air dispersion modeling as is typical for residential receptors (all hours of a year for the entire period of the meteorological data set).

b. Determine the coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule.

c. Calculate the worker adjustment factor (WAF) using Equation 2.1. When assessing inhalation cancer health impacts, a discount factor (*DF*) may also be applied if the offsite worker's schedule partially overlaps with the source's emission schedule. The discount factor is based on the number of coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule (see Equation 2.2). The DF is always less than or equal to one.

Please note that worker adjustment factor does not apply if the source's emission schedule and the offsite worker's schedule do not overlap. Since the worker is not around during the time that the source is emitting, the worker is not exposed to the source's emission (i.e., the DF in Equation 2.2 becomes 0).

$$WAF = \frac{H_{residential}}{H_{source}} \times \frac{D_{residential}}{D_{source}} \times DF$$

Eq. 2.1

Where:

WAF = the worker adjustment factor

$H_{residential}$= the number of hours per day the long-term residential concentration is based on

(always 24 hours)

H_{source} = the number of hours the source operates per day

$D_{residential}$ = the number of days per week the long-term residential concentration is based on

(always 7 days).

D_{source}= the number of days the source operates per week.

DF = a discount factor for when the offsite worker's schedule partially overlaps the source's emission schedule. Use 1 if the offsite worker's schedule occurs within the source's emission schedule. If the offsite worker's schedule partially overlaps with the source's emission schedule, then calculate the discount factor using Equation 2.2 below.

$$DF = \frac{H_{coincident}}{H_{worker}} \times \frac{D_{coincident}}{D_{worker}}$$

Eq. 2.2

Where:

DF = the discount factor for assessing cancer impacts

$H_{coincident}$ = the number of hours per day the offsite worker's schedule and the source's emission schedule overlap

$D_{coincident}$= the number of days per week the offsite worker's schedule and the source's emission schedule overlap.

H_{worker} = the number of hours the offsite worker works per day

D_{worker}= the number of days the offsite worker works per week.

d. The final step is to estimate the offsite worker's inhalation concentration by multiplying the worker adjustment factor with the long-term residential concentration. The worker's concentration is then plugged into the dose equation and risk calculation.

The HARP software has the ability to calculate worker impacts using an approximation factor and, in the future, it will have the ability to post-process refined worker concentrations using the hourly raw results from an air dispersion analysis.

2.8.1.1.2 Continuous Sources

If the source is continuously emitting, then the worker is assumed to breathe the long-term annual average concentration during their work shift. Equation 2.1 becomes one and no concentration adjustments are necessary in this situation when estimating the inhalation cancer risk. Note however, if an assessor does not wish to apply the assumption the worker breathes the long-term annual average concentration during the work shift, then a refined concentration can be post-processed as described in Appendix M. All alternative assumptions should be approved by the reviewing authority and supported in the presentation of results.

2.8.1.2 Modeling and Adjustments for 8-Hour RELs

For 8-hour noncancer health impacts, we evaluate if the receptor (e.g., worker or resident) is exposed to a daily (e.g., 8-hour) average concentration that exceeds the 8-hour REL. For ease, we use a worker receptor in this discussion and in the discussion below for a non-continuously emitting source. The daily average concentration is intended to represent the long-term average concentration the worker is breathing during their work shift. In general, there are two approaches for estimating the concentration used for the 8-hour hazard index. The more refined, complex, and

time consuming approach is to post-process the hourly dispersion model output and use only the hourly concentrations that are coincident with the offsite worker hours to obtain the long-term concentration. See Appendix M for information on how to simulate the daily average concentration through air dispersion modeling. Before proceeding through a refined analysis described in Appendix M, the assessor may wish to approximate the long-term concentration, as described below, and calculate the 8-hour hazard index. Based on those results, the assessor can contact OEHHA for assistance in determining whether further evaluation may be necessary. The results from the 8-hour hazard index calculations are not combined with the chronic or acute hazard indices. All potential noncancer health impacts should be reported independently.

In lieu of post-processing the hourly dispersion model output described in Appendix M, the more typical approach is to obtain the long-term average concentration as you would for modeling a residential receptor and approximate the worker's inhalation concentration using an adjustment factor. The method for applying the adjustment factor is described below.

2.8.1.2.1 Non-Continuous Sources

When modeling a non-continuously emitting source (e.g., operating for eight hours per day and five days per week), the modeled long-term average concentrations are based on 24 hours a day and seven days per week for the period of the meteorological data set. Even though the emitting source is modeled using a non-continuous emissions schedule, the long-term concentration is still based on 24 hours a day and seven days per week. Thus, this concentration includes the zero hours when the source was not operating. For the offsite worker 8-hour hazard index, we want to determine the long-term average daily concentration the worker may be breathing during their work shift. This is similar to the cancer approximation adjustment method with one difference; there is no adjustment for partial overlap between the worker's schedule and the source's emission schedule. The reason for this difference in methodology is because the 8-hour REL health factors are designed for repeated 8-hour exposures and cannot readily be adjusted to other durations of exposure.

When calculating the long-term average daily concentration for the 8-hour REL comparison, the long-term residential concentration needs to be adjusted so it is based only on the operating hours of the emitting source with the assumption the offsite worker's shift falls within the emitting source's schedule. For example, assuming the emitting source operates 8 hours per day, 5 days per week and the offsite worker's schedules fall within this period of emissions, then the adjustment factor is 4.2 = (24 hours per day/8 hours of emissions per day)x(7 days in a week/5 days of emissions per week). In this example, the long term residential exposure is adjusted upward to represent the 8-hour exposure to a worker. No adjustments are applied for partial work shift overlap with the emitting source. If the source emits at night, then see Appendix N for additional recommendations.

Using the approximation factor is a screening method. If the 8-hour hazard index is above a threshold of concern with this method, the district or assessor should contact OEHHA for further guidance regarding the substance of concern. If necessary, further evaluation can be performed using the refined daily average modeling methodology discussed in Appendix M.

The calculation of the adjustment factor from a non-continuous emitting source is summarized in the following steps.

a. Obtain the long-term concentrations from air dispersion modeling as is typical for residential receptors (all hours of a year for the entire period of the meteorological data set).

b. Calculate the worker adjustment factor (WAF) using Equation 2.3. The source's emission schedule is assumed to overlap offsite worker's schedule. Note that the worker adjustment factor and the 8-hour REL do not apply if the source's emission schedule and the offsite worker's schedule do not overlap at some point.

$$WAF = \frac{H_{residential}}{H_{source}} \times \frac{D_{residential}}{D_{source}}$$

Eq. 2.3

Where:

WAF = the worker adjustment factor
$H_{residential}$= the number of hours per day the long-term residential concentration is based on (always 24 hours)
H_{source} = the number of hours the source operates per day
$D_{residential}$ = the number of days per week the long-term residential concentration is based on (always 7 days).
D_{source}= the number of days the source operates per week.

c. The final step is to estimate the offsite worker's daily average inhalation concentration by multiplying the WAF with the long-term residential concentration. The worker's concentration is then used to calculate the 8-hour hazard index. This method using the approximation factor is a screening method. If the 8-hour hazard index is above a threshold of concern, the district or assessor should contact OEHHA for further guidance regarding the substance of concern.

In the future, the HARP software will have the ability to use 8-hour RELs, calculate worker impacts using an approximation factor, and to post-process worker concentrations using the hourly raw results from an air dispersion analysis.

2.8.1.2.2 Continuous Sources

If the source is continuously emitting, then the worker is assumed to breathe the long-term annual average concentration during their work shift and no concentration adjustments are made when estimating 8-hour health impacts. Note however, if an assessor does not wish to assume the worker breathes the long-term annual average concentration during the work shift, then a refined concentration can be post-processed as described in Appendix M. All alternative assumptions should be approved by the reviewing authority and supported in the presentation of results.

Eight-hour RELs are not used for residential receptors that are exposed to continuously emitting sources. In this situation, chronic RELs are used.

2.8.1.3 Modeling and Adjustment Factors for Chronic RELs

Potential chronic noncancer health impacts use the long-term annual average concentration regardless of the emitting facility's schedule. No adjustment factors should be used to adjust this concentration. Chronic RELs are used to assess both residential or worker health impacts. The results from the chronic hazard index calculations are not combined with the 8-hour or acute hazard indices. All potential noncancer results should be reported independently.

2.8.1.4 Modeling and Adjustments for Oral Cancer Potencies and Oral RELs

When estimating the cancer risk or noncancer health impacts from noninhalation pathways, no adjustment is made to the long-term annual average concentration regardless of the emitting facility's schedule. Since the media (e.g., soil) at the receptor location where deposition takes place for noninhalation pathways is continuously present, the concentrations used for all noninhalation pathways are not adjusted (up or down) by an adjustment factor. However, some adjustments are made to the concentration once the pollutants reach the media, for example, pollutants undergo decay in soils. In addition, when the dose for each pathway is calculated, exposure adjustments may also be made. See the individual chapters for each exposure pathway to get more information on these types of adjustments. Oral cancer potencies and oral RELs are used to assess both residential or worker health impacts.

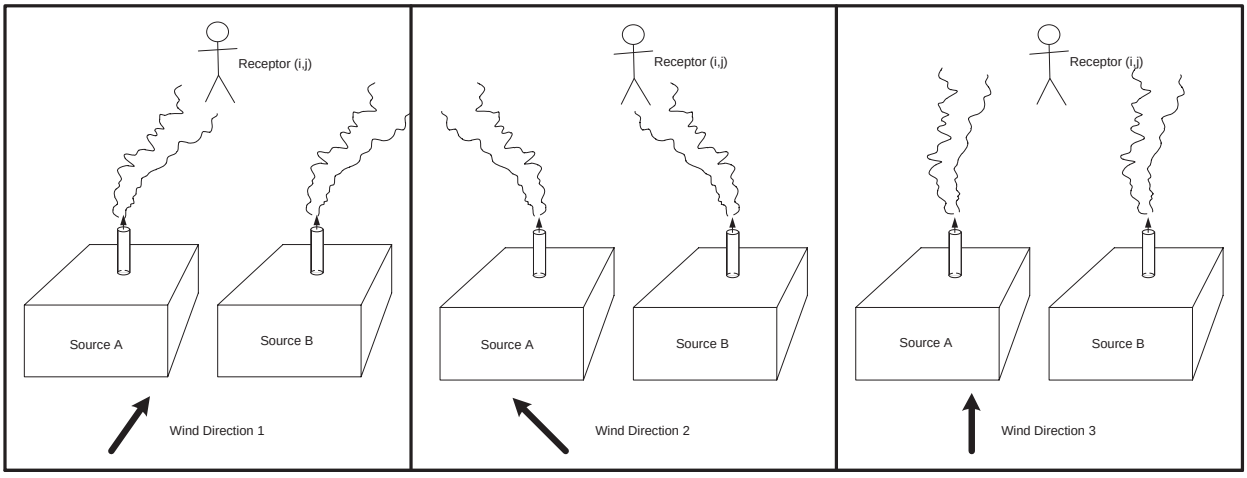
2.8.2 Modeling One-Hour Concentrations using Simple and Refined Acute Calculations

Modeled one-hour concentrations are needed for the acute health hazard index calculations. HARP has two methods to calculate this concentration; Simple and Refined. As an aid to understanding the differences between Simple and Refined, Figure 3 shows three possible conditions showing how wind direction may vary and impact a downwind receptor (i,j) differently from just two sources (A and B).

For the Simple calculation, HARP stores only the maximum one-hour concentration at each receptor (i,j) from each source (A and B) as the dispersion model marches down each hour of the simulation (e.g., one to five years of hourly data). At the end of the simulation period, HARP reports back only the maximum impacts at each receptor from each source regardless of which hour of the simulation period this occurred. For example, the Simple Maximum Acute Impacts would be the summation of Source A impacts from Wind Direction 1 and Source B impacts from Wind Direction 2 as shown in Figure 3.

For the Refined simulation, HARP stores each hourly concentration at each receptor (i,j) from each source. At the end of the simulation period, HARP evaluates the coincident impact at each receptor from all sources for each hour of the simulation period. In this case the maximum impacts will be identified by a particular hour of the period with associated wind speed, direction, and atmospheric conditions. For example, the Refined Maximum Acute impact from Sources A and B on receptor (i,j) could be from any wind direction (1,2, or 3) as shown in Figure 3. As HARP stores all simulations for all sources – at all receptors – for all hours to calculate the refined impacts, there is great potential to fill large amounts of disk storage space. However the Refined simulation provides a more representative picture of the Maximum acute hazard index from a facility. The Simple calculation will provide an upper bound to the acute hazard index.

FIGURE 3 – ACUTE SCENARIOS



The following sections, taken mostly from the document “On-Site Meteorological Program Guidance for Regulatory Modeling Applications” (U.S. EPA, 1995e), provide general information on data formats and representativeness. Some Districts may have slightly different recommendations from those given here.

2.8.3 Meteorological Data Formats

Most short-term dispersion models require input of hourly meteorological data in a format which depends on the model. U.S. EPA provides software for processing meteorological data for use in U.S. EPA recommended dispersion models. U.S. EPA recommended meteorological processors include the Meteorological Processor for Regulatory Models (MPRM), PCRAMMET, and AERMET. Use of these processors will ensure that the meteorological data used in an U.S. EPA recommended dispersion model will be processed in a manner consistent with the requirements of the model.

Meteorological data for a subset of NWS stations are available on the World Wide Web at the U.S. EPA SCRAM address, http://www.epa.gov/scram001.

2.8.4 Treatment of Calms

Calms are hours when the wind speed is below the starting threshold of the anemometer. Gaussian plume models require a wind speed and direction to estimate plume dispersion in the downwind direction.

U.S. EPA's policy is to disregard calms until such time as an appropriate analytical approach is available. The recommended U.S. EPA models contain a routine that eliminates the effect of the calms by nullifying concentrations during calm hours and recalculating short-term and annual average concentrations. Certain models lacking this built-in feature can have their output processed by U.S. EPA's CALMPRO program (U.S. EPA, 1984a) to achieve the same effect. Because the adjustments to the concentrations for calms are made by either the models or the postprocessor, actual measured on-site wind speeds should always be input to the preprocessor. These actual wind speeds should then be adjusted as appropriate under the current U.S. EPA guidance by the preprocessor.

Following the U.S. EPA methodology, measured on-site wind speeds of less than l.0 m/s, but above the instrument threshold, should be set equal to l.0 m/s by the preprocessor when used as input to Gaussian models. Calms are identified in the preprocessed data file by a wind speed of 1.0 m/s and a wind direction equal to the previous hour. For input to AERMOD, no adjustment should be made to the site specific wind data. AERMOD can produce model estimates for conditions when the wind speed may be less than 1 m/s but still greater than the instrument threshold. Some air districts provide pre-processed meteorological data for use in their district that treats calms differently. Local air districts should be consulted for available meteorological data.

If the fraction of calm hours is excessive, then an alternative approach may need to be considered to characterize dispersion. The Calpuff model modeling system can simulate calm winds as well as complex wind flow and therefore is a viable alternative. The local air district should be consulted for alternative approaches.

2.8.5 Treatment of Missing Data

Missing data refer to those hours for which no meteorological data are available from the primary on-site source for the variable in question. When missing values arise, they should be handled in one of the following ways listed below, in the following order of preference:

(1) If there are other on-site data, such as measurements at another height, they may be used when the primary data are missing. If the height differences are significant, corrections based on established vertical profiles should be made. Site-specific vertical profiles based on historical on-site data may also be appropriate to use if their determination is approved by the reviewing authority. If there is question as to the representativeness of the other on-site data, they should not be used.

(2) If there are only one or two missing hours, then linear interpolation of missing data may be acceptable, however, caution should be used when the missing hour(s) occur(s) during day/night transition periods.

(3) If representative off-site data exist, they may be used. In many cases this approach may be acceptable for cloud cover, ceiling height, mixing height, and temperature. This approach will rarely be acceptable for wind speed and direction. The representativeness of off-site data should be discussed and agreed upon in advance with the reviewing authority.

(4) Failing any of the above, the data field should be coded as missing using missing data codes appropriate to the applicable meteorological pre-processor.

Appropriate model options for treating missing data, if available in the model, should be employed. Substitutions for missing data should only be made in order to complete the data set for modeling applications, and should not be used to attain the “regulatory completeness” requirement of 90%. That is, the meteorological data base must be 90% complete on a monthly basis (before substitution) in order to be acceptable for use in air dispersion modeling.

2.8.6 Representativeness of Meteorological Data

The atmospheric dispersion characteristics at an emission source need to be evaluated to determine if the collected meteorological data can be used to adequately represent atmospheric dispersion for the project.

Such determinations are required when the available meteorological data are acquired at a location other than that of the proposed source. In some instances, even though meteorological data are acquired at the location of the pollutant source, they still may not correctly characterize the important atmospheric dispersion conditions.

Considerations of representativeness are always made in atmospheric dispersion modeling whether the data base is "on-site" or "off-site." These considerations call for the judgment of a meteorologist or an equivalent professional with expertise in atmospheric dispersion modeling. If in doubt, the District should be consulted.

2.8.6.1 Spatial Dependence

The location where the meteorological data are acquired should be compared to the source location for similarity of terrain features. For example, in complex terrain, the following considerations should be addressed in consultation with the District:

- Aspect ratio of terrain, i.e., ratio of:

  Height of valley walls to width of valley;

  Height of ridge to length of ridge; and

  Height of isolated hill to width of hill at base.

- Slope of terrain
- Ratio of terrain height to stack/plume height.
- Distance of source from terrain (i.e., how close to valley wall, ridge, isolated hill)
- Correlation of terrain feature to prevailing meteorological conditions

Likewise, if the source is located on a plateau or plain, the source of meteorological data used should be from a similar plateau or plain.

Judgments of representativeness should be made only when sites are climatologically similar. Sites in nearby, but different air sheds, often exhibit different weather patterns. For instance, meteorological data acquired along a shoreline are not normally representative of inland sites and vice versa.

Meteorological data collected need to be examined to determine if drainage, transition, and synoptic flow patterns are characteristics of the source, especially those critical to the regulatory application. Consideration of orientation, temperature, and ground cover should be included in the review.

An important aspect of space dependence is height above the ground. Where practical, meteorological data should be acquired at the release height, as well as above or below, depending on the buoyancy of the source's emissions. AERMOD at a minimum requires wind observations at a height above ground between seven times the local surface roughness height and 100 meters.

2.8.6.2 Temporal Dependence

To be representative, meteorological data must be of sufficient duration to define the range of sequential atmospheric conditions anticipated at a site. As a minimum, one full year of on-site meteorological data is necessary to prescribe this time series. Multiple years of data are used to describe variations in annual and short-term impacts. Consecutive years from the most recent, readily available 5-year period are preferred to represent these yearly variations.

2.8.6.3 Further Considerations

It may be necessary to recognize the non-homogeneity of meteorological variables in the air mass in which pollutants disperse. This non-homogeneity may be essential in correctly describing the dispersion phenomena. Therefore, measurements of meteorological variables at multiple locations and heights may be required to correctly represent these meteorological fields. Such measurements are generally required in complex terrain or near large land-water body interfaces.

It is important to recognize that, although certain meteorological variables may be considered unrepresentative of another site (for instance, wind direction or wind speed), other variables may be representative (such as temperature, dew point, cloud cover). Exclusion of one variable does not necessarily exclude all. For instance, one can argue that weather observations made at different locations are likely to be similar if the observers at each location are within sight of one another - a stronger argument can be made for some types of observations (e.g., cloud cover) than others. Although by no means a sufficient condition, the fact that two observers can “see” one another supports a conclusion that they would observe similar weather conditions.

Other factors affecting representativeness include change in surface roughness, topography and atmospheric stability. Currently there are no established analytical or statistical techniques to determine representativeness of meteorological data. The establishment and maintenance of an on-site data collection program generally fulfills the requirement for “representative” data. If in doubt, the District should be consulted.

2.8.7 Alternative Meteorological Data Sources

It is necessary, in the consideration of most air pollution problems, to obtain data on site-specific atmospheric dispersion. Frequently, an on-site measurement program must be initiated. As discussed in Section 2.8.5, representative off-site data may be used to substitute for missing periods of on-site data. There are also situations where current or past meteorological records from a National Weather Service station may suffice. These considerations call for the judgment of a meteorologist or an equivalent professional with expertise in atmospheric dispersion modeling. More information on Weather Stations including: National Weather Service (NWS), military observations, supplementary airways reporting stations, upper air and private networks, is provided in

"On-Site Meteorological Program Guidance for Regulatory Modeling Applications" (U.S. EPA, 1995e).

2.8.7.1 Recommendations

On-site meteorological data should be processed to provide input data in a format consistent with the particular models being used. The input format for U.S. EPA short-term regulatory models is defined in U.S. EPA's MPRM. The input format for AERMOD is defined in the AERMET meteorological pre-processor. Processors are available on the SCRAM web site. The actual wind speeds should be coded on the original input data set. Wind speeds less than 1.0 m/s but above the instrument threshold should be set equal to 1.0 m/s by the preprocessor when used as input to Gaussian models. Wind speeds below the instrument threshold of the cup or vane, whichever is greater, should be considered calm, and are identified in the preprocessed data file by a wind speed of 1.0 m/s and a wind direction equal to the previous hour. For input to AERMOD, no adjustment should be made to the site specific wind data. AERMOD can produce model estimates for conditions when the wind speed may be less than 1 m/s but still greater than the instrument threshold.

If data are missing from the primary source, they should be handled as follows, in order of preference: (l) substitution of other representative on-site data; (2) linear interpolation of one or two missing hours; (3) substitution of representative off-site data; or (4) coding as a missing data field, according to the discussions in Section 2.8.5.

If the data processing recommendations in this section cannot be achieved, then alternative approaches should be developed in conjunction with the District.

2.8.8 Quality Assurance and Control

The purpose of quality assurance and maintenance is the generation of a representative amount (90% of hourly values for a year on a monthly basis) of valid data. For more information on data validation consult reference U.S. EPA (1995e). Maintenance may be considered the physical activity necessary to keep the measurement system operating as it should. Quality assurance is the management effort to achieve the goal of valid data through plans of action and documentation of compliance with the plans.

Quality assurance (QA) will be most effective when following a QA Plan which has been signed-off by appropriate project or organizational authority. The QA Plan should contain the following information (paraphrased and particularized to meteorology from Lockhart):

1. Project description - how meteorology data are to be used
2. Project organization - how data validity is supported
3. QA objective - how QA will document validity claims
4. Calibration method and frequency - for data
5. Data flow - from samples to archived valid values

6. Validation and reporting methods - for data
7. Audits - performance and system
8. Preventive maintenance
9. Procedures to implement QA objectives - details
10. Management support - corrective action and reports

It is important for the person providing the quality assurance (QA) function to be independent of the organization responsible for the collection of the data and the maintenance of the measurement systems. Ideally, the QA auditor works for a separate company.

2.9 Model Selection

There are several air dispersion models that can be used to estimate pollutant concentrations and new ones are likely to be developed. U.S. EPA added AERMOD, which incorporates the PRIME downwash algorithm, to the list of preferred models in 2005 as a replacement to ISCST3. CalPuff was added in 2003. The latest version of the U.S. EPA recommended models can be found at the SCRAM Bulletin board located at http://www.epa.gov/scram001. However, any model, whether a U.S. EPA guideline model or otherwise, must be approved for use by the local air district. Recommended models and guidelines for using alternative models are presented in this section. All air dispersion models used to estimate pollutant concentrations for risk assessment analyses must be in the public domain. Classification according to terrain, source type and level of analysis is necessary before selecting a model (see Section 2.4). The selection of averaging times in the modeling analysis is based on the health effects of concern. Annual average concentrations are required for an analysis of carcinogenic or other chronic effects. One-hour maximum concentrations are generally required for analysis of acute effects.

2.9.1 Recommended Models

Recommended air dispersion models to estimate concentrations for risk assessment analyses are generally referenced in US EPA's Guideline on Air Quality Models available at http://www.epa.gov/scram001. Currently AERMOD is recommended for most refined risk assessments in flat or complex terrain and in rural or urban environments[3]. In addition, CalPuff is available where spatial wind fields are highly variable or transport distances are large (e.g., 50 km). AERSCREEN is a screening model based on AERMOD. AERSCREEN can be used when representative meteorological data are unavailable. CTSCREEN is available for screening risk assessments in complex terrain. The most current version of the models should be used for risk assessment analysis. Some facilities may also require models capable of

[3] AERMOD was promulgated by U.S. EPA as a replacement to ISCST3 on November 9, 2006.

special circumstances such as dispersion near coastal areas. For more information on modeling special cases see Sections 2.12 and 2.13.

Most air dispersion models contain provisions that allow the user to select among alternative algorithms to calculate pollutant concentrations. Only some of these algorithms are approved for regulatory application such as the preparation of health risk assessments. The sections in this guideline that provide a description of each recommended model contain information on the specific switches and/or algorithms that must be selected for regulatory application.

To further facilitate the model selection, the District should be consulted for additional recommendations on the appropriate model(s) or a protocol submitted for District review and approval (see Section 2.14.1).

2.9.2 Alternative Models

Alternative models are acceptable if applicability is demonstrated or if they produce results identical or superior to those obtained using one of the preferred models referenced in Section 2.9.1. For more information on the applicability of alternative models refer to the following documents:

- U.S. EPA (2005). "Guideline on Air Quality Models" Section 3.2.2
- U.S. EPA (1992). "Protocol for Determining the Best Performing Model"
- U.S. EPA (1985a). "Interim Procedures for Evaluating Air Quality Models – Experience with Implementation"
- U.S. EPA (1984b). "Interim Procedures for Evaluating Air Quality Models (Revised)"

2.10 Screening Air Dispersion Models

A screening model may be used to provide a maximum concentration that is biased toward overestimation of public exposure. Use of screening models in place of refined modeling procedures is optional unless the District specifically requires the use of a refined model. Screening models are normally used when no representative meteorological data are available and may be used as a preliminary estimate to determine if a more detailed assessment is warranted.

Some screening models provide only 1-hour average concentration estimates. Other averaging periods can be estimated based on the maximum 1-hour average concentration in consultation and approval of the responsible air district. Because of variations in local meteorology, the exact factor selected may vary from one district to another. Table 2.2 provides guidance on the range and typical values applied. The conversion factors are designed to bias predicted longer term averaging periods towards overestimation.

TABLE 2.2 RECOMMENDED FACTORS TO CONVERT MAXIMUM 1-HOUR AVG. CONCENTRATIONS TO OTHER AVERAGING PERIODS (U.S. EPA, 2011, 1995A; ARB, 1994).

Averaging Time	Range	Typical SCREEN3 Recommended	AERSCREEN Recommended
3 hours	0.8 - 1.0	0.9	1.0
8 hours	0.5 - 0.9	0.7	0.9
24 hours	0.2 - 0.6	0.4	0.6
30 days	0.2 - 0.3	0.3	
Annual	0.06 - 0.1	0.08	0.1

AERSCREEN automatically provides the converted concentration for longer than 1-hour averaging periods. For area sources, the AERSCREEN 3, 8, and 24-hour average concentration are equal to the 1-hour concentration. No annual average concentration is calculated. SCREEN3 values are shown for comparison purposes.

2.10.1 AERSCREEN

The AERSCREEN (U.S. EPA, 2011) model is now available and should be used in lieu of SCREEN3 with approval of the local District. AERSCREEN is a screening level air quality model based on AERMOD. AERSCREEN does not require the gathering of hourly meteorological data. Rather, AERSCREEN requires the use of the MAKEMET program which generates a site specific matrix of meteorological conditions for input to the AERMOD model. MAKEMET generates a matrix of meteorological conditions based on local surface characteristics, ambient temperatures, minimum wind speed, and anemometer height.

AERSCREEN is currently limited to modeling a single point, capped stack, horizontal stack, rectangular area, circular area, flare, or volume source. More than one source may be modeled by consolidating the emissions into one emission source.

2.10.2 Valley Screening

The Valley model is designed to simulate a specific worst-case condition in complex terrain, namely that of a plume impaction on terrain under stable atmospheric conditions. The algorithms of the VALLEY model are included in other models such as SCREEN3 and their use is recommended in place of the VALLEY model. The usefulness of the VALLEY model and its algorithms is limited to pollutants for which only long-term average concentrations are required. For more information on the Valley model consult the user's guide (Burt, 1977).

2.10.2.1 Regulatory Options

Regulatory application of the Valley model requires the setting of the following values during a model run:

- Class F Stability (rural) and Class E Stability (urban)
- Wind Speed = 2.5 m/s
- 6 hours of occurrence of a single wind direction (not exceeding a 22.5 deg sector)
- 2.6 stable plume rise factor

2.10.3 CTSCREEN

The CTSCREEN model (Perry et al., 1990) is the screening mode of the Complex Terrain Dispersion Model (CTDMPLUS). CTSCREEN can be used to model single point sources only. It may be used in a screening mode for multiple sources on a case by case basis in consultation with the District. CTSCREEN is designed to provide conservative, yet theoretically more sound, worst-case 1-hour concentration estimates for receptors located on terrain above stack height. Internally-coded time-scaling factors are applied to obtain other averages (see Table 2.3). These factors were developed by comparing the results of simulations between CTSCREEN and CTDMPLUS for a variety of scenarios and provide conservative estimates (Perry et al., 1990). CTSCREEN produces identical results as CTDMPLUS if the same meteorology is used in both models. CTSCREEN accounts for the three-dimensional nature of the plume and terrain interaction and requires detailed terrain data representative of the modeling domain. A summary of the input parameters required to run CTSCREEN is given in Table 2.4. The input parameters are provided in three separate text files. The terrain topography file (TERRAIN) and the receptor information file (RECEPTOR) may be generated with a preprocessor that is included in the CTSCREEN package. In order to generate the terrain topography file the analyst must have digitized contour information.

TABLE 2.3. TIME-SCALING FACTORS INTERNALLY CODED IN CTSCREEN

Averaging Period	Scaling Factor
3 hours	0.7
24 hour	0.15
Annual	0.03

TABLE 2.4. INPUT PARAMETERS REQUIRED TO RUN CTSCREEN

Parameter	File
Miscellaneous program switches	CTDM.IN
Site latitude and longitude (degrees)	CTDM.IN
Site TIME ZONE	CTDM.IN
Meteorology Tower Coordinates (user units)	CTDM.IN
Source Coordinates: x and y (user units)	CTDM.IN
Source Base Elevation (user units)	CTDM.IN
Stack Height (m)	CTDM.IN
Stack Diameter (m)	CTDM.IN
Stack Gas Temperature (K)	CTDM.IN
Stack Gas Exit Velocity (m/s)	CTDM.IN
Emission Rate (g/s)	CTDM.IN
Surface Roughness for each Hill (m)	CTDM.IN
Meteorology: Wind Direction (optional)	CTDM.IN
Terrain Topography	TERRAIN
Receptor Information (coordinates and associated hill number)	RECEPTOR

2.11 Refined Air Dispersion Models

Refined air dispersion models are designed to provide more representative concentration estimates than screening models. In general, the algorithms of refined models are more robust and have the capability to account for site-specific meteorological conditions.

2.11.1 AERMOD

For a wide variety of applications in all types of terrain, the recommended model is AERMOD. AERMOD is a steady-state plume dispersion model for assessment of pollutant concentrations from a variety of sources. AERMOD simulates transport and dispersion from multiple point, area, or volume sources based on an up-to-date characterization of the atmospheric boundary layer. Sources may be located in rural or urban areas and receptors may be located in simple or complex terrain. AERMOD

accounts for building wake effects (i.e., plume downwash) based on the PRIME building downwash algorithms. The model employs hourly sequential preprocessed meteorological data to estimate concentrations for averaging times from one hour to one year (also multiple years). AERMOD is designed to operate in concert with two pre-processor codes: AERMET processes meteorological data for input to AERMOD, and AERMAP processes terrain elevation data and generates receptor information for input to AERMOD. Guidance on input requirements may be found in the AERMOD Users Guide.

2.11.1.1 Regulatory Options

U.S. EPA regulatory application of AERMOD requires the selection of specific switches (i.e., algorithms) during a model run. All the regulatory options can be set by selecting the DFAULT keyword. The U.S. EPA regulatory options, automatically selected when the DFAULT keyword is used, are:

- Stack-tip downwash
- Incorporates the effects of elevated terrain
- Includes calms and missing data processing routines
- Does not allow for exponential decay for applications other than a 4-hour half life for SO_2

Additional information on these options is available in the AERMOD User's Guide.

2.11.1.2 Special Cases

a. Building Downwash:
AERMOD automatically determines if the plume is affected by the wake region of buildings when their dimensions are given. The specification of building dimensions does not necessarily mean that there will be downwash. See section 2.12.1 for guidance on how to determine when downwash is likely to occur.

b. Area Sources:
The area source algorithm in AERMOD does not account for the area that is 1 m upwind from the receptor and, therefore, caution should be exercised when modeling very small area sources (e.g., a few meters wide) with receptors placed within them or within 1 m from the downwind boundary.

c. Volume Sources:
The volume source algorithms in AERMOD require an estimate of the initial distribution of the emission source. Tables that provide information on how to estimate the initial distribution for different sources are given in the AERMOD User's Guide (U.S. EPA, 2004a).

d. Line Sources:
Line sources are a special case of a series of volume or area sources. Where

the emission source is neutrally buoyant, such as a conveyor belt, AERMOD can be used according to the user guide. In the event that the line source is a roadway, then additional considerations are required.

At the present time, CALINE (CALINE3, CAL3QHCR, and CALINE4) is the only model dedicated to modeling the enhanced mechanical and thermal turbulence created by motor vehicles traveling on a roadway. Of these, CAL3QHCR is the only model that accepts hourly meteorological data and can estimate annual average concentrations. However, CALINE uses the Pasquill-Gifford stability categories which are used in the ISCST model. AERMOD is now the preferred plume model over ISCST3 with continuous plume dispersion calculations based on observations but AERMOD does not include the enhanced roadway turbulence.

In the case where roadway emissions dominate the risk assessment, it may be most important to simulate the enhanced thermal and mechanical turbulence from motor vehicles with the CAL3QHCR model. In the case where roadway emissions are a subset of all emissions for the risk assessment, in the case of including roadway emissions along with facility emissions, it may be best to use AERMOD for all emissions, roadway and facility, in order to maintain continuity with one dispersion model for the risk assessment. Most importantly, roadway modeling should be treated on a case-by-case basis in consultation with the District.

Line sources inputs include a composite fleetwide emission factor, roadway geometry, hourly vehicle activity (i.e., diurnal vehicle per hour pattern), hourly meteorological data, and receptor placement. For practical information on how to simulate roadway emissions using these models, see CAPCOA's website at http://www.capcoa.org or the Sacramento Metropolitan AQMD (SMAQMD) website at http://www.airquality.org/ceqa/RoadwayProtocol.shtml. The SMAQMD has a document titled, "Recommended Protocol for Evaluating the Location of Sensitive Land Uses Adjacent to Major Roadways"(January , 2010).

e. Complex Terrain :
AERMOD uses the Dividing Streamline (Hc) concept for complex terrain. Above Hc, the plume is assumed to be "terrain following" in the convective boundary layer. Below Hc, the plume is assumed to be "terrain impacting" in the stable boundary layer. AERMOD computes the concentration at any receptor as a weighted function between the two plume states (U.S. EPA, 2004b)

f. Deposition:
AERMOD contains algorithms to model settling and deposition and require additional information to do so including particle size distribution. For more information consult the AERMOD User's Guide (U.S. EPA, 2004a).

g. Diurnal Considerations:
Systematic diurnal changes in atmospheric conditions are expected along the coast (or any large body of water) or in substantially hilly terrain. The wind speed and direction are highly dependent on time of day as the sun rises and begins to heat the Earth. The sun heats the surface of the land faster than the water surface. Therefore the air above the land warms up sooner than over water. This creates a buoyant effect of warm air rising over land and the cool air from over water moves in to fill the void. Near large bodies of water (e.g., the ocean) this is known as a sea breeze. In complex terrain this is known as upslope flow as the hot air follows the terrain upwards. When the sun sets and the surface of the land begins to cool, the air above also cools and creates a draining effect. Near the water this is the land breeze; in complex terrain this is known as downslope or drainage flow. In addition, for the sea breeze, the atmospheric conditions change rapidly from neutral or stable conditions over water to unstable conditions over land.

Near the large bodies of water the sea breeze is typical in the afternoon and the land breeze is typical for the early morning before sunrise. In complex terrain upslope flow is typical in the afternoon, while drainage flow is typical at night. For these reasons, it is especially important to simulate facility emissions with a hourly diurnal pattern reflective of source activity so that the risk assessment is representative of daily conditions.

h. 8-hour Modeling for the Offsite Worker's Exposure and Residential Exposure:
If the ground level air concentrations from a facility operation 5 days a week/ 8 hours per day have been estimated by a 24 hour per day annual average, an adjustment factor can be applied to estimate the air concentration that offsite worker with the same schedule would be exposed to. The 24 hour annual average concentration is multiplied times 4.2.

If the meteorology during the time that the facility is emitting is used, hourly model simulations need to be post-processed to cull out the data needed for the offsite worker exposure. See Appendix M for information on how to calculate the refined offsite worker concentrations using the hourly raw results from the AERMOD air dispersion model. For more discussion on worker exposure, see Section 2.8.1.

2.11.2 CTDMPLUS

CTDMPLUS is a Gaussian air quality model for use in all stability conditions in complex terrain. In comparison with other models, CTDMPLUS requires considerably more detailed meteorological data and terrain information that must be supplied using specifically designed preprocessors.

CTDMPLUS was designed to handle up to 40 point sources.

2.12 Modeling Special Cases

Special situations arise in modeling some sources that require considerable professional judgment; a few of which are outlined below. It is recommended that the reader consider retaining professional consultation services if the procedures are unfamiliar.

2.12.1 Building Downwash

The entrainment of a plume in the wake of a building can result in the "downwash" of the plume to the ground. This effect can increase the maximum ground-level concentration downwind of the source. Therefore, stack sources must be evaluated to determine whether building downwash is a factor in the calculation of maximum ground-level concentrations.

The PRIME algorithm, included with AERMOD, has several advances in modeling building downwash effects including enhanced dispersion in the wake, reduced plume rise due to streamline deflection and increased turbulence, and continuous treatment of the near and far wakes (Schulman, 2000).

Complicated situations involving more than one building may necessitate the use of the Building Profile Input Program (BPIP) which can be used to generate the building dimension section of the input file of the ISC models (U.S. EPA, 1993). The BPIP program calculates each building's direction-specific projected width. The Building Profile Input Program for PRIME (BPIPPRM) is the same as BPIP but includes an algorithm for calculating downwash values for input into the PRIME algorithm which is contained in such models as AERMOD. The input structure of BPIPPRM is the same as that of BPIP.

2.12.2 Deposition

There are two types of deposition; wet deposition and dry deposition. Wet deposition is the incorporation of gases and particles into rain-, fog- or cloud water followed by a precipitation event and also rain scavenging of particles during a precipitation event. Wet deposition of gases is therefore more important for water soluble chemicals; particles (and hence particle-phase chemicals) are efficiently removed by precipitation events (Bidleman, 1988). Dry deposition refers to the removal of gases and particles from the atmosphere.

In the Air Toxics "Hot Spots" program, deposition is quantified for particle-bound pollutants and not gases. Wet deposition of water-soluble gas phase chemicals is thus not considered. When calculating pollutant mass deposited to surfaces without including depletion of pollutant mass from the plume airborne concentrations remaining in the plume and deposition to surfaces can be overestimated, thereby resulting in overestimates of both the inhalation and multi-pathway risk estimates. However, neglecting deposition in the air dispersion model, while accounting for it in the multi-

pathway health risk assessment, is a conservative, health protective approach (CAPCOA, 1987; Croes, 1988). Misapplication of plume depletion can also lead to possible underestimates of multi-pathway risk and for that reason no depletion is the default assumption. If plume depletion is incorporated, then some consideration for possible resuspension is warranted. An alternative modeling methodology accounting for plume depletion can be discussed with the Air District and used in an approved modeling protocol.

Although not generally used, several air dispersion models can provide downwind concentration estimates that take into account the upwind deposition of pollutants to surfaces and the consequential reduction of mass remaining in the plume. Air dispersion models having deposition and plume depletion algorithms require particle distribution data that are not always readily available. These variables include particle size, mass fraction, and density for input to AERMOD. In addition, the meteorological fields need to include additional parameters including relative humidity, precipitation, cloud cover, and surface pressure. Consequently, depletion of pollutant mass from the plume often is not taken into account.

In conclusion, multipathway risk assessment analyses normally incorporate deposition to surfaces in a screening mode, specifically by assigning a default deposition velocity of 2 cm/s for controlled sources and 5 cm/s for uncontrolled sources in lieu of actual measured size distributions (ARB, 1989). For particles (and particle-phase chemicals), the deposition velocity depends on particle size and is minimal for particles of diameter approximately 0.1-1 micrometer; smaller and larger particles are removed more rapidly.

2.12.3 Short Duration Emissions

Short-duration emissions (i.e., much less than an hour) require special consideration. In general, “puff models” provide a better characterization of the dispersion of pollutants having short-duration emissions. Continuous Gaussian plume models have traditionally been used for averaging periods as short as about 10 minutes and are not recommended for modeling sources having shorter continuous emission duration.

2.12.4 Fumigation

Fumigation occurs when a plume that was originally emitted into a stable layer in the atmosphere is mixed rapidly to ground-level when unstable air below the plume reaches plume level. Fumigation can cause very high ground-level concentrations. Typical situations in which fumigation occurs are:

- Breaking up of a nocturnal radiation inversion by solar warming of the ground surface (rising warm unstable air); note that the break-up of a nocturnal radiation inversion is a short-lived event and should be modeled accordingly.

- Shoreline fumigation caused by advection of pollutants from a stable marine environment to an unstable inland environment

- Advection of pollutants from a stable rural environment to a turbulent urban environment

SCREEN3 incorporates concentrations due to inversion break-up and shoreline fumigation and is limited to maximum hourly evaluations. The Offshore and Coastal Dispersion Model incorporates overwater plume transport and dispersion as well as changes that occur as the plume crosses the shoreline – hourly meteorological data are needed from both offshore and onshore locations.

2.12.5 Raincap on Stack

The presence of a raincap or any obstacle at the top of the stack hinders the momentum of the exiting gas. The extent of the effect is a function of the distance from the stack exit to the obstruction and of the dimensions and shape of the obstruction.

On the conservative side, the stack could be modeled as having a non-zero, but negligible exiting velocity, effectively eliminating any momentum rise. Such an approach would result in final plume heights closer to the ground and therefore higher concentrations nearby. There are situations where such a procedure might lower the actual population-dose and a comparison with and without reduced exit velocity should be examined.

Plume buoyancy is not strongly reduced by the occurrence of a raincap. Therefore, if the plume rise is dominated by buoyancy, it is not necessary to adjust the stack conditions. (The air dispersion models determine plume rise by either buoyancy or momentum, whichever is greater.)

The stack conditions should be modified when the plume rise is dominated by momentum and in the presence of a raincap or a horizontal stack. Sensitivity studies with the SCREEN3 model, on a case-by-case basis, can be used to determine whether plume rise is dominated by buoyancy or momentum. The District should be consulted before applying these procedures.

- Set exit velocity to 0.001 m/sec
- Turn stack tip downwash off
- Reduce stack height by 3 times the stack diameter

Stack tip downwash is a function of stack diameter, exit velocity, and wind speed. The maximum stack tip downwash is limited to three times the stack diameter in the AERMOD air dispersion model. In the event of a horizontal stack, stack tip downwash should be turned off and no stack height adjustments should be made.

Note: This approach may not be valid for large (several meter) diameter stacks.

An alternative, more refined, approach could be considered for stack gas temperatures which are slightly above ambient (e.g., ten to twenty degrees Fahrenheit above

ambient). In this approach, the buoyancy and the volume of the plume remain constant and the momentum is minimized.

- Turn stack tip downwash off
- Reduce stack height by 3 times the stack diameter ($3D_o$)
- Set the stack diameter (D_b) to a large value (e.g., 10 meters)
- Set the stack velocity to $V_b = V_o (D_o/D_b)^2$

Where V_o and D_o are the original stack velocity and diameter and V_b and D_b are the alternative stack velocity and diameter for constant buoyancy. This approach is advantageous when $D_b >> D_o$ and $V_b << V_o$ and should only be used with District approval.

In the presence of building downwash and in the event that PRIME downwash is being utilized in AERMOD, an alternative approach is recommended. PRIME algorithms use the stack diameter to define initial plume radius and to solve conservation laws. The user should input the actual stack diameter and exit temperature but set the exit velocity to a nominally low value (e.g., 0.001 m/s). Also since PRIME does not explicitly consider stack-tip downwash, no adjustments to stack height should be made.

Currently US-EPA is BETA testing options for capped and horizontal releases in AERMOD. It is expected that these options will replace the above guidance when BETA testing is complete.

2.12.6 Landfill Sites

Landfills should be modeled as area sources. The possibility of non-uniform emission rates throughout the landfill area should be investigated. A potential cause of non-uniform emission rates would be the existence of cracks or fissures in the landfill cap (where emissions may be much larger). If non-uniform emissions exist, the landfill should be modeled with several smaller areas assigning an appropriate emission factor to each one of them, especially if there are nearby receptors (distances on the same order as the dimensions of the landfill).

2.13 Specialized Models

Some models have been developed for application to very specific conditions. Examples include models capable of simulating sources where both land and water surfaces affect the dispersion of pollutants and models designed to simulate emissions from specific industries.

2.13.1 Buoyant Line and Point Source Dispersion Model (BLP)

BLP is a Gaussian plume dispersion model designed for the unique modeling problems associated with aluminum reduction plants, and other industrial sources where plume rise and downwash effects from stationary line sources are important.

2.13.1.1 Regulatory Application

Regulatory application of BLP model requires the selection of the following options:

- rural (IRU=l) mixing height option;
- default (no selection) for all of the following: plume rise wind shear (LSHEAR), transitional point source plume rise (LTRANS), vertical potential temperature gradient (DTHTA), vertical wind speed power law profile exponents (PEXP), maximum variation in number of stability classes per hour (IDELS), pollutant decay (DECFAC), the constant in Briggs' stable plume rise equation (CONST2), constant in Briggs' neutral plume rise equation (CONST3), convergence criterion for the line source calculations (CRIT), and maximum iterations allowed for line source calculations (MAXIT); and
- terrain option (TERAN) set equal to 0.0, 0.0, 0.0, 0.0, 0.0, 0.0

For more information on the BLP model consult the user's guide (Schulman and Scire, 1980).

2.13.2 Offshore and Coastal Dispersion Model (OCD)

OCD (DiCristofaro and Hanna, 1989) is a straight-line Gaussian model developed to determine the impact of offshore emissions from point, area or line sources on the air quality of coastal regions. OCD incorporates "over-water" plume transport and dispersion as well as changes that occur as the plume crosses the shoreline. Hourly meteorological data are needed from both offshore and onshore locations. Additional data needed for OCD are water surface temperature, over-water air temperature, mixing height, and relative humidity.

Some of the key features include platform building downwash, partial plume penetration into elevated inversions, direct use of turbulence intensities for plume dispersion, interaction with the overland internal boundary layer, and continuous shoreline fumigation.

2.13.2.1 Regulatory Application

OCD has been recommended for use by the Minerals Management Service for emissions located on the Outer Continental Shelf (50 FR 12248; 28 March 1985). OCD is applicable for over-water sources where onshore receptors are below the lowest source height. Where onshore receptors are above the lowest source height, offshore plume transport and dispersion may be modeled on a case-by-case basis in consultation with the District.

2.13.3 Shoreline Dispersion Model (SDM)

SDM (PEI, 1988) is a hybrid multipoint Gaussian dispersion model that calculates source impact for those hours during the year when fumigation events are expected using a special fumigation algorithm and the MPTER regulatory model for the remaining hours.

SDM may be used on a case-by-case basis for the following applications:

- tall stationary point sources located at a shoreline of any large body of water;
- rural or urban areas;
- flat terrain;
- transport distances less than 50 km;
- 1-hour to 1-year averaging times.

2.14 Interaction with the District

The risk assessor must contact the District to determine if there are any specific requirements. Examples of such requirements may include: specific receptor location guidance, specific usage of meteorological data and specific report format (input and output).

2.14.1 Submittal of Modeling Protocol

It is strongly recommended that a modeling protocol be submitted to the District for review and approval prior to extensive analysis with an air dispersion model. The modeling protocol is a plan of the steps to be taken during the air dispersion modeling process. Following is an example of the format that may be followed in the preparation of the modeling protocol. Consult with the District to confirm format and content requirements or to determine the availability of District modeling guidelines before submitting the protocol.

Emissions

- Specify that emission estimates for all substances for which emissions were required to be quantified will be included in the risk assessment. This includes both annual average emissions and maximum one-hour emissions of each pollutant from each process.
- Specify the format in which the emissions information will be provided (consult with the District concerning format prior to submitting the protocol).
- Specify the basis for using emissions data, other than that included in the previously submitted emission inventory report, for the risk assessment (consult with the District concerning the use of updated emissions data prior to submitting the protocol).

- Specify the format for presenting release parameters (e.g., stack height and diameter, stack gas exit velocity, release temperature) for each process as part of the risk assessment (consult with the District concerning the format prior to submitting the protocol).
- A revised emission inventory report must be submitted to the District and forwarded by the District to the CARB if revised emission data are used.

Models

- Identify the model(s) to be used, including the version number.
- Identify any additional models to be run if receptors are found above stack height.
- Specify which model results will be used for receptors above stack height.
- Specify the format for presenting the model options selected for each run (consult with the District concerning the format prior to submitting the protocol).

Meteorological Data

- Specify type, source, and year (e.g., hourly surface data, upper air mixing height information).
- Evaluate whether the data are representative.
- Describe QA/QC procedures.
- Identify any gaps in the data; if so, describe how the data gaps are filled.

Deposition

- Specify method to calculate deposition (if applicable).

Receptors

- Identify the method to determine maximum exposed individual for residential and occupational areas for long-term exposures (e.g., a Cartesian grid at 20-meter grid increments).
- Identify whether spatially averaged supplemental results will be submitted in addition to the modeling results from the maximum concentration at the single location. Identify the spatial average grid receptor domain and resolution and procedure for centering the grid on the maximum concentration. For tilted spatial average fields, identify whether rectangular or polar fields will be used. This information should be provided for each receptor type (e.g., PMI, MEIR, and MEIW) and any water body or pasture land that will use spataial averaging for determining multipathway disposition exposure.
- Identify method to determine maximum short-term impact.
- Identify the methods and data sources for population and land-use that will be used to evaluate cancer risk in the vicinity of the facility for purposes of

calculating cancer burden or population exposure estimates (e.g., centroids of the census tracts in the area within the zone of impact).

- Specify that UTM coordinates and street addresses, where possible, will be provided for specified receptor locations.

Maps

- Specify which cancer risk isopleths will be plotted (e.g., 10^{-6}, 10^{-7}; see Section 2.6.1).
- Specify which hazard indices will be plotted for acute and chronic (e.g., 0.1, 1, 10).

2.15 Report Preparation

This section describes the information related to the air dispersion modeling process that needs to be reported in the risk assessment. The District may have specific requirements regarding format and content (see Section 2.14). Sample calculations should be provided at each step to indicate how reported emissions data were used. Reviewing agencies must receive input, output, and supporting files of various model analyses on computer-readable media (e.g., CD). See the Air Toxics Risk Assessment Guidance Manual on the ARB website (http://www.arb.ca.gov/toxics/harp/harp.htm) for information on which files that should be included with a HARP risk assessments.

2.15.1 Information on the Facility and its Surroundings

Report the following information regarding the facility and its surroundings:

- Facility Name
- Location (UTM coordinates and street address)
- Land use type (see Section 2.4)
- Local topography
- Facility plot plan identifying:
  - source locations
  - property line
  - horizontal scale
  - building heights
  - emission sources

2.15.2 Source and Emission Inventory Information†

2.15.2.1 Source Description and Release Parameters

Report the following information for each source in table format:

- Source identification number used by the facility
- Source name
- Source location using UTM coordinates
- Source height (m)
- Source dimensions (e.g., stack diameter, building dimensions, area size) (m)
- Exhaust gas exit velocity (m/s)
- Exhaust gas volumetric flow rate (ACFM)
- Exhaust gas exit temperature (K)

2.15.2.2 Source Operating Schedule

The operating schedule for each source should be reported in table form including the following information:

- Number of operating hours per day and per year (e.g., 0800-1700, 2700 hr/yr)
- Number of operating days per week (e.g., Mon-Sat)
- Number of operating days or weeks per year (e.g., 52 wk/yr excluding major holidays)

2.15.2.3 Emission Control Equipment and Efficiency

Report emission control equipment and efficiency by source and by substance

2.15.2.4 Emissions Data Grouped By Source

Report emission rates for each toxic substance, grouped by source (i.e., emitting device or process identified in Inventory Report), in table form including the following information:

- Source name
- Source identification number
- Substance name and CAS number (from Inventory Guidelines)
- Annual average emissions for each substance (lb/yr)
- Hourly maximum emissions for each substance (lb/hr)

2.15.2.5 Emissions Data Grouped by Substance

Report facility total emission rate by substance for all emitted substances listed in the Air Toxics “Hot Spots” Program including the following information:

- Substance name and CAS number (from Inventory Guidelines)
- Annual average emissions for each substance (lb/yr)
- Hourly maximum emissions for each substance (lb/hr)

2.15.2.6 Emission Estimation Methods

Report the methods used in obtaining the emissions data indicating whether emissions were measured or estimated. Clearly indicate any emission data that are not reflected

in the previously submitted emission inventory report and submit a revised emission inventory report to the district. A reader should be able to reproduce the risk assessment without the need for clarification.

2.15.2.7 List of Substances

Include tables listing all "Hot Spots" Program substances which are emitted, plus any other substances required by the District. Indicate substances to be evaluated for cancer risks and noncancer effects.

2.15.3 Exposed Population and Receptor Location

Report the following information regarding exposed population and receptor locations:

- Description of zone of impact including map showing the location of the facility, boundaries of zone of impact, census tracts, emission sources, sites of maximum exposure, and the location of all appropriate receptors. This should be a true map (one that shows roads, structures, etc.), drawn to scale, and not just a schematic drawing. USGS 7.5 minute maps or GIS based maps are usually the most appropriate choices. (If significant development has occurred since the user's survey, this should be indicated.)
- Separate maps for the cancer risk zone of impact and the hazard index (noncancer) zone of impact. The cancer zone of impact should include isopleths down to at least the 1/1,000,000 risk level. Because some districts use a level below 1/1,000,000 to define the zone of impact, the District should be consulted. Two separate isopleths (to represent both chronic and acute HI) should be created to define the zone of impact for the hazard index from both inhalation and noninhalation pathways greater than or equal to 0.5. The point of maximum impact (PMI), maximum exposed individual at a residential receptor (MEIR), and maximum exposed individual worker (MEIW) for both cancer and noncancer risks should be located on the maps.
- Tables identifying population units and sensitive receptors (UTM coordinates and street addresses of specified receptors).
- Heights or elevations of the receptor points.
- For each receptor type (e.g., PMI, MEIR, and MEIW) that will utilize spatial averaging, the domain size and grid resolution must be clearly identified. If another domain or grid resolution other than 20 meters by 20 meters with 5-meter grid spacing will be used for a receptor, then care should be taken to determine the proper domain size and grid resolution that should be used. For a worker, the HRA shall support all assumptions used, including, but not limited to, documentation for all workers showing the area where each worker routinely performs their duties. The final domain size should not be greater than the smallest area of worker movement. Other considerations for determining domain size and grid spacing resolution may include an evaluation of the concentration gradients across the worker area. The grid spacing used within the domain should be sufficient in number and detail to obtain a representative concentration

across the area of interest. When spatial averaging over the deposition area of a pasture or water body, care should be taken to determine the proper domain size to make sure it includes all reasonable areas of potential deposition. The size and shape of the pasture or water body of interest should be identified and used for the modeling domain. The grid spacing or resolution used within the domain should be sufficient in detail to obtain a representative deposition concentration across the area of interest. One way to determine the grid resolution is to include an evaluation of the concentration gradients across the deposition area. The HRA shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the pasture or water body, maps, representative coordinates, grid resolution, concentration gradients, etc.). The use or spatial averaging is subject to approval by the reviewing authority. This includes the size of the domain and grid resolution that is used for spatial averaging of a worksite or multipathway deposition area.

2.15.4 Meteorological Data

If meteorological data were not obtained directly from the District, then the report must clearly indicate the data source and time period used. Meteorological data not obtained from the District must be submitted in electronic form along with justification for their use including information regarding representativeness and quality assurance.

The risk assessment should indicate if the District required the use of a specified meteorological data set. All memos indicating the District's approval of meteorological data should be attached in an appendix.

2.15.5 Model Selection and Modeling Rationale

The report should include an explanation of the model chosen to perform the analysis and any other decisions made during the modeling process. The report should clearly indicate the name of the models that were used, the level of detail (screening or refined analysis) and the rationale behind the selection.

Also report the following information for each air dispersion model used:

- version number.
- selected options and parameters in table form.

2.15.6 Air Dispersion Modeling Results

- Maximum hourly and annual average concentrations of chemicals at appropriate receptors such as the residential and worker MEI receptors
- Annual average and maximum one-hour (and 30-day average for lead only) concentrations of chemicals at appropriate receptors listed and referenced to computer printouts of model outputs

- Model printouts (numbered), annual concentrations, maximum hourly concentrations
- Disk with input/output files for air dispersion program (e.g., the AERMOD input file containing the regulatory options and emission parameters, receptor locations, meteorology, etc.)
- Include tables that summarize the annual average concentrations that are calculated for all the substances at each site. The use of tables that present the relative contribution of each emission point to the receptor concentration is recommended. (These tables should have clear reference to the computer model which generated the data. It should be made clear to any reader how data from the computer output was transferred to these tables.) [As an alternative, the above two tables could contain just the values for sites of maximum impact (i.e., PMI, MEIR and MEIW), and sensitive receptors, if required. All the values would be found in the Appendices.]

(†) Health and Safety Code section 44346 authorizes facility operators to designate certain "Hot Spots" information as trade secret. Section 44361(a) requires districts to make health risk assessments available for public review upon request. Section 44346 specifies procedures to be followed upon receipt of a request for the release of trade secret information. See also the Inventory Guidelines Report regarding the designation of trade secret information in the Inventory Reports.

2.16 References

Auer Jr., A.H. (1978). Correlation of Land Use and Cover with Meteorological Anomalies. *Journal of Applied Meteorology*, 17:(5):636-643.

ARB (1994). ARB memorandum dated 4/11/94 from A. Ranzieri to J. Brooks on the subject, "One-hour to Thirty-day Average Screening Factor."

ARB (1989). "Screening Deposition Velocities," Internal memorandum from A. Ranzieri to G. Shiroma dated 8/17/89.

Bidleman, T.F. (1988). "Atmospheric processes", Environmental Science & Technology, 22, pp. 361-367

Bjorklund, J.R. and J.F. Bowers (1982). User's Instructions for the SHORTZ and LONGZ Computer Programs, Volumes I and II. EPA-903/9-82-004A and B. U.S. Environmental Protection Agency. Philadelphia, PA.

Burt, E.W. (1977). Valley Model User's Guide. EPA-450/2-77-018. U.S. Environmental Protection Agency, Research Triangle Park, NC.

CAPCOA (1987). "Deposition Rate Calculations for Air Toxics Source Assessments," in Air Toxics Assessment Manual, Appendix C.7.

Catalano, J.A., D.B. Turner and H. Novak (1987). User's Guide for RAM - Second Edition. U.S. Environmental Protection Agency. Research Triangle Park, NC. (Distributed as part of UNAMAP Version 6 Documentation)

Chico, T. and J. A. Catalano (1986). Addendum to the User's Guide for MPTER. U. S. Environmental Protection Agency. Research Triangle Park, NC.

Croes, B. (1988). "Deposition Rate Calculations for Air Toxic Risk Assessments in California," Proceedings of the 81st Annual Meeting of the Air Pollution Control Association, Dallas, TX, June 20-24, 1988.

DiCristofaro, D. C. and S. R. Hanna (1989). OCD: The Offshore and Coastal Dispersion Model, Version 4. Volume I: User's Guide, and Volume II: Appendices. Sigma Research Corporation, Westford, MA. (NTIS Nos. PB 93-144384 and PB 93-144392)

Irwin, J.S. (1978). Proposed Criteria for Selection of Urban Versus Rural Dispersion Coefficients. (Draft Staff Report). Meteorology and Assessment Division. U.S. Environmental Protection Agency, Research Triangle Park, NC. (Docket No. A-80-46, II-B-8).

PEI Associates (1988). User's Guide to SDM - A Shoreline Dispersion Model. U.S. EPA Publication No. EPA-450/4-88-017. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Perry, S.G., D.J. Burns, A.J. Cimorelli (1990). User's Guide to CTDMPLUS: Volume 2. The Screening Mode (CTSCREEN). EPA-600/8-90-087. Office of Research and Development, U.S. Environmental Protection Agency, Research Triangle Park, NC.

Pierce, T.E., D.B. Turner, J.A. Catalano, and F.V. Hale (1982). PTPLU - A Single Source Gaussian Dispersion Algorithm User's Guide. EPA-600-/8-82-014. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Pierce, T.E. (1986). Addendum to PTPLU - A Single Source Gaussian Dispersion Algorithm. EPA/600/8-86-042. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Schulman, L.L., and J.S. Scire (l980). Buoyant Line and Point Source (BLP) Dispersion Model User's Guide. Document P-7304B. Environmental Research and Technology, Inc., Concord, MA. (NTIS No. PB 8l-l64642)

Schulman, L.L., Strimaitis, D. G., and Scire, J. S. (2000). "Development and Evaluation of the PRIME Plume Rise and Building Downwash Model." Journal of the Air and Waste Management Association, Volume 50:378-390, March 2000.

Tikvart, J. (1993). "Proposal for Calculating Plume Rise for Stacks with Horizontal Releases or Rain Caps for Cookson Pigment, Newark, New Jersey," Internal memorandum from J. Tikvart to K. Eng dated 7/9/93.

Turner, D. and J.H. Novak (1978). User's Guide for RAM. Vol. 1. Algorithm Description and Use, Vol. II. Data Preparation and Listings. EPA-600/8-78-016a and b. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1984a). Calms Processor (CALMPRO) User's Guide. EPA-901/9-84-001. U.S. Environmental Protection Agency, Region I, Boston, MA.

U.S. EPA (1984b). Interim Procedures for Evaluating Air Quality Models (Revised). EPA-450/4-84-023. U.S. Environmental Protection Agency, Research Triangle Park, NC. (NTIS No. PB 85-106060)

U.S. EPA (1985a). Interim Procedures for Evaluating Air Quality Models: Experience with Implementation. U.S. EPA Publication No. EPA-450/4-85-006. U.S. Environmental Protection Agency, Research Triangle Park, NC. (NTIS No. PB 85-242477)

U.S. EPA (1985b). Guideline for Determination of Good Engineering Practice Stack Height (Technical Support Document for the Stack Height Regulations) - Revised EPA-450/4-80-023R, U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1992). Protocol for Determining the Best Performing Model. U.S. EPA Publication No. EPA-454/R-92-025. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1993). User's Guide to the Building Profile Input Program (BPIP). Revised February, 1995. EPA-454/R-93-038. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1995a). Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised. EPA-450/R-92-019. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1995b). User's Guide for the Industrial Source Complex (ISC) Dispersion Models. Volume I: User Instructions. EPA-454/B-95-003a. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1995c). User's Guide for the Industrial Source Complex (ISC) Dispersion Models. Volume II: User Instructions. EPA-454/B-95-003a. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1995d). SCREEN3 Model User's Guide. EPA-454/B-95-004. U.S. Environmental Protection Agency. Research Triangle Park, NC.

U.S. EPA (1995e). On-Site Meteorological Program Guidance For Regulatory Modeling Applications. EPA-450/4-87-013. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2004a). User's Guide for the AMS/EPA Regulatory Model - AERMOD. EPA-454/B-03-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2004b). AERMOD: Description of Model Formulation. EPA-454/R-03-004. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2005). Federal Register / Volume 70, Number 216 / November 9, 2005 / Rules and Regulations, 40 CFR Part 51 Appendix W, Revision to the Guideline on Air Quality Models, U.S. Environmental Protection Agency.

U.S. EPA (2011). AERSCREEN User's Guide. EPA-454/B-11-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

3 Daily Breathing Rates

3.1 Introduction

This chapter presents age-specific breathing rates for use in health risk assessments for short-term exposure to maximum 1-hour facility emissions and for long-term daily average exposures resulting from continuous or repeated 8-hour exposure. The specified age ranges of interest in the "Hot Spots" program are ages third trimester, 0<2, 2<9, 2<16, 16<30 and 16-70 years.

The term ventilation rate has been frequently used for the metric of volume of air inhaled per minute (i.e., mL/min) and is used in this document to describe short-term, one hour exposures. For convenience, the term "breathing rate" is applied throughout this chapter for chronic daily exposure, both to the metric of volume of air inhaled per day (L/day) and the volume of air inhaled per kg body weight per day (L/kg-day). The normalized daily breathing rate in L/kg-day is the preferred metric for use in the "Hot Spots" program. The term "respiratory rate" is not used in this chapter interchangeably with "breathing rate" because respiratory rate usually represents the number of breaths taken per unit time, and not the volume of air taken in per unit time.

The 8-hour breathing rates were developed for specialized exposure scenarios that involve exposures only during facility operations of about 8-12 hours/day. Eight-hour breathing rates reflect exposures to off-site workers or exposures that may occur in schools when class is in session. Ventilation rates for 1-hour exposure were developed to meet the SB-352 mandate for school districts to conduct a risk assessment at school sites located within 100 meters of a freeway or busy roadway. These ventilation rates were developed for exposures to 1-hour maximum facility emissions that may occur during passive activities such as sitting at a desk during class instruction or during higher intensity activities such as play during recess.

OEHHA recommends the breathing rates presented in Section 3.2. Various published methods for deriving daily breathing rates and their advantages and limitations are discussed in Sections 3.3 to 3.7. Where possible, the breathing rates from these reports were re-evaluated to correspond with the five specific age groups used in OEHHA's risk assessment guidelines.

At elevations above 5000 feet, the ventilation rate will increase due to lower air pressure (NOLS, 2012). The respiratory rate at this elevation peaks at one week and then slowly decreases over the next few months, although it tends to remain higher than its normal rate at sea level. There have been a few facilities located at 5000 feet or higher that have been required to produce a Hot Spots risk assessment. However, long-term residents at high altitude will have breathing rates near what is found in residents at sea level. OEHHA does not anticipate any adjustments will be needed to the breathing rates at higher altitudes in California, although the Districts should consider this issue and adjust if needed for very high altitude facilities.

3.2 Breathing Rate Recommendations

3.2.1 Long-Term Breathing Rates

The recommended long-term daily breathing rate point estimates in Table 3.1 are based on a mean of two different methods used to determine daily breathing rates, the doubly labeled water method and an energy intake approach based on food consumption data from the Continuing Survey of Food Intake of Individuals (CSFII) (See Section 3.5.5). These methods are described in detail below. The recommended distributions for stochastic analysis are presented in Tables 3.2a-b. The breathing rates normalized to body weight are expressed in L/kg-day, and the non-body weight-normalized breathing rates are expressed in m^3/day. All values were rounded to two or three significant figures.

Table 3.1. Recommended Point Estimates for Long-Term Daily Breathing Rates

	3rd Trimester	0<2 years	2<9 years	2<16 years	16<30 years	16<70 years
	L/kg-day					
Mean	225	658	535	452	210	185
95th Percentile	361	1090	861	745	335	290
	m^3/day					
Mean	15.3	6.2	10.7	13.3	15.0	13.9
95th Percentile	23.4	11.2	16.4	22.6	23.5	22.9

OEHHA calculated mean and high end breathing rates for the third trimester assuming the dose to the fetus during the third trimester was the same as that to the mother.

TABLE 3.2a. Recommended Breathing Rate Distributions (L/kg-day) by Age Group for Stochastic Analysis

	3rd Trimester	0<2 years	2<9 years	2<16 years	16<30 years	16-70 years
Distribution	Max extreme	Max extreme	Max extreme	Log-normal	Logistic	Logistic
Minimum	78	196	156	57	40	13
Maximum	491	2,584	1,713	1,692	635	860
Scale	59.31	568.09	125.59		40.92	36.19
Likeliest	191.50	152.12	462.61			
Location				-144.06		
Mean	225	658	535	452	210	185
Std Dev	72	217	168	172	75	67
Skewness	0.83	2.01	1.64	1.11	0.83	1.32
Kurtosis	3.68	10.61	7.88	6.02	5.17	10.83
Percentiles						
5%	127	416	328	216	96	86
10%	142	454	367	259	118	104
25%	179	525	427	331	161	141
50%	212	618	504	432	207	181
75%	260	723	602	545	252	222
80%	273	758	631	572	261	233
90%	333	934	732	659	307	262
95%	361	1090	861	745	335	290
99%	412	1430	1,140	996	432	361

TABLE 3.2b. Recommended Breathing Rate Distributions (M^3/day) by Age Group for Stochastic Analysis

	3rd Trimester	0<2 years	2<9 years	2<16 years	16<30 years	16-70 years
Distribution	Logistic	Log-normal	Log-normal	Log-normal	Logistic	Log-normal
Minimum	4.0	0.8	2.7	2.7	1.5	1.8
Maximum	29.0	20.1	31.7	52.3	75.4	75.4
Scale	2,403.72				2,992.97	
Location		-650.7	-1,072.8	598.9		-8,251.3
Mean	15.1	6.2	10.7	13.3	15.0	13.9
Std Dev	4.3	2.6	3.1	4.9	5.4	5.4
Skewness	0.48	1.06	0.912	1.39	1.16	1.42
Kurtosis	3.73	4.69	5.18	7.14	12.22	11.19
Percentiles						
5%	8.6	2.9	6.1	6.9	6.4	6.3
10%	10.4	3.3	6.9	8.1	8.5	7.6
25%	12.3	4.4	8.5	9.9	11.8	10.3
50%	15.1	5.8	10.4	12.3	14.7	13.6
75%	17.6	7.6	12.4	15.9	18.0	16.8
80%	18.2	8.1	13.0	16.7	18.9	17.6
90%	21.4	9.6	14.8	19.5	21.5	20.1
95%	23.4	11.2	16.4	22.6	23.5	22.9
99%	28.8	13.9	20.0	28.1	29.9	28.0

3.2.2 Eight-hour Breathing Rate Point Estimates

The 8-hour breathing rates are based on minute ventilation rates derived by U.S. EPA (2009). The minute ventilation rates, presented in Section 3.6, were multiplied by 480 (60 min x 8) to generate 8-hour breathing rate point estimates shown in Table 3.3. The 8-hour breathing rates may be useful for cancer risk assessment for the off-site worker exposure scenario, and school exposures to facility emissions. They may also be useful for evaluating residential exposures where the facility operates non-continuously. The 8-hour breathing rates vary depending on the intensity of the activity. Exposed individuals may be engaged in activities ranging from watching TV to desk work, which would reflect breathing rates of sedentary/passive or light activities, to yard work or farm worker activities, which would reflect breathing rates of moderate intensity or greater. Breathing rates resulting from high intensity activities generally cannot be sustained for an 8-hour period (see Section 3.6).

OEHHA recommends using point estimate 8-hour breathing rates in L/kg-8-hrs based on the mean and 95th percentile of moderate intensity activities, 170 and 230 L/kg-8-hrs, respectively, for adults 16-70 yrs old. Point estimates for lower breathing rates of

sedentary/passive and light intensity work activities may be used in site-specific scenarios (i.e., work in which activity is limited to desk jobs or similar work). Pregnant women will generally participate in lower intensity activities than non-pregnant women, but as shown in Tables 3.1 and 3.2, breathing rate normalized to body weight will be slightly greater than breathing rates of adult men and non-pregnant women combined. OEHHA recommends using the mean and 95th percentile 8-hour breathing rates based on moderate intensity activity of 16<30 year-olds for third trimester women.

Table 3.3a. Eight Hour Breathing Rate (L/kg-8 Hr) Point Estimates for Males and Females Combined

	0<2 years	2<9 years	2<16 years	16<30 years	16-70 years
	Sedentary & Passive Activities (METS ≤ 1.5)				
Mean	200	100	80	30	30
95th Percentile	250	140	120	40	40
	Light Intensity Activities (1.5 < METs ≤ 3.0)				
Mean	490	250	200	80	80
95th Percentile	600	340	270	100	100
	Moderate Intensity Activities (3.0 < METs ≤ 6.0)				
Mean	890	470	380	170	170
95th Percentile	1200	640	520	240	230

Table 3.3b. Eight-Hour Breathing Rate (M^3/8-Hr) Point Estimates for Males and females Combined

	0<2 years	2<9 years	2<16 years	16<30 years	16-70 years
	Sedentary & Passive Activities (METS ≤ 1.5)				
Mean	1.86	2.24	2.37	2.33	2.53
95th Percentile	2.69	2.99	3.20	3.23	3.34
	Light Intensity Activities (1.5 < METs ≤ 3.0)				
Mean	4.61	5.44	5.66	5.72	6.03
95th Percentile	6.51	7.10	7.52	7.75	7.80
	Moderate Intensity Activities (3.0 < METs ≤ 6.0)				
Mean	8.50	10.20	10.84	12.52	12.94
95th Percentile	12.36	13.47	14.52	18.08	18.07

3.2.3 Short-term (1-Hour) Ventilation Rate Point Estimates

One-hour ventilation rates (Tables 3.4a-b) were calculated from U.S. EPA (2009) minute ventilation rates (e.g., minute ventilation rate x 60) to meet the SB-352 mandate for school districts to conduct a risk assessment for school sites located within 100 M of a freeway or busy roadway. These ventilation rates allow assessment of exposures to facility emissions during the course of the school day.

The age groups for children mostly deviate from those child age groupings designed for AB2588. The age groups attempt to address specific school categories (e.g., kindergarten, grade school, high school) under SB-352. However, if 1-hr ventilation rates are required that fit the AB2588 age groups, 1-hr ventilation rates can be calculated from the 8-hr breathing rates shown in Tables 3.28a-b.

Table 3.4a. One-Hour Breathing Rates for SB352 School Sites in L/kg-60 min (Males and Females Combined)

	0<2 Years	2<6 years	6<11 years	11<16 years	16-70 years
	Sedentary & Passive Activities (METS ≤ 1.5)				
Mean	25	17	10	6	4
95th Percentile	31	23	14	8	5
	Light Intensity Activities (1.5 < METS ≤ 3.0)				
Mean	61	41	23	14	10
95th Percentile	75	54	32	19	13
	Moderate Intensity Activities (3.0 < METS ≤ 6.0)				
Mean	110	76	44	28	21
95th Percentile	140	100	62	39	29
	High Intensity Activities (METS ≥ 6.0)				
Mean	-	140	82	55	38
95th Percentile	-	190	110	80	56

Table 3.4b. One-Hour Breathing Rates for SB352 School Sites in M^3/60 min (Males and Females Combined)

	0<2 Years	2<6 years	6<11 years	11<16 years	16-70 years
	Sedentary & Passive Activities (METS ≤ 1.5)				
Mean	0.23	0.27	0.29	0.33	0.32
95th Percentile	0.34	0.36	0.39	0.45	0.42
	Light Intensity Activities (1.5 < METS ≤ 3.0)				
Mean	0.58	0.68	0.68	0.76	0.75
95th Percentile	0.81	0.86	0.91	1.03	0.97
	Moderate Intensity Activities (3.0 < METS ≤ 6.0)				
Mean	1.06	1.25	1.30	1.50	1.62
95th Percentile	1.54	1.63	1.73	2.05	2.26
	High Intensity Activities (METS ≥ 6.0)				
Mean	-	2.24	2.49	2.92	3.01
95th Percentile	-	2.98	3.51	4.18	4.39

For children at school, MET activity levels equivalent to sitting at a desk during instruction and outside at play can be used as guidance for determining 1-hour breathing rates. As shown in Table 3.26 below, sitting was assigned a MET of 1.5, while play outdoors, recess and physical education had mean MET values in the range

of 4.5 to 5.0 (U.S. EPA, 2009). Thus, 1-hour breathing rates based on sedentary/passive or light activities to represent activities within the class room and moderate intensity activities to represent activities during recess and some physical education classes, are recommended.

U. S. EPA (2009) also determined ventilation rates for high intensity activities with MET values $\geq$ 6.0. The distributions generated by U.S. EPA for hrs/day spent at MET values ≥6.0 for infants (age 0<2 yrs) suggests that this level of activity is unlikely for this age group. However, there is a subgroup of children in the older child age groups that exercise at this level for at least one hr/day, although this level of activity may not happen all in one hour's time. OEHHA recommends using 1-hr high intensity ventilatory rates for after-school sports and training that require high energy output such as track, football, tennis etc. This MET category may also be used for demanding sports during physical education classes.

3.3 Estimation of Daily Breathing Rates

3.3.1 Inhalation Dose and Cancer Risk

The approach to estimating cancer risk from long-term inhalation exposure to carcinogens requires calculating a range of potential doses and multiplying by cancer potency factors in units of inverse dose to obtain a range of cancer risks. This range reflects variability in exposure rather than in the dose-response. In equation 3-1, the daily breathing rate (L/kg BW-day) is the variate which is varied for each age group.

The general algorithm for estimating dose via the inhalation route is as follows:

$$DOSEair = Cair \times [BR/BW] \times A \times EF \times (1 \times 10^{-6}) \quad \textbf{(Eq. 3-1)}$$

where:

DOSEair	= dose by inhalation (mg/kg BW-day)
Cair	= concentration in air (μg/m^3)
[BR/BW]	= daily breathing rate normalized to body weight (L/kg BW-day)
A	= inhalation absorption factor, if applicable (default = 1)
EF	= exposure frequency (days/365 days)
1×10^{-6}	= conversion factors (μg to mg, L to m^3)

The inhalation absorption factor (A) is a unitless factor that is only used if the cancer potency factor itself includes a correction for absorption across the lung. It is inappropriate to adjust a dose for absorption if the cancer potency factor is based on applied rather than absorbed dose. The exposure frequency (EF) is set at 350 days per year (i.e., per 365 days) to allow for a two week period away from home each year.(US EPA, (1991). Another factor may come into consideration in the inhalation dose equation, the fraction of time at home (FAH). See Chapter 11 for more details.
For cancer risk, the risk is calculated for each age group using the appropriate age sensitivity factors (ASFs) and the chemical-specific cancer potency factor (CPF), expressed in units of $(mg/kg\text{-}day)^{-1}$.

$$RISKair = DOSEair * CPF * ASF * ED/AT \quad \textbf{(Eq. 3-2)}$$

RISK is the predicted risk of cancer (unitless) over a lifetime as a result of the exposure, and is usually expressed as chances per million persons exposed (e.g., 5×10^{-6} would be 5 chances per million persons exposed).

The dose-response phase of a cancer risk assessment aims to characterize the relationship between an applied dose of a carcinogen and the risk of tumor appearance in a human. This is usually expressed as a cancer potency factor, or CPF, in the above equation. The CPF is the slope of the extrapolated dose-response curve and is expressed as units of inverse dose $(mg/kg\text{-}d)^{-1}$, or inverse concentration $(\mu g/m^3)^{-1}$.

Exposure duration (ED) is the number of years within the age groupings. In order to accommodate the use of the ASFs (OEHHA, 2009), the exposure for each age grouping must be separately calculated. Thus, the DOSEair and ED are different for each age grouping. The ASF, as shown below, is 10 for the third trimester and infants 0<2 years of age, is 3 for children age 2<16 years of age, and is 1 for adults 16 to 70 years of age.

ED = exposure duration (yrs):

0.25 yrs for third trimester	(ASF = 10)
2 yrs for 0<2 age group	(ASF = 10)
7 yrs for 2<9 age group	(ASF = 3)
14 yrs for 2<16 age group	(ASF = 3)
14 yrs for 16<30 age group	(ASF = 1)
54 yrs for 16-70 age group	(ASF = 1)

AT, the averaging time for lifetime cancer risks, is 70 years in all cases. To determine lifetime cancer risks, the risks are then summed across the age groups:

$$RISKair_{(lifetime)} = RISKair_{(3rdtri)} + RISKair_{(0<2\ yr)} + RISKair_{(2<16\ yr)} + RISKair_{(16\text{-}70yr)} \quad \textbf{(Eq. 3-3)}$$

As explained in Chapter 1, we also need to accommodate cancer risk estimates for the average (9 years) and high-end (30 years) length of time at a single residence, as well as the traditional 70 year lifetime cancer risk estimate. For example, assessing risk in a 9 year residential scenario assumes exposure during the most sensitive period, from the third trimester to 9 years of age and would be presented as follows:

$$RISKair_{(9\text{-}yr\ residency)} = RISKair_{(3rdtri)} + RISKair_{(0<2\ yr)} + RISKair_{(2<9\ yr)} \quad \textbf{(Eq. 3-4)}$$

For 30-year residential exposure scenario, the 2<16 and 16<30 age group RISKair would be added to the risk from exposures in the third trimester and ages 0<2yrs. For 70 year residency risk, Eq 3-3 would apply.

3.3.2 Methods for Estimating Daily Breathing Rates

Two basic techniques have been developed to indirectly estimate daily breathing rates: the time-activity-ventilation (TAV) approach and an energy expenditure derivation

method. Ideally, daily breathing rates would be directly measured. However, the equipment for direct measurement is bulky and obtrusive and thus impractical for measuring breathing rates over an entire 24-hour period, especially on children performing their typical activities. Thus, ventilation measurements are typically taken for shorter time periods under specific conditions (e.g., running or walking on a treadmill).

The TAV approach relies on estimates or measurements of ventilation rates at varying physical activity levels, and estimates of time spent each day at those activity levels. An average daily breathing rate is generated by summing the products of ventilation rate (L/min) and time spent (min/day) at each activity level.

The second approach derives breathing rates based on daily energy expenditure and was first proposed by Layton (1993). Layton reasoned that breathing rate is primarily controlled by the amount of oxygen needed to metabolically convert food into energy the body can use. Because the volume of oxygen required to produce one kcal of energy and the ratio of the volume of oxygen consumed to the volume of air inhaled per unit time are both constant values, the amount of energy a person expends is directly proportional to the volume of air the person breathes. Layton (1993) developed an equation that models this relationship and that can be used to derive breathing rates from energy expenditure data:

$$VE = H \times VQ \times EE \qquad \textbf{(Eq. 3-5)}$$

where:

VE = the volume of air breathed per day (L/day),
H = the volume of oxygen consumed to produce 1 kcal of energy (L/kcal),
VQ = the ratio of the volume of air to the volume of oxygen breathed per unit time and is referred to as the breathing equivalent (unitless)
EE = energy (kcal) expended per day

Layton calculated an H value of 0.21 L/kcal for noninfant children. Arcus-Arth and Blaisdell (2007) calculated essentially the same H value of 0.22 L/kcal from data of non -breastfed infants based on food surveys. For VQ, Layton calculated a value of 27 from adult data. Children have different respiratory minute ventilation rates, as well as other respiratory parameter values, relative to adults. Therefore, children's VQ values can be different from those of adults. Arcus-Arth and Blaisdell (2007) calculated VQ values for children from which daily breathing rates can be derived (Table 3.5).

Table 3.5. Mean VQ Values Calculated for Children

	Weighted mean VQ	Recommended VQ
Infants 0-11 mo.	nd[a]	33.5
Boys & girls 1-3 yrs	nd[a]	33.5
Boys & girls 4-8 yrs	33.5	33.5
Boys 9-18 yrs	30.6	30.6
Girls 9-18 yrs	31.5	31.5

[a] Insufficient or no data

Three variations of estimating EE have been used based on conversion of metabolic energy to derive a breathing rate: (1) from the caloric content of daily food intake, (2) as the product of basal metabolic rate (BMR) and ratios of average daily energy expenditure to BMR, and (3) as time-weighted averages of energy expenditure (expressed as multiples of BMR) across different levels of physical activity during the course of a day. Published reports applying these variations in metabolic energy conversion to arrive at breathing rates using Layton's equation are summarized below.

In addition to using energy intake data with Layton's method to derive breathing rates, an approach called the doubly labeled water (DLW) technique has also been used to derive total energy expenditure and is summarized below. The DLW data have been shown to be quite accurate, but the approach has only been applied to specific sub-populations.

3.4 Available Daily Breathing Rate Estimates

There are a number of sources of information on daily breathing rates for various age groups and other subpopulations that have been derived via the methods described above. Some sources have compiled breathing rates from other studies.

3.4.1 Traditional Breathing Rate Estimation

The book Reference Man (Snyder et al., 1975), a report by the International Commission on Radiological Protection (ICRP), presents breathing rates based on about 10 limited studies. Using an assumption of 8 hour (hr) resting activity and 16 hr light activity and the breathing rates (see Table 3.6), ICRP recommended daily breathing rates of 23 m^3/day for adult males, 21 m^3/day for adult females, and 15 m^3/day for a 10 year old child. In addition, assuming 10 hr resting and 14 hr light activity each day, ICRP recommends a daily breathing rate of 3.8 m^3/day for a 1 year old. Finally, assuming 23 hr resting and 1 hr light activity, ICRP recommends a daily breathing rate of 0.8 m^3/day for a newborn. The breathing rates estimated by the ICRP used sources that had a small sample size and were limited in scope. Table 3.6 is the minute volume data upon which the daily breathing rates were based.

Table 3.6. Minute Volumes from ICRP'S Reference Man [a]

	Resting L/min (m^3/hr)	Light Activity L/min (m^3/hr)
Adult male	7.5 (0.45)	20 (1.2)
Adult female	6.0 (0.36)	19 (1.14)
Child, 10 yr	4.8 (0.29)	13 (0.78)
Child, 1 yr	1.5 (0.09)	4.2 (0.25)
Newborn	0.5 (0.03)	1.5 (0.09)

[a] Data compiled from available studies measuring minute volume at various activities by age/sex categories

This report provided the approach used in traditional risk assessment, in that a single estimate of daily breathing was employed, often 20 m^3/day for a 70-kg person.

3.4.2 Daily Breathing Rate Estimates Based on Time-Activity-Ventilation (TAV) Data

3.4.2.1 Marty et al. (2002)

Marty et al. (2002) derived California-specific distributions of daily breathing rates using estimates and measurements of ventilation rates at varying physical activity levels, and estimates of time spent each day at those activity levels. Two activity pattern studies were conducted in which activities of a randomly sampled population of 1762 adults and 1200 children were recorded retrospectively for the previous 24 hours via telephone interview (Phillips et al., 1991; Wiley et al., 1991a; Wiley et al., 1991b; Jenkins et al., 1992). Measured breathing rates in people performing various laboratory and field protocols were conducted by Adams et al. (1993). The subjects in this study were 160 healthy individuals of both sexes, ranging in age from 6 to 77 years. An additional forty 6 to 12 year olds and twelve 3 to 5 year olds were recruited for specific protocols.

For adults, each activity was assigned to a resting, light, moderate, moderately heavy, or heavy activity category to reflect the ventilation rate that could reasonably be associated with that activity. For children there were only resting, light, moderate, and heavy activity categories. The ventilation rates were classified into similar levels (e.g., the lying down protocol was considered the resting category of ventilation rate). The measured ventilation for each individual in the lab and field protocols was divided by that person's body weight. For each individual, the time spent at each activity level was summed over the day. The mean ventilation rate for each category (resting, etc.) was then multiplied by the summed number of minutes per day in that category to derive the daily breathing rate for each category. The breathing rates were then summed over categories to give a total daily breathing rate. The moments and percentiles for the raw derived breathing rates as well as for the breathing rates fit to a gamma distribution are presented in Tables 3.7 and 3.8 for the combined group of adolescents and adults (i.e., >12 years age) and for children (<12 years age). OEHHA staff also derived distributions of breathing rates for the equivalent of a 63-kg adult and

an 18-kg child. These breathing rates form the basis of the current risk assessment guidelines (OEHHA, 2000), which this document is revising.

Table 3.7 Children's (<12 Years) Daily Breathing Rates (L/Kg-Day)

	Moments and Percentiles from Empirical Data	Moments and Percentiles, Fitted Gamma Parametric Model	Breathing Rate Equivalent for a 18 kg Child, m^3/Day (Empirical Data)
N	1200		
Mean	452	451	8.1
Std Dev	67.7	66.1	1.22
Skewness	0.957	0.9	
Kurtosis	1.19	4.32	
%TILES	**L/kg-day**		
1%	342.5	(not calculated)	6.17
5%	364.5	360.3	6.56
10%	375	374.9	6.75
25%	401.5	402.7	7.23
50%	441	440.7	7.94
75%	489.5	488.4	8.81
90%	540.5	537.9	9.73
95%	580.5	572.1	10.5
99%	663.3	(not calculated)	11.9
Sample Max	747.5		13.5

Table 3.8 Adult/Adolescent (>12 Years) Breathing Rates (L/kg-Day)

	Moments and Percentiles from Empirical Data	Moments and Percentiles, Fitted Gamma Parametric Model	Breathing Rate Equivalent for a 63 kg Adult, m^3/Day
N	1579		
Mean	232	233	14.6
Std Dev	64.6	56.0	4.07
Skewness	2.07	1.63	
Kurtosis	6.41	6.89	
%TILES	**L/kg-day**		
1%	174	(Not calculated)	11.0
5%	179	172.3	11.3
10%	181	178.0	11.4
25%	187	192.4	11.8
50%	209	218.9	13.2
75%	254	257.9	16.0
90%	307	307.8	19.3
95%	381	342.8	24.0
99%	494.0	(Not calculated)	31.1
Sample Max	693		43.7

Advantages of these rates are that the activity pattern data were from a large randomly sampled population of California adults and children, and that ventilation rates were normalized by body weight for each individual in the ventilation rate study. However, body weight information was not available for the activity pattern subjects. Measured breathing rates during specified activities were also collected from California participants with the intention that the data would be used in conjunction with the activity pattern data to derive daily breathing rates.

Limitations include the use of one-day activity pattern survey data that may tend to overestimate long-term daily breathing rates because both intraindividual variability and interindividual variability are poorly characterized. However, intraindividual variability is believed to be small relative to interindividual variability, which would make the breathing rate distributions reasonably accurate for chronic exposure assessment. Despite these limitations, the derived breathing rates were reasonably similar to those measured by the doubly-labeled water method (described in (OEHHA, 2000)).

Because the time-weighted average method involves professional judgment in assigning a breathing rate measured during a specific activity to various other types of activities, some uncertainty is introduced into the resulting daily breathing rates. Lastly, there is a paucity of breathing rate data for specific activities in children in the 3 to 6

year age range, and no data for children and infants younger than 3 years old. Thus, only a broad age range (i.e., < 12 years old) could be used for estimating daily breathing rates in children. Daily breathing rates cannot be reliably estimated from this study for children and infants over narrow age ranges, such as the critical 0<2 year age group.

3.4.2.2 Allan et al. (2008)

Allan et al. (2008) also estimated breathing rates for specified age groups by the TAV approach, but employed a greater number of time-activity data sets than that used by Marty et al. (2002). This study updated TAV inhalation rate distributions from a previous report by Allan and Richardson (1998) by incorporating supplemental minute volume and time-activity data, and by correlating minute volume with metabolic equivalents (METs) for performing the physical activities at the time of measurement. Published time-activity and minute volume data used by Marty et al. (2002) were also used by the authors to develop the distributions (Wiley et al., 1991a; Wiley et al., 1991b; Adams, 1993), but also a number of other reports primarily conducted in the USA and Canada.

Their TAV approach calculated mean expected breathing rates for five different activity levels (i.e., level 1 – resting; level 2 – very light activity; level 3 – light activity; level 4 – light to moderate activity, level 5 – moderate to heavy activity). For infants, only three levels of activity were defined (i.e., sleeping or napping, awake but not crying, and crying).

Probability density functions describing 24-hour inhalation rates were generated using Monte Carlo simulation and can be described with lognormal distributions. Table 3.9 presents the estimated breathing rates in m^3/day for males and females (combined) by age groupings commonly used in Canada for risk assessment purposes. In their report, Allan et al. (2008) also provided breathing rates for males and females separately. However, breathing rate distributions adjusted for body weight **(**m^3/day-kg) were not included in the report.

Table 3.9. Allan et al. (2008) TAV-Derived Daily Breathing Rates (m^3/Day) for Males And Females Combined

Age Category	Males and Females Combined (m^3/day)			
	Mean + SD	50%-ile[a]	90%-ile[a]	95%-ile[a]
Infants (0-6 mo)	2.18 ± 0.59	2.06	2.87	3.12
Toddlers (7 mo-4 yr)	8.31 ± 2.19	7.88	10.82	11.72
Children (5-11 yr)	14.52 ± 3.38	13.95	18.49	19.83
Teenagers (12-19 yr)	15.57 ± 4.00	14.80	20.09	21.69
Adults (20-59 yr)	16.57 ± 4.05	15.88	21.30	22.92
Seniors (60+ yr)	15.02 ± 3.94	14.35	19.72	21.36

[a] Percentiles provided courtesy of Allan (e-mail communication)

Allan et al. (2008) compared the breathing rate distribution derived by the DLW method (see below, Table 3.12) to their TAV breathing rate probability density function results and found that there appeared to be longer tails in the upper bounds for all age groups except teenagers and infants for the TAV method, suggesting the TAV distribution gives

a better representation of the more exposed members of the population such as athletes. For teenagers, the TAV and DLW distributions show considerable overlap. But for infants, lower breathing rates were observed by the TAV approach compared with the DLW approach. The authors could not explain this discrepancy. Unlike the Marty et al. (2002) study, daily breathing rates could be estimated in infants and toddlers. However, there is still a shortage of TAV data in children in the younger age groups relative to adults.

Uncertainty was reduced by grouping activities by expected METs. However, Allen et al. (2008) noted that there is still uncertainty about actual physical exertion at an activity level because of the way some source studies grouped activities (e.g., grouping walking with running). Uncertainty was also reduced by using, wherever possible, studies that documented all activities over a multi-day period rather than studies that considered only a few hours of behavior. Nevertheless, there is some uncertainty in combining data from disparate studies and in assigning ventilation rates to activities that are not described by energy expenditure levels. In particular, interpolations and extrapolations were used to fill in minute volume data gaps and may have resulted in overestimates or underestimates. For example, minute volume data for some activity levels in toddlers and children were considered insufficient to adequately characterize their minute volumes.

3.4.3 Daily Breathing Rate Estimates Based on Energy Expenditure

As discussed above, Layton (1993) developed a mathematical equation to estimate daily breathing rates based on energy expenditure. The paper also presented examples of breathing rates that had been derived using this method.

3.4.3.1 Layton (1993)

Layton took three approaches to estimating breathing rates from energy estimates. The first approach used the U.S.D.A.'s National Food Consumption Survey (1977-78) data to estimate energy (caloric) intake. The National Food Consumption Survey used a retrospective questionnaire to record three days of food consumption by individuals in households across the nation, and across all four seasons. Layton recognized that food intake is underreported for individuals 9 years of age and older in these surveys and therefore adjusted the reported caloric intake for these ages. These data are no longer the most current population based energy intake data available. Further, the breathing rates are not normalized to body weight.

The second approach to estimating breathing rates multiplied the BMR estimated for a given age-gender group by the estimated ratio of energy intake to basal metabolic rate (EFD/BMR) for that age-gender group. The BMR can be determined as a linear function of body weight, after accounting for gender and age. An activity multiplier can then be applied which is derived from previously reported ratios of daily food intake to BMR. The advantages of this approach include linking breathing rates to BMR, which is valuable since breathing rates are considered to be determined primarily by BMR.

However, the BMR for each age-gender group was calculated from equations derived from empirical but non-representative data. Further, these data were collected using techniques that may be outdated (e.g., for the 0-3 year age group, 9 of the 11 studies were conducted between 1914 and 1952). These data may no longer be representative of the current population. The EFD/BMR ratios for males and females over 18 years of age were estimated from data collected over one year in one study while those for other age groups were estimated based on the consistency of the value in calculating energy expenditures similar to other studies. Average body weights do not capture the variability of body weights in the population. Thus the BMR values may not be as accurate as current technology can provide nor are they representative of the population.

Layton's third approach to calculate daily breathing rates involves the metabolic equivalent (MET) approach, which is a multiple of the BMR and reflects the proportional increase in BMR for a specific activity. For example, the MET for standing is 1.5 (i.e., 1.5*BMR), and the MET for cycling and swimming is 5.3. Layton categorized METs into 5 levels (from light activity with a MET = 1 to very strenuous activities with a MET = 10). MET levels were then assigned to each activity in a study that had categorized activities by energy expenditure level and recorded the time study participants spent at each activity. The energy expended at each activity was converted to a breathing rate and then summed over the day to give a daily breathing rate. However, the time-activity data used in this approach were only available for ages over 18 years.

The results of Layton's approaches are presented in Table 3.10. Layton did not report statistical distributions of the breathing rates that he derived. Other limitations, for our purposes, are that the breathing rates in Table 3.6 are not representative of the current U.S. population, are not normalized to body weight, and were for broad age ranges. In addition, no distributions were reported in the paper.

Table 3.10. Layton (1993) Estimates of Breathing Rate Based on Caloric and Energy Expenditure

Method	Breathing Rate – Men m^3/day	Breathing Rate – Women m^3/day
Time-weighted average lifetime breathing rates based on food intake	14	10
Average daily breathing rates based on the ratio of daily energy intake to BMR	13-17 (over 10 years of age)	9.9-12 (over 10 years of age)
Breathing rates based on average energy expenditure	18	13

Finley et al. (1994) presented probability distributions for several exposure factors, including inhalation rates. Based on the data Layton used to derive point estimates via his third approach (i.e., with energy expenditure equivalent to a multiple of BMR), Finley

et al. (1994) expanded on Layton's results to develop a probability distribution for breathing rate for several age groups (Table 3.11).

Table 3.11. Selected Distribution Percentiles from Finley et al. (1994) for Breathing Rates by Age

Age Category (years)	Percentile (m^3/day)		
	50th	90th	95th
<3	4.7	6.2	6.7
3 -10	8.4	10.9	11.8
10 – 18	13.1	17.7	19.3
18 – 30	14.8	19.5	21.0
30 – 60	11.8	15.4	16.7
>60	11.9	15.6	16.7

Because Finley largely used the same data as Layton to develop breathing rate distributions, the same limitations apply.

3.4.3.2 Arcus-Arth and Blaisdell (2007)

Arcus-Arth and Blaisdell (2007) derived daily breathing rates for narrow age ranges of children and characterized statistical distributions for these rates. The rates were derived using the metabolic conversion method of Layton (1993) and energy intake data (calories consumed per day) from the Continuing Survey of Food Intake of Individuals (CSFII) 1994–1996, 1998 conducted by the USDA (2000). The CSFII provided the most recent population based energy data at the time. The CSFII dataset consisted of two days of recorded food intake for each individual along with self-reported body weights. The individual data allowed for the assessment of interindividual variability. Because one-day intakes may be less typical of average daily intake, the two-day intakes were averaged to obtain a better estimate of typical intake available from these limited repeated measures. The CSFII energy intakes were weighted to represent the U.S. population. The rates were intended to be more representative of the current U.S. children's population than prior rates that had been derived using older or non-representative data.

The premise for Layton's equation is that breathing rate is proportional to the oxygen required for energy expenditure. While there are no energy expenditure data that are representative of the population, there are population representative energy intake data (i.e., calories consumed per day). Energy intake data can be used in Layton's equation when energy intake equals energy expenditure. Energy intake is equal to energy expended when the individual is neither gaining nor losing body weight (i.e., all energy intake is expended). Because the percentage of daily energy intake that is needed to result in a discernible change in body weight for adults is very small, it can be assumed that for adults energy intake equals energy expended. However, in young infants, a significant portion of their daily energy intake is deposited in new tissue (e.g., adipose, bone and muscle). The deposited energy is referred to as the energy cost of deposition (ECD). Therefore, the daily energy intake needed for normal growth of infants is used

both for energy expenditure (EE) and ECD (i.e., energy intake = EE + ECD). If the breathing rate is to be estimated by the caloric intake approach for growing infants, the ECD must be subtracted from the total daily energy intake in order to determine an accurate breathing rate.

Accounting for the ECD is primarily important for newborn infants (Butte et al., 1990; Butte et al., 2000). For example, at ages 3 and 6 months the energy cost for growth constituted 22 and 6%, respectively, of total energy requirements. In older children the energy cost is only 2-3% of total energy requirements. By the age of 25 years in males and 19 years in females, the ECD has essentially decreased to zero and remains at that level throughout adulthood (Brochu et al., 2006a).

Because Layton's equation requires only energy expenditure to derive the breathing rate, a small modification to Eq. 3-5 is made when deriving the infant breathing rate using the caloric intake approach:

$$VE = H \times VQ \times (TDEI - ECD) \times 10^{-3} \qquad \text{(Eq. 3-6)}$$

where:

TDEI = Total daily energy intake (kcal/day)
ECD = Daily energy cost of deposition (kcal/day)

Arcus-Arth and Blaisdell (2007) subtracted the ECD from the TDEI to give a more accurate estimate of energy expended. The ECD for each month of age for infants up to 11 months of age was estimated from Scrimshaw et al. (1996). Although there is typically a burst of growth just prior to and during adolescence, Arcus-Arth and Blaisdell did not subtract the ECD during adolescence because investigators considered it negligible relative to total energy intake (Spady, 1981; Butte et al., 1989).

Layton (1993) reported on the bias associated with underreporting of dietary intakes by older children. He calculated a correction factor for this bias (1.2) and multiplied the daily energy intake of each child nine years of age and older by 1.2. Arcus-Arth and Blaisdell, having evaluated the literature and finding Layton's adjustment to be reasonable, likewise multiplied daily energy intake of adolescent ages by 1.2.

Arcus-Arth and Blaisdell (2007) also evaluated the numerical values used by Layton for the VQ and H conversion factors in his metabolic equation. Their estimated value for the conversion factor H was similar to that found by Layton. However, they found data in the literature indicating that other values of VQ may be more specific to children than those used by Layton (see Table 3.5). The VQ values Arcus-Arth and Blaisdell calculated were used to derive breathing rates.

Non-normalized (L/day) and normalized (L/kg-day) breathing rates shown in Tables 3.8a-e) were derived for both children and adults from the CSFII dataset using the methodology described in Arcus-Arth and Blaisdell (2007). Briefly, the CSFII used a multistage complex sampling design to select individuals to be surveyed from the population. The CSFII recommended using a Jacknife Replication (JK) statistical

method (Gossett et al., 2002; Arcus-Arth and Blaisdell, 2007), which is a nonparametric technique that is preferred to analyze data from multistage complex surveys.

For each age group, the mean, standard error of the mean, percentiles (50th, 90th, and 95th) of non-normalized and normalized breathing rates, derived as described, are presented in Tables 3.12a and 3.12b, respectively. Child breathing rates are for males and females combined, except for the 9-18 yr adolescent age group breathing rates shown at the bottom of the tables.

TABLE 3.12a. Non-Normalized Daily Breathing Rates (L/Day) for Children and Adults Using CSFII Energy Intake and Layton's Equation

Age	Sample Size Nonweighted	Mean	SEM	50%-ile	90%-ile	95%-ile	SE of 95%-ile
Age (months)	**Infancy**						
0-2	182	3630	137	3299	5444 [1]	7104 [1]	643
3-5	294	4920	135	4561	6859	7720	481
6-8	261	6089	149	5666	8383	9760	856
9-11	283	7407	203	6959	10,212	11,772	**
0-11	1020	5703	98	5323	8740	9954	553
Age (years)	**Children**						
1	934	8770	75	8297	12,192	13,788	252
2	989	9758	100	9381	13,563	14,807	348
3	1644	10,642	97	10,277	14,586	16,032	269
4	1673	11,400	90	11,046	15,525	17,569	234
5	790	12,070	133	11,557	15,723	18,257	468
6	525	12,254	183	11,953	16,342	17,973	868
7	270	12,858	206	12,514	16,957	19,057	1269
8	253	13,045	251	12,423	17,462	19,019	1075
9	271	14,925	286	14,451	19,680	22,449 [1]	1345
10	234	15,373	354	15,186	20,873	22,898 [1]	1021
11	233	15,487	319	15,074	21,035	23,914 [1]	1615
12	170	17,586	541	17,112	25,070 [1]	29,166 [1]	1613
13	194	15,873	436	14,915	22,811 [1]	26,234 [1]	1106
14	193	17,871	615	15,896	25,748 [1]	29,447 [1]	4382
15	185	18,551	553	17,913	28,110 [1]	29,928 [1]	1787
16	201	18,340	536	17,370	27,555	31,012	2065
17	159	17,984	957	15,904	31,421 [1]	36,690 [1]	**
18	135	18,591	778	17,339	28,800 [1]	35,243 [1]	4244
0<2	1954	7502	75	7193	11,502	12,860	170
2<16	7624	14,090	120	13,128	20,993	23,879	498
	Adolescent Boys						
9-18	983	19,267	278	17,959	28,776	32,821	1388
	Adolescent Girls						
9-18	992	14,268	223	13,985	21,166	23,298	607

[1] Value may be less statistically reliable than other estimates due to small cell size

** Unable to calculate

Table 3.12b. Normalized Daily Breathing Rates (L/kg-Day) for Children and Adults Using CSFII Energy Intake and Layton's Equation

Age	Sample Size Nonweighted	Mean	SEM	50%-ile	90%-ile	95%-ile	SE of 95%-ile
Age (months)	**Infancy**						
0-2	182	839	42	725	1305	1614	290
3-5	294	709	24	669	1031	1232	170
6-8	261	727	16	684	1017	1136	73
9-11	283	760	20	710	1137	1283	96
0-11	1020	751	11	694	1122	1304	36
Age (years)	3.4.3.3 **Children**						
1	934	752	7	716	1077	1210	33
2	989	698	9	670	986	1107	31
3	1644	680	6	648	966	1082	18
4	1673	645	5	614	904	1011	19
5	790	602	7	587	823	922	25
6	525	550	10	535	765	849	28
7	270	508	9	495	682	788	39
8	253	458	11	439	657	727	37
9	271	466	11	445	673	766 [1]	21
10	234	438	12	425	661	754 [1]	38
11	233	378	9	350	566	616 [1]	32
12	170	373	13	356	545 [1]	588 [1]	46
13	194	311	12	289	459 [1]	588 [1]	55
14	193	313	12	298	443 [1]	572 [1]	92
15	185	299	10	285	461 [1]	524 [1]	25
16	201	278	10	258	434	505	46
17	159	276	15	251	453 [1]	538 [1]	**
18	135	277	10	244	410 [1]	451 [1]	42
0<2	1954	752	6	706	1094	1241	24
2<16	7624	481	3	451	764	869	6
	Adolescent Boys						
9-18	983	367	5	343	567	647	14
	Adolescent Girls						
9-18	992	315	6	288	507	580	24

[1] Value may be less statistically reliable than other estimates due to small cell size

** Unable to calculate

Ideally, breathing rates and other variates used in risk assessment should be as representative as possible of the exposed population. Population representative daily energy (caloric) intake can be estimated from national food consumption surveys, such as the CSFII and the National Health and Nutrition Examination Survey (NHANES). These surveys can be analyzed to provide results that are representative of the nation

and of several subpopulations, including narrow age groups. The sample sizes are large with these surveys and thus provide relatively robust results, which is of particular concern for the tails of probability distributions.

Limitations for the CSFII energy intake-derived breathing rates include the underreporting of food intakes discussed above. Underestimation of energy intake leads to underestimation of breathing rates. Another limitation is that only two days of food intake data had been collected. Although collection of two consecutive days of food intake is an improvement over earlier collections of one day of food intake, the repeated measures in the survey were still too limited to reduce the impact of daily variations in food intake and would tend to overestimate the upper and lower percentiles. Typical intake is not captured by the caloric intake of two days, and breathing rate and dietary intake on any given day are not tightly coupled.

3.4.3.4 US EPA (2009) Metabolic Equivalent-Derived Daily Breathing Rate Estimates

Similar to one of the approaches Layton (1993) used to estimate the breathing rate, U.S. EPA employed a metabolic equivalent (METS) approach for estimating breathing rates. This method determines daily time-weighted averages of energy expenditure (expressed as multipliers of the basal metabolic rate) across different levels of physical activity. METs provide a scale for comparing the physical intensities of different activities. Recent energy expenditure data including the 1999-2002 NHANES and U.S EPA's Consolidated Human Activity Database (CHAD) were used that considers variability due to age, gender, and activities. NHANES (CDC, 2000; 2002) was used as the source of body weight data, and CHAD (U.S. EPA, 2002) was the central source of information on activity patterns and METS values for individuals. The 4-year sampling weights assigned to the individuals within NHANES 1999-2002 were used to weight each individual's data values in the calculations of these statistics.

Data were grouped into age categories and a simulated 24-hour activity pattern was generated by randomly sampling activity patterns from the set of participants with the same gender and age. Each activity was assigned a METS value based on statistical sampling of the distribution assigned by CHAD to each activity code. Using statistical software, equations for METS based on normal, lognormal, exponential, triangular and uniform distributions were generated as needed for the various activity codes. The METS values were then translated into energy expenditure (EE) by multiplying the METS by the basal metabolic rate (BMR), which was calculated as a linear function of body weight. The VO2 was calculated by multiplying EE by H, the volume of oxygen consumed per unit energy.

The inhalation rate for each activity within the 24-hour simulated activity pattern for each individual was then estimated as a function of VO2, body weight, age, and gender. Following this, the average inhalation rate was calculated for each individual for the entire 24-hour period, as well as for four separate classes of activities based on METS value (sedentary/passive [METS less than or equal to 1.5], light intensity [METS greater than 1.5 and less than or equal to 3.0], moderate intensity [METS greater than 3.0 and less than or equal to 6.0], and high intensity [METS greater than 6.0]. Data for

individuals were then used to generate summary tables with distributional data based on gender and age categories (Tables 3.13a and 3.13b). No parametric distributional assumptions were placed on the observed data distributions before these statistics were calculated.

Table 3.13a. US EPA (2009) Metabolically-Derived Daily Breathing Rate (m^3/Day in Males and Females Unadjusted For Body Weight

Age Category (years)	Means and Percentiles in m^3/day							
	Males				Females			
	Mean	50th	90th	95th	Mean	50th	90th	95th
Birth to <1	8.76	8.70	11.93	12.69	8.53	8.41	11.65	12.66
1	13.49	13.11	17.03	17.89	13.31	13.03	17.45	18.62
2	13.23	13.19	16.27	17.71	12.74	12.60	15.58	16.37
3 to <6	12.65	12.58	14.63	15.41	12.16	12.02	14.03	14.93
6 to <11	13.42	13.09	16.56	17.72	12.41	11.95	15.13	16.34
11 to <16	15.32	14.79	19.54	21.21	13.44	13.08	16.25	17.41
16 to <21	17.22	16.63	21.94	23.38	13.59	13.20	17.12	18.29
21 to <31	18.82	18.18	24.57	27.14	14.57	14.10	19.32	21.14
31 to <41	20.29	19.83	26.77	28.90	14.98	14.68	18.51	20.45
41 to <51	20.93	20.60	26.71	28.37	16.20	15.88	19.91	21.35
51 to <61	20.91	20.41	27.01	29.09	16.18	15.90	19.93	21.22
61 to <71	17.94	17.60	21.78	23.50	12.99	12.92	15.40	16.15

Table 3.13b. US EPA (2009) Metabolically-Derived Daily Breathing Rate (m^3/Kg-Day) in Males and Females Adjusted for Body Weight

Age Category (years)	Means and Percentiles in m^3/kg-day							
	Males				Females			
	Mean	50th	90th	95th	Mean	50th	90th	95th
Birth to <1	1.09	1.09	1.26	1.29	1.14	1.13	1.33	1.38
1	1.19	1.17	1.37	1.48	1.20	1.18	1.41	1.46
2	0.95	0.94	1.09	1.13	0.95	0.96	1.07	1.11
3 to <6	0.70	0.69	0.87	0.92	0.69	0.68	0.88	0.92
6 to <11	0.44	0.43	0.55	0.58	0.43	0.43	0.55	0.58
11 to <16	0.28	0.28	0.36	0.38	0.25	0.24	0.31	0.34
16 to <21	0.23	0.23	0.28	0.30	0.21	0.21	0.27	0.28
21 to <31	0.23	0.22	0.30	0.32	0.21	0.20	0.26	0.28
31 to <41	0.24	0.23	0.31	0.34	0.21	0.20	0.27	0.30
41 to <51	0.24	0.23	0.32	0.34	0.22	0.21	0.28	0.31
51 to <61	0.24	0.24	0.30	0.34	0.22	0.21	0.28	0.30
61 to <71	0.21	0.20	0.24	0.25	0.18	0.17	0.21	0.22

US EPA (2009) described the strengths and weaknesses of their approach. The strengths of this metabolically-derived method include nationally representative data sets with a large sample size, even within the age and gender categories. This approach also yields an estimate of ventilation rate that is a function of VO2 rather than

an indirect measure of oxygen consumption such as VQ as other researchers have used.

Another strength is that the breathing rates included a BMR component which had been derived from NHANES body weights and to which NHANES sampling weights were linked. The BMR component of the breathing rates was representative of the population because of the sampling weights. That is, the degree of association between body weight and breathing rate was incorporated into the distribution of breathing rate distributions.

However, the degree of association between breathing rate and other characteristics (e.g., race, geographic region) was not incorporated into the distributions (US EPA, 2009). These non-body weight characteristics can be highly associated with variability in activity patterns. Although BMR may contribute the greatest percent to the quantitative breathing rate value, the variability in breathing rates is most likely driven by differing levels of physical activity by different persons. Because the activity data was collected over a 24-hour period, day-to-day variability is not well characterized (US EPA, 2009; US EPA, 2011). The outcome is that the simulated 24-hour activity pattern assigned to an NHANES participant is likely to contain a greater variety of different types of activities than one person may typically experience in a day.

Furthermore, because the simulated activity profiles did not consider possible limits on the "maximum possible METS value" that would account for previous activities, ventilation rates may be overestimated (US EPA, 2009). This happens, in part, because the MET approach does not take into consideration correlations that may exist between body weight and activity patterns. For example, high physical activity levels can be associated with individuals of high body weight, leading to unrealistically high inhalation rates at the upper percentiles levels (US EPA 2011). The result is that the central tendency of the MET breathing rates may be fairly representative of the population, but the breathing rates may not appropriately capture the variability within the population. This limitation was probably most evident in children <3 years of age where the data used to calculate BMR values may be less representative of the current population (US EPA, 2009).

3.4.4 Daily Breathing Rate Estimates from Doubly Labeled Water Measurements

In another method used to quantify human energy expenditure, published doubly-labeled water (DLW) energy expenditure data can be used in conjunction with Layton's equation to convert metabolic energy to daily inhalation rates (Brochu et al., 2006a; 2006b; Stifelman, 2007). In the DLW method, isotopically labeled water containing $^{2}H_{2}0$ (i.e., heavy water) and $H_{2}{}^{18}0$ is given orally to the study participant. The isotopes then distribute in the body and disappear from body water pools by dilution from new unlabeled water into the body, by the excretion of the labeled isotope from the body, or by the production of CO_2. The difference in disappearance rates between the two isotopes represents CO_2 production over an optimal period of 1–3 half-lives (7 to 21 days in most human subjects) of the labeled water. CO_2 production is an indirect

measure of metabolic rate and can be converted into units of energy using knowledge of the chemical composition of the foods consumed.

A major advantage of the DLW method is that it provides an index of total energy expenditure over a period of 1 to 3 weeks, which is a more biologically meaningful period of time compared to the other methods, and can reduce the impact of daily variations in physical activity or food intake (IOM, 2005). In addition, the DLW method is non-invasive, requiring only that the subject drink the stable isotopes and provide at least three urine samples over the study period. Thus, measurements can be made in subjects leading their normal daily lives (i.e., free-living individuals). The DLW method is considered to be the most accurate method for determining the breathing rate of an individual (IOM, 2005).

A disadvantage is that the DLW method is expensive to undertake, and that essentially all the available studies investigated different age ranges but the subjects were not randomly selected to be representative of populations. However, measurements are available in a substantial number of men, women and children whose ages, body weights, heights and physical activities varied over wide ranges.

DLW measurements of total daily energy expenditures (TDEE) include basal metabolism, physical activity level, thermogenesis, and the synthetic cost of growth (Butte et al., 2000). The synthetic cost of growth is the energy that is expended to synthesize the molecules that will be stored. This is different from the energy deposited for growth (ECD), which is the energy intake that is deposited in the body for new tissue. The ECD is an important factor in newborn infants and is not accounted for in DLW measurements. Thus, the derivation of breathing rates using Layton's equation does not require an adjustment to subtract out the ECD to determine TDEE, as was necessary for deriving the breathing rates of infants by the caloric intake approach (Section 3.5.3.2).

3.4.4.1 Brochu et al. (2006a,b)

Brochu et al. (2006a) calculated daily inhalation rates for 2210 individuals aged 3 weeks to 96 years using DLW energy expenditure data mainly from the IOM (2005). The IOM database is a compilation of DLW-derived energy expenditure results and other raw data from individuals collected from numerous studies. Breathing rates were estimated for different groups of individuals including healthy normal-weight males and females with normal active lifestyles (n=1252), overweight/obese individuals with normal active lifestyles (n=679), individuals from less affluent societies (n=59), underweight adults (n=34), and individuals during various extreme physical activities (n=170). Normal weight adults age 20 yrs and above were categorized as having BMIs between 18.5 and 25 kg/m^2. Overweight/obese adults had BMIs above 25 kg/m^2. For children and teenagers aged 4 to 19 yrs, BMIs corresponding to the 85^{th} percentile or below were considered normal. The breathing rate data were presented as 5^{th}, 10^{th}, 25^{th}, 50^{th}, 75^{th}, 90^{th}, 95^{th}, and 99^{th} percentile values as well as mean and SEM values for the derived inhalation rates for narrow age groups ranging from 1 month to 96 years. A partial

listing of the breathing rate percentiles for normal weight individuals by age group are shown in Tables 3.14a and 3.14b.

Table 3.14a. Means and Percentiles of Daily Breathing Rates (in m^3/Day) for Free-Living Normal-Weight Males and Females Derived from DLW Measurements by Brochu et al. (2006a)

Age Category (years)	Means and Percentiles in m^3/day									
	Males[a]					Females[a]				
	N	Mean	50th	90th	95th	N	Mean	50th	90th	95th
0.22 to <0.5	32	3.38	3.38	4.30	4.57	53	3.26	3.26	4.11	4.36
0.5 to <1	40	4.22	4.22	5.23	5.51	63	3.96	3.96	4.88	5.14
1 to <2	35	5.12	5.12	6.25	6.56	66	4.78	4.78	6.01	6.36
2 to <5	25	7.60	7.60	9.25	9.71	36	7.06	7.06	8.54	8.97
5 to <7	96	8.64	8.64	10.21	10.66	102	8.22	8.22	9.90	10.38
7 to <11	38	10.59	10.59	13.14	13.87	161	9.84	9.84	12.00	12.61
11 to <23	30	17.23	17.23	21.93	23.26	87	13.28	13.28	16.61	17.56
23 to <30	34	17.48	17.48	21.08	22.11	68	13.67	13.67	16.59	17.42
30 to <40	41	16.88	16.88	20.09	21.00	59	13.68	13.68	15.94	16.58
40 to <65	33	16.24	16.24	19.67	20.64	58	12.31	12.31	14.96	15.71
65 to <96	50	12.96	12.96	16.13	17.03	45	9.80	9.80	12.58	13.37

[a] Percentiles based on a normal distribution assumption for all age groups

Table 3.14b. Means and Percentiles of Daily Breathing Rates (in m^3/kg-Day) for Free-Living Normal-Weight Males and Females Derived from DLW Measurements by Brochu et al. (2006a)

Age Category (years)	Mean and Percentiles in m^3/kg-day									
	Males[a]					Females[a]				
	N	Mean	50th	90th	95th	N	Mean	50th	90th	95th
0.22 to <0.5	32	0.509	0.509	0.627	0.661	53	0.504	0.504	0.623	0.657
0.5 to <1	40	0.479	0.479	0.570	0.595	63	0.463	0.463	0.545	0.568
1 to <2	35	0.480	0.480	0.556	0.578	66	0.451	0.451	0.549	0.577
2 to <5	25	0.444	0.444	0.497	0.512	36	0.441	0.441	0.532	0.559
5 to <7	96	0.415	0.415	0.475	0.492	102	0.395	0.395	0.457	0.474
7 to <11	38	0.372	0.372	0.451	0.474	161	0.352	0.352	0.431	0.453
11 to <23	30	0.300	0.300	0.360	0.377	87	0.269	0.269	0.331	0.349
23 to <30	34	0.247	0.247	0.297	0.311	68	0.233	0.233	0.287	0.302
30 to <40	41	0.237	0.237	0.281	0.293	59	0.235	0.235	0.279	0.292
40 to <65	33	0.230	0.230	0.284	0.299	58	0.211	0.211	0.257	0.270
65 to <96	50	0.188	0.188	0.228	0.239	45	0.172	0.172	0.220	0.233

[a] Percentiles based on a normal distribution assumption for all age groups

Comparing the largest subgroups (i.e., overweight/obese individuals vs. normal-weight individuals), Brochu et al. observed that overweight/obese individuals inhaled between 0.8 to 3.0 m^3 more air per day than normal-weight individuals, but their physiological daily breathing rates are 6 to 21% lower than that of their leaner counterparts when

expressed in m^3/kg-day. Also of interest is that the daily inhalation rates (in m^3/kg-day) of newborns and normal-weight infants aged 2.6 to less than 6 months are 2.1 to 5.1 times higher than those of normal-weight and overweight/obese adults aged 18 to 96 years with normal lifestyles.

Besides the lack of randomly selected individuals representative of a population for estimating energy expenditure, much of the DLW data used to derive the breathing rate percentiles relied heavily on adults with sedentary lifestyles (Black et al., 1996). Occupations of many participants included professionals, white collar workers or other sedentary occupations, and almost no participants were in manual labor occupations that are known to result in higher breathing rates. Although a small group of athletic individuals appear to be included in the DLW database by Brochu et al. (2006a), it was suggested by Black et al. (1996) that not enough participants involved in manual labor are represented in the DLW database. This may result in breathing rate percentiles that are lower than what might be obtained from a population-based study. Nevertheless, as noted above, the DLW method provides an index of total energy expenditure over a period of 1 to 3 weeks, which is a better determinant of long-term breathing rate than other methods described that rely on 1 to 2 days of energy intake or expenditure to estimate long-term breathing rates. Thus, the DLW method is considered to be the most accurate method for determining an average daily breathing rate of a free-living individual.

3.4.4.2 Stifelman (2007)

Using energy expenditure data based on extensive DLW measurements from two sources (FAO, 2004a; 2004b; IOM, 2005), Stifelman (2007) calculated inhalation rates with Layton's equation for long-term physical activity levels categorized as active to very active individuals. The breathing rate data are presented in Table 3.15 in one year age groupings for infants and children and in three age groupings for adults up to age 70.

TABLE 3.15. Equivalent Breathing Rates Based on Institute of Medicine Energy Expenditure Recommendations for Active and Very Active People

Age (Years)	Inhalation rate – males active – very active (m^3/day)	Inhalation rate – females active – very active (m^3/day)
<1	3.4	3.4
1	4.9	4.9
2	5.9	5.5
3	8.4 – 9.5	7.9 – 9.3
4	8.8 – 10.1	8.3 – 9.9
5	9.4 – 10.7	8.8 – 10.5
6	9.8 – 11.3	9.3 – 11.1
7	10.4 – 11.9	9.7 – 11.6
8	10.9 – 12.6	10.2 – 12.3
9	11.5 – 13.3	10.7 – 12.8
10	12.1 – 14.0	11.1 – 13.4
11	12.9 – 14.9	11.7 – 14.1
12	13.7 – 15.9	12.3 – 14.9
13	14.8 – 17.2	12.9 – 15.6
14	16.0 – 18.5	13.2 – 16.0
15	17.0 – 19.8	13.3 – 16.2
16	17.8 – 20.7	13.4 – 16.3
17	18.2 – 21.2	13.3 – 16.2
18	18.6 – 21.5	13.2 – 16.1
19-30	17.0 – 19.7	13.4 – 15.2
31-50	16.2 – 18.9	12.8 – 14.5
51-70	15.1 – 17.8	12.0 – 13.8

Physical activity levels (PALs) were categorized into four levels of activity by the IOM, two of which were the active and very active levels. A PAL is the ratio of total energy expended (TEE) divided by the basal metabolic rate, defined as the minimum level of energy needed to support essential physiologic functions in free-living people. Stifelman (2007) also calculated the breathing rate associated with each level, as shown in Table 3.16. It is believed unlikely that the PAL "very active" category (i.e., PAL range 1.9-2.5) would be exceeded over a duration of years. PALs exceeding the IOM and FAO ranges are generally not sustainable over long periods of time, but can be quite high for limited periods of time (Westerterp, 2001). For example, highly trained athletes during periods of high-intensity training competition, including cross-country skiers and Tour de France bicycle racers, can reach a PAL of 3.5-5.5.

The IOM and FAO PALs describe a range of 1.4-2.5 in accord with ranges of sustainable PALs described by others, including people actively engaged in non-mechanized agriculture, deployed military personnel, and long-distance runners (Stifleman, 2007; Westerterp, 2001; Westerterp, 1998; Black et a., 1996; Haggerty et al., 1994). Individuals among the general population exceeding PALs of 2-2.5 for long

periods of time are expected to experience negative energy balance (i.e., weight loss) mainly because an important limit to sustainable metabolic rate is the energy intake (Westerterp 1998; Westerterp, 2001).

TABLE 3.16. IOM Physical Activity Categories, Associated Breathing Rates and Equivalent Walking Distance

PAL Category	PAL midpoint value (range)	Breathing rate midpoint value	Equivalent walking distance (km /day)[a]
Sedentary	1.25 (1.0-1.39)	14.4 m^3/day	0
Low active	1.5 (1.4-1.59)	15.7 m^3/day	3.5
Active	1.75 (1.6-1.89)	17.3 m^3/day	11.7
Very active	2.2 (1.9-2.5)	19.4 m^3/day	26.9

[a] Equivalent walking distance in addition to energy expended during normal daily life, based on a 70 kg adult walking 5-6 km per hour. Adapted from Stifelman (2007) and Brooks et al. (2004)

Based on the DLW data, Stifelman's analysis indicates that human energy expenditure occurs within a fairly narrow range of activity levels (PAL in the range of 1.4-2.5), and that for breathing rates estimated by the DLW method, a breathing rate of 19.4 m^3/day (equivalent to a PAL of 2.2) is near the maximum energy expenditure that can be sustained for long periods of time in adults. This finding supports the idea that the traditional 20 m^3/day is an upper end breathing rate (Snyder et al. (1975).

The narrow range in breathing rates was found to be consistent with the daily energy expenditure estimated from the adult breathing rate distribution in Marty et al. (2002) where the range is slightly over 2-fold between the 5th and 95th percentile in Table 3.7. A roughly 2-fold range in between the 5th and 95th percentiles is also exhibited in the MET-derived breathing rates by US EPA (2009).

3.4.4.3 Limits of Sustainable Breathing Rates Derived from PALs

As noted above, DLW studies have shown that a PAL of approximately 2 to 2.5 in the general population of adults is the limit of sustainable energy expenditure for long periods of time (Westerterp, 2001; IOM, 2005; Stifelman, 2007). The PAL of novice athletes training for endurance runs and soldiers during field training falls within this range (Westerterp, 1998; 2001). The PAL has been found to be twice the upper limit (PALs = 3.5 to 5.5) in professional endurance athletes in the most demanding sports (cross-country skiing and cycling) during training and competition. The PALs of these professional athletes are in the right tail of the breathing rate distribution of the general population (Westerterp, 2001). However, the high PALs are not expected to be sustained at these high levels when averaged over years.

Knowing the average basal energy expenditure (BEE) for adults and the upper range of daily energy expenditure, the upper limit of long-term daily breathing rates for the general population can be estimated from Layton's equation (eq. 3.1). Marty et al. (2002) observed that the 95th percentile breathing rate should be found within this PAL range of 2 to 2.5. Thus, it might be reasonable to compare the 95th percentile adult

breathing rate calculated by other methods to the breathing rates derived from an upper limit PAL range of 2 to 2.5.

Table 3.17 show the expected breathing rates of adults in a PAL range of 2.0 to 2.5. The mean BEE in kcal/day for the adult age groups is obtained from Brooks et al. (2004). Mean weights for the adult age groups were also obtained from this reference in order to convert breathing rates in L/day to L/kg-day. The results from the DLW-derived energy expenditure data suggest that for normal weight adults (i.e., adults with BMIs within the healthy range of 18.5 to 25), the upper limit of breathing rates for males and females combined would be 16,629 to 20,787 L/day, or 256 to 320 L/kg-day.

Table 3.17. Description of the Normative Adult DLW Data from Brooks et al. (2004) for Persons with a Healthy BMI, and the Resulting Calculations of Breathing Rate Within the Sustainable PAL Range of 2.0 to 2.5

	Age years	n	Mean BEE kcal/d	TEE limits[a] kcal/d	Breathing rate L/d	Mean weight kg	Breathing rate L/kg-d
Males	19-30	48	1769	3538 - 4423	20,060 - 25,078	71.0	283 - 353
	31-50	59	1675	3350 - 4188	18,995 - 23,746	71.4	266 - 333
	51-70	24	1524	3048 - 3810	17,282 - 21,603	70.0	247 - 309
	19-70[b]	-	-	-	18,582 - 23,229	-	263 - 328
Females	19-30	82	1361	2722 - 3403	15,434 - 19,295	59.3	260 - 325
	31-50	61	1322	2644 - 3305	14,991 - 18,739	58.6	256 - 320
	51-70	71	1226	2452 - 3065	13,903 - 17,379	59.1	235 - 294
	19-70[b]	-	-	-	14,675 - 18,344	-	249 - 311
Males/ females[c]	19-70	-	-	-	16,629 - 20,787	-	256 - 320

[a] Sustainable PAL range (2.0 to 2.5) multiplied by mean BEE equals the daily total energy expenditure (TEE) that can be sustained over long periods of time.
[b] 19-70 yr breathing rates calculated as a weighted average from the three smaller age groupings
[c] Average breathing rates of males and females combined, assuming each gender represents 50% of the population.

Although the PAL limits were estimated for adults, it might also be useful to estimate high-end sustainable breathing rates for adolescents using the same assumption that a PAL of 2 to 2.5 represents the limit of sustainable energy expenditure over a long-term period. Some of the highest daily breathing rates in L/day were calculated for adolescents from the CSFII caloric intake data (Arcus-Arth and Blaisdell, 2007).

For deriving adolescent breathing rates from the mean BEE in Brooks et al. (2004) for 14-18 year olds, an upper limit of sustainable energy expenditure would be in the range of 3458-4323 kcal/d for males, and 2722-3403 kcal/d for females. Using Layton's equation to derive the breathing rates from these daily energy expenditures, sustainable upper limit breathing rates of 22,221-27,780 L/day for adolescent males, and 18,006-22,511 L/day for adolescent females were calculated. After normalizing for weight using the mean weights for the 14-18 year age groups in Brooks et al. (2004),

upper range daily breathing rates of 378-472 L/kg-day for males and 332-513 L/kg-day for females were calculated.

3.4.5 Compilations of Breathing Rate Data

In the US EPA (2011) Exposure Factors Handbook, ranges of measured breathing rate values were compiled for infants, children and adults by age and sex. Table 3.18 presents the recommended breathing rate values for males and females combined for specific age groups up to age ≥81 yrs based on the average of the inhalation rate data from four recent key studies: Brochu et al. (2006a); U.S. EPA, (2009); Arcus-Arth and Blaisdell, (2007); and Stifelman (2007). The Table represents the unweighted means and 95th percentiles for each age group from the key studies. U.S. EPA noted that there is a high degree of uncertainty associated with the upper percentiles, including the 95th percentile shown in Table 3.18, thus they should be used with caution. The upper percentiles represent unusually high inhalation rates for long-term exposures, but were included in the handbook to provide exposure assessors a sense of the possible range of inhalation rates for children.

Table 3.18. US EPA (2011) Recommended Long-Term Exposure (More than 30 Days) Breathing Rate Values for Infants and Children (Males and Females Combined) Averaged From Four Key Studies

Age Group	Mean m^3/day	Sources Used for Means	95th Percentile m^3/day	Sources Used for 95th-ile
Birth to <1 month	3.6	a	7.1	a
1 to <3 months	3.5	a,b	5.8	a,b
3 to <6 months	4.1	a,b	6.1	a,b
6 to <12 months	5.4	a,b	8.0	a,b
Birth to <1 year	5.4	a,b,c,d	9.2	a,b,c
1 to <2 years	8.0	a,b,c,d,	12.8	a,b,c
2 to <3 years	8.9	a,b,c,d	13.7	a,b,c
3 to <6 years	10.1	a,b,c,d	13.8	a,b,c
6 to <11 years	12.0	a,b,c,d	16.6	a,b,c
11 to <16 years	15.2	a,b,c,d	21.9	a,b,c
16 to <21 years	16.3	a,b,c,d	24.6	a,b,c
21 to <31 years	15.7	b,c,d	21.3	b,c
31 to <41 years	16.0	b,c,d	21.4	b,c
41 to <51 years	16.0	b,c,d	21.2	b,c
51 to <61 years	15.7	b,c,d	21.3	b,c
61 to <71 years	15.7	b,c,d	18.1	b,c
71 to <81 years	14.2	b,c	16.6	b,c
≥91 years	12.2	b,c	15.7	b,c

a Arcus-Arth and Blaisdell, 2007; b Brochu et al. 2006a;
c U.S. EPA, (2009) d Stifelman 2007

3.5 OEHHA-Derived Breathing Rate Distributions for the Required Age Groupings Using Existing Data.

The summarized published reports provide breathing rate distributions by month/year of age or in specific age groups, but seldom in age groups applicable to OEHHA's age groupings for cancer risk assessment. However, individual data were obtainable from the CSFII food intake study and the DLW database in the IOM (2005) report, from which breathing rate distributions could be derived in the specific age groups of third trimester, 0<2, 2<9, 2<16, 16<30, and 16-70 years. In addition, the U.S. EPA's breathing rate distributions based on the MET approach, shown in Tables 3.13a and 3.13b, can be merged to obtain the necessary age group breathing rates.

3.5.1 OEHHA-derived breathing rates based on CSFII energy intake data

In Tables 3.19a-e, non-normalized (L/day) and normalized (L/kg-day) breathing rates for the specific OEHHA age groups were derived for both children and adults from the CSFII dataset using the Jacknife Replication statistical method (Arcus-Arth and Blaisdell, 2007). Breathing rates for pregnant women, for determination of third trimester breathing rates, are presented in Section 3.5.4.

In addition, each age group was also fit to a lognormal distribution using Crystal Ball® (Oracle Corp., Redwood Shores, CA, 2009). Crystal Ball® was also used to determine the best parametric model fit for the distribution of breathing rates for each age group. The Anderson-Darling test was chosen over other goodness-of-fit tests available in Crystal Ball® because this test specifically gives greater weight to the tails than to the center of the distribution. OEHHA is interested in the tails since the right tail represents the high-end (e.g., 95^{th} percentile) breathing rates.

Tables 3.19a-e. Breathing Rate Distributions by Age Group (Males and Females Combined) Derived from CSFII Food Intake Data Using Jacknife Methodology and Parameter Estimates of Log-Normally and Best Fit Distributions

Table 3.19a. Breathing Rate Distributions for the 0<2 Year Age Group

	Jacknife Approach		Lognormal Parametric Model		Best Fit Parametric Model	
					Max Extreme	Lognormal
N (sample)	1954	1954	-	-	-	-
Skewness	na[a]	na	0.74	0.77	1.47	0.77
Kurtosis	na	na	3.96	4.34	7.81	4.34
%-ile or mean	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
Sample Min	43	79	-	-	-	-
Mean (SE)[b]	752 (9)	7502 (91)	752 (1)	7568 (13)	752 (1)	7568 (13)
50%-ile (SE)	706 (7)	7193 (91)	720	7282	706	7282
75%-ile (SE)	870 (11)	9128 (91)	909	9201	871	9201
90%-ile (SE)	1094 (19)	11,502 (120)	1107	11,523	1094	11,523
95%-ile (SE)	1241 (24)	12,860 (170)	1241	12,895	1241	12,895
Sample Max	2584	24,411	-	-	-	-

[a] Not applicable [b] SE = Standard error

Table 3.19b. Breathing Rate Distributions For the 2<9 Year Age Group

	Jacknife Approach		Lognormal Parametric Model		Best Fit Parametric Model	
					Log-normal	Lognormal
N (sample)	6144	6144	-	-	-	-
Skewness	na[a]	na	0.95	0.86	0.95	0.86
Kurtosis	na	na	4.63	4.96	4.63	4.96
%-ile or mean	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
Sample Min	144	2661	-	-	-	-
Mean (SE)[b]	595 (4)	11,684 (82)	595 (1)	11,680 (16)	595 (1)	11,680 (16)
50%-ile (SE)	567 (5)	11,303 (70)	567	11,303	567	11,303
75%-ile (SE)	702 (5)	13,611 (110)	702	13,606	702	13,606
90%-ile (SE)	857 (7)	16,010 (170)	857	16,012	857	16,012
95%-ile (SE)	975 (9)	17,760 (229)	975	17,758	975	17,758
Sample Max	1713	31,739	-	-	-	-

[a] Not applicable [b] SE = Standard error

Table 3.19c. Breathing Rate Distributions for the 2<16 Year Age Group

	Jacknife Approach		Lognormal Parametric Model		Best Fit Parametric Model	
					Gamma	Max Extreme
N (sample)	7624	7624	-	-	-	-
Skewness	na[a]	na	0.74	0.75	0.91	1.46
Kurtosis	na	na	3.97	4.02	4.38	7.26
%-ile or mean	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
Sample Min	57	2661	-	-	-	-
Mean (SE)[b]	481 (5)	14,090 (135)	481 (1)	14,094 (24)	481 (1)	14,095 (24)
50%-ile (SE)	450 (5)	13,128 (110)	456	13,465	451	13,131
75%-ile (SE)	603 (4)	16,644 (189)	606	17,239	603	16,655
90%-ile (SE)	764 (6)	20,993 (361)	763	21,214	763	20,993
95%-ile (SE)	869 (6)	23,879 (498)	868	23,870	868	23,886
Sample Max	1713	53,295	-	-	-	-

[a] Not applicable [b] SE = Standard error

Table 3.19d. Breathing Rate Distributions for the 16<30 Year Age Group

	Jacknife Approach		Lognormal Parametric Model		Best Fit Parametric Model	
					Max Extreme	Lognormal
N (sample)	2155	2155	-	-	-	-
Skewness	na[a]	na	0.69	1.90	1.69	1.90
Kurtosis	na	na	3.75	11.15	8.94	11.15
%-ile or mean	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
Sample Min	23	1029	-	-	-	-
Mean (SE)[b]	197 (3)	13,759 (204)	200 (<1)	13,899 (31)	200 (<1)	13,899 (31)
50%-ile (SE)	180 (3)	12,473 (125)	190	12,494	182	12,494
75%-ile (SE)	238 (4)	16,975 (245)	259	17,192	242	17,192
90%-ile (SE)	320 (4)	21,749 (305)	331	22,136	323	22,136
95%-ile (SE)	373 (11)	26,014 (634)	378	26,481	377	26,481
Sample Max	976	75,392	-	-	-	-

[a] Not applicable [b] SE = Standard error

Table 3.19e. Breathing Rate Distributions for the 16-70 Year Age Group

	Jacknife Approach		Lognormal Parametric Model		Best Fit Parametric Model	
					Max Extreme	Lognormal
N (sample)	8512	8512	-	-	-	-
Skewness	na[a]	na	0.67	2.05	1.87	2.05
Kurtosis	na	na	3.74	12.35	10.67	12.35
%-ile or mean	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
Sample Min	13	740	-	-	-	-
Mean (SE)[b]	165 (2)	12,078 (134)	165 (<1)	12,074 (26)	165 (<1)	12,074 (26)
50%-ile (SE)	152 (1)	10,951 (86)	157	10,951	152	10,951
75%-ile (SE)	200 (1)	14,687 (141)	212	14,685	200	14,685
90%-ile (SE)	257 (3)	18,838 (173)	269	18,834	257	18,834
95%-ile (SE)	307 (4)	21,812 (371)	307	21,831	307	21,831
Sample Max	975	75,392	-	-		

[a] Not applicable

[b] SE = Standard error

3.5.2 OEHHA-derived breathing rates based on the IOM DLW Database

The Institute of Medicine (IOM) 2005 dietary reference report includes an extensive database that is a compilation of DLW-derived energy expenditure results and other raw data for individuals collected from numerous studies. An advantage of this dataset over the U.S. EPA MET approach and the TAV approaches is that individual data on energy expenditure are matched with the weight and age of the individuals. The disadvantage is that the data are not necessarily representative of a random sample of a population.

When breathing rates were calculated from the energy expenditure data, it became apparent that there were some extreme individual breathing rates that did not appear physically possible. Using the results from the PAL limits (Section 3.4.4.3), breathing rates with a PAL greater than 2.5 were removed. Additionally, some breathing rates were below the expected BMR for an individual. Based on evidence that energy expenditure during sleep is 5 to 10% lower than the BMR, derived breathing rates that were 10% or more below the expected BMR were also removed (Brooks et al., 2004). However, relatively few individuals were removed due to an extreme breathing rate; <1 to 6% of the values were removed from any one age group.

Rather than assume a normal distribution for the age groupings as Brochu et al. (2006a) had done, OEHHA arranged the data to be more representative of a population by weighting the energy expenditure data by age and gender. The modeled populations were weighted towards an equal number of persons per year of age and the assumption was used that males and females in a population are at a ratio of 50:50. In addition, the IOM database separated individuals by weight, or more specifically, by body mass index

(BMI). Children 3 to 18 years of age are considered at risk of overweight when their BMI is greater than the 85th percentile, and overweight when their BMI is greater than the 95th percentile (Kuczmarski et al., 2000). Thus, the IOM (2005) placed overweight/obese children in a separate dataset. For the modeled populations, an 85:15 weighting for normal:overweight children in the 2<9 and 2<16 age groups was used. Adults (>19 years of age) were placed in the overweight/obese dataset if they had BMIs of 25 kg/m^2 and higher by the IOM. The results from USDA's 1994-96 Diet and Health Knowledge Survey (Tippett and Clevelend, 2001) found that 54.6% of the U.S. population have a BMI of 25 kg/m^2 or greater (n=5530). Thus, for the adult age groups (16<30 and 16-70 yrs), 45:55 weighting for normal:overweight adults was used to model the populations.

For infants, the source of the raw data in the IOM (2005) database was from Butte et al. (2000), a DLW study conducted at the Children's Nutrition Research Center in Houston, TX. Butte et al. (2000) monitored energy expenditure in 76 healthy infants by the DLW method up to six times during the study, at 3, 6, 9, 12, 18, and 24 months of age, generating a total of 351 measurements that fell within the OEHHA-specified 0<2 year age group. Thus, many of the infants were tested more than once during the study period. Following each administration of DLW by mouth, urine samples were collected over 10 days and analyzed for the hydrogen and oxygen isotopes to calculate energy expenditure.

The percentage of breast-fed infants at ages 3, 6, 9, 12, 18, and 24 months were 100%, 80%, 58%, 38%, 15%, and 5%, respectively in the Butte et al. (2000) study. The racial distribution by maternal lineage was 55 white, 7 African American, 11 Hispanic, and 3 Asian infants. The NCHS growth reference (Hamill et al., 1979) was used to evaluate the adequacy of growth in these infants. The growth performance of these infants was comparable with that of other breast-fed and formula-fed infant populations in whom socioeconomic and environmental constraints would not be expected to limit growth. Relative to the NCHS reference and compared with other breast-fed and formula-fed study populations, the growth of the children was considered satisfactory by the researchers.

Although the study did not choose subjects representative of any particular population, the range of activities that individuals of this age engage in is not as variable as the range of activities engaged in by older children and adults. In addition, even though many of the infants were tested more than once during the study period, repeated measures on the same individuals can reduce the amount of intraindividual variability in the distribution of measurements because a better estimate of typical energy expenditure is captured. Considering the limitations, the study results were judged by OEHHA to be similar enough to a randomly sampled population to calculate distributional statistics for breathing rate.

An additional observation from Butte et al. (2000) was that total energy expenditure measurements differed by age and by feeding group, but not by sex, when adjusted for weight. As expected, PAL increased significantly with age from 1.2 at 3 months to 1.4 at 24 months.

Breathing rates determined by the DLW method for women in their third trimester of pregnancy are presented separately in Section 3.5.4.

To obtain the daily breathing rate distributions for all age groups shown in Table 3.20a-e, OEHHA fit the data to a lognormal distribution using Crystal Ball® and sampled 250,000 times using Latin-Hypercube. The lognormal distribution is commonly used in stochastic risk assessment and has been found to be a reasonable parametric model for a variety of exposure parameters, including breathing rate. Latin-Hypercube analysis in Crystal Ball® was also used to determine the best parametric model fit for the distribution of breathing rates. The Anderson-Darling statistic was used for the goodness-of-fit test because it gives greater weight to the tails than to the center of the distribution.

Tables 3.20a-e. Breathing Rate Distributions by Age Group (Males and Females Combined) Derived from IOM (2005) DLW Database Using Parameter Estimates of Lognormal and Best Fit Distributions

Table 3.20a. 0<2 Year Age Group Breathing Rate Distribution

	Moments and Percentiles, Empirical Data		Moments and Percentiles, Lognormal Parametric Model		Moments and Percentiles, Best Fit Parametric Model	
N	281	281				
Skewness	-0.044	0.28	-0.001	0.44	-0.044	0.28
Kurtosis	2.10	2.59	3.00	3.35	2.10	2.59
	L/kg-day	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
					Beta	Beta
Sample Min	357	2228	-	-	-	-
Mean (SE)	567	5031	567	5031	567	5031
50%-ile	562	4967	567	4925	568	4943
80%-ile	657	6323	644	6232	655	6325
90%-ile	689	6889	685	6981	691	7042
95%-ile	713	7595	718	7638	714	7607
Sample Max	752	9210	-	-	-	-

Table 3.20b. 2<9 Year Age Group Breathing Rate Distribution

	Moments and Percentiles, Empirical Data		Moments and Percentiles, Lognormal Parametric Model		Moments and Percentiles, Best Fit Parametric Model	
N	810	810				
Skewness	0.0759	0.4676	0.0796	0.4763	0.0796	0.0290
Kurtosis	2.93	3.62	3.00	3.40	3.00	3.50
	L/kg-day	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
					Log-normal	Student's T
Sample Min	240	5085	-	-	-	-
Mean (SE)	482	9708	482	9708	482	9711
50%-ile	479	9637	481	9521	481	9708
80%-ile	551	11,478	555	11,650	555	11,641
90%-ile	597	12,629	595	12,880	595	12,704
95%-ile	631	13,626	628	13,962	628	13,632
Sample Max	703	21,152	-	-	-	-

Table 3.20c. 2<16 Year Age Group Breathing Rate Distribution

	Moments and Percentiles, Empirical Data		Moments and Percentiles, Lognormal Parametric Model		Moments and Percentiles, Best Fit Parametric Model	
N	1227	1237				
Skewness	0.2729	0.8705	0.4613	1.12	0.2729	1.14
Kurtosis	2.45	3.70	3.38	5.32	2.45	5.43
	L/kg-day	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
					Beta	Max Ext.
Sample Min	168	5328	-	-	-	-
Mean (SE)	423	12,695	423	12,700	423	12,695
50%-ile	411	11,829	414	12,000	416	11,988
80%-ile	529	16,184	517	15,833	527	15,788
90%-ile	580	18,944	576	18,328	583	18,303
95%-ile	623	20,630	628	20,694	626	20,716
Sample Max	737	27,803	-	-	-	-

Table 3.20d. 16<30 Year Age Group Breathing Rate Distribution

	Moments and Percentiles, Empirical Data		Moments and Percentiles, Lognormal Parametric Model		Moments and Percentiles, Best Fit Parametric Model	
N	245	245				
Skewness	0.3471	0.4786	0.4008	0.6962	0.4008	0.6962
Kurtosis	3.03	3.11	3.28	3.88	3.28	3.88
	L/kg-day	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
					Log-normal	Log-normal
Sample Min	135	7246	-	-	-	-
Mean (SE)	222	16,458	222	16,464	222	16,464
50%-ile	220	16,148	219	16,053	219	16,053
80%-ile	256	19,468	259	19,395	259	19,395
90%-ile	282	21,954	282	21,410	282	21,410
95%-ile	308	23,295	302	23,231	302	23,231
Sample Max	387	26,670	-	-	-	-

Table 3.20e. 16-70 Year Age Group Breathing Rate Distribution

	Moments and Percentiles, Empirical Data		Moments and Percentiles, Lognormal Parametric Model		Moments and Percentiles, Best Fit Parametric Model	
N	842	846				
Skewness	0.4264	0.6323	0.4506	0.7346	0.4506	0.7346
Kurtosis	3.18	3.32	3.36	3.98	3.36	3.98
	L/kg-day	**L/day**	**L/kg-day**	**L/day**	**L/kg-day**	**L/day**
					Log-normal	Log-normal
Sample Min	95	7235	-	-	-	-
Mean (SE)	206	15,713	206	15,715	206	15,715
50%-ile	204	15,313	203	15,282	203	15,282
80%-ile	241	18,773	243	18,664	243	18,664
90%-ile	268	20,612	266	20,687	266	20,687
95%-ile	286	22,889	286	22,541	286	22,541
Sample Max	387	29,136	-	-	-	-

3.5.3 OEHHA Age Group Breathing Rate Distributions Derived From U.S. EPA (2009) MET Approach

In Tables 3.21a-e, non-normalized (L/day) and normalized (L/kg-day) breathing rates for the specific OEHHA age groups were derived for both children and adults from the data included in the U.S. EPA (2009) report and presented above. Values for males and females were combined by taking weighted averages for each age range provided, assuming that the numbers of males and females in the population are equal. Ages were combined by the same means to create the age ranges of toxicological interest to the "Hot Spots" program.

The breathing rates used in preparation of the U.S. EPA report were derived by selecting an activity pattern set from a compilation of daily activity pattern sets (CHAD) and assigning them to a person in NHANES of the same sex and age group, although the age groups are fairly narrow for the very young (i.e., 3-month or 1-year intervals), the older age groups consist of broad age categories (i.e., 3 to 5 year intervals). These broad age groups include periods, for example 3 to <6 years, when activity can vary greatly by year of age. In addition, NHANES calculates a "sampling weight" for each participant, which represents the number of individuals in the population with the same set of these characteristics. When an individual in CHAD is matched to an individual in NHANES only on sex and age group, the set of characteristics that belonged to the CHAD individual are ignored, which could result in significantly different weighting. Thus the derived breathing rates cannot be considered representative of the population.

For these reasons and other limitations of the EPA data, as stated in Section 3.3.3.3, OEHHA chose to fit a selected set of parametric distributions to the percentile data given by U.S. EPA, rather than attempting to use the raw data to determine the best fit parametric model. A gamma distribution was fit to each age group using Crystal Ball®, which is usually one of the better fitting distributions for the right-skewed distributions typical of intake variability. The gamma distribution is a three parameter distribution with fewer shape constraints than two parameter distributions such as a lognormal distribution.

Table 3.21a-e. Normalized and Non-Normalized Breathing Rate Distributions by Age Group (Males and Females Combined) Derived From U.S. EPA (2009) Breathing Rates Using a Gamma Parameter Estimate Distribution

Table 3.21a. 0<2 Year Age Group Breathing Rate Distribution

	Moments and Percentiles, Gamma Parametric Model	
N	1601	1601
	L/kg-day	**L/day**
Mean	1125	10,711
50%-ile	1104	10,489
75%-ile	1199	12,301
90%-ile	1302	14,104
95%-ile	1372	15,271

Table 3.21b. 2<9 Year Age Group Breathing Rate Distribution[a]

	Moments and Percentiles, Gamma Parametric Model	
N	4396	4396
	L/kg-day	**L/day**
Mean	597	12,758
50%-ile	591	12,518
75%-ile	662	13,911
90%-ile	732	15,375
95%-ile	776	16,176

[a] Breathing rate data for this age range were actually available for 2<11 years of age

Table 3.21c. 2<16 Year Age Group Breathing Rate Distribution

	Moments and Percentiles, Gamma Parametric Model	
N	7657	7657
	L/kg-day	**L/day**
	449	13,365
50%-ile	440	13,106
75%-ile	496	14,694
90%-ile	555	16,426
95%-ile	595	17,609

Table 3.21d. 16<30 Year Age Group Breathing Rate Distribution[a]

	Moments and Percentiles, Gamma Parametric Model	
N	6111	6111
	L/kg-day	**L/day**
Mean	221	16,005
50%-ile	215	15,469
75%-ile	244	17,984
90%-ile	275	20,699
95%-ile	296	22,535

[a] Breathing rate data for this age range were actually available for 16<31 years of age

Table 3.21e. 16-70[a] Year Age Group Breathing Rate Distribution

	Moments and Percentiles, Gamma Parametric Model	
N	16,651	16,651
	L/kg-day	**L/day**
Mean	219	16,937
50%-ile	214	16,515
75%-ile	245	18,924
90%-ile	278	21,443
95%-ile	299	23,128

[a] Breathing rate data for this age range were given as 16<71 years of age

A limitation in calculating these breathing rates is that equal weighting by year of age was assumed when merging the U.S. EPA breathing rates into larger age groups used by OEHHA. However, this may not be a significant factor for the smaller age groups (i.e., 3rd trimester, 0<2, 2<9, 2<16, 16<30 yr old age groups), but could affect the breathing rate estimate for the 16-70 year olds. This is because a random sample of the population would find proportionally fewer adults in the 61 to 70 year age range, for example, compared to 21 to 30 year age range.

Another limitation is that merging the U.S. EPA age groups into the OEHHA age groupings does not yield the precise age range for 2<9 and 16 to <30 year olds. The actual age range in the US EPA data used to get the 16 to <30 year olds is 16 to <31, which we do not consider a significant deviation. However, the actual age range in the US EPA data used to get the 2 to <9 year olds is 2 to <11 years. The addition of 9 and 10 year olds would slightly reduce the normalized breathing rate in L/kg-day because younger children (i.e., 2<9 year olds) have higher normalized breathing rates than older children (i.e., 9-10 year olds). Alternatively, addition of 9 and 10 year olds to the 2<9 year age group would slightly increase the absolute breathing rate in L/day due to

higher volumes of air breathed per day by 9 and 10 year olds compared to younger children.

3.5.4 OEHHA-Derived Third Trimester Breathing Rates

For third trimester exposure, OEHHA calculated breathing rates using the assumption that the dose to the fetus during the third trimester was the same as that to the mother. Both the CSFII and DLW data sets included data from pregnant women that could be used to calculate breathing rates (Table 3.22). The DLW data included a code for trimester of pregnancy, while the CSFII data did not. Thus, breathing rates by the CSFII method was estimated using data for women in all stages of pregnancy with no means for separation by stage of pregnancy. OEHHA believes this would not underestimate the third trimester breathing rates, since the CSFII breathing rate data tend to overestimate the breathing rate in the upper (e.g., 95th percentile) and lower percentiles for the reasons cited in Section 3.4.3.2. Since breathing rate increases over the course of pregnancy, we felt that we could successfully combine these data with the DLW data and produce a reasonable set of point estimates for the third trimester.

In order to create a set of breathing rate data suitable for use in a stochastic risk assessment for third trimester pregnant women, we selected 1,000 observations from each set of data, normalized and non-normalized, using a Monte Carlo simulation in Crystal Ball®. Because the data sets from the two sources were similar in size, a relatively small set of simulated data was sufficient. We combined these data to create two sets of pooled data (see Section 3.2 above). We then fit a parametric distribution to each of the pooled samples, using Crystal Ball® and the Anderson-Darling goodness-of-fit test.

Table 3.22. Normalized and Non-Normalized Breathing Rate Distributions for Women in Their Third Trimester of Pregnancy: OEHHA-Derived Values from Doubly-Labeled Water (DLW) and Continuing Survey of Food Intake of Individuals (CSFII) Databases

	DLW L/kg BW-day	CSFII L/kg BW-day	DLW L/day	CSFII L/day
Distribution	Lognormal	Gamma	Lognormal	Gamma
Minimum	150	78	10,316	4,025
Maximum	348	491	23,932	29,041
Mean	220	232	15,610	14,830
Median	210	216	15,196	14,311
Std Dev	46	92	3,118	5,326
Skewness	1.19	0.5575	0.7744	0.4393
Kurtosis	4.04	2.57	3.57	3.02
Percentiles				
1%	150	84	10,316	4,025
5%	161	104	10,809	7,714
10%	174	127	11,846	8,201
25%	192	155	13,750	11,010
50%	210	216	15,196	14,311
75%	241	302	17,343	18,153
80%	246	323	17,832	19,114
90%	280	363	18,552	21,799
95%	322	392	22,763	24,349
99%	348	490	23,932	28,848

3.5.5 Summary of Long-Term Daily Breathing Rate Distributions

Table 3.23 presents a summary of the long-term daily mean and high end (i.e., 95th percentile) breathing rates derived by OEHHA from different sets of energy expenditure data. The breathing rate distributions for women in their third trimester of pregnancy are presented separately in Table 3.22 above. The MET- (non-normalized only), CSFII- and DLW-derived breathing rates in Table 3.22 are based on the best fit parametric models for each age group, although little variation in the breathing rate was observed between models within each breathing rate method. Also included are data from TAV studies that estimated breathing rates in age groupings reasonably similar to that used by OEHHA.

As noted in Table 3.23, some of the age groupings for the MET-derived breathing rates, and all age groups in the TAV-derived breathing rates do not precisely reflect the age ranges used in the “Hot Spots” program. This was primarily due to methodological differences in data collection which did not allow individual breathing rates matched with the age of the individual. However, the differences in the age ranges were small

enough in many cases to allow a rough comparison among the various breathing rate estimation methods, so they were included in the table.

TABLE 3.23. Summary of Breathing Rate by Study and Age Group

	0<2 yrs L/kg-day		2<9 yrs L/kg-day		2<16 yrs L/kg-day		16<30 yrs L/kg-day		16-70 yrs L/kg-day	
	mean	**95th**	**mean**	**95th**	**mean**	**95th**	**mean**	**95th**	**mean**	**95th**
MET[a]	1125	1372	597[b]	776[b]	449	595	221[c]	296[c]	219	299
CSFII[d]	752	1241	595	975	481	868	200	377	165	307
DLW[e]	567	713	482	628	423	626	222	302	206	286
TAV[f]										
Marty et al.	-	-	-	-	452[g]	580.5[g]	-	-	232[h]	381[h]
Allan et al.	-	-	-	-	-	-	-	-	201[e]	280[e]
	0<2 yrs L/day		**2<9 yrs L/day**		**2<16 yrs L/day**		**16<30 yrs L/day**		**16-70 yrs L/day**	
	mean	**95th**	**mean**	**95th**	**mean**	**95th**	**mean**	**95th**	**mean**	**95th**
MET[a]	10,711	15,271	12,758	16,176	13,365	17,609	16,005	22,535	16,937	23,128
CSFII[d]	7568	12,895	11,680	17,758	14,095	23,886	13,899	26,481	12,074	21,831
DLW[e]	5031	7595	9711	13,632	12,695	20,716	16,464	23,231	15,715	22,541
TAV[f]										
Marty et al.	-	-	-	-	8,100[g]	10,500[g]	-	-	14,600[h]	24,000[h]
Allan et al.	-	-	-	-	-	-	-	-	16,160[i]	22,480[i]

[a] U.S. EPA metabolic equivalent (MET) approach breathing rate point estimates shown were derived using the best fit parametric model from Tables 3.20a-e.
[b] All MET-derived breathing rates for the 2<9 yr age group actually represent 2<11 yr olds.
[c] All MET-derived breathing rates for the 16<30 yr age group actually represent 16<31 yr olds.
[d] CSFII food intake-derived breathing rate point estimates shown were derived using the best fit parametric model as presented in Tables 3.18a-e.
[e] Doubly-labeled water-derived (DLW) breathing rate point estimates shown were derived using the best fit parametric model as shown in Tables 3.19a-e.
[f] Time-activity-ventilation (TAV) breathing rate point estimates are from Table 3.3 (Marty et al. 2002) and Table 3.5 (Allan et al., 2008).
[g] The breathing rate point estimates from Table 3.3 actually represent an age range of about 3 to <12 yrs old. The non-normalized breathing rate point estimates in L/day is the equivalent for an 18 kg child.
[h] The breathing rate point estimates from Table 3.4 actually represent an age range of 12 to 70 years old. Non-normalized breathing rate point estimates in L/day are the equivalent for a 63 kg adult.
[i] Breathing rate point estimates were derived from Table 3.5 and represent an age range of 12 to 60+ years. The point estimates were calculated assuming equal weighting for each age group (12-19 yrs, 20-59 yrs, 60+ yrs) and combined. Breathing rates in Table 3.5 were available only in L/day, so the non-normalized point estimates were both divided by the mean body weight for the 16-70 age group (80.3 kg) to generate breathing rates in L/kg-day.

The DLW energy expenditure data likely result in daily breathing rates that are slightly lower in some cases than what would be expected in a random population sample, particularly for adults (Black et al., 1996). On the other hand, U.S. EPA (2008) observed that the upper percentile breathing rates for the MET and CSFII approaches are unusually high for long-term daily exposures. Based on the limits of sustainable daily breathing rates for adolescents and adults discussed in Section 3.4.4.3, the 95th percentile breathing rates in Table 3.22 appear to be above sustainable limits for some age groups. For example, the CSFII-generated upper percentile breathing rates are

highest in the age groups containing older adolescents. The 16<30 year age group upper percentile breathing rate from the CSFII study is 377 L/kg-d. This breathing rate is above the sustainable breathing rate (based on PAL) of 283-353 L/kg-d for males 19-30 years of age shown in Table 3.16 (but is not above the sustainable breathing rates for the subgroup of males and females 14-18 yrs of age with a breathing rate of 332-513 L/kg-d).

A limitation of the estimated PALs for daily breathing rates determined in Tables 3.15 and 3.17 is that the participants used in the study may not reflect a random sample of the population. Nevertheless, the observed PAL of novice athletes training for endurance runs and soldiers during field training falls within this range of 2.0-2.5 (Westerterp, 1998; 2001). Thus, the breathing rates based on physical activity limits should be accurate for the general population, with the exception of professional endurance athletes in the most demanding sports (cross-country skiing and cycling) during training and competition.

With the advantages and disadvantages of the breathing rate datasets described in Section 3.2, OEHHA recommends using a daily breathing rate point estimates based on a mean of the DLW and CSFII approaches. The main benefit is the use of individual data from these two datasets, including individual body weights, which can be combined into one distribution. In order to create a set of breathing rate data suitable for use in a stochastic risk assessment of long-term daily average exposures, OEHHA combined data for each age range within the two sources of breathing rate data, CSFII and DLW. We selected an equal number of observations from each source for the five age ranges, normalized and non-normalized, using a Monte Carlo simulation in Crystal Ball® to create pooled data for each group. We then fit a parametric distribution to each of the pooled samples, using Crystal Ball® and the Anderson-Darling goodness-of-fit test.

For infants 0<2 yrs of age, OEHHA used the DLW data by Butte et al. (2000) for combining with CSFII study 0<2 yr data. This longitudinal study followed a group of about 40 infants collecting urine every 3 months after DLW administration from age 3 months to two years of age. The sample size was not considered large enough to use this data exclusively for determining the 0<2 yr breathing rates, so was combined with CSFII data of infants in the same age range.

3.6 8-Hour Breathing Rates

Specialized exposure scenarios for estimating cancer risk to offsite workers, neighborhood residents, and school children may involve evaluating exposure in the 8-12 hour range. Therefore, 8-hour breathing rates were estimated for exposed individuals engaged in activities that bracket the range of breathing rates including minimal inhalation exposure such as reading a book and desk work, and high breathing rates such as farm work or yard work, that can be reasonably sustained for an 8-hour period.

As part of the development of average daily breathing rates, U.S. EPA (2009) used existing data on minute ventilation rates (in ml/min or ml/kg-min) for a range of activities and assigned MET values depending on the intensity level of activity:

- Sedentary/Passive Activities: Activities with MET values no higher than 1.5
- Light Intensity Activities: Activities with MET values exceeding 1.5 to $\leq$3.0
- Moderate Intensity Activities: Activities with MET values exceeding 3.0 to $\leq$6.0
- High Intensity Activities: Activities with MET values exceeding 6.0

An additional ventilation rate distribution was developed for sleeping/napping only, although the sedentary/passive activity category (MET values ≤1.5) also includes sleeping and napping. Table 3.23 shows selected MET values for various workplace activities and activities in the home or neighborhood that were used to calculate daily breathing rates by U.S. EPA (2009).

Table 3.23. METS Distributions for Workplace and Home Activities

Activity Description	Mean	Median	SD	Min	Max
Workplace Activities					
Administrative office work	1.7	1.7	0.3	1.4	2.7
Sales work	2.9	2.7	1.0	1.2	5.6
Professional	2.9	2.7	1.0	1.2	5.6
Precision/production/craft/repair	3.3	3.3	0.4	2.5	4.5
Technicians	3.3	3.3	0.4	2.5	4.5
Private household work	3.6	3.5	0.8	2.5	6.0
Service	5.2	5.3	1.4	1.6	8.4
Machinists	5.3	5.3	0.7	4.0	6.5
Farming activities	7.5	7.0	3.0	3.6	17.0
Work breaks	1.8	1.8	0.4	1.0	2.5
Household/Neighborhood Activities					
Sleep or nap	0.9	0.9	0.1	0.8	1.1
Watch TV	1.0	1.0	-	1.0	1.0
General reading	1.3	1.3	0.2	1.0	1.6
Eat	1.8	1.8	0.1	1.5	2.0
Do homework	1.8	1.8	-	1.8	1.8
General personal needs and care	2.0	2.0	0.6	1.0	3.0
Indoor chores	3.4	3.0	1.4	2.0	5.0
Care of plants	3.5	3.5	0.9	2.0	5.0
Clean house	4.1	3.5	1.9	2.2	5.0
Home repairs	4.7	4.5	0.7	4.0	6.0
General household chores	4.7	4.6	1.3	1.5	8.0
Outdoor chores	5.0	5.0	1.0	2.0	7.0
Walk/bike/jog (not in transit) age 20	5.8	5.5	1.8	1.8	11.3
Walk/bike/jog (not in transit) age 30	5.7	5.7	1.2	2.1	9.3
Walk/bike/jog (not in transit) age 40	4.7	4.7	1.8	2.3	7.1

MET values and hr/day spent at these various activities were used by U.S. EPA (2009) to calculate selected minute ventilation rates shown in Table 3.24a-b.

Table 3.24a. Descriptive Statistics for Minute Ventilation Rates (L/min-kg) While Performing Activities Within the Specified Activity Category (US EPA, 2009)

Age Category (years)	Males				Females			
	Mean	50th	90th	95th	Mean	50th	90th	95th
	Sedentary & Passive Activities[a] (METS ≤ 1.5)							
Birth to <1	0.40	0.39	0.47	0.50	0.40	0.40	0.48	0.52
1	0.41	0.40	0.49	0.52	0.43	0.42	0.51	0.54
2	0.34	0.34	0.41	0.45	0.36	0.35	0.42	0.44
3 to <6	0.25	0.25	0.33	0.35	0.25	0.25	0.33	0.36
6 to <11	0.16	0.16	0.21	0.22	0.16	0.16	0.21	0.23
11 to <16	0.10	0.10	0.13	0.14	0.10	0.09	0.12	0.13
16 to <21	0.08	0.08	0.09	0.10	0.07	0.07	0.10	0.10
21 to <31	0.06	0.06	0.08	0.08	0.06	0.06	0.07	0.08
31 to <41	0.07	0.07	0.08	0.09	0.06	0.06	0.08	0.08
41 to <51	0.07	0.07	0.09	0.09	0.06	0.06	0.08	0.09
51 to <61	0.07	0.07	0.09	0.09	0.07	0.07	0.08	0.09
61 to <71	0.08	0.08	0.09	0.09	0.07	0.07	0.08	0.08
	Light Intensity Activities (1.5 < METS ≤ 3.0)							
Birth to <1	0.99	0.97	1.17	1.20	0.98	0.96	1.18	1.23
1	1.02	1.01	1.22	1.30	1.05	1.04	1.25	1.27
2	0.84	0.83	1.00	1.03	0.90	0.89	1.04	1.10
3 to <6	0.63	0.63	0.79	0.87	0.62	0.60	0.78	0.83
6 to <11	0.38	0.38	0.49	0.53	0.38	0.38	0.50	0.54
11 to <16	0.25	0.24	0.31	0.33	0.23	0.22	0.28	0.31
16 to <21	0.18	0.18	0.22	0.23	0.17	0.17	0.21	0.22
21 to <31	0.16	0.15	0.19	0.21	0.15	0.15	0.18	0.19
31 to <41	0.16	0.16	0.20	0.21	0.15	0.15	0.19	0.20
41 to <51	0.17	0.16	0.20	0.21	0.16	0.16	0.20	0.22
51 to <61	0.17	0.16	0.20	0.22	0.16	0.16	0.20	0.21
61 to <71	0.16	0.16	0.19	0.20	0.15	0.14	0.17	0.18
	Moderate Intensity Activities (3.0 < METS ≤ 6.0)							
Birth to <1	1.80	1.78	2.18	2.28	1.87	1.85	2.25	2.40
1	1.88	1.82	2.33	2.53	1.90	1.87	2.24	2.37
2	1.55	1.54	1.84	2.02	1.60	1.58	1.92	2.02
3 to <6	1.17	1.12	1.56	1.68	1.14	1.11	1.45	1.56
6 to <11	0.74	0.71	0.96	1.04	0.72	0.71	0.94	1.01
11 to <16	0.49	0.47	0.64	0.68	0.44	0.43	0.55	0.61
16 to <21	0.39	0.38	0.49	0.52	0.36	0.35	0.46	0.49
21 to <31	0.36	0.34	0.47	0.51	0.33	0.32	0.42	0.45
31 to <41	0.36	0.34	0.47	0.52	0.32	0.30	0.41	0.46
41 to <51	0.37	0.35	0.47	0.52	0.33	0.32	0.44	0.49
51 to <61	0.38	0.37	0.48	0.55	0.34	0.33	0.44	0.49
61 to <71	0.34	0.34	0.40	0.42	0.29	0.28	0.35	0.37

[a] Sedentary and passive activities includes sleeping and napping

Table 3.24b. Descriptive Statistics for Minute Ventilation Rates (L/min) While Performing Activities Within the Specified Activity Category (US EPA, 2009)

Age Category (years)	Males				Females			
	Mean	50th	90th	95th	Mean	50th	90th	95th
	Sedentary & Passive Activities[a] (METS ≤ 1.5)							
Birth to <1	3.18	3.80	4.40	4.88	3.00	2.97	4.11	4.44
1	4.62	5.03	5.95	6.44	4.71	4.73	5.95	6.63
2	4.79	5.35	6.05	6.71	4.73	4.67	5.75	6.22
3 to <6	4.58	5.03	5.58	5.82	4.40	4.34	5.29	5.73
6 to <11	4.87	5.40	6.03	6.58	4.64	4.51	5.88	6.28
11 to <16	5.64	6.26	7.20	7.87	5.21	5.09	6.53	7.06
16 to <21	5.76	6.43	7.15	7.76	4.76	4.69	6.05	6.60
21 to <31	5.11	5.64	6.42	6.98	4.19	4.00	5.38	6.02
31 to <41	5.57	6.17	6.99	7.43	4.33	4.24	5.33	5.79
41 to <51	6.11	6.65	7.46	7.77	4.75	4.65	5.74	6.26
51 to <61	6.27	6.89	7.60	8.14	4.96	4.87	6.06	6.44
61 to <71	6.54	7.12	7.87	8.22	4.89	4.81	5.86	6.29
	Light Intensity Activities (1.5 < METS ≤ 3.0)							
Birth to <1	7.94	7.95	10.76	11.90	7.32	7.19	9.82	10.80
1	11.56	11.42	14.39	15.76	11.62	11.20	15.17	15.80
2	11.67	11.37	14.66	15.31	11.99	11.69	15.63	16.34
3 to <6	11.36	11.12	13.40	14.00	10.92	10.69	12.85	13.81
6 to <11	11.64	11.26	14.60	15.60	11.07	10.79	13.47	14.67
11 to <16	13.22	12.84	16.42	18.65	12.02	11.76	14.66	15.82
16 to <21	13.41	12.95	16.95	18.00	11.08	10.76	13.80	14.92
21 to <31	12.97	12.42	16.46	17.74	10.55	10.24	13.40	14.26
31 to <41	13.64	13.33	16.46	18.10	11.07	10.94	13.11	13.87
41 to <51	14.38	14.11	17.39	18.25	11.78	11.61	13.85	14.54
51 to <61	14.56	14.35	17.96	19.37	12.02	11.79	14.23	14.87
61 to <71	14.12	13.87	16.91	17.97	10.82	10.64	12.62	13.21
	Moderate Intensity Activities (3.0 < METS ≤ 6.0)							
Birth to <1	14.49	14.35	20.08	22.50	13.98	13.53	19.41	22.30
1	21.35	20.62	26.94	28.90	20.98	20.14	27.09	29.25
2	21.54	20.82	26.87	29.68	21.34	21.45	27.61	28.76
3 to <6	21.03	20.55	25.60	27.06	20.01	19.76	23.83	25.89
6 to <11	22.28	21.64	27.59	29.50	21.00	20.39	26.06	28.08
11 to <16	26.40	25.41	33.77	36.93	23.55	23.04	28.42	31.41
16 to <21	29.02	27.97	38.15	42.14	23.22	22.39	30.28	31.98
21 to <31	29.19	27.92	38.79	43.11	22.93	21.94	30.02	32.84
31 to <41	30.30	29.09	39.60	43.48	22.70	21.95	28.94	31.10
41 to <51	31.58	30.44	40.28	44.97	24.49	23.94	30.79	33.58
51 to <61	32.71	31.40	41.66	45.77	25.24	24.30	31.87	35.02
61 to <71	29.76	29.22	36.93	39.98	21.42	20.86	25.72	27.32

[a] Sedentary and passive activities includes sleeping and napping

In order to obtain minute ventilation rates that represent age ranges used in risk assessment for the "Hot Spots" program, age groups in Tables 3.25a-b were weighted equally by year of age and combined by OEHHA. The male and female data were also merged assuming 50:50 ratio in the California population. Two of the age groups combined from the U.S. EPA MET data do not exactly reflect the age ranges used by OEHHA, but they were judged reasonably close enough to use (i.e., combined MET ages 2 to <11 yrs represents OEHHA's 2<9 yr age group; combined MET ages 16 to <31 yrs represents OEHHA's 16<30 yr age group).

Table 3.25a. Minute Ventilation Rates for OEHHA Age Groups in L/kg-min (Males and Females Combined)

	0<2 years	2<9 years	2<16 years	16<30 years	16-70 years
	Sedentary & Passive Activities (METS ≤ 1.5)				
Mean	0.41	0.21	0.17	0.07	0.07
95th Percentile	0.52	0.29	0.24	0.09	0.09
	Light Intensity Activities (1.5 < METS ≤ 3.0)				
Mean	1.01	0.52	0.42	0.16	0.16
95th Percentile	1.25	0.70	0.56	0.21	0.21
	Moderate Intensity Activities (3.0 < METS ≤ 6.0)				
Mean	1.86	0.97	0.79	0.36	0.35
95th Percentile	2.40	1.33	1.09	0.49	0.48

Table 3.25b. Minute Ventilation Rates for OEHHA Age Groups in L/min (Males and Females Combined)

	0<2 years	2<9 years	2<16 years	16<30 years	16-70 years
	Sedentary & Passive Activities (METS ≤ 1.5)				
Mean	3.88	4.67	4.94	4.85	5.27
95th Percentile	5.60	6.22	6.66	6.73	6.96
	Light Intensity Activities (1.5 < METS ≤ 3.0)				
Mean	9.61	11.34	11.79	11.92	12.56
95th Percentile	13.57	14.80	15.67	16.15	16.24
	Moderate Intensity Activities (3.0 < METS ≤ 6.0)				
Mean	17.70	21.25	22.58	26.08	26.95
95th Percentile	25.74	28.07	30.25	37.67	37.65

From these tables, the 8-hour breathing rates were calculated by OEHHA based on age groupings used in the Hot Spots program and are presented in Section 3.2. Eight-hour breathing rates based on high intensity activities (MET values >6.0) were not considered here because even at the 95th percentile, U.S. EPA (2009) showed that individuals spent only about 1 hour or less per day at this intensity. For moderate intensity activities, the 95th percentile was at or near 8 hours/day for some age groups. For women in their third trimester of pregnancy, we are recommending using 8-hour breathing rates based on moderate intensity activities.

3.7 Short-term (1-Hour) Ventilation Rates

SB-352 mandates school districts to conduct a risk assessment for school sites located within 100 meters of a freeway or busy roadway, and also mandates that the AB-2588 risk assessment guidance be used in the risk assessment. Assessing cancer risks due to exposure at a school site requires less than 24 hour breathing rates. OEHHA recommends breathing rates derived from the USEPA (2009) age-specific ventilation rates for these purposes.

The U.S. EPA ventilation rates were developed for various levels of activity and can be used to estimate inhalation cancer risk from short-term maximal emissions from facilities. Breathing rates for children at school can range from sedentary in the classroom to active on the playground or sports field. OEHHA assumes that in some cases, a day care facility will be present on the school site where children may be as young as 0<2 years of age. The age ranges that U.S. EPA (2009) presents are useful for estimating the impact of early-in-life exposure for school-age children. Classroom instructors (i.e., adults) are also considered under SB-352. If the soil ingestion or dermal pathways need to be assessed, OEHHA recommends the exposure variates presented elsewhere in this document. The public health protective approach is to assume that all daily dermal and soil ingestion exposure occurs at school.

As discussed in Section 3.6 above, U.S. EPA (2009) used existing data of ventilation rates (in ml/min or ml/kg-min) from a range of activities and assigned MET values depending on the intensity level of activity. Table 3.26 shows MET values various school-related activities collected from the CHAD database (U.S. EPA, 2009).

Table 3.26. METS Distributions for School-Related Activities

Activity Description	Mean	Median	SD	Min	Max
Passive sitting	1.5	1.5	0.2	1.2	1.8
Use of computers	1.6	1.6	0.2	1.2	2.0
Do homework	1.8	1.8	-	1.8	1.8
Use library	2.3	2.3	0.4	1.5	3.0
Attending day-care	2.3	2.3	0.4	1.5	3.0
Attending K-12 schools	2.1	2.1	0.4	1.4	2.8
Play indoors	2.8	2.8	0.1	2.5	3.0
Play outdoors	4.5	4.5	0.3	4.0	5.0
Recess and physical education	5.0	5.0	1.7	2.0	8.0

For OEHHA's purposes, the minute ventilation rates of males and females from Tables 3.24a-b were combined assuming a 50:50 proportional population distribution, and some age groups were combined assuming equal number of individuals in the population per year of age (Table 3.27a-b). For the SB-352, the child age groups were 0<2 years (infants), 2<6 years (preschool, kindergarten), 6<11 years (grade school), 11<16 (junior high and high school). From these minute ventilation rates, 1-hour ventilation rates are derived and presented in Section 3.2.

Table 3.27a. Minute Ventilation Rates for SB352 School Sites in L/kg-min (Males and Females Combined)

	0<2 years	2<6 years	6<11 years	11<16 years	16-70 years
	Sedentary & Passive Activities (METS ≤ 1.5)				
Mean	0.41	0.28	0.16	0.10	0.07
95th Percentile	0.52	0.38	0.23	0.14	0.09
	Light Intensity Activities (1.5 < METS ≤ 3.0)				
Mean	1.01	0.69	0.38	0.24	0.16
95th Percentile	1.25	0.90	0.54	0.32	0.21
	Moderate Intensity Activities (3.0 < METS ≤ 6.0)				
Mean	1.86	1.26	0.73	0.47	0.35
95th Percentile	2.40	1.72	1.03	0.65	0.48
	High Intensity Activities (METS ≥ 6.0)				
Mean	-	2.27	1.37	0.92	0.64
95th Percentile	-	3.12	1.87	1.34	0.93

Table 3.25b. Minute Ventilation Rates for SB352 School Sites in L/min (Males and Females Combined)

	0<2 years	2<6 years	6<11 years	11<16 years	16-70 years
	Sedentary & Passive Activities (METS ≤ 1.5)				
Mean	3.88	4.56	4.76	5.43	5.27
95th Percentile	5.60	5.95	6.43	7.47	6.96
	Light Intensity Activities (1.5 < METS ≤ 3.0)				
Mean	9.61	11.31	11.36	12.62	12.56
95th Percentile	13.57	14.38	15.14	17.24	16.24
	Moderate Intensity Activities (3.0 < METS ≤ 6.0)				
Mean	17.70	20.75	21.64	24.98	26.95
95th Percentile	25.74	27.16	28.79	34.17	37.66
	High Intensity Activities (METS ≥ 6.0)				
Mean	-	37.34	41.51	48.69	50.10
95th Percentile	-	49.66	58.50	69.62	73.23

No high intensity minute ventilation rates are included in Tables 3.25a-b for infants age 0<2 yrs. The distributions generated by U.S. EPA (2009) for hrs/day spent at MET values ≥6.0 for infants (age 0<2 yrs) suggest that this level of activity for a 1-hr duration is unlikely for this age group.

SB-352 is also designed to protect adults working at the schools, including pregnant women. For women in their third trimester of pregnancy, OEHHA is recommending using ventilation rates of moderate intensity activities based on the same reasoning cited above in Section 3.6.

3.8 References

Adams WC. (1993). *Measurement of Breathing Rate and Volume in Routinely Performed Daily Activities. Final Report*. Human Performance Laboratory, Physical Education Department, University of California, Davis. Prepared for the California Air Resources Board, Contract No. A033-205, April 1993.

Allan M and Richardson GM (1998). Probability density distributions describing 24-hour inhalation rates for use in human health risk assessments. Hum Ecol Risk Assess 4(2): 379-408.

Allan M, Richardson GM and Jones-Otazo H (2008). Probability density functions describing 24-hour inhalation rates for use in human health risk assessments: An update and comparison. Hum Ecol Risk Assess 14: 372-91.

Arcus-Arth A and Blaisdell RJ (2007). Statistical distributions of daily breathing rates for narrow age groups of infants and children. Risk Anal 27(1): 97-110.

Black AE, Coward WA, Cole TJ and Prentice AM (1996). Human energy expenditure in affluent societies: an analysis of 574 doubly-labelled water measurements. Eur J Clin Nutr 50(2): 72-92.

Brochu P, Ducre-Robitaille J-F and Brodeur J (2006a). Physiological daily inhalation rates for free-living individuals aged 1 month to 96 years, using data from doubly-labeled water measurements: A proposal for air quality criteria, standard calculations and health risk assessment. Hum Ecol Risk Assess 12: 675-701.

Brochu P, Ducre-Robitaille J-F and Brodeur J (2006b). Physiological daily inhalation rates for free-living individuals aged 2.6 months to 96 years based on doubly-labeled water measurements: Comparison with time-activity-ventilation and metabolic energy conversion estimates. Hum Ecol Risk Assess 12: 736-61.

Brooks GA, Butte NF, Rand WM, Flatt JP and Caballero B (2004). Chronicle of the Institute of Medicine physical activity recommendation: how a physical activity recommendation came to be among dietary recommendations. Am J Clin Nutr 79(5): 921S-930S.

Butte NF, Wong WW, Ferlic L, Smith EO, Klein PD and Garza C (1990). Energy expenditure and deposition of breast-fed and formula-fed infants during early infancy. Pediatr Res 28(6): 631-40.

Butte NF, Wong WW and Garza C (1989). Energy cost of growth during infancy. Proc Nutr Soc 48(2): 303-12.

Butte NF, Wong WW, Hopkinson JM, Heinz CJ, Mehta NR and Smith EO (2000). Energy requirements derived from total energy expenditure and energy deposition during the first 2 y of life. Am J Clin Nutr 72(6): 1558-69.

CDC. (2000). Centers for Disease Control and Prevention, National Health and Nutrition Examination Survey (NHANES) 1999-2000. U.S. Department of Health and Human Services, National Center for Health Statistics (NCHS), Hyattsville, MD. Available online at: http://www.cdc.gov/nchc/about/major/nhanes/nhanes99_00.htm.

CDC. (2002). Centers for Disease Control and Prevention, National Health and Nutrition Examination Survey (NHANES) 1999-2000. U.S. Department of Health and Human Services, National Center for Health Statistics (NCHS), Hyattsville, MD. Available online at: http://www.cdc.gov/nchc/about/major/nhanes/nhanes01-02.htm.

FAO. (2004a). *Human Energy Requirements: Report of a Joint FAO/WHO/UNU Expert Consultation*. United Nations, World Health Organization, Food and Agriculture Organization: Roma, Italy.pp. 107. ISBN: 9251052123; ISSN: 1813-3932. Available online at: http://www.fao.org/documents/show_cdr.asp?url_file=/docrep/007/y5686e/y5686e00.htm.

FAO. (2004b). *Population energy requirements software (included in the Human energy requirements: Report of a Joint FAO/WHO/UNU Expert Consultation)*. Food and Agriculture Organization of the United Nations, World Health Organization: Roma, Italy. Vol. 2005. pp. 107. Available online at: http://www.fao.org/documents/show_cdr.asp?url_file=/docrep/007/y5686e/y5686e00.htm.

Finley B, Proctor D, Scott P, Harrington N, Paustenbach DJ and Price P (1994). Recommended distributions for exposure factors frequently used in health risk assessment. Risk Anal 14(4): 533-53.

Gossett JM, Simpson P, Parker JG and Simon WL (2002). How complex can complex survey analysis be with SAS? [abstract]. Proceedings, SAS Users Group International Meeting.Orlandao, April 14-17, 2002 (Cary, NC: SAS Institute Inc.) Paper 266-27.

Hamill PVV, Drizd TA, Johnson CL, Reed RB, Roche AF and Moore WM (1979). Physical growth: National Center for Health Statistics percentiles. Am J Clin Nutr 32: 607-29.

IOM (2005). Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. Institute of Medicine, Food and Nutrition Board. National Academy Press, Washington DC. Available online at: http://books.nap.edu/openbook.php?isbn=0309085373.

Jenkins PL, Phillips TJ, Mulberg EJ and Hui SP (1992). Activity patterns of Californians: Use of and proximity to indoor pollutant sources. Atmos Environ 26A: 2141-8.

Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, Flegal KM, Guo SS, Wei R, Mei Z, Curtin LR, Roche AF and Johnson CL (2000). CDC growth charts: United States. Adv Data(314): 1-27.

Layton DW (1993). Metabolically consistent breathing rates for use in dose assessments. Health Phys 64(1): 23-36.

Marty MA, Blaisdell RJ, Broadwin RL, Hill M, Shimer D and Jenkins M (2002). Distribution of daily breathing rates for use in California's Air Toxics Hot Spots Program risk assessments. Hum Ecol Risk Assess 8(7): 1723-37.

NOLS (2012). Exerpt from the National Outdoor Leadership School First Aid manual. Online at: http://www.elbrus.org/eng1/high_altitude1.htm.

OEHHA (2000). Air Toxics Hot Spots Program Risk Assessment Guidelines. Part IV. Exposure Assessment and Stochastic Technical Support Document. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA. Available online at: http://www.oehha.ca.gov.

OEHHA (2009). Technical Support Document for Cancer Potency Factors:Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environrmental Health Hazard Assessment. Online at:http://www.oehha.ca.gov/air/hot_spots/2009/TSDCancerPotency.pdf.

Phillips TJ, Jenkins PL and Mulberg EJ (1991). Children in California: Activity patterns and presence of pollutant sources. Proceedings of the 84th Annual Meeting and Exhibition of the Air and Waste Management Association, June 16-21, 1991.Vol 17: Vancouver, British Columbia, Canada, 91-172.5.

Scrimshaw NS, Waterlow JC and Schurch B (1996). Energy and Protein Requirements. Proceedings of an International Dietary and Energy Consultancy Group Workshop, 1994 Oct 31-Nov 4.London, UK: Stockton Press.

Snyder WS, Cook MJ and Nasset ES, et al. (1975). Report to the Task Group on Reference Man, International Commision on Radiological Protection, No 23. Pergamon Press, Oxnard, CA, USA, pp. 338-47.

Spady DW. (1981). *Determination and expression of energy requirements*. Joint FAO/WHO/UNU Expert Consultation on Energy and Protein Requirements: EPR Repository M2760/E; 2002. Available online at: <http://www.fao.org/docrep/meeting/004/M2760E/M2760E00.htm>.

Stifelman M (2007). Using doubly-labeled water measurements of human energy expenditure to estimate inhalation rates. Sci Total Environ 373(2-3): 585-90.

Tippett KS and Clevelend LE. (2001). *Results From USDA's 1994-6 Diet and Health Knowledge Survey*. U.S. Department of Agriculture, Nationwide Food Survey Report No. 96-4. Available online at: http://www.ars.usda.gov/SP2UserFiles/Place/12355000/pdf/dhks9496.PDF.

U.S. EPA (1991). OSWER Directive 9285.6-03 Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors". PB91-921314.

U.S. EPA. (2002). *CHAD user's guide: extracting human activity information from CHAD on the PC.* Prepared by ManTech Environmental Technologies and modified March 22 2002 by Science Applications International Corporation for the National Exposure Research Laboratory, U.S. Environmental Protection Agency. Research Triangle Park, NC. Available online at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=146583.

U.S. EPA (2008). Child-Specific Exposure Factors Handbook (Final Report). Chapter 6 - Inhalation Rates. U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-06/096F, 2008. Available online at: http://cfpub.epa.gov/ncea/CFM/recordisplay.cfm?deid=199243.

U.S. EPA. (2009). *Metabolically derived human ventilation rates: A revised approach based upon oxygen consumption rates*. U.S. Environmental Protection Agency, National Center for Environmental Assessment, Washington, DC; EPA/600/R-06/129F.

U.S. EPA. (2011). *Exposure Factors Handbook: 2011 Edition. U.S. Environmental Protection Agency*. EPA/600/R-090/052F, Washington DC.

USDA. (2000). *Continuing Survey of Food Intake by Individuals (CSFII) 1994-96, 1998. CD-ROM*. U. S. Department of Agriculture, Agricultural Research Service.

Westerterp KR (1998). Alterations in energy balance with exercise. Am J Clin Nutr 68(4): 970S-974S.

Westerterp KR (2001). Limits to sustainable human metabolic rate. J Exp Biol 204(Pt 18): 3183-7.

Wiley JA, Robinson JP, Cheng YT, Piazza T, Stork L and Pladsen K. (1991b). *Study of Children's Activity Patterns, Final Report*. Prepared for California Air Resources Board, Contract No. A733-149, September 1991.

Wiley JA, Robinson JP, Piazza T, Garrett K, Cirkensa K, Cheng YT and Martin G. (1991a). *Activity Patterns of California Residents. Final Report*. Survey Research Center, University of California, Berkeley. Prepared for California Air Resources Board, Contract No. A6-177-33, May 1991.

4 SOIL INGESTION

4.1 Introduction

There is general consensus that hand-to-mouth activity results in incidental soil ingestion, and children ingest more soil than adults. Soil ingestion rates vary depending on the age of the individual, frequency of hand-to-mouth contact, seasonal climate, amount and type of outdoor activity, the surface on which that activity occurs, and personal hygiene practices. The specified age ranges of interest in the "Hot Spots" program are ages third trimester<2, 0<2, 2<9, 2<16, 16<30 and 16-70 years.

At present, the knowledge of soil ingestion patterns within the United States is limited. A few researchers in the U.S. have attempted to quantify soil ingestion patterns in children, and have performed studies in a few locales mainly in the northern parts of the United States. The limited information shows that children may ingest fairly substantial amounts of soil on a per-kilogram-body-weight basis, and their soil ingestion pattern is important in understanding and estimating their overall exposures to environmental toxicants from contaminated soil.

The Centers for Disease Control and Prevention's Agency for Toxic Substances and Disease Registry (ATSDR) has developed definitions for soil ingestion, soil-pica, and geophagy, to distinguish aspects of soil ingestion patterns that are important from a research perspective (ATSDR, 2001):

- ***Soil ingestion*** is defined as the intentional or unintentional consumption of soil. This may result from various behaviors including, but not limited to, mouthing, contacting dirty hands, eating dropped food, or consuming soil directly.
- ***Soil-pica*** is a form of intentional ingestion of unusually high amounts of soil (i.e., on the order of 1,000 - 5,000 milligrams per day).
- ***Geophagy*** is a form of soil ingestion defined as the intentional ingestion of earths usually associated with cultural practices.

The "soil" ingested could be from outdoor soil, containerized soil for indoor plants, or a combination of both. The soil ingestion recommendations in this document represent ingestion of combined "soil" and outdoor settled dust. Outdoor settled dust is derived from particles that deposited or settled on outdoor objects and surfaces. It is not possible to differentiate between soil and outdoor settled dust. The "dust" found indoors includes soil tracked inside the building or blown indoors through opened windows and doors, particles from building materials or consumer products, human and animal dander, and particles drawn in by the house's heating and air conditioning system.

The source of "dust" in indoor environments can be quite variable. Many studies provided dust or soil ingestion estimates on pollutants that have both indoor and outdoor sources. For some pollutants it is often difficult to determine the percentage which each of these sources contributed to the amount of soil or dust ingested. Many

pollutants emitted from stationary outdoor sources can also come from important indoor sources. For example, lead from lead paint is probably the major source of lead found in indoor dust. The contribution of lead emitted from stationary sources to indoor dust is probably minor compared to that from lead paint but is difficult to pinpoint. Thus, pollutants found in indoor dust from many studies may poorly reflect the amount contributed from stationary sources.

Soil ingestion has been documented in U.S. children and adults in several studies that use a "tracer element" methodology. The tracer element methodology attempts to quantify amounts of soil ingested by analyzing samples of soil from residences, and by analyzing samples of excreta (feces, and sometimes also urine). The soil, fecal, and urine samples are analyzed for the presence and quantity of tracer elements - typically, aluminum, silicon, titanium, and yttrium, and other elements. Because these metals/metalloids are not metabolized or absorbed to an appreciable extent in the gut, their presence in feces and urine can be used to estimate the quantity of soil ingested.

However, there is some evidence that tracer elements such as aluminum and silicon can be absorbed in small amounts from the digestive tract (Davis and Mirick, 2006). None of the studies using this methodology attempt to quantify amounts excreted in perspiration, tears, glandular secretions, shed skin, hair or nails. Entry into the body via the dermal and inhalation routes was not examined. Early studies usually did not account for the contribution of tracer elements from non-soil substances (food, medications, and non-food sources such as toothpaste) that children might swallow. Some studies adjusted the soil ingestion estimates to account for the potential contribution of tracer elements found in household dust as well as soil.

The amount of soil ingested is calculated from the quantity of the tracer element measured in the feces and urine minus that present in the food and medicine consumed. This number is then divided by the soil concentration of the tracer element to yield an estimate of ingested soil. Most of the studies assumed a lag time of 24 to 28 hours between ingestion and resulting fecal and urine output. Thus, the previous day's food, medications and non-food quantity of the tracer element is subtracted from that found in the current day's feces and urine excreted. An estimation of the amount of soil ingested daily can be obtained by dividing the total amount of soil ingested by the number of days in which the feces and urine were collected.

In the *Child-Specific Exposure Factors Handbook* (U.S. EPA, 2008), U.S. EPA includes the "biokinetic model comparison" and "survey response" methods in the document to assess soil and dust ingestion in children. The biokinetic model methodology is used mainly to estimate children's exposure to lead. This model compares lead exposure and uptake to predict children's blood lead levels with biomarker blood measurements. The model predictions are made using assumptions about ingested soil and dust amounts that are based on the tracer element methodology. The survey response method uses the responses to survey questions regarding soil and dust ingestion. This method includes questions about children's soil and dust ingestion behaviors, frequency, and sometimes the quantity ingested. The respondents are the children themselves, or their caregivers.

4.2 Soil Ingestion Recommendations

4.2.1 Incidental Soil Ingestion

Before 1997, the U.S. EPA (1989, 1991) used 200 mg/day as a soil ingestion rate for children one through six years of age. In 1997, in the *Exposure Factors Handbook*, U.S. EPA recommends 100 mg/day as a mean for children under six, but indicates 200 mg could be used as a conservative estimate of the mean as it is consistent with the data.

U.S. EPA (2008) in the *Child-Specific Exposure Factors Handbook* recommended values (central tendency, mg/d) for soil, and soil and dust combined of 30, 60 (age 6 to <12 months), 50, 100 (age 1 to <6 years), and 50, 100 (age 6 to <21 years), respectively. The 90th and 95th percentile values from the key studies were used together with other data to derive a number for pica soil ingestion (above 1000 mg/d). We think that it is not appropriate to assume that the 90th and 95th percentile values in the children's studies are due to pica behavior as in any group of children there will be those that will consume more soil than the average.

OEHHA supports the U.S. EPA (2008) recommendations of 100 mg/day as the central tendency of the combined soil and dust ingestion rate for children aged 1 to <6 years. This number was rounded down from the actual number of 110 mg/d. Using 110 mg/day for soil and dust ingestion for the age group 1 to <6 years old (Table 4-13), and assuming this group has combined indoor and outdoor hand-to-mouth contacts of 14.8/hour (from Figure 4-17), soil and dust ingestion in other age groups are estimated (Table 4-18 and Table 4-19).

OEHHA calculated mean and 95th percentile soil and dust ingestions estimates (mg/kg BW-day) for the 3rd trimester < 2 by assuming that the soil and dust ingestions rate in mg/kg-day for the fetus was the same as for the mother (ages 16<30) and doing a time weighted average for the third trimester and ages 0 < 2.

OEHHA recommends the following point estimate soil and dust ingestion rates for children of various age groups and adults. Due to insufficient data, OEHHA has not developed distributions of soil ingestion data. Thus, this pathway is evaluated through the point estimate approach only.

Table 4.1 Recommended Soil Ingestion Estimates for Adults and Children (mg/kg-day)*

Age Groups (years)	Mean (mg/kg-day)	95th % (mg/kg-day)
3rd Trimester[a]	0.7	3
0<2	20	40
2<9	5	20
2<16	3	10
9<16[b]	2	7
16<30	0.7	3
16 to 70	0.6	3
PICA children[c]	200	-
PICA adult	NR	-

The mean weights for various age groups (with exceptions, see below) are from Chapter 10, Table 10.8
[a] Assumed to be the mother's soil ingestion rate (adult age 16 <30)
[b] Estimated mean body weight for this age group 55 kg
[c] Estimated mean body weight used for the PICA children 30 kg
* Soil includes outdoor settled dust
NR = No recommendation

4.3 Algorithm for Dose from Soil Ingestion

4.3.1 Inadvertent Soil Ingestion by Adults and Children

The dose from inadvertent soil ingestion by adults can be estimated using the following general equation:

$$DOSEsoil = Csoil \times GRAF \times SIR \times EF \times (1 \times 10^{-9}) \qquad \text{(Eq. 4-1)}$$

where:

DOSEsoil = dose from soil ingestion (mg/kg body weight-day)
1×10^{-9} = conversion factor (μg to mg of contaminant, and kg to mg soil)
Csoil = concentration of contaminant in soil (μg/kg soil)
GRAF = gastrointestinal relative absorption fraction, unitless
SIR = soil ingestion rate (mg/kg BW-day)
EF = exposure frequency (days/year), EF = 350 d/yr (allows 2 week vacation away from residence)

The annual average soil concentration in the Hot Spots model is determined by air dispersion models and the half-life of the chemical in the soil. The term GRAF, or gastrointestinal relative absorption factor, is defined as the fraction of contaminant absorbed by the GI tract relative to the fraction of contaminant absorbed from the matrix (feed, water, other) used in the study(ies) that is the basis of either the cancer potency factor (CPF) or the reference exposure level (REL). If no data are available to distinguish absorption in the toxicity study from absorption from the environmental

matrix in question, soil in this case, then the default assumption is that the GRAF = 1. The GRAF allows for adjustment for absorption from a soil matrix if it is known to be different from absorption across the GI tract in the study used to calculate the CPF or REL. At present that information is available only for polychlorinated dibenzo-p-dioxins and dibenzofurans. The GRAF for those compounds is 0.43. All others have a GRAF of 1.

The exposure frequency (EF) is the fraction of time spent at a residence or offsite work place, and is set at 350 days per year (i.e., per 365 days) to allow for two weeks per year away from home (US EPA,1991).

For cancer risk, the risk is calculated for each age group using the appropriate age sensitivity factors (ASFs) and the chemical-specific cancer potency factor (CPF), expressed in units of $(mg/kg\text{-}day)^{-1}$.

$$RISKsoil = DOSEsoil * CPF * ASF * ED/AT \qquad \textbf{(Eq. 4-2)}$$

Exposure duration (ED) is the number of years within the age groupings. In order to accommodate the use of the ASFs (see OEHHA, 2009), the exposure for each age grouping must be separately calculated. Thus, the DOSEsoil and ED are different for each age grouping. The ASF, as shown below, is 10 for the third trimester and infants 0<2 years of age, is 3 for children age 2<16 years of age, and is 1 for adults 16 to 70 years of age.

ED = exposure duration (yrs):

0.25 yrs for third trimester	(ASF = 10)
2 yrs for 0<2 age group	(ASF = 10)
7 yrs for 2<9 age group	(ASF = 3)
14 yrs for 2<16 age group	(ASF = 3)
14 yrs for 16<30 age group	(ASF = 1)
54 yrs for 16-70 age group	(ASF = 1)

AT, the averaging time for lifetime cancer risks, is 70 years in all cases. To determine lifetime cancer risks, the risks are then summed across the age groups:

$$RISKsoil_{(lifetime)} = RISKsoil_{(3rdtri)} + RISKsoil_{(0<2\ yr)} + RISKsoil_{(2<16\ yr)} + RISKsoil_{(16\text{-}70yr)} \qquad \textbf{(Eq. 4-3)}$$

As explained in Chapter 1, we also need to accommodate cancer risk estimates for the average (9 years) and high-end (30 years) length of time at a single residence, as well as the traditional 70 year lifetime cancer risk estimate. For example, assessing risk in a 9 year residential exposure scenario assumes exposure during the most sensitive period, from the third trimester to 9 years of age and would be presented as such:

$$RISKsoil_{(9\text{-}yr\ residency)} = RISKsoil_{(3rdtri)} + RISKsoil_{(0<2\ yr)} + RISKsoil_{(2<9\ yr)} \qquad \textbf{(Eq. 4-4)}$$

For 30-year residential exposure scenario, the 2<16 and 16<30 age group RISKsoil would be added to the risks for third trimester and age 0<2. For 70 year residential risk, Eq 4-3 would apply.

As described earlier, children have been divided into the following age groups with respect to soil ingestion rate: 0 to <2 years, 2 to <9 years, and 2 to <16 years of age. In addition, soil ingestion estimates are calculated for the adult age groups, 16 to < 30 years, and 16 to 70 years of age. In Section 4.7, OEHHA recommends soil ingestion rates for the 9, 30 and 70 year exposure duration scenarios.

The exposure duration scenarios evaluate the first 9, 30 and 70 years of an individual's life. The evaluation of the 9, 30 and 70 year exposure durations represent central tendency, ≈90^{th}- 95^{th} and lifetime of residency time, respectively. The evaluation of the 0 to <2 years, 2 to <9 years, 9 < 16 years, 16 to < 30 years, and 30 to 70 years age groupings are needed in order to properly estimate cancer risk for the age ranges as specified in *The Technical Support Document for Cancer Potency Factors: Methodologies for Derivation, Listing of Available Values, and Adjustments to Allow for Early Life Stage Exposures* (OEHHA, 2009).

For children, OEHHA is recommending that 9.7, 21.9, and 37.0 kg be used for the body weight for the 0 to <2, 2 to <9 and 2 to <16 year-old groups, respectively, for determination of dose from soil ingestion (Chapter 10). For the 16 to <30 and 16 to 70 year exposure duration scenarios, OEHHA recommends that 75.9 and 80.0 kg body weight, respectively, be used for the body weight term (Chapter 10). These body weights have been incorporated into the recommended soil consumption rates (mg/kg body weight-day). Care should be taken in using the appropriate ED and EF values for each sub-age grouping. Pica children are analyzed separately as described in Section 4.6.

4.3.2 Inadvertent Soil Ingestion by Offsite Workers

The impact zone of a facility may include offsite workplaces. Risk estimates for those offsite workers include exposure from incidental soil ingestion for multi-pathway chemicals. Equation 4-3 can be used, but the exposure is adjusted for the time at work by multiplying by 5/7 days, and 46/70 years (a total adjustment of 0.15). This adjustment is meant to account for soil ingestion occurring while at work. The assumption inherent in the exposure adjustment is that one third of the daily soil ingestion occurs at work. For those who work outdoors this assumption may underestimate exposure, and could be an overestimation for those who work mainly indoors.

4.4 Soil Intake - Key Children Studies

4.4.1 Davis and Co-workers Studies

4.4.1.1 Davis et al. (1990)

In this study, 104 toilet-trained children between the ages of 2 and 7 years were randomly recruited from a three-city area in southeastern Washington State. The study was conducted over a seven day period, primarily during the summer. A mass-balance/tracer technique was used to estimate soil ingestion. Daily soil ingestion was evaluated by analyzing soil and house dust, feces, urine, and duplicate food samples for aluminum, silicon, and titanium. In addition, information on dietary habits and demographics was collected in an attempt to identify behavioral and demographic characteristics that influence soil intake rates among children. The soil intake rates were corrected for the amount of tracer in vitamins and medications.

Soil ingestion rates were highly variable, especially those based on titanium. Mean daily soil ingestion estimates were 39 mg/day for aluminum, 82 mg/day for silicon and 246 mg/day for titanium (Table 4-2). Median values were 25 mg/day for aluminum, 59 mg/day for silicon, and 81 mg/day for titanium. The differences in concentrations of the tracer elements in house dust and yard soil were adjusted to estimate soil ingestion rates.

Table 4.2 Soil Ingestion Values From Davis et al. (1990)

Tracer Element[a]	Mean (mg/d)	Median (mg/d)	Standard Error of the Mean(mg/d)	Range(mg/d)[b]
Aluminum	38.9	25.3	14.4	279.0 to 904.5
Silicon	82.4	59.4	12.2	-404.0 to 534.6
Titanium	245.5	81.3	119.7	-5,820.8 to 6,182.2

a Excludes three children who did not provide any samples (n=101).
b Negative values occurred as a result of correction for non-soil sources of the tracer elements.

The adjusted mean soil/dust intake rates were 65 mg/day for aluminum, 160 mg/day for silicon, and 268 mg/day for titanium. Adjusted median soil/dust intake rates were: 52 mg/day for aluminum, 112 mg/day for silicon, and 117 mg/day for titanium.

The soil ingestion range includes negative numbers, which is indicative of a basic difficulty in estimating soil ingestion rates using the mass balance approach. If fecal output does not correspond to the food/medicines sampled due to factors such as the variation in transit time in the gut, then the calculated soil ingestion rate will be inaccurate. Overcorrecting for the presence of tracer elements in foods and medicines can bias the soil ingestion estimates downward, producing negative soil ingestion estimates which are obviously impossible. Likewise, if the food that was digested to produce the fecal sample contained more tracer elements than the food that was sampled, the soil ingestion rate can be biased in the positive.

In addition, the following demographic characteristics were found to be associated with high soil intake rates: male sex, racial groups other than white, low income, operator/laborer as the principal occupation of the parent, and city of residence. However, none of these factors were predictive of soil intake rates when tested using multiple linear regression.

Although a relatively large sample population was surveyed, these children were all from a single area of the U.S. and may not be representative of the U.S. population as a whole. The study was conducted over a one-week period during the summer and may not be representative of long term (i.e., annual) or seasonal patterns of soil intake.

4.4.1.2 Davis and Mirick, 2006

The study used a subset of the 104 families who participated in the soil ingestion study by Davis *et al.* (1990). The data for this study were collected one year prior to the Davis *et al.* (1990) study. Nineteen families were selected in this study. Each family consisted of one child participant between the age of 3 and 7, and one female and one male parent or guardian living in the same house. Samples were collected for 11 consecutive days of all food items consumed, all feces excreted, twice-daily urine, and soil/house dust. Tracer elements for this study included aluminum, silicon and titanium. In addition, parents completed a daily diary of the activities for 4 consecutive days for themselves and the participant child during the study period.

For children, the mean and median estimates for all three tracers ranged from 36.7 to 206.9 mg/day and 26.4 to 46.7 mg/day, respectively, and fall within the range of those reported by Davis *et al.* (1990). Adult soil ingestion estimates ranged from 23.2 to 624.9 mg/day for mean values and from 0 to 259.5 mg/day for median values, and were more variable than for the children in the study regardless of the tracer element used. The authors believed that this higher variability in adult soil ingestion rates may be attributed to occupational exposure in some, but not all, of the adults. Similar to the Davis *et al.* (1990) study, the soil ingestion estimates were the highest for titanium.

Various behaviors were found to be associated with increased soil ingestion in this study such as reported eating of dirt (for children), occupational contact with soil (for adults), and hand washing before meals (for both children and adults). Within the same family, a child's soil ingestion was not found to be associated with the parent's soil ingestion, nor did the mother and father's soil ingestion appear to be correlated. Although toothpaste is a known source of titanium, the titanium content of the toothpaste used by study participants was not determined.

An advantage of this study is that it examines soil ingestion among children and adults in the same family. However, the sample population was small and the families were a subset of those in a previous study, chosen for their high compliance to the study protocol. Thus, the uncertainties from the previous study still exist.

Table 4.3 Soil Ingestion Values From Davis and Mirick (2006)

Participant	Tracer Element	Estimated Soil Ingestion (mg/day)[a]			
		Mean	Median	Standard Deviation	Maximum
Child[b]	Aluminum	36.7	33.3	35.4	107.9
	Silicon	38.1	26.4	31.4	95.0
	Titanium	206.9	46.7	277.5	808.3
Mother[c]	Aluminum	92.1	0	218.3	813.6
	Silicon	23.2	5.2	37.0	138.1
	Titanium	359.0	259.5	421.5	1394.3
Father[d]	Aluminum	68.4	23.2	129.9	537.4
	Silicon	26.1	0.2	49.0	196.8
	Titanium	624.9	198.7	835.0	2899.1

[a] For some study participants, estimated soil ingestion resulted in a negative value. These estimates have been set to 0 mg/day for tabulation and analysis.
[b] Results based on 12 children with complete food, excreta, and soil data.
[c] Results based on 16 mothers with complete food, excreta, and soil data.
[d] Results based on 17 fathers with complete food, excreta, and soil data.

4.4.2 Binder and Co-workers Study

4.4.2.1 Binder et al. (1986)

Binder *et al.* (1986) used a tracer technique modified from a method previously used to measure soil ingestion among grazing animals to study the ingestion of soil among children. The children were studied during the summer of 1984 as part of a larger study of residents living near a lead smelter in East Helena, Montana.

Binder *et al.* (1986) measured tracer elements in feces to estimate soil ingestion by young children 1 to 3 years of age who wore diapers. Soiled diapers collected over a three day period from 65 children (42 males and 23 females), and composite samples of soil obtained from 59 of these children's yards were analyzed for aluminum, silicon, and titanium. It was assumed that the soil ingested by these children originated largely from their own yards. The soil tracer elements were assumed to be minimally absorbed in the GI tract and minimally present in the children's diet. Soil ingestion by each child was estimated based on an assumed fecal dry weight of 15 g/day. Tracer elements were assumed to be neither lost nor introduced during sampling.

Daily soil ingestion rates based on aluminum, silicon and titanium are presented in Table 4.4. The minimum soil ingestion presented in the table is based on the lowest of three estimates of soil ingestion in each subject. The minimum is presented because of the failure to account for the presence of the three tracers in ingested foods, medicines, and other sources such as toothpaste. Estimates from aluminum and silicon were comparable. However, much higher soil ingestion estimates were obtained using titanium as a tracer suggesting that there may be an unrecognized source of titanium

that the children were ingesting or the tracer element was introduced during the laboratory processing of stool samples.

Table 4.4 Soil Ingestion Rates (mg/day) From Binder et al. (1986)

Tracer:	Aluminum	Silicon	Titanium
Mean	181	184	1834
Standard deviation	203	175	3091
Range	25-1324	31-799	4-17,076
Median	121	136	618
95th percentile	584	578	9590
Geometric mean	128	130	401

The advantages of this study are that a relatively large number of children were studied and tracer elements were used to estimate soil ingestion. However, there were several methodological difficulties with the protocol pointed out by the investigators. The tracers ingested in foods and medicines were not accounted for which leads to overestimation of soil ingestion rates. Rather than using measured fecal weights, the investigators assumed a dry fecal weight of 15 g/day for each child. This may lead to either over- or underestimation of soil ingestion rates. Measuring fecal weights was difficult because the entire diaper (including urine) was collected, and as much stool as possible recovered from the diaper.

This was a short-term study and, as with all the studies on soil ingestion rates, the data may not be entirely representative of longer-term soil ingestion rates. Finally, the children may not be a representative sample of the U.S. population.

4.4.3.1 Amherst, Massachusetts Studies

4.4.3.1.1 Calabrese et al. (1989)

Sixty-four children between one and four years old in the Amherst, Massachusetts area were studied. Soil ingestion rate was based on measurements of eight tracer elements: aluminum, barium, manganese, silicon, titanium, vanadium, yttrium, and zirconium, and a method similar to Binder *et al.* (1986) but including a mass balance approach was used. Duplicate meal samples, including vitamins and medicines, were collected for all children from Monday through Wednesday of two consecutive weeks, while fecal and urine samples were collected over four 24-hour periods from noon Monday through noon Friday in the corresponding weeks.

Soil and dust samples were collected from each child's home and play areas. Children were given toothpaste, diaper rash ointment and other hygiene products that contained trace to no levels of the tracer elements. Blanks of diaper and commode specimens using distilled water were collected to control for introduced tracer. Waste samples from a single 24-hour period were pooled as were soil samples which represented composite samples from the three areas in which the child played the most.

In addition, these investigators also provided a validation study in six adult volunteers, age 25-41, for three consecutive days (Monday to Wednesday, breakfast and dinner) for three weeks. The volunteers ingested empty gelatin capsules in week one, gel capsules containing 50 mg sterilized soil in week two, and gel capsules containing 250 mg soil in week three. Duplicate food samples were collected as in the children's study and total excretion was collected Monday through Friday for the three study weeks. Soil was determined to be non-contaminated in terms of priority pollutants and contained enough of each tracer element to be detectable in the excreta.

The adult validation study indicated that study methodology could adequately detect soil ingestion at rates expected by children. The ingestion of soil in the second week was accompanied by a marked increase in fecal excretion of tracer that could not be accounted for by variability of tracer in food. Recovery data from the adult study indicated that aluminum, silicon, yttrium, and zirconium had the best recoveries (closest to 100%) while barium and manganese grossly exceeded 100% recovery. Both these elements were deemed unreliable due to their relatively higher concentrations in food relative to soil. Zirconium as a tracer was highly variable and titanium was not reliable in the adult studies. The investigators conclude that aluminum, silicon, and yttrium are the most reliable tracers for soil ingestion. Also see description of Calabrese *et al.* (1990).

The results of the soil ingestion calculations for children based on excretory tracer levels minus food tracer levels (Table 4.5) indicate a median value between 9 mg/day for yttrium and 96 mg/day for vanadium. There was a large degree of interindividual variation, with one or two extreme outliers. The mean estimates were considerably higher than the median in most cases.

Table 4.5 Soil Ingestion Results (mg/day) for Children Aged 1 to 4 Years from Calabrese et al. (1989)

Tracer:	Aluminum	Silicon	Titanium	Vanadium	Yttrium	Zirconium
Mean	153	154	218	459	85	21
Median	29	40	55	96	9	16
SD	852	693	1150	1037	890	209
95^{th} %	223	276	1432	1903	106	110
Max	6837	5549	6707	5676	6736	1391

One child in this study exhibited pica behavior. The high soil ingestion rates for this child may or may not be applicable to other soil pica children or, over time, even to this one child. However, it is interesting to note that this study did pick up a child with this behavior.

There are a number of methodological difficulties in attempting to quantify soil ingestion using the tracer methodology. Food (including vitamins and medicines), soil, and fecal material are analyzed for specific tracer elements in a mass balance approach to estimate soil ingestion. The assumption is that the tracer elements measured in the feces are exclusively from the food and medicines analyzed. However, transit time

through the gut varies widely. The fecal sample may not represent the food/medicine sample input. This input-output misalignment can underestimate soil ingestion and could result in negative soil ingestion estimates.

The other main type of error in tracer studies for estimating soil ingestion is source error. Source error occurs when an unknown or unaccounted for source of the tracer element is ingested by the study subjects. The soil ingestion estimate can be inflated since it is assumed that soil is the source of tracer.

However, this study is useful in several ways. The mass balance approach attempts to correct for ingestion of tracer such as titanium in foods, medicines, and toothpaste. The validation regimen in adults points out the most reliable tracers and validates the overall methodology. The complete sample collection of urine and feces in this study obviates the need to assume a fecal weight for calculating soil ingestion estimates. A relatively large population was studied, but it may not be entirely representative of the U.S. population because it was selected from a single location. The results presented in this paper have been superseded by more refined analyses of the same data by the authors (Stanek and Calabrese, 1995a and 1995b).

4.4.3.1.2 Calabrese and Stanek (1992)

This study estimated the amount of outdoor soil in indoor dust using statistical modeling. Data from 60 homes in the Calabrese *et al.* (1989) study were used to develop scatter plots of each tracer concentration in soil (outdoor) versus dust (indoor) for the subject population. The scatter plots show little evidence of a consistent relationship between outdoor soil and indoor dust concentrations.

The assumption is that 50% of excess fecal tracers were from indoor origin. Multiplying this by the model prediction that 31.3% of indoor dust came from outdoor soil resulted in an estimate that 15% of excess fecal tracers were from soil material present in indoor dust. These analyses indicate that approximately 65% of the total fecal tracer was of soil origin and the estimates of median outdoor soil ingestion presented in the earlier study should be reduced by 35%. The revised soil ingestion estimates are reduced from 29 to19 mg/d based on aluminum, 40 to 26 mg/d based on silicon, and 9 to 6 mg/d based on yttrium.

The model uses several simplifying assumptions: a) the amount of dust produced every day from both indoor and outdoor sources in a house is constant for all houses, b) the proportion of indoor dust due to outdoor soil is constant for all houses, and c) the concentration of the tracer element in dust produced from indoor sources is constant for all houses. The validity of these assumptions cannot be evaluated and subsequent papers by the authors did not make use of this adjustment.

4.4.3.1.3 Stanek and Calabrese (1995a)

Stanek and Calabrese (1995a) reanalyzed the soil ingestion study by Calabrese *et al.* (1989). The individual daily soil ingestion estimates (64 subjects for 8 days) were used to develop distributions of values for 365 days for each subject using an assumed

lognormal distribution. All soil ingested was assumed to come from outdoors and food intake was directly linked with fecal output. Daily soil ingestion estimates were made for each element and each study subject. The study links the food samples with the fecal samples in an attempt to more accurately estimate soil ingestion rates. In addition, the tracers were ranked according to their usefulness, and criteria for excluding certain soil ingestion estimates were incorporated into the reanalysis.

Negative estimates were replaced with a value of 1 mg/day. For each day and subject, medians, and lower and upper bounds of soil ingestion rate were calculated for the eight tracers. The lower and upper bounds functioned as exclusion criteria. If a soil ingestion rate estimate fell outside the bounds, it was assumed to be invalid and discarded. The investigators took estimates of the means and medians of the subjects' daily soil ingestion and constructed their cumulative distributions.

The results indicate that mean soil ingestion estimates over the study period of four to eight days were 45 mg/day or less for 50% of the children and 208 mg/day or less for 95% of the children. The median daily soil ingestion estimates were 13 mg/day or less for 50% of the children studied, and 138 mg/day or less for 95% of the children studied.

The median of the distribution of average daily soil ingestion extrapolated over 365 days is 75 mg, while the 95th percentile is 1751 mg/day. The median of the distribution of median soil ingestion estimates is 14 mg/day while the 95th percentile is 252 mg/day. The range of upper 95th percentiles of the median soil ingestion rate estimates for 63 kids (exclusive of the one pica child) is 1 to 5623 mg/day.

Stanek and Calabrese (1995a) also evaluated the presence of soil pica using their distribution methodology. They estimated that on 35-40 days of the year, 16% of children would ingest more than 1 gram/d of soil and 1.6% would ingest more than 10 grams/d.

Table 4.6 Estimates of Children (%) Exceeding Certain Soil Ingestion Rates from Stanek and Calabrese (1995a)

Soil Ingestion Rate	Days per year of excessive soil ingestion		
	1-2	7-10	35-40
> 1 gram	63%	41%	16%
> 5 grams	42%	20%	1.6%
>10 grams	33%	9%	1.6%

There are many limitations to the study, one of which is the assumption of lognormal distributions to estimate daily soil ingestion over 365 days. There is little empirical evidence to support its use. The number of samples needed to capture typical intake over a year would be considerably more and seasonal variability would need to be taken into account. There are methodological difficulties in quantifying the distribution of soil ingestion rates such as assuming that the transit time in the gut was the same for all subjects and did not vary within subjects. The correction used is unlikely to be

adequate to account for the input-output misalignment error, probably resulting in the negative soil ingestion estimates as obtained in Calabrese *et al.* (1989).

There are large discrepancies between trace elements estimates of soil ingestion for the same subject on the same day. The outlier criterion was used to correct for the likelihood that ingestion of some tracers occurred from other sources than food or soil. The exclusion methodology (using the median as a reference point rather than the mean) did not indicate how many data points were excluded or what those data points were. However, the effect of these exclusions is probably small as indicated by comparing the distributions of the mean estimates (where three or fewer elements are used following exclusion) with the distribution of the mean estimates (where no elements are excluded).

Short term studies are often all that are available to extrapolate to long term intakes needed for risk assessment. However, the limitations need to be acknowledged and the data available must be sufficient to perform the quantification.

4.4.3.1.4 Stanek and Calabrese (1995b)

Stanek and Calabrese (1995b) reanalyzed the data from their 1989 study with data from Davis *et al.* (1990) using a different methodology from that used in Stanek and Calabrese (1995a). The Best Tracer Method (BTM), based on the food to soil ratio, is designed to overcome inter-tracer inconsistencies in the estimation of soil ingestion rates. It is assumed that tracers with a low food to soil ratio lead to more precise soil ingestion estimates because confounding from the tracer content of food is decreased.

The combined data from the two studies (Calabrese *et al.* 1989 and Davis *et al.* 1990) were used to construct estimates of the food to soil (F/S) ratio for each trace element for each subject/week. The F/S ratio was calculated by dividing the average daily amount of a trace element ingested from food by the soil trace element concentration per gram soil. For each subject/week, these ratios were ranked lowest to highest. The F/S ratio is small when the tracer concentration in food is almost zero compared to the tracer concentration in soil. A small F/S ratio is desirable because it lessens the impact of transit time error. This error occurs when fecal output does not reflect food ingestion, due to fluctuation in gastrointestinal transit time. Distributions of soil ingestion estimates are presented based on the various ranked tracers for both children (Calabrese *et al.* 1989; Davis *et al.* 1990) and adults (Calabrese *et al.* 1990).

In contrast to the Stanek and Calabrese (1995a) study, negative values for soil ingestion estimates were included in the distributions. This would shift the distribution towards lower ingestion estimates. While it is valuable to eliminate source error as much as possible by utilizing elements with low F/S ratios, the presence of negative soil ingestion estimates is indicative that there still is a problem with input-output misalignment. Negative soil ingestion estimates are biologically meaningless, and incorporating these values into a distribution is problematic. Distributions of soil ingestion estimates from the combined studies for children are presented in Table 4.7.

Table 4.7 Distributions of Soil Ingestion Estimates (mg/d) in Children from Stanek and Calabrese (1995b)

Studies	Percentiles						Mean ± SD	Min	Max
	10th	25th	50th	90th	95th	99th			
A[a]	-6	9	33	110	154	226	132 ± 1006	-97	11,415
B[b]	-52	-15	44	210	246	535	69 ± 146	-404	905
A and B	-12	10	37	156	217	535	104 ± 758	-404	11,415

Table based on element groupings formed by ranked food:soil ratios.
[a] Study A: data from Calabrese et al., 1989 [b] Study B: data from Davis et al., 1990

Based on the 64 children in the Calabrese *et al.* (1989) study and using the median soil ingestion estimates from the best four tracers, the mean soil ingestion rate was 132 mg/day and the median soil ingestion rate was 33 mg/day. The 95th percentile value was 154 mg/day. For the 101 children in the Davis *et al.* (1990) study, the mean soil ingestion rate was 69 mg/day and the median soil ingestion rate was 44 mg/day. The 95th percentile estimate was 246 mg/day. When the Calabrese *et al.* (1989) and Davis *et al.* (1990) studies were combined, soil ingestion rates for children were estimated to be 104 mg/day (mean), 37 mg/day (median) and 217 mg/day (95th percentile), using the BTM. When the adult data from the Calabrese *et al.* (1990) study were reevaluated, soil ingestion rates were estimated to be 64 mg/day (mean), 87 mg/day (median), and 142 mg/day (95th percentile), using the BTM.

This study combines data from two studies of children, one from southwestern Washington and one from Massachusetts, thus increasing the number of observations. It also corrects for some differences associated with tracer metabolism. The limitations associated with the data used in this study are the same as the limitations described earlier in the summaries of the Calabrese *et al.* (1989), Davis *et al.* (1990) and Calabrese *et al.* (1990) studies.

4.4.3.2 Anaconda, Montana Studies

4.4.3.2.1 Calabrese et al. (1997)

Sixty-four children ages 1-3 years and predominantly from two-parent households living on a Superfund site in Anaconda, Montana were selected for this study. Thirty-six of the 64 children were male, and the children ranged in age from 1 to 3 years with approximately an equal number of children in each age group. The study was conducted for seven consecutive days during a two week period in the month of September.

Duplicate samples of meals, beverages, and over- the-counter medicines and vitamins were collected over the seven day period, along with fecal samples. In addition, soil and dust samples were collected from the children's home and play areas. Toothpaste containing non-detectable levels of the tracer elements, with the exception of silica, was provided to all of the children. Infants were provided with baby cornstarch, diaper rash

cream, and soap which were found to contain low levels of the tracer elements. The mass-balance methodology similar to that in Calabrese *et al.* (1989) was used.

As in Calabrese *et al.* (1989), an additional study was conducted in which the mass-balance methodology was used on adults in order to validate that soil ingestion could be detected. Known amounts of soil were administered to ten adults (5 males, 5 females) from Western Massachusetts over a period of 28 days. Each adult ingested for 7 consecutive days: a) no soil during Week 1, b) 20 mg of sterilized soil during Week 2, c) 100 mg of sterilized soil during Week 3, and d) 500 mg of sterilized soil during Week 4. Duplicate food and fecal samples were collected every day during each study week and analyzed for the eight tracer elements (aluminum, silicon, titanium, cerium, lanthanum, neodymium, yttrium, and zirconium). The authors determined that a soil ingestion of 200 to 500 mg/day could be detected in a reliable manner.

Soil ingestion by each tracer element was estimated using the Best Tracer Method (BTM), which allows for the selection of the most recoverable tracer for a group of subjects (Stanek and Calabrese, 1995b). The median soil ingestion estimates for the four best trace elements based on food:soil ratios for the 64 children are presented in Table 4-8. The best estimate was calculated by taking the median of these four trace elements. Based on the soil ingestion estimate for the best tracer, the mean soil ingestion rate was 66 mg/day and the median was 20 mg/day. The 95th percentile value was 283 mg/day. Using the median of the 4 tracers, the mean was 7 mg/day and the 95th percentile was 160 mg/day.

These results are lower than the soil ingestion estimates obtained by Stanek and Calabrese (1995a). The investigators believed that families, who participated in this study, were aware that they lived on an EPA Superfund site and this knowledge might have resulted in reduced exposure. There was no statistically significant difference found in soil ingestion estimates by gender or age, by housing or yard characteristics (i.e., porch, deck, door mat, etc.), or between children with or without pets.

The advantages of this study were a consecutive seven day study period rather than two periods of 3 and 4 days (Stanek and Calabrese, 1995a), the use of the BTM, and the use of a dietary education program to reduce food tracer input and variability.

Table 4.8 Soil Ingestion Estimates for 64 Anaconda Children (mg/day) Based on Food:Soil Ratios for Aluminum, Silicon, Titanium, Yttrium, and Zirconium [b]

Tracer	Soil Ingestion (mg/day)[a]										
	Percentile							Min	Max	Mean	SD
	5th	10th	25th	50th	75th	90th	95th				
Median[b]	-91.0	-53.8	-38.0	-2.4	26.8	73.1	159.8	-101.3	380.2	6.8	74.5
Best	-24.4	-14.4	2.2	20.1	68.9	223.6	282.4	-53.4	609.9	65.5	120.3
2nd best	-62.1	-48.6	-26.6	1.5	38.4	119.5	262.3	-115.9	928.5	33.2	144.8
3rd best	-88.9	-67.0	-52.0	-18.8	25.6	154.7	376.1	-170.5	1293.5	31.2	199.6
4th best	-171.0	-131.9	-74.7	-29.3	0.2	74.8	116.8	-298.3	139.1	-34.6	79.7

[a] Negative values occurred as a result of calculating child-specific estimates for multiple days. For example, negative estimates of soil ingestion occurred when an individual child had low, but positive, soil ingestion, but the standard deviation was large.
[b] Median value of best four tracers

Table 4.9 Dust Ingestion Estimates for 64 Anaconda Children (mg/day) Based on Food/Dust Ratios for Aluminum, Silicon, Titanium, Yttrium, and Zirconium [b]

Tracer	Dust Ingestion (mg/day)[a]										
	Percentile							Min	Max	Mean	SD
	5th	10th	25th	50th	75th	90th	95th				
Median[b]	-186.2	-152.7	-69.5	-5.5	62.8	209.2	353.0	-261.5	683.9	16.5	160.9
Best	-193.8	-91.0	-20.8	26.81	198.1	558.6	613.6	-377.0	1499.4	127.2	299.1
2nd best	-147.2	-137.1	-59.1	7.6	153.1	356.4	409.5	-239.8	1685.1	82.7	283.6
3rd best	-247.5	-203.1	-81.7	-14.4	49.4	406.5	500.5	-375.7	913.2	25.5	235.9
4th best	-365.6	-277.7	-161.5	-55.1	52.4	277.3	248.8	-542.7	6120.5	81.8	840.3

[a] Negative values occurred as a result of calculating child-specific estimates for multiple days. For example, negative estimates of dust ingestion occurred when an individual child had low, but positive, dust ingestion, but the standard deviation was large.
[b] Median value of best four tracers.

However, the data presented in this study are from a single seven-day period during September which may not reflect soil ingestion rates for longer time-periods or other seasonal months. The net residual negative error indicates probably an underestimation in the soil ingestion rates. The investigators estimated that this error is unlikely to affect the median value by more than 40 mg/day. Since the data from half of the distribution are negative, it is difficult to place a lot of confidence in the soil and dust ingestion estimates obtained.

4.4.3.2.2 Calabrese et al. (1996)

In this study Calabrese *et al.*, (1996) examined the hypothesis that differences in soil tracer concentrations could be related to soil particle size. Soil that was used by Calabrese *et al.* (1997) from Anaconda, Montana was reanalyzed for the tracer concentration after it had been sieved to a particle size of <250 μm in diameter (<2 mm soil particle size in the original study). The smaller particle size was examined based on the assumption that children and adults principally ingest soil of small particle size adhering to fingertips and under fingernails.

Soil concentration was not changed by particle size for five of the tracers used in the original study (aluminum, silicon, titanium, yttrium, and zirconium). However, the soil concentrations of three tracers (cerium, lanthanum and neodymium) were increased two- to four-fold at the smaller soil particle size. Soil ingestion estimates for these three tracers were decreased by approximately 60% at the 95th percentile, when the effect of particle size on tracer concentration is taken into account.

4.4.3.2.3 Stanek et al. (1999)

Stanek *et al.* (1999) extended the findings from their earlier study (Calabrese *et al.* 1996) by quantifying trace element concentrations in soil of different particle sizes. The soil was sieved to particle sizes of 100 to 250 μm and to particle sizes of 53 to < 100 μm. This study used the data from soil concentrations from the Anaconda, Montana site reported by Calabrese *et al.* (1997).

Results of the study indicated that soil concentrations of aluminum, silicon, and titanium did not increase at the two finer particle size ranges measured. However, soil concentrations of cerium, lanthanum and neodymium increased by a factor of 2.5 to 4.0 in the 100-250 μm particle size range when compared with the 0 to 2 μm particle size range. There was not a significant increase in concentration in the 53 to 100 μm particle size range. The importance of this study and that published in 1996 is that they provide further insights regarding the selection of tracers for soil ingestion studies.

4.4.3.2.4 Stanek and Calabrese (2000)

In this study the soil ingestion data from the Anaconda, Montana study were reanalyzed, assuming a lognormal distribution for the soil ingestion estimates. Average soil ingestion for children was predicted over time periods of 7 days, 30 days, 90 days, and 365 days. The 95th percentile soil ingestion values predicted were 133 mg/day over 7 days, 112 mg/day over 30 days, 108 mg/day over 90 days, and 106 mg/day over 365 days. Based on this analysis, estimates of the distribution of longer term average soil ingestion are expected to be narrower, with the 95th percentile estimates being as much as 25% lower. The limitations to this analysis were similar to that discussed in Stanek and Calabrese (1995a) in Section 4.4.3.1.3.

4.4.4 Clausing and Co-workers Studies

4.4.4.1 Clausing et al. (1987)

This soil ingestion study was conducted with Dutch children using the Limiting Tracer Method (LTM). Aluminum, titanium, and acid-insoluble residue (AIR) contents were determined for fecal samples from children aged 2 to 4 years attending a nursery school and for samples of playground dirt at that school.

Twenty seven daily fecal samples were obtained over a 5-day period for the 18 children examined. Using the average soil concentrations present at the school, and assuming a standard fecal dry weight of 10 g/day, soil ingestion was estimated for each tracer. Eight daily fecal samples were also collected from six hospitalized, bedridden children. These children served as a control group, representing children who had little access to soil. The average quantity of soil ingested by the school children in this study was 230 mg/day (range 23 to 979 mg/day) for aluminum; 129 mg/day (range 48 to 362 mg/day) for AIR; and 1,430 mg/day (range 64 to 11,620 mg/day) for titanium. As in the Binder et al. (1986) study, a fraction of the children (6/19) showed titanium values well above 1,000 mg/day.

Table 4.10 Soil Ingestion Results (mg/day) From Clausing et al. (1987)

	School Children	Hospitalized Children	Difference
Mean	105	49	56
Standard Deviation	67	22	
Range	23-362	26-84	
Geometric Mean	90	45	

Mean soil intake for the school children was estimated to be 105 mg/day with a standard deviation of 67 mg/day (range 23 to 362 mg/day). Geometric mean soil intake was estimated to be 90 mg/day. The soil intake for this group of children was much higher when compared to the hospitalized children used as the control group (mean 49 mg/day, standard deviation 22 mg/day).

Mean (arithmetic) soil intake for the hospitalized children was estimated to be 56 mg/day based on aluminum. For titanium, three of these children had estimates well in excess of 1,000 mg/day, with the remaining three children in the range of 28 to 58 mg/day. The mean soil ingestion rate was estimated to be 49 mg/day with a population standard deviation of 22 mg/day (range 26 to 84 mg/day). The geometric mean soil intake rate was 45 mg/day (Table 4-10).

The data on hospitalized children suggest a non-soil source of titanium and aluminum. However, conditions specific to hospitalization (e.g., medications) were not considered. Assuming that soil ingestion rates observed in hospitalized children actually represent background tracer intake from dietary and other non-soil sources, mean soil ingestion by nursery school children was estimated to be 56 mg/day (i.e., 105 mg/day for nursery school children minus 49 mg/day for hospitalized children).

The advantages of this study are that the investigators evaluated soil ingestion among children that had differences in access to soil and soil intake rates were corrected based on background estimates derived from the hospitalized group. However, the number of children used in this study was small. Tracer elements in foods or medicines were not evaluated. Also, the study was a short-term study and the intake rates may not be representative of soil intake over the long-term. The children's activities were not monitored. For example, hand washing frequency could impact soil ingestion.

4.4.4.2 Van Wïjnen et al. (1990)

In this study soil ingestion among Dutch children ranging in age from 1 to 5 years was evaluated using the tracer element methodology (LTM) used by Clausing *et al.* (1987). Three tracers (titanium, aluminum, and acid insoluble residue (AIR)) were measured in soil and feces and soil ingestion was estimated from the measurements. An average daily feces dry weight of 15 g was assumed. A total of 292 children attending daycare centers were sampled during the first sampling period and 187 children were sampled in the second. A total of 78 children were sampled at campgrounds. Samples taken from 15 hospitalized children were used as controls.

The mean soil ingestion values for these groups were: 162 mg/day for children in daycare centers, 213 mg/day for campers and 93 mg/day for hospitalized children. Geometric means were estimated to be 111 mg/day for children in daycare centers, 174 mg/day for children vacationing at campgrounds and 74 mg/day for hospitalized children (70-120 mg/day based on the 95th percent confidence limits of the mean) (Table 4-11). AIR was the limiting tracer in about 80 percent of the samples. Among children attending daycare centers, soil intake was also found to be higher when the weather was good.

The investigators used the mean value (93 mg/day) for hospitalized children as the background intake of tracers. Using the mean value to correct the soil intake rates, corrected soil intake rates were 69 mg/day for daycare children and 120 mg/day for campers. Corrected geometric mean soil intake was estimated to range from 0 to 90 mg/day with a 90th percentile value of 190 mg/day for the various age categories within the daycare group and 30 to 200 mg/day with a 90th percentile value of 300 mg/day for the various age categories within the camping group.

The major limitation of this study is that tracer concentrations in food and medicine were not evaluated. Although the population of children studied was relatively large, it may not be representative of the U.S. population. This study was conducted over a relatively short time period and estimated intake rates may not reflect long-term patterns, especially at the high-end of the distribution. Another limitation of this study is that values were not reported element-by-element, and the children's daily activities such as hand washing frequency were not monitored.

Table 4.11 Soil Ingestion Values Using the LTM Methodology for Children at Daycare Centers and Campgrounds

Age (Years)	Sex	Daycare centers			Campgrounds		
		N	Geometric Mean(mg/d)	Geometric Standard Deviation(mg/d)	N	Geometric Mean(mg/d)	Geometric Standard Deviation(mg/d)
birth to <1	**Girls**	3	81	1.09	-	-	-
	Boys	1	75	-	-	-	-
1 to <2	**Girls**	20	124	1.87	3	207	1.99
	Boys	17	114	1.47	5	312	2.58
2 to <3	**Girls**	34	118	1.74	4	367	2.44
	Boys	17	96	1.53	8	232	2.15
3 to <4	**Girls**	26	111	1.57	6	164	1.27
	Boys	29	110	1.32	8	148	1.42
4 to <5	**Girls**	1	180	-	19	164	1.48
	Boys	4	99	1.62	18	136	1.30
CombinedAll ages	**Girls**	86	117	1.70	36	179	1.679
	Boys	72	104	1.46	42	169	1.7
Total		162[a]	111	1.60	78[b]	174	1.73

[a] Age and/or sex not registered for eight children.

[b] Age not registered for seven children.

4.4.5 Other Relevant Studies and Analyses

4.4.5.1 Thompson and Burmaster (1991)

Thompson and Burmaster (1991) developed parameterized distributions of soil ingestion rates for children based on a reanalysis of the key study data collected by Binder *et al.* (1986). In the original Binder *et al.* (1986) study, an assumed dry fecal weight of 15 g/day was used. Thompson and Burmaster re-estimated the soil ingestion rates from the Binder *et al.* (1986) study using the actual stool weights of the study participants instead of the assumed stool weights. Because the actual stool weights averaged only 7.5 g/day, the soil ingestion estimates presented by Thompson and Burmaster (1991) are approximately one-half of those reported by Binder *et al.* (1986).

The mean soil intake rates were 97 mg/day for aluminum, 85 mg/day for silicon, and 1,004 mg/day for titanium. The 90th percentile estimates were 197 mg/day for aluminum, 166 mg/day for silicon, and 2,105 mg/day for titanium. Based on the arithmetic average of aluminum and silicon for each child, mean soil intake was estimated to be 91 mg/day and 90th percentile intake was estimated to be 143 mg/day (Table 4-12).

Table 4.12 Distribution of Soil Ingestion Estimates For Children by Thompson and Burmaster (1991)

	Soil Intake (mg/d)			
	Aluminum	**Silicon**	**Titanium**	**Mean[a]**
Mean	97	85	1004	91
Median	45	60	293	59
90th %	197	166	2105	143

[a] Arithmetic average of soil ingestion based on aluminum and silicon

Thompson and Burmaster (1991) also adjusted Binder *et al.* (1986) data for aluminum, and silicon for lognormal distribution. No adjustment was made for titanium because titanium may be present in high concentrations in food and the Binder *et al.* (1986) study did not correct for food sources of titanium. Statistical tests indicated that only silicon and the average of the silicon and aluminum tracers were lognormally distributed.

The advantages of this study are that it provides percentile data and defines the shape of soil intake distributions. However, the number of data points used to fit the distribution was limited. This analysis is based on a study that did not correct for tracer intake from food or medicine and the methodological difficulties encountered in the original Binder *et al.* study still exist including difficulty in obtaining the entire fecal sample from a diaper.

4.4.5.2 Sedman and Mahmood (1994)

The data of two previous studies, Calabrese *et al.* 1989 and Davis *et al.* 1990, were used to obtain estimates of the average daily soil ingestion in young children. The soil ingestion in these children was determined by dividing the excess tracer intake (the quantity of tracer recovered in the feces in excess of the measured intake) by the average concentration of tracer in soil samples from each child's dwelling.

The mean estimates of soil ingestion in children for each tracer were adjusted from both studies to reflect that of a 2-year old child. The mean of the adjusted levels of soil ingestion for a two year old child was 220 mg/kg for the Calabrese *et al.* (1989) study and 170 mg/kg for the Davis *et al.* (1990) study. Based on a normal distribution of means, the mean estimate for a 2-year old child was 195 mg/day. Based on uncertainties associated with the method employed, the authors recommended a conservative estimate of soil ingestion in young children of 250 mg/day. Based on the 250 mg/day ingestion rate in a 2-year old child, a lifetime intake was estimated to be 70 mg/day.

4.4.5.3 Calabrese and Stanek (1995)

Calabrese and Stanek (1995) examined the various sources and magnitude of positive and negative errors in soil ingestion estimates for children.

Possible sources of positive errors include:

a) ingestion of high levels of tracer elements before the start of the study and low ingestion during the study period, and
b) ingestion of tracer elements from a non-food or non-soil source during the study period.

Possible sources of negative bias include:

a) ingestion of tracer elements in food, but they are not captured in the fecal sample either due to slow transit time or not having a fecal sample available on the final study day, and
b) diminished detection of tracer element levels in fecal, but not in soil samples.

The data of Calabrese *et al.* (1989) were quantified to reduce the magnitude of error in the individual trace element ingestion estimates. A lag period of 28 hours was assumed for the passage of tracers ingested in food to the feces. A daily soil ingestion rate was estimated for each tracer for each 24-hr day fecal sample. Daily soil ingestion rates for tracers that fell beyond the upper and lower ranges were excluded from subsequent calculations, and the median soil ingestion rates of the remaining tracer elements were considered the best estimate for that particular day.

The positive and negative errors for six tracer elements from the 1989 Calabrese *et al.* study were estimated. The original mean soil ingestion rates ranged from a low of 21 mg/day based on zirconium to a high of 459 mg/day based on titanium. The adjusted mean soil ingestion rate after correcting for negative and positive errors ranged from 97 mg/day based on yttrium to 208 mg/day based on titanium.

The authors concluded that correcting for errors at the individual level for each tracer element provides more reliable estimates of soil ingestion. However, this approach is based on the hypothesis that the median tracer value is the most accurate estimate of soil ingestion, and the validity of this assumption depends on the specific set of tracers used in the study. The estimation of daily tracer intake is the same as in Stanek and Calabrese (1995a), and the same limitations mentioned earlier in Calabrese *et al.*(1989) still exist.

4.4.5.4 Stanek et al. (2001)

The authors developed a simulation model to identify and evaluate biasing factors for soil ingestion estimates from data taken from Calabrese *et al.* (1989), Davis *et al.* (1990), and Calabrese *et al.* (1997). Only the data from the aluminum and silicon trace element estimates were used.

Study duration has the most positive bias in all the biasing factors explored, with a bias of more than 100% for the 95th percentile estimates in the 4-day mass balance study. A smaller bias was observed for the impact of absorption of trace elements from food. Although the trace elements selected for use in the mass balance studies are believed to have low absorption, the amount unaccounted for will result in an underestimation of the soil ingestion distribution. In these simulations, the absorption of trace elements from food of up to 30% was shown to negatively bias the estimated soil ingestion distribution by less than 20 mg/day.

4.4.5.5 Zartarian et al. (2005)

Zartarian *et al.* (2005) conducted an analysis of soil ingestion rates using data from several studies as input for the Stochastic Human Exposure and Dose Simulation (SHEDS) model for the U.S. EPA. Data from Calabrese's Amherst and Anaconda studies (Calabrese *et al.* 1989, 1997) were used to fit distributions of soil/dust ingestion rates. The statistical distributions relied upon two tracers only, aluminum and silicon, in estimating the parameters of the lognormal variability and uncertainty distributions.

Using a Monte-Carlo sampling method, values from the fitted distribution were separated into those values under 500 mg/day and values that exceeded 500 mg/day. Soil ingestion values that exceed 500 mg/day are assumed to represent pica behavior. Using the SHEDS model, the soil ingestion rate distribution for non-pica behavior children has a mean of 61, standard deviation of 81, median of 30, 95th percentile of 236, and 99th percentile of 402 (mg/day). For children exhibiting pica behavior, the mean is 962, standard deviation 758, median 735, 95th percentile 2130, and 99th percentile 3852 (mg/day).

A limitation of this analysis is that pica children and incidental ingestion were simulated separately. The distribution for incidental soil ingestion does not take into account that children may have days where they ingest unusually high levels of soil, which may not be indicative of long-term pica behavior.

4.4.5.6 Hogan et al. (1998)

Hogan *et al.* (1998) published a paper that compares observed and predicted children's blood lead levels as applied to the Integrated Exposure and Uptake Biokinetic (IEUBK) model for lead in children. The IEUBK model is being used by the U.S. EPA and state regulatory agencies as a model for lead uptake from environmental media for risk assessments. The model functions primarily to estimate the risk and probability of children having blood lead concentrations exceeding a specific level of concern. It predicts children's blood levels by using measurements of lead in house dust, soil, drinking water, food and air together with default inputs such as child-specific estimates of intake for each exposure medium.

One of the parameters that the IEUBK model uses to estimate child blood lead concentration is the ingestion of soil and household dust. Young children are primarily exposed to lead through fine particles of surface soil and household dust that adhere to

their hands and are incidentally ingested during normal hand-to-mouth activities. The age-specific default soil and dust ingestion rates recommended for use in the IEUBK model (version 0.99d) are 50 and 60 mg/day (averaged over children ages 1 through 6), respectively. The combined soil and dust ingestion is 110 mg/day. The default soil ingestion values used in the IEUBK model are based on several observational studies by Binder *et al.* (1986), Clausing *et al.* (1987), Calabrese *et al.* (1989, 1991), van Wijnen *et al.* (1990) and Davis *et al.* (1990), utilizing the trace element methodology (U.S. EPA, 1994).

Hogan *et al*. (1998) applied an empirical comparisons exercise of the IEUBK method to evaluate three epidemiologic datasets consisting of blood lead levels of 478 children. These children were a subset of the entire population of children living in three historic lead smelting communities: Palmerton, Pennsylvania; Southern Kansas/southwestern Missouri; and Madison County, Illinois. The children's measured blood lead levels were compared with the IEUBK's blood lead predictions using measured lead levels in drinking water, soil and dust together with the model's default inputs such as soil/dust ingestion rates and lead bioavailability.

Results showed that there was reasonably close agreement between observed and IEUBK predicted blood lead distributions in the three studies. The geometric means for the observed and predicted blood lead levels were within 0.7 µg/dl. U.S. EPA (2008) used this study to do a back calculation on the soil and dust ingestion rates and concluded that the numbers (50 mg/d soil; 60 mg/d dust; and 110 mg/d combined) are "roughly accurate in representing the central tendency soil and dust ingestion rates" of children ages 1 to 6.

4.4.6 U.S. EPA (2008)

The U.S. EPA (2008) *Child-Specific Exposure Factors Handbook* considered certain studies as "key" for developing recommendations for children's soil ingestion rates. Key tracer element methodology, biokinetic model comparison, and survey response studies were selected based on judgment about the study's design features, applicability, and utility of the data to U.S. children, clarity and completeness, and characterization of uncertainty and variability in ingestion estimates. Most of the key studies selected are the same as those described in this Section.

The soil ingestion recommendations represented ingestion of a combination of soil and outdoor settled dust. The dust ingestion recommendations included soil tracked into indoor environment, indoor settled dust and air-suspended particulate matter that is inhaled and swallowed. The recommended values for soil and dust are on a dry weight basis.

The recommended central tendency soil and dust ingestion for infants 6 months up to their first birthday is 60 mg/d (soil 30 mg/d, dust 30 mg/d), and for children ages 1 to <6 years is 100 mg/d (soil 50 mg/d, dust 60 mg/d, sum rounded to 100 mg/d). In the absence of data that can be used to develop specific central tendency soil and dust ingestion recommendations for children aged 6 to <11 years, 11 to <16 years and 16 to

<21 years, U.S. EPA (2008) recommends using the central tendency soil and dust ingestion rate of 100 mg/d developed for children ages 1 to <6 years. An important factor is that the recommendations did not extend to issues regarding bioavailability of the contaminants present in the soil and dust.

Table 4.13 Recommended Values for Daily Soil and Dust Ingestion From U.S. EPA (2008)

Age Group	Central Tendency Values, mg/day		
	Soil	Dust	Soil and Dust
6 to <12 m	30	30	60
1 to <6 y	50	60	100[a]
6 to <21 y	50	60	100[a]

[a] Sum of 110 mg/d rounded to one significant figure
Adapted from Child-Specific Exposure Factors Handbook, U.S. EPA (2008)

4.5 Soil Ingestion Adult Studies

There are few studies that estimated adult soil ingestion. The three studies that provide data used in the estimation of soil ingestion in adults did not provide the ages of the individuals studied. They were not designed as adult soil ingestion studies but rather as a validation of the methodology used to study soil ingestion in children.

4.5.1 Hawley (1985)

Hawley (1985) suggested a value of 480 mg/day for adults engaged in outdoor activities, a range of 0.6 to 110 mg/day of house dust during indoor activities, and an annual average of 60.5 mg/day. These estimates were derived from assumptions about soil/dust levels on hands, mouthing behavior, and frequencies of certain indoor and outdoor activities, without supporting measurements.

4.5.2 Calabrese et al (1990)

This study was originally part of the study in children in Calabrese *et al.* (1989). The soil ingestion rates for the 6 volunteer adults were estimated by subtracting out the tracer quantities in food and soil capsules from the amounts excreted. The four most reliable tracers were aluminum, silicon, yttrium, and zirconium. Median soil ingestion rates were as follows: aluminum, 57 mg; silicon, 1 mg; yttrium, 65 mg; and zirconium, -4 mg. Mean values were: aluminum, 77 mg; silicon, 5 mg; yttrium, 53 mg, and zirconium, 22 mg. The average of the soil ingestion means based on the four tracers is 39 mg. The sample size is very small (n = 6) and the study was not designed to look at soil ingestion by the adults but rather as a validation of the overall soil ingestion tracer methodology.

4.5.3 Stanek and Calabrese (1995b)

Stanek and Calabrese (1995b) reanalyzed the data from their 1989 study of children with data from Davis *et al.* (1990), and their adult study (Calabrese *et al.* 1990) using the Best Tracer Method (BTM). Distributions of soil ingestion estimates were based on the various ranked tracers for both children and adults. A description of this study is provided in Section 4.4.3. When the adult data from the Calabrese *et al.* (1990) study were reevaluated, soil ingestion rates were estimated to be 64 mg/day (mean), 87 mg/day (median), and 142 mg/day (95^{th} percentile), using the BTM.

4.5.4 Stanek et al. (1997)

Soil ingestion was evaluated in 10 adults as part of a larger study to evaluate soil ingestion in children. The average daily soil ingestion (taken over 4 weeks) was 6 mg/day. The estimation was based on four tracer elements aluminum, silicon, titanium, and zirconium, although 8 tracers were measured. The authors reported that "the broad range in estimates for different trace elements implies that a simple average estimate (over the eight trace elements) provides little insight into adult soil ingestion, since estimates based on different trace elements for the same adults and time periods are so highly variable". To account for variability and bias, the authors decided to base the estimate of soil ingestion on trace elements whose concentrations in soil are relatively homogeneous across different particle sizes. Trace elements that satisfied this criterion include aluminum, silicon, titanium, yttrium and zirconium, and they were considered for estimating soil ingestion by the authors.

However, this study has some complications. One of the ten adults in the study had a high soil ingestion estimate (2 grams) on the first day. The subject also had 4 times higher freeze-dried fecal weight than on any day of the study suggesting that this may be due to days of fecal accumulation. The result is an inflated 95^{th} percentile soil ingestion estimate.

Calabrese (2003) recommended that the upper 75^{th} percentile estimate soil ingestion of 49 mg/day be used as an estimate of high-end soil ingestion by adults (letter to the General Electric Company concerning the U.S. EPA's Human Health Assessment for the Housatonic River) (Calabrese *et al.* 2003). Although the outlier subject in the study causes the 95th percentile soil ingestion estimate to be inflated, it should not be ignored as enhanced adult ingestion could occur among agricultural or utility workers. The study itself also shows that there are problems in the use of tracers and the results varied depending upon which set of tracers was used.

4.5.5 Davis and Mirick (2006)

This study estimated soil ingestion in children aged 3 to 8 years and their parents (16 mothers and 17 fathers) for 11 consecutive days. Three trace elements (Al, Si, and Ti) were measured. The ages of the adults were not provided.

Since titanium exhibits much greater variability compared to other tracer elements due to its presence in various non-soil sources, only Al and Si were used to estimate the adult daily soil ingestion. The means of the mothers and fathers are calculated to be 58 and 47 mg/day, respectively. The weighted average for the combined adults is 53 mg/day.

Table 4.14 Adult Soil Ingestion Estimates from Davis and Mirick (2006)

Tracer Element	Mean Adult Soil Ingestion (mg/day)	
	Mothers	Fathers
Al	92.1	68.4
Si	23.2	26.1
Mean	57.7	47.3
Mean of All Adults	52.5	

4.5.6 Summary of Adult Soil Ingestion Estimates

The mean and 95th percentile adult soil ingestion rates are calculated from the studies as shown in Table 4-15. For soil ingestion in adults, the average of the mean and the 95th percentile are 41 and 213 mg/day, respectively.

Table 4.15 Summary of Soil Ingestion Estimates (mg/day) in Adults

Study	Mean	P95
Calabrese et al (1990) and Stanek and Calabrese (1995b)	64	142 331
Stanek et al (1997)	6	168[a]
Davis and Mirick (2006)	53	
Average	41	213

[a] The 95th percentile adult soil ingestion from Davis and Mirick (2006) was calculated from data in the paper assuming lognormal distribution.

4.6 PICA

4.6.1 General Pica

General pica is the repeated eating of non-nutritive substances including sand, clay, paint, plaster, hair, string, cloth, glass, matches, paper, feces, and various other items (Feldman, 1986). There are numerous reports on general pica among various populations and this behavior appears to occur in approximately half of all children between 1-3 years of age (Sayetta, 1986). Danford (1982) reported that the incidence of general pica was higher for black children (30%) than for white children (10-18%) between 1-6 years of age. There appears to be no sex differences in the incidence rates (Kaplan and Sadock, 1985).

However, general pica is reported to be higher among children in lower socioeconomic groups (50-60%) than in higher income families (about 30%) and is more common in rural areas (Lourie *et al.* 1963, Vermeer and Frate, 1979). A higher rate of general pica has also been reported in pregnant women, individuals with poor nutritional status, and mentally retarded children (Behrman and Vaughan 1983, Danford 1982, Illingworth 1983, Sayetta 1986).

General pica does not include the consumption of some condiments that contain clay or soil. Examples are the Hawaiian Red Alaea sea salt (containing the red volcanic clay called Alaea) and black sea salt found in many parts of the world (containing lava and other substances). These salts have characteristic taste and are used in cooking and food preservation.

4.6.2 Soil Pica

ASTDR (2001) defines soil pica as the recurrent ingestion of unusually high amounts of soil of between 1,000 - 5,000 mg/day. Bruhn and Pangborn (1971) studied dirt ingestion in migrant agricultural workers among 91 non-black, low-income families in California. The incidence of pica was 19% in children, 14% in pregnant women, and 3% in non-pregnant women. However, in this study “dirt” was not clearly defined and may include non-soil substances.

Data from tracer studies (Binder *et al.*, 1986; Clausing *et al.,* 1987; Van Wïjnen *et al.*, 1990; Davis *et al.*, 1990; and Calabrese *et al.*, 1989) showed that only one child out of the more than 600 children studied ingested soil in significantly large amounts to indicate pica behavior. In addition, parental observations regarding children who are likely to be high soil ingesters were reported to be often inaccurate (Calabrese *et al.*, 1997).

A study by Vermeer and Frate (1979) showed that the incidence of geophagia (i.e., intentional earth eating) was about 16% among children from a rural black community in Mississippi. In this study, the intentional earth eating was described as a cultural practice in the community surveyed and may not be representative of the general

population. However, there are cultures in many parts of the world where soil eating is practiced in religious or sacred rituals.

4.6.3 Soil Pica Behavior in Children

Information on the amount of soil ingested by children with pica behavior is very limited. There is no study on pica children and infrequent pica behavior is often observed in normal children in soil ingestion studies.

4.6.3.1 Calabrese et al. (1991); Calabrese and Stanek (1992)

Calabrese *et al.* (1991) reported a pica child among the 64 children who participated in the soil ingestion study. One 3.5-year-old female child had extremely high soil ingestion, from 74-2200 mg/day during the first week and from 10.1-13.6 g/day during the second week of observation. The upper soil ingestion values for this pica child range from approximately 5 to 7 g/day.

Using a methodology that compared differential element ratios, Calabrese and Stanek (1992b) quantitatively attempt to distinguish outdoor soil ingestion from indoor dust ingestion in this pica child. Using tracer ratios of soil, dust, and residual fecal samples, an analysis was performed which indicates that from 71 to 99% of the tracer originated from soil. The authors concluded that the predominant proportion of the fecal tracers originated from outdoor soil and not from indoor dust.

4.6.3.2 Wong (1988) as reviewed by Calabrese and Stanek (1993)

Wong (1988) in his doctoral thesis studied soil ingestion by 52 children in two government institutions in Jamaica. This study was reviewed by Calabrese and Stanek (1993). The younger group contained 24 children with an average age of 3.1 years (range of 0.3 to 7.6 years). The older group contained 28 children with an average age of 7.2 years (range of 1.8 to 14 years).

Fecal samples were obtained from the children and the amount of silicon in dry feces was measured to estimate soil ingestion. An unspecified number of daily fecal samples were collected from a control group consisting of 30 hospital children with an average age of 4.8 years (range of 0.3 to 12 years). Dry feces were observed to contain 1.45% silicon, or 14.5 mg Si per gram of dry feces. This quantity was used as a baseline representing the background level of silicon ingestion from dietary sources. Observed quantities of silicon greater than 1.45% were interpreted as originating from soil ingestion.

For the 28 children in the older group, soil ingestion was estimated to be 58 mg/day, based on the mean minus one outlier, and 1520 mg/day, based on the mean of all the children. The outlier was a child with an estimated average soil ingestion of 41 g/day over the 4-month period. This child was stated to be “developmentally disabled”, but no information was provided on the nature or severity of the disability. Of the 28 children in the group, 7 had average soil ingestion greater than 100 mg/day, 4 had average soil

ingestion greater than 200 mg/day, and one had average soil ingestion greater than 300 mg/day. Eight children showed no indication of soil ingestion. The mean soil ingestion of all the children was 470 ± 370 mg/day.

Of the 24 children in the younger group, 14 had average soil ingestion of less than 100 mg/day, 10 had average soil ingestion greater than 100 mg/day, 5 had average soil ingestion greater than 600 mg/day, and 4 had average soil ingestion greater than 1000 mg/day. Five children showed no indication of soil ingestion. Of the 52 children studied, 6 displayed soil pica behavior.

The use of a single soil tracer in this study may introduce error in the sampling because there may be other sources of the tracer in the children's environment. For example, certain types of toothpastes have extremely high silica concentrations, and children may ingest significant quantities during brushing. Silica may also be found in indoor dust that children could ingest. Despite these uncertainties, the results indicate that soil pica is not a rare occurrence in younger children in this study population. Results from this Jamaica study may not be indicative of similar behavior in children in the United States.

4.6.3.3 ATSDR (2001)

ATSDR (2001) held a workshop to discuss and review the state of the science on soil pica behavior. The review acknowledges that soil pica clearly exists, but there were insufficient data to determine the prevalence of this behavior in children and in adults. The present ATSDR assumption that soil pica children ingest 5 g of soil/day is supported by only a few subjects (i.e., two children in Massachusetts and six children in Jamaica). The ATSDR (2001) committee advises ATSDR to err on the side of being health protective and to continue using the 5 g/day pica ingestion number until more data become available.

4.6.3.4 Zartarian et al. (2005)

Zartarian *et al.* (2005) conducted an analysis of soil ingestion rates from several studies in the literature using the Stochastic Human Exposure and Dose Simulation (SHEDS) model of the U.S. EPA. Data from Calabrese's Amherst and Anaconda studies were used to fit distributions of soil/dust ingestion rates. A soil pica distribution was obtained by sampling from the fitted lognormal distribution and retaining values above 500 mg/day. The mean and 95th percentile values for this population were estimated to be 963 mg/day and 2170 mg/day, respectively (See Section 4.4.5.6).

4.6.3.5 U.S. EPA (1984)

In a risk assessment for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), U.S. EPA (1984) used 5 g/day to represent the soil intake rate for pica children. The Centers for Disease Control (CDC) in an investigation on the exposure potential to 2,3,7,8-TCDD via soil ingestion used a value of 10 g/day to represent the amount of soil that a child with pica behavior might ingest (Kimbrough *et al.,* 1984). These values are based on only one pica child observed in the Calabrese *et al.* (1989) study where the intake ranged from 10-14 g/day during the second week of observation. The CDC suggests that an

ingestion rate of 10 g/day is a reasonable value for use in acute exposure assessments, based on the available information.

4.6.3.6 U.S. EPA (2008)

In the 2008 U.S. EPA's *Child-Specific Exposure Factors Handbook*, U.S. EPA redefined children's "soil-pica" as the quantity of soil ingested by children above 1000 mg/d. Using this definition, the upper 90th and 95th percentiles of soil ingestion from all the key primary studies were included in the assessment of children's pica soil ingestion. The soil-pica ingestion estimate for children up to age 14 ranged from 400 to 41,000 mg/d. The recommended value for soil pica in children was then set at 1000 mg/day. No data were available for individuals above 14-21 years. We believe this number is probably too low based on our calculations (see Table 4.16).

4.6.3.7 Summary of Pica Behavior Studies in Children

Soil ingestion in 8 children that exhibited pica behavior from two studies is given in Table 4-16. It is important to note that soil pica behavior in children in the studies used was observed over a very short period of time and may not reflect long-term pica behavior. In the absence of data, the ATSDR panelists recommended in the *Summary Report for the ATSDR Soil-Pica Workshop* (2001) that "ATSDR should err on the side of being protective and should use 5000 mg until more data are collected". We concur with this recommendation. Our calculation on pica children in two studies shows that the amount ingested is about 5000 mg/day (Table 4-16).

Table 4.16 Pica Behavior in Children

Sample Size	Observation (days)	Age	Soil Ingestion (mg/day)	Source
1 1	2 4	2.5 -	20,000; 22,000 1000-2000	Calabrese et al. (1989, 1991)
1 1 1 1 1	1 1 "different days" "different days" 1	3.1[a]	1447 7924 1016; 2690; 898 10343; 4222; 1404; 5341 5341	Wong (1988) doctoral thesis. Study reviewed and presented by Calabrese and Stanek (1993)
1[c]	"different days"	7.2[b]	48,300; 60,692; 51,422; 3782	Wong (1988) doctoral thesis. Study reviewed and presented by Calabrese and Stanek (1993)

Number of Children	Average Pica Soil Ingestion (mg/day)
8	10,600
7[d]	5500

[a] Average age of 24 children
[b] Average age of 28 children
[c] This child was stated to be "developmentally disabled" by the author
[d] Excluding last child

4.6.4 Soil Pica Behavior In Adults

The ASTDR report (2001) views adult soil pica to be an extremely rare behavior that has not been characterized. Deliberate consumption of clays or soil (geophagy) has been reported in many parts of the world and is particularly prevalent among certain cultural groups especially during certain rituals or religious ceremonies. However, the clay or soil is typically from known uncontaminated sources. Thus, surface soils are generally not the source of geophagical materials consumed. Very little data are available to establish an unintentional soil ingestion rate for adults with pica behavior.

4.7 Hand-To-Mouth Transfer

The studies discussed earlier examined soil intake using a mass balance methodology that measures trace elements in feces and soil. These studies have various shortcomings one of which is the paucity of data for estimating soil ingestion to a broader age range in children and adults. Data are lacking for children less than 1 and above 7 years of age, and for adults where ages are often not given in the studies.

U.S. EPA (2005) provides guidance on the appropriate age groups to consider when assessing children's exposure and potential dose of environmental contaminants. The recommended childhood age groups for exposure and risk assessments are: birth to <1 month, 1 to < 3 months, 3 to < 6 months, 6 to < 12 months, 1 to < 2 years, 2 to < 3 years, 3 to < 6 years, 6 to < 11 years, 11 to < 16 years, 16 to < 18 years, and 18 to < 21 years. These age groupings take into consideration human developmental and physiological changes that impact exposure and potential dose intake. Hand-to-mouth activities may provide information that may be useful in assessing the ingestion of soil in age groups that do not have direct soil ingestion data.

4.7.1 Hand-to-Mouth Transfer Behavior in Children

Children often put their hands, toys, and other objects in their mouths during normal exploration of their environment, as a sucking reflex and as a habit. This hand-to-mouth behavior may result in the ingestion of soil and dust, from outside and/or indoors. Transfer from the hand to the mouth can occur directly by handling of contaminated soil and indirectly by using products, materials and equipment that come in contact with contaminated soil. This can happen in both occupational and non-occupational settings. Soil ingestion can occur by touching the mouth with the hand, nail biting, finger sucking, eating food (especially with bare hands), smoking cigarettes, and other hand-to-mouth activities.

Generally, children's mouthing behavior is studied using both direct observation and videotaping methodologies (Zartarian *et al.* 1998; Reed *et al.* 1999; Freeman *et al.* 2001, 2005; AuYeung *et al.* 2006, 2008; Black *et al.* 2005; Ferguson *et al.* 2005). Observations may be conducted by an instructed parent, or by a trained person. Videotaping the child's behavior is usually done by a trained technician, and information from these recordings is obtained by a trained person who watches the videotapes.

4.7.2 Probabilistic Models of Hand-to-Mouth Transfer

Estimation of non-dietary ingestion of a chemical via hand-to-mouth contact includes information of the hand residue/soil loading (µg/cm² or µg/g), hand-to-mouth frequency (number of contacts/hr), area of hand surface mouthed (cm²), and exposure duration (hr/day). Probabilistic models have been developed to estimate non-dietary ingestion of a chemical via hand-to-mouth contact (e.g., Calendex™ by Exponent Inc.; CARES™ by International Life Science Institute; Lifeline™ by Lifeline Group; and Residential-SHEDS by U.S. EPA's Office of Research and Development).

These models have certain limitations as the calculations are based on data from the few studies available on non-dietary ingestion via hand-to-mouth contact. The studies used in the models have their own limitations such as the different methods of data collection, analysis and reporting, different age groupings of research subjects, and even different definition of "mouthing". Models such as SHEDS that deal with various microenvironments assume a strong relationship between the total dust ingested and indoor dust loading. Although the ratio of ingested outdoor soil to ingested indoor dust is important, factors influencing exposure and risk such as the types of exposures, chemical pollutants indoors and outdoors, amount of track-in, resuspension and particle size, seasonal effects, and fate and transport are some of the issues still largely uncharacterized.

4.7.3 Relevant Hand-to-Mouth Transfer Studies (Summary)

Studies that provide estimates for a hand load transfer factor or transfer efficiency include the analyses of Dubé *et al.* (2004), Beyer *et al.* (2003), and the report from the Consumer Product Safety Commission (CPSC, 2003).

4.7.3.1 Dubé et al. (2004)

Using data from Stanek and Calabrese (1995a), Dubé *et al.* (2004) estimated the fraction of "dislodgeable" residue on the hands of children that was incidentally ingested daily. The estimate was 25% hand load per day (range: 7 – 100%) for 2 to 6 year olds, and 13% hand load per day (range: 3.5 – 50%) for 7 to 31 year olds. This assumed that individuals 7 years old and up would ingest half the amount of soil as 2 to 6 year olds. Information was not provided for a direct hand-to-mouth transfer factor for soil, the fraction of material on the hand in contact with the mouth that is transferred, the number of hand to mouth contacts, and losses through intermediate contacts.

4.7.3.2 Beyer et al. (2003)

Beyer *et al* (2003), in their assessment of incidental ingestion of metals from laundered shop towels in the workplace, used a value of 13% as the fraction dislodged from the hands that was incidentally ingested on a daily basis by adults.

4.7.3.3 CPSC (2003)

The Consumer Product Safety Commission (CPSC, 2003) developed an estimate of the percent of residue dislodged on the hands that is ingested on a daily basis by children. The estimate was based on data on soil ingestion, soil–skin adherence, and contact surface area of the hand with soil from multiple studies. There are large uncertainties in the available data analyzed. The daily intake estimates for children ranged from 3% to 700% of the mass loaded on the hand (i.e., "handload"), with an average of 43% for both direct and indirect hand-to-mouth activities combined.

4.7.3.4 Zartarian et al. (2000)

Zartarian *et al.* (2000) used the U.S. EPA's Residential Stochastic Human Exposure and Dose Simulation (Residential-SHEDS, 2000) model for pesticides to estimate children's exposure to chlorpyrifos. The primary purpose of the study is to demonstrate the capabilities of the model by simulating the exposures and doses of children who contacted chlorpyrifos residues inside treated residences and on turf-treated residential yards. The hand-to-mouth transfer efficiency of chlorpyrifos was estimated to range from 10% to 50%, based on the data of Zartarian *et al.* (1997); Leckie *el al.* (1999); Kissel *et al.* (1998) and Camann *et al.* (2000). The 50% hand-to-mouth transfer efficiency has been used by the CPSC (1997) in estimating hand-to-mouth exposure to lead from polyvinyl chloride products, and by the U.S. EPA's Office of Pesticide Programs as a default value for hand-to-mouth exposure to pesticides (U.S. EPA, 2001).

4.7.3.5 Zartarian et al. (2005)

Zartarian *et al.* (2005) working under a contract from the U.S. EPA derived a statistical distribution for hand-to-mouth transfer efficiency for arsenic from chromated copper arsenate (CCA)-treated wood. Hand-to-mouth transfer efficiency is defined as the fraction of chemical mass that enters the mouth and remains in the mouth as a result of one hand-to-mouth contact. The value of 50% was used as the lower bound on the transfer efficiency, with 100% assigned as the upper bound and the mode of distribution set to 75%. The resulting fitted beta distribution of the hand-to-mouth transfer efficiency for arsenic had a mean value of 78% and a 75th percentile value of 84.9% per hand-to-mouth contact.

4.7.3.6 OEHHA (2008)

OEHHA (2008) published a lead exposure guideline for calculating the hand-to-mouth transfer of lead from the use of fishing tackle in recreational fishing. The guideline examined both direct and indirect hand-to-mouth activities. No data were available from the scientific literature on the amount of lead transferred from the hand to the mouth as a result of handling fishing tackle products, but data from two studies (Camann *et al*, 2000; Kissel *et al*, 1998) were found to be useful. The study by Camann *et al*. (2000) provides data on the removal of three pesticides from the hands of three adults. The study by Kissel *et al.* (1998) provides estimates on the total soil loading on the hand,

and its transfer to the mouth from particular parts of the hand (i.e., thumb; two fingers; palm) in four adults. After reviewing the data from these and other studies, OEHHA (2008) selected a value of 50% as the direct, and 25% as the indirect hand-to-mouth transfer factors for lead in fishing tackle products for adults.

U.S. EPA (2002) concluded from the data of Reed *et al.* (1999) and Zartarian *et al.* (1998) that hand-to-mouth contacts of 9 contacts/hour was a reasonable estimate for children 2 to 6 years old. Since then other published studies (Black *et al*., 2005 and Ko *et al*., 2007) reported that the hand-to-mouth value of 9 contacts/hour probably underestimates the frequency of children's hand-to-mouth activity and the frequency could be over 20 contacts/hour. OEHHA (2008) selected 9 contacts/hour as the average estimate, and 20 as the upper bound estimate of direct hand-to-mouth contact frequency for adults during fishing in contact with lead fishing tackle products.

4.7.3.7 Xue et al. (2007)

A meta-analysis was conducted by Xue and colleagues (2007) to examine hand-to-mouth frequency based on study, age groups, gender, and location (indoor vs. outdoor). Data were gathered from 9 studies (Zartarian *et al.* 1998; Reed *et al.* 1999; Leckie *et al.* 2000; Freeman *et al.* 2001; Greene, 2002; Tulve *et al.* 2002; Hore, 2003; Black *et al.* 2005; Beamer *et al.* 2008). The combined studies represent 429 subjects and more than 2,000 hours of behavior observations. To pool and analyze the data from these studies collectively, Xue *et al* (2007) contacted the authors of the 9 studies to obtain and clarify needed and missing data for the analysis.

Results of the analysis indicate that age and location are important for hand-to-mouth frequency, but not gender. As age increases, both indoor and outdoor hand-to-mouth frequencies decrease, and this behavior is higher indoors than outdoors. Average indoor hand-to-mouth frequency ranged from 6.7 to 28.0 contacts/hour, with the lowest value corresponding to the 6 years to <11 years age group and the highest value corresponding to the 3 months to <6 months group. Average outdoor hand-to-mouth frequency ranged from 2.9 to 14.5 contacts/hour, with the lowest value corresponding to the 6 years to <11 years age group and the highest value corresponding to the 6 months to <12 months group. For the 3 months to < 6 months age group, outdoor hand-to-mouth contact frequency data were not available.

The study is an important effort to provide data on hand-to-mouth contact frequency by indoor/outdoor location and age groups based on the recommendations by the U.S. EPA (2005) for assessing childhood exposures. However, it did not analyze or collect data on other mouthing behaviors such as object-to-mouth. Also, data for older children, ages 11 and above, are not included; they are likely to have very different behaviors from the younger children.

Table 4.17 Hand-to-Mouth Frequency (contacts/hour) in Children

Age Group	No. of Observations	Mean	Std Dev	P25	P50	P75	P95
		INDOORS					
3m to < 6m	23	28	21.7	8.0	23.0	48.0	65.0
6m to < 12m	119	18.9	17.4	6.6	14.0	26.4	52.0
1y to < 6y[a]	575	16.2	-	4.5	11.1	22.1	53.1
6y to < 11y	14	6.7	5.5	2.4	5.7	10.2	20.6
		OUTDOORS					
3m to < 6m	0	-	-	-	-	-	-
6m to < 12m	10	14.5	12.3	7.6	11.6	16.0	46.7
1y to < 6y[a]	133	8.7	-	1.1	5.1	11.6	32.0
6 to < 11y	15	2.9	4.3	0.1	0.5	4.7	11.9
		COMBINED					
3m to < 6m	23	28	21.7	8.0	23.0	48.0	65.0
6m to < 12m	129	18.6	-	6.7	13.8	25.6	51.6
1y to < 6y[a]	708	14.8	-	3.8	10.0	20.2	49.1
6y to < 11y	29	4.7	-	1.2	3.0	7.4	16.1

Adapted from Xue *et al.*, 2007; results are from 9 studies using Weibull distributions.
[a] Three age groups, 1y to < 2 y, 2y to <3y, and 3y to <6y, combined.

4.7.4 Extrapolation of Soil Ingestion from Hand-to-Mouth Contact

U.S. EPA (2008) in their *Child-Specific Exposure Factors Handbook* recommends 100 mg/d as the central tendency value for daily soil and dust ingestion in children 1 year to <6 years. The actual sum (soil and dust) is 110 mg/d but rounded to 100 mg/d (to one significant figure) (U.S. EPA, 2008). In the absence of data that can be used to develop soil and dust recommendations for children aged 6 to <11 years, 11 to <16 years and 16 to <21 years, U.S. EPA (2008) recommended using 100 mg/d as the central tendency value for children aged 6 to <21 years.

Using the mean weighed average value of 110 mg/day for soil and dust ingestion for the age group 1 to <6 years old (from Table 4.13 derived from the 2008 U.S. EPA document), and assuming this age group has combined indoor and outdoor hand-to-mouth contacts of 14.8/hour (from Table 4.17), soil ingestion in other age groups can be estimated (Table 4.18).

OEHHA (2008) selects 9 and 20 as the average and upper bound estimates, respectively, of direct hand-to-mouth contact frequency for adults from the use of lead tackle in recreational fishing. Using the same extrapolation procedure above, the mean and the upper bound soil ingestion estimates were obtained. The combined soil and dust ingestion rate estimated from Xue *et al.* (2007) data for children aged 6 months to < 12 months is higher than that provided by the U.S. EPA (2008) – 133 mg/d versus 60 mg/d, respectively. We believe that the value of 133 mg/d better reflects the soil and dust ingestion rate in children aged 6 months to < 12 months because children in this age group are known to have much higher hand-to-mouth contact behavior as they explore their environment (Xue *et al.* 2007).

Table 4.18 Soil and Dust Ingestion Rates (mg/day) Extrapolated from Xue et al. (2007) Hand-to-Mouth Contact Data to Three Age Groups

Age Groups	Mean	P95
3m to < 6m	NC[a]	NC
6m to < 12m	133	370
1y to < 6y	106	352
6 to < 11y	34[b]	115[b]
Adult	64	143

[a] Not calculated as there is no hand-to-mouth contact in this group
[b] Low confidence level for this number due to low number of observations

OEHHA supports the U.S. EPA (2008) recommendations of 100 mg/day as the central tendency of the combined soil and dust ingestion rate for children aged 1 to <6 years. This number was rounded down from the actual number of 110 mg/d. Using 110 mg/day for soil and dust ingestion for the age group 1 to <6 years old (Table 4-13), and assuming this group has combined indoor and outdoor hand-to-mouth contacts of 14.8/hour (from Figure 4-17), soil and dust ingestion in other age groups are extrapolated from hand-to-mouth data (Table 4-18). The value for the 6 to <11 year old group is not used because of the low number of hand-to-mouth observations in this group. The soil ingestion values for adults and children (mg/day) estimated for the various age groups are shown in Table 4.19.

Table 4.19 Soil Ingestion Estimates for Adults and Children (mg/day)*

Age Groups (years)	Mean (mg/day)	95th percentile (mg/day)
3rd Trimester[a]	**50**	**200**
0 < 2	150	400
2<9	100	400
2<16	100	400
9<16	100	400
16<30	50	200
30>70	50	200
PICA children	5000	-
PICA adult	NR[b]	-

[a] Assumed to be the mother's soil ingestion rate (adult age 16 <30)
[b] No recommendation
* Soil includes outdoor settled dust

4.8 References

ATSDR. 2001. Summary report for the ATSDR soil-pica workshop. Agency for Toxic Substances and Disease Control Registry, US Centers for Disease Control, Atlanta, GA.

Auyeung W, Canales RA, Beamer P, Ferguson AC, Leckie JO. 2006. Young children's hand contact activities: An observational study via videotaping in primarily outdoor residential settings. J Expo Sci Environment Epidemiol 16(5): 434-446.

AuYeung W, Canales RA, Leckie JO. 2008. The fraction of total hand surface area involved in young children's outdoor hand-to-object contacts. Environ Res 108(3): 294-299.

Beamer P, Key ME, Ferguson AC, Canales RA, Auyeung W, James O. 2008. Quantified activity pattern data from 6 to 27-month-old farmworker children for use in exposure assessment. Environ Res 108(2): 239-246.

Beamer PI, Canales RA, Bradman A, Leckie JO. 2009. Farmworker children's residential non-dietary exposure estimates from micro-level activity time series. Environ Int 35(8): 1202-1209.

Behrman LE, Vaughan VCI. 1983. Textbook of Pediatrics. Philadelphia, PA: W.B. Saunders Company.

Beyer LA, Seeley MR, Beck BD. 2003. Evaluation of potential exposure to metals in laundered shop towels. International Nonwovens Journal Winter: 22-37.

Binder S, Sokal D, Maughan D. 1986. Estimating soil ingestion - the use of tracer elements in estimating the amount of soil ingested by young children. Arch Environ Health 41(6): 341-345.

Black K, Shalat SL, Freeman NCG, Jimenez M, Donnelly KC, Calvin JA. 2005. Children's mouthing and food-handling behavior in an agricultural community on the US/Mexico border. J Expo Anal Environment Epidemiol 15(3): 244-251.

Bruhn CM, Pangborn RM. 1971. Reported incidence of pica among migrant families. J Am Diet Assoc 58(5): 417-420.

Calabrese EJ. 2003. Letter to Mr. Kevin W. Holtzelaw. Exhibit E.1 in GE-Pittsfield/Housatonic River Site, Rest of River (GECD850) Corrective Measures Study Report submitted to U.S. EPA, Region 1.

Calabrese EJ, Barnes R, Stanek EJ, 3rd, Pastides H, Gilbert CE, Veneman P, et al. 1989. How much soil do young children ingest: an epidemiologic study. Regul Toxicol Pharmacol 10(2): 123-137.

Calabrese EJ, Stanek EJ. 1992. What proportion of household dust is derived from outdoor soil? Soil Sediment Contam 1(3): 253-263.

Calabrese EJ, Stanek EJ. 1993. Soil pica - not a rare event. Journal of Environmental Science and Health Part A - Environ Sci Engineer Toxic Hazard Subs Control 28(2): 373-384.

Calabrese EJ, Stanek EJ, 3rd. 1995. Resolving intertracer inconsistencies in soil ingestion estimation. Environ Health Perspect 103(5): 454-457.

Calabrese EJ, Stanek EJ, Barnes R. 1997. Soil ingestion rates in children identified by parental observation as likely high soil ingesters. J Soil Contam 6(3): 271-279.

Calabrese EJ, Stanek EJ, Barnes R, Burmaster DE, Callahan BG, Heath JS, et al. 1996. Methodology to estimate the amount and particle size of soil ingested by children: implications for exposure assessment at waste sites. Regul Toxicol Pharmacol 24(3): 264-268.

Calabrese EJ, Stanek EJ, Gilbert CE. 1991. Evidence of soil-pica behavior and quantification of soil ingested. Hum Exper Toxicol 10(4): 245-249.

Calabrese EJ, Stanek EJ, Gilbert CE, Barnes RM. 1990. Preliminary adult soil ingestion estimates: results of a pilot study. Regul Toxicol Pharmacol 12(1): 88-95.

Calabrese EJ, Stanek EJ, 3rd, Pekow P, Barnes RM. 1997. Soil ingestion estimates for children residing on a superfund site. Ecotoxicol Environ Saf 36(3): 258-268.

Calabrese EJ, Stanek ES. 1992. Distinguishing outdoor soil ingestion from indoor dust ingestion in a soil pica child. Regul Toxicol Pharmacol 15(1): 83-85.

Camann DE, Majumdar TK, Geno PW. 2000. Evaluation of saliva and artificial salivary fluids for removal of pesticide residues from human skin. Research Triangle Park, NC: U.S. EPA, National Exposure Research Laboratory, Research Triangle Park, NC.

Clausing P, Brunekreef B, van Wijnen JH. 1987. A method for estimating soil ingestion by children. Int Arch Occup Environ Health 59(1): 73-82.

CPSC. 1997. CPSC Staff Report on Lead and Cadmium in Children's Polyvinyl chloride (PVC) Products. Consumer Product Safety Commission, Washington, DC.

CPSC. 2003. Briefing Package. Petition to ban chromated copper arsenate (CCA)-treated wood in playground equipment. Consumer Product Safety Commission, Washington, DC.

Danford DE. 1982. Pica and nutrition. Ann Rev Nutrition 2: 303-322.

Davis S, Mirick DK. 2006. Soil ingestion in children and adults in the same family. J Expo Sci Environ Epidemiol 16(1): 63-75.

Davis S, Waller P, Buschbom R, Ballou J, White P. 1990. Quantitative estimates of soil ingestion in normal children between the ages of 2-years and 7-years - population-based estimates using aluminum, silicon, and titanium as soil tracer elements. Arch Environ Health 45(2): 112-122.

Dube EM, Boyce CP, Beck BD, Lewandowski T, Schettler S. 2004. Assessment of potential human health risks from arsenic in CCA-treated wood. Hum Ecol Risk Assess 10(6): 1019-1067.

Feldman MD. 1986. Pica: current perspectives. Psychosomatics 27(7): 519-523.

Ferguson AC, Canales RA, Beamer P, Auyeung W, Key M, Munninghoff A, et al. 2006. Video methods in the quantification of children's exposures. Journal of Expo Sci Environ Epidemiol 16(3): 287-298.

Freeman NCG, Hore P, Black K, Jimenez M, Sheldon L, Tulve N, et al. 2005. Contributions of children's activities to pesticide hand loadings following residential pesticide application. J Expo Anal Environ Epidemiol 15(1): 81-88.

Freeman NCG, Jimenez M, Reed KJ, Gurunathan S, Edwards RD, Roy A, et al. 2001. Quantitative analysis of children's microactivity patterns: The Minnesota Children's Pesticide Exposure Study. J Expo Anal Environ Epidemiol 11(6): 501-509.

Greene MA. 2002. Mouthing times among young children from observational data. U.S. Consumer Product Safety Commission, Bethesda, MD.

Hawley JK. 1985. Assessment of health risk from exposure to contaminated soil. Risk Anal 5(4): 289-302.

Hemond HF, Solo-Gabriele HM. 2004. Children's exposure to arsenic from CCA-treated wooden decks and playground structures. Risk Anal 24(1): 51-64.

Hogan K, Marcus A, Smith P, White P. 1998. Integrated exposure uptake biokinetic model for lead in children: Empirical comparisons with epidemiologic data. Environ Health Perspect 106: 1557-1567.

Hore P, Robson M, Freeman N, Zhang J, Wartenberg D, Ozkaynak H, et al. 2005. Chlorpyrifos accumulation patterns for child-accessible surfaces and objects and urinary metabolite excretion by children for 2 weeks after crack-and-crevice application. Environ Health Perspect 113(2): 211-219.

Illingworth RS. 1983. The Normal Child. New York, NY: Churchill Livingstone.

IPSC. 1982. Environmental Health Criteria 24: Titanium. International Programme on Chemical Safety. Geneva, Switzerland: United Nations Environment Programme, the International Labour Organization, and the World Health Organization.

Kaplan HI, Sadock BJ. 1985. Comprehensive Textbook of Psychiatry. Baltimore, MD: Williams and Wilkins.

Kimbrough RD, Falk H, Stehr P, Fries G. 1984. Health implications of 2,3,7,8-tetrachlorodibenzodioxin (TCDD) contamination of residential soil. J Toxicol Environ Health 14(1): 47-93.

Kissel JC, Shirai JH, Richter KY, Fenske RA. 1998. Empirical investigation of hand-to-mouth transfer of soil. Bull Environ Contam Toxicol 60(3): 379-386.

Ko S, Schaefer PD, Vicario CM, Binns HJ. 2007. Relationships of video assessments of touching and mouthing behaviors during outdoor play in urban residential yards to parental perceptions of child behaviors and blood lead levels. J Expo Sci Environ Epidemiol 17(1): 47-57.

Lacey EP. 1990. Broadening the perspective of pica: literature review. Public Health Reports 105(1): 29-35.

Leckie JO, Naylor KA, Canales RA, Ferguson AC, Cabrera NL, Hurtado AL, et al. 2000. Quantifying Children's Microlevel Activity Data from Existing Videotapes. Stanford University, Palo Alto, CA. Reference #U2F112OT-RT-99-001182

Lourie RS. 1963. The Contributions of Child Psychiatry to the Pathogenesis of Hyperactivity in Children. Clinical Proceedings – Children's Hospital of the District of Columbia 19: 247-253.

OEHHA. 2008. Guideline for hand-to-mouth transfer of lead through exposure to fishing tackle products. Reproductive and Cancer Hazard Assessment Branch, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA. Proposition 65 Interpretive Guideline No. 2008-001

Reed KJ, Jimenez M, Freeman NC, Lioy PJ. 1999. Quantification of children's hand and mouthing activities through a videotaping methodology. J Expo Anal Environ Epidemiol 9(5): 513-520.

Sayetta RB. 1986. Pica: an overview. American Family Physician 33(5): 181-185.

Sedman RM, Mahmood RJ. 1994. Soil ingestion by children and adults reconsidered using the results of recent tracer studies. Air Waste 44(2): 141-144.

Stanek EJ, Calabrese EJ. 1994. Bias and the detection limit model for soil ingestion. Soil Sed Contam 3(2): 183-189.

Stanek EJ, Calabrese EJ. 1995a. Daily estimates of soil ingestion in children. Environ Health Perspect 103(3): 276-285.

Stanek EJ, Calabrese EJ. 1995b. Soil ingestion estimates for use in site evaluations based on best tracer method. Hum Ecol Risk Assess 1: 133-156.

Stanek EJ, Calabrese EJ. 2000. Daily soil ingestion estimates for children at a superfund site. Risk Anal 20(5): 627-635.

Stanek EJ, Calabrese EJ, Barnes R, Pekow P. 1997. Soil ingestion in adults - Results of a second pilot study. Ecotoxicol Environ Safety 36(3): 249-257.

Stanek EJ, Calabrese EJ, Barnes RM. 1999. Soil ingestion estimates for children in Anaconda using trace element concentrations in different particle size fractions. Hum Ecol Risk Assess 5(3): 547-558.

Stanek EJ, Calabrese EJ, Mundt K, Pekow P, Yeatts KB. 1998. Prevalence of soil mouthing/ingestion among healthy children aged 1 to 6. J Soil Contam 7(2): 227-242.

Stanek EJ, Calabrese EJ, Zorn M. 2001. Soil ingestion distributions for Monte Carlo risk assessment in children. Hum Ecol Risk Assess 7(2): 357-368.

Thompson KM, Burmaster DE. 1991. Parametric distributions for soil ingestion by children. Risk Anal 11(2): 339-342.

Tulve NS, Suggs JC, McCurdy T, Hubal EAC, Moya J. 2002. Frequency of mouthing behavior in young children. J Expo Anal Environ Epidemiol 12(4): 259-264.

U.S. EPA. 1985. Health Assessment Document for Polychlorinated Dibenzo-p-Dioxin. U.S. Environmental Protection Agency, Office of Research and Development, Cincinnati, OH. EPA600/8-84-014F

U.S. EPA. 1989. Risk Assessment Guidance for Superfund. Volume 1. Human Health Evaluation Manual (Part A, Baseline Risk Assessment). U.S. Environmental Protection Agency, Washington, DC. EPA/540/1-89/002

U.S. EPA. 1991. OSWER Directive 9285.6-03 Human Health Evaluation Manual. Supplemental Guidance: Standard Default Exposure Factors. U.S. Environmental Protection Agency, Washington, DC. PB91-921314

U.S. EPA. 2001. Draft Protocol for Measuring Children's Non-Occupational Exposures to Pesticides by All Relevant Pathways. U.S. Environmental Protection Agency, Office of Research and Development, Research Triangle Park, NC. EPA/600/R-03/026

U.S. EPA. 2005. Summary report of the U.S. EPA colloquium on soil/dust ingestion rates and mouthing behavior for children and adults. U.S. Environmental Protection Agency, Washington, DC. EP-C-04-027

U.S. EPA. 2005. Guidance on selecting age groups for monitoring and assessing childhood exposures to environmental contaminants. U.S. Environmental Protection Agency, Washington, DC. EPA/630/P-03/003F

U.S. EPA. 2008. Child-Specific Exposure Factors Handbook (Final Report), Chapter 5: Ingestion of Soil and Dust. U.S. Environmental Protection Agency, Washington, DC. EPA/600/R-05/096F

Van Wijnen JH, Clausing P, Brunekreef B. 1990. Estimated soil ingestion by children. Environ Res 51(2): 147-162.

Vermeer DE, Frate DA. 1979. Geophagia in rural Mississippi - environmental and cultural contexts and nutritional implications. Am J Clin Nutrition 32(10): 2129-2135.

Wong MS. 1988. The Role of Environmental and Host Behavioural Factors in Determining Exposure to Infection with Ascaris Lumbricoldes and Trichlura. Ph.D. Thesis. Faculty of Natural Sciences, University of West Indies, West Indies.

Xue JP, Zartarian V, Moya J, Freeman N, Beamer P, Black K, et al. 2007. A meta-analysis of children's hand-to-mouth frequency data for estimating nondietary ingestion exposure. Risk Anal 27(2): 411-420.

Zartarian VG, Ferguson AC, Leckie JO. 1997. Quantified dermal activity data from a four-child pilot field study. J Expo Anal Environ Epidemiol 7(4): 543-552.

Zartarian VG, Ozkaynak H, Burke JM, Zufall MJ, Rigas ML, Furtaw EJ, Jr. 2000. A modeling framework for estimating children's residential exposure and dose to chlorpyrifos via dermal residue contact and nondietary ingestion. Environ Health Perspect 108(6): 505-514.

Zartarian VG, Xue J, Ozkaynak HA, Dang W, Smith L, Stallings C. 2005. A Probabilistic Exposure Assessment for Children who Contact CCA-Treated Playsets and Decks Using the Stochastic Human Exposure and Dose Simulation Model for the Wood Preservation Scenario (SHEDs-WOOD), Final Report. U.S. Environmental Protection Agency, Washington, DC. EPA/600/X-05/009

5 Breast Milk Intake Rates

5.1 Terminology and Nomenclature

In this chapter, we review breast milk intake estimates reported in the published literature. In the prior version of these guidelines, published rates as well as unpublished rates derived by OEHHA were presented. The OEHHA derived rates have been updated and revised to reflect breastfeeding practices most likely to occur in the United States (U.S.) (i.e., following the American Academy of Pediatrics recommendations). The revised OEHHA derived rates have been published in a peer-reviewed journal (Arcus-Arth et al, 2005) and are presented along with other published rates in these guidelines.

Specific terms and definitions have been adopted for use throughout this chapter (Table 5.1), because different and sometimes contradictory terms for various breastfeeding patterns are used in the literature.

Table 5.1 Breastfeeding Terminology[a]

Term	Definition
Fully breastfed	
Exclusively breastfed	Breast milk is sole source of calories.
Almost exclusively breastfed	Breast milk is primary if not sole milk source with no significant calories from other liquid or solid food sources.
Predominantly breastfed	Breast milk is the primary if not sole milk source with significant calories from other liquid or solid food sources.
Partially breastfed	Combined breast milk and other milk intake where non-breast milk (e.g., formula) is a significant milk source whether or not the infant is consuming significant calories from other liquid or solid food sources.
Token breastfeeding	Minimal, irregular or occasional breastfeeding contributing minimal nutrition and few calories.
Extended breastfeeding	Breastfeeding beyond 12 months of age.
Weaning	Discontinuation of breastfeeding.

[a] Adapted from Labbok and Krasovec (1990)

These terms are important for our discussion in this section because breastfeeding patterns are important determinants of breast milk intake rates.

Fully breastfed infants are those that receive breast milk as the primary, if not sole, source of milk. This category encompasses three specific patterns of breastfeeding. Thus, the term "fully breastfed" is probably most often applied to the entire lactation period (0-12 months). For example, an infant who was exclusively breastfed for the first 6 months, then predominantly breastfed from 6 through 12 months, would be considered fully breastfed for the lactation period. We use the term "almost exclusively breastfed" particularly for the common practice of exclusive breastfeeding during the day with a small bottle of formula fed at night. Older infants who are breastfed and do not receive significant amounts of formula (or other non-breast milk) but do receive supplementary solid foods would fit into the category of "predominantly breastfed." Partially breastfed infants, like fully breastfed infants, receive some breast milk but unlike fully breastfed infants they also receive significant amounts of milk, or formula from non-breast milk sources.

A few words about units and nomenclature are provided to avoid confusion. In toxicology and pharmacology "dose" is typically expressed as the amount received over time divided by body weight (e.g., mg/kg-day). Analogously, breast milk intake rates can be expressed as the amount received by the infant over time divided by the infant's body weight. Daily breast milk intake rate (e.g., g/kg BW-day) is the most commonly used unit of measure. If multiple days of breast milk intake rate for a single infant are averaged together, the result is the "average daily breast milk intake rate." This averaging is over time rather than over individuals. This term is useful for characterizing an average intake over time (e.g., over the first 6 months of life).

A final note is that the means and standard deviations (SDs) reported in these guidelines are arithmetic means and arithmetic SDs, unless otherwise indicated.

5.2 Recommendations

OEHHA recommends the following to estimate dose to the infant through breast milk.

5.2.1 Default Point Estimate for Daily Breast Milk Intake During the First Year

For the default point estimate approach to assess dose and risk from breast milk intake by breastfed infants during the first year, OEHHA recommends using the mean and high-end estimates presented in Table 5.2. The average and high end point estimates are 101 and 139 g/kg BW *day.

Table 5.2 Point Estimates of Breast Milk Intake for Breastfed Infants

Infant Group	Intake (g/kg-day)
Fully breastfed over the first year (i.e., fed in accordance with AAP recommendations)[1]	
Mean	101
90th percentile	130
95th percentile	139
Exclusively breastfed during first year[2]	
Mean	113
90th percentile	141
95th percentile	149
Fully breastfed over first 6 months (i.e., fed in accordance with AAP recommendations) [1]	
Mean	130
90th percentile	138
95th percentile	165

[1] AAP = dataset based on American Academy of Pediatrics (1997) infant feeding recommendations;

[2] EBF = dataset of exclusively breastfed infants

As discussed in Section 5.1, fully breastfed infants are those that receive breast milk as the primary, if not sole, source of milk. Thus, the term "fully breastfed" is probably most often applied to the entire lactation period (0-12 months). An infant who was exclusively breastfed for the first 6 months, then predominantly breastfed from 6 through 12 months, would be considered fully breastfed for the lactation period. Exclusively breastfed infants are those in which breast milk is the sole source of calories.

5.2.2 Stochastic Approach to Breast Milk Intake Among Individuals During the First Year of Life

For a stochastic analysis of exposure and dose through the breast milk intake pathway, a normal distribution with a mean of 101 g/kg-day and standard deviation 23 g/kg-day, is recommended as a distribution for breast milk intake (Table 5.3).

Table 5.3 Recommended Breast Milk Intake Rates Among Breastfed Infants (Averaged Over an Individual's First Year of Life)

	Mean (SD)	Percentile							
		5	10	25	50	75	90	95	99
Intake (g/kg-day)	101 (23)	62	71	85	101	116	130	139	154

The recommended values for average and high end breast milk consumption rates are the mean and 95th percentiles (101 and 139 g/kg BW -day) for fully breastfed infants.

The recommended parametric model for stochastic risk assessment is a normal distribution with a mean and standard deviation of 101 ± 23

5.2.3 Consideration of Variable Age of Breastfeeding Mothers

Because some environmental toxicants continue to accumulate, older primiparous mothers could excrete higher concentrations of the toxicant in breast milk than younger mothers could when daily intake is constant over time. For example, Hedley et al (2007) reported that breast milk concentrations of POPs increased in a population of Asian women by 1.45 pg/g-fat/yr. Incorporating a distribution or range of age among breastfeeding mothers into the risk assessment is a refinement that could be considered in the future.

5.2.4 Analysis for Population-wide Impacts from Breast Milk Exposure

If the risk assessor is evaluating a population-wide risk (e.g., for the purpose of developing a range of cancer burden estimates from this pathway), it may be appropriate to incorporate information on the percent of the infant population that is breastfed at various ages. Information on the prevalence of breastfeeding by age of infant in California from the National Immunization Survey (NIS) specific to California is available in Appendix 5A, Table 5A-11 for this purpose. Alternatively, values in Table 5A-17 could be used. This information should be re-evaluated periodically to take into account recent trends in breastfeeding and the outcome of the breastfeeding promotion policies of the last decade.

5.3 Conceptual Framework for Variable Breast Milk Intake Rates

The Hot Spots program provides a tiered approach to risk assessment. Point estimate and stochastic approaches are available. The stochastic approach uses probability distributions for variates with sufficient data to estimate variability. The point estimate approach for the breast milk pathway uses average and high-end breast milk consumption values. Data on the distribution of breast milk intake rates allow selection of point estimates that represent average breast milk consumption and a specified percentile of high-end consumption. To incorporate the variability of breast milk intake into the infant dose of toxicant from breast milk, we use a stochastic approach to characterize parameters related to the breast milk pathway.

The data set that we use for breast milk intake rate distributions includes 130 infants for whom there are at least two measurement time points separated by at least 7 days during the lactation period. This is an unusually robust data set for evaluating variability in breast milk intake rates. The repeated measures help ensure that typical intake over time is captured, thus reducing the effect of intraindividual variability on the distribution of values. Further, milk intake measurements and body weight for individual infants are included and, therefore, breast milk intake can be normalized to body weight for each infant. Breast milk intake is correlated with infant body weight (e.g., large babies

consume greater amounts of milk than small ones) and thus the variability simply due to body weight can be eliminated.

The correlation of intake and body weight is taken into account by normalizing intake by body weight for each individual infant. That is, for each infant, their daily intake at that measurement is divided by his/her body weight at that measurement to give intake in units of g/kg-day. Because larger infants consume greater amounts of milk, normalizing to body weight reduces much of the variability due to differences in body weight among infants.

Interindividual variability is explicitly addressed through the distributional approach used in these guidelines. A distribution of intake rate quantifies the probability of the array of intake rate values in the population. This describes variability between individuals in the population.

Intraindividual variability is addressed by allowing intake to be a function of time (e.g., see Arcus-Arth et al., 2005; Burmaster and Maxwell, 1993), thus taking into account variability of an individual's intake over time. Intraindividual variability can also be addressed by assessing the impact of different methods of averaging over time (e.g., Arcus-Arth et al., 2005).

Exposure through mother's milk ingestion ($Dose_m$) is a function of the average substance concentration in mother's milk and the amount of mother's milk ingested. The minimum pathways that the nursing mother is exposed to include inhalation, soil ingestion and dermal, since the chemicals evaluated by the mother's milk pathway are multipathway chemicals. Other pathways may be appropriate depending on site conditions (e.g., presence of vegetable gardens or home grown chickens or the fish consumption). The nursing mother in the mother's milk pathway is not herself subject to the mother's milk pathway. The summed average daily dose (mg/kg BW-day) from all pathways is calculated for the nursing mother using equations in the other chapters of this document.

The general algorithm for estimating dose to the infant via the mother's milk pathway is as follows:

$$Dose_m = C_m * BMI_{bw} * EF * (1x10^{-3}) \qquad \text{(Eq 5-1)}$$

where:

$Dose_m$ = Dose to the infant through ingestion of mother's milk (mg/kg BW/day)

C_m = Concentration of contaminant in mother's milk is a function of the mother's exposure through all routes and the contaminant half-life in the body (mg/kg milk). Various equations for estimating Cm are presented in Appendix J

BMI_{bw} = Daily breast-milk ingestion rate (g-milk/kg BW/day). See Table 5.2 for point estimates. See Table 5.3 for distribution for Tier 3 stochastic risk assessments.

EF = Frequency of exposure, unitless, (days/365 days)

$1x10^{-3}$ = Conversion factor (g to kg for milk,)

The exposure frequency (EF) is the fraction of time the infant is exposed daily during the first year (i.e., 365 days) of breast-feeding. Thus, the EF is set at 1. For cancer risk assessment, the risk via the mother's milk pathway ($RISKm_{(0<2\ yr)}$) occurs only during the first year in the 0<2 age group.

The risk is calculated for this age group using the appropriate, unitless, age sensitivity factor (ASF) of 10, (see OEHHA, 2009) and the chemical-specific cancer potency factor (CPF), expressed in units of $(mg/kg\text{-}day)^{-1}$.

$$RISKm_{(0<2\ yr)} = Dose_m * CPF * ASF * ED * 0.5 \qquad \textbf{(Eq. 5-2)}$$

The cancer risk, $RISKm_{(0<2\ yr)}$ is the predicted number of expected cases of cancer over a lifetime as a result of the exposure (e.g., expressed as 1×10^{-6} or 1 case per million people exposed)

Exposure duration (ED) is the number of years within the age grouping, which is 2 years for the 0<2 year age group. Since risk for the mother's milk pathway is assessed only during the first year of the 0<2 year age group, a 0.5 adjustment factor is included in Eq. 5-2. The risk from other exposure pathways (e.g., the inhalation pathway) would not include this factor in the 0<2 age group.

To determine lifetime cancer risks (i.e., 70 years), the total risk for the 0<2 age group is then summed across the total risk of the other age groups:

$$RISK_{(lifetime)} = RISK_{(3rdtri)} + RISK_{(0<2\ yr)} + RISK_{(2<16\ yr)} + RISK_{(16\text{-}70yr)} \qquad \textbf{(Eq. 5-3)}$$

As explained in Chapter 1, different age groups for assessing risk are needed due to different ASFs for each group. We also need to accommodate cancer risk estimates for the average (9 years) and high-end (30 years) length of time at a single residence, as well as the traditional 70-year lifetime cancer risk estimate. For example, assessing risk in a 9-year residential exposure scenario assumes exposure during the most sensitive period, from the third trimester to 9 years of age and would be presented as such:

$$RISK_{(9\text{-}yr\ residency)} = RISK_{(3rdtri)} + RISK_{(0<2\ yr)} + RISK_{(2<9\ yr)} \qquad \textbf{(Eq. 5-4)}$$

For the 30-year residential exposure scenario, the risk for 2<16 and 16<30 age groups would be added to the risks from third trimester and 0<2 exposures. For the 70-year residential exposure scenario risk, Eq 5-3 would apply.

The risk algorithm for the stochastic approach and for the point estimate approach is the same. In the stochastic approach, the distribution of mother's milk consumption is reflected as a distribution of dose to the infant.

The chemicals with human milk transfer coefficients (Tco_{hm}) to be analyzed in the breast milk exposure pathway are described in Appendix J.

5.3.1 Transfer Coefficients for Chemicals From Mother into Milk

Tco_{hm} represent the transfer relationship between the chemical concentration found in milk and the mother's chronic daily dose (i.e. concentration (µg/kg-milk)/dose (µg/day) under steady state conditions. Transfer coefficients can be applied to the mother's chronic daily dose estimated by the Hot Spots exposure model for all applicable exposure pathways to estimate a Cm for a specific chemical concentration in her milk by equation 5-5. Appendix J has additional detail of the derivation of transfer coefficients for specific chemicals.

$$Cm = [DOSEair + DOSEwater + DOSEfood + DOSEsoil + DOSEdermal] \times Tco_{hm} \times BW \quad \textbf{(Eq. 5-5)}$$

where:
- DOSEair = dose to the mother through inhalation (Eq 3-1) (mg/kg/day)
- Dwi = dose though drinking water ingestion (mg/kg/day)
- DOSEfood = dose through ingestion of food sources (Eq 7-1) (mg/kg/day)
- DOSEsoil = dose through incidental ingestion of soil (Eq 4-1) (mg/kg/day)
- DOSEdermal = dose from dermal absorption from contaminated soil (Eq 6-1) (mg/kg/day)
- DOSEwater = dose through ingestion of surface water (Eq 8-2) (mg/kg/day)
- Tco_{hm} = transfer coefficient (see Table 5-4) (day/kg-milk)
- BW = body weight of the mother (default = 70.7 (kg)

However, if bio-transfer information is available for an individual exposure route, route-specific Tcos can be developed resulting in a modification of Eq. 5.5:

$$Cm = [(DOSEair \times Tco_{mi}) + (DOSEwater \times Tco_{mw}) + DOSEfood \times Tco_{mf}) + (DOSEsoil \times Tco_{ms}) + (DOSEdermal \times Tco_{md}] \times BW \quad \textbf{(Eq. 5-6)}$$

where:
- Tcomi = biotransfer coefficient from inhalation to mother's milk (day/kg-milk)
- Tcomw = biotransfer coefficient from drinking water to mother's milk (day/kg-milk)
- Tcomf = biotransfer coefficient from food to mother's milk (day/kg-milk)
- Tcoms = biotransfer coefficient from incidental soil ingestion to mother's milk (day/kg-milk)
- Tcomd = biotransfer coefficient from dermal absorption from contaminated soil (day/kg-milk)

Estimates of toxicant bio-transfer to breast milk are chemical-specific. Table 5.4 shows the transfer coefficients for dioxin-like compounds, carcinogenic PAHs and lead that OEHHA has estimated from data found in the peer-reviewed literature. One key factor that plays a role in the difference between oral and inhalation transfer coefficient (e.g., for PAHs) is first pass metabolism which is lacking in dermal and inhalation exposures. Thus, for simplicity, OEHHA applies the transfer coefficients from inhalation to the dermal absorption pathway for lead and PAHs. For lead, we are using the inhalation Tco for all the other pathways of exposure to the mother. Likewise for PCDD/Fs and

dioxin-like PCBs, we are using the oral Tco for the other pathways of exposure to the mother in Eq. 5-7.

$$Cm = [(D\_inh \times Tco_{m\_inh}) + (D\_ing \times Tco_{m\_ing})] \times BW \quad \textbf{(Eq. 5-7)}$$

where: D_ing = the sum of DOSEfood + DOSEsoil + DOSEwater through ingestion (mg/kg-BW-day)
D_inh = the sum of DOSEair + DOSEdermal through inhalation and dermal absorption (mg/kg-BW-day)
Tcom_inh = biotransfer coefficient from inhalation to mother's milk (d/kg-milk)
Tcom_ing = biotransfer coefficient from ingestion to mother's milk (d/kg-milk)

Table 5.4 Mother's Milk Transfer Coefficients (Tcos) (Taken from Appendix J)

Chemical/chem. group	Tco (day/kg-milk)
PCDDs - oral	3.7
PCDFs - oral	1.8
Dioxin-like PCBs - oral	1.7
PAHs – inhalation	1.55
PAHs – oral	0.401
Lead - inhalation	0.064

The chemicals evaluated in the mother's milk pathway are multipathway chemicals (Appendix E) for which sufficient data were available to estimate a Tco.

Each Tco estimate accounts for biological processes from intake to milk that affect the transfer of a toxicant in the mother's body. Appendix J further describes OEHHA's recommendations for estimating the concentration of chemicals in breast milk.

5.4 Available Breast Milk Intake Rate Estimates

The literature contains several studies reporting measured breast milk intakes for infants at various ages and of different breastfeeding patterns. These studies typically have small sample sizes, are cross-sectional and do not represent the U.S. population of breastfeeding infants. However, the U.S. EPA (1997) Exposure Factors Handbook, the prior Hot Spots Exposure guidelines (OEHHA, 2000), and Arcus-Arth et al. (2005) compiled data from selected studies to derive summary intake rates for the population or certain subgroups of the infant population. Below we briefly summarize these reports.

5.4.1 U.S. EPA Exposure Factors Handbook (1997) and Child Specific Exposure Factors Handbook (2008)

The U.S. EPA National Center for Environmental Assessment published an Exposure Factors Handbook in 1997 (U.S. EPA, 19997) that provides a review of the breast milk pathway intake rates, and recommends values for breast milk intake rate, lipid intake rate, and lipid content. The 1997 Exposure Factors Handbook recommended breast milk intake rate values based on data from five publications identified as "key studies" by the Agency: Butte *et al.* (1984a), Dewey and Lonnerdal (1983), Dewey *et al.* (1991a; 1991b), Neville *et al.* (1988), and Pao *et al.* (1980). The Handbook recommended mean time-weighted average milk intakes of 742 ml/day and 688 ml/day for infants 0-6 months and 0-12 months of age, respectively. The Handbook also recommends upper-percentiles for time-weighted average daily intakes of 980 ml/day and 1033 ml/day for 0-6 and 0-12 months of age, respectively. The upper percentiles were calculated as the "mean plus 2 standard deviations." These estimates can be converted from ml to grams of breast milk by multiplying by 1.03. A disadvantage of these rates is that they are not normalized to infant body weight.

In September 2008, the U.S. EPA released the Child-specific Exposure Factors Handbook (CEFH). The CEFH reviewed relevant breast milk intake studies and provided recommended values (Table 5.3). In order to conform to the new standardized age groupings used in the CEFH, U.S. EPA used breast milk intake data from Pao et al. (1980), Dewey and Lönnerdal (1983), Butte et al. (1984), Neville et al. (1988), Dewey et al. (1991a), Dewey et al. (1991b), Butte et al. (2000) and Arcus-Arth et al. (2005). These data were compiled for each month of the first year of life.

Recommendations were converted to mL/day using a density of human milk of 1.03 g/mL rounded up to two significant figures. Only two studies (i.e., Butte et al., 1984 and Arcus-Arth et al., 2005) provided data on a body weight basis. For some months multiple studies were available; for others only one study was available. Weighted means were calculated for each age in months. When upper percentiles were not available from a study, these were estimated by adding two standard deviations to the mean value. Recommendations for upper percentiles, when multiple studies were available, were calculated as the midpoint of the range of upper percentile values of the studies available for each age in months. These month-by-month intakes were composited to yield intake rates for the standardized age groups by calculating a weighted average.

U.S.EPA provides recommendations for the population of exclusively breastfed infants (Table 5.5) since this population may have higher exposures than partially breastfed infants. For U.S. EPA, exclusively breastfed refers to infants whose sole source of milk comes from human milk, with no other milk substitutes. Partially breastfed refers to infants whose source of milk comes from both human milk and milk substitutes (i.e., formula). Note that some studies define partially breastfed as infants whose dietary intake comes from not only human milk and formula, but also from other solid foods (e.g., strained fruits, vegetables, meats).

Table 5.5. Recommended Values for Human Milk and Lipid Intake Rates for Exclusively Breastfed Infants by U.S. EPA Child-specific Exposure Factors Handbook (2008)

Age Group	Mean (mL/day)	Upper %ile[a] (mL/day)	Mean (mL/kg BW-day)	Upper %ile[a] (mL/kg BW-day)	Source
Human Milk Intake					
Birth to <1 month	510	950	150	220	b
1 to <3 months	690	980	140	190	b, c, d, e, f
3 to <6 months	770	1,000	110	150	b, c, d, e, f, g
6 to <12 months	620	1,000	83	130	b, c, e, g
Lipid Intake [h]					
Birth to <1 month	20	38	6.0	8.7	i
1 to <3 months	27	40	5.5	8.0	d, i
3 to <6 months	30	42	4.2	6.0	d, i
6 to <12 months	25	42	3.3	5.2	i

a Upper percentile is reported as mean plus 2 standard deviations
b. Neville et al., 1988.
c. Pao et al., 1980.
d. Butte et al., 1984.
e. Dewey and Lönnerdal, 1983.
f. Butte et al., 2000.
g. Dewey et al., 1991b.
h. The recommended value for the lipid content of human milk is 4.0 percent.
i. Arcus- Arth et al., 2005.

5.4.2 OEHHA Hot Spots Exposure Assessment and Stochastic Analysis Guidelines (OEHHA, 2000)

In the prior version of this document (OEHHA, 2000), breast milk intake studies were identified using specified criteria (described in the prior guidelines). The studies are briefly described in the prior guidelines and are divided into two categories: those for which breast milk intake is reported as amount (e.g., ml or grams) per day and those for which intake is reported as amount per body weight per day. Mothers were described as healthy, well-nourished, and at or near normal body weight. Infants were described as healthy, near- or full-term, and single born.

In reviewing and evaluating studies, several factors potentially affecting the accuracy of breast milk intake estimates and their applicability to the general population of infants were considered. These are discussed in the prior guidelines and include (1) the methods for measuring the volume of breast milk consumed, (2) the correlation of breast milk intake with age and with body weight, (3) insensible water loss, and (4) the effect of maternal factors on breast milk intake.

In the prior version of this document (OEHHA, 2000), two datasets were selected with which to derive breast milk intake rates: Hofvander *et al.* (1982) and Dewey *et al.* (1991a; 1991b). These datasets were selected because the data were on a body weight and individual infant basis and the combined datasets provided data covering the 1-12 month age period (the majority of the typical breastfeeding period). For the Hofvander study, all infants were exclusively breast fed while infants in the Dewey et al. study were exclusively breastfed to about 4 months of age and many through 6 months of age. However, in Dewey et al., some infants (exactly who and how many were unspecified) were introduced to solid foods as early as 4 months of age (based on the age of food introduction of 5.3 ± 1.1 months reported in the published report). Therefore, the Dewey et al. infants did not fit the AAP recommendations at 6 months of age (i.e., exclusively breastfed). Nonetheless, the 3 (exclusive breastfeeding), 9 (fully breastfeeding), and 12 (fully breastfeeding) month ages were in accordance with AAP recommendations.

The normal distribution described the combined datasets fairly well and fit much better than the log normal distribution. The means at the 3-month age group were not statistically different between the Hofvander et al. and Dewey et al. studies. There was considerable variability in the intakes reported at any given age, with the range (60-120 g/kg-day) and standard deviation (18-25 g/kg-day) consistent among the different age groups.

There is an overall trend of decreasing consumption on a per kg basis with increasing age, with daily intake greatest at 30 days of age. A linear relationship fits the age versus consumption rate data fairly well. From this combined data set, an intake averaged across breastfeeding infants during the first year of life is estimated to be 102.4 g/kg-day. Assuming a normal distribution of intake among the infants in this population (with mean and standard deviation 102.4 and 21.82 g/kg-day, respectively), the different levels of intake are derived and provided in Table 5.6. Similarly, an estimate of average intake during the first 6 months of life is estimated to be 131.4 g/kg-day.

Table 5.6 OEHHA (2000) - Distribution of daily breast milk intake (g/kg-day) for fully breastfed infants during their first 6 and 12 months of life*

Percentile	6 months	12 months
5	95.5	66.5
10	103	74.3
15	109	79.7
20	113	84.1
25	116	87.7
30	120	90.9
35	123	94.0
50	131	102
65	140	111
70	143	114
75	146	117
80	150	121
85	154	125
90	159	130
95	167	138
99	182	153

*Data from Hofvander et al. (1982) and Dewey et al. (1991a; 1991b), analysis conducted by OEHHA (2000).

5.4.3 Arcus-Arth et al. (2005)

Arcus-Arth et al. (2005) extended the work presented in OEHHA (2000) and reported statistical distributions (i.e., percentiles and parameters) of breast milk intake rates for infants fed in accordance with the 1997 American Academy of Pediatrics recommendations (AAP, 1997). The AAP recommendations were for infants to be exclusively breast fed through 6 months of age, and then to receive breast milk as the sole source of milk through 12 months of age during which time solid foods and non-milk liquids are being introduced.

Arcus-Arth et al. also presented distributions of breast milk intake rates for infants exclusively breastfed for 0-12 months. The Arcus-Arth et al. rates are based on breast milk intakes normalized to body weight (g/kg-day) of individual infants seven days to one year of age, with many infants providing data at more than one age period but no infant providing intake measurements from early to late infancy (i.e., at periodic time points throughout the first year). The rates were found to be normally distributed at each measurement age (e.g., at 3 months) as well as over the one year age period (i.e., 7 days through 12 months).

Two methods were used to analyze the data. In the first method (Method 1), the daily intake per kg infant body weight was regressed on age. Intake was integrated over a 6 or 12 month period, and divided by 182.5 or 365 days, respectively. This resulted in a

daily intake rate averaged over that period, i.e., an average daily intake. A pooled SD was calculated using the SD's at each measurement age. A distribution was then derived using an integrated average value calculated from the regression, the pooled SD, and an assumption of normality.

For the second method (Method 2), a dataset of breast milk intake over each of 6 or 12 months for 2500 hypothetical infants was created by randomly selecting values at each measurement age from the empirical distribution at that age and assuming normality. For each hypothetical infant, a line was fit using the generated "intake versus age" data, and an average daily intake for each infant was derived. The results are presented in Table 5.7 below.

Table 5.7 Daily Breast Milk Intake Rates Averaged Over 6 or 12 Months (g/kg-day)

Averaging Period	Mean (SD)	Population Percentile							
		5	10	25	50	75	90	95	99
AAP [1] 0-6 Months Method 1	129.6 (21.3)	94.5	102.3	115.2	129.6	144.0	157.0	164.6	179.3
AAP [1] 0-6 Months Method 2	126.3 (6.8)	115.2	117.7	121.8	126.3	130.9	135.0	137.5	142.1
AAP [1] 0-12 Months Method 1	100.7 (22.7)	62.4	70.9	85.0	100.7	116.3	130.4	138.9	154.9
AAP [1] 0-12 Months Method 2	101.6 (5.3)	92.8	94.8	98.0	101.6	105.2	108.4	110.3	113.4
EBF [2] 0-12 Months	113.0 (21.8)	77.1	85.0	98.3	113.0	127.7	140.9	148.8	163.8

[1] AAP = dataset based on American Academy of Pediatrics (1997) infant feeding recommendations

[2] EBF = dataset of exclusively breastfed infants

The variability, as measured by the SD and the range in values of the distribution, differ between Methods 1 and 2. Method 1 incorporated the correlation for an individual infant over time in their intake pattern (e.g., high-end consumers remained high-end consumers throughout the lactation period). Method 2 randomly selected intake values for a hypothetical infant at each age (measurement) point, and thus did not incorporate correlation between intakes. Because higher-end consumers tended to remain higher-end consumers while lower-end consumers remained lower-end, the range of values from the 5th-percentile to the 99th-percentile is much greater for Method 1 than for Method 2.

In comparison to the breast milk intake rates derived for the prior Hot Spots Exposure guidelines (2000), the Arcus-Arth et al. (2005) rates are based on a larger sample size, include intake measurements as young as 7 days of age (the prior guidelines used data from infants only as young as 3 months), and are in accordance with AAP recommendations. Because pediatricians tend to refer to AAP guidance, it is likely that they would encourage mothers to follow AAP breastfeeding recommendations.

5.5 Representativeness of Breast Milk Intake Estimates

The Exposure Factors Handbook (1997), prior Hot Spots Exposure and Stochastic Guidelines (2000), and Arcus-Arth et al. (2005) used data from mothers who were predominantly white, well-nourished and of relatively high socioeconomic (SES) and educational status, and therefore do not represent a cross-section of all California mothers. However, the literature indicates that SES does not affect the amount of breast milk produced by the mother or the amount of breast milk consumed by the infant, except when the mother is severely undernourished. This was the conclusion made by Ahn and MacLean (1980) who reported that studies generally agreed "that the milk output of mothers in [developing and industrialized countries are] comparable, except in populations of markedly undernourished women." Further, the World Health Organization (WHO, 1985) concluded that, for most mother-infant pairs, the volume of breast milk consumed by the infant is considerably less than the mother's potential supply. Thus, the breast milk intake rates reviewed in these guidelines are likely representative of the population of California infants.

5.6 Conclusion

Breastfeeding is an important indirect pathway of exposure for environmental toxicants, particularly persistent lipophilic chemicals, other substances that may accumulate in the body, and substances that are preferentially transferred into breast milk. Significantly larger quantities of some environmental toxicants stored in maternal tissue are delivered to breastfed infants compared to non-breastfed infants. Factors such as the duration of breastfeeding and maternal age at first breast feeding period can influence dose estimates. Breast milk intake should be considered when evaluating risks from environmental toxicants transferred to breast milk. This chapter provides a framework and the values needed for estimating the range of exposures to breast milk pollutants for breastfeeding infants.

The benefits of breastfeeding are widely recognized, and public health institutions promote and encourage breast feeding. In most situations, the benefits for the general infant population appear to outweigh the risks from exposure to toxicants in breast milk. It is a public health goal to minimize the risk and to understand the magnitude of the risk. Because the patterns of breastfeeding are changing, the duration of breastfeeding and intake of breast milk at different ages should be re-evaluated periodically to ensure a sound basis for such calculations.

Appendix 5A

Appendix 5A includes some background information on the mother's milk exposure pathway that may be useful for some specialized risk assessment applications but is not currently used in the Hot Spots exposure assessment model.

5A-1 Breast Milk Lipid

5A-1.1 Breast Milk Lipid Content

Many chemicals of concern in breast milk are primarily found in the breast milk lipid. Thus information on the lipid content of breast milk may be useful for some risk assessment applications. The average lipid composition of breast milk is significantly different among women (Harmann, et al., 1998). Some researchers have reported monthly increases in breast milk lipid during the breastfeeding period (Ferris et al. 1988; Clark *et al.* 1982), while others have found that breast milk lipid does not change significantly over time (Butte *et al.* 1984b; Dewey and Lonnerdal, 1983). Mean reported values from various studies are provided in Table 5A-1.

Nommsen et al. (1991) measured lipid content in breast milk of 39 women at four measurement periods (3, 6, 9, and 12 months of infant age). The data were collected to be representative of a 24-hour nursing duration, thus accounting for within feeding and diurnal variation in lipid content. Examination of the subjects' lipid levels longitudinally reveals that a subject with high lipid levels in breast milk produced at three months will tend to have high levels at subsequent months. An analysis of variance (ANOVA) using the 39 subjects for which four lipid level measurements are available confirms that there is a highly significant subject effect. Some studies have reported that lipid levels increase over the lactation period (Allen et al., 1991). For the Nommsen et al. study, the average lipid levels among the 39 subjects increase from 3.63 g/100 ml at 3 months to 4.02 g/100 ml at 12 months. However, for 14 of the 39 individuals, the lipid level shows a downward trend (e.g., the 12-month lipid level is lower than the 3 month). There is increased variability in lipid content at later measurement periods relative to earlier periods.

Table 5A-1 Lipid Content of Breast Milk Reported by Various Researchers

Study	Study Findings
Butte *et al.* (1984c)	3.92 g lipid /dl - mean for preterm infants 4.31 g lipid /dl - mean for full term infants For infants aged 2 to 12 weeks. 13 full term and 8 preterm infants. Measurements taken at 2, 4, 6, 8, 10, 12 weeks postpartum. No significant changes in content noted over time. Standard deviations ranged from 0.78 to 1.57 g lipid /dl.
Clark *et al.* (1982)	Mean total lipid content in units g/100 ml increased between 2 and 16 weeks postpartum for 10 subjects: 3.9, 4.1, 4.6 and 5.2 at 2, 6, 12, and 16 weeks postpartum.
Ferris *et al.* (1988)	Mean lipid in g/100 ml were 3.98, 4.41, 4.87, and 5.50 at, respectively, 2, 6, 12, and 16 weeks postpartum in 12 subjects. Standard deviations ranged from 0.99 to 1.09 g/100 ml.
Dewey and Lonnerdal (1983)	Overall mean lipid content ranged from 4.3 to 4.9 g/100 ml 1-6 months postpartum, without significant differences at different months. Standard deviations ranged from 0.97 to 1.96 g/100 ml. Measurements taken at 1, 2, 3, 4, 5, and 6 months postpartum. Number of subjects at each month ranged from 13 to 18.
Dewey *et al.* (1991a; 1991b) – raw data provided by K. Dewey	Percent of Lipid in Breast Milk (mean +/- SD) (n=sample size) 3 Months age = 3.67 +/- 0.84 (n=72) 6 Months age = 3.92 +/- 1.04 (n=53) 9 Months age = 4.16 +/- 1.07 (n=46) 12 Months age = 4.02 +/- 1.55 (n=39) All ages = 3.9 +/- 1.1 (n=210)
Mitoulas et al. (2003)	3.55 g lipid/dl (mean for 1-12 months)

5A-1.2 Breast Milk Lipid Intake Rates – Point Estimates

The Exposure Factors Handbook (U.S. EPA, 1997) recommends values for breast milk lipid intake rates (Table 5A-2). Values for infants under one year were based on data of Butte *et al.* (1984a) and the Maxwell and Burmaster (1993) analysis of the Dewey *et al.* (1991a) study. A lipid intake rate of 26 ml/day (equivalent to 26.8 g/day) was recommended for risk assessment purposes, with an upper percentile value of 40.4 ml/day (equivalent to 41.6 g/day) ("based on the mean plus 2 standard deviations"). The high-end value is based on a statistical model but falls within the range of empirical values (maximum 51.2 g/day) from Dewey et al. (1991a). A disadvantage of these rates is that they are not normalized to infant body weight.

Table 5A-2. Recommended Values for Lipid Intake Rates for Exclusively Breastfed Infants by U.S. EPA Child-specific Exposure Factors Handbook[a] (2008)

Age Group	Mean (mL/day)	Upper 95 %ile (mL/day)	Mean (mL/kg BW-day)	Upper 95 %ile (mL/kg BW-day)	Source
Birth to <1 month	20	38	6.0	8.7	b
1 to <3 months	27	40	5.5	8.0	b,c
3 to <6 months	30	42	4.2	6.0	b,c,
6 to <12 months	25	42	3.3	5.2	b

a The recommended value for the lipid content of human milk is 4.0 percent.
b. Arcus- Arth et al., 2005
c. Butte et al., 1984.

Mitoulas et al. (2003) studied breast milk intake and lipid levels in 30 Australian mother-infant pairs. The infants were fully breastfed for at least 4 months, with complementary foods added between 4 and 6 months age. Measurements were made at 1, 2, 4, 6, 9, and 12 months of age. For the 0-6 and 0-12 month periods, the mean lipid intake was 13.50 and 12.96 g/day, respectively. For the period of exclusive breastfeeding (1-4 months age), mean lipid intake was 13.33 g/day.

5A-1.3 Breast Milk Lipid Intake Rates - Distributions

The Maxwell and Burmaster (1993) study presented a distribution of breast milk lipid intake by infants less than one year of age. They report that, at any given time, "approximately 22% of infants less than one year of age are being breastfed, the remaining 78% have no exposure to chemicals in their mother's breast milk." They found the mean lipid intake among nursing infants to be characterized by a normal distribution with mean 26.81 g/day and standard deviation 7.39 g/day. Their results are based on the fraction of infants at different ages being breastfed according to the reports of Ryan *et al.* (1991a, 1991b) and "on data for lipid intake from a sample of white, middle- to upper-income, highly educated women living near Davis, California" (Dewey *et al.*, 1991a).

Advantages of this study include the detailed analysis of the breast milk pathway, which addressed several of the key factors contributing to variable intakes among individual infants. However, some features of this study limit its usefulness for evaluation of acute and chronic exposure of breastfed infants to environmental toxicants. First, the study did not analyze data on breast milk intake during the first three months of life and instead extrapolated from the Davis study to predict intake during this period. Second, intake was expressed as amount per day, rather than amount per body weight per day;

the latter would facilitate more accurate dose calculations. Third, estimates of the breastfeeding population are made for the fraction of current feeders on any given day rather than the fraction of infants who breastfed at any time during their first year of life. For chronic exposure analyses it is important to consider prior intakes in addition to current intake of individual infants.

Arcus-Arth et al. (2005) presented lipid intake rates normalized to body weight by combining measured milk intake values with lipid content values. The first set of lipid intakes was derived using only Dewey et al. data (raw data provided by K. Dewey, and methodology described in Dewey et al. 1991a, b). The infants were exclusively breastfed through 3 months of age and fully breast fed thereafter. Milk intake and lipid content were measured at 3 (n=72), 6 (n=53), 9 (n=46), and 12 (n=39) months of age. The milk intake from each infant was multiplied by the corresponding measured lipid content value for that infant at that age to give lipid intake. These lipid intake rates were normally distributed at the 6-, 9-, and 12-month measurement ages.

The researchers also derived a second set of lipid intakes using the same milk intake values of Dewey et al. and a 4% lipid content value, which is the lipid content value commonly used as a default in risk assessment. The 4% lipid content derived rates differed by 2-10% from the measured lipid content derived rates, with probable overestimation at the mean and underestimation at the low- and high-end percentiles. Because the differences were not substantial, and because a dataset of lipid content values representing the population is not available, the 4% lipid content value was considered a reasonable default.

A third set of lipid intakes was derived to represent the subpopulation of infants fed in accordance with AAP recommendations (AAP, 1997). Because a few infants in the Dewey et al. study had consumed solid foods between 4 and 6 months of age, and because it is not known which infants these were, the 6-month data did not follow AAP recommendations and thus could not be used for this purpose. Therefore, Arcus-Arth et al. used the AAP dataset they had created and the default 4% lipid content value to derive a set of “AAP lipid intake rates.”

For each set of lipid intakes, the values were regressed by age to derive average daily lipid intake rates over the 0-6 and 0-12 month periods. While the 0-12 month derived lipid intake rates were available in the Arcus-Arth et al. journal article, the 0-6 month rates were not published but were obtained from the authors (Arcus-Arth, personal communication, 2008).

Arcus-Arth et al. derived lipid intakes and average daily lipid intake rates only for breastfed infants, not the entire infant population, resulting in intakes that are not directly comparable to those of Maxwell and Burmaster (1993). An advantage of the Arcus-Arth et al. derived rates is that they are normalized to infant body weight. A disadvantage is that lipid intake values for infants 0-3 months of age were derived using extrapolation because measured values for this age group were not available.

Inter- and intraindividual variation of lipid content over time should be considered when evaluating lipid intake for the infant population. We chose to use the average daily lipid intake rates of Arcus-Arth et al. because they have incorporated variability over time and have been normalized to body weight. The mean and selected percentiles of the average daily lipid intake rates are presented in Tables 5A-4, below.

Table 5A-3 suggests that assuming a 4% lipid content value tends to slightly overestimate the mean and slightly underestimate the high-end percentile of average daily lipid intake. Nonetheless, the values are similar, supporting the use of a 4% lipid content value as a reasonable default. Further, the Exposure Factors Handbook (U.S. EPA, 1997) recommends assigning a value of 4% (i.e., 4 g/dl) to breast milk lipid content based on data of the National Research Council (1991), Butte *et al.* (1984a), and Maxwell and Burmaster (1993).

Table 5A-3 Comparison of Lipid Content Assumptions: average daily lipid intake (g/kg day) of breastfed infants for the 0–12 month age period*

	Mean	Population Percentiles							
		5	10	25	50	75	90	95	99
Measured lipid content [a]	3.70	2.01	2.38	3.00	3.70	4.39	5.01	5.38	6.08
4% lipid content [b]	4.03	2.53	2.85	3.37	3.96	4.54	5.07	5.38	5.98

[a] Lipid intake derived by multiplying the lipid content measurement by the milk intake measurement for each infant in the dataset provided by K. Dewey. Includes a few infants who may have received some solid foods between 4-6 months age.

[b] Lipid intake derived by multiplying a 4% lipid content value by the milk intake measurements provided by K. Dewey. Includes a few infants who may have received some solid foods between 4-6 months age.

* Data source: Arcus-Arth et al. (2005)

Assuming a 4% lipid content value, the distribution of average daily lipid intake rates for the AAP dataset is presented in Table 5A-4, below.

Table 5A-4 Distributions of Average Daily Lipid Intake (g/kg day) over the 0-6 and 0–12 month age periods for AAP infants and assuming 4% milk lipid content*

Age	Mean	Population Percentiles							
		5	10	25	50	75	90	95	99
0-6 months	5.18	3.78	4.09	4.61	5.18	5.76	6.28	6.58	7.17
0-12 [a] months	4.03	2.50	2.84	3.40	4.03	4.65	5.22	5.56	6.20

[a] includes infants exclusively breast fed through 6 months age and thereafter fully breast fed

* Data source: Arcus-Arth et al. (2005)

5A-2 Prevalence of Breastfeeding

Information on the prevalence of breastfeeding may be useful for assessing population impacts of pollutants. The majority of infants receive at least some breast milk during infancy. Of these infants, a significant number receive breast milk through at least 12 months of age. Using survey data, the prevalence of breastfeeding (i.e., percent of infants who are breastfed) can be estimated. The prevalence of in-hospital and early postpartum breastfeeding provides information regarding the initiation of breastfeeding and therefore the potential number of infants that may be exposed via the breast milk pathway. The prevalence of breastfeeding at later ages in the lactation period provides information on the duration of breastfeeding, which is a key determinant of the amount of breast milk, and therefore the total dose, to an infant over the lactation period.

Until recently, the only nationwide survey of breastfeeding prevalence was the Ross Mothers Survey (Ross Products Division, Abbott Laboratories). More recently, the National Immunization Survey and the National Survey of Children's Health have collected national data on breastfeeding prevalence, while the California Newborn Screening Program has collected data on infants in California (but only at an early postpartum (in-hospital) age). In addition, Hammer et al. (1999) provide prevalence data on a subpopulation of California infants (i.e., SF Bay area infants). These studies are briefly described below, and results are presented in Tables 5A-5 and 5A-6. The prevalence data could potentially be used in conjunction with breast milk intake rates to derive breast milk intake rates over the entire population of infants for the estimation of population cancer burden.

5A-2.1 The Ross Mothers Survey

The Ross Mothers Survey (RMS) is an annual nationwide mail survey conducted by Ross Products Division of Abbott Laboratories and is sent periodically to a probability sample of new mothers. Prior to January 1997, mothers received the survey at the time their babies turned six months of age. Since that time, surveys are sent to mothers at each month of age, from one through 12 months.

The survey asks mothers to recall the types of milk their babies received (1) in the hospital, (2) at one week of age, (3) in the last 30 days, and (4) most often in the last week. By using a multiple choice question, mothers select the kinds of milk fed to their infants from a listing that includes breast milk, commercially available infant formulas, and cow milk.

The weighting of the results reflects national demographics associated with the geography, race, age, and education of mothers throughout the United States. The 1998-2002 rates were weighted using U.S. Department of Health and Human Services 1997 natality data, while the 2002-2003 rates were weighted using year 2000 natality data. For 2002, the response rate was 21% (290,000 questionnaires returned out of 1,380,000 mailed) (Ryan, 2005).

The majority of infants in the U.S. receive breast milk at some time. The survey has consistently found that the percent of mothers breastfeeding in the U.S. varies considerably with geographic region. The highest rates of breastfeeding are in the Mountain and Pacific states (U.S. census regions). In the Pacific states in 2001, 82.9% of newborns were breastfed in-hospital, and 44.2% of infants were breastfed at 6 months (Ryan et al., 2002).

These rates are higher than the 1996 rates (75.1% and 30.9%, respectively for in-hospital and at 6 months age) reported in the prior guidelines. In addition to geographic differences, breastfeeding patterns vary considerably with maternal age and education, race/ethnicity, and economic status (National Research Council, 1991; Ross Products Division, Abbott Laboratories, 1996).

5A-2.2 The National Immunization Survey

The National Immunization Survey is conducted annually with approximately 35,600 questionnaires completed each year. Beginning July 2001 and continuing through December 2002, a sample of respondents was asked about breastfeeding using a set of breastfeeding questions. Starting January 2003, all respondents to the household telephone survey were asked these breastfeeding questions.

The NIS uses random-digit dialing to survey households about childhood immunization for children aged 19–35 months of age. The response rates for NIS years 2001–2006 ranged from 64.5% to 76.1%. Because children are 19–35 months of age at the time of the parent interview, each survey year represents children born sometime during a three calendar year period (Table A2 in NIS report). All analyses were conducted using statistical software that accounts for complex sample design. A more detailed description of the methods can be found at http://www.cdc.gov/nis.
Three modifications were made to the breastfeeding questions in 2004 and 2006. Only the change in January 2006 to Question 3, which consisted of asking the one question as two separate questions, resulted in significant effects on the prevalence rates (i.e., yielded significantly lower estimates of exclusive breastfeeding). Because of this large effect, the trends of exclusive breastfeeding by year of birth are shown separately for children whose caregivers were interviewed before and after January 2006.

Advantages of the NIS study include the relatively high response rates, California-specific data, and the inclusion in the survey of specific questions regarding the consumption by the infant of other foods or liquids in addition to breast milk. A disadvantage is the lengthy time interval between when the infant was breastfed and when the parent was asked questions pertinent to breastfeeding that infant, which may lead to inaccuracies in recall.

Table 5A-5 Prevalence of breastfeeding in the United States by birth year (percent ± ½ of confidence interval)

Age	Year					
	1999	2000	2001	2002	2003	2004
Early postpartum	68 ± 3	71 ± 2	71 ± 1	71 ± 1	73 ± 1	74 ± 1
At 6 months	33 ± 3	34 ± 2	37 ± 1	38 ± 1	39 ± 1	42 ± 1
At 12 months	15 ± 2	16 ± 2	18 ± 1	19 ± 1	20 ± 1	21 ± 1

* Exclusive breastfeeding information is from 2006 NIS survey data only and is defined as only breast milk — no solids, water, or other liquids.
* percent represents the proportion of infants
* Source: National Immunization Survey, Centers for Disease Control and Prevention, Department of Health and Human Services

Table 5A-6 Prevalence of Breastfeeding California Infants by Birth Year and Type of Breastfeeding (percent ± ½ of confidence interval)[1]

	N	Ever Breast-fed	Breast-fed at 6 Months	Breast-fed at 12 Months	N	Exclusive Breast-fed[2] at 3 Months	Exclusive Breast-fed[2] at 6 Months
Born in 2004	1702	83.8 ± 3.3	52.9 ± 4.3	30.4 ± 4.0	1438	38.7 ± 4.5	17.4 ± 3.5
Born in 2003	1688	83.8 ± 3.2	49.3 ± 4.0	26.6 ± 3.5			

[1] percent represents the proportion of infants
[2] Exclusive breastfeeding information is from 2006 NIS survey data only and is defined as only breast milk — no solids, water, and other liquids.
* Source: National Immunization Survey, Centers for Disease Control and Prevention, Department of Health and Human Services

5A-2.3 California Newborn Screening Program (MCAH, 2007)

In-hospital infant feeding practices in California are monitored using data collected by the Newborn Screening (NBS) Program. All non-military hospitals providing maternity services are required to complete the Newborn Screening Test Form prior to an infant's discharge. In addition to tracking genetic diseases and metabolic disorders, the NBS program gathers data on all infant feedings from birth to time of collecting the specimen for the genetic disease/metabolic disorder. The Maternal, Child and Adolescent Health (MCAH) Program staff, of the California Department of Public Health, analyze these data and publish the in-hospital breastfeeding rates (accessible at: http://www.cdph.ca.gov/data/statistics/Pages/BreastfeedingStatistics.aspx).

In September 2007, the MCAH published rates using 2006 Newborn Screening Program data. The prevalence rate for any breastfeeding in-hospital was 86.5% of mothers, while the rate of exclusive breastfeeding was 42.8%. The relatively low exclusive breastfeeding rate is only applicable to the in-hospital stay and not to the later period at home. This is because infants frequently receive some formula while in the hospital to prevent infant hypoglycemia which may result from an inability of the infant to properly nurse (e.g., latch on) initially or from the mother not producing sufficient milk for nursing yet.

5A-2.4 Hammer et al. (1999)

Hammer et al. (1999) prospectively studied the feeding patterns of 216 infants in the San Francisco Bay area from birth through weaning. Information on infant feeding practices was collected via an Infant Feeding Report form completed by the mother for a 3-day period at the end of every month. Parent-infant pairs were recruited from the well newborn nurseries at a university hospital, community hospital, and health maintenance organization (HMO). The parents' intention to feed the infant by a particular feeding pattern (e.g., bottle feeding) was not considered in selecting infants for the study.

Investigators or their staff in the laboratory did not give information or advice on feeding practices to parents, and all infants received routine health maintenance care from local physicians or clinics. Thus, the feeding patterns for these infants were not dictated by the study but instead are likely to have reflected prevalent feeding patterns in the general infant population of the SF Bay area. These patterns are likely to also be applicable to similar areas (e.g., urban) in California.

5A-2.5 Taylor (2006)

Taylor et al. (2006) analyzed data of singleton children of primiparous mothers from the 2002 National Survey of Family Growth. The data set included information on 3229 mother-child pairs when the child was 1-18 years of age. Women were asked if they had breastfed their child, and, if so, the number of completed weeks. A limitation of this study is the sometimes lengthy interval between infancy and when the mother was asked about infant feeding practices. An advantage of this study is the inclusion of only primiparous women, which is consistent with the assumption of the child being from a primiparous mother in these guidelines.

5A-2.6 Summary of Prevalence Data

Breastfeeding prevalence rates from the above studies are summarized in Table 5A-7, below. For the Ross Mothers Survey, rates for the Pacific region are presented because the Pacific region better represents California than the entire U.S.

Table 5A-7 Prevalence of Breastfeeding

Study	NIS [1]	Ross Mothers Survey [2] (Pacific region)	New Born Screening Program [3]	Hammer et al. (1999) [4]	Taylor et al. (2006) [5]
Study Background					
Sample Size	1702	39,600 (estimated 1999 sample size)	506,442	175	3229 primiparous, singleton
Geographic Region	U.S.	Pacific region	California	SF Bay Area, northern CA	U.S.
Year	2004	2001	2006	1997-1998 (presumed)	2002 (interview)1986-2001(birth year)
Percent of Infants Breastfeeding – Any Breastfeeding Pattern					
Ever breastfed	83.3%			90%	62%
In-hospital		82.9%	86.5%		
At 3 months					36%of all infants,58% of those who ever breastfed
At 6 months	52.9%	44.2%		48%	23% of all 38% of those who ever breastfed
At 12 months	30.4%			19%	6% of all,13% of those who ever breastfed

Table 5A-7 Prevalence of Breastfeeding (Cont.)

Study	NIS [1]	Ross Mothers Survey [2] (Pacific region)	New Born Screening Program [3]	Hammer et al. (1999) [4]	Taylor et al. (2006) [5]
Study Background					
Sample Size	1702	39,600 (estimated 1999 sample size)	506,442	175	3229 primiparous, singleton
Geographic Region	U.S.	Pacific region	California	SF Bay Area, northern CA	U.S.
Year	2004	2001	2006	1997-1998 (presumed)	2002 (interview)1986-2001(birth year)
Percent of Infants Breastfeeding - Exclusive Breastfeeding					
In-hospital		54.2%	42.8%		
At 2 months				31%	
At 3 months	38.7%				
At 6 months	17.4%	24.1%		14%	
At 12 months				7% ("sole breast-feeding")	

[1] National Immunization Survey, Centers for Disease Control and Prevention, Department of Health and Human Services
[2] Ryan et al. (2002)
[3] MCAH of the California Department of Public Health
[4] fed directly from the breast, does not include feedings from a bottle of breast milk
[5] data from the National Survey of Family Growth (2002)

5A-2.7 Trends in Breastfeeding at Early-postpartum, 6 month, and 12 Month Ages

The Ross Mothers Survey, National Immunization Survey, National Survey of Children's Health, and Hammer et al. (1999) collected data on the prevalence of breastfeeding at various times of the lactation period, and thus provide information on the initiation and duration of breastfeeding. The California Newborn Screening Program only provides information on in-hospital infants (i.e., initiation of breastfeeding).

The Ross Mothers Survey showed increases in breastfeeding both for in-hospital and at 6 months age between 1993 and 2003 for California (Mothers Survey, Ross Products Division of Abbott (2004) (Table 5A-8). It is of note that the in-hospital rate stabilized at about 80% from 1999-2002 but then decreased to 73.9% in 2003. Upon examination of rates for the other states (not shown here), a similar decrease of in-hospital rates occurred for 47 of the other 49 states (the exceptions being Delaware and North Dakota, which were noted as having 'variable' data associated with low sample sizes). A systematic calculation in the rates or a change in hospital policy might be responsible for this decrease. A decrease from 2002 to 2003 is also seen in 6-month rates for California and a little over half of the other states, but the decrease is much less than for the in-hospital rates and possibly not statistically significant. Thus, there appears to be a sudden unexplained decrease in the initiation of breastfeeding but the duration of breastfeeding has not significantly changed.

Table 5A-8 California-specific Breastfeeding Rates from the Ross Mothers Survey*

	In-hospital	At 6 months
1993	69.5	25.8
1994	70.6	27.1
1995	73.2	29.8
1996	72.0	29.4
1997	75.2	35.0
1998	76.9	38.4
1999	79.1	39.1
2000	80.2	40.1
2001	81.7	43.6
2002	79.7	41.7
2003	73.9	39.8

* Source: Mothers Survey, Ross Products Division of Abbott, 2004

The prevalence of infants who are exclusively breastfed at 6 months has also increased according to the RMS data (Table 5A-9, below). However, in-hospital exclusive breastfeeding does not appear to have changed. This might be because the mother's milk has not yet come in or that the infant has not yet learned how to latch on during the short stay in the hospital. Hospital staff may be anxious to feed the infant formula due to concern over hypoglycemia, which can occur very quickly in neonates.

Table 5A-9 Prevalence (percent of infants) of Breastfeeding for the United States from the Ross Mothers' Survey[1]

	Breastfeeding		Exclusive Breastfeeding	
	In-hospital	At 6 months	In-hospital	At 6 months
1994	57.4	19.7	46.8	11.2
1995	58.9	20.8	47.6	11.9
1996	59.2	21.7	47.3	12.2
1997	62.4	26.0	46.1	12.7
1998	64.3	28.6	46.2	13.8
1999	67.2	30.7	46.3	15.8
2000	68.4	31.4	46.0	16.0
2001	69.5	32.5	46.3	17.2
2001 – Pacific Region			54.2	24.1

[1] source: Ryan et al. (2002)

The National Immunization Survey Study (NIS) provides data from 1999 to 2004 for the entire U.S, which is sufficient for the assessment of trend over time. The NIS U.S. data show that from 2001 to 2006 slight to moderate progressive increases in breastfeeding prevalence occurred at the early postpartum period and at 6 and 12 months of age (Table 5A-10). California-specific data are available, but only for 2003 and 2004, which is insufficient for evaluating statistical trends over time (Table 5A.11). However, the data do reveal an increase from 2003 to 2004 in 6- and 12-month prevalence rates for California.

Table 5A-10 Prevalence of Breastfeeding in the United States by Birth Year (percent ± ½ of confidence interval)[1,2]

	Birth Year					
	1999	**2000**	**2001**	**2002**	**2003**	**2004**
Early postpartum	68 ± 3	71 ± 2	71 ± 1	71 ± 1	73 ± 1	74 ± 1
At 6 months	33 ± 3	34 ± 2	37 ± 1	38 ± 1	39 ± 1	42 ± 1
At 12 months	15 ± 2	16 ± 2	18 ± 1	19 ± 1	20 ± 1	21 ± 1

[1] Percent represents the proportion of infants
[2] Source: National Immunization Survey, Centers for Disease Control and Prevention, Department of Health and Human Services

Table 5A-11 Prevalence of Breastfeeding for California Infants by Birth Year and Type of Breastfeeding (percent ± ½ of confidence interval)[1]

	N	**Ever Breast-fed**	**Breast-fed at 6 Months**	**Breast-fed at 12 Months**		**N**	**Exclusively Breastfed [2] at 3 Months (2006)**	**Exclusively Breastfed[2] at 6 Months (2006)**
Birth Year 2003	1688	83.8 ± 3.2	49.3 ± 4.0	26.6 ± 3.5				
Birth Year 2004	1702	83.8 ± 3.3	52.9 ± 4.3	30.4 ± 4.0		1438	38.7 ± 4.5	17.4 ± 3.5

[1] Percent represents the proportion of infants
[2] Exclusive breastfeeding information is from interviews in 2006 and is defined as consumption of only breast milk (i.e., no solids, water, or other liquids).
* Source: National Immunization Survey, Centers for Disease Control and Prevention, Department of Health and Human Services

Maternal education and age, and family socioeconomic status have been correlated with both initiation and duration of breastfeeding (NIS, National Research Council, 1991; Ross Products Division, Abbott Laboratories, 1996). The NIS data for infants born in 2004 shows that infants were more likely to have ever been breastfed, breastfed at 6 months, or exclusively breastfed if they were born to mothers 30 years of age or older, born to mothers who were college graduates, or born to families at the highest income level studied (i.e., the highest level over the poverty-to-income ratio).

Because the above data demonstrate continued trends towards increases in the initiation and duration of breastfeeding (including exclusive breastfeeding), these trends should be re-evaluated periodically. Factors affecting breastfeeding prevalence, such as maternal age and the promotion of breastfeeding (both discussed below), can help to assess breastfeeding trends.

5A-2.8 Age at Weaning

A few studies have examined the rate of breastfeeding cessation. Maxwell and Burmaster (1993) found that the fraction of infants breastfeeding (f) in the U.S. in 1989 was well described by a negative exponential distribution (e.g., $f = a\ e^{-c\ t}$) with a cessation rate of 0.5% per day for the 0-12 month period. Arcus-Arth et al. (2005) used Ross Mothers Survey data from the year 2000 and found a cessation rate of 0.2027% per day for the 0-6 month period and 0.07563% for the 6-12 month period.

We evaluated data from the National Survey of Children's Health (NSCH, CDC, 2003) to assess age of weaning data that are more recent and that are specific to California. The NSCH is a national survey funded by the Maternal and Child Health Bureau, U.S. Department of Health and Human Services, and administered by the National Center for Health Statistics, Centers for Disease Control and Prevention. The survey collects data on national and state-level prevalence of a variety of physical, emotional, and behavioral child health indicators, including the age at which the child was completely weaned from breast milk.

The survey uses the State and Local Area Integrated Telephone Survey, which provides a consistent means to collect data across states. Phone numbers are selected randomly to identify households with one or more children less than 18 years of age. For these households, one child is randomly selected for inclusion in the study. Over 102,350 surveys were completed for children 0-17 years of age.

Survey results are weighted to represent the population of non-institutionalized children 0-17 years of age on both national and state levels. For the question on the age of weaning from breast milk, NSCH used only data from mothers whose children were 0-5 years of age at the time of interview. The reported age at weaning was reported as age intervals rather than age points.

These age intervals were <3, 3-6, 7-12, and over 12 months of age. Some women were still breastfeeding their child at the time of interview so it is unknown when these children were weaned. Data were available specific to California, with the most recent year being 2003. Results were based on those infants who were fed breast milk (versus based on all breastfed plus non-breastfed infants).

The NSCH Data Resource Center provides a website with an interactive data query feature for hands-on access to the survey data (http://www.nschdata.org/DataQuery/SurveyAreas.aspx). We used the website query system to assess age at weaning in California, by selecting "Survey Sections", then "California", "2003" and "Early Childhood", then "at what age did young children completely stop breastfeeding? (S6Q60 -- ages 0-5 who have been breastfed)." Results are presented in Table 5A-12, below.

Table 5A.12 Age interval when completely weaned from breast milk – California Infants[1]

	< 3 months	3-6 months	7-12 months	> 12 months	Total
Percent of breastfed infants[2]	19.9	30.2	31.3	18.6	100
Sample size	118	179	185	110	592

[1] Data from the National Survey of Children's Health from 2003
[2] Excluding those still breastfeeding at time of interview

To evaluate the distribution of breast milk weaning age in California we used the data in Table 5A-13 and applied simulation and curve fitting functions in Crystal Ball version 7.2.1 (Decisioneering, 2007) to find the best-fit distribution and to identify distributional parameters. We excluded infants (N=67) who were still breastfeeding at the time of interview, and adjusted the remaining data (i.e., percent weaned, N=592) to account for the exclusions. We found that the data best fit a gamma distribution with location = -0.17, scale = 3.60, and shape = 2.41464. The median age of weaning was 7.0 months and 75% of infants were weaned by 12 months, 90% by 16 months, and 95% by 18 months of age. It is noteworthy that a significant percentage of infants can be considered extended breast feeders (i.e., breastfed past 12 months of age). Our results are presented in Table 5A.13.

Table 5A.13 Mean and percentiles of the parametric model of age at weaning from breast milk for California infants in 2003 (in months) [1,2]

	mean	50%-ile	75%-ile	90%-ile	95%-ile
Weaning age (months)	8	8	12	16	18

[1] derived by OEHHA from the National Survey of Children's Health 2003 data
[2] excludes infants still breastfeeding at time of interview with mother

Other studies that provide information on the cessation of breastfeeding (weaning) include Hammer et al. (1999) (described above in Section 5A-2.4 and Rempel (2004). These two studies are summarized in Table 5A-14, below.

The Rempel (2004) study followed a cohort of Canadian mother-infant pairs from birth until 12 months of age. Of the 317 mothers who agreed to participate in the study, 289 initiated breastfeeding. The results are based on the 289 infants that breastfed. At 9 months of infant age, 27% of infants were still consuming some breast milk and 14% of the original 289 weaned between 9 and 12 months. Though the Rempel (2004) study involved Canadian mother-infant pairs, the results are likely similar to similar subpopulations in the U.S.

The mothers in the Rempel study were from Ontario (a fairly large cosmopolitan city), 16-42 years of age, had a mean +/- SD number years of education of 15 ± 2.8, 59% were employed full-time, 16% were employed part-time, 67% were married, 13% were born outside Canada. According to the authors "the participants represented a wide variety of cultural backgrounds." These demographics may be similar to some subpopulations of women in California cities.

Table 5A.14 Age at Weaning

Study	N	Infants Studied	Infant Age at Weaning (month)	Year(s) of Study	Comments
Hammer et al. (1999)	175	General population	Median: 6.0 Range: 0.9-39.1	1996-1998 (approx)	SF Bay area
Rempel (2004)	312	General population	13% weaned between 9 &12	1999-2000	Canada

5A-3 Subpopulations of Special Concern

5A.3.1 Infants Breastfed for an Extended Period of Time

Documentation of extended breastfeeding is quite limited in this country both because there is little socio-cultural support for extended nursing (Stein et al., 2004) and because many health care practitioners do not consider asking about it (Sugarman and Kendall-Tackett, 1995). However, recent increases in the duration of breastfeeding (see Section 5A-2.7, above) as well as efforts by public agencies and the American Academy of Pediatrics to promote and support breastfeeding would suggest that the number (and proportion) of infants being breastfed beyond the first year of life may be increasing as well. Few studies have evaluated information on extended breastfeeding. These studies are described, and summarized in Table 5A-15, below.

Sugarman and Kendall-Tackett (1995) found that among a group of American women (n = 179) who breastfed past 6 months of infant age, the age of weaning averaged between 2.5 and 3.0 years, with a high end value of 7 years 4 months. Forty-three percent of children in this sample (i.e., breastfed past 6 months) were breastfed beyond their third birthday. The researchers also found in examining mothers who breastfed more than one child past 6 months of age, that in subsequent lactations the younger children were breastfed for longer periods of time than the older child(ren) had been.

Dettwyler (2004) reported results of an informal survey of children who were breastfed for periods greater than 3 years. The sample included 1280 children, most during the 1990s, but some in the 1980s and earlier. The average age at weaning was 4.24 years, with a median of 4.00, a mode of 3.50, and a standard deviation of 1.08 years. Close to half of the children weaned between 3.00 and 4.00 years of age.

Children whose weaning was characterized as "child led" weaned at an average age of 4.39 years, whereas those whose weaning was characterized as "mother led" were weaned at an average age of 3.83 years. The mothers were most often middle-class and upper-class, worked outside the home, and highly educated. More than 50% of the mothers were college graduates, and the sample included numerous women with advanced degrees. Of those who responded to the question on ethnicity of the mother, most said they were European-American. These characteristics mirror those found in previous studies of extended breastfeeding in the U.S. (Sugarman and Kendall-Tackett, 1995).

Although most infants in California are weaned during their first year (see Table 5A-14, above)), there is a subpopulation of infants who are breastfed for an extended period. The Hammer et al. (1999) study (see description in Section 5A-2.8, above), which did not seek to identify extended breastfeeding infants, demonstrates that extended breastfeeding may be more prevalent than is commonly thought. Of the 175 infants who were breastfed, the oldest age at complete weaning from the breast was 39.1 months (extended breastfeeding).

Table 5A.15 Age at Weaning for Extended Breastfeeding Infants

Study	N	Infants Studied	Infant Age at Weaning	Year(s) of Study	Comments
Dettwyler (2004)	1280	Infants breastfed to at least 3 years	Mean: 4.24 yrs Median: 4.0 yrs SD: 1.08	1995-2000	U.S.
Hammer et al. (1999)	175	General population	Median: 6.0 mos Range: 0.9-39.1 mos	1996-1998 (presumed)	SF Bay area
Sugarman and Kendall-Tackett (1995)	134	Infants breastfed to at least 6 months	Mean: 2.5-3.0 yrs Range: 6 mo - 7 yrs 4 mos 43% breastfed past 3 yrs	1989-1991	U.S.

Immigrants to the U.S. may be more likely to practice extended breastfeeding, if they retain breast feeding practices from the home country. The 2003 joint WHO/UNICEF released a joint recommendation in 2003 that advocates exclusive breastfeeding for the first 6 months followed by breastfeeding with supplementation of complementary foods for at least the first two years of life (UNICEF/WHO, 1990). In the study by Buckley (2002), ten Hispanic mothers from Caribbean, South American or Central American countries, residing in the U.S. who breastfed their infant(s) beyond one year of age, stated that breastfeeding a child up to 4 years of age was common in their countries of origin.

Stein et al (2004) report a personal communication with Anne Seshadri (2002) who states “mothers in India frequently breastfeed their infants until 3 or 4 years of age”. Immigration into the U.S. from locations, where extended breast feeding is practiced such as Hispanic countries and India, could cause an overall increase in the incidence of extended breastfeeding.

Currently there are little data on the composition of breast milk during extended breastfeeding. Studies have found that when milk volume decreases (e.g., near the time of weaning) that lipid content increases, while other studies have found the opposite result. It would be helpful to know the lipid content of breast milk during extended breastfeeding to better understand the importance of lipophillic chemical transfer to an extended breastfed infant.

Exposures to infants who are breastfed for an extended period should be further investigated and could potentially be taken into account in non-default analyses. See Appendix J for a more detailed discussion about the accumulation and transfer of chemicals in maternal body tissue and its potential impact on extended breastfed infants.

5A-3.2 Infants of Older Mothers

Older primiparous mothers have longer to accumulate toxicants with long body tissue half-lives (i.e., more than six years) and could therefore eliminate more toxicant to their breast milk than younger mothers would. Furthermore, older mothers tend to breastfeed for a longer duration than younger mothers do (Section 5A.3.1, above). Both conditions could lead to higher dosing of primiparous infants from the breast milk of older mothers than of infants from younger primiparous mothers’ breast milk.

Many chemicals will reach a steady state in the mother’s body before age 25. On the other hand, other substances do not reach steady state within 25 years. For example, lead continues to accumulate in cortical bone over the human lifetime (O’Flaherty 1998). Thus, women giving birth after 25 years of age will have accumulated greater amounts of lead that can be passed to the infant in breast milk relative to mothers 25 years of age and younger.

Older mothers tend to initiate breastfeeding of their infants and breastfeed for longer periods of time. Because substances such as lead can accumulate in maternal tissues past the default 25 years for exposure to facility emissions before birth of a child, it is important to consider maternal age in assessing infant exposure to such toxicants via breast milk.

5A-3.2.1 Breastfeeding Practices of Older Mothers

In Section 5A-2.1, we provide background on the Ross Mothers Survey and the NIS. These surveys have consistently found that both the initiation and duration of breastfeeding increased with maternal age. The Ross Mothers Survey data (Table 5A-

16) show an increasing trend from 1996 to 2001 of older mothers to initiate breastfeeding and to continue to breastfeed for at least 6 months. The NIS data (Table 5A.17) show that older mothers are more likely to breastfeed and to exclusively breastfeed through 6 months in accordance with AAP recommendations (NSCH, 2007).

Table 5A-16 Prevalence (percent) of Breastfeeding by Maternal Age, Ross Mothers Survey

	Maternal Age				
	<20 years	**20-24 years**	**25-29 years**	**30-34 years**	**≥35 years**
In-hospital					
1996	43	53	62	68	69
2001	57	66	73	76	76
At 6 months					
1996	10	15	23	29	34
2001	20	26	35	42	44

* Source: Ryan et al. (2002)

Table 5A-17 Prevalence (percent) of Types of Breastfeeding by Maternal Age, Infants born in 2004

	Maternal Age		
	<20 years age	**20-29 years age**	**>=30 years age**
Ever Breastfeed	53	69	77
Breastfeed at 6 months	18	31	46
Breastfeed at 12 months	6	15	24
Exclusively breastfed at 3 months	17	26	35
Exclusively breastfed at 6 months	6	8	14

* Source: National Immunization Survey, Centers for Disease Control and Prevention, Department of Health and Human Services

5A-3.2.2 Prevalence of Older Women Giving Birth in California

There is an increasing trend toward older women giving birth in California. Births to women 35 years of age and older showed a progressive increase from 1990 to 2006 (Table 5A-18, below) (CDPH, 2006).

Table 5A-18 California Births by Maternal Age and Year of Birth (percent of total births for that year)

	Maternal Age		
	35-39 years	40-44 years	>=45 years
1990	9	1.6	0.07
1995	11	2.3	0.12
2000	13	2.9	0.18
2006	14	3.3	0.25

Data source: California Department of Public Health, birth records

It should be noted that the above data are for maternal age at primiparous and multiparous births. Data on primiparous-only births are not readily available. For some lipophilic toxicants, primiparous birth is an important parity as this can be when the greatest amount of toxicant may be excreted in the mother's breast milk, and the mother's body burden is reduced, thus lowering the dose to subsequent children.

Increases in maternal age may continue due to the increasing use of in-vitro fertilization for older women, though such increases are likely to be very small relative to the population of women giving birth.

5A-3.3 High-end Consumers

Under certain circumstances, information on individuals exposed at very high levels is of interest. For assessing high-end exposures, Table 5A-19 may be of use. It provides upper-end breast milk and lipid intake rate estimates for the breastfeeding population.

Table 5A-19 Intake estimates for the breastfeeding infant population

	Breast Milk Intake[1] (g/kg-day)		Lipid Intake[2] (g/kg-day)	
	6 month average	1 year average	6 month average	1 year average
99th percentile	179	155	7.1	6.2

[1] From Arcus-Arth et al. (2005)
[2] From correspondence with author (Arcus-Arth et al.) and based on lipid intakes at 3 and 6 months

Arcus-Arth et al. (2005) found that the rate of breast milk intake was highest during the second week of life. At this age, when susceptibility to certain toxicants is high, the mean intake is 160.6 g/kg-day and the 99th percentile is 257.8 g/kg-day.

5.7 References

Ahn CH, MacLean WC (1980). Growth of the exclusively breast-fed infant. Am J Clin Nutr 33:183-192.

AIHC (1994). Exposure Factors Sourcebook. Washington, D.C.: American Industrial Health Council.

American Academy of Pediatrics (1997). Breastfeeding and the use of human milk. Pediatr 100(6):1035-1039.

American Academy of Pediatrics (1982). The promotion of breastfeeding; policy statement based on task force report. Pediatr 69(5):654-661.

American Academy of Pediatrics Committee on Nutrition (1993). Supplemental foods for infants. In: Pediatric Nutrition Handbook. Third Edition. Barnes LA, editor. Elk Grove, IL: American Academy of Pediatrics; pp. 23-32.

Arcus-Arth A, Krowech G, Zeise L.(2005). Breast milk and lipid intake distributions for assessing cumulative exposure and risk. J Expo Anal Environ Epidemiol. 15(4):357-65.

Borschel MW, Kirksey A, Hannemann RE (1986). Evaluation of test-weighing for the assessment of milk volume intake of formula-fed infants and its application to breast-fed infants. Am J Clin Nutr 43:367-373.

Brown KH, Black RE, Robertson AD, Akhtar NA, Ahmed G, Becker S (1982). Clinical and field studies of human lactation: methodological considerations. Am J Clin Nutr 35:745-756.

Brown KH, Robertson AD, Akhtar NA (1986). Lactational capacity of marginally nourished mothers: infants' milk nutrient consumption and patterns of growth. Pediatrics 78(5):920-927.

Buckley, K.M. (2002). A comparison of long-term breastfeeding between Hispanics and Non-Hispanics, *Current Issues in Clinical Lactation, 2*, 23-36.

Butte NF, Garza C, Smith EO, Nichols BL (1983). Evaluation of the deuterium dilution technique against the test-weighing procedure for the determination of breast milk intake. Am J Clin Nutr 37:996-1003.

Butte NF; Garza C; Smith EO, Nichols BL (1984a). Human milk intake and growth in exclusively breast-fed infants. J Pediatr 104:187-194.

Butte NF, Garza C, Stuff JE, Smith EO, Nichols BL (1984b). Effects of maternal diet and body composition on lactational performance. Am J Clin Nutr 39:296-306.

Butte NF, Garza C, Johnson CA, Smith EO, Nichols BL (1984c). Longitudinal changes in milk composition of mothers delivering preterm and term infants. Early Hum Dev 9:153-162.

California Department of Health Services (1996). Vital Statistics of California 1994. August 1996.

Clark RM, Ferris AM, Fey M, Brown PB, Humdrieser KE, Jensen RG (1982). Changes in the lipids of human milk from 2 to 16 weeks postpartum. J Pediatr Gastroenterol Nutr 1:311-315.

Cohen R, Mrtek M (1994). The impact of two corporate lactation programs on the incidence and duration of breastfeeding by employed mothers. Am J Health Promot 8(6):436-441.

Crump KS and Howe RB (1984). The multistage model with time dependent dose pattern: applications to carcinogenesic risk assessment. Risk Anal 4(3): 163-176.

Decisioneering Inc., Denver, CO. Crystal Ball version 7.2.1. 2007.

Dewey KG; Heinig MJ; Nommsen LA, and Lonnerdal B (1991a). Adequacy of energy intake among breast-fed infants in the DARLING study: Relationships to growth velocity, morbidity, and activity levels. J Pediatr 119:538-547.

Dewey KG, Heinig MS, Nommsen MS and Lonnerdal B (1991b). Maternal versus infant factors related to breast milk intake and residual milk volume: The DARLING study. Pediatr 87(6):829-837.

Dewey KG and Lonnerdal B (1983). Milk and nutrient intake of breast-fed infants from 1 to 6 months: relation to growth and fatness. J Pediatr Gastroenterol Nutr 3(2):497-506.

Dorea JG, Donangelo CM. (2006). Early (in uterus and infant) exposure to mercury and lead. Clin Nutri 25:369-376.

DTSC (1993). Parameter values and ranges for CALTOX. Sacramento, CA: California Department of Toxic Substances Control, Office of Scientific Affairs, California Environmental Protection Agency; (DRAFT).

Ferris AM, Dotts MA, Clark RM, Ezrin M, Jensen RG (1988). Macronutrients in human milk at 2, 12, and 16 weeks postpartum. Journal of the American Dietetic Association 88(6):694-697.

Ferris AM, Jensen RG (1984). Lipids in human milk: A review. 1: Sampling, determination, and content. J Pediatr Gastroenterol Nutr 3(1):108-122.

Ferris AM; Neubauer SH; Bendel RB; Green KW; Ingardia CJ, and Reece EA (1993). Perinatal lactation protocol and outcome in mothers with and without insulin-dependent diabetes mellitus. Am J Clin Nutr 58:43-48.

Grandjean P, Weihe P, White RF. Milestone development in infants exposed to methylmercury from human milk. 1995. Neurotoxicol 16(1):27-34.

Harmann PE, Sherriff JL, Mitoulas LR, Homeostatic mechansisms that regulate lactation during energetic stress. Am Soc Nut Sci 1998; 128:394S-99S.

Hedley, A. J., T. W. Wong, et al. (2006). Breast milk dioxins in Hong Kong and Pearl River Delta. Environ Health Perspect 114(2): 202-8.

Hofvander Y; Hagman U; Hillervik C, and Sjolin S (1982). The amount of milk consumed by 1-3 months old breast- or bottle-fed infants. Acta Paediatr Scand 71:953-958.

Hoover S, Zeise L, Krowech G (1991). Exposure to environmental toxicants through breast milk: In: The analysis, communication and perception of risk. Garrick BJ, Gekler WC, editors. Advances in Risk Analysis. New York: Plenum Publishing.

Jelliffe DB, Jelliffe EFP (1978). The volume and composition of human milk in poorly nourished communities. Am J Clin Nutr 31:492-515.

Kershaw TG, Dhahir PH, Clarkson TW. 1980. The relationship between blood levels and dose of methylmercury in man. Arch Environ Health 35:28-36.

Labbok M and Krasovec K (1990). Toward consistency in breastfeeding definitions. Studies in Family Planning 21:226-230.

Kohler L, Meeuwisse G, Mortensson W (1984). Food intake and growth of infants between six and twenty-six weeks of age on breast milk, cow's milk formula, or soy formula. Acta Paediatr Scand 73:40-48.

Mata L, Perez MD, Puyol P, Calvo M. (1995). Distribution of added lead and cadmium in human and bovine milk. J Food Prot 58(3):305-309.

Matheny R and Picciano MF (1986). Feeding and growth characteristics of human milk-fed infants. J Am Diet Assoc 86(3):327-331.

Maxwell NI, Burmaster DE (1993). A simulation model to estimate a distribution of lipid intake from breast milk during the first year of life. J Exp Analysis Environ Epidemiol 3(4):383-406.

Michaelsen KF, Larsen PS, Thomsen BL, Samuelson G (1994). The Copenhagen Cohort Study on Infant Nutrition and Growth: breast-milk intake, human milk macronutrient content, and influencing factors. Am J Clin Nutr 59:600-611.

Montandon CM, Wills C, Garza C, O'Brian-Smith E, Nichols BL (1986). Formula intake of 1- and 4-month-old infants. J of Pediatr Gastroenterol Nutr 5:434-438.

Morrow, A. L., Guerrero, M. L., Shults, J., Calva, J. J., Lutter, C., Bravo, J., Ruiz-Palacion, G., Morrow, R. C. & Butterfoss, F. D. (1999) Efficacy of home-based peer counselling to promote exclusive breastfeeding: a randomized controlled trial. Lancet 353: 1226–1231.

Morse JM, Harrison MJ (1992). Social Coercion for Weaning. In: Qualitative Health Research. Morse JM, editor. Newbury Park, CA: Sage Publications, Inc. pp.363-375.

National Research Council (1991). Nutrition During Lactation. Washington DC: National Academy Press.

National Research Council (1993). Pesticides in the Diets of Infants and Children. NRC Committee on Pesticides in the Diets of Infants and Children. Washington DC.: National Academy Press.

National Immunization Survey. Breastfeeding practices – results from the National Immunization Survey: infants born in 2004. Hyatsville, Maryland: National Center for Health Statistics. 2007.

National Survey of Children's Health, National Children's Survey. Maternal, Child and Adolescent Health. (data accessed online via the Data Resource Center for Child and Adolescent Health at: http://www.nschdata.org/Content/Default.aspx).

National Survey of Children's Health 2003 (Centers for Disease Control and Prevention, National Center for Health Statistics, Division of Health Interview Statistics, State and Local Area Integrated Telephone Survey, National Survey of Children's Health (NSCH), 2003

Neubauer SH, Ferris AM, Chase CG, Fanelli J, Thompson CA, Lammi-Keefe CJ, Clark RM, Jensen RG, Bendel RB, Green KW (1993). Delayed lactogenesis in women with insulin-dependent diabetes mellitus. Am J Clin Nutr 58:54-60.

Neville MC; Keller R; Seacat J; Lutes V; Neifert M; Casey C; Allen J, and Archer P (1988). Studies in human lactation: milk volumes in lactating women during the onset of lactation and full lactation. Am J Clin Nutr 48:1375-1386.

Nommsen LA, Lovelady CA, Heinig MJ, Lonnerdal B, Dewey KG (1991). Determinants of energy, protein, lipid, and lactose concentrations in human milk during the first 12 mo of lactation: the DARLING study. Am J Clin Nutr 53:457-465.

OEHHA (2009). Technical Support Document for Cancer Potency Factors:Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office

of Environrmental Health Hazard Assessment. Online at:http://www.oehha.ca.gov/air/hot_spots/2009/TSDCancerPotency.pdf.

O'Flaherty, E J (1998). Physiologically based models of metal kinetics. Crit Rev Toxicol 28(3): 271-317.

Oskarsson A, Schutz A, Skerfving S, Hallen IP, Lagerkvist BJ. 1996. Total and inorganic mercury in breast milk and blood in relation to fish consumption and amalgam fillings in lactating women. Arch Environ Health 51:234–241.

Pao EM, Himes JM, Roche AF (1980). Milk intakes and feeding patterns of breast-fed infants. J Am Diet Assoc. 77:540-545.

Philipp BL, Merewood A, Miller LW, Chawla N, Murphy-Smith MM, Gomes JS, Cimo S, Cook JT. (2001). Baby-friendly hospital initiative improves breastfeeding initiation rates in a US hospital setting. Pediatrics. 108(3):766-8.

Piovanetti Y. (2001). Breastfeeding beyond 12 months: a historical perspective. Pediatr Clin North Am. 48:199–206.

Ross Products Division, Abbott Laboratories (1996). Updated breastfeeding trend. 1987-1995. Columbus, OH. Unpublished draft supplied to OEHHA by Ross Products Division, Abbott Laboratories.

Ross Products Division, Abbott Laboratories (1994). Updated breastfeeding trend: 1986-1993. Columbus, OH. Unpublished draft supplied to OEHHA by Ross Products Division, Abbott Laboratories.

Ryan AS, Rush D, Krieger FW, Lewandowski GE (1991). Recent declines in breastfeeding in the United States, 1984-1989. Pediatrics 88(4):719-727.

Ryan AS, Pratt WF, Wyson JL, Lewandowski G, McNally JW, Krieger FW (1991). A comparison of breastfeeding data from the national surveys of Children's Health and the Ross Laboratories mothers surveys. Am J Public Health 81(8):1049-1052.

Ryan AS, Zhou W; and Acosta A (December 2002). Breastfeeding conintues to increase into the new millennium. Pediatrics 110 (6):1103- 1109.

Sakamoto M, Kubota M, Matsumoto S, Nakano A, Akagi H. 2002. Declining risk of methylmercury exposure to infants during lactation. Environ Res 90:185–189.

Salmenpera L, Perheentupa J, Siimes MA (1985). Exclusively breast-fed health infants grow slower than reference infants. Pediatr Res 19(3):307-312.

Smith AH (1987). Infant exposure assessment for breast milk dioxins and furans derived from incineration emissions. Risk Anal 7(3):347-353.

Stein, MT, Boies EG, Snyder D. (2004).Parental Concerns About Extended Breastfeeding in a Toddler. Challenging Case: Family. J Developmental Behavioral Pediatrics 25:S107-S111.

Stuff JE and Nichols BL (1989). Nutrient intake and growth performance of older infants fed human milk. J Pediatr 115(6):959-68.

Sugarman M, Kendall-Tackett KA (1995). Weaning ages in a sample of American women who practice extended breastfeeding. Clin Pediatr 642-649.

Sundberg J, Ersson B, Lonnerdal B, Oskarsson A. 1999. Protein binding of mercury in milk and plasma from mice and man—a comparison between methylmercury and inorganic mercury. Toxicology 137:169–184.

Sundberg J, Jonsson S, Karlsson MO, Pallminger Hallen I, Oskarsson A. 1998. Kinetics of methylmercury and inorganic mercury in lactating and nonlactating mice. Toxicol Appl Pharmacol 151:319–329.

U.S. EPA (1989). Exposure Factors Handbook, 1989 U.S. Environmental Protection Agency, National Center for Environmental Assessment Washington, D.C.: EPA/600/8-89/043.

U.S. EPA (1997). Exposure Factors Handbook, August 1997 U.S. Environmental Protection Agency, National Center for Environmental Assessment Washington, D.C.: EPA/600/P-95/002Fb.

U.S. EPA. Child-Specific Exposure Factors Handbook (2008). U.S. Environmental Protection Agency, Washington, D.C., EPA/600/R-06/096F, 2008.

Wang RY, Needham LL. (2007). Environmental chemicals: from the environment to food, to breast milk, to the infant. J Toxicol Environ Health Part B, 10:597-609.

Whitehead RG, Paul AA (1981). Infant growth and human milk requirements. A fresh approach. Lancet. 2:161-163.

Woolridge MW, Butte N, Dewey KG, Ferris AM, Garza C, Keller RP (1985). Methods for the measurement of milk volume intake of the breast-fed infant. in: Jensen RG, Neville MC, eds. Human Lactation: Milk Components and Methodologies. New York: Plenum; pp. 5-21.

World Health Organization (1985). The quantity and quality of breast milk. Geneva: World Health Organization.

Wright A, Rice S, Wells S (1996). Changing hospital practices to increase the duration of breastfeeding. Pediatrics 97(5):669-675.

6 Dermal Exposure Assessment

6.1 Introduction

Semi-volatile and nonvolatile contaminants emitted into the air can be subsequently deposited onto soil or other surfaces. Exposure to chemicals can occur through skin contact with the contaminated soil. This exposure pathway is considered under the Air Toxics "Hot Spots" Act when evaluating chronic exposure.

For semi-volatile organic compounds (SVOCs), OEHHA has not quantified exposure via the air-to-skin transdermal pathway for the Hot Spots Program. This pathway is inherently included in human and animal whole-body inhalation exposures to chemicals in toxicology and epidemiology studies for both VOCs and SVOCs. Whole-body inhalation studies almost always form the basis for determining Reference Exposure Levels (RELs) and Cancer Potency Factors (CPFs) where the metric of exposure is the airborne concentration. As such, exposure via the air-to-skin pathway is incorporated into the RELs and CPFs for individual chemicals.

The significance of the air-to-skin transdermal pathway for some Hot Spots SVOCs has been shown in a modeling study that utilized physical and chemical principles combined with empirical evidence to critically assess the significance of the dermal pathway as a contributor to total human exposure to SVOCs (Weschler and Nazaroff, 2012). In this study, it is proposed that intake by the air-to-skin transdermal pathway can exceed intake by inhalation for several SVOCs that humans can be exposed to. The air-to-skin pathway is of particular concern for the relatively more volatile SVOCs that both equilibrate rapidly with skin-surface lipids and also permeate the skin relatively quickly. Amphiphilic SVOCs (i.e., containing both hydrophilic and lipophilic properties) in particular are included in this group. Hot Spots chemicals that fall into this group probably include the smaller molecular weight PCBs such as PCB77 and PCB81.

For a second group of SVOCs, direct air-to-skin transport can also contribute to total uptake, but perhaps not to the same fractional extent as the first group owing to slower equilibration with skin-surface lipids or slower migration through the stratum corneum (Weschler and Nazaroff, 2012). Hot Spots chemicals that fall into this group include many of the PAHs such as B(a)P and chrysene. In a third group of SVOCs, the equilibrium time is too long for air-to-skin transport to be important. Hot Spots chemicals in this third group include diethylhexylphthalate and probably the dioxins and furans (e.g., TCDD). However, skin contact with these SVOC-containing materials or surfaces (such as contaminated soil) may contribute to elevated levels in skin-surface lipids. Once sorbed at the skin surface, subsequent migration through the stratum corneum and viable epidermis can be relatively fast.

Although the air-to-skin transdermal pathway is generally taken into account in RELs and CPFs, the importance of this route should be discussed in the event RELs or CPFs are developed for some SVOCs based on studies that use other than whole-body

inhalation (e.g., nose-only inhalation). Note that chronic inhalation exposures are always “whole body” for logistic reasons.

Likewise absorption of chemicals dissolved or deposited into water while swimming, bathing, or showering could be significant under certain exposure scenarios but usually not under the airborne release scenario considered in the “Hot Spots” program.

The significance of each of the above exposure pathways varies by type of chemical, but dermal uptake of chemicals from soil and other surfaces is considered the most relevant. This route applies to semivolatile organic chemicals such as PAHs, dioxins and PCBs, and some inorganic metals such as lead and lead compounds. Under the “Hot Spots” program, dermal exposure to soils contaminated with these chemicals is considered the principal dermal exposure pathway. The concentrations in soil around a specific facility due to long term deposition are estimated from facility emissions estimates, air modeling, estimates of soil half-life and soil mixing depth.

As discussed in Section 6.5 below, OEHHA devised a new variate called the Annual Dermal Load, or ADL. This variate is a composite of three variates described in the previous version of this document (OEHHA, 2000): the body surface area (BSA) per kg body weight, exposure frequency, and soil adherence variates, which simplifies the calculation for risk assessors. In addition, ADLs have been determined for California climate zones, expressed as warm, mixed and cold. These climate zones recognize the different amount of time one spends outside during the year (depending on the climate zone), and the amount of clothing one wears in these different climate zones. All of which influences the ADL value.

6.2 Recommended Dermal Exposure Values

For assessing dermal exposure, we are recommending point estimates using the ADL variates presented in Table 6.1. These point estimates are the mean and 95th percentile values from the stochastic distributions shown in Tables 6.2a-d. Using Eq. 6-8 (see below), the variables that are needed to assess dermal exposure include the climate-dependent ADL, the soil concentration of contaminant and the ABS (dermal absorption value from soil).

Table 6.1. Recommended Annual Dermal Load Point Estimates (in mg/kg-yr) for Dermal Exposure

	3rd Trimester	Children 0<2 yrs	Children 2<9 yrs	Children 2<16 yrs	Adults[a]	Off-Site Worker
Warm climate						
Mean	1.2×10^3	3.6×10^3	7.5×10^3	6.4×10^3	1.2×10^3	2.6×10^3
95th percentile	2.6×10^3	4.3×10^3	9.1×10^3	8.5×10^3	2.6×10^3	5.0×10^3
Mixed climate						
Mean	1.1×10^3	2.2×10^3	6.6×10^3	5.7×10^3	1.1×10^3	2.6×10^3
95th percentile	2.4×10^3	2.9×10^3	8.7×10^3	8.1×10^3	2.4×10^3	5.0×10^3
Cold climate						
Mean	0.7×10^3	1.2×10^3	3.1×10^3	2.8×10^3	0.7×10^3	2.6×10^3
95th percentile	2.1×10^3	1.9×10^3	5.2×10^3	5.1×10^3	2.1×10^3	5.0×10^3

[a] Residential adults includes 16<30 and 16-70 year age groups

ADL distributions in Tables 6.2a-d are by age group and climate, with the adult age groups (16-30 and 16-70 years of age) sharing the same values. The ADL for the third trimester of the fetus is based on the ADL of the mother; when normalized to body weight, we assume that exposure to the mother and the fetus will be the same. The mother's exposure is based on the adults age 16-30 years of age in Table 6.2d.

Tables 6.2a-d. Annual Dermal Load Distributions by Age Group and ClimateTable

6.2a. Annual Dermal Load (mg/kg-yr) Distributions for the 0<2 Year Age Group

Climate Type	Warm climate	Mixed climate	Cold climate
Distribution	Student's t	Logistic	Triangular
Minimum			0.2×10^3
Likeliest			0.7×10^3
Maximum			2.6×10^3
Scale	0.41	0.28	
Deg. freedom	3		
Midpoint	3.6×10^3		
Mean	3.6×10^3	2.2×10^3	1.2×10^3
50th percentile	3.6×10^3	2.2×10^3	0.9×10^3
90th percentile	4.1×10^3	2.8×10^3	1.9×10^3
95th percentile	4.3×10^3	2.9×10^3	1.9×10^3
99th percentile	4.7×10^3	3.1×10^3	2.1×10^3

Table 6.2b. Annual Dermal Load (mg/kg-yr) Distributions for the 2<9 Year Age Group

Climate Type	Warm climate	Mixed climate	Cold climate
Distribution	Min extreme	Min extreme	Triangular
Minimum			0.4×10^3
Likeliest	8.0×10^3	7.3×10^3	1.9×10^3
Maximum			6.9×10^3
Scale	0.1	1.3	
Mean	7.5×10^3	6.6×10^3	3.1×10^3
50 th percentile	7.7×10^3	6.5×10^3	2.3×10^3
90 th percentile	8.7×10^3	8.4×10^3	5.1×10^3
95 th percentile	9.1×10^3	8.7×10^3	5.2×10^3
99 th percentile	9.7×10^3	9.4×10^3	5.7×10^3

Table 6.2c. Annual Dermal Load (mg/kg-yr) Distributions for the 2<16 Year Age Group

Climate Type	Warm Climate	Mixed climate	Cold climate
Distribution	Min extreme	Logistic	Triangular
Minimum			0.3×10^3
Likeliest	7.2×10^3		1.6×10^3
Maximum			6.9×10^3
Scale	1.29	0.91	
Mean	6.4×10^3	5.7×10^3	2.8×10^3
50 th percentile	6.6×10^3	5.7×10^3	2.2×10^3
90 th percentile	8.1×10^3	7.7×10^3	4.8×10^3
95 th percentile	8.5×10^3	8.1×10^3	5.1×10^3
99 th percentile	9.3×10^3	8.9×10^3	5.6×10^3

Table 6.2d. Annual Dermal Load (mg/kg-yr) Distributions for Residential Adults (Age 16-30 and 16-70 Years) and Offsite Workers

Receptor	Residential Adult			Offsite Worker
Climate Type	**Warm**	**Mixed**	**Cold**	**All Climates[a]**
Distribution	Beta	Beta	Gamma	Lognormal
Minimum	0.2×10^3	0.02×10^3		
Maximum	3.3×10^3	0.3×10^3		
Scale			0.07	
Mean	1.2×10^3	1.1×10^3	0.7×10^3	2.6×10^3
50 th percentile	1.2×10^3	1.0×10^3	0.5×10^3	2.3×10^3
90 th percentile	2.4×10^3	2.1×10^3	1.6×10^3	4.5×10^3
95 th percentile	2.6×10^3	2.4×10^3	2.1×10^3	5.0×10^3
99 th percentile	2.9×10^3	2.6×10^3	2.3×10^3	6.4×10^3

[a] Face, hands and forearms are exposed only, regardless of climate

There are several advantages for stochastically combining the four variates from the original dermal dose equation (see Equation 6-1 below) into an annual dermal load variate (OEHHA, 2000). First, using one variate (annual dermal load) rather than four separate variates simplifies calculations for risk assessors. Also, distributional information that previously was separate is now integrated into one distribution. In addition, selecting a high-end value from the annual dermal load distribution reduces the possibility of over-conservatism that can occur when high-end values of the variates are multiplied together as was done with Equation 6-1 in the prior edition of the Stochastic guidelines (OEHHA, 2000).

6.3 Dermal Uptake from Contaminated Soil Contact

Although the dermal exposure route is generally considered a minor exposure pathway, a screening study by Johnson and Kissel (1996) of over 200 risk assessments for Superfund sites resulted in identification of 37 sites at which projected lifetime excess cancer risks attributed to dermal contact with contaminated soil were greater than 1 in 10,000. Dermal exposure was the dominant exposure route at 9 sites. Thus it is possible for dermal exposure to reach a level of significance, although the soil concentrations resulting from airborne deposition tend to be lower than when more concentrated pollutants are present in hazardous waste sites. The primary soil contaminants in these dermal risk assessments included dioxins, PAHs, PCBs and arsenic. Johnson and Kissel (1996) highlighted early concern for the dermal pathway and the need for better information for dermal exposure variates, such as the chemical fractional skin absorption, surface area exposure and soil adherence, in order to better assess dermal absorption potential.

The potential for skin contact with soil near the home can be significant. In a national survey known as the Soil Contact Survey, almost half of households reported the presence of bare spots (44.7%) other than gardens in their yards (Wong et al., 2000a).

A majority (63.7%) of respondents with homes also reported a vacant lot or field within walking distance of the home.

As discussed above, dermal absorption varies by exposure pathway and with the properties of the chemical. Other major factors which influence dermal absorption include the anatomical region exposed (Maibach et al., 1971; Wester and Maibach, 1985), the amount of skin exposed, soil or particle type and size, amount of soil adhering to skin (Duff and Kissel, 1996; Choate et al., 2006), type of surface contacted, chemical concentration (Nomeir et al., 1992; Sartorelli et al., 2003), duration of exposure, ambient temperature and humidity (Chang and Riviere, 1991), and activities which limit exposure (e.g., washing the skin).

The inherent variability in some of the exposure factors can be estimated, such as in total skin surface area of children and adults. In other cases, the actual variation is not as well known, such as soil loading on specific body parts in young children. Also, the factor involved may be well known but the net effect on dermal absorption of chemicals may not be readily described or quantified. For example, dermal absorption varies with skin temperature and blood flow, which tends to vary with ambient temperature and physical activity. However, the magnitude of this effect is insufficiently documented to support distribution modeling. Overall, there is generally not enough information to generate probability distributions for all of the key variates for estimating dermal absorption, although ranges are available for some variates.

This discussion of dermal exposure estimates includes the primary variates involved and can be reasonably quantified or estimated, based on the more common human activities that result in soil skin contact (e.g., gardening). Dermal exposure is expressed as a variate called the dermal dose (Eq. 6-1). The dermal dose is defined as the amount of contaminant absorbed through the skin per unit of body weight per day (mg/kg-day). For the Air Toxics "Hot Spots" program, the dermal dose resulting from contact with contaminated soil can be estimated using the following equation:

$$DOSEdermal = (C_s \times SA \times SL \times EF \times ABS) \ / \ (BW \times 1x10^6) \qquad \textbf{(Eq. 6-1)}$$

where:

DOSEdermal = exposure dose through dermal absorption (mg/kg-d)
C_s = average concentration of chemical in soil (µg/kg)
SA = surface area of exposed skin (m^2)
SL = soil loading on skin (g/m^2-d)
EF = exposure frequency (d/365 d)
ABS = fraction of chemical absorbed across skin
BW = body weight (kg)
$1x10^6$ = conversion factors for chemical and soil (µg to mg, g to kg)

The dermal absorption factor (ABS) is a chemical-specific, unitless factor that is discussed in Section 6.4.1 below. The exposure frequency (EF) is set at 350 days per year (i.e., per 365 days) to allow for a two-week vacation away from home each year (US EPA (1991).

Equation 6-1 requires multiplying values together, which could lead to overly conservative exposure estimates when high-end values for variates are used. By combining information from several variates into one composite distribution, over-conservatism may be avoided (see Section 6.5). To this end, OEHHA created a new variate, "annual dermal load", or ADL, which is a composite of the body surface area (BSA) per kg body weight, exposure frequency, and soil adherence variates:

$$ADL = (BSA / BW)^* [(SL_b)(SA_b\%_b)] * EF \quad \textbf{(Eq. 6-2)}$$

Where:

ADL = Annual dermal load (mg/kg BW-yr)
EF = Exposure frequency (d /yr)

Thus, the dermal-dose equation (Eq. 6-1) can be reduced to the following:

$$\text{Dermal dose (mg/kg-d)} = ADL * C_s * ABS * (yr/365\ d) * 1x10^{-9} \quad \textbf{(Eq. 6-3)}$$

Where:

yr/365 d = Conversion factor (years to days)
$1x10^{-9}$ = Conversion factor for chemical and soil (µg to mg, mg to kg)

For cancer risk, the risk is calculated for each age group using the appropriate age sensitivity factors (ASFs) and the chemical-specific cancer potency factor (CPF) expressed in units of $(mg/kg\text{-}day)^{-1}$:

$$RISKdermal = DOSEdermal * CPF * ASF * ED/AT \quad \textbf{(Eq. 6-4)}$$

RISK is the predicted risk of cancer (unitless) over a lifetime as a result of the exposure, and is usually expressed as chances per million persons exposed (e.g., 5×10^{-6} would be 5 chances per million persons exposed).

The dose-response phase of a cancer risk assessment aims to characterize the relationship between an applied dose of a carcinogen and the risk of tumor appearance in a human. This is usually expressed as a cancer potency factor, or CPF, in the above equation. The CPF is the slope of the extrapolated dose-response curve and is expressed as units of inverse dose $(mg/kg\text{-}d)^{-1}$, or inverse concentration $(\mu g/m^3)^{-1}$.

Exposure duration (ED) is the number of years within the age groupings. In order to accommodate the use of the ASFs (OEHHA, 2009), the exposure for each age grouping must be separately calculated. Because cancer risk has been shown to be greater in sensitive age groups, different ASFs are applied to different life stages used for cancer risk assessment (see below). DOSEdermal can vary depending on the type of outdoor activities that involve soil exposure. The type of outdoor activities may be specific for the age of the individual, such as general outdoor play on bare soil by young children, or gardening by adults. Thus, the DOSEdermal and ED are different for each age grouping.

ED = exposure duration (yrs):

0.25 yrs for third trimester	(ASF = 10)
2 yrs for 0<2 age group	(ASF = 10)
7 yrs for 2<9 age group	(ASF = 3)
14 yrs for 2<16 age group	(ASF = 3)
14 yrs for 16<30 age group	(ASF = 1)
54 yrs for 16-70 age group	(ASF = 1)

DOSEdermal includes indirect exposure to the fetus via direct exposure to the mother during the third trimester of pregnancy. Fetal exposure during the third trimester will be the same as that of the mother on a body weight-normalized basis, and is taken into account in the final determination of the annual dermal load presented in Section 6.2.

AT, the averaging time for lifetime cancer risks, is 70 years in all cases. To determine lifetime cancer risks, the risks are then summed across the age groups:

$$RISKdermal_{(lifetime)} = RISKdermal_{(3rdtri)} + RISKdermal_{(0<2\ yr)} + RISKdermal_{(2<16\ yr)} + RISKdermal_{(16\text{-}70yr)} \quad \textbf{(Eq. 6-5)}$$

As explained in Chapter 1, we also need to accommodate cancer risk estimates for the average (9 years) and high-end (30 years) length of time at a single residence, as well as the traditional 70 year lifetime cancer risk estimate. For example, assessing risk in a 9 year residential scenario assumes exposure during the most sensitive period, from the third trimester to 9 years of age and would be presented as follows:

$$RISKdermal_{(9\text{-}yr\ residency)} = RISKdermal_{(3rdtri)} + RISKdermal_{(0<2\ yr)} + RISKdermal_{(2<9\ yr)} \quad \textbf{(Eq. 6-6)}$$

For 30-year residential exposure scenario, the 2<16 and 16<30 age group RISKdermal would be added to the risk from the third trimester to 0<2 age group. For 70 year residency risk, Eq 6-5 would apply.

Because distributional data are available for the total surface area, body weight and exposure frequency variates, a stochastic approach can be used to derive one distribution by combining these variates for the specified age groups. This stochastic approach provides an alternative means for estimating dermal exposure and is presented below in Section 6.2.

The term Cs, concentration of the contaminant in soil, can be derived in the Hot Spots Analysis and Reporting Program (HARP) using air dispersion and deposition modeling (CARB, 2003). The concentration is a function of the deposition, accumulation period, chemical-specific soil half-life, mixing depth, and soil bulk density. The formula used is:

$$C_s = [Dep \times X)] \ / \ [K_s \times SD \times BD \times T_t] \quad \textbf{(Eq. 6-7)}$$

where:

C_s	= average soil concentration over the evaluation period (μg/kg)
Dep	= deposition on the affected soil area per day (μg/m2-d)
X	= integral function accounting for soil half-life (d)
K_s	= soil elimination time constant = $0.693/T_{1/2}$
SD	= soil mixing depth = 0.01 m for playground setting and 0.15 m for agricultural setting
BD	= bulk density of soil = 1333 kg/m3
T_t	= 25,550 days (70 yrs), total averaging time for the chemical accumulation period (i.e., 70 yrs, the presumed life of the facility emitting chemicals)

The deposition on the affected soil area per day is expressed as:

$$Dep = GLC \times Dep\text{-}rate \times 86{,}400 \quad \textbf{(Eq. 6-8)}$$

where:

GLC	= ground level concentration from air dispersion modeling (μg/m3)
Dep-rate	= vertical rate of deposition (m/sec) (see Chapter 2 for values)
86,400	= seconds per day conversion factor (sec/d)

The integral function, X, is as follows:

$$X = [\{Exp\ (-K_s \times T_f) - Exp\ (-K_s \times T0)\} / K_s] + T_t \quad \textbf{(Eq. 6-9)}$$

where:

Exp	= exponent base e = 2.718
K_s	= soil elimination constant = $0.693/ T_{1/2}$
$T_{1/2}$	= chemical-specific soil half-life (d)
T_f	= end of exposure period (d)
T_0	= beginning of exposure period (d) = 0 days
T_t	= total days of exposure period = Tf - T0 (d)

Chemical-specific soil half-lives ($T_{1/2}$) are presented in Appendix G.

T_f = 25,500 d = 70 yrs. Identifies the total number of days of soil deposition.

T_f = 9,490 d = 25 yr for nursing mother in mother's milk pathway.

The assumptions in the soil concentration algorithm include:

1) Uniform mixing of pollutants in the soil and a constant concentration over the duration of the exposure.
2) The bulk density (BD) of soils is similar over a wide variety of soil types.
3) Substances are not leached or washed away, except where evidence exists to the contrary
4) For the mother's milk pathway, the mother is exposed for 25 years, the child receives milk for one year (from mother's 25th birthday to 26th birthday), and then is exposed to all other pathways.

6.4 Derivation of Key Dermal Exposure Variates

Other than the soil concentration of a chemical, which is estimated from the emission, meteorological, terrain, and other data using HARP (or other software), the key variates in equation 6-1 are the chemical-specific fractional absorption factor (ABS), the surface area of exposed skin (SA), body weight, the soil loading or soil adherence of contaminated soil on skin (SL) in mg soil per cm^2 skin, and the exposure frequency (EF) in number of days exposed per year. The description of how point estimates or distributions were derived for each of these variates using existing literature sources are summarized below, and in Appendix F for the chemical ABS.

6.4.1 Chemical-specific Absorption Factors

Skin permeability is related to the solubility or strength of binding of the chemical in the delivery matrix (soil or other particles) versus the receptor matrix, the skin's stratum corneum. This skin layer, which is the major skin permeability barrier, is essentially multiple lipophilic and hydrophilic layers comprised of flattened, dead, epidermal cells. The greatest rate of skin permeation occurs with small moderately lipophilic organic chemicals. However, such chemicals may not have the greatest total uptake, because they may evaporate off the skin. The highest penetration thus is expected from larger, moderately lipophilic chemicals with negligible vapor pressures. Organic chemicals which dissociate in solution, or metal salts that are more soluble in the aqueous phase of stratum corneum and insoluble in the lipid phase, will not penetrate the skin readily.

These principles of skin absorption are presented in US EPA (1992), and summarized in Appendix F of this document as it pertains to dermal absorption from contact with contaminated soil. Fractional dermal absorption point estimate values were derived by OEHHA from available literature sources for the semi-volatile and nonvolatile chemicals in the "Hot Spots" program (Table 6.3). The rationale for the chemical-specific dermal absorption fraction values, and the use of default values in cases where sufficient data are lacking, can be found in Appendix F.

Table 6.3. Dermal Absorption Fraction Factors (ABS) as Percent from Soil for Semi-Volatile and Solid Chemicals under the OEHHA "Hot Spots" Program

Chemical	ABS
Inorganic chemicals	
Arsenic	6
Beryllium	3
Cadmium	0.2
Chromium (VI)	2
Fluorides (soluble compounds)	3
Lead	3
Mercury	4
Nickel	2
Selenium	3
Organic chemicals	
Creosotes	13
Diethylhexylphthalate	9
Hexachlorobenzene	4
Hexachlorocyclohexanes	3
4,4'methylene dianiline	10
Pentachlorophenol	[a]
Polychlorinated biphenyls	14
Polychlorinated dibenzo-p-dioxins and dibenzofurans	3
Polycyclic aromatic hydrocarbons	13

[a] To be assessed for dermal absorption

Most exposure estimates have utilized a single value for presumed dermal uptake rate or percent without distinguishing between the specific skin regions that might be involved under different scenarios. However, it is known that the permeability of skin to chemicals may vary depending on the skin site of absorption. In general, hands are least permeable, and face and neck are most permeable (Maibach et al., 1971; Wester and Maibach, 1985). Other site-specific and scenario-specific factors are involved in dermal absorption, as discussed in Appendix F, which can result in significant differences in dermal uptake under different conditions. Data are inadequate to describe potential changes in fractional dermal absorption with changing scenarios. Thus, point estimate values are used for the ABS.

6.4.2 Body Surface Area / Body Weight Distributional Variate

Total body surface area (BSA) and body weight are known to be highly correlated with a reported correlation coefficient (r) ranging from 0.88-0.96 (Durnin, 1959). Although there are distributional human body weight data, there are no directly measured data for BSA representative of the population. However, Gehan and George (1970) derived a BSA formula based on direct measurements of BSA from 401 individuals. Their formula

accounted for over 99% of the variation in BSA and was derived using more BSA measurements that were directly measured than other BSA formulae. The Gehan and George formula is shown as:

$$BSA\ (m^2) = (Wt^{0.51456}) \times (Ht^{0.42246}) \times 0.02350 \quad \textbf{(Eq. 6-10)}$$

where:
Wt = body weight (kg)
Ht = body height (cm)

For body weight and height data, OEHHA used the National Health and Nutrition Examination Survey (NHANES) 1999-2004 dataset (CDC, 2007). NHANES provides weights for each individual in the dataset and for the study design so that estimates using NHANES data can be weighted to be nationally representative. Total body surface estimates for each individual in the NHANES 1999-2004 dataset were derived using these individuals' body weight and height and equation 6-5. Means and specific percentiles are shown in Table 6.4 and 6.5. The sample size for NHANES, and for many subpopulations within NHANES (e.g., each year of age), is sufficiently large to provide information on interindividual variability and distributions. There are other sources of body weight and height data, but NHANES is the most recent national dataset, thus reflecting the current population, and has data on each individual for the assessment of interindividual variability.

Table 6.4. Summary Distribution Estimates of Total Body Surface Area (in m^2) by Age Group[a]

	Children 0<2 years	**Children 2<9 years**	**Children 2<16 years**	**Adults >16 years**
Sample size	2106	3250	9007	16,718
Mean	0.459	0.884	1.177	1.942
SEM	0.003	0.005	0.006	0.003
50^{th} percentile	0.470	0.824	1.124	1.923
90^{th} percentile	0.564	1.107	1.730	2.302
95^{th} percentile	0.583	1.212	1.880	2.414

[a] Derived using the equation 6.3 and the body height and weight data of the NHANES 1999-2004 study

Table 6.5. Summary Estimates of Total Body Surface Area over Body Weight (m^2/kg) by Age Group[a]

	All ages	Children 0<2 years	Children 2<9 years	Children 2<16 years	Adults >16 years
Sample size	27831	2106	3250	9007	16718
Min	0.016	0.034	0.022	0.016	0.016
Max	0.077	0.077	0.054	0.054	0.040
Mean	0.028	0.049	0.039	0.035	0.025
SEM	0.000068	0.0001	0.000019	0.000097	0.000038
50^{th} percentile	0.026	0.048	0.040	0.035	0.025
75^{th} percentile	0.029	0.051	0.043	0.040	0.027
90^{th} percentile	0.038	0.056	0.045	0.043	0.029
95^{th} percentile	0.043	0.059	0.046	0.045	0.029
99^{th} percentile	0.049	0.063	0.048	0.047	0.031

[a] Derived from NHANES 1999-2004 data

6.4.3 Skin Surface Area Exposed

The amount of skin or body region that is exposed to soil contact is dependent on the type of clothing worn. Clothing is expected to significantly reduce exposure to the covered skin area from contaminated soil. Dermal risk assessment procedures used by U.S. EPA (2004) assumes no exposure of skin that is covered with clothing. The few studies that investigated this issue found that clothing had a protective effect for soil exposure, although some exposure may occur under clothing (Kissel et al., 1998; Dor et al., 2000). Considering Kissel et al. (1998) showed incomplete coverage of exposed body parts occurred in a soil exposure study, it appears unlikely that the limited soil exposure that occurs under clothing will underestimate total exposure. Consequently, the model OEHHA uses assumes no exposure to covered skin. Exposed skin is essentially limited to face, hands, forearms, lower legs, feet, or some combination thereof (U.S. EPA, 2004). However, the amount of skin exposed as a result of clothing choices is dependent on exposure activity, age group, and the climatic conditions. Because California has geographically diverse climatic regions, studies investigating clothing choices by children and adults during warm and cold weather outdoor activities were used to estimate skin exposure for different climate regions within the state.

6.4.3.1 Fractional Body Part Surface Area

U.S. EPA (2004) provides data on the percent of surface area for different body parts that may be exposed to soil. When the fractional surface area of a specific body part, such as hands, is multiplied by total surface area, the surface area of the specified body part in m^2 or cm^2 is determined. As mentioned above, normalized surface area can be derived for each individual in the NHANES dataset. Multiplying normalized surface area for each individual by the percent surface area of each body part gives an estimated normalized surface area of each body part for that individual. Individuals are then grouped by age to derive the surface area for each body part for each age group. Because the percent surface area is a constant, multiplying normalized total surface

area by the percent surface area maintains the same probability distribution of the NHANES normalized total body surface area. That is, the probability distribution of body surface area from the nationally representative NHANES data is preserved.

In the children's Soil Contact Survey by Wong et al. (2000b), the activity patterns of children (≤18 years) that would result in dermal soil contact were investigated. Of 680 households, 500 (73.5%) had children that were reported to play outdoors on bare dirt or mixed grass and dirt surfaces. An age breakdown of the children showed that those reporting little outdoor play were either very young (≤1 year) or relatively old (≥14 years for females; ≥16 years for males).

The Soil Contact Survey also asked about clothing choices during outdoor play in warm weather and determined estimated percentage skin surface area exposed (Table 6.6). For children under 5 years of age, outdoor play was treated as a single activity. Information on outdoor activity of children aged 5 to 17 was categorized as gardening/yardwork and as organized team sports. The combination of short sleeves and short pants was a common clothing choice for outdoor activities. Skin exposure was lowest for participants in organized team sports because that group had the highest fraction wearing shoes and high socks.

The mean skin area exposed for children age 5-17 during gardening and yardwork (33.8%) is essentially the same as the default mean surface area value of 33.9% used by U.S. EPA (2004), based on soil adherence data, for children age 6 years and up. Together, the findings indicate that soil contact exposure in warm weather is primarily limited to face, hands, forearms, and lower legs, with feet exposure most common in young children up to about 6 years of age.

Table 6.6. Estimated Skin Surface Area Exposed During Selected Warm Weather Outdoor Activities by Children[a]

	Skin area exposed (% of total) based on expressed clothing choices		
	Outdoor play (age <5 yrs)	Gardening/yardwork (age 5-17 yrs)	Organized team sports (age 5-17 yrs)
Mean	38.0	33.8	29.0
Median	36.5	33.0	30.0
SD	6.0	8.3	10.5

[a] Table adapted from data in Wong et al. (2000)

In the Soil Contact Survey of adults, Garlock et al. (1999) conducted a regional (Washington and Oregon state) and national telephone survey for four outdoor activities among 450 adults for each sample. The activities included gardening, other yard work, outdoor team sports and home construction or repair with digging. The reported participation rate for any activity was 89% for the regional survey and 79% for the national survey, with more than half of the respondents reporting participation in 2 or 3 of the activities. Table 6.7 presents both the national and regional (in parentheses) percentage skin area exposed during warm and cold months among the outdoor

participants for these activities. Warm- and cold-weather months were defined by the respondent.

Table 6.7. Estimated Skin Surface Exposed During Outdoor Activities by Adults in the National and Regional (in parentheses) Surveys[a]

	Skin area exposed (% of total) based on expressed clothing choices			
	Gardening	Other yard work	Team sports	Repair/Digging
Warm months				
Median	33 (33)	33 (31)	33 (33)	28 (28)
95th %tile	69 (68)	68 (68)	43 (68)	67 (67)
Cold months				
Median	8 (3)	3 (3)	8 (8)	3 (3)
95th %tile	33 (14)	31 (12)	33 (30)	14 (14)

[a] Table adapted from data by Garlock et al. (1999).

In most activities, the median and 95th percentiles were remarkably similar between the two surveys. Current U.S. EPA guidelines (U.S. EPA, 2004; 2011) for skin area exposed to soil contact assumes roughly 25% exposure for adults, corresponding to head, forearms, lower legs and hands. These findings show that the median exposure during warm months exceeds 25%, suggesting some exposures occur with no shoes or no shirt (males) or with a halter (women).

Based on the results of the Soil Contact Surveys and the activity-dependent soil adherence data in U.S. EPA (2004), the anticipated exposed body parts for children and adults during cold and warm weather are shown in Table 6.8. In cold weather, the findings by Garlock et al. (1999) for adults suggest that the hands and face are most often exposed for some activities (e.g., gardening and team sports), but that only the face is most often exposed or partially exposed for other activities (e.g., other yard work and repair/digging), corresponding to wearing gloves. Given that the most common activities in this study, gardening and team sports, suggest both hands and face were exposed, our assessment will include both body parts for soil exposure of adults and children in a cold climate. Very limited data suggested body part exposure in young children during cold weather months was similar to findings in adults (Holmes et al., 1999). Accordingly, we will also use hands and faces as the exposed body parts for the cold climate assessments in children.

In warm weather, the adult fractional skin exposure during outdoor activities in the Soil Contact Study had a median ranging from 28-33% (Garlock et al., 1999). This finding is only slightly higher than the median fractional skin exposure of about 27% for face, hands, forearms and lower legs combined shown in Table 6.8. Review of the U.S. EPA (2004) soil adherence data for adults shows that shoes are predominantly worn during outdoor activities, and that a halter (for women) or no shirt were choices of some participants as indicated by the Garlock et al. study. For the stochastic assessment, only face, forearms, hands and lower legs were considered “exposed” in warm weather.

For the offsite worker, fractional skin exposure is similar, but since full length pants are worn, assessments only included faces, hands and forearms.

For children in warm weather climates, the survey by Wong et al. (2000b) observed that in addition to the face, hands, forearms and lower legs, the feet were often exposed. For example, young daycare children ages 1 to 6.5 years with free access to both the indoors and outdoors were all found to go without shoes, exposing bare feet or socks, at least once during the day. No data were presented for children less than one year of age. Nevertheless, for the warm weather exposure assessment of the 0<2 age group, the body parts considered exposed include feet, face, hands, forearms and lower legs.

For older children, Wong et al. (2000b) noted that organized team sports are common activities in children ages 5<17 years which may result in soil contact with skin. However, shoes are likely worn during many of these activities. In another study that monitored children's microactivity patterns, it was observed among children ages 3-13 years that younger children were more likely to be barefoot both indoors and outdoors compared to older children (Freeman et al., 2001). The average age of the barefoot children was 5.8 years, and the average age of children that wore shoes was 8.2 years. To account for the greater tendency of younger children in the 2< 9 and 2<16 year age group to go barefoot during outdoor play, OEHHA designated that feet exposure will be given 2/3 and 1/3 weighting for the 2<9 and 2<16 year age groups, respectively, during warm weather activities. This feet exposure adjustment was assessed in the soil adherence section below, in which the soil adherence value for 2< 9 and 2<16 year-olds was reduced to 2/3 and 1/3, respectively, of the initial soil load.

Table 6.8. Exposed Body Parts by Age Group and Weather Conditions, with the Corresponding Mean Values for the Percentage of Total Body Surface for each Body Part in Parenthesis.

	Children 0<2 yrs[a]	Children 2<9 yrs[a]	Children 2<16 yrs[a]	Residential Adult[b]	Offsite Worker[b]
Body Part Exposed	**Cold Weather**				
	Hands (5.5) Face (5.8)	Hands (5.3) Face (4.4)	Hands (5.4) Face (3.7)	Hands (5.2) Face (2.5)	Hands (5.2) Face (2.5)
	Warm Weather				
	Hands (5.5) Face (5.8) Forearms (6.0) Lower legs (8.7) Feet (6.4)	Hands (5.3) Face (4.4) Forearms (5.9) Lower legs (10.8) Feet (7.2)	Hands (5.4) Face (3.7) Forearms (6.0) Lower legs (11.8) Feet (7.2)	Hands (5.2) Face (2.5) Forearms (6.1) Lower legs (12.8)	Hands (5.2) Face (2.5) Forearms (6.1)

[a] The percentage of total body surface area for the specified body parts was estimated for each age group from data in Exhibit C-1 of U.S. EPA (2004). All values are averages for males and females combined.

[b] Body part percentage estimated from data in Table B-3 of U.S. EPA (1985).

OEHHA believes the surface area exposure estimates in Table 6.8 are health protective, but not overly conservative. For example, soil exposure under clothing is not included in the algorithm, even though some studies have shown that a limited degree of exposure may occur under clothing (Kissel et al., 1998; Dor et al., 2000). Also, the neck is not included as an exposed skin region in this document, even though a field study by Dor et al. (2000) showed that soil contact on the exposed neck can occur. Future studies of soil contact to skin may need to include the neck as a potential skin region for soil contact.

6.4.3.2 California Climate Regions and Skin Exposure

Climate will strongly influence people's choice of clothing. Due to California's varied climatic regions and existing data on clothing choices at different temperatures, three levels of climatic conditions, warm, mixed, and cold, are used to describe California's climate regions. The type of climate will, in turn, be used to assess the fraction of exposed skin for soil contact.

The "warm" climate is characteristic of Southern California areas such as Los Angeles, which can have warm to hot temperatures throughout the year. The "cold" climate is representative of San Francisco, Eureka, and other northern coastal communities, which have cool temperatures (daily highs of less than 65 degrees) for the majority of the year and can receive a considerable amount of fog and rainfall. The "mixed" climate is one that has warm-to-hot temperatures during much of the year (daily highs over 80 degrees are common), roughly from April to October, and cold temperatures (lows near or below freezing) during the remainder of the year. The mountains and central valley are examples of a mixed climate. Specifically, the mixed climate is described as seven months/year of warm temperatures, resulting in warm-temperature clothing choices, and the remaining five months a year as a cold climate with cold-temperature clothing choices. Thus, the average surface area exposed over a year is proportional to seven months of warm weather skin exposure and five months of cold weather skin exposure.

6.4.4 Soil Adherence Factors

Assessing risk from dermal exposure with contaminated soil requires an estimate of the amount of soil that will stick to skin long enough for the chemical to transfer from the soil and into the skin. This estimate has been given the term soil loading, or soil adherence, and is expressed in mass of soil per area of skin (usually in mg/cm^2). Because some body parts may have substantially greater soil adherence rates relative to other body parts, we assigned body part-specific soil adherence values to the corresponding body part surface area. Soil adherence estimates utilized published studies that were body part-specific, measuring soil adherence to hands, forearms, face, lower legs, and feet resulting from specific outdoor activities. Knowledge of body-part specific soil adherence and surface area exposure can be applied in equation 6-6 below to determine a weighted soil adherence factor (U.S. EPA, 2004; 2011). The example equation presented here is based on potential skin exposure resulting from a choice of clothing that allows soil contact with face, hands, forearms, lower legs and feet (e.g., children in a warm weather climate):

Weighted AF = (**Eq. 6-9)**

$$\frac{(AF_{face})(SA_{face}) + (AF_{forearms})(SA_{forearms}) + (AF_{hands})(SA_{hands}) + (AF_{feet})(SA_{feet}) + (AF_{lower\ legs})(SA_{lower\ legs})}{SA_{face} + SA_{forearms} + SA_{hands} + SA_{lower\ legs} + SA_{feet}}$$

where:

Weighted AF = overall weighted adherence factor of soil to skin (mg/cm^2-event)
AF_i = adherence factor for specific body part (mg/cm^2-event)
SA_i = specific skin surface area exposed for soil contact (cm^2)

U.S. EPA (2004) provided individual data on body-part-specific soil adherence for numerous activities (e.g., playing in dry soil, gardening, etc.), which were derived from published work (Kissel et al., 1996b; Kissel et al., 1998; Holmes et al., 1999). Although soil load was measured for quite a few activities, the number of individuals measured was small for each activity and soil adherence data for some body parts were not available for certain activities and age groups. Thus, OEHHA chose to use the arithmetic average of the soil loading rate for each body part rather than attempt to define a distribution for soil adherence. Table 6.9 presents the body part-specific soil adherence factors, in g/m^2, resulting from common outdoor activities in children and adults.

Lack of soil adherence data is particularly evident among children in the 0<2 year age group. Soil adherence data are essentially absent under one year of age. For children 1<2 yrs of age, soil adherence on specific body parts can be calculated from a small group of daycare children that had roamed freely indoors and outdoors and had access to outdoor soil (Holmes et al., 1999; U.S. EPA, 2004).

For infants less than 1 yr of age, Wong et al. (2000b) observed that these children remained mostly indoors and were likely given little opportunity for direct contact with soil when outdoors. In another children activity survey, parents reported that only 17% of infants age 7-12 months had contact with outdoor dirt the previous day, while 70% of children age 1 to 4 yrs had contact with outdoor soil the previous day (Black et al., 2005).

Notably, the outdoor soil contact findings by Black et al. (2005) contrast with their findings of time spent by children playing indoors on the floor, with considerably greater time spent on the floor among infants compared to older children. Although this chapter is focused on exposure to contaminated outdoor soil, there is much evidence that shows a significant amount of outdoor soil can be found in indoor house dust (Culbard and Johnson, 1984; Davies et al., 1985; Thornton et al., 1985; Culbard et al., 1988; Fergusson and Kim, 1991; Stanek and Calabrese, 1992). From these studies, an average of about one-third of indoor house dust is composed of soil (range: 20-78%). Because infants <1 year old spend more time indoors and play on the floor more frequently than older children, soil exposure from indoor sources may be important source of dermal contact for this age group. However, lack of soil adherence data for infants and lack of soil adherence data due to indoor soil exposure prevent an estimation of the extent of the risk.

To avoid underestimating indoor soil exposure in infants of the 0<2 age group, the infants (i.e., 0≤1 yr olds) are assumed to have the same soil adherence levels on specified body parts as the 1<2 yr old children in a daycare facility (Holmes et al., 1999; U.S. EPA, 2004). Thus, the average soil adherence for the entire 0<2 age group is based on the 1<2 yr old daycare children and is presented in Table 6.9.

A limitation of this data is the lack of soil adherence data for the faces of the young children. To avoid non-participation in the studies, the faces of the children were not examined for soil adherence. As a surrogate, soil adherence data on the faces of 8-12 yr old children playing in dry and wet soil were averaged and used to represent soil adherence on faces of the 0<2 yr age group (Kissel et al., 1998b; U.S. EPA, 2004).

For the 2<9 and 2<16 year-old child groups, equal weighting for soil adherence was given to three groups of children: those that played in dry soil, those that played in wet soil, and those that played team sports (Kissel et al., 1996b; Kissel et al., 1998; U.S. EPA, 2004). Team sports were included to account for the greater tendency of older children to play team sports as opposed to general play in dry or wet soil (Wong et al., 2000b).

The methodology for outdoor play by the children stipulated that shoes be worn. However, studies show that during unrestricted play by children <8 years of age many go barefoot during outdoor play (Freeman et al., 2001). To account for the tendency of younger children in the 2<9 and 2<16 age groups to be barefoot during outdoor play, the soil adherence data on feet of children with access indoors and outdoors at a daycare facility were used (Holmes et al., 1999; U.S. EPA, 2004). Although the ages of the daycare children ranged from 1 to 6.5 years, these data represent the best information currently available for soil adherence on feet of children. OEHHA decided feet exposure during warm weather activities will be given 2/3 weighting for the 2<9 year-olds and 1/3 weighting for the 2<16 year-olds, corresponding to frequent exposure of bare feet to soil primarily in younger children.

For residential adults, a number of outdoor activities that resulted in soil contact were investigated (U.S. EPA, 2004; 2011). Among these activities, gardeners were chosen to estimate body part-specific soil adherence for adults (Table 6.9). Outdoor gardening represents not only one of the more common activities resulting in soil contact, but is also a high-end soil contact activity relative to some of the other outdoor activities examined.

In addition, a number of soil contact activities by adult workers have been examined for soil adherence (U.S. EPA, 2004). The calculated geometric mean weighted soil adherence factors from these data range from 0.02 (grounds keepers) to 0.6 mg/cm^2 (pipe layers in wet soil). Soil adherence values for adult workers in Table 6.9 were based on utility workers, as soil adherence in this line of work appears to be near the median for soil-contact related jobs presented by the U.S. EPA report.

Table 6.9. Body Part-Specific Soil Adherence Factors (in g/m^2) Resulting from Common Outdoor Activities in Children and Adults

	Children 0<2 years	Children 2<9 years	Children 2<16 years	Residential Adults	Adult Workers
Activity	General outdoor play	Sports, play in wet & dry soil	Sports, play in wet & dry soil	Gardening	Utility workers
Hands	1.334	5.919	5.919	3.179	3.487
Face	0.063[a]	0.082	0.082	0.574	1.102
Forearms	0.306	0.228	0.228	0.819	3.279
Lower legs	0.183	1.332	1.332	0.42	na[b]
Feet	0.744	1.23	0.41	na	na

[a] No soil adherence data for the face are available for young children. Soil adherence data for the face in 8-12 year old children playing in wet and dry soil were used as a surrogate.
[b] Not applicable
[c] Soil adherence to bare feet based on 1 to 6.5 year olds. Exposure reduced in 2<9 and 2<16 age groups due to less frequent exposure of bare feet in older children.

There are a number of limitations in these types of soil adherence studies that may result in greater or lesser dermal absorption of contaminants in contact with skin. Equation 6-1 assumes uniform soil coverage over the specific body-parts exposed. Gardening studies in a greenhouse using soil amended with fluorescent marker shows that soil contact is uneven and occurs most predictably on those specific body parts, such as hands and knees, that routinely come in direct contact with surfaces (Kissel et al., 1998). This is potentially significant because contaminant absorption is likely reduced in absolute terms as contact area is reduced and as a percent of total contaminant available as soil loading increases beyond monolayer coverage (Duff and Kissel, 1996). As discussed in greater detail in Appendix F, increasing soil loading beyond monolayer coverage will likely reduce fractional absorption of a chemical in soil, as a portion of the soil-bound chemical will not be in direct contact with skin.

Alternatively, there are factors related to soil loading that may underestimate adherence or chemical absorption estimates. A potential underestimation of risk is that hands were washed before hand press studies to estimate pre-loading soil levels (Kissel et al., 1996; Kissel et al., 1998b). Choate et al. (2006) observed that nonwashed hands had considerably greater soil loading after exposure to soil when compared to soil loading on recently washed hands. The lower adhered mass on prewashed hands was probably due to the removal of oils from the skin that aid in the adherence of soil particles. In addition, Sheppard and Evenden (1992) observed a 30% increase in the concentration of a contaminant in soil adhering to the hands compared to the bulk soil that the hands were pressed in. Sparingly soluble contaminants were observed to accumulate in the clay fraction of the bulk soil, characterized as the smallest particles in soil, which was the fraction adhering to hands in greatest abundance.

6.4.5 Duration and Frequency of Exposure to Contaminated Soil

Frequencies (in days/year) and durations (in hours/day) of soil exposures have not been well characterized in past studies. Recent surveys of adult and child activity patterns in relation to soil contact behavior are now available to help reduce the uncertainty associated with these variates. Regarding soil contact duration, the ABS of a particular chemical is dependent on duration of exposure. Thus, dermal absorption studies that most closely reflect the expected duration of soil contact are the most useful for estimating a chemical-specific ABS.

6.4.5.1 Exposure Duration

US EPA (2004) recommends a soil exposure time of 24 hrs and one soil exposure event per day. The exposure duration of 24 hrs assumes soil adhered to skin for 24-hrs starting from the time of first soil contact with skin to soil removal by hand washing and bathing.

One event per day can be defined as one period of exposure to soil per day. Algorithms have also been developed to assess multiple exposure events per day, which can be thought of as replenishment or replacement with a fresh layer of soil on skin (Bunge and Parks, 1997). If soil replacement is frequent enough, the soil concentration is not depleted before the next exposure, and the concentration remains essentially constant for the entire exposure period. Notably, activities involving multiple soil contacts may be better represented by a single contact scenario, if soil from the initial contact interferes with direct exposure to subsequent soil encounters. For the purposes of simplicity, one exposure event per day will be synonymous to a daily exposure, with the assumption that soil depletion of the chemical does not occur before removal from the skin with washing.

For children, exposure durations of 24 hrs are supported by national survey data reported in Wong et al. (2000b) which showed a median child bathing of one time per day. Similarly, regional data from Washington and Oregon reported median child bathing of 7 times per week. The 5th percentile for bathing was 2 and 3 times/week for cold and warm weather, respectively. However, Shoaf et al. (2005) reported a median value of two times per week for child bathing. The deviance from the national survey results was considered to be due to parents being more relaxed in interviews and less inclined to report conservative estimates.

Hand washings were more frequent than bathing among children. Wong et al. (2000b) reported median hand washing of 3 to 5 times per day in the national survey and a median hand washing of 4 times per day in the regional survey. The 5th percentile for hand washing was 2 times/day. Again, Shoaf et al. (2005) reported a less frequent median value of one time per day for hand washings. Videotaping of children's microactivity patterns by Freeman et al. (2001) also tends to support fewer hand washings per day than the national and regional surveys reported by Wong et al. (2000b).

Considering that hands tend to have higher soil loadings than other parts of the body, except perhaps the feet, but are washed more frequently than other body parts, 24 hr exposure to contaminated soil is supported by OEHHA as a reasonable estimate for an overall default assumption for exposure duration. This health protective approach is not considered overly conservative given that some studies show bathing behaviors in children may be as few as 2 times per week.

National and regional bathing and hand washing patterns in adults were reported by Garlock et al. (1999). Nearly all respondents in both surveys (72 to 99%) reported washing hands right away after soil contact activities including gardening, yard work, team sports and home repair and digging. Bathing was reported to occur mainly within 1 hr or later in the day after an activity. Only 1 to 8% did not bathe until the next day. Similar to the child bathing/hand washing survey data, the authors cautioned that the washing/bathing findings may be biased towards more socially desirable responses and should be interpreted with caution. Accordingly, the health protective assumption is to also use a soil contact duration of 24 hrs for adults, as recommended by U.S. EPA (2004).

The duration of the activity does not appear to be a good predictor of soil loading. Kissel et al. (1998) noted that initial soil contact involves a substantial portion of key body parts and is followed by continual gain and loss of soil during activity due to abrasion of skin surfaces. Soil amended with fluorescent marker does suggest increasing involvement of skin surfaces with time, but this outcome was not clearly reflected in the gravimetric results.

6.4.5.2 Exposure Frequency

Soil exposure frequency is the final parameter of significance in these exposure estimates. Prior research by Hawley (1985) based estimates for frequency of contact with soils largely on professional judgment. The U.S. EPA (1992) used Hawley's estimate in arriving at a default value for frequency of contact with soil of 40 events (days) per year as typical for adults, with a high-end estimate of 350 events per year. Hawley also estimated soil contact in young (<2-5 years of age) and older children at 130 events per year. In the revised U.S. EPA dermal risk assessment guidelines (U.S. EPA, 2004), a reasonable maximum exposure (RME) frequency for a residential scenario is 350 days/year for both adults and children.

The Soil Contact Surveys in adults (Garlock et al., 1999) and children (Wong et al., 2000b) provided more specific estimates of time or days spent involved in outdoor activities that may result in soil contact. For the child Soil Contact Survey, adult participants with children recorded outdoor play activities of their children in both warm and cold weather. The play participation rate was 73.5% of all children surveyed. The term "play" or "player" referred specifically to participation in outdoor play on bare soil or mixed grass and soil. Of the 500 children reported to play outdoors, 407 were reported to play outdoors during warm weather months and 390 were reported to play outdoors in cold months. Child players in both seasons were 57.4%.

The child frequency in days/week and hours/day for participants of outdoor play activities is shown in Table 6.10. Among child players, the median play frequency was 7 days/week in warm weather (April-October) and 3 days/week in cold weather (November-March). Arithmetic or geometric means were not reported in the study.

Table 6.10. Frequency of Outdoor Activities with Soil Contact Among Child* Participants in Warm and Cold Climates

Percentile	Cold Months (November-March)		Warm Months (April-October)	
	days/week	hours/day	days/week	hours/day
5	1	1	2	1
50	3	1	7	3
95	7	4	7	8

* Data from Wong et al. (2000b) for children <18 years of age

The exposure frequencies of outdoor play activities in days/week were multiplied by 50 weeks/year (assumes a two-week vacation per year away from the contaminated environment) to arrive at exposure frequencies in days/year (Table 6.11). For a mixed climate, outdoor play activity in days/year was calculated as 7 months of warm climate (e.g., April-October) and 5 months of cold climate (e.g., November-March), with the assumption of one week vacation away from the contaminated environment during each of the cold and warm climate periods.

Table 6.11. Estimated Frequency of Outdoor Activities with Soil Contact in Days/Year for Children <18 Years of Age*

Percentile	Cold	Mixed	Warm
5	50	60	100
50	150	267	350
95	350	350	350

* Extrapolated from data of Wong et al. (2000b)

For adults, outdoor activities in the Soil Contact Survey by Garlock et al. (1999) were categorized as (1) gardening, (2) other yardwork, (3) team sports, and (4) home repair involving digging. The reported participation rate for the first three activities ranged from 79 to 89% while that for the last activity was 30 and 18% for regional and national surveys, respectively. The report presented activity frequency for warm and cold climates, with climate defined by the survey respondents. Results were presented for “doers”, or participants, of the activity as well as all survey respondents. The survey was conducted on a national basis and for a regional area around Hanford, Washington. Because the Hanford area does not get the extreme weather conditions that some areas of the nation outside of California do, the Hanford area data were considered more likely representative of California than the national data. For three of the activities, gardening, other yardwork, and team sports, the results were presented in hours/month. These soil contact frequency data are not directly applicable to the Hot Spots dermal exposure algorithm because the algorithm requires a different unit of measure

(days/year). The frequency of each of these three activities was combined and the results are presented in Table 6.12.

Table 6.12. Total Reported Activity Duration (hrs/mo) Among Adult Participants of Three Activities: Gardening, Other Yard Work, and Team Sports[a]

Hanford (regional) Survey[b]		
Percentile	Cold	Warm
5	1	4
50	6	27
95	31	126
National Survey		
Percentile	Cold	Warm
5	2	4
50	9	22
95	130	108

[a] Data from Garlock et al. (1999)
[b] Participants of regional survey were from counties in Oregon and Washington surrounding the Hanford Nuclear Reservation.

The fourth activity surveyed by Garlock et al. (1999), home repair involving digging, was reported in event days per season. No statistical difference was found between the two survey regions in terms of event days/season among participants for this activity. OEHHA chose not to use the "home repair involving digging" activity data because these data add uncertainty (significant bias may exist in the "digging" data due to the low participation rate) with only small gain in sample size. Table 6.13 presents the results for the home repair involving digging activity.

Table 6.13. Frequency of Home Repair Involving Digging in Events/Season (Days/Season)

	Cold	**Warm**
	Hanford	
50th percentile	3	4
95th percentile	24	28
	National	
50th percentile	4	6
95th percentile	35	31

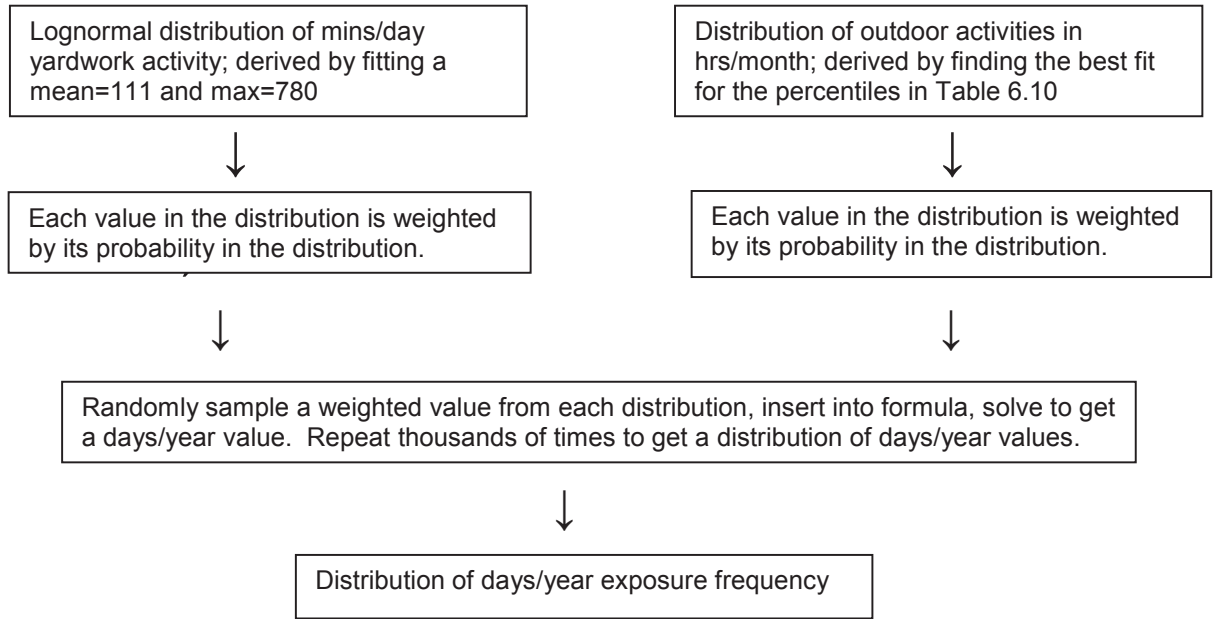
OEHHA chose to use the first three of the Garlock et al. activities (gardening, other yardwork, and team sports) for estimating soil contact frequency of adults. Using Monte Carlo simulation in Crystal Ball (Decisioneering, 2008), OEHHA calculated the best fit distribution for exposure frequency in hours/month for each climate (Table 6.12). In order to use these distributions for the exposure variate in these guidelines, the units need to be converted from hours/month to days/year. To do so, a similar activity survey by Jenkins et al. (1992) was employed. The Jenkins et al. study was a statewide survey of Californians' activity patterns, including "yard work/outdoor chores." Results were

reported in minutes/day and were given for both participants of the activity as well as extrapolated to the population. OEHHA used only the participant results to convert the Garlock et al. study's hours/month data to estimates of days/year. The following formula was used for the conversion:

Days/year = (hrs/mo * 60 mins/hr *12 mos/yr) / (mins/day)

For the time spent by California participants in the "yardwork" activities, Jenkins et al. reported a mean and maximum of 111 and 780 minutes/day, respectively. We fit a lognormal distribution to the mean and maximum values using Monte Carlo simulation (Decisioneering, 2008). For this fit, we considered the maximum to be the 99th percentile. We applied Monte Carlo methods to solve the above formula using the minutes/day and hours/month distributions. We repeated the Monte Carlo analysis of the formula for each climate. As was done for the child exposure frequencies, a mixed climate was considered to have seven months of warm climate (e.g., April-October) and five months of cold climate (e.g., November-March). Diagram 1 outlines the derivation of the distribution of days per year.

Diagram 1. Derivation of distribution of days/year using Monte Carlo methods



In order to perform a Monte Carlo analysis, we assumed a correlation exists between the number of minutes per day and the number of hours per month spent in outdoor activities. We also assumed a maximum exposure frequency of 350 days/year in the analyses. The analyses resulted in distributions of days/year for each climate (Table 6.14).

Table 6.14. Days/Year of Soil Contact Activities by Adults*

Climate	Cold	Mixed	Warm
Mean	97	150	168
Percentiles 5th	11	25	31
50th	70	135	161
75th	140	220	241
90th	227	290	302
95th	276	318	326
99th	331	343	345

* Derived from data of Garlock et al. (1999) and Jenkins et al. (1992)

Several potential limitations exist for using an unrelated activity survey to estimate exposure frequency in days/year from the Soil Contact Survey. The category yard work/outdoor chores in the California survey may include activities not involving soil contact, and the two survey populations (i.e., Jenkins' California survey and Garlock's regional/national survey) were mainly from different states. The Jenkins study included participants age >11 years, whereas the adult Soil Contact Survey was conducted with adults 18 years and older. However, these survey data together provide the best available estimate for daily exposure to soil in California resulting from common outdoor activities.

Although specific soil exposure frequency of adult workers was not part of the Soil Contact Survey, a reasonable estimate would assume exposure five d/wk with roughly two weeks off per year, regardless of the California climate region, resulting in an exposure frequency of 250 d/yr. U.S. EPA (2004) uses 350 d/yr as a Reasonably Maximally Exposed individual for industrial workers, and an exposure frequency of 219 d/yr as a central tendency for this variate.

Soil exposure frequency estimates in d/yr for use in Hot Spots programs are summarized below in Table 6.15. The exposure frequency percentiles from the child Soil Contact Survey are most representative for children in the 2<9 and 2<16 year age group. Only about 10% of the children in the Survey were under 2 yrs of age. For the 0<2 year age group, as noted above, Wong et al. (2000b) observed that most newborns (20% or less) up to the first year after birth generally stay indoors and are not exposed to outdoor surfaces with bare dirt. However, most children age 1<2 years participate in outdoor play activities, similar to older children.

As discussed above in Section 6.3.3, about 30% of indoor dust is composed of soil that is brought in from outside. The tendency of infants to play on the floor and be exposed to soil in the dust is much greater when compared to older children. Although infants spend significantly less time outdoors than older children, they may be exposed to contaminated soil via indoor dust as often as older children are exposed to soil outdoors. To address this issue, which involves a sensitive age group, OEHHA used a health-protective approach by assuming that the same exposure frequency occurred for the 0<2 age group as the older child age groups (Table 6.15).

Table 6.15. Cumulative Probability Distributions of Soil Exposure Frequency for Children and Adults in Days/Year

Age Group	Cumulative Probability	Warm Climate	Mixed Climate	Cold Climate
0<2 years	5% 50% 95%	100 350 350	79 267 350	50 150 350
2< 9 and 2<16 years	5% 50% 95%	100 350 350	79 267 350	50 150 350
Adult – residential	5th 50th 95th	31 165 326	25 137 318	11 70 276
Adult – offsite worker	central tendency	250	250	250

6.5 *Point Estimates and Stochastic Approach for Dermal Dose Assessment*

The dermal exposure pathway generally contributes only a small portion of the risk of airborne substances under the typical facility operation and exposure scenarios in the Air Toxics "Hot Spots" program. In the previous edition of this exposure guidelines document (OEHHA, 2000), OEHHA recommended using specified average and high-end point estimate values for four of the variates in equation 6-1:

body weight (Table 6.5)

exposed surface area of skin (SA) (Table 6.5)

soil load on skin (SL) (Table 6.9)

frequency of exposure (EF) (Table 6.15)

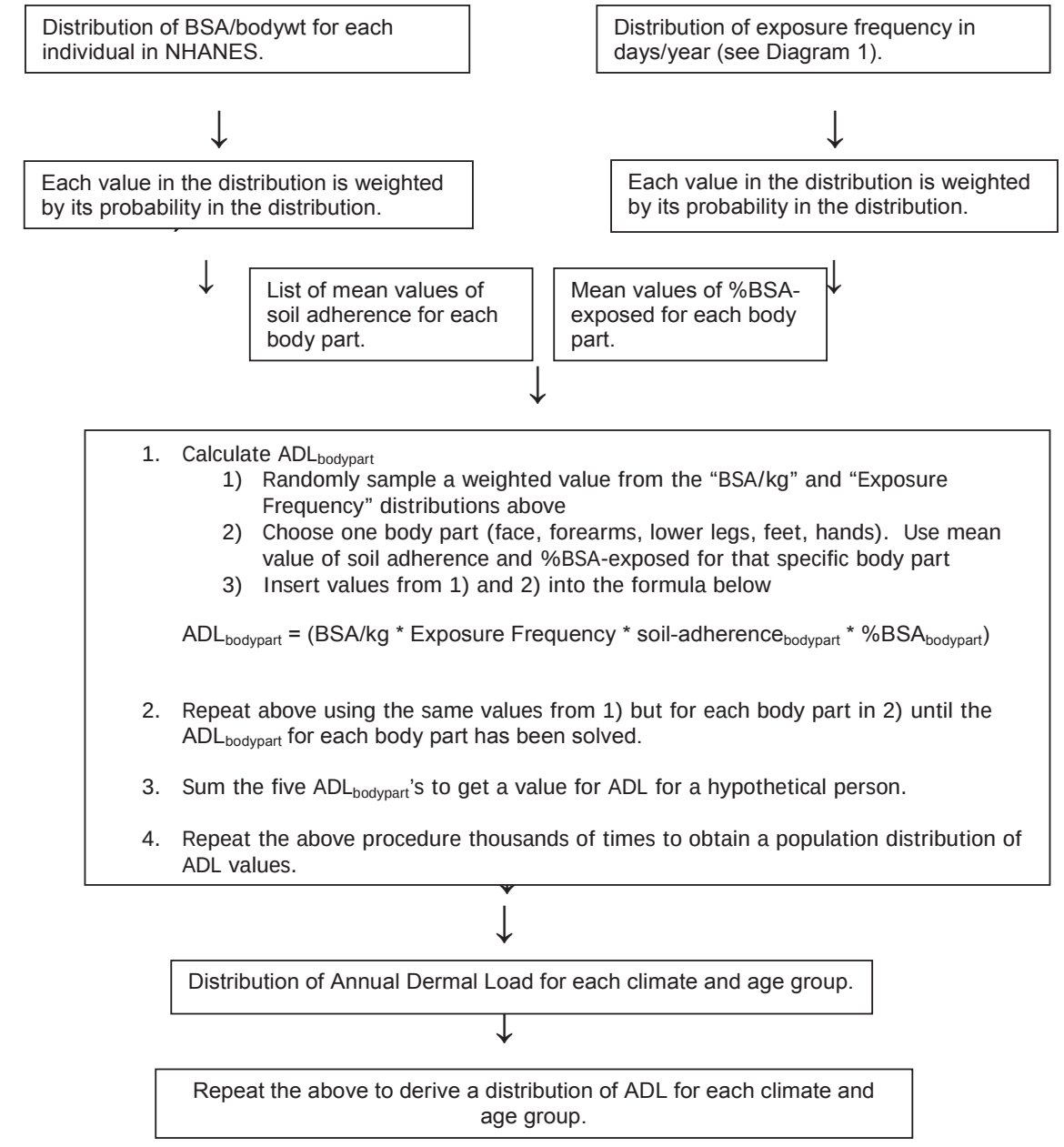
As explained in Section 6.3, OEHHA created a new variate, "annual dermal load", or ADL, which is a composite of the body surface area (BSA) per kg body weight, exposure frequency, and soil adherence variates. Point estimates from the composite "annual dermal load" can be used for point estimate assessments while parameters and information on the type of distribution (e.g., lognormal) can be used for stochastic assessments.

Distributional data are available for the body surface area per kg of body weight (BSA/BW) and exposure frequency variates. Thus, a stochastic approach could be used to derive a distribution by combining these variates. On the other hand, only point estimates for soil loading and percent of surface area for specific body parts for activities that result in soil contact are available. These constant values (means) can be used in the stochastic derivation of a composite distribution because they will not affect the distributional type or shape of the combined BSA/KG and exposure frequency distribution. Using a Monte Carlo simulation in Crystal Ball (Decisioneering, 2008) a distribution for the ADL was derived combining these variates. The ADL is in units of mg of soil loaded onto skin per kg body weight per year (mg / kg-yr)

To derive a distribution of ADL values that can be used to stochastically derive dermal dose, nationally representative values of "BSA per kg body weight" and "exposure frequency" distribution data are used together with mean values of "soil adherence" and "%BSA-exposed". For each age group and climate, a value is sampled from each of the "BSA/BW" and "Exposure Frequency" distributions based on its probability in the distribution. These values are multiplied by the mean "soil adherence" and "%BSA-exposed" values for a given body part (and age group and climate). This product gives an ADL for that body part ($ADL_{bodypart}$). This process is repeated for up to four more times using the same "BSA/kg" and "Exposure Frequency" values but with "soil adherence" and "%BSA-exposed" values for a different body part each time. This results in five $ADL_{bodypart}$ values, one for each of face, hands, feet, forearms, and lower legs. The five $ADL_{bodypart}$'s are summed to give an ADL for a hypothetical person for a specific age group and climate.

This process of deriving an ADL for a hypothetical person is repeated thousands of times to give a distribution of ADL values (for that age group and climate). This distribution of ADL values has incorporated the population distribution information from the "body surface area normalized to body weight" and "exposure frequency" variates. Diagram 2 outlines the procedure of stochastically estimating a probability distribution of ADL values and Table 6.2 in Section 6.2 above present the stochastically-derived ADL distributions for each of the five age groupings.

Diagram 2. Derivation of Annual Dermal Load (ADL) using Monte Carlo methodology



6.6 Dermal Uptake Equations by Other Agencies

6.6.1 U.S. EPA Exposure Estimates

The U.S. EPA (2004) suggested using the following equation for estimating dermal exposure to chemicals from soil:

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT} \quad \textbf{(Eq. 6-12)}$$

where:

DAD = dermal absorbed dose (mg/kg-d)
DAevent = absorbed dose per event (mg/cm2-event)
EV = event frequency (events/d)
EF = exposure frequency (d/yr)
ED = exposure duration (yrs)
SA = skin surface area available for contact (cm2)
BW = body weight (kg)
AT = averaging time (d); for noncarcinogenic effects, AT = ED x 365 d/yr
for carcinogenic effects, AT = 70 yrs or 25,550 d

The absorbed dose per event, DA_{event}, uses a percent absorption calculation which considers chemical-specific absorption estimates and the soil type and skin adherence factor:

$$DA_{event} = C_{soil} \times CF \times AF \times ABS_d \quad \textbf{Eq. 6-13}$$

where:

DAevent = absorbed dose per event (mg/cm2-event)
Csoil = chemical concentration in soil (mg/kg)
CF = conversion factor (10-6/mg)
AF = adherence factor of soil to skin (mg/cm2-event)
ABSd = dermal absorption fraction

US EPA (2004) recommends an age-adjusted dermal exposure factor (SFS_{adj}) when dermal exposure is expected throughout childhood and into the adult years. This accounts for changes in surface area, body weight and adherence factors over time. The SFS_{adj} is calculated using the US EPA age groupings of 1-6 years (children) and 7-31 years (adult):

$$SFS_{adj} = \frac{(SA_{1\text{-}6})(AF_{1\text{-}6})(ED_{1\text{-}6})}{(BW_{1\text{-}6})} + \frac{(SA_{7\text{-}31})(AF_{7\text{-}31})(ED_{7\text{-}31})}{(BW_{7\text{-}31})} \quad \textbf{Eq. 6-14}$$

where:

SFSadj = age-adjusted dermal exposure factor (mg-yrs/kg-events)
AF1-6 = adherence factor of soil to skin for a child 1-6 yrs (mg/cm^2-event)
AF7-31 = adherence factor of soil to skin for an adult 7-31 yrs (mg/cm^2-event)
SA1-6 = skin surface area available for contact during ages 1-6 yrs (cm^2)
SA7-31 = skin surface area available for contact during ages 7-31 yrs (cm^2)
ED1-6 = exposure duration during ages 1-6 (yrs)
ED7-31 = exposure duration during ages 7-31 (yrs)
BW1-6 = average body weight during ages 1-6 yrs (kg)
BW7-31 = average body weight during ages 7-31 yrs (kg)

6.6.2 Cal/EPA Department of Pesticide Regulation Guidance for the Preparation of Human Pesticide Exposure Assessment Documents

The Department of Pesticide Regulation (DPR) has developed guidelines for exposure assessment that include a dermal absorption component for occupational exposure to pesticides. The guidelines are currently under revision and have not been posted as of this writing (DPR, 2007). Previously, the DPR dermal absorption estimate procedure used a default uptake value of 100% unless a pesticide registrant chooses to collect specific data. However, DPR has revised the dermal absorption default for pesticides to 50% absorption on the basis of a survey of previous pesticide absorption studies, and the finding that 100% absorption in humans has not been observed for any pesticide (DPR, 1996). Experimental absorption values prior to the current revision process were calculated from *in vivo* data as follows:

$$\text{Percent dermal absorption} = \frac{\text{Applied dose} - \text{Unabsorbed dose}}{\text{Applied dose}} \times 100 \quad \text{Eq. 6-15}$$

The absorbed portion may also be calculated from the sum of all residues found in excreta, expired air, blood, carcass, and skin at the site of application (after washing), or estimated from the asymptotic plot of all (radioactively-labelled) residues excreted in feces, urine, and air. Absorption rate in an animal experiment in vivo is assumed to be applicable to humans, unless it can be corrected with the ratio of *in vitro* uptake in animal vs. human skin.

6.6.3 CalTOX

The Department of Toxic Substances Control (DTSC) developed the CalTOX computer program to estimate potential exposure to chemicals at hazardous waste sites (DTSC, 1993; 1994). The program incorporates variable parameters in each exposure pathway to estimate multimedia uptake of a chemical by all exposure routes, with the uncertainty assumptions explicitly presented. The program provides a mechanism for screening health risks at hazardous waste sites. CalTOX incorporates explicit assumptions for distributions of all exposure parameters, but with regard to dermal exposure, is focused on dermal uptake of contaminants poured directly onto soil, and at concentrations higher than one would anticipate from airborne deposition. The basic uptake model is:

$$ADD = AR_s \times SA_b \times 0.3 \times 15 \times EF_{sl}/365 \times C_g \qquad \text{(Eq.6-16)}$$

where:

- ADD = average daily dose in mg/kg-day, for one exposure event/d
- AR_s = ratio of the absorbed dose to the soil concentration, e.g., uptake per unit area of skin per unit concentration in soil in mg/cm^2 per mg/cm^3
- SA_b = body surface area per kg, in m^2/kg
- 0.3 = fraction of total body exposed to soil, default value; coefficient of variation (CV) assumed = 0.04
- 15 = conversion factor for soil density, in kg/cm-m^2, based on a soil bulk density of 1500 kg/m^3
- $EF_{sl}/365$ = exposure frequency in days/year, divided by the days in a year; mean assumed = 137, CV = 0.6
- C_g = chemical concentration in soil (mg chemical/kg soil).

The absorbed dose for each event is calculated with the following equation:

$$AR_s = T_s \times \left\{ 1 - \exp\left[\frac{-K^s_p \times ET_{sl}}{T_s} \right] \right\} \qquad \text{(Eq. 6-17)}$$

where:

AR_s = skin uptake as defined above

- T_s = thickness of soil layer on skin, in cm
- K^s_p = permeability factor for chemical movement from soil into skin, in cm/hr
- ET_{sl} = soil exposure time, in hrs/d

The thickness of the soil layer on skin, T_s, depends on the soil loading factor, which was assumed to be 0.5 mg/cm^2, with CV = 0.4. The permeability factor, K^s_p, is derived from permeability values, K_p, from water, with a correction for decreased skin hydration. ET_{sl} is set equal to half the total exposure time at home.

6.7 References

Black K, Shalat SL, Freeman NC, Jimenez M, Donnelly KC and Calvin JA (2005). Children's mouthing and food-handling behavior in an agricultural community on the US/Mexico border. J Expo Anal Environ Epidemiol 15(3): 244-51.

Bunge AL and Parks JM (1997). Predicting dermal absorption from contact with chemically contaminated soils. Dwyer FJ, Doane TR and Hinman ML, eds., ASTM STP, 1317. Environmental Toxicology and Risk Assessment: Modeling and Risk Assessment (Sixth Volume); Sixth Symposium on Environmental Toxicology and Risk Assessment, Orlando, FL, USA, April 15-18, 1996. American Society for Testing and Materials: Philadelphia, PA, pp. 227-244.

CARB (2003). California Air Resources Board. Hot Spots Analysis Reporting Program. California Environmental Protection Agency, Sacramento, CA. Online at: http://www.arb.ca.gov/toxics/harp/harp.htm. .

CDC. (2007). Centers for Disease Control and Prevention (CDC). National Center for Health Statistics (NCHS). National Health and Nutrition Examination Survey Data. Hyattsville, MD: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 1999-2004. Available online a*t: www.cdc.gov/nchs/about/major/nhanes/datalink.htm*.

Chang SK and Riviere JE (1991). Percutaneous absorption of parathion in vitro in porcine skin: effects of dose, temperature, humidity, and perfusate composition on absorptive flux. Fundam Appl Toxicol 17(3): 494-504.

Choate LM, Ranville JF, Bunge AL and Macalady DL (2006). Dermally adhered soil: 1. Amount and particle-size distribution. Integr Environ Assess Manag 2(4): 375-84.

Culbard EB and Johnson LR (1984). Elevated arsenic concentrations in house dusts located in a mineralized area of Southwest England: implication for human health. Trace Subst Environ Health 18: 311-19.

Culbard EB, Thornton I, Watt J, Wheatley M and Moorcroft ST, M. (1988). Metal contamination in British urban streets. J Environ Qual 17: 226-34.

Davies BE, Elwood PC, Gallacher J and Ginnever RC (1985). The relationships between heavy metals in garden soils and house dusts in an old lead mining area of North Wales, Great Britain. Environ Pollut (Series B) 9: 255-66.

Decisioneering (2008). Crystal Ball, Version 11, Fusion Edition, Oracle Corporation, Redwood Shores, CA.

Dor F, Jongeneelen F, Zmirou D, Empereur-Bissonnet P, Nedellec V, Haguenoer JM, Person A, Ferguson C and Dab W (2000). Feasibility of assessing dermal exposure to

PAHs of workers on gaswork sites--the SOLEX study. Sci Total Environ 263(1-3): 47-55.

DPR (1996). Revised Policy on Dermal Absorption Default for Pesticides, Memorandum. Department of Pesticide Regulation, California Environmental Protection Agency, Sacramento, CA. HSM-96005. Online at: www.cdpr.ca.gov/docs/whs/eampmemo.htm#exposuredocument.

DPR (2007). Exposure Assessment Guidelines HS-1612, Exposure Assessment and Mitigation policies and procedures webpage. Department of Pesticide Regulation, California Environmental Protection Agency, Sacramento, CA. Online at: www.cdpr.ca.gov/docs/whs/eampmemo.htm.

DTSC (1993). CalTOX, A Multimedia Total Exposure Model for Hazardous Waste Sites, Part III: The Multiple Pathway Exposure Model. The Office of Scientific Affairs, Department of Toxic Substances Control, California Environmental Protection Agency, Sacramento, CA. Online at: www.dtsc.ca.gov/AssessingRisk/ctox_model.cfm.

DTSC (1994). Preliminary Endangerment Assessment Guidance Manual (A guidance manual for evaluating hazardous substances release sites). Chapter 3, Preparation of the PEA report; Appendix A, Tables for use with screening evaluations; Appendix B, Derivations for equations and complete equation for VOC emission model. Department of Toxic Substances Control, California Environmental Protection Agency, Sacramento, CA. Online at: www.dtsc.ca.gov/PublicationsForms/pubs_index.cfm.

Duff RM and Kissel JC (1996). Effect of soil loading on dermal absorption efficiency from contaminated soils. J Toxicol Environ Health 48(1): 93-106.

Durnin JVGA (1959). The use of surface area and of body-weight as standards of reference in studies on human energy expenditure. Br J Nutr 13: 68-71.

Fergusson JE and Kim ND (1991). Trace elements in street and house dusts: sources and speciation. Sci Total Environ 100 Spec No: 125-50.

Freeman NC, Jimenez M, Reed KJ, Gurunathan S, Edwards RD, Roy A, Adgate JL, Pellizzari ED, Quackenboss J, Sexton K and Lioy PJ (2001). Quantitative analysis of children's microactivity patterns: The Minnesota Children's Pesticide Exposure Study. J Expo Anal Environ Epidemiol 11(6): 501-9.

Garlock TJ, Shirai JH and Kissel JC (1999). Adult responses to a survey of soil contact-related behaviors. J Expo Anal Environ Epidemiol 9(2): 134-42.

Gehan EA and George SL (1970). Estimation of human body surface area from height and weight. Cancer Chemother Rep 54(4): 225-35.

Hawley JK (1985). Assessment of health risk from exposure to contaminated soil. Risk Anal 5(4): 289-302.

Holmes KK, Jr., Shirai JH, Richter KY and Kissel JC (1999). Field measurement of dermal soil loadings in occupational and recreational activities. Environ Res 80(2 Pt 1): 148-57.

Jenkins PL, Phillips TJ, Mulberg EJ and Hui SP (1992). Activity patterns of Californians: Use of and proximity to indoor pollutant sources. Atmos Environ 26A(12): 2141-48.

Johnson JE and Kissel JC (1996). Prevalence of dermal pathway dominance in risk assessment of contaminated soils: A survey of superfund risk assessments, 1989-1992. Hum Ecol Risk Assess 2(2): 356-365.

Kissel JC, Richter KY and Fenske RA (1996). Factors affecting soil adherence to skin in hand-press trials. Bull Environ Contam Toxicol 56(5): 722-8.

Kissel JC, Richter KY and Fenske RA (1996b). Field measurement of dermal soil loading attributable to various activities: implications for exposure assessment. Risk Anal 16(1): 115-25.

Kissel JC, Shirai JH, Richter KY and Fenske RA (1998). Investigation of dermal contact with soil in controlled trials. J Soil Contam 7(6): 737-52.

Kissel JC, Shirai JH, Richter KY and Fenske RA (1998b). Empirical investigation of hand-to-mouth transfer of soil. Bull Environ Contam Toxicol 60(3): 379-86.

Maibach HI, Feldman RJ, Milby TH and Serat WF (1971). Regional variation in percutaneous penetration in man. Pesticides. Arch Environ Health 23(3): 208-11.

Nomeir AA, Markham PM, Mongan AL, Silveira DM and Chadwick M (1992). Effect of dose on the percutaneous absorption of 2- and 4-chloronitrobenzene in rats. Drug Metab Dispos 20(3): 436-9.

OEHHA (2000). Air Toxics Hot Spots Program Risk Assessment Guidelines. Part IV. Exposure Assessment and Stochastic Technical Support Document. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA. Available online at: http://www.oehha.ca.gov.

OEHHA (2009). Technical Support Document for Cancer Potency Factors:Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environrmental Health Hazard Assessment. Online at:http://www.oehha.ca.gov/air/hot_spots/2009/TSDCancerPotency.pdf.

Sartorelli P, Montomoli L, Sisinni AG, Barabesi L, Bussani R and Cherubini Di Simplicio F (2003). Percutaneous penetration of inorganic mercury from soil: an in vitro study. Bull Environ Contam Toxicol 71(6): 1091-9.

Sheppard SC and Evenden WG (1992). Concentration enrichment of sparingly soluble contaminants (U, Th and Pb) by erosion and by soil adhesion to plants and skin. Environ Geochem Health 14(4): 121-31.

Shoaf MB, Shirai JH, Kedan G, Schaum J and Kissel JC (2005). Child dermal sediment loads following play in a tide flat. J Expo Anal Environ Epidemiol 15(5): 407-12.

Stanek EJ and Calabrese EJ (1992). Soil ingestion in children: Outdoor soil or indoor dust? J Soil Contam 1(1): 1-28.

Thornton I, Culbard E, Moorcroft S, Watt J, Wheatley M and Thompson M (1985). Metals in urban dusts and soils. Environ Technol Lett 6: 137-44.

U.S. EPA. (1985). *Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessment. Final Report* U.S. Environmental Protection Agency, Washington D.C. August 1985. EPA/600/8-85/010, pp. 9-32 and B-9.

U.S. EPA (1991). OSWER Directive 9285.6-03 Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors". PB91-921314.

U.S. EPA (1992). Dermal Exposure Assessment: Principles and Applications. Interim Report. U.S. Environmental Protection Agency, Washington D.C. January 1992. EPA/600/8-91/011B, pp. 6-1 to 6-43. Available online at: http://www.epa.gov/nceawww1/pdfs/derexp.pdf.

U.S. EPA (2004). Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual (Part E, supplemental guidance for dermal risk assessment). Final. Office of Superfund Remediation and Technology Innovation, U.S. Environmental Protection Agency, Washington DC.

U.S. EPA. (2011). *Exposure Factors Handbook: 2011 Edition. U.S. Environmental Protection Agency*. EPA/600/R-090/052F, Washington DC.

Weschler CJ and Nazaroff WW (2012). SVOC exposure indoors: fresh look at dermal pathways. Indoor air.

Wester RC and Maibach HI (1985). In vivo percutaneous absorption and decontamination of pesticides in humans. J Toxicol Environ Health 16(1): 25-37.

Wong EY, Shirai JH, Garlock TJ and Kissel JC (2000a). Survey of selected activities relevant to exposures to soils. Bull Environ Contam Toxicol 65(4): 443-50.

Wong EY, Shirai JH, Garlock TJ and Kissel JC (2000b). Adult proxy responses to a survey of children's dermal soil contact activities. J Expo Anal Environ Epidemiol 10(6 Pt 1): 509-17.

7 Home Produced Food Exposure Assessment

7.1 Introduction

Semivolatile organic toxicants and toxic heavy metals emitted into the air by California facilities (e.g., dioxin and lead) are subject to deposition onto vegetation, soil, and surface water bodies. Homegrown produce can become contaminated through the deposition of the toxicant onto the surface of edible leaves, exposed edible portions of vegetables, and fruit, or, in the case of metals, may be taken up from the soil into the roots of the plant. Food animals may become contaminated from consuming contaminated vegetation (e.g., pasture, grains), water, or soil, or from inhaling the airborne toxicants. Humans may then be exposed by consuming the contaminated produce (leafy greens, fruits, vegetables), or animal products (meat, milk, and eggs).

Commercially grown produce or commercially raised beef, chicken, pork, cow's milk, and eggs come from diverse sources, so that the potential public health impacts from a single Hot Spots facility impacting a commercial operation are minimal. Therefore, only the risks from Hot Spots facility contamination of homegrown produce and home-raised beef, chicken, pork, eggs, and milk are assessed.

In order to quantify risks (cancer and chronic noncancer) from homegrown, or home raised food exposures, the dose from these sources must be determined. Dose is proportional to the consumption rate of the homegrown food items and the concentration of the toxicant in the homegrown products (i.e., produce, meat, eggs, and milk). In this chapter, we discuss and present consumption rates (both probability distributions and point estimate values) and methods to determine toxicant concentration levels for homegrown foods. The equation for determining the dose from home grown foods is shown in Equation 7.1.

7.2 Home Produced Food Exposure Recommendations

OEHHA has used the National Health and Nutrition Examination Survey (NHANES) 1999-2004 survey data to generate per capita consumption distributions for produce (exposed, leafy, protected, and root categories), meat (beef, chicken, and pork), dairy products, and eggs. The NHANES data are the most recent data available with which to estimate consumption rates for the food categories discussed and that are relatively representative of the California population. The variability in food consumption that may be associated with interindividual variability in body weight was accounted for by presenting the rates on a body weight basis.

There is uncertainty in the estimations of produce, meat, dairy products, and eggs. The consumption rates are based on a single day of surveyed food intake. One day of survey data per individual is not adequate for capturing typical intake, which means that the lower percentile is likely to be underestimated and upper percentile is overestimated. Unfortunately these data are the best representative data for the United States population.

7.2.1 Point Estimates

OEHHA is recommending that the default values presented in Table 7.1 be used, as needed, for the point estimate approach (Tier 1). These default values represent the mean and 95th percentiles of the empirical distributions presented in Tables 7.8 through 7.13. When the food pathway is a dominant pathway, and multiple homegrown produce, home raised meat, milk, and eggs categories all are assessed, the 95th percentile default consumption rate for the highest risk category (e.g. leafy produce) should be used. OEHHA recommends using the mean consumption values for the remaining categories. This procedure will help avoid overly conservative estimation of risk that would arise from assuming that a single receptor would be a high consumer of all homegrown categories.

Table 7.1 Recommended Average and High End Point Estimate Values for Home Produced Food Consumption (g/kg-day)

Food Category	Third Trimester		Ages 0<2		Ages 2<9	
Produce	Avg.	High End	Avg.	High End	Avg.	High End
Exposed	1.9	5.9	11.7	30.2	7.4	21.7
Leafy	0.9	3.2	3.8	10.8	2.5	7.9
Protected	1.7	5.8	5.9	17.5	4.7	13.3
Root	1.7	4.6	5.7	15.3	3.9	10.8
Meat						
Beef	2.0	4.8	3.9	11.3	3.5	8.6
Poultry	0.9	2.9	2.9	10.5	2.2	7.8
Pork	1.8	4.7	4.5	11.4	3.7	9.0
Milk	5.4	15.9	50.9	116	23.3	61.4
Eggs	1.6	4.2	6.1	15.0	3.9	9.4
	Ages 2>16		Ages 16<30		Ages 16-70	
Produce	Avg.	High End	Avg.	High End	Avg.	High End
Exposed	1.9	5.9	1.9	5.9	1.8	5.6
Leafy	0.9	3.2	0.9	3.2	1.1	3.4
Protected	1.7	5.8	1.7	5.8	1.6	5.2
Root	1.7	4.6	1.7	4.6	1.5	4.2
Meat						
Beef	2.0	4.8	2.0	4.8	1.7	4.4
Poultry	0.9	2.9	0.9	2.9	0.9	2.8
Pork	1.8	4.7	1.8	4.7	1.5	3.8
Milk	5.4	15.9	5.4	15.9	4.3	13.2
Eggs	1.6	4.2	1.6	4.2	1.3	3.4

[a] Food consumption values for 3rd trimester calculated by assuming that the fetus receives the same amount of contaminated food on a per kg BW basis as the mother (adult age 16 to less than 30).

7.2.2 Stochastic Approach

OEHHA is recommending that the parametric models for food consumption distributions presented in Tables 7.2 through 7.7 be used as needed in Tier III stochastic risk assessments. The methods leading to these distributions are described in Section 7.4.1.

Table 7.2 Parametric Models of Food Consumption (g/kg-day) for All Ages

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	LogN	62	11.8	11.9			
Leafy	Gamma	88			0.0	1.26	0.9664
Protected	Gamma	95			0.0	2.49	0.8076
Root	Gamma	70			0.0	1.77	1.0592
Meat							
Beef	LogN	16	1.97	1.73			
Poultry	LogN	19	1.84	1.64			
Pork	LogN	144	1.08	1.76			
Dairy	LogN	358	8.74	21			
Eggs	LogN	114	1.62	1.55			

Table 7.3 Parametric Models of Food Consumption (g/kg-day) for 0 <2 Years.

Food Category	Distrib. Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape	Like-liest
Produce								
Exposed	Gamma	60			0.01	6.56	0.830	
Leafy	Gamma	167			0.01	3.30	1.161	
Protected	LogN	67	6.03	7.31				
Root	Gamma	83			0.06	4.44	1.28	
Meat								
Beef	LogN	16	1.97	1.73				
Poultry	LogN	58	4.5	4.08				
Pork	LogN	230	3.00	4.46				
Dairy	Max Ext.	169				27.82		33.79
Eggs	LogN	172	6.11	4.21				

Table 7.4 Parametric Models of Food Consumption (g/kg-day) for Ages 2<9

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape	Rate
Produce								
Exposed	Exponential	206						0.14
Leafy	LogN	127	2.64	3.89				
Protected	Weibull	68			0.02	4.76	1.063	
Root	LogN	60	3.95	3.85				
Meat								
Beef	LogN	35	3.55	2.79				
Poultry	LogN	17	3.71	2.67				
Pork	LogN	66	2.25	2.84				
Milk	LogN	12	23.4	20.78				
Eggs	LogN	38	3.93	3.00				

Table 7.5 Parametric Models of Food Consumption (g/kg-day) for Ages 2<16

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	Gamma	60			0.01	6.54	0.8325
Leafy	LogN	68	1.83	2.91			
Protected	Gamma	47			0.00	3.69	0.9729
Root	LogN	51	3.10	3.44			
Meat							
Beef	LogN	10	2.96	2.49			
Poultry	LogN	27	2.98	2.52			
Pork	LogN	48	1.84	2.79			
Milk	LogN	35	16.8	19.2			
Eggs	LogN	71	3.16	2.95			

Table 7.6 Parametric Models of Food Consumption (g/kg-day) for Ages 16-30[a]

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	Gamma	70			0.01	2.05	0.9220
Leafy	Weibull	191			0.00	0.88	0.8732
Protected	LogN	93	1.81	3.31			
Root	LogN	43	1.69	1.69			
Meat							
Beef	LogN	26	1.98	1.54			
Poultry	LogN	26	1.80	1.42			
Pork	LogN	242	1.01	1.74			
Milk	Gamma	22			0.02	5.66	0.9421
Eggs	LogN	29	1.55	1.36			

[a] These distributions are also recommended for the third trimester.

Table 7.7 Parametric Models of Food Consumption (g/kg-day) for Ages 16-70

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	Gamma	148			0.01	2.07	0.8628
Leafy	Gamma	83			0.00	1.15	0.9713
Protected	Gamma	78			0.01	1.90	0.8325
Root	Gamma	14			0.00	1.28	1.166
Meat							
Beef	LogN	20	1.75	1.40			
Poultry	LogN	18	1.53	1.18			
Pork	LogN	190	0.97	1.59			
Milk	Gamma	20			0.00	4.50	0.9627
Eggs	LogN	30	1.3	1.01			

7.3 Home Grown Food Intake Dose

7.3.1 Point Estimate (Deterministic) Algorithm

The general algorithm for estimating dose via the food pathway is as follows:

$$DOSEfood = (Cf * IF * GRAF * L)* EF* (1 \times 10^{-6}) \quad \textbf{(Eq. 7-1)}$$

Where: DOSEfood = (mg/kg-day)
- Cf = concentration of toxicant in food type F (μg/kg)
- IF = consumption for food type F (g/kg body weight per day)
- GRAF = gastrointestinal relative absorption factor (unitless)
- L = fraction of food type consumed from contaminated source (unitless)
- 1×10^{-6} = conversion factor (μg/kg to mg/g) for Cf term
- EF = exposure frequency (days/365 days)

The gastrointestinal relative absorption factor (GRAF) is currently only available for dioxins and furans. In most cases, a GRAF factor of one is used because it assumed that absorption would be similar in the animal oral studies as it would for humans consuming the contaminated food. In addition, data for estimating a GRAF are almost never available. The exposure frequency (EF) is set at 350 days per year (i.e., per 365 days) (US EPA, 1991).

For cancer risk, the risk is calculated for each age group using the appropriate age sensitivity factors (ASFs) and the chemical-specific cancer potency factor (CPF), expressed in units of $(mg/kg\text{-}day)^{-1}$.:

$$RISKfood = DOSEfood *(CPF)*ASF*ED/AT \quad \textbf{(Eq. 7-2)}$$

Exposure duration (ED) is the number of years within the age groupings. In order to accommodate the use of the ASFs (see OEHHA, 2009), the exposure for each age grouping must be separately calculated. Thus, the DOSEfood and ED are different for each age grouping. The ASF, as shown below, is 10 for the third trimester and infants 0<2 years of age, is 3 for children age 2<16 years of age, and is 1 for adults 16 to 70 years of age.

ED = exposure duration (yrs):

0.25 yrs for third trimester	(ASF = 10)
2 yrs for 0<2 age group	(ASF = 10)
7 yrs for 2<9 age group	(ASF = 3)
14 yrs for 2<16 age group	(ASF = 3)
14 yrs for 16<30 age group	(ASF = 1)
54 yrs for 16-70 age group	(ASF = 1)

AT, the averaging time for lifetime cancer risks, is 70 years in all cases. To determine lifetime cancer risks, the risks are then summed across the age groups:

$$RISKfood_{(lifetime)} = RISKfood_{(3rdtri)} + RISKfood_{(0<2\ yr)} + RISKfood_{(2<16\ yr)} + RISKfood_{(16\text{-}70yr)} \quad \textbf{(Eq. 7-3)}$$

As explained in Chapter 1, we also need to accommodate cancer risk estimates for the average (9 years) and high-end (30 years) length of time at a single residence, as well as the traditional 70 year lifetime cancer risk estimate. For example, assessing risk in a 9 year residential exposure scenario assumes exposure during the most sensitive period, from the third trimester to 9 years of age and would be presented as such:

$$RISKfood_{(9\text{-}yr\ residency)} = RISKfood_{(3rdtri)} + RISKfood_{(0<2\ yr)} + RISKfood_{(2<9\ yr)} \quad \textbf{(Eq. 7-4)}$$

For the 30-year residential exposure scenario, the risk for the 2<16 and 16<30 age group would be added in to the risk from exposures in the third trimester and from age 0<2 yr. For 70 year residency risk, Eq 7-3 would apply.

7.3.2 Stochastic Algorithm

The algorithm for the stochastic method is the same as the point estimate algorithm. Recommended distributions, as parametric model of empirical data on variability, are available to substitute for single values, where data permit.

7.4 Food Consumption Variates for the Hot Spots Exposure Model

The homegrown produce and home-raised meat, eggs, and milk pathways in the Hot Spots program are used to assess chronic noncancer risks and cancer risks. Separate consumption estimates are needed for the third trimester, 0 to <2 years, 2<16 years, 16<30 years and 30 to 70 years in g/kg body weight per day, in order to account for the greater exposure of children and the differential impact of early in life exposure.

The ideal data for such long-term exposure determinations would be recent, representative of the California population, and have repeated measures on the same individuals to characterize typical intake over time. The amount of homegrown produce, and home-raised meat, eggs and milk would be addressed. Such data are not available. The available data, while not perfect, are nonetheless useful for the purposes of chronic exposure assessment. In the next Section, we review the currently available data and discuss the reasons for our recommendations.

7.4.1 Derivation of Consumption Rates

7.4.1.1 Data

Several survey methods have been used to estimate consumption of various foods or food items by a population. These include market basket, food frequency, diary, and consumption recall methods. The USDA has conducted market basket surveys in which the amount of food that enters into the wholesale and retail markets was measured (Putnam and Allshouse, 1992). These amounts are then divided by the U.S.

population to give per capita consumption. This methodology does not allow determination of food consumption rates for individuals in the age ranges that are needed. It provides data on the amount bought at the market, not the amount consumed, which differ due to trimming, water and fat loss during processing and cooking (Putnam and Allshouse, 1992). The USDA market basket studies are thus not useful for assessing chronic exposure in our model because of these limitations.

The food frequency method asks subjects to recall the frequency with which they consumed certain food items over a previous period of time. Typically, information is collected on specific food items (e.g., green tea) or food groups (e.g., grilled red meat) that are being evaluated for their relationship to a certain disease (e.g., cancer). These surveys are conducted on relatively small groups of individuals or on large groups of a certain subpopulation (e.g., nurses in the Nurses Health Study). The food frequency method could provide very helpful information for estimating 'usual' consumption of foods that are typically consumed on a less than daily basis (e.g., berries), and for assessing intraindividual variability (Block, 1992). However, food frequency data from current studies are not representative of the general population and thus not ideal for assessing chronic exposure in the Hot Spots model.

The U.S. Department of Agriculture (USDA) conducted seven Nationwide Food Consumption Surveys (NFCS) beginning in 1935 and ending in 1987-88 that collected data on household food consumption (http://www.ars.usda.gov/Services/docs.htm). The two most recent NFCS studies (1977-78 and 1987-88) included data on individuals. Because one of our objectives for food consumption rates was that the rates reflect current dietary patterns, the NFCS were considered too old to meet our needs. The USDA also conducted a series of food consumption surveys called the Continuing Survey of Food Intake of Individuals (CSFII) (1985, 1986, 1987, 1989, 1990, 1991, 1994-96, and 1998). OEHHA used the 1989-91 CSFII data to determine distributions of food consumption rates for the previous version of the Hot Spots Exposure Assessment and Stochastic Analysis Guidelines (OEHHA, 2000).

The three days of consumption data per individual in the CSFII 1989-1991 capture typical intake better than the fewer days in more recent surveys but are still not considered a sufficient number of repeated measures for a good determination of intraindividual variability (Andersen, 2006). The CSFII 1994-96, 1998 and the National Health and Nutrition Examination Survey (NHANES) 1999-2004, with more recent data, have become available. We therefore chose to consider the more recent datasets because the advantages of the more recent data outweighed the greater number of individual measures on the same individual in the older surveys.

The CSFII 1994-1996, 1998 survey (hereafter referred to as CSFII) collected data on two non-consecutive days of consumption, 3-10 days apart, by over 20,000 individuals, while the NHANES 1999-2004 (hereafter referred to as NHANES) dataset provided only one day of consumption (with the exception of the 2004 year) on over 30,000 individuals. OEHHA considered that the two days of intake of the CSFII did not provide sufficient additional information on typical intake to outweigh the advantage of the more recent NHANES data.

Further, the number of days between data collection for each individual in the CSFII was not available in the dataset and CSFII reported that there was no standard procedure used to determine the second day of food consumption. This likely resulted in the interval between the first and second days of data collection to be widely variable

California specific food consumption data are not available. The CSFII data are available for the Pacific region, but not for California alone. Neither California-specific nor Pacific region-specific data are available for NHANES. Therefore, OEHHA chose to use the NHANES dataset since the need for the most recent data was considered more important than having data specific to California.

7.4.1.2 The NHANES Data

The NHANES uses a multistage sampling design to select individuals for the survey. Some of these stages do not use simple random sampling to select units to be surveyed (i.e., “sampled”) resulting in uneven probability and non-independent selection. Therefore, statisticians also created weights to account for these issues. These weights allow for proper estimation of variance, the standard error of the mean (SEM), and confidence intervals (CIs). These parameters (variance, SEM, CIs) estimate confidence that the value of a statistic (e.g., the mean) is the true population value. Therefore, accounting for a multistage survey design is important for estimating confidence in the numerical value of the results. This differs from the sampling weights that provided results that best represent the targeted population.

It is common that some individuals selected to participate in a survey end up either voluntarily or for other reasons, such as incomplete responses, not participating or contributing to the survey. This may result in a surveyed sample of individuals that do not reflect the targeted demographics of the survey. In NHANES, the statisticians created “sample weights” that account for non-participation. Using these weights in statistical analyses provides results that are more representative of the population.

NHANES is designed to collect the most accurate information possible. Participants are interviewed in a private setting, the mobile examination center (MEC), which consists of several mobile units specially designed and equipped for the survey. The MEC is used by NHANES to collect dietary information as well as body measurements (e.g., height, X-rays) and body specimens (e.g., urine) that are also part of the total survey for some participants. The privacy and professional setting of the MEC is thought to encourage greater accuracy in food consumption reporting. The dietary interview room of the MEC contains measuring devices (e.g., cups, spoons, photos) to help participants better estimate the amounts of various foods consumed. In 2002, NHANES implemented the automated multiple pass method, a method intended to solicit greater and more accurate recall of food consumption.

The NHANES survey is quite comprehensive in the range of prepared and non-prepared foods for which data are collected. These foods include beverages, sweets, and condiments, as well as items more commonly considered foods. Further, some

food entries contain very detailed information about the food (e.g., peaches, sliced, canned, in light syrup).

We chose to use NHANES data for the derivation of consumption rates because the data are the most recent available, have a larger sample size than CSFII, use detailed procedures to best estimate consumption (e.g., automated pass), and provide weights (sampling and multistage) with which to generate results that are the most representative of the population. Further, because NHANES is now considered a continuous survey (a complete nationwide survey is completed every two years), past results can be compared with future ones due to consistent operating procedures and study design, and future data can be added to past data to provide a more statistically sound sample size.

The disadvantage of the NHANES data is that the single day of data will tend to exaggerate the higher percentiles of the distribution. For example, if chicken consumption were investigated for 2 separate days, and the individual indicates consumption on one day but not on the second survey day, then chicken consumption would be the average of the two survey days. The average of the two days is probably closer to typical intake for the individual than the one day of chicken consumption that is captured by the NHANES survey.

7.4.1.3 Methodology for the Derivation of Food Consumption Rates

Since 1999, NHANES has been conducted in two-year increments on a continuous basis. The two-year increment is needed to collect data on the full national sample of selected participants. Thus, the NHANES data are composed of datasets from the 1999-2000, 2001-2002, and 2003-2004 periods and the survey is sometimes called the "Continuous NHANES."

The NHANES collected two days of intake for some individuals in the 1999-2004 period. In 2002, a pilot test of collecting two days of intake was conducted on 10 percent of the participants. The pilot study results were not publicly released because of confidentiality issues. In 2003-2004, two days of intake were collected. However, the 2003-2004 dataset has a much smaller sample size relative to the 1999-2004 dataset. We decided that the increased interindividual information available from the larger sample size of one-day intake from the 1999-2000 dataset was advantageous to the two-day intake from a smaller sample size of the 2003-2004 dataset.

7.4.1.4 Categorization of Produce

For the risk assessment of home produced foods, food items can be grouped into food categories to simplify calculations. For produce (i.e., fruits and vegetables), we reviewed the study of Baes et al. (1984) who considered exposure to radionuclides from produce consumption. The physical processes by which plants can be contaminated by airborne radionuclides are analogous to the processes by which airborne low volatility chemical contamination may occur. In the Baes et al. study, produce is divided into

three categories based on the manner in which contamination from air deposition could occur.

The first category, leafy produce, consists of broad-leafed vegetables in which the leaf is the edible part with a large surface area and can be contaminated by deposition of the toxicant onto its surface (e.g., spinach). The next category, exposed produce, includes produce with a small surface area subject to air deposition (e.g., strawberries, green peppers). The third category, protected produce, includes produce in which the edible part is not exposed to air deposition (e.g., oranges, peas).

OEHHA has chosen to use an additional category, root produce, which includes produce for which root translocation could be a source of contamination (e.g., potatoes). In Baes et al., root produce had been placed into one of the other three categories. For the semi-volatile organic and heavy metal toxicants addressed in the AB-2588 program, the produce items from NHANES are classified into the four categories of leafy, exposed, protected, and root produce.

7.4.1.5 Categorization of Meat, Eggs, and Dairy

In addition to homegrown produce, animals are sometimes raised at home, depending on space and zoning regulations, for meat, egg, and milk consumption. Animal derived food items such as lamb, goat meat, or goat milk where consumption rates are small are not included in our risk assessment model.

Cattle, pigs, and poultry differ in the types (e.g., pasture vs. grain) and quantities (g/kg-body weight) of feed consumed and thus food products from these animals are likely to differ in contaminant concentrations. The transfer of contaminant into meat differs from that into eggs and milk. Therefore, we categorized animal derived foods into beef, pork, poultry, eggs, and milk product groups. These groups include the main food item (e.g., milk) as well as products from that item (e.g., cheese).

7.4.1.6 Estimating and Analyzing Consumption Rate Distributions

We used the NHANES 1999-2004 data to estimate consumption rates for the third trimester, 0 to<2 years, 2<9 years, 9<16 years, 16<30 years, 30 to 70 years, and 0-70 years age groups. The NHANES dataset contained data on food items as eaten (e.g., grams of raw apple or grams of cheeseburger), which resulted in two issues for data analysis. In order to estimate the dose of toxicant from the beef component of the hamburger, we need to estimate the grams of beef in hamburger. Toxicant concentration is calculated based on grams of raw or harvested food. Therefore, for foods composed of multiple food items (e.g., ground beef, cheese, tomato, lettuce), the weight of each food item in the food was estimated based on the food item's typical proportion in that type of food. For example, ground beef is considered to be 50 percent of the weight of the cheeseburger while tomatoes in a lettuce and tomato salad are estimated at 50 percent of the reported weight of salad.

The second issue was that ideally we would use the weight of the raw food (rather than the food as eaten) because the concentration of toxicant in a food group (e.g., exposed

produce) is based on the raw food at the time of produce harvesting, meat butchering, milking, or egg laying. In particular, the gram weight of food consumed was adjusted for food items such as jams, jellies, juices, and cheese (a complete list of adjustments, including adjustments to the grams consumed for other reasons, is presented in Appendix D). This is because it takes one part fruit to make 2/3 part juice while one needs 1.5 parts milk to make 1 part cheese. OEHHA did not adjust meats for the amount of moisture lost during cooking. This is because the percent moisture can be highly variable but the majority of the time it is less than 10 percent of initial raw weight, and a default adjustment would have introduced significant uncertainty due to highly variable methods of cooking.

For each participant in the survey, the grams of each food item eaten at each eating occasion was divided by that participant's body weight in kg to give g/kg for each food item-occasion. For food items (e.g., cheeseburger) with multiple components (e.g., ground beef, cheese, lettuce, tomato) the proportional g/kg of each food component was determined (e.g., g/kg ground beef, g/kg cheese). For some food item components the consumption amounts were adjusted, as described above, to account for differences in "as eaten" weights and raw/harvested weights.

We then summed the g/kg of the food item components across eating occasions during the day (e.g., ground beef in cheeseburger at lunch and in meatballs at dinner) to give g/kg-day for each food item component. The sum of the g/kg-day of each food item component was then assigned to its appropriate food group category (an example of this is described in the paragraph following this one). The g/kg-day of all food item components in a food group category were summed to give g/kg-day of the food group category for that participant (e.g., g/kg-day exposed produce).

As an example of assigning food item components to food group categories, we can use a study participant who consumed the following foods: strawberries on cereal at breakfast; a tomato, lettuce and cheese salad and strawberry shake for lunch; chicken, a baked potato, and broccoli, and a slice of apple pie for dinner.

In this example, the g/kg of strawberries at breakfast and at lunch would be added together and then added to the g/kg of the summed g/kg tomatoes, and apples to give the g/kg daily intake for the exposed produce group. Likewise, the g/kg of lettuce at lunch, and broccoli at dinner would be added together for the leafy produce group, the g/kg of onion (in the salad) and potato would be added together for the root produce group. For the poultry food group, the g/kg of chicken at lunch would have been the daily intake for the poultry food group. Beverages were also included as food items so that the g/kg of milk on cereal and in the shake would be added together. These intake rates of milk would then be added to the g/kg of cheese on the salad for the milk products food group for that survey participant. In this manner we obtain the g/kg-day values for each participant for each food group.

Foods that could not be grown in California (e.g., bananas, pineapple) or are only available commercially (e.g., canned milk) were excluded from our analyses. Some food items were not easily identified as to whether they were commercial or home

produced (e.g., frozen berries). In these cases, the assumption was made that they were home produced. Canned produce was also included because the product of home canning is sometimes referred to as canned (e.g., "canned peaches"). The list of foods eligible to be used in deriving the food consumption rates for these guidelines is in Appendix D.

Resultant g/kg-day values for each food group category were analyzed across all ages and the third trimester to <2 years, 2<9 years, 9<16 years, 16<30 years, 16<70 years age groups. It was assumed that during the third trimester that food consumption (and exposure to food borne contaminants) was the same as during ages 16<30 years. This is clearly a simplification but the third trimester is a short time period and the error introduced by this assumption is likely to be small. The "Proc Surveymeans" procedure in SAS 9.1 (SAS Institute, 2007) was used to derive mean, SEM, and 50th-, 90th-, 95th-, and 99th-percentile values. The "Proc Surveymeans" procedure incorporates information from each stage of the sampling, which is needed to provide non-biased variance estimates (e.g., the SEM), as well as incorporating information from the sampling weights to provide results that are the most representative of the population.

7.4.1.7 Produce, Meat, Dairy and Egg Consumption Distributions

Produce, meat, dairy and egg consumption empirical distributions are presented for 0-70, 0<2 years, 2<9 years, 2<16 years, 16<30 years, and 16-70 years (Tables 7.8, 7.9, 7.10, 7.11, 7.12, and 7.13 respectively). The empirical distribution for 16<30 is also recommended for the third trimester because the fetus is assumed to receive the same dose (mg/kg BW) as the mother, and this age category is most representative of the child-bearing years. Consumption is expressed in terms of grams of food per kilogram body weight per day in these tables. The average and high end point estimate recommendations are presented above in Table 7.4.1. These point estimates are the mean and 95th percentiles from the distributions.

The parametric model that best fit each distribution was estimated using the fitting function in Crystal Ball® version 7.2.1 (Oracle, 2007) and presented in Tables 7.2, through 7.7. Of the three goodness-of-fit tests available in Crystal Ball, the Anderson-Darling test was chosen to identify the best-fit distribution since this test is more sensitive to the tails of the distributions than the other two goodness-of-fit tests (the Chi-Square and the Kolmogorov-Smirnov). For an individual dataset and distribution, the better the distribution fits the data set, the smaller the Anderson-Darling statistic will be.

There are 20 distributions that Crystal Ball can test for distributional fit to the dataset of interest, including the Lognormal, Beta, Gamma, Logistic, Beta, and Pareto. For a few consumption rate stratifications (i.e., for a specific age group and food category), the best fit was determined to be Pareto. However, the mean and percentiles estimated for the Pareto distribution were significantly different from the empirically derived mean and percentiles. For these consumption rate strata, we chose to use the second best fit rather than the Pareto, which more clearly fit the empirically derived mean. Tables 7.2 – 7.7 present the best fit distribution for the consumption rates (noted in the column labeled "distribution type").

Table 7.8 Empirical Distributions of Food Consumption (g/kg-day) for All Ages 0-70 years

Food Category	N	Mean	SEM	Min	Max	50th-%ile	75th-%ile	80th-%ile	90th-%ile	95th-%ile	99th-%ile
Produce											
Exposed	9683	3.1	0.05	0.0	84.3	1.7	3.5	4.3	7.2	10.8	23.5
Leafy	7049	1.2	0.03	0.0	19.9	0.8	1.6	1.8	2.7	3.8	7.0
Protected	7033	2.0	0.04	0.0	49.8	1.2	2.5	3.0	4.8	6.8	13.3
Root	11,467	1.9	0.01	0.0	39.5	1.3	2.4	2.8	4.0	5.6	10.8
Meat											
Beef	9043	2.0	0.03	0.0	26.8	1.5	2.5	2.9	4.0	5.2	8.5
Pork	3585	1.1	0.03	0.0	21.4	0.6	1.4	1.6	2.4	3.5	6.8
Poultry	8813	1.9	0.02	0.0	22.5	1.4	2.3	2.6	3.8	5.1	8.7
Milk	17,635	8.4	0.14	0.0	285.3	4.2	9.1	11.3	19.5	31.3	70.6
Eggs	5056	1.7	0.03	0.0	27.1	1.2	2.0	2.3	3.6	5.1	9.3

Table 7.9 Empirical Distributions of Food Consumption (g/kg-day) for Ages 0<2 Yrs

Food Category	N	Mean	SEM	Min	Max	50th-%ile	75th-%ile	80th-%ile	90th-%ile	95th-%ile	99th-%ile
Produce											
Exposed	941	11.7	0.05	0.1	84.3	8.9	15.4	17.6	23.9	30.2	55.3
Leafy	169	3.8	0.04	0.0	19.9	2.8	5.3	6.6	9.2	10.8	14.5
Protected	464	5.9	0.04	0.1	49.8	3.9	7.5	9.1	12.8	17.5	28.8
Root	783	5.7	0.02	0.1	51.4	4.2	8.2	9.2	12.3	15.3	24.0
Meat											
Beef	301	3.9	0.03	0.1	17.7	3.1	5.6	6.4	8.4	11.3	15.6
Pork	91	2.9	0.37	0.0	14.0	1.7	3.8	4.9	6.8	10.5	14.0
Poultry	472	4.5	0.02	0.0	21.8	3.5	5.9	6.7	9.3	11.4	19.6
Milk	924	50.9	1.9	0.0	285.3	44.1	72.3	80.4	100.1	116.1	167.6
Eggs	330	6.1	0.03	0.1	27.1	4.9	7.7	8.5	13.4	15.0	18.8

Table 7.10 Empirical Distributions of Food Consumption (g/kg-day) for Ages 2<9 Years

Food Category	N	Mean	SEM	Min	Max	50th-%ile	75th-%ile	80th-%ile	90th-%ile	95th-%ile	99th-%ile
Produce											
Exposed	1944	7.4	0.26	0.0	74.2	5.6	9.9	11.0	15.6	21.7	35.2
Leafy	689	2.5	0.15	0.0	14.0	1.6	3.3	3.9	6.0	7.9	12.3
Protected	970	4.7	0.17	0.0	33.9	3.5	6.3	7.3	10.2	13.3	19.3
Root	643	3.9	0.12	0.0	34.9	3.1	5.0	5.7	8.0	10.8	17.7
Meat											
Beef	1288	3.5	0.10	0.0	26.8	2.9	4.6	5.0	6.8	8.6	13.6
Pork	434	2.2	0.17	0.0	21.4	1.4	2.7	3.4	4.6	7.8	10.6
Poultry	1430	3.7	0.10	0.0	22.5	3.1	4.7	5.2	7.0	9.0	14.1
Milk	3294	23.3	0.59	0.0	181.8	18.0	30.6	35.2	47.4	61.4	91.2
Eggs	782	3.9	0.15	0.1	19.7	3.4	5.0	5.7	7.4	9.4	15.2

Table 7.11 Empirical Distributions of Food Consumption (g/kg-day) for Ages 2<16 Years

Food Category	N	Mean	SEM	Min	Max	50th-%ile	75th-%ile	80th-%ile	90th-%ile	95th-%ile	99th-%ile
Produce											
Exposed	3764	5.5	0.15	0.0	74.2	3.5	7.3	8.4	12.4	16.6	32.1
Leafy	1833	1.7	0.09	0.0	14.5	1.0	2.3	2.6	4.0	5.8	11.3
Protected	2128	3.6	0.11	0.0	34.7	2.5	4.9	5.6	8.5	10.6	17.5
Root	3599	3.0	0.06	0.0	34.9	2.2	3.9	4.5	6.4	8.7	15.5
Meat											
Beef	3119	3.0	0.07	0.0	26.8	2.3	3.9	4.3	5.7	7.6	11.8
Pork	1018	1.8	0.10	0.0	21.4	1.1	2.2	2.7	4.0	5.7	10.4
Poultry	3093	3.0	0.06	0.0	22.5	2.4	3.9	4.4	5.9	7.5	11.4
Milk	7082	16.5	0.34	0.0	181.8	11.6	21.8	25.2	36.7	48.4	78.6
Eggs	1500	3.1	0.09	0.0	19.7	2.4	4.2	4.6	6.4	8.1	13.5

Table 7.12 Empirical Distributions of Food Consumption (g/kg-day) for Ages 16<30 Years

Food Category	N	Mean	SEM	Min	Max	50th-%ile	75th-%ile	80th-%ile	90th-%ile	95th-%ile	99th-%ile
Produce											
Exposed	1757	1.9	0.06	0.0	20.6	1.4	2.6	3.2	4.3	5.9	9.1
Leafy	1774	0.9	0.04	0.0	11.4	0.6	1.3	1.6	2.2	3.2	5.2
Protected	1523	1.7	0.09	0.0	22.7	1.0	2.1	2.5	3.9	5.8	10.7
Root	2703	1.7	0.05	0.0	13.0	1.2	2.2	2.5	3.6	4.6	7.5
Meat											
Beef	2462	2.0	0.05	0.0	19.4	1.6	2.6	2.9	3.9	4.8	7.4
Pork	843	0.9	0.04	0.0	9.0	0.5	1.4	1.6	2.3	2.9	4.9
Poultry	2208	1.8	0.04	0.0	12.1	1.4	2.3	2.5	3.5	4.7	7.5
Milk	3806	5.4	0.16	0.0	116.3	3.6	7.1	8.4	12.4	15.9	27.6
Eggs	1053	1.6	0.06	0.0	11.6	1.2	1.9	2.3	3.2	4.2	5.8

Table 7.13 Empirical Distributions of Food Consumption (g/kg-day) for Ages 16-70 Years

Food Category	N	Mean	SEM	Min	Max	50th-%ile	75th-%ile	80th-%ile	90th-%ile	95th-%ile	99th-%ile
Produce											
Exposed	4978	1.8	0.06	0.0	23.2	1.3	2.4	2.8	4.1	5.6	8.8
Leafy	5047	1.1	0.03	0.0	15.6	0.8	1.5	1.7	2.5	3.4	5.8
Protected	4441	1.6	0.05	0.0	30.6	1.0	2.1	2.4	3.7	5.2	9.7
Root	6852	1.5	0.02	0.0	13.0	1.1	2.1	2.3	3.2	4.2	6.6
Meat											
Beef	5623	1.7	0.03	0.0	19.4	1.4	2.3	2.5	3.4	4.4	6.8
Pork	2476	0.9	0.03	0.0	14.6	0.5	1.3	1.5	2.2	2.8	4.8
Poultry	5248	1.5	0.02	0.0	12.1	1.3	2.0	2.2	2.9	3.8	6.1
Milk	9629	4.3	0.08	0.0	116.3	3.0	5.8	6.6	9.9	13.2	22.6
Eggs	3226	1.3	0.03	0.0	11.6	1.0	1.6	1.8	2.5	3.4	5.4

*Min = 0 (zero) is due to amounts consumed <0.05 that were rounded to 0.0 (zero)

7.5 Calculating Contaminant Concentrations in Food

The previous sections focused on consumption rates for a variety of foods, and included development of means and distributions for those consumption rates. Consumption rates represent one exposure variate in the algorithm for calculating human exposure to contaminants through the food chain. As in Eq. 7-1, concentrations of contaminants in food products, Cf, must also be estimated. The following sections describe the algorithms and default values for exposure variates used in estimating concentrations in foods.

7.5.1 Algorithms used to Estimate Concentration in Vegetation (Food and Feed)

Vegetation that is consumed directly by humans will be referred to as 'food', while that consumed by animals is termed 'feed'. Humans can be exposed to contaminants from vegetation either directly through food consumption or indirectly through the consumption of animal products derived from animals that have consumed contaminated feed.

The concentration of contaminants in plants is a function of both direct deposition and root uptake. These two processes are estimated through the following equations:

$$Cf = (Cdep)*(GRAF) + Ctrans \quad \textbf{(Eq. 7-5)}$$

where: Cf = concentration in the food (μg/kg)
Cdep = concentration due to direct deposition (μg/kg)
GRAF = gastrointestinal relative absorption fraction
Ctrans = concentration due to translocation from the roots (μg/kg)

7.5.1.1 GRAF

A gastrointestinal relative absorption fraction (GRAF) is included in the calculation of concentration via deposition to account for decreased absorption in the GI tract of materials bound to fly ash or fly ash-like particulate matter relative to absorption of a contaminant added to the diet in animal feeding studies (i.e., laboratory animal studies used to determine oral chronic Reference Exposure Levels). At the present time, GRAF data are only available for polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/F), based on the 2,3,7,8-TCDD congener. The GRAF for those compounds is 0.43. All other compounds have a GRAF of 1.0. There are no data available to describe differential absorption onto feed from fly ash particles as compared to other compounds. Consequently, the factor comes into play only in calculating dose of PCDD/F through this pathway. Note that the factor is not applied to the material translocated through the roots, as toxicants taken up by the roots are assumed to be absorbed to the same extent as that in the feed of the experimental animals in the study, which is the basis for both the cancer potency factor and reference exposure level.

7.5.1.2 Deposition onto Crops

The factor Cdep is calculated by the following equation:

$$Cdep = [(Dep)\ (IF)/(k)\ (Y)] \times (1\text{-}e^{-kT}) \qquad \textbf{(Eq. 7-6)}$$

where: Cdep = amount of toxicant depositing on the vegetation per kg crop (µg-toxicant / kg-crop)
Dep = deposition rate on impacted vegetation ($\mu g/m^2 day$)
IF = interception fraction
k = weathering constant (d^{-1})
Y = crop yield (kg/m^2)
e = base of natural logarithm (~2.718)
T = growth period (days)

The variate, Dep, is a function of the modeled (or measured) ground level concentration, and the vertical rate of deposition of emitted materials, and is calculated as follows:

$$Dep = GLC \times Dep\text{-}rate \times 86{,}400 \qquad \textbf{(Eq. 7-7)}$$

where: GLC = ground level concentration of contaminant in air ($\mu g/m^3$)
Dep-rate = vertical deposition rate (m/sec)
86,400 = seconds per day (sec/day)

The ground level concentration is calculated in the air dispersion modeling (see Chapter 2). The deposition rate is assumed to be 0.02 meters per second for a controlled source and 0.05 meters/second for an uncontrolled source (see Chapter 2).

The interception fraction in Eq. 7-6 above is crop specific. The work of Baes et al. (1984), examining the transport of radionuclides through agriculture, describes interception fraction as a factor which accounts for the fact that not all airborne material depositing in a given area initially deposits on edible vegetation surfaces. That fraction will be somewhere between zero and one.

There are no data on interception fraction for leafy and exposed produce but interception fractions for these produce categories were modeled by Baes et al. (1984). Baes et al. used assumptions based on typical methods of cultivating leafy and exposed produce in the U.S., and on the following equations:

$$If\ e = 1 - e^{(-0.0324Ye)}$$
$$If\ l = 1 - e^{(-0.0846Yl)}$$

where: If e = interception fraction for exposed produce
If l = interception fraction for leafy produce
Y = yield of exposed produce (kg/m^2, dry)
Y = yield of leafy produce (kg/m^2, dry).

Baes et al. calculated an average interception fraction of 0.15 for leafy produce and 0.052 for exposed produce. For these guidelines, the interception fractions were rounded off to 0.2 and 0.1 for leafy and exposed produce, respectively.

Some information is available from studies of radioactive isotopes for pasture grasses. The empirical relationship for grasses is given by:

$$IFpg = 1-e^{-2.88\,Y} \qquad \textbf{(Eq. 7-8)}$$

where: IFpg = interception fraction for pasture grasses
Y = yield in kg/m^2 (dry)

Assuming that the wet yield is 2 kg/m^2, and 80 percent of the wet weight is water, then the IFpg is approximately 0.7 (Baes et al., 1984). This value compares well with the Baes modeled interception fractions for leafy and exposed produce since grasses are more densely packed into a given area relative to home grown leafy and exposed produce.

For protected and root produce, there are no known interception fractions (modeled or empirical) and it is difficult to arrive at a wet yield value. OEHHA recommends that the 2 kg/m^2 wet yield value be used for the protected and root categories of produce.

Additional default values for variates in Eq. 7-6 are obtained from *Multi-pathway Health Risk Assessment Parameters Guidance Document* prepared for South Coast Air Quality Management District (Clement Associates, 1988). The weathering constant, k, is based on experimental observations from studies of particulate radionuclides on plant surfaces. This weathering constant does not include volatilization from the leaf surface since the radionuclides used were not volatile, nor does it include biotransformation or chemical transformation on the leaf surface. Baes et al. (1984) describe particulate half-lives ranging from 2.8 to 34 days with a geometric mean of 10 days for radionuclides depositing on plants. OEHHA proposes using a weathering constant of 10 days based on Baes et al. (1984).

The growth period, T, in Equation 7-6 above is based on the time from planting to harvest. OEHHA recommends a value of 45 days for leafy and root crops and 90 days for exposed and protected produce (time from fruit set to harvest). The assumptions in the interception fraction include the issue of increasing surface area with growth. Therefore, no additional adjustment is necessary.

7.5.1.3 Translocation from the Roots

The variate, C_{trans}, in Equation 7-9, represents the amount of contaminant that is translocated, or absorbed, from the soil into the roots of homegrown crops that are food sources for humans. Once absorbed, the contaminant may accumulate in edible roots (e.g., carrots) and be translocated to other parts of the plant that are consumed including the leaves and fruit. The equation for calculating concentration in the plant from root uptake is as follows:

$$C_{trans} = C_s \times UF \quad \textbf{(Eq. 7-9)}$$

Where: C_s = concentration in the soil (see Chapter 6)
UF = soil-to-plant uptake factor

The soil-to-plant uptake factor (UF) is the ratio of the fresh weight contaminant concentration in the edible plant or plant part over the total concentration of the contaminant in soil wet weight. The UFs (Eq. 7-9) recommended by OEHHA are from the scientific literature. Due to the large volume of studies investigating metal concentrations in edible plants grown in contaminated soils, OEHHA created a database to assemble the data and calculate UFs. The database and methods used to estimate the UFs are described in Appendix H.

The concentration in the soil (C_s) is calculated as described in Chapter 6 using air dispersion and deposition modeling. The UF for specified metals can then be applied in Eq. 7-9 in order to estimate C_{trans}.

Due to lack of root absorption and translocation, the soil-to-plant uptake from the roots of organic compounds under the "Hot Spots" program (e.g., dioxins and PCBs) is not included. Therefore, the soil-to-plant UFs are currently limited to the inorganic metals and chemicals.

The soil-to-plant UFs of edible plants, shown in Table 7.14, are divided into four types: leafy, root, protected, and exposed. The foods in each of these produce categories are presented in Appendix D. The classification of edible plants into these four groups reflects the potential differences in contaminant concentrations that may occur in the plant parts resulting not only from soil-to-plant uptake, but also from airborne deposition.

Table 7.14 Soil-to-plant uptake factors for inorganic metals and chemicals in edible crops[a]

Element	Leafy	Exposed	Protected	Root
Arsenic	1×10^{-2}	2×10^{-2}	7×10^{-2}	8×10^{-3}
Beryllium	2×10^{-4}	8×10^{-3}	3×10^{-4}	5×10^{-3}
Cadmium	1×10^{-1}	2×10^{-2}	1×10^{-2}	8×10^{-2}
Chromium (VI)	3×10^{-1}	2×10^{-2}	7×10^{-2}	3×10^{0}
Fluoride	4×10^{-2}	4×10^{-3}	4×10^{-3}	9×10^{-3}
Lead	8×10^{-3}	7×10^{-3}	3×10^{-3}	4×10^{-3}
Mercury	2×10^{-2}	9×10^{-3}	1×10^{-2}	2×10^{-2}
Nickel	1×10^{-2}	3×10^{-3}	3×10^{-2}	6×10^{-3}
Selenium	6×10^{-2}	4×10^{-2}	3×10^{-1}	7×10^{-2}

[a] Soil-to-plant UFs represent the fresh weight concentration of a contaminant in the plant part over the wet weight concentration of contaminant in the soil.

7.5.2 Algorithms used to Estimate Dose to the Food Animal

The general formula for estimating concentrations of contaminants in animal products is as follows:

$$Cfa = [Dinh + Dwi + Dfeed + Dpast + Dsi] \times Tco \quad \textbf{(Eq. 7-10)}$$

where: Dinh = dose through inhalation (μg/day)
Dwi = dose through water intake (μg/day)
Dfeed = dose through feed consumption (μg/day)
Dpast = dose through pasturing/grazing (μg/day)
Dsi = dose through soil ingestion (μg/day)
Tco = transfer coefficient from consumed media to meat/milk products

Ideally, the Tco values would be evaluated separately for the inhalation and oral routes but the data do not exist to separately evaluate the inhalation route. The Tco values are based on oral studies, and are presented in Appendix K, and summarized in Table 7.16 and 7.17.

7.5.2.1 Dose via Inhalation

The dose via inhalation is proportional to the concentration of the contaminant in the air and the amount of air breathed by the animal in a single day. It is assumed that 100 percent of the chemical is absorbed. The dose via inhalation is calculated as follows:

$$Dinh = BR \times GLC \quad \textbf{(Eq.7-11)}$$

where: Dinh = dose to the animal via inhalation (μg/day)
BR = daily breathing rate of the animal (m^3/day)
GLC = ground level concentration (μg/m^3)

7.5.2.2 Dose via Water Consumption

Airborne contaminants depositing in surface water sources of drinking water for food animals can end up in the human food chain. The dose to the food animal from water consumption is proportional to the concentration of the contaminant in the drinking water and the amount of water consumed by the animal daily. In addition, the fraction of the water consumed daily that comes from a contaminated body of water is used to adjust the dose to the food animal. That fraction is a site-specific value that must be estimated for the site. The dose via water consumption can be calculated as follows:

$$Dwi = WI \times Cw \times Fr \quad \textbf{(Eq. 7-12)}$$

where: Dwi = dose to the food animal through water intake (μg/day)
WI = water intake rate (L/day)
Cw = concentration of contaminant in water (μg/L)
Fr = fraction of animal's water intake from the impacted source

Cw is calculated as in Chapter 8. Water consumption rates for food animals are shown in Table 7.15. The fraction of the animals' water intake that comes from the source impacted by emissions is a site-specific variable.

7.5.2.3 Dose from Feed Consumption, Pasturing and Grazing

Airborne contaminants may deposit on pastureland and on fields growing feed for animals. The default assumption is that the feed is not contaminated because most feed would be purchased from offsite sources. However, if feed is produced onsite, the dose from contaminated feed should be determined. Deposited contaminant contributes to the total burden of contaminants in the meat and milk. The dose to the animal from feed and pasture/grazing can be calculated as follows:

$$Dfeed = (1 - G) \times FI \times L \times Cf \quad \textbf{(Eq. 7-13)}$$

where:
- Dfeed = dose through feed intake (μg/day)
- G = fraction of diet provided by grazing
- FI = feed consumption rate (kg/d)
- L = fraction of feed that is locally grown and impacted by facility emissions
- Cf = concentration of contaminant in feed (μg/kg) (calculated in Eq. 7-2)

$$Dpast = G \times Cf \times FI \quad \textbf{(Eq. 7-14)}$$

where:
- Dpast = dose from pasture grazing (μg/day)
- G = fraction of diet provided by grazing
- FI = pasture consumption rate (kg/day)
- Cf = concentration of contaminant in pasture (μg/kg)

DMI, kg dry matter intake (feed), is given for food animals in Table 7.15. The percent of the diet that comes from pasture and feed, and the fraction of feed that is locally grown and impacted by emissions are site-specific variables and values for these variables need to be assessed by surveying farmers in the impacted area. Concentration in the feed and pasture are calculated as in Equations 7-10 and 7-11 above. It is considered likely that feed will come from sources not subject to contamination from the stationary source under evaluation.

Table 7.15 Point Estimates for Animal Pathway

Parameter	Beef Cattle	Lactating Dairy Cattle	Pigs	Meat Poultry	Egg-laying Poultry
BW (body weight in kg)	533	575	55	1.7	1.6
BR (inhalation rate in m^3/d)	107	115	7	0.4	0.4
WI (water consumption in kg/d)	45	110	6.6	0.16	0.23
DMI (kg/d)[1]	9	22			
Feed Intake			2.4	0.13	0.12
%Sf (soil fraction of feed)	0.01	0.01	NA	NA	NA
%Sp (soil fraction of pasture)	0.05	0.05	0.04	0.02	0.02

[1] Dry matter intake

7.5.2.4 Transfer Coefficients from Feed to Animal Products

The derivation and use of transfer coefficients for specific chemicals is explained in Appendix K. Tables 7.16 and 7.17 contain the recommended values for multipathway organic and inorganic chemicals, respectively.

Table 7.16 Food Animal Transfer Coefficients for Organic Chemicals

Organic Chemical	Tcos (d/kg)[a]				
	Cow's Milk	Chicken Egg	Chicken Meat	Cattle Meat	Pig Meat
Diethylhexylphthalate	9×10^{-5}	0.04	0.002	6×10^{-4}	5×10^{-4}
Hexachlorobenzene	0.02	20	10	0.2	0.08
Hexachlorocyclohexanes	0.01	7	5	0.2	0.09
PAHs	0.01	0.003	0.003	0.07	0.06
Polychlorinated biphenyls					
Congener 77	0.001	6	4	0.07	0.4
81	0.004	10	7	0.2	0.4
105	0.01	10	7	0.6	0.7
114	0.02	10	7	0.9	0.7
118	0.03	10	7	1	0.7
123	0.004	10	7	0.2	0.7
126	0.04	10	7	2.	0.7
156	0.02	10	8	0.9	2
157	0.01	10	8	0.5	2
167	0.02	10	8	1	2
169	0.04	10	8	2	2
189	0.005	10	8	0.2	1
Unspeciated	0.01	10	7	0.2	0.5
PCDD/Fs					
Congener 2378-TCDD	0.02	10	9	0.7	0.1
12378-PeCDD	0.01	10	9	0.3	0.09
123478-HxCDD	0.009	10	6	0.3	0.2
123678-HxCDD	0.01	10	6	0.4	0.1
123789-HxCDD	0.007	7	3	0.06	0.02
1234678-HpCDD	0.001	5	2	0.05	0.2
OCDD	0.0006	3	1	0.02	0.1
2378-TCDF	0.004	10	6	0.1	0.02
12378-PeCDF	0.004	30	10	0.1	0.01
23478-PeCDF	0.02	10	8	0.7	0.09
123478-HxCDF	0.009	10	5	0.3	0.1
123678-HxCDF	0.009	10	6	0.3	0.09
234678-HxCDF	0.008	5	3	0.3	0.06
123789-HxCDF	0.009	3	3	0.3	0.03
1234678-HpCDF	0.002	3	1	0.07	0.06
1234789-HpCDF	0.003	3	1	0.1	0.02
OCDF	0.002	1	0.6	0.02	0.03
Unspeciated	0.001	6	5	0.03	0.09

[a] All Tco values were rounded to the nearest whole number.
[b] NA – no data available or not applicable

Table 7.17 Food Animal Transfer Coefficients for Inorganic Chemicals

Inorganic Metals and Chemicals	Tcos (d/kg)[a]				
	Cow's Milk	Chicken Egg	Chicken Meat	Cattle Meat	Pig Meat
Arsenic	5×10^{-5}	0.07	0.03	2×10^{-3}	0.01[b]
Beryllium	9×10^{-7}	0.09	0.2	3×10^{-4}	0.001
Cadmium	5×10^{-6}	0.01	0.5	2×10^{-4}	0.005
Chromium (VI)	9×10^{-6}	NA[c]	NA	NA	NA
Fluoride	3×10^{-4}	0.008	0.03	8×10^{-4}	0.004[b]
Lead	6×10^{-5}	0.04	0.4	3×10^{-4}	0.001[b]
Mercury	7×10^{-5}	0.8	0.1	4×10^{-4}	0.002[b]
Nickel	3×10^{-5}	0.02	0.02	3×10^{-4}	0.001
Selenium	0.009	3	0.9	0.04	0.5

[a] All Tco values were rounded to the nearest whole number.
[b] The meat Tco was estimated using the metabolic weight adjustment ratio of 4.8 from cattle to pig
[c] NA – no data available or was not applicable

7.6 Default Values for Calculation of Contaminant Concentration in Animal Products

7.6.1 Body Weight Defaults

Cows used for milk production will be adults (i.e., full body weight) and females, so only adult female weights should be used for the home produced milk pathway. OEHHA recommends the central tendency weight of 575 kg for the home raised milk cow (midpoint of the adult cow range). A cow or bull raised for home produced beef may be of any age, gender or strain. We recommend 533 kg (midpoint of the beef cattle range) for the home produced beef pathways (National Research Council, 2000). Beef cattle are growing while being raised and thus transitioning through lower body weights to reach the mature body weight. We therefore propose a default central tendency value.

Mean pig body weights of 30.9-80 kg at age 13-23 weeks have been reported (Agricultural Research Council, London, 1967). The 4H club, which encourages children to participate in the home raising of pigs, recommends that the pigs weigh between 200 and 240 pounds (90.9 and 109 kg) at the end of the project (http://www.goats4h.com/Pigs.html#weight). OEHHA recommends half of 240 pounds, 120 pounds or 55 kg, as the average weight of the pig while being raised.

The National Research Council (1994) in Table 2.5 lists the weight of broiler chickens by week up to 9 weeks. The weight for the males is 3.5 kg after 9 weeks. The average weight over the 9-week period is 1.7 kg, which is the OEHHA's recommendation for a default body weight for chickens raised for meat. The OEHHA recommends the average weight of white and brown egg laying chickens at 18 weeks to first egg laying (1.5 kg) in Table 2-1 National Research Council (1994).

7.6.2 Breathing Rate Defaults

Animal breathing rate defaults were calculated based upon a relationship of tidal volume to body weight. Each pound of body weight has been reported to correspond to approximately 2.76 ml of tidal volume (2.76 ml/lb ≅ 6.07 ml/kg body weight) (Breazile, 1971). Using this relationship, the default animal body weight, and breathing cycle frequencies provided in Breazile (1971), we generated breathing rates. Reported breathing frequencies for cattle, pigs, and poultry were 18-28, 8-18, and 15-30 respirations per minute, respectively. The body weight defaults described above were used in the calculations. Use of these values generated a range of breathing rates and the default value was derived as the average of the range limits. Default breathing rates for dairy cattle, beef cattle, pigs, and poultry are 116, 107, 6.2, and 0.33 m^3/day, respectively. The default value for cattle falls within the range of that reported by Altman et al. (1958).

7.6.3 Feed Consumption Defaults

Backyard farmers could raise cattle, swine, and chickens from birth to early adulthood for meat. There is a large change in body weight that correlates with feed-consumption rates during that period of the animal's life. For meat animals, the OEHHA attempted to identify the consumption rate at the mid-point of the meat animals' pre-slaughter life span. In contrast, the adult cows and chicken that produce milk and eggs have relatively constant feed-consumption rates and body weights. For these cows and chickens, OEHHA attempted to identify the consumption rate of the fully-grown adult.

OEHHA's risk assessment model assumes that the source contaminates the pasture or hay from that pasture. A regulated source could contaminate a pasture that provides a cow with 100 percent of its nutrition. In contrast, homeowners usually procure feed for backyard swine and chicken that is produced off-site. Therefore, the default assumptions are that the regulated source contaminates 0 percent of the swine or chicken feed, and 100 percent of cows' feed. Site-specific conditions may require that different percent contamination be used.

7.6.3.1 Bovine Feed Ingestion

Most published literature on bovine feed ingestion is on commercial production. While the backyard and commercial animals are the same breeds, the feeding patterns can be different. It is likely that home raised cattle will be fed a higher percentage of forage, for example. DMI is the feed consumption rate with the units of kilograms feed per day (kg/d). Feed is dried before it is weighed to obtain a DMI because water content varies. The NRC identifies several factors that affect DMI (NRC, 2001). These include fiber content of the forage, initial size of the animal, and time preceding parturition. Two types of feed are reported in the literature: forage (grass, hay, alfalfa, etc.) and concentrate (high-energy feeds like corn, soybean or oats). As concentrate increases, consumption of forage decreases.

As the animal gets larger, it eats more food; therefore, DMI is correlated with body weight. Body weight does not change greatly during the majority of the milk producing years of dairy cows. Therefore, we assume the backyard dairy cow consumes the same amount as those in the studies described below. In contrast, the body weight of beef cattle varies greatly as they grow from calves to adults. Papers often report the starting body weight for beef cattle. OEHHA selected peer-reviewed papers in which DMI was reported with adequate description of the methods. DMI was measured in these studies but was not necessarily the objective of the study.

Cows eat about as much pasture as they do hay or silage. Holden et al. (1994) compared DMIs of pasture, hay, and silage in three non-lactating, non-pregnant dairy cows. The pasture was identical to that used for the hay and silage. The cows ate pasture, hay, and silage in sequential 19-day exposures. Chromium oxide, an indigestible component of vegetation, was used to estimate consumption. This study showed that fecal chromium oxide accurately predicts DMI of hay and silage. More importantly, intake rates (kg/d) showed no difference among pasture, silage or hay using fecal chromium oxide estimates. Therefore, OEHHA selected studies that measured silage or hay consumptions assuming they are the same as pasture consumption.

Britt et al. (2003) measured DMI in 13 herds of lactating Holstein dairy cows in Kentucky, Tennessee, and Mexico at different times throughout the year. The mean ± standard deviation of 34 measurements is 21.8 ±1.6 kg/day with a range of 16.8 to 24.5. Holcomb et al. (2001) reported an average DMI for 40 Holsteins of 21.6 kg/day. Rastani et al. (2005) measured DMI for 20 weeks around birth. Ten weeks prior to birth, the DMI was 20 kg/day and gradually decreased to 10 kg/day at birth, and then it gradually increased to 23 kg/day ten weeks post-partum. The OEHHA recommendation for DMI for dairy cows is 22 kg/day, the mean of these three reports.

As described in the Bovine section above, a number of factors influence the uncertainty and variability of pasture DMI of backyard dairy cows. As Rastani et al. (2005) show, lactating cows consume about twice as much as cows not lactating. We did not consider non-lactating cows since milk is the vehicle of human exposure. Cows fed supplements such as corn, soybean, or oats would eat less pasture.

The NRC (2000) has developed an equation predicting DMI based on the energy content in mega-calories per kg of dry matter of the forage (Mcal/kg). A graph of DMI vs. energy content using this equation peaks at about 9 kg/d with cows fed medium energy content forage. The DMI gradually decreases to about 7.6 kg/day with both high and low energy content forages. A second graph in the NRC report shows DMI plotted against initial body weight. The smallest steers (200 kg) ate the least (4 kg/d) and larger animals ate the most (12 kg/d for 350 kg steers). Burns et al. (2000) reported DMI in six Angus steers (initial mean BW = 334 kg) fed with an average DMI of 9.7 kg/d. Stanley et al. (1993) measured DMI in four Hereford x Angus cows at seven time points. The total duration was 83 days during which there was a linear increase in DMI from 8.8 to 14.9 kg/day. Unfortunately, the authors did not report body weights at the seven time

points. OEHHA recommends a default DMI of 9 kg/day for cattle home raised for beef to estimate average food consumption during the home raising period.

The uncertainties described for dairy cows apply to beef cattle. In addition, DMI correlates with body weight and the body weight varies greatly in beef cattle grown from calves to young adults for slaughter. The OEHHA value is an average over this period. It could over-estimate intake if calves are slaughtered for veal or under-estimate intake of cattle slaughtered long after reaching maturity.

7.6.3.2 Swine Feed Ingestion

Since it is likely that most backyard swine would eat feed produced off-site, this exposure pathway to the swine should be included only when feed is grown on-site. OEHHA assumes people obtain backyard swine as weanlings and slaughter them at early adulthood when they weigh about 110 kg. The food consumption varies with body weight and calorie density of the feed. The NRC has developed a mathematical model from simultaneous observations of body weight and feed intake of a nutritionally adequate corn/soybean mix to over 8,000 swine. The model (NRC, 1998) predicts the digestible energy requirement (in kcal/day) as a function of body weight (from 10 to 120 kg). The equation predicts that swine at the average body weight of 55 kg would require about 8000 kcal/d. Corn has a digestible energy content of about 3,300 kcal/kg (Feoli et al.(2007). Thus, a 55 kg swine would consume about 2.4 kg/d.

Generally, backyard swine consume restaurant waste or other feed not produced on-site. Therefore, risk assessors should assume the amount of contaminated feed consumed by backyard swine is zero, as the default. If the dry weight digestible energy content of this feed is known, it can be used to convert 8,000 kcal into kg of feed consumed per day. When swine eat supplements not raised on-site, the risk assessor will need to determine the fraction of feed raised on-site.

7.6.3.3 Chicken Feed Ingestion

Since most backyard chickens would eat feed produced off-site, this exposure pathway for chickens should be included only when chickens' feed is known to be grown on-site. Chicken feed consumption from onsite could contaminate the meat and/or eggs.

7.6.3.4 Feed Ingestion by Chickens Raised for Meat

Ingestion of homegrown feed by chickens, which are home-raised for meat, is only an exposure pathway if the feed is also grown on site, which is unlikely. If the feed is grown on site then the following feed consumption value is provided. The National Research Council (1994) report in Table 2.5 of their document shows data on chicken food consumption for broilers from one to nine weeks of age. Males, the most likely to be eaten by homeowners, weigh 3.5 kg at 9 weeks and consume 0.23 kg/d of feed. Males at the midpoint, 4 weeks, weigh 1 kg and consume 0.132 kg/d. If only a fraction of the feed at a particular site is grown on site, this fraction should be used to reduce the consumption rate.

7.6.3.5 Laying Hen Feed Ingestion

Ingestion of homegrown feed by chickens home-raised for eggs, is only an exposure pathway if the feed is grown on site, which is unlikely. If the feed is grown on site, then the following feed consumption value is provided. Table 2.2 of the NRC report (1994) shows consumption rates for laying hens from 2 to 20 weeks of age. At 20 weeks, the average weight of strains laying brown eggs and strains laying white eggs is 1.6 kg and the average food consumption at 20 weeks is 0.12 kg/d, which is recommended as the default for egg laying chickens. If only a fraction of the feed which chickens at a particular site ingest is grown on site, this fraction should be used to reduce the consumption rate.

7.6.4 Water Consumption Defaults

Water consumption for home raised beef cattle, dairy cattle, pigs, and chickens would be an exposure pathway for these animals only if surface waters are used as a water source (e.g., a farm pond). If municipal or well water were used, the water supply would not be contaminated by the facility under evaluation under the assumptions of the Hot Spots risk assessment model.

7.6.4.1 Bovine Water Consumption

Literature reported bovine water intake rates are generally expressed in relation to dry matter consumption on a weight basis. Water intake also generally increases with increasing temperature. Water intakes for cattle of 3.1-5.9 kg/kg dry matter at temperatures ranging from 12°C to 29.4°C have been reported (Winchester and Morris, 1956, as summarized by the Agricultural Research Council, London, 1965).

Water intakes of 6.6-10.2 kg/kg dry matter consumed for shorthorn cows at 27°C and 3.2-3.8 kg/kg dry matter consumed at 10°C have been reported (Johnson et al., 1958). Water intake for shorthorn cows at 18-21°C of 4.2-5.0 kg/kg dry matter consumed have also been reported (Balch et al., 1953). Water intake at lower temperatures (-18 to 4°C) of 3.5 kg/kg dry matter consumed has also been reported (MacDonald and Bell, 1958). Friesian cattle water intake was estimated at 3.3-4.3 kg/kg dry matter consumed (Atkeson et al., 1934).

The National Research Council (2001) has several equations for calculating water intake of dairy cows that take into account ambient temperature, sodium intake, DMI, and milk production to produce a refined estimate of water intake. Given the feed intake for both non-lactating and lactating cattle as described above, a reasonable default estimate of water consumption is approximately 5-fold the dry matter consumption. If this exposure pathway to beef cattle or dairy cows is applicable, the resulting default water consumption rates for beef cattle and lactating dairy cattle are 45 and 110 kg/day, respectively.

7.6.4.2 Swine Water Consumption Rates

Water consumption has been estimated for pigs at 1 kg/day for 15 kg pigs, increasing to 5 kg/day at 90 kg body weight (Agricultural Research Council, London, 1967). Non-pregnant sow water consumption was estimated at 5 kg/day, pregnant sows at 5-8 kg/day, and lactating sows at 15-20 kg/day. The National Research Council (1998) estimates 120 mL water/kg BW day for growing (30 to 40 kg) nonlactating pigs and 80 mL water/kg BW-day for nonlactating adult pigs (157 kg). A default value of 6.6 L/day is recommended based on the 120 mL/kg BW day figure in the National Research Council (1998).

7.6.4.3 Water Consumption Rates by Chickens

The water consumption exposure pathway would only be applicable as an exposure pathway for chickens if surface water were used as a drinking water source (e.g., a farm pond). If municipal water or well water is used as the water supply for home raised chicken, the water is assumed uncontaminated from airborne emissions of a facility. Water consumption by chickens has been reported to fall in the range of 1-3 times the food consumption on a weight basis (Agricultural Research Council, London, 1975). They established a 2:1 ratio of water to feed consumption as the default value. Given a daily feed consumption rate of 0.1 kg/day, the resulting daily water consumption rate for chickens is 0.2 kg/day.

The National Research Council (1994) estimated water consumption over an eight-week period for broilers and brown egg layers. The average water consumption rate is 0.16 L/day for broilers. The daily water consumption rate is 0.23 L/day for brown egg layers at 20 weeks (National Research Council, 1994). A default water consumption rate of 0.16 L/day is recommended for broilers and 0.23 L/day is recommended for egg laying chickens, if the water exposure pathway is applicable to chickens.

7.6.5 Soil Ingestion Defaults

Soil ingestion was estimated for dairy cattle based upon fecal titanium content (Fries et al., 1982). Among yearling heifers and non-lactating cattle receiving feed (vs. pasture), soil ranged from 0.25 to 3.77 percent of dry matter ingested, depending on the management system used, with those cattle with access to pasture having the greatest soil ingestion. For cattle on feed, a reasonable estimate of 1 percent soil ingestion was made. For cattle grazing pasture, soil intake estimates of 4-8 percent dry matter ingestion have been made for cattle receiving no supplemental feed (Healy, 1968).

Soil ingestion varies seasonally, with the greatest soil ingestion during times of poor plant growth (14 percent) and the least soil ingestion during lush growth (2 percent). In a study of several farms in England, beef and dairy cattle were found to have soil ingestion rates ranging from 0.2 to 17.9 percent of dry matter consumed, depending both on the location and the time of year (Thornton and Abrahams, 1983). The two largest sets of data evaluated showed a range of soil ingestion of 1.1-4.4 percent dry

matter consumed. Thus, a reasonable estimate of soil ingestion by beef and dairy cattle as percent of pasture consumed is 5 percent.

Soil ingestion estimates have been made for pigs (Healy and Drew, 1970). A mean weekly soil ingestion estimate of 1 kg soil/week was made for pigs grazing swedes (rutabaga), corresponding to 0.014 kg soil/day. Other estimates for animals grazing swedes, swedes with hay, and pasture only were 0.084, 0.048, and 0.030 kg soil/day, respectively. Assuming total feed ingestion of 2 kg/day, the soil ingestion as percent of grazed feed (pasture) ranged from 1.5 to 7 percent, with a best estimate of 4 percent. In the absence of information concerning soil content of feed for pigs, no estimate has been made for soil ingestion from feed. For risk assessment purposes, pigs are assumed to consume 4 percent soil from pasture ingestion.

As a digestive aid, chickens normally consume approximately 2 percent grit in their diet (McKone, 1993). This value was used as an estimate of the fraction of soil ingestion for chickens with access to pasture. Chickens were assumed to have access to pasture/soil and therefore, no estimate was made for soil ingestion strictly from feed.

7.7 Fraction of Food Intake that is Home-Produced

The Child-Specific Exposure Factors Handbook (USEPA, 2008) has information on the fraction of food intake that is home produced (Table 13.6). This information is from a U.S. EPA analysis of the 1987-1988 National Food Consumption Survey. The Table contains information on a number of specific home produced items as well as broad categories such as total vegetables and fruits.

Table 7.18 Fraction of Food Intake that is Home-Produced

	All Households	**Households that Garden**	**Households that Farm**
Total Fruits	0.04	0.101	0.161
Total Vegetables	0.068	0.173	0.308
Avg. Total Veg & Fruits	0.054	0.137	0.235
	All Households	Households that Raise Animals/Hunt	Households that Farm
Beef	0.038	0.485	0.478
Pork	0.013	0.242	0.239
Poultry	0.011	0.156	0.151
Eggs	0.014	0.146	0.214
Total Dairy	0.012	0.207	0.254

The data on the fraction of food intake that is home produced are older than would be considered optimal and there is no data on variability in percent consumption in the populations of concern. There are many factors that could affect the percent of home-

produced fruits and vegetables. These may include lot size, employment status, avidity and income. As a default for home-produced leafy, exposed, protected and root produce, OEHHA recommends 0.137 as the fraction of produce that is home raised (Table 7.18). The households that grow their own vegetables and fruits are the population of concern. In rural situations where the receptor is engaged in farming, OEHHA recommends 0.235 as the default value for fraction of leafy, exposed, protected and root produce that is home produced.

OEHHA recommends the fraction home-raised under “Households that raise animals/hunt” (Table 7.18) for beef, pork, poultry (chicken), eggs and dairy (milk), with the exception of rural household receptors engaged in farming. OEHHA recommends that the fractions listed under “Households that farm” be used for the rural household receptors.

7.8 References

Agricultural Research Council, London (1975). The Nutrient Requirements of Farm Livestock, No. 1, Poultry.

Agricultural Research Council, London (1965). The Nutrient Requirements of Farm Livestock, No. 2, Ruminants.

Agricultural Research Council, London (1967). The Nutrient Requirements of Farm Livestock, No. 3, Pigs.

Altman, PI, Gibson, JF, and Wang, CC. (1958). Handbook of Respiration. Dittmer, D.S. and Grebe, R.M. (eds.). W.B. Saunders Company, Philadelphia.

Atkeson, FW, Warren, TR, and Anderson, GC. (1934). Water requirements of dairy calves. J Dairy Sci, 17:249.

Baes, C, Sharp, R, Sjoreen, A, and Shor, R. (1984). A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides Through Agriculture. Oak Ridge National Laboratory, and The Office of Radiation Programs, U.S. Environmental Protection Agency. Interagency Agreement AD-89-F-2-A106, September.

Block, G. (1992). A review of validations of dietary assessment methods. Am J Epidem 115: 492-505.

Breazile, JE (ed.) (1971). Textbook of Veterinary Physiology. Lea & Febiger, Philadelphia.

Britt, JS, Thomas RC, et al. (2003). "Efficiency of converting nutrient dry matter to milk in Holstein herds." J Dairy Sci 86(11): 3796-3801.

Clement (1988). Multi-pathway Health Risk Assessment Input Parameters Guidance Document. Prepared for the South Coast Air Quality Management District by Clement Associates, Inc., Fairfax, Virginia, June 1988.

Feoli, C, Hancock JD, Monge C, Gugle TL, Carter SD, and Cole NA (2007) Digestible Energy Content Of Corn- Vs Sorghum-Based Dried Distillers Grains With Solubles And Their Effects On Growth Performance And Carcass Characteristics. In Finishing Pigs. American Society of Animal Science

Fries, GF, Marrow, GS, and Snow, PA (1982). Soil ingestion by dairy cattle. J Dairy Sci, 65:611-8.

Healy, W.B. (1968). Ingestion of soil by dairy cows. New Zealand J Agricul Res, 11:487-99.

Healy, WB and Drew, KR (1970). Ingestion of soil by hoggets grazing swedes. New Zealand J Agricult Res, 13:940-4.

Holcomb, CS, Van Horn, HH, et al. (2001). Effects of prepartum dry matter intake and forage percentage on postpartum performance of lactating dairy cows. J Dairy Sci **84**(9): 2051-2058.

Holden, LA, LD Muller, GA Varga and Hillard PJ (1994) Ruminal Digestion and Duodenal Nutrient Flows in Dairy Cows Consuming Grass as Pasture, Hay or Silage. J Dairy Sci 77(10):3034-42.

MacDonald, MA and Bell, JM (1958). Effects of low fluctuating temperatures on farm animals. II Influence of ambient air temperatures on water intake of lactating Holstein-Fresian cows. Canadian Journal of Animal Science, 38: 23-32.

McKone, TE (1993). CalTOX, A Multimedia Total-Exposure Model for Hazardous-Wastes Sites, Parts I-III. Prepared for the State of California, Department of Toxic Substances Control, Lawrence Livermore National Laboratory, Livermore, CA, UCRL-111456.

NRC (1994). Nutrient Requirements of Poultry. National Research Council, Washington, D.C., National Academy Press.

NRC (1998). Nutrient Requirements of Swine. National Research Council, Washington, D.C., National Academy Press.

NRC (2000). Nutrient Requirements of Beef Cattle. National Research Council, Washington, D.C., National Academy Press.

NRC (2001). Nutrient requirements of Dairy Cattle. National Research Council, Washington, D.C., National Academy Press

NHANES II (1976-80). The National Health and Nutrition Examination Surveys. Total Nutrient Intakes, Food Frequency and Other Related Dietary Data Tape. National Center for Health Statistics, 1983. Public use data tape documentation (Tape No. 5701).

Oracle (2007) Crystal Ball® version 7.2.1

Putnam, J and Allshouse, J (1992). Food Consumption, Prices, and Expenditures 1970-92. United States Department of Agriculture, Economic Research Service, Statistical Bulletin Number 867.

Rastani, RR, Grummer RR, et al. (2005). Reducing dry period length to simplify feeding transition cows: Milk production, energy balance, and metabolic profiles. Journal of Dairy Science **88**(3): 1004-1014.

Stanley, TA, Cochran RC, et al. (1993). "Periparturient Changes in Intake, Ruminal Capacity, and Digestive Characteristics in Beef-Cows Consuming Alfalfa Hay." Journal of Animal Science 71(3): 788-795.

Thornton, I and Abrahams, P. (1983). Soil ingestion - A major pathway of heavy metals into livestock grazing contaminated land. Sci Total Environ, 28:287-94.

USDA (1983) United States Department of Agriculture. Nationwide Food Consumption Survey Food Intakes: Individuals in 48 States, Year 1977-78, Report No 1-1. Hyattsville, Md: Consumer Nutrition Division, Human Nutrition Information Service.

USDA (1985)United States Department of Agriculture. Nationwide Food Consumption Survey. Continuing Survey of Food Intakes of Individuals, Women 19-50 Years and Their Children 1-5 Years, 1 Day, 1985. Report No 85-1, Hyattsville, Md: Nutrition Monitoring Division, Human Nutrition Information Service.

USDA (1986a)United States Department of Agriculture. Nationwide Food Consumption Survey. Continuing Survey of Food Intakes of Individuals, Women 19-50 Years and Their Children 1-5 Years, 1 Day, 1985. Report No 85-2, Hyattsville, Md: Nutrition Monitoring Division, Human Nutrition Information Service.

USDA (1986b) United States Department of Agriculture. Nationwide Food Consumption Survey. Continuing Survey of Food Intakes of Individuals, Men 19-50 Years, 1 day, 1985 Report No 85-3, Hyattsville, Md: Nutrition Monitoring Division, Human Nutrition Information Service.

USDA (1987a)United States Department of Agriculture. Nationwide Food Consumption Survey. Continuing Survey of Food Intakes of Individuals, Low-Income Women 19-50 Years and Their Children 1-5 Years, 1 Day, 1985. Report No 86-2, Hyattsville, Md: Nutrition Monitoring Division, Human Nutrition Information Service.

USDA (1987b) United States Department of Agriculture. Nationwide Food Consumption Survey. Continuing Survey of Food Intakes of Individuals, Women 19-50 Years and Their Children 1-5 Years, 1 Day, 1985. Report No 86-1, Hyattsville, Md: Nutrition Monitoring Division, Human Nutrition Information Service.

USDA (1987c) United States Department of Agriculture. Nationwide Food Consumption Survey. Continuing Survey of Food Intakes of Individuals, Women 19-50 Years and Their Children 1-5 Years, 4 Days, 1985. Report No 85-4, Hyattsville, Md: Nutrition Monitoring Division, Human Nutrition Information Service.

USDA (1988) United States Department of Agriculture Nationwide Food Consumption Survey. Continuing Survey of Food Intakes of Individuals, Low-Income Women 19-50 Years and Their Children 1-5 Years, 4 Days, 1985. Report No 85-5, Hyattsville, Md: Nutrition Monitoring Division, Human Nutrition Information Service.

Technical Support Document for Exposure Assessment and Stochastic Analysis, FINAL, August, 2012

USDA (1989-91) United States Department of Agriculture. Nationwide Food Consumption Survey. Continuing Survey of Food Intakes of Individuals (Data Tapes) Hyattsville, Md: Nutrition Monitoring Division, Human Nutrition Information Service.

USEPA, 2008 Child-Specific Exposure Factors Handbook (Final Report) 2008. U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-06/096F

8 Water Intake Rates

8.1 Introduction

Surface water can serve as a source of domestic water in some locations, particularly rural areas. Airborne contaminants from facilities can deposit directly on surface water bodies, thus exposing humans to contaminants through water consumption. Hot Spots facilities having non-municipal surface bodies of water, which are within the facility's zone of impact and which are used as a source of drinking water, need to include the water pathway in their risk assessments. Note that this pathway is rarely invoked for typical facilities in the Air Toxics Hot Spots program. Hot Spots risk assessments do not include municipal or commercial water sources. Municipal water is excluded because surface reservoirs are generally so large that contaminants from a single source become highly diluted once they enter the surface water body. Further, the level of some contaminants in municipal water may be reduced by water treatment processes typically used for municipal water supplies.

OEHHA does not recommend water pathway algorithms for municipal water source evaluation because the simple model used in the Hot Spots program is not adequate for this purpose. In these guidelines, the algorithm for calculating the water concentration of contaminants only includes that amount of chemical that directly deposits onto the surface of the water and not amounts that deposit onto surface soil and then enter the water body via runoff. It is assumed that contaminants initially deposited onto the water body surface remain suspended in the water column.

Water can be consumed by individuals through various forms of foods and beverages. For Hot Spots program risk assessments, the assessment only considers plain drinking water, water added for reconstituting foods and beverages, and water absorbed by food during cooking. This is because these foods and beverages could be made with water from a non-municipal surface water body. The risk assessment does not include water from commercial food or drink, or water that occurs naturally in fresh foods (e.g., water in an apple). The reasons for these exclusions are given in the paragraph above.

8.2 Recommendations

8.2.1 Point Estimate Approach

Currently there are no water intake distributions specific for California residents. However, OEHHA's derived water intake rate distributions provide a reasonable basis for exposure assessments of the California population. Chemical specific properties such as volatility may influence alternate route exposures via tap water, e.g., by bathing, showering, flushing toilets, etc. In the Air Toxics "Hot Spots" program, these exposure routes are currently not considered. However, they are treated in Superfund risk assessments where ground water contamination is a larger issue. The following

recommendations are based on currently available data. Depending on the nature of the analysis, one or more of the recommendations may apply.

For ages involving infants, OEHHA recommends using intake rates based on reconstituted formula intake. This is to protect the sizable subpopulation of infants who typically receive significant amounts of water through reconstituted formula. Breastfed infants, particularly during the first 6 months of age, are essentially non-consumers of water, and should not be included in the derivation of water intake rates designed to protect exposed infants.

For cancer risk assessment, the cancer risk estimates for exposures in the third trimester and from 0<2 years are weighted by an age sensitivity factor of 10 and exposures for the 2<16 year age groups are weighted by an age sensitivity factor of 3 (OEHHA, 2009). These age groups do not completely fit the 0-9, 0-30, and 0-70 year exposure duration scenario age groups. In order to properly weight for these periods and evaluate risk over each of the exposure duration scenarios, water intake rates specific for the third trimester, 0<2, 2<9, 2<16, 16-30, and 16-70 year age groups are needed. For example, for the 9 year scenario, intake rates are needed for the third trimester, the period from 0<2 year (for which the cancer risk will be weighted 10X), and for the 2-9 year period (for which the cancer risk will be weighted 3X). Likewise, for the 30 year exposure scenario, water intake rates are needed for the third trimester, 0<2 year, 2<16 year, and 16-30 year periods. Similarly, for the 70 year exposure scenario, water intake rates are needed for the third trimester, 0<2, 2<16, and 16-70 year periods. OEHHA has derived water intake rates for these additional age groups using the steps and methods outlined in Section 8.2.9 (“OEHHA Derived Water Intake Rates”) below.

Table 8.1 presents recommended point estimate water intake rates for Air Toxics Hot Spots risk assessments. The derivation is described below in section 8.4.13.

8.2.2 The Stochastic Approach

When using distributions it is appropriate to truncate them to avoid impossibly large or small values. For drinking water ingestion, the minimum should be set to zero while the maximum should be set to the maximum value listed in Table 8.11.

Recommended water intake rates for stochastic analyses are presented in Table 8.2.

Table 8.1 Recommended Point Estimate Tap Water Intake Rates (ml/kg-day)

Point Estimates				
Using Mean Values	**For the Age Period**	**9-year scenario**	**30-year scenario**	**70-year scenario**
	3rd trimester	18	18	18
	0<2 years	113	113	113
	2<9 years	26	-	-
	2<16 years	-	24	24
	16-30 years	-	18	-
	16-70 years	-	-	18
Using 95th-percentile values	**For the Age Period**	**9-year scenario**	**30-year scenario**	**70-year scenario**
	3rd trimester	47	47	47
	0<2 years	196	196	196
	2<9 years	66	-	-
	2<16 years	-	61	61
	16-30 years	-	47	-
	16-70 years	-	-	45

Table 8.2 Recommended Distributions of Tap Water Intake Rates (ml/kg-day) for Stochastic Risk Assessment

	9-year scenario	**30-year scenario**	**70-year scenario**
0<2 years	**Max Extreme** Likeliest = 93 Scale = 35	**Max Extreme** Likeliest = 93 Scale = 35	**Max Extreme** Likeliest = 93 Scale = 35
2<9 years	**Weibull** Location = 0.02 Scale = 29 Shape = 1.3		
2<16 years		**Gamma** Location = 0.19 Scale = 15.0 Shape = 1.6	**Gamma** Location = 0.19 Scale = 15.0 Shape = 1.6
16-30 years		**Gamma** location=0.49 scale=13.6 shape=1.26	
16-70 years			**Beta** min=0.17 max=178 alpha=1.5 beta= 12.9

8.2.3 Recommended Water Intake Rates for Lactating Subpopulations

OEHHA also recommends water intake rates specific for lactating subpopulations. These recommendations are presented in Table 8.18 in Section 8.5.2. In the point estimate approach, the mean and 95th percentile intake rate for lactating women should be used for the drinking water exposure of a mother when evaluating contaminant concentrations in breast milk. For stochastic analyses, OEHHA recommends using the percentile data for the lactating subpopulations in Table 8.18 and fitting each to distributional models using the procedure outlined in Sections 8.4.13 and 8.4.14. Although the same study derived water intake rates for pregnant women, we utilized the water intake rates for adults ages for the third trimester as they were slightly more health protective than the values derived for pregnant women by U.S. EPA (2004) and presented in Section 8.5.2 below.

8.2.4 Recommended Water Intake Rates for High Activity Levels / Hot Climates

For groups who may be highly physically active or who may live or work in hot climates, OEHHA recommends using the 95th percentile value in Table 8.1 for the age group for which the sensitive endpoint has been identified. For stochastic analyses, OEHHA recommends using the distributions for 9-year or 30-year scenarios in Table 8.2.

8.3 Water Intake Algorithm

The equation to calculate contaminant concentration in surface water for the Air Toxics "Hot Spots" risk assessment model is:

$$Cw = GLC * Dep\text{-}rate * 86{,}400 * SA * 365 / (WV * VC) \qquad \textbf{(Eq. 8-1)}$$

where: Cw = Average concentration in water (µg/kg)

GLC = Ground-level concentration of the pollutant (µg/m^3)

Dep-rate = Vertical rate of deposition (m/sec) (0.02 meters/second for controlled, or 0.05 meters/second for uncontrolled, sources.)

86,400 = Seconds per day conversion factor (sec/d)

SA = Water surface area (m^2)

365 = Days per year (d/yr)

WV = Water volume (kg) (1L = 1 kg)

VC = Number of volume changes per year

Site-specific values for SA, WV, and VC are needed for evaluating the surface water exposure pathway and can be estimated from data collected on-site or public data sources. The equation assumes that all material deposited into the water remains in the water column and that the deposition rate remains constant for a 9, 30 or 70-year exposure duration.

Estimating the daily oral dose of contaminants via the water intake pathway requires information on typical daily water intake of individuals. Typical water intake varies

among individuals. Characterizing this inter-individual variability allows more accurate estimates of average and high end intake as well as characterizing a range of exposures to the population.

Water intake can be classified as tap water or total water. Tap water is water consumed directly from the tap (i.e., plain drinking water) as well as water used to reconstitute beverages (e.g., coffee, OJ) or foods (e.g., baby cereal), and water absorbed during cooking of foods (e.g., cooked oatmeal) in the home or at a food service establishment (e.g., school, restaurant). "Total water" consists of tap water, plus water found naturally in foods (e.g., in a fresh apple), and water that is in commercial beverages (e.g., soft drinks) and foods (e.g., canned spaghetti). The term "direct" is used by the USEPA (2008) to describe tap water consumed from the tap. The term "indirect" is used to describe tap water used to make foods or beverages. Water in purchased items such as canned soup and intrinsic water in items such as lettuce were not included in the indirect category.

For the Hot Spots program, we are interested in tap water intake rates of consumers. We use tap water intake rates because tap water does not include water from commercial sources and from fresh foods. Commercial food and beverages are excluded because they are almost certainly prepared using water from municipal sources. In addition, commercial food and drink are typically from diverse sources resulting in minimization of the likelihood of a person being exposed from a single source (i.e., facility) from commercial products. Water in fresh foods is excluded because it does not come from a local water source. We use consumer-only data because consumers are the population being exposed. Thus, for example, data from non-consumers, such as individuals who exclusively drink bottled water, would be excluded from the data we use to quantify tap water intake rates.

The sources for tap water are municipal (public) water, household wells or cisterns, and household or public springs. The Hot Spots program water pathway risk assessments apply to water obtained from non-municipal surface water sources impacted by a given facility's emissions. Because non-municipal surface water is delivered via the tap (faucet) to consumers, and because most studies that have measured water consumption do not specify non-municipal surface water sources, we will use "tap" water data for the estimation of intake rates.

For stochastic evaluation of exposures from the water pathway, probability distributions reflecting variability within the population are needed. There are intake data that are available in ml/kg-day. By normalizing water intake by body weight, the variability associated with the correlation between water intake and body weight is reduced.

Historically, when estimating exposures via drinking water, risk assessors assumed that children ingest 1 liter/day of water, while adults ingest 2 liters/day (NAS, 1977). These values have been used in guidance documents and regulations issued by the U.S. Environmental Protection Agency (U.S. EPA). The purpose of this section is to briefly assess data on water intake rates for use in stochastic types of exposure assessments that employ distributions of water intake. In addition, point estimates of intake can be

identified from the distribution and used in the point estimate approach (Tier 1 and 2).

The algorithm for determining dose from surface drinking water sources is:

$$DOSEwater = 1 \times 10^{-6} * Cw * WIR * ABSwa * Fdw * EF \quad \textbf{(Eq. 8-2)}$$

where:

DOSEwater = daily oral dose of contaminant, mg/kg-d

1×10^{-6} = conversion factor (1 mg/1000 µg) (1L/1000 ml)

Cw = Concentration of contaminant in drinking water, µg/L

WIR = Water intake rate for receptor of concern in ml/kg BW-day

ABSwa = GI tract absorption factor (default = 100%)

Fdw = Fraction of drinking water from contaminated source (default = 100%)

EF = Exposure frequency (days/year)

In practice, the GI tract absorption factor (ABSwa) is only used if the cancer potency factor itself includes a correction for absorption across the GI tract. It is inappropriate to adjust a dose for absorption if the cancer potency factor is based on applied rather than absorbed dose. The Fdw variate is always 1 (i.e., 100%) for Tier 1 risk assessments. This variate may only be adjusted under Tier 2-4 risk assessments. The exposure frequency (EF) is set at 350 days per year (i.e., per 365 days) following U.S. EPA (1991).

For cancer risk, the risk is calculated for each age group using the appropriate age sensitivity factors (ASF) and the chemical-specific cancer potency factor (CPF), expressed in units of $(mg/kg\text{-}day)^{-1}$.

$$RISKwater = DOSEwater * CPF * ASF * ED/AT \quad \textbf{(Eq. 8-3)}$$

Exposure duration (ED) is the number of years within the age groupings. In order to accommodate the use of the ASFs (see OEHHA, 2009), the exposure for each age grouping must be separately calculated. Thus, the DOSEwater and ED are different for each age grouping. The ASF, as shown below, is 10 for the third trimester and infants 0<2 years of age, is 3 for children age 2<16 years of age, and is 1 for adults 16 to 70 years of age.

ED = Exposure duration (years):

0.25 yrs for third trimester	(ASF = 10)
2 yrs for 0<2 age group	(ASF = 10)
7 yrs for 2<9 age group	(ASF = 3)
14 yrs for 2<16 age group	(ASF = 3)
14 yrs for 16<30 age group	(ASF = 1)
54 yrs for 16-70 age group	(ASF = 1)

AT, the averaging time for lifetime cancer risks, is 70 years in all cases. To determine

lifetime cancer risks, the risks are then summed across the age groups:

$$RISKwater_{(lifetime)} = RISKwater_{(3rdtri)} + RISKwater_{(0<2\ yr)} + RISKwater_{(2<16\ yr)} + RISKwater_{(16\ yr\ onward)} \quad \textbf{(Eq. 8-4)}$$

As explained in Chapter 1, we also need to accommodate cancer risk estimates for the average (9 years) and high-end (30 years) length of time at a single residence, as well as the traditional 70 year lifetime cancer risk estimate. For example, assessing risk for a 9 year residential exposure scenario assumes exposure during the most sensitive period, from the third trimester to 9 years of age and would be presented as such:

$$RISKwater_{(9\text{-}yr\ residency)} = RISKwater_{(3rdtri)} + RISKwater_{(0<2\ yr)} + RISKwater_{(2<9\ yr)} \quad \textbf{(Eq. 8-5)}$$

For the 30-year residential exposure scenario, risk for the 2<16 and 16<30 age groups would be added to risks for exposures in the third trimester and ages 0<2 years. For the 70 year lifetime risk, Eq 8-4 would apply.

8.4 Water Intake Rate Studies

Water intake rates have been estimated through the collection of empirical (measured or self-reported) intake data. Some studies have modeled these data by fitting them to distributions. Both U.S. EPA and Cal/EPA (OEHHA) have reviewed and made recommendations for water intake rates in their exposure guidelines. In this section (8.4) we will present background on the major studies that have collected or modeled water intake rate data as well as summarize U.S. EPA (Exposure Factors Handbooks) and OEHHA (Air Toxics "Hot Spots" Program Exposure and Stochastic guidelines) exposure guidelines. We review and present water intake values in ml/kg-day because these rates are needed for Equation 8.2 (above). The studies and guidelines are presented chronologically, below. We also describe and present the estimates derived by OEHHA for the current guidelines.

It is important to note that currently available water intake data were collected over short-term periods (one to three days). These data do not reflect long-term typical water intake rates because repeated measures are not available on the same individual over long periods. Therefore, the variability of currently available estimates includes both intra- and inter-individual variability. These two types of variability cannot be separately evaluated with the current data. The average long term intake is better estimated by such data than high end intake.

8.4.1 Canadian Ministry of National Health and Welfare (1981)

The Canadian Ministry of National Health and Welfare (1981) study was conducted in the summer of 1977, the winter of 1978, and involved 970 individuals in 295 households. Interview and questionnaire techniques were used to determine per capita intake of tap water in all beverages (water, tea, coffee, reconstituted milk, soft drinks, homemade alcoholic beverages, etc.). Patterns of water intake were analyzed with

respect to age, sex, season, geographical location, and physical activity. Average daily intake rates by age group are presented in Table 8.3 (below). OEHHA did not use data from the Canadian study because the overall climate of Canada tends to be colder than California, the estimates are not likely representative of the current demographics of the U.S. population, and the raw data necessary to determine distributional characteristics were not available.

Table 8.3 Average Daily Water Intake (ml/kg-day) from the Canadian Ministry of National Health and Welfare (1981)

Age	Females	Males	Both sexes
<3 years	53	35	45
3-5 years	49	48	48
6-17 years	24	27	26
18-34 years	23	19	21
35-54 years	25	19	22
55+ years	24	21	22
All Ages	24	21	22

8.4.2 Ershow and Cantor (1989), Ershow et al. (1991)

The Ershow and Cantor (1989) and Ershow et al. (1991) studies analyzed drinking water intake rates using the 1977-1978 Nationwide Food Consumption Survey (NFCS) data. Tap water intakes include tap water consumed as plain water and tap water added, while at home or at restaurants, in the preparation of food and beverages. There were approximately 20,000 study participants. Data were analyzed by age group, sex, season, and geographic region (including the Western Region), and separately for pregnant women, lactating women, and breast-fed children. Intakes were normalized to body weight using self-reported body weights. Because the Western Region estimates of the NFCS most closely reflect intake patterns of California, the Western Region estimates were recommended in the prior version of the Air Toxics Hot Spots Program Exposure Guidelines (OEHHA, 2000).

The Western Region estimates are presented by age group in Table 8.4. These estimates are based on about 16 percent of the total data set. Note that the traditional assumption of 2 liters daily water intake for a 70 kg body weight person corresponds to approximately the 75th percentile on Ershow and Cantor's distribution (28 ml/kg-day, see Table 8.4). Table 8.5 summarizes the intake estimates for pregnant women, lactating women, and breast-fed children of the Ershow and Cantor study. Though the Ershow and Cantor (1989) and Ershow et al. (1991) studies presented extensive analyses of the NFCS data, more recent intake data that more closely reflect current water intake patterns are now available.

Table 8.4 Tap Water Intake Rates (ml/kg-day) of the Western Region, from Ershow and Cantor (1989) [1]

	Mean (SD)	50%	75%-ile	90%-ile	95%-ile
All Ages	24 (17)	21	30	43	54
< 1 year	53 (51)	39	67	106	141
1-10 years	39 (24)	34	49	70	88
11-19 years	18 (11)	17	24	32	39
20-64 years	21 (12)	19	27	37	44
65+ years	23 (10)	21	28	37	42

[1] Pregnant and lactating women, and breast-fed children excluded

Table 8.5 Tap Water Intake Rates (ml/kg-day) for Control, Pregnant and Lactating Women, and Breast-fed Children, from Ershow et al. (1991) [1]

	Mean (SD)	50%	75%-ile	90%-ile	95%-ile
Control [1]	19 (11)	17	24	33	29
Pregnant	18 (10)	16	24	35	40
Lactating	21 (10)	21	27	35	37
Breast-fed	22 (25)	12	38	56	60

[1] Control = women 15-49 years age who were not pregnant or lactating

8.4.3 Roseberry and Burmaster (1992)

Roseberry and Burmaster fit lognormal distributions to the datasets of Ershow and Cantor (1989) (discussed above). In tabulating the data they adjusted the data that were originally collected in 1977-78 to better represent the U.S. age group distribution of 1988. Although this study provided distributions of water intake, which is an essential component of stochastic analyses, OEHHA chose to not use these estimates because more recent water intake data are available. Further, the estimates are not normalized to body weight so they cannot be used or compared to the water estimates recommended in this document.

8.4.4 Levy et al. (1995)

Levy et al. (1995) evaluated fluoride intake of infants at 6 weeks, and 3, 6, and 9 months of age. At 6 weeks age, the sample size was 124, while at 9 months of age it was 77. Mothers were asked to record the average number of ounces of water per day over the past week that the infant consumed as plain water or that were used to make formula, juices and other beverages, baby food, cereal, and other foods consumed by the infant. These amounts were used to determine water intake. However, we did not use data from this study because only the mean and range were reported and because results were given as ounces per day, and were not normalized to body weight.

8.4.5 Exposure Factors Handbook (U.S. EPA, 1997)

The U.S. EPA's Exposure Factors Handbook (EFH) (U.S. EPA, 1997) reviewed water intake studies conducted before 1997 and made recommendations for water intake rate values in U.S. EPA risk assessments. The EFH (1997) used three key studies as the basis for their water intake recommendations: Canadian Ministry of National Health and Welfare (1981), Ershow and Cantor (1989), and Roseberry and Burmaster (1992) (see above). These studies were selected based on the applicability of their survey designs to exposure assessment of the entire United States population. U.S. EPA recommended 21 ml/kg-day as the average tap water intake rate for adults. This value is the population-weighted mean of the data from the Canadian Ministry of National Health and Welfare (1981) and Ershow and Cantor (1989). For the high-end adult value, U.S. EPA averaged the 90th percentile values from the same two studies to obtain a value of 34.2 ml/kg-day. The U.S. EPA recommended using the estimates of Roseberry and Burmaster (1992) for a characterization of the lognormal distribution of water intake estimates. However, U.S. EPA cautioned against using Roseberry and Burmaster (1992) for post-1997 estimates since these distributions reflect 1978 data adjusted to the U.S. age distribution of 1988. In addition to intake rates for adults, U.S. EPA also provided a table of intake rates for children, by age category, also from Ershow and Cantor (1989) and the Canadian Ministry of National Health and Welfare (1981).

OEHHA chose to not use the U.S. EPA (1997) estimates for these Hot Spots Exposure and Stochastic Guidelines because more recent data are available and different age groupings are needed for the Hot Spots risk assessment.

It should be noted that the USEPA released an external review draft of an updated Exposure Factors Handbook in 2009. The final version of the Exposure Factors Handbook was released in October, 2011 (U.S. EPA, 2011).

8.4.6 OEHHA (2000) Exposure Assessment and Stochastic Analysis Guidance

The previous version of the Hot Spots Exposure and Stochastic guidance (2000) recommended the "Western Region" water intake values of Ershow and Cantor (1989), which are presented in Table 8.4 (above). The Western Region was considered more applicable to California than the entire U.S. due to climate and lifestyle (e.g., physical activity) factors.

OEHHA (2000) provided point and distributional recommendations for the 9-, 30-, and 70-year exposure durations used with that guidance. For the 9-year scenario, OEHHA simulated a distribution using the tap water distributions presented by Ershow and Cantor (1989) for children <1 year of age and for children 1 to 10 years of age using Crystal Ball®. This distribution is presented below in Table 8.6. The distribution was fit to a lognormal parametric model with an arithmetic mean and standard deviation of 40.3 ±□21.6, μ ± [illegible] exp(3.57 ±0.50). The Anderson Darling Statistic is 0.65.

Table 8.6 OEHHA (2000) Tap Water Intake Rates Fit to a Lognormal Model for the 9-year Scenario (ml/kg-day) [1]

mean	SD	Percentiles										
		5	10	20	30	40	50	60	70	80	90	95
40	22	16	19	23	27	31	35	40	46	54	68	81

[1] Derived by OEHHA from data of ages 0-10 years from Ershow and Cantor (1989) fit to a lognormal distribution. Results presented in OEHHA Exposure Assessment and Stochastic Analysis Guidelines (2000)

For the 30- and 70-year scenarios, OEHHA used data for all ages of females from Ershow and Cantor (1989) to fit to a lognormal distribution with a mean of 24.0 and standard deviation (SD) of 17.2. The female mean was chosen because it is slightly higher than the male mean. Estimates of the fit to a lognormal model distribution are presented in Table 8.7, below.

Table 8.7 OEHHA (2000) Tap Water Intake Rates Fit to a Lognormal Distribution for the 30- and 70-year Scenarios (ml/kg-day) [1]

mean	SD	Percentiles										
		5	10	20	30	40	50	60	70	80	90	95
24	17	7	9	12	14	17	20	23	31	34	45	56

[1] Derived by OEHHA using data of females of all ages from Ershow and Cantor (1989) fit to a lognormal distribution. Results presented in OEHHA Exposure Assessment and Stochastic Analysis Guidelines (2000)

The OEHHA (2000) Exposure and Stochastic Guidance recommended using the mean and 95th percent-ile values from Table 8.6 and 8.7 (above) for each of the 9-, 30-, and 70-year scenarios. These recommended point values are presented in Table 8.8, below.

Table 8.8 Previously Recommended Point-Value Estimates for Daily Water Intake Rates (ml/kg-day) for the Exposure and Stochastic Guidelines of OEHHA (2000)

	9-year scenario (children)	30- and 70-year scenario
Average	40	24
High-end	81	54

For stochastic analyses using the OEHHA (2000) Exposure and Stochastic Guidance, the distributional values presented in Tables 8.6 and 8.7 (above) and fit to a lognormal distribution were recommended.

8.4.7 U.S. EPA Office of Water (2004)

The Office of Water, U.S. EPA, derived estimated water intakes using data from the Continuing Survey of Food Intake of Individuals (CSFII) 1994-1996, 1998 dataset. The CSFII 1994-1996, 1998 (hereafter referred to as CSFII) is a nationwide survey that collected data on food and beverage intakes for two 24-hour non-consecutive periods, 3-10 days apart, on approximately 20,000 individuals during the years 1994-1996 and 1998. The Office of Water estimated the amount of water consumed by each individual, including both direct and indirect water intake. Direct water intake is water consumed as plain water from the tap, while indirect water intake is water used to prepare beverages and foods, either at home or at a food service establishment.

Two-day average water intakes for each participant were used in the analyses. Results are presented by water source (tap, bottled, other sources, or all water sources), type of water (direct, indirect or both), consumption type (consumer-only or combined consumer plus non-consumer ("per capita")), and in units of L/day or L/kg-day. Fine and broad age groups were analyzed. This report provides the most recent published analysis of water intake rates that are representative of the U.S. population. The report includes results for both combined and separate analyses of direct and indirect water intakes. However, the Office of Water (2004) intake estimates are from data that is the average of two non-consecutive days of intake and thus do not reflect a person's long-term typical intake. The combined direct plus indirect, community water intake rates by age group from the Office of Water (2004) report are presented in Table 8.9, below. For all ages, the mean and 95th percentile water intake rates were 17 and 44 ml/kg-d.

Table 8.9 Direct + Indirect, Community Water Intake Rates From U.S. EPA (2004) Table IV-8 (ml/kg-day)

			Percentiles							
Age in Years	**Sample Size**	**Mean**	**5**	**10**	**25**	**50**	**75**	**90**	**95**	**99**
0<0.5	414	95	5	7	37	91	133	184	221	294
0.5<0.9	534	53	3	5	12	47	81	112	129	186
0<2	1828	44	2	4	11	28	62	109	137	215
1-3	3230	26	2	4	9	20	35	53	68	110
4-6	2715	22	1	3	8	18	31	47	63	91
0<6	6410	30	2	4	9	21	38	67	93	162
7-10	956	16	1	3	6	13	22	33	40	59
11-14	736	13	1	2	5	10	17	27	36	54
15-19	771	12	1	1	4	9	16	26	32	62
20+	8459	16	1	3	7	13	22	32	39	62
20-24	637	15	1	2	5	11	18	31	39	80
25-54	4512	16	1	3	7	13	21	32	40	65
55-64	1383	17	1	3	8	14	23	32	38	58
65+	1927	18	2	5	10	16	24	32	37	53
All Ages	17,815	17	1	3	7	13	22	33	44	77

8.4.8 U.S. EPA Child-Specific Exposure Factors Handbook (2008)

The U.S. EPA Child-Specific Exposure Factors Handbook (CEFH) provides exposure factor recommendations, including recommended water intake rate values for exposure assessments that are specific for infants and children.

The U.S. EPA (2008) undertook an analysis of the CSFII 1994-1996, 1998 dataset to derive water intake rates specific for the CEFH age groups. U.S. EPA (2008) defined direct water as water consumed as a beverage. They defined indirect as water used to make beverages or foods. In their analysis, the U.S. EPA did not differentiate between direct and indirect water resulting in intake estimates for combined direct plus indirect water.

The U.S. EPA (2008) presented separate analyses of water intake by water source (i.e., community, bottled, other sources, and all sources). The U.S. EPA (2008) presented both ml/day and ml/kg-day intake rate values, and mean, minimum, maximum, and eleven percentile bins of intake estimates. No recommendations for

fitted distributions for water intake rates were made in the CEFH (U.S. EPA, 2008). Both per capita and consumer only water consumption rates were presented.

8.4.9 CEFH Table 3-19

Of the tables in CEFH (U.S. EPA, 2008), Table 3-19 provides water intake estimates that were of the most relevance to OEHHA because these rates are for combined direct plus indirect community water intake. The table includes percentile values for consumer-only rates. Table 3-19 is presented in Table 8.10, below. OEHHA chose to use the estimates for some of these age groups in deriving OEHHA-specific age group water intake rates (see Section 8.4.13, below). This information is also published in Kahn and Stralka (2009).

Table 8.10 Table 3-19 U.S. EPA CEFH (2008). Consumer-only, Direct plus Indirect, Community Water Intake Rates By Age Group for U.S. Infants and Children (ml/kg-day)

	Sample Size	Mean	50th	90th	95th	99th
0<1 month	37	137	138	235	238	263
1<3 months	108	119	107	228	285	345
3<6 months	269	80	77	148	173	222
6<12 months	534	53	47	112	129	186
1<2 years	880	27	20	56	75	109
2<3 years	879	26	21	52	62	121
3<6 years	3703	24	19	49	65	97
6<11 years	1439	17	13	35	45	72
11<16 years	911	13	10	26	34	54
16>18 years	339	12	9	24	32	58
18<21 years	361	13	10	29	35	63

* Source of Data: USDA Continuing Survey of Food Intakes by Individuals (CSFII), 1994-96, 1998

8.4.10 Michaud et al. (2007)

Michaud et al. (2007) investigated the relationship between total fluid intake and bladder cancer. Participants were asked via questionnaire about the volume and frequency of specific beverages during the 5 years prior to the study interview. The researchers calculated total fluid intake by multiplying the volume and frequency of each beverage and summing the result. Because the fluid intake included fluids from commercial beverages, and because water absorbed into foods during cooking was not included,

we did not use these intakes. Further, intakes were only given as ml/day and results were reported as quintiles so only intervals of intake were reported (e.g., 29 ml/day, 29-40 ml/day, 41-55 ml/day, etc.).

8.4.11 Barraj et al. (2008)

Barraj et al. (2008) collected drinking water consumption data over a 7-day period on a nationwide sample of persons of all ages during two 'waves' (survey periods meant to represent winter and summer seasons). Diaries were used to record frequency and amounts of plain drinking water consumed. The final dataset contained data from 4198 individuals from 2154 households. The response rate was 33 percent and 36 percent for wave 1 and wave 2, respectively. The proportion of study participants by age-sex groups and U.S. region was comparable to those of the U.S. 2000 census, with the exception of women over 50 years of age. The proportion of whites in the study was greater than the U.S. census. Results included 24-hour drinking water consumption rates, number of occasions of drinking water, amount per occasion, and inter- and intra-individual variability in water consumption patterns. This study was restricted to plain drinking water, while we are interested in water used for reconstituting food and beverages and water absorbed during cooking, in addition to plain drinking water. Therefore we cannot use these data to quantify water intake rates. Nonetheless, the study did evaluate inter- and intra-individual variability in daily water intake (ounces per day) and found that inter-individual variability was greater than intra-individual variability. There were significant day-to-day differences in water intake (ounces per day) in "wave 1" (summer) for women 13-49 years of age and men 20-49 years of age, and in "wave 2" (winter/early spring) for children 0-5 and boys 13-19 years of age. There was also a significant weekend effect.

8.4.12 Kahn and Stralka (2009)

Kahn and Stralka (2009) published in a peer-reviewed journal the water intake rates that they had derived for the U.S. EPA, Office of Drinking Water (2004) report. This publication will not be discussed here because the methodology and results are presented in Section 8.4.7, above. However, we make note of this publication and that it has been reviewed for these guidelines.

8.4.13 OEHHA Derived Water Intake Rates for Hot Spots Program Age Groups and Exposure Duration Scenarios

OEHHA chose to use water intake estimates from the Office of Water, U.S. EPA (2004) and USEPA's CEFH (U.S. EPA, 2008) Table 3-19 as the basis for OEHHA's water intake rate recommendations (with the exception of the infant age group, see below). Both the Office of Water (2007) and U.S. EPA (2008) CEFH Table 3.19 intake estimates are representative of demographics (e.g., age, sex, income, etc.) of the U.S. population because they have been weighted using the data-specific sample and variance weights. The rates are in ml/kg-day, which is the unit of measure specified for the current Hot Spots program guidance (see Equation 8.1,

above). The Office of Water report and U.S. EPA (2008) CEFH Table 3.19 include consumer-only tap (community) water intake rates, which are of particular relevance for OEHHA because water consumed from local surface water bodies is likely to be made available to consumers via the tap at home. Though more recent water intake data are now available (NHANES 1999-2004), the NHANES water intake data are limited because information on whether the water was from the tap or not was not collected, and the water source (e.g., municipal, bottled, etc.) is not specified for several of the years. Further, although direct intake rates are in the NHANES dataset, to obtain the indirect intake rates that OEHHA needs would require calculations using recipe code books and other data manipulation. Thus, the Office of Water and U.S. EPA (2008) CEFH Table 3.19 rates, which are based on 1994-1996 and 1998 data, are the most recent derivation of direct and indirect water intake rates that are representative of the population.

It should be noted, though, that the Office of Water (2004) and U.S. EPA (2008) CEFH Table 3.19 intake rates are not available on a state-by-state basis. Thus, the rates used by OEHHA are not specific to California and therefore may differ from those of the California population due to different climate and lifestyle factors. However, it is likely that the rates would not be substantially different overall since there are other areas of the U.S. with climate and lifestyle patterns similar to those of California. Further, the California population represents a significant fraction (over 10%) of the national population and thus would have contributed some weight to the CSFII survey.

Because the age groups in the Office of Water report (2004) and U.S. EPA (2008) CEFH Table 3.19 differ from the age groups and exposure duration scenarios to be used for Hot Spots risk assessments, OEHHA derived water intake rates specific for the Hot Spots program ages. Table 8.11, below, lists the data sources used to derive water intake rates for the Hot Spots program.

Table 8.11 Data Used to Derive Water Intake Rates for Hot Spots Program Age Groups and Exposure Duration Scenarios

Hot Spots Age Group	Derived by OEHHA [1]	CEFH Revised Table 3-19 (2008)	Office of Water (2004)
0<2 years	0<1 year [1]	1<2 years	
2-9 years		2<3 years 3<6 years 6<11 years	
2<16 years		2<3 years 3<6 years 6<11 years 11<16 years	
16-30 years		16<21 years	20-24 years 25-54 years [2]
16-70 years		16<21 years	20-24 years 25-54 years 55-64 years [3]
>=16 years		16<21 years	20-24 years 25-54 years 55-64 years 65+ years
Hot Spots Exposure Duration	Derived by OEHHA [1]	CEFH Table 3-19 (2008)	Office of Water (2004)
9-year	0<1 year [1]	1<2 years 2<3 years 3<6 years 6<11 years	
30-year	0<1 year [1]	1<2 years 2<3 years 3<6 years 6<11 years 11<16 years 16<21 years	20-24 years 25-54 years [2]
70-year	0<1 year [1]	1<2 years 2<3 years 3<6 years 6<11 years 11<16 years 16<21 years	20-24 years 25-54 years 55-64 years [3]

[1] Using intakes of water in reconstituted formula consumed by infants in CSFII 1994-1996, 1998

[2] Because intake rates are relatively stable after 16 years of age, the 25-54 year age group was used to represent the 25-30 year age group but with population size adjusted to the 25-30 year age group

[3] Because intake rates are relatively stable between the 55-64 year and 65+ year age groups (mean of 17 vs. 18 and 95%-ile of 38 vs. 37, for the 55-64 and 65+ year age groups, respectively), OEHHA chose to use the 55-64 year age group to represent the 65-70 year age group and adjust for the additional 65-70 years of age population.

For the derivation of Hot Spots program age groups and exposure duration scenarios, OEHHA used Crystal Ball version 7.2 (Oracle, 2008) to find the best fit for distributions, to simulate values of distributions, and to identify distributional parameters (mean, scale, location, etc.). Crystal Ball was also used to derive percentiles and summary statistics. In identifying the best fit for a distribution, the Anderson-Darling test, one of three goodness-of-fit tests available in Crystal Ball, was used because it gives extra weight to the tails of the distribution, which the other goodness-of-fit tests do not. The tails of the distribution are of particular interest to OEHHA because the right tail defines high-end intake rates.

OEHHA did not use the Office of Water (2004) or U.S. EPA (2008) CEFH Table 3.19 water intake estimates for infant (0<1 year of age) intake rates. Instead, OEHHA derived water intake rates of infants consuming reconstituted formula. The reasons for this are described below in Section 8.5.1. OEHHA used data from the CSFII 1994-1996, 1998 dataset to derive infant water intake rates. To identify infants who received reconstituted formula, the food description provided for the formula consumed by each infant was reviewed. Breast-fed infants were excluded from analysis. To calculate the amount of water consumed by each infant, the amount of reconstituted formula consumed was multiplied by the percent of indirect water in each type of reconstituted formula (these values were obtained from Appendix-D of the U.S. EPA Office of Drinking Water report (2004)). Two outliers were identified and excluded from analyses. Sample weights were available in the dataset in order to weight each individual's intake according to the number of infants in the population that he/she represented (see USDA, 2000 for a more detailed description). Each infant's water intake was paired with her/his sample weight in Crystal Ball (version 7.2) to derive a distribution of intakes representative of the population. The Anderson-Darling goodness-of-fit test was used to find the best fit distribution for the weighted data. This weighting and best fit procedure was conducted for each infant age group (0<1, 1<2, 0<3, 3<6, and 0<12 months of age).

The OEHHA–derived water intake rates for these infant age groups are used in conjunction with other data to derive Hot Spots program age group and exposure duration scenario water intake rates (as outlined in Table 8.11, above). By doing so, the Hot Spots program water intake rates reflect intake rates of the truly exposed infants (those receiving reconstituted formula). The results are presented in Table 8.12, below, along with the Office of Water (2004) or U.S. EPA (2008) CEFH Table 3.19 estimates (direct plus indirect consumer-only community water intake rates) for comparison.

Table 8.12 Water Intake Rates of Infants by Age Group (ml/kg-day) – Derived by OEHHA (2008) or U.S. EPA (2004 or 2008)

Study	Age in Months	Sample Size	Mean	50%-ile	90%-ile	95%-ile	99%-ile
OEHHA CSFII [2]	0<1	45	184	171	253	300	466
U.S. EPA Table 3-19 [3]	0<1	37	137	155	236	269	269
OEHHA CSFII [2]	1<2	61	134	113	294	301	375
OEHHA CSFII [2]	0<3	137	122	113	206	294	375
U.S. EPA Table 3-19 [3]	0<3	108	119	107	247	289	375
OEHHA CSFII [2]	0<6	467	127	123	200	237	333
U.S. EPA (2004) [3]	0<6	414	95	91	184	221	294
OEHHA CSFII [2]	0<12	906	142	148	213	228	276
U.S. EPA (2004) [3]	0<12	948	71	62	145	185	261

[1]N = sample size. However, results have been weighted to adjust sample to the population.
[2]OEHHA analyses include water intake only from reconstituted formula
[3]U.S. EPA (2008) includes any direct or indirect intake of community water by consumers-only

A limitation of using intake data from infants receiving reconstituted formula is that the intakes do not include water added to food and non-formula drink, which results in possible underestimation of water intake. This limitation is likely only applicable to the second half of infancy when infants typically receive supplemental food and drink in addition to formula. A second limitation to the OEHHA derived infant intake rates are that the source of water (e.g., tap) used to reconstitute the formula is unknown. However, it is probable that a large fraction of infants are fed reconstituted formula prepared with tap water (see Section 8.5.1, below, for results of Levallois et al. 2007).

The Office of Water (2004) mean estimates are lower than the OEHHA mean estimates because they include data from infants who may have been almost exclusively (i.e., received an insignificant amount of calories from other non-milk food or drink), or exclusively, breast-fed. The 90th-, 95th-, and 99th-percentile estimates are similar among the analyses because these values likely represent

infants who are exclusively fed formula reconstituted with water. These values support the consistency of results among analyses, and indicate that some infants consuming reconstituted formula may have very high water intake rates.

To estimate intake rates for the Hot Spots 0<2 year age group, the percentiles of the distribution and associated intake values for the 0<1 year age group (OEHHA derived, see Table 8.12, above) were entered into Crystal Ball and used to characterize the probability distribution of the intake rates. The best fit for the distribution was identified using the Anderson Darling goodness-of-fit test. The parameters for the modeled distribution were then derived using the empirical minimum and maximum to truncate unrealistically low and high values. This process (characterizing the probability distribution) was repeated for the water intake values of the 1<2 year age group of the CEFH Table 3-19 (2008). Table IV-8 of the Office of Water (2004) provided data on the population size of each age group (0<1 year and 1<2 years) relative to the full age group (0<2 years).

The population proportion was multiplied by 60,000 to give the number of infants for each age group in a hypothetical population of 60,000 infants. The Latin Hypercube method of Monte Carlo simulation in Crystal Ball was then used to generate simulated values for the 0<1 year age group based on the calculated number of infants in the hypothetical population. The same simulation procedure was applied to the 1<2 year age group distribution. The simulated values were then combined into one dataset. The best fit for the distribution of the combined values was characterized using the empirical minimum and maximum values for truncation to eliminate potentially unrealistic extreme values. The parameters of the combined (0<2 year age group) distribution were identified and summary statistics calculated.

To derive distributions for the other Hot Spots age groups and exposure duration scenarios, the above described procedure was also used. That is, using the data outlined in Table 8.11 for each Hot Spots program age group and exposure duration scenario, the probability distribution was characterized, population proportions were calculated (using Office of Water Table IV-8), and values proportional to population size were simulated. The simulated values were then combined, the best fit for the resultant distribution was identified, and parameters and summary statistics for the distribution were found. It may be noted that when calculating population proportions, the age groups of Table IV-8 of the Office of Water (2004) did not always fit the CEFH Table 3-19 age groups. In these cases, some approximations were required.

Values for the OEHHA derived Hot Spots age groups and exposure duration scenarios are presented in Table 8.13, below.

Table 8.13 OEHHA Derived Consumer-only Water Intake Rates (ml/kg-day) for Hot Spots Program Age Groups and Exposure Duration Scenarios[1]

Age	Mean	50th	Variance	90th	95th	99th	Max
Third Trimester	18	14	218	38	47	67	117
0<1 year [2]	143	149	3240	213	228	276	491 [5]
0<2 years [2]	113	106	1915	172	196	247	491 [4]
2-9 years [3]	26	22	414	54	66	92	190 [5]
2<16 years [3]	24	19	362	49	61	88	152
>=16 years [3]	19	16	208	38	47	67	135 [5]
16-30 years [3]	18	14	218	38	47	67	117
16-70 years [3]	18	15	191	37	45	62	116
Duration							
0-9 year [2]	45	25	3052	102	152	288	491
0-30 year [2]	28	15	1219	59	87	177	450
0-70 year [2]	23	14	886	51	73	141	442

[1]OEHHA recommends the mean and 95th percentiles as the average and high end point estimate values.

[2]Includes the OEHHA derived 0<1 year of age group water intake rates derived from the water in reconstituted formula for infants in CSFII

[2]OEHHA derived – data sources are **c**onsumer-only, direct + indirect, community water intake rates from Office of Water (2004) and U.S. EPA CEFH (2008) Table 3.19.

[4]Right tail outliers deleted

[5]fit distribution has maximum of infinity

8.4.14 Fitted Distributions of OEHHA Derived Water Intake Rates

The steps involved in deriving water intake rates specific for the Hot Spots program age group and exposure duration scenarios are described above, and briefly discussed here. OEHHA characterized the probability distributions for certain age group datasets from the Office of Water (2004) or Table 3-19 (2008) using Crystal Ball version 7.2 (Oracle, 2008). The best fit distributional type (e.g., gamma) was then found using the Anderson-Darling goodness-of-fit test. The parameters of the best fit distribution were then determined. Distributions were combined as listed in Table 8.11 to provide age groups matching the age groups needed for the Air Toxics Hot Spots program. The distributions were combined proportionate to population size which was approximated using the population numbers in U.S. EPA (2004). The mean and percentiles were calculated for the combined age group distributions using Crystal Ball 7.2 (Oracle, 2008)

and the results are presented in Table 8.13, above. The combined age group distributions were characterized using Crystal Ball to find the best fit distribution, the Anderson-Darling statistic for that fit, and the parameters that fit that distribution. The distributional characteristics and values are presented in Table 8.14, below.

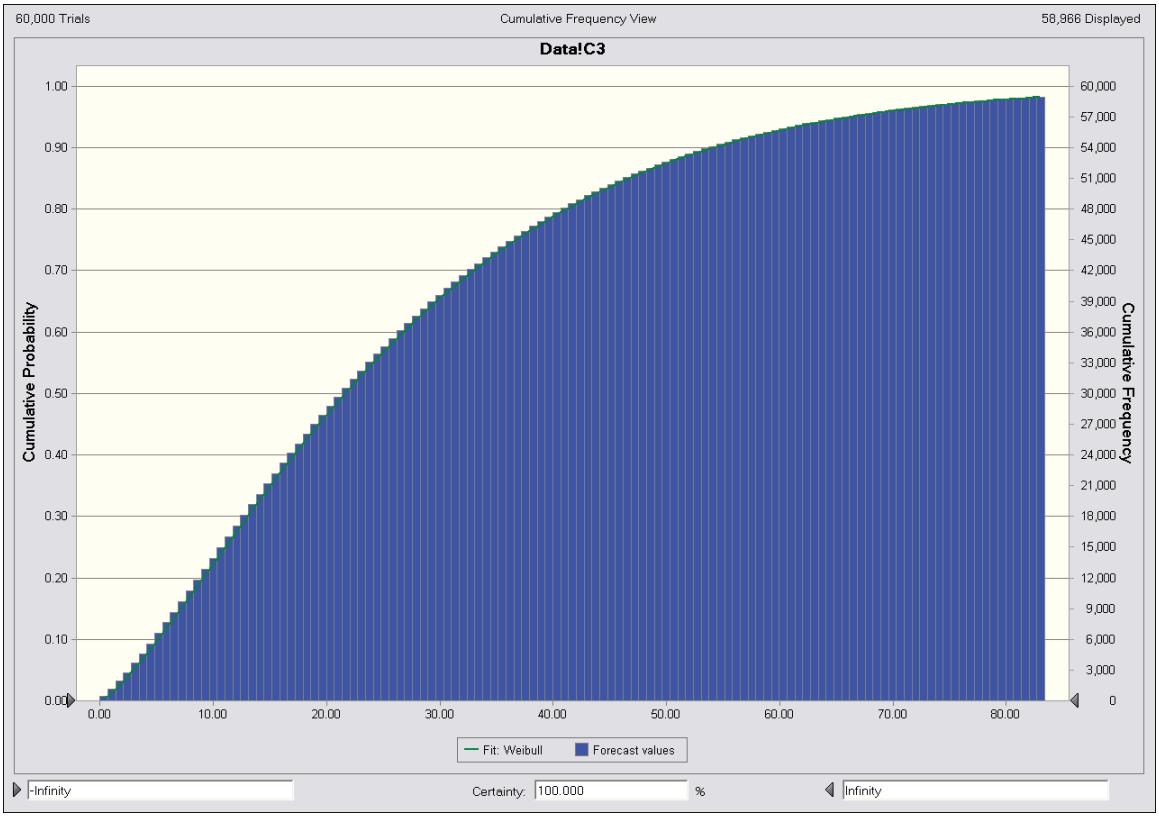
Table 8.14 Recommended Distributions of OEHHA Derived Water Intake Rates for Stochastic Analysis (ml/kg-day)

Age	Best Fit [1]	A-D statistic [2]	Parameters of Distribution [3]
0<1 year	Beta	23.2	Min = 60 Max = 264 Alpha = 4.1 Beta = 2.5
0<2 years [5]	Max Extreme	1.06	Likeliest = 93 Scale = 35
2<9 years	Weibull	0.01	Location = 0.02 Scale = 29 Shape = 1.3
2<16 years [6]	Gamma	0.11	Location = 0.19 Scale = 15.0 Shape = 1.6
≥16 years [6]	Gamma	0.52	Location = 0.17 Scale = 10.7 Shape = 1.8
16-30 year[7]	Gamma	10.6	location=0.49, scale=13.6, shape=1.26
16-70 year	Beta	1.09	min=0.17, max=178, alpha=1.5beta= 12.9
Duration			
0-9 year scenario	Lognormal	2.7	Mean = 45 SD = 70
0-30 year scenario	Lognormal	0.31	Mean = 26 SD = 39
0-70 year scenario	Lognormal	0.04	Mean = 23 SD = 29

[1]Best Fit refers to the distribution found to best fit the empirical data according to the Anderson-Darling goodness-of-fit test
[2]A-D statistic = Anderson-Darling statistic
[3]Parameters of Distribution refers to the parameters of the best fit distribution
[4]Taken directly from U.S. EPA CEFH (2008) Table 3.19.
[5]0<2 year age group derived by combining water in reconstituted formula only for 0<12 month ages from CSFII and the 1<2 year age group from U.S. EPA CEFH (2008) Table 3.19
[6]OEHHA analyses that derived alternate age groups using U.S. EPA (2004) and U.S. EPA CEFH (2008) Table 3.19.
[7]This distribution is recommended for the third trimester also.

To give a graphical example of the OEHHA derived distributions, the cumulative probability of the 2-9 year of age distribution (best fit) is shown below, in Figure 8.1.

Figure 8.1. Cumulative Probability Distribution for Water Intake Rates (ml/kg-day) for 2-9 Years of Age



8.5 Special Subpopulations of Concern

8.5.1 Infants

Infants may be more sensitive and exposed (on a body weight basis) to some toxicants than non-infant children and adults. Further, infants have unique nutritional needs, necessitating the feeding of milk or milk substitutes through at least three, and more commonly through four to six months of age. For the first 4-6 months, infants who are fed breast milk typically receive little, if any, other fluid. This is primarily because continued lactation is dependent on continued nursing. If nursing is reduced or discontinued for any length of time, the milk production quickly ceases. Thus, breast-fed infants tend to receive breast milk as their sole source of fluid and nutrition during the first half of infancy.

On the other hand, infants who are not breast-fed receive formula. The Ross Mothers Survey (Ross Products Division, Abbott, 2003) reported that in 2003,

44 percent, 18 percent, and 10 percent of infants were exclusively breastfed (no other liquids) in the hospital (i.e., soon after birth), at 6 months of age, and at 12 months of age, respectively. This suggests that the percent of infants who receive at least some formula may be up to 56 percent soon after birth and 82 percent at 6 months of age.

Formula can be bought ready-to-feed or in a form requiring the addition of water before it can be fed to the infant (i.e., powder or concentrated liquid). OEHHA analyzed the CSFII 1994-1996, 1998 and NHANES (National Health and Nutrition Examination Survey) 1999-2004 dataset to assess the proportion of infants who received reconstituted formula, relative to all types of formula. The food code descriptions were reviewed to identify the type of formula each infant received, including reconstituted formula. The results are presented in Table 8.15, below. These results provide evidence that a large fraction of formula-fed infants receive reconstituted formula, especially so for the youngest ages. These results also suggest that there may be a trend over time toward greater consumption of reconstituted formula relative to ready-to-feed formula.

Table 8.15 Percent of formula-fed infants consuming reconstituted formula

Age	CSFII	NHANES
0 < 1 month	82% (45 / 55) [1]	94% (31 / 32)
0<6 months	71% (467 / 658)	87% (398 / 457)
0<12 months	75% (906 / 1201)	87% (886 / 1013)

[1] ( ) = # receiving reconstituted formula / # receiving any type formula

Additionally, a study of 2-month old infants in rural Canada (with a sample size of approximately 300) found that 91 percent of formula-fed infants received formula reconstituted with water (Levallois et al., 2007). This is consistent with the results in Table 8.15, above. Because OEHHA is particularly interested in tap water intake rates, it is important to note that, of the Canadian infants receiving reconstituted formula, 60 percent received formula reconstituted with tap water.

Because the majority of formula-fed infants receive formula that has been reconstituted with water, which is often tap water (60 percent per Levallois et al., 2007), during the first half of infancy, the infant population is dichotomized into infants who receive little, or no, tap water (breast-fed infants) and infants who receive significant amounts of tap water every day (reconstituted formula fed infants).

While the infant's diet during first half of infancy typically consists almost exclusively of breast milk or formula, infant diet during the second half is much more varied and includes the gradual introduction of food and non-milk beverages. (The term

'second half of infancy' is used loosely here because the age at which food and non-milk drink is introduced varies but is typically between 4-6 months of age). Nonetheless, during this second half of infancy, the dichotomization of infants into two groups based on water intake rates continues, though the difference between the groups may be somewhat less pronounced.

The American Academy of Pediatrics (AAP, 1997) recommends that infants be exclusively breast-fed through 6 months of age and continue to receive breast milk as their sole source of milk while being introduced to solid food through 12 months. Thus, breast-fed infants may begin to receive some food and drink prepared with water but often not until at least 6 months of age. Further, breast-fed infants frequently continue to receive breast milk as a significant source of fluid and nutrition for several months past the introduction of supplemental food and drink. For formula-fed infants, because the accepted medical recommendation is to not feed cow's milk until at least 12 months of age, formula-fed infants typically continue to receive formula as their sole milk source. Like breast-fed infants, formula-fed infants may increase their intake of food and non-formula drink prepared with water during this period. Both breast-fed and formula-fed infants tend to decrease their consumption of breast milk or formula, respectively, while their consumption of food and drink prepared with water is likely to increase. Thus, during the second half of infancy, overall water intake of breast-fed infants likely increases, though probably not dramatically, while intake of formula-fed infants likely varies considerably between infants but with the potential for some infants to have even greater intake rates than during the first half of infancy.

The above information supports the existence of a sizable subpopulation of infants who are exclusively (or almost exclusively) fed formula reconstituted with water, which is often tap water, for the first 4-6 months and thereafter receive significant quantities of tap water through 12 months of age. These infants could receive significant tap water intake over the first year of life. In the past few years, there has been heightened awareness of the probable increased susceptibility of infants and children to some environmental toxicants. Therefore, it is prudent to identify subpopulations of infants who may be the most highly exposed. For the water pathway, reconstituted formula-fed infants can have a very high rate of tap water intake over the first year of life. Thus, water intake rates representative of this subpopulation (reconstituted formula fed infants) should be used for assessments of infants to exposures via the water pathway.

In risk assessment, we are interested in the dose to those who are exposed; in the case of the water pathway, those who consume water. With water intake, some individuals may not consume water on one or more days, or consume insignificant amounts of water (e.g., breast-fed infants). For the 'consumer-only' groups of infants in the Office of Water report, (U.S. EPA, 2004), only mean (average) values were given and these were only for the 0<6 and 0<12 month ages (i.e., relatively broad age groups for infants). In Table 3-19, consumer-only rates include percentiles of the distribution and the ages are stratified into narrower age groups (i.e., 0<1, 1<3, 3<6, and 6<12 months of age).

Of interest to OEHHA are rates of direct plus indirect community water intakes for narrow age groups of consumer-only infants. With such rates, both central tendency plus high-end rates of potentially more susceptible and exposed infants can be identified. U.S. EPA (2008) CEFH Table 3.19 provides these estimates. The U.S. EPA (2008) CEFH Table 3.19 infant estimates are presented in Table 8.16, below. However, the data used to derive these estimates included infants who were breast-fed. Therefore, these values do not represent the high-end exposure subpopulation of formula-fed infants.

Table 8.16 Infants Only -- U.S. EPA (2008), Child-Specific Exposure Factors Handbook Table 3-19. Estimates of Direct + Indirect, Consumer-only, Community Water Intake By Age Group (ml/kg-day)

Age (years)	Mean	Min	Percentiles (ml/kg-day)								
			5	10	25	50	75	90	95	99	Max
0<1 month	137	5	11	11	67	155	198	236	269	269	269
1<3 months	119	3	9	12	72	107	153	247	289	375	375
3<6 months	80	1	3	7	28	77	118	149	174	224	288
6<12 months	53	0	3	5	12	48	81	112	130	186	254

8.5.2 Pregnant and Lactating Women

Pregnant and lactating women have greater water requirements than non-pregnant or non-lactating women. A pregnant woman requires increased water intake in order to support fetal circulation, amniotic fluid, and a higher maternal blood volume, while a lactating woman requires increased water to replace the water excreted in breast milk. Values from the literature support this hypothesis. OEHHA (2000) Exposure Assessment and Stochastic Analysis Guidelines presented a table based on Ershow and Cantor (1989) that compared water intake rates of pregnant and lactating women with 'control' (not lactating, not pregnant) women of the same ages (see Table 8.17, below). These estimates demonstrate that lactating women consume significantly more water than non-lactating and pregnant women. More recent data are available than the values in Table 8.17. Therefore the values from Table 8.17 will not be used for Hot Spots guidance values.

Table 8.17 Water Intake Estimates For Pregnant and Lactating Women from Ershow and Cantor (1989) (ml/kg-day) – Tap Water

Group	Sample size	mean	Percentiles			
			50th	75th	90th	95th
Control	6201	19	17	24	33	39
Pregnant	188	18	16	24	35	40
Lactating	77	21	21	27	35	37

* Data from Ershow et al. 1991 based on data from the USDA Nationwide Food Consumption Survey (NFCS 1977-78)

The Office of Water, U.S. EPA (2004) report presented estimates of water intake rates for pregnant and lactating women. These rates are derived from CSFII 1994-1996, 1998 data. The consumer-only intake rates of direct plus indirect community water intakes are presented in Table 8.18 below.

Table 8.18 Water Intake Rates of Direct + Indirect Community Water for Consumers-only (ml/kg-day) for Pregnant, Lactating, and Non-pregnant / Non-lactating Women 15-40 Years of Age

Group	Sample size	mean	Percentiles				
			50th	75th	90th	95th	99th
Pregnant	65	14	9	22	33	43	47
Lactating	33	26	20	41	54	55	57
Non-pregnant, non-lactating, aged 15-44 yrs	2028	15	12	21	32	38	68

- From Part IV Table A3 of U.S. EPA (2004)
- Data used were from CSFII 1994-1996, 1998

8.5.3 High Activity Levels / Hot Climates

In the Exposure Factors handbook (1997), the U.S. EPA also addresses the issue of water consumption for those individuals performing strenuous activities under various environmental conditions, including desert climates (U.S. EPA, 1997). Data on these intake rates are very limited, and since the populations in the available studies are not considered representative of the general U.S. population, U.S. EPA did not use these data as the basis of their recommendations. Instead, they used the data from two studies to provide bounding intake values for those individuals engaged in strenuous activities in hot climates (McNall and Schlegel, 1968; U.S. Army, 1983).

McNall and Schlegel (1968) measured water intake of adult males working under varying degrees of physical activity, and varying temperatures. The results of this study indicate that hourly intake can range from 0.21 to 0.65 L/hour depending on the temperature and activity level.

U.S. EPA notes that these intake rates cannot be multiplied by 24 hours/day to convert to daily intake rates because they are only representative of water intakes during the 8-hour study periods of the test protocol. Intakes of the subjects for the rest of the day are not known.

The U.S. Army has developed water consumption planning factors to enable them to transport an adequate amount of water to soldiers in the field under various conditions (U.S. Army, 1983 and 1999). According to their estimates, intake among physically active individuals can range from 6 L/day in temperate climates to 11 L/day in hot climates. The Army's water consumption planning factors are based on military operations and may over-estimate civilian water consumption.

8.6 References

Abbott Laboratories. Ross Laboratories Mothers Survey. Ross Products Division, Columbus, OH, 1999-2001.

American Academy of Pediatrics (AAP) (1997). Breastfeeding and the use of human milk. Pediatrics; 100: 1035-1039.

Barraj L, Scrafford C, Lantz J, Daniels D, Mihlan G. (2008). Within-day drinking water consumption patterns: Results from a drinking water consumption survey. J Expo Sci Environ Epidemiol 19(4):382-95.

Cantor KP, Hoover R, Hartge P, et al. (1987). Bladder cancer, drinking water source, and tap water consumption: A case-control study. J Natl Cancer Inst 79:1269-1279.

CEHD (1981). *Tapwater Consumption in Canada.* Health Protection Branch, Environmental Health Directorate, Department of the Minister of National Health and Welfare, Ottawa, Ontario, Canada.

Ershow AG and Cantor KP (1989). *Total Water and Tapwater Intake in the United States: Population-Based Estimates of Quantities and Sources*. Life Sciences Research Office, Federation of American Societies for Experimental Biology, Bethesda, MD.

Ershow AG, Brown LM and Cantor KP (1991). Intake of tap water and total water by pregnant and lactating women. Amer J Pub Hlth 81: 328-334.

Gillies ME and Paulin HV (1983). Variability in mineral intakes from drinking water: A possible explanation for the controversy over the relationship of water quality to cardiovascular disease. Intl J Epidemiol 12:45-50.

Kahn HD, Stralka K (2009). Estimated daily average per capita water ingestion by child and adult age categories based on USDA's 1994–1996 and 1998 continuing survey of food intakes by individuals. J Expos Sci Environ Epidemiol 19(4)396-404.

Levallois P, Gingras S, Caron M,Phaneuf D. (2008) Drinking water intake by infants living in rural Quebec (Canada). Sci Total Environ 397(1-3):82-5.

Levy SM, Kohout FJ, Guha-Chowdhury N, Kiritsyl MC, Heilman JR, Wefel JS (1995). Infants' fluoride intake from drinking water alone, and from water added to formula, beverages, and food. J Dent Res 74(7):1399-1407.

McNall PE and Schlegel JC (1968). Practical thermal environmental limits for young adult males working in hot, humid environments. American Society of Heating, Refrigeration and Air-Conditioning Engineers (ASHRAE) Transactions, 74:225-235.

Michaud DS, Kogevinas M, Cantor KP, Villanueva CM, Garcia-Closas M, Rothman N (2007). Total fluid and water consumption and the joint effect of exposure to disinfection by-products on risk of bladder cancer. Environ Health Perspect 115:1569-1572.

NAS (1977). *Drinking Water and Health*. Volume 1. National Academy of Sciences-National Research Council, Washington, DC.

OEHHA (2009) Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for earlylife stage exposures. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots/tsd052909.html

Pennington JAT (1983). Revision of the total diet study food list and diets. J Am Diet Assoc 82:166-173.

Roseberry AM and Burmaster DE (1992). Lognormal distributions for water intake by children and adults. Risk Anal 12:99-104.

Steinmaus C, Caraway CA, Arcus A, Howd R, Fan AM. Drinking Water Consumption Default Values: Incorporating Updated Data on Susceptible Subpopulations. Council of State and Territorial Epidemiologists Annual Conference June 4-8, 2006 Anaheim, CA poster.

U.S. Army (1983, as cited in U.S. EPA 1989a). Water Consumption Planning Factors Study. Directorate of Combat Developments, United States Army Quartermaster School, Fort Lee, Virginia.

U.S. Army (1999). Study Report, Water Consumption Planning Factors. Prepared by Directorate of Combat Developments (Quartermaster), U.S. Army Combined Arms Support Command, Ft. Lee, Virginia, June 15, 1999. Available on the internet at: www.cascom.army.mil/Quartermaster/Water/Planning_Factors/

U.S. EPA (1984). An Estimation of the Daily Food Intake Based on Data from the 1977-1978 USDA Nationwide Food Consumption Survey. Office of Radiation Programs, United States Environmental Protection Agency, EPA/520/1-84/021, Washington, DC.

U.S. EPA (1989a). *Exposure Factors Handbook*. Office of Health and Environmental Assessment, United States Environmental Protection Agency, EPA/600/8-89/043, Washington, DC

U.S. EPA (1989b). Risk Assessment Guidance for Superfund Volume 1 Human Health Evaluation Manual (Part A). Office of Emergency and Remedial Response, United States Environmental Protection Agency, EPA/540/1-89/002, Washington, DC.

U. S. EPA (1991). U.S. Environmental Protection Agency. OSWER Directive No. 9285.6-03 Office of Solid Waste and Emergency Response. March 25, 1991.

U.S. EPA (1997). *Exposure Factors Handbook*. Office of Research and Development, National Center for Environmental Assessment, United States Environmental Protection Agency, EPA/600/P95/002Fa August 1997, Washington DC.

U.S.EPA (2011). Exposure Factors Handbook (Final). U.S. Environmental Protection Agency, Washington, D.C.. EPA/600/R-09/052F.

U.S. EPA (2004). Estimated Per Capita Water Ingestion and Body Weight in the United States–An Update Based on Data Collected by the United States Department of Agriculture's 1994–1996 and 1998 Continuing Survey of Food Intakes by Individuals. Office of Water, Office of Science and Technology, United States Environmental Protection Agency, EPA/822/R/00/001 October 2004, Washington DC.

U.S. EPA (1997b). Guiding principles for Monte Carlo analysis. Risk Assessment Forum. Office of Research and Development, U.S. Environmental Protection Agency EPA/630/R-97/001.

U.S. EPA (2000a). Estimated per capita water ingestion in the United States. Office of Science and Technology, Office of Water, U.S. Environmental Protection Agency, Washington, DC.

U.S. EPA (2000b). Options for development of parametric probability distributions for exposure factors, U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC, EPA/600/R-00/058.

U.S. EPA (2008). Child-Specific Exposure Factors Handbook EPA/600/R-06/096F | September 2008 www.epa.gov/ncea

9 Fish Consumption

9.1 Introduction

The “Hot Spots” (AB-2588) risk assessment process addresses contamination of bodies of water near facilities emitting air pollutants. The consumption of fish from contaminated bodies of water can be a significant exposure pathway for persistent bioaccumulative organic compounds and some heavy metals. Sport fishing in freshwater lakes and ponds is the primary concern for this exposure pathway, as deposited contaminants have the greatest potential to concentrate in these types of water bodies. Although regional air contaminants depositing into the ocean, bays and estuaries are a significant problem, the risks predicted from a single source are expected to be relatively insignificant due to tidal flows and dilution. Possible exceptions could be estuaries, salt marshes or sloughs with very low tidal flow that lead to accumulation of pollutants from nearby emission sources.

Commercial store-bought fish generally come from a number of sources. Consequently, the health risks of concern are due to noncommercial, or sport, fishing. The sport fish consumption rate is a critical variate in the assessment of potential health risks to individuals consuming fish from waters impacted by facility emissions. Other synonymous terms used for sport fishing include “self-caught fish” and “wild-caught fish”. The term “angler” or “sport fisher” refers to persons who catch sport fish or shellfish. These groups may include subsistence fishers.

Estimates of sport fish consumption by fishers tend to be greater than estimates of commercial fish consumption rates for the general population (Puffer et al., 1982a; Puffer et al., 1982b; SCCWRP and MBC, 1994; OEHHA, 2001). The higher intake rate of sport fish consumption by fishers creates a sensitive subpopulation relative to the general population when a facility’s emissions impact a fishable body of water. For this reason, consumption rates that apply to the general sport fisher population, rather than per capita estimates of fish consumption, are used here to characterize fish consumption by the subpopulation that is at risk from consuming fish contaminated by air emissions from stationary sources.

Sport fish consumption rates may also vary by geographic location and for specific subpopulations. The U.S. EPA recommends using data on local consumption patterns and population characteristics whenever possible (U.S. EPA, 2000). For instance, subsistence fishers, as well as certain cultural groups, can have particularly high consumption rates relative to the general population (Harnly et al., 1997; SFEI, 2000; U.S. EPA, 2000). Use of national averages can seriously underestimate risks to these subpopulations.

Because freshwater bodies such as lakes and ponds have the greatest potential for concentrating deposited contaminants, the ideal fish consumption study to use for the Hot Spots program would be a study of California freshwater sport fish consumption. Unfortunately, there are no such studies available. However, comprehensive studies

have been conducted in California surveying consumption rates of saltwater or Central Valley Delta fishers (Puffer et al., 1982a; Puffer et al., 1982b; SCCWRP and MBC, 1994; Wong, 1997; SFEI, 2000; Shilling et al., 2010). One strength of the California marine surveys is that the survey population is ethnically diverse, which may better approximate the consumption patterns for the California population, relative to studies that surveyed more homogeneous populations.

The application of the results of an ideal single fish consumption study conducted elsewhere to an impacted water body will always be uncertain because factors such as individual water body productivity, size, and local angler water body preferences will influence fish consumption. Conducting a site-specific sport fish consumption survey, in most cases, would not be a cost-effective alternative to use of the values presented in this chapter. Thus, OEHHA encourages the description of factors in the risk assessment which might significantly reduce or increase the estimated quantity of sport fish consumed for the consideration of the risk managers.

9.2 Recommendations for Angler-Caught Fish Consumption Rates

Recommended point estimates for angler-caught fish consumption rates are shown in Table 9.1. The fish consumption estimates are used to calculate individual cancer risk and noncancer chronic risk to those who eat sport (angler-caught) fish. Under the “Hot Spots” program, these consumption estimates apply principally to the general freshwater fishing population and encompass consumption of all sport fish species at a given location.

The risks should be presented using the high-end estimate in Tier 1 risk assessments, if the fish ingestion pathway is a dominant pathway. As noted in Chapter 1, dominant pathways are defined as the two pathways contributing the most to cancer risk when high-end estimates of intake are used in the risk calculation. The risks estimated from the average value would be used where fish ingestion is not a dominant pathway and may also be presented for comparison in assessments where fish ingestion is a dominant pathway.

However, if high fish-consuming groups including ethnic groups and/or subsistence fishers are known to be present, OEHHA recommends that the intake rate at the 95th percentile be used to reflect the upper bound estimate of consumption rates for these subpopulations, and when aiming to protect the target population as a whole.

Table 9.1 Point Estimate Values for Sport Fish Consumption by Age Group

	Third Trimester	0 <2 Years	2<9 Years	2<16 Years	16<30 Years	16-70 Years
	Consumption rates in g/day					
Average	-	2.1	7.9	13.3	28.8	28.8
High End[a]	-	6.6	25.4	42.9	92.4	92.4
	Consumption rates normalized by body weight, in g/kg-day					
Average	0.38	0.18	0.36	0.36	0.38	0.36
High-End[a]	1.22	0.58	1.16	1.16	1.22	1.16

[a] High end fish consumption values are the 95th percentiles. OEHHA recommends using the g/kg-day values.

Distributional analysis rather than single point estimates of fish consumption rates may be used to describe exposure within a population. Using a stochastic analysis will allow a more complete characterization of the variability in consumption in a population.

OEHHA recommends that the avidity-bias corrected distribution derived from the San Francisco Bay study (see Section 9.5) be used in Tier 3 and 4 risk assessments. The data in Table 9.2, expressed in g/kg-d, were obtained by dividing the adult fish consumption lognormal distribution data (in g/day) in Table 9.6 by the mean body weight of 80.0 kg derived in Section 10 for adults age 16-70 years. This was necessary because individual body weights were not collected in the fish consumption surveys.

Table 9.2. Empirical Distribution for Avidity Bias Adjusted Sport-Caught Fish Consumption Expressed in g/kg-day

Mean	Percentiles									
	10th	20th	30th	40th	50th	60th	70th	80th	90th	95th
Third trimester, 2<9, 2<16, 16<30 and 16-70-year age groups										
0.36	0.06	0.09	0.12	0.16	0.21	0.28	0.36	0.50	0.79	1.16
0<2-year age group										
0.18	0.03	0.05	0.06	0.08	0.11	0.14	0.18	0.25	0.40	0.58

As discussed below, there were no data available to clearly ascertain sport fish consumption rates of children. Estimates from studies for children in households of anglers indicate both potentially higher consumption rates than the anglers themselves (Mayfield et al., 2007; Shilling et al., 2010), and lower consumption rates than the anglers themselves (US EPA, 2002). We therefore assumed that sport fish consumption rate for adults 16-70 years of age would be proportional to body weight for the child age groupings of 2<9 and 2<16-year olds. Multiplying the adult consumption rate point estimates in g/kg-day by the time-weighted average body weight of 21.9 kg from Section 10 for the 2<9 year olds yields a mean and high-end fish consumption rate of 7.9 and 25.4 g/day, respectively. Performing the same calculation for the 2<16 age group with an average body weight of 37.0 kg results in a mean and high-end fish consumption rate of 13.3 and 42.9 g/day, respectively.

For the 0<2 age group, no fish consumption is expected in the first year, and fish consumption during the second year was assumed proportional on a gram per kg body weight basis to that of older children and adults. Thus, the fish consumption rate is based on the mean body weight of children during the second year (11.4 kg for 1<2 year age group) and divided by two to represent the first 2 years after birth. The resulting mean and high-end fish consumption rates are 2.1 and 6.6 g/day, respectively (See Table 9.1 above).

Fetal exposure via the mother's consumption of fish during the third trimester is represented in g/kg-day only; no estimate was determined based on g/day. To account for the third trimester of fetal exposure we assumed sport fish consumption for both the fetus and the mother will be the same during this three-month period using the sport fish consumption rate of 0.38 g/kg-day for adults age 16<30 years.

9.3 List of "Hot Spots" Chemicals for Which Evaluation of the Fish Pathway Is Recommended

The subset of organic and metal compounds that exhibit multipathway exposure are semi-volatile or nonvolatile, and are therefore partially or wholly in the solid or liquid phase and subject to deposition on water bodies. Fate and transport of the deposited chemical are estimated in order to assess the impact on fish that humans may catch and consume. The basis for the selection of these compounds as Hot Spots multipathway substances can be found in Appendix E. If the chemical has a long half-life and accumulates in fish, the multipathway analysis becomes more important. Below are the compounds on the Air Toxics "Hot Spots" list for which evaluation of the fish pathway is recommended:

Organic Compounds
Diethylhexylphthalate
Hexachlorobenzene
Hexachlorocyclohexanes
Pentachlorophenol
Polychlorinated biphenyls
Polychlorinated dibenzo-p-dioxins and dibenzofurans
Polycyclic aromatic hydrocarbons

Inorganic Metals and Semi-Metals
Arsenic & arsenic compounds
Beryllium & beryllium compounds
Cadmium & cadmium compounds
Soluble compounds of hexavalent chromium
Lead & inorganic lead compounds
Inorganic mercury
Nickel & nickel compounds
Selenium & selenium compounds

9.4 Algorithm for Dose via Fish Ingestion

In the Air Toxics "Hot Spots" program, the concentration of a chemical in fish, Cf, is a product of the modeled concentration in water, Cw, and the bioaccumulation factor (BAF) for the chemical of concern.

$$Cf = Cw \times BAF \qquad \textbf{(Eq. 9-1)}$$

where: Cf = concentration in fish (μg/kg)

Cw = concentration in water (μg/kg)

BAF = chemical-specific bioaccumulation factor for fish

Bioaccumulation refers to the uptake and retention of a chemical by an aquatic organism such as fish from all surrounding media (e.g., water, food, sediment). A BAF is the ratio of the chemical concentration in the fish tissue to the concentration in water, taking into account uptake through contaminated food, sediment and water. There are a number of factors that can affect the BAF of a chemical in fish. Appendix I presents the derivation of the BAF for each chemical, and provides a brief discussion of the various factors influencing the BAF in fish.

Airborne contaminants can deposit directly into a body of water or be carried there by runoff. As discussed in chapter 8, the Air Toxics "Hot Spots" algorithm only considers direct deposition onto the surface of the water body. OEHHA has not currently endorsed a modeling approach for runoff. If runoff into a water body is thought to significantly impact risk from a particular facility, the risk assessor should include discussion of this problem. The concentration in the water in the model below is a function of what is directly deposited into the body of water. This is calculated as follows:

$$Cw = Dep\ (SA)\ (365) / (WV)\ (VC) \qquad \textbf{(Eq. 9-2a)}$$

and

$$Dep = GLC \times \text{dep-rate} \times 86{,}400 \qquad \textbf{(Eq. 9.2b)}$$

where: Cw = concentration in water due to direct deposition (μg/kg)

Dep = amount deposited/day (μg/m2/day) = GLC x dep-rate x 86,400

GLC = modeled ground level concentration ($\mu g/m^3$)

dep-rate = vertical rate of deposition (m/sec)

86,400 = seconds/day

SA = surface area of water body (m^2)

365 = days per year

WV = water volume (L = kg)

VC = number of volume changes per year

The deposition rate is assumed to be 0.02 m/sec for a controlled source and 0.05 m/sec for an uncontrolled source (see Chapter 2). The terms SA, WV, and VC are site-specific factors; values for these terms need to be ascertained by the risk assessor.

Calculating dose of contaminant via fish ingestion requires an estimate of the fish concentration and the amount of fish an individual consumes. The following equation can be used to calculate dose via ingestion of contaminated fish:

$$DOSEfish = (Cf \times Ifish \times GI \times Fsf \times EF \times (1 \times 10^{-6}) \quad \textbf{(Eq. 9-3)}$$

where:
- DOSEfish = dose of contaminant via ingestion of fish (mg/kg BW-day)
- Cf = concentration in fish (μg/kg)
- Ifish = sport fish ingestion rate (g/kg BW-day)
- GI = gastrointestinal absorption fraction, unitless
- Fsf = fraction of sport fish caught at contaminated site, unitless
- EF = exposure frequency (days/365 days)
- 1×10^{-6} = conversion factor (μg/mg) (kg/gm)

The value of Cf is calculated using equations 9-1 and 9-2. The default gastrointestinal absorption fraction is 1. There are currently no data to support a value different from 1 for any of the chemicals that are evaluated for this pathway. The factor, Fsf, is a site-specific factor; the risk assessor must evaluate site-specific data to ascertain what fraction of the sport fish consumed by an individual comes from the impacted body of water. If such data are unobtainable, then Fsf should be set to 1. We provide both point estimates and a distribution of sport fish consumption rates normalized to body weight in this chapter. The exposure frequency (EF) is set at 350 days per year (i.e., per 365 days) to allow for a two week period of time away from home (US EPA (1991).

For cancer risk, the risk is calculated for each age group using the appropriate age sensitivity factors (ASFs) and the chemical-specific cancer potency factor (CPF) expressed in units of $(mg/kg\text{-}day)^{-1}$.

$$RISKfish = DOSEfish * CPF * ASF * ED/AT \quad \textbf{(Eq. 9-4)}$$

RISK is the predicted risk of cancer (unitless) over a lifetime as a result of the exposure, and is usually expressed as chances per million persons exposed (e.g., 5×10^{-6} would be 5 chances per million persons exposed).

The dose-response phase of a cancer risk assessment aims to characterize the relationship between an applied dose of a carcinogen and the risk of tumor appearance in a human. This is usually expressed as a cancer potency factor, or CPF, in the above equation. The CPF is the slope of the extrapolated dose-response curve and is expressed as units of inverse dose $(mg/kg\text{-}d)^{-1}$.

Exposure duration (ED) is the number of years within the age groupings. In order to

accommodate the use of the ASFs (OEHHA, 2009), the exposure for each age grouping must be separately calculated. Thus, the ED is different for each age grouping. The ASF, as shown below, is 10 for the third trimester and infants 0<2 years of age, is 3 for children age 2<16 years of age, and is 1 for adults 16 to 70 years of age.

ED =	exposure duration (yrs):	
	0.25 yrs for third trimester	(ASF = 10)
	2 yrs for 0<2 age group	(ASF = 10)
	7 yrs for 2<9 age group	(ASF = 3)
	14 yrs for 2<16 age group	(ASF = 3)
	14 yrs for 16<30 age group	(ASF = 1)
	54 yrs for 16-70 age group	(ASF = 1)

AT, the averaging time for lifetime cancer risks, is 70 years in all cases. To determine lifetime cancer risks, the risks are then summed across the age groups:

$$RISKfish_{(lifetime)} = RISKfish_{(3rdtri)} + RISKfish_{(0<2\ yr)} + RISKfish_{(2<16\ yr)} + RISKfish_{(16\text{-}70yr)} \quad \textbf{(Eq. 9-5)}$$

As explained in Chapter 1, we also need to accommodate cancer risk estimates for the average (9 years) and high-end (30 years) length of time at a single residence, as well as the traditional 70 year lifetime cancer risk estimate. For example, assessing risk in a 9 year residential exposure scenario assumes exposure during the most sensitive period, from the third trimester to 9 years of age and would be presented as such:

$$RISKfish_{(9\text{-}yr\ residency)} = RISKfish_{(3rdtri)} + RISKfish_{(0<2\ yr)} + RISKfish_{(2<9\ yr)} \quad \textbf{(Eq. 9-6)}$$

For the 30-year residential exposure scenario, the risk for the 2<16 and 16<30 age groups would be added to the risks from exposure during the third trimester and from ages 0<2 yr. For 70 year residency risk, Eq 9-5 would apply.

The fetus can be exposed via the mother's consumption of fish during the third trimester of pregnancy. Fetal exposure during the third trimester via fish consumption by the mother is taken into account in the final determination of the point estimate values presented in Section 9.2. For the 0<2 yr age group, no fish consumption by the infant is expected from birth to one year of age.

9.5 Studies Evaluated for Sport Fish Consumption Rate

In order to determine the dose of a contaminant via ingestion of fish, reasonable point estimates and distributions for the rate of California sport fish ingestion are required. The most comprehensive studies of noncommercial fish consumption in California are the Santa Monica Bay Seafood Consumption Study (SCCWRP and MBC, 1994) and the San Francisco Bay Seafood Consumption Study (SFEI, 2000). These studies were undertaken to describe the demographic characteristics of anglers that fish the Santa Monica Bay and San Francisco Bay, to assess their sport seafood consumption rates, and to identify ethnic subgroups that may have high rates of seafood consumption.

Other California fish consumption studies that provide estimates of fish consumption rates are also reviewed here. Since comprehensive freshwater fish consumption rate studies in California are lacking, the best freshwater fish studies performed elsewhere in the U.S. are also summarized. Studies that discussed consumption of sport fish by household members are also summarized. Household members may represent a more sensitive subgroup of people consuming contaminated sport fish brought home by anglers. Sensitive household members include children and pregnant and lactating women.

9.5.1 Marine and Delta Fish Consumption Studies

9.5.1.1 1998-1999 San Francisco Bay Seafood Consumption Study

Between July 1998 and June 1999, the California Department of Health Services conducted over 150 fishing site visits and approached over 1700 San Francisco Bay (SF Bay) anglers (SFEI, 2000). The sites chosen for interviews included public piers and adjacent beaches or banks, public boat launches, and party boats. Anglers were asked how many times they ate Bay fish in the four weeks prior to being interviewed - a time period within which anglers were assumed to have reasonably accurate recall. Anglers were also asked the portion size of the meal compared to a plastic model of an eight-ounce fish fillet. The portion size question was asked only once and was used to calculate all fish consumption rates. Angler fish-consumption rates were determined by multiplying the two variables, meal frequency and portion size, and converted to grams per day (g/d). Consumption rates are described primarily for two populations, consumers and recent consumers. Consumers are anglers who reported eating Bay fish. Recent consumers are a subset of consumers who reported consuming Bay fish in the last four weeks.

Of 1738 eligible (i.e., not previously interviewed) anglers interviewed, 501 individuals identified as recent consumers provided adequate information for deriving a consumption rate. The researchers had determined a sample size of 500 recent consumers would be needed to derive a reasonably precise mean consumption rate (i.e., 95% confidence interval of ± 10% around the geometric mean consumption rate and 95% confidence interval of ± 15% around the upper percentiles). The mean and 95^{th} percentile for fish consumption rate among recent consumers based on 4-week recall was 28 and 108 g/d, respectively.

The SF Bay report also included a distribution of consumption rates for recent consumers adjusted for avidity bias (See section 9.8.2.1 for discussion on avidity bias). In on-site surveys such as the SF Bay study, avid anglers are over-represented in the sample and infrequent anglers are under-represented, resulting in avidity bias. This bias occurs because an individual who fishes frequently has a greater chance of being interviewed than a person who fishes infrequently. Thus the distribution will over-represent the consumption of frequent fishers. Further information about avidity bias is discussed below. The mean and 95^{th} percentile for the avidity adjusted fish consumption rate among recent consumers based on 4-week recall was 23 and 80 g/d, respectively.

Although less reliable than the four week recall, consumers (n=1019) were asked to report the number of times they ate Bay fish in the past 12 months. The unadjusted mean and 95th percentile for fish consumption rate based on 12-month recall was 11 and 44 g/d, respectively. Consumption rates for the 12-month period prior to the interview could not be adjusted for avidity bias due to insufficient fishing frequency data over the same time period.

Due to historic mercury contamination in the region, the SF Bay report also surveyed angler households for pregnant or lactating women. The developing fetus and infants are particularly sensitive to mercury contamination. The SF Bay report found that only 2% of anglers reported that pregnant or lactating women in their household ate SF Bay sport-caught fish. However, 46% of anglers reported that women of childbearing age (18-45 years) in their household ate SF Bay sport-caught fish, and 13% reported that children younger than six years of age ate SF Bay sport-caught fish.

9.5.1.2 1991-1992 Santa Monica Bay Seafood Consumption Study

For the Santa Monica Bay study, surveys were conducted at 29 sites on 99 days, from September 1991 to August 1992 (SCCWRP and MBC, 1994; Allen et al., 1996). Fishers on piers and jetties, private boats, party boats, and beaches were interviewed using a questionnaire. The fish consumption estimates applied only to consumption of Santa Monica Bay sport fish, and did not include consumption of fish from all sport and commercial sources. Anglers were questioned about consumption of eight commonly consumed species of fish as well as about fish they had in hand. Anglers were also asked to estimate how much fish he/she consumed per meal, compared to a wood model representing a 150 gram (0.33 pound) portion of a fish fillet. Similar to the SF Bay study, fishers were asked the number of times they had consumed sport fish in the 4 weeks prior to the interview, but unlike the SF Bay study, the frequency of fish consumption was increased by one meal to account for consumption of catch present at the time of the interview. Fishers who had eaten any of the 8 species in the survey in the 4 weeks prior to the interview were included in consumption rate estimates. Of the 1,243 fishers interviewed, 554 provided information that could be used for calculating consumption rates. Average daily sport fish consumption rates (g/day) were calculated by multiplying the fisher's estimate of the typical meal size relative to the model, by the frequency of consumption in the four weeks prior to the interview, divided by 28 days. The mean and 95th percentile consumption rates for the overall surveyed population were 49.6 and 161 g/d, respectively.

OEHHA utilized a basic inverse-weighting scheme to adjust the fish consumption rate data for avidity bias, resulting in a mean of 29.4 g/d (OEHHA, 2000). Additionally, the analysis adjusted for four separate factors producing potential bias in the sampling procedure (i.e., number of times fished, frequency of site selection, proportion of successful interviews, and week days versus weekend days sampled). The four-factor corrected mean was 30.5 g/d, and differed from the avidity-corrected mean by only 3%. The four-factor adjusted high end (95th percentile) fish consumption rate estimate was 85.2 g/d.

9.5.1.3 1980 Los Angeles Metropolitan Area Survey

In 1980, an intercept survey was conducted in the Los Angeles metropolitan area (including Santa Monica Bay) to assess noncommercial fish and shellfish consumption rates by local fishers, and to identify subgroups that have significantly larger consumption rates (Puffer et al., 1982a; Puffer et al., 1982b). The intercept survey method surveys fishers at a fishing site or sites about fish consumption, catch or other questions of interest. During the one-year study period, a total of 1,059 fishers were interviewed at 12 sites, including piers, jetties, and party boats. Average daily consumption rates were estimated based on the number of fish in the catch, the average weight of the fish in the catch, the edible portion of the species, the number of fish eaters in the family and the frequency of fishing per year. The fish consumption rate data were presented as a cumulative percentile distribution, with a median of 37 g/d and 90^{th} and 95^{th} percentiles of 225 and 339 g/d, respectively. Mean estimates of fish consumption were not presented.

While this study was quite extensive, there were several limitations. Consumption data were collected from over 1,000 individuals representing various ethnic groups in the survey population (i.e., Caucasian, Black, Mexican-American, and Oriental/Samoan), but only English speaking fishers were included in the study. The Santa Monica and SF Bay Seafood Consumption Studies interviewed a number of different ethnic groups in their native languages. In addition, the survey did not ask fishers for direct estimates of the amount of fish they consumed, correction for avidity bias was not performed, and no recall was included of sport fish consumption over a previous period of time.

Price et al. (1994) attempted to correct for avidity bias using the general assumption that sampling probability is proportional to the inverse of fishing frequency. The adjusted consumption rate distribution was considerably lower than that obtained by Puffer et al. studies; the median and 90^{th} percentile were estimated at 2.9 and 35 g/d, respectively. U.S. EPA (1997) notes that an avidity-correction assumption is not completely valid, as interviewers visited sites numerous times and anglers were not interviewed more than once. However, U.S. EPA (1997) does state that the estimates of Price et al. (1994) are probably better estimates of the fish consumption of the entire population that fishes the area than the non-adjusted survey results.

9.5.1.4 1988-1989 San Diego Bay Health Risk Study

The San Diego Department of Health Services conducted a survey of fishers fishing the San Diego Bay (SDCDHS, 1990) to identify the demographics of this fisher population and to characterize their noncommercial fish consumption patterns. The authors derived an overall bay-wide fishing population mean of 31.2 g/d. Only 59 fishers provided all of the necessary data for calculating individual noncommercial fish consumption rates and subsets of the 59 interviews were used to calculate species and ethnic-specific rates. Thus, there is more uncertainty about the fish consumption values because of the small number of subjects in the study population, particularly for the subsets for specific species and influence of ethnicity. In addition, the consumption

rate overestimates consumption in the general fishing population because the rate only includes fishers who were known to catch and consume fish year-round.

9.5.1.5 1993 San Francisco Bay Seafood Consumption and Information Project

In an earlier study of fish consumption habits of people fishing in San Francisco Bay, Wong (1997) conducted personal interviews with approximately 200 people fishing or crabbing from ten public piers during September to November 1996. A fish fillet model, representing 150 grams, was used to assist with estimating the amount of fish consumed per meal. Sixty-two respondents (29 percent) reported consumption of SF Bay fish in the 7-day period preceding the interview. A calculated median consumption rate of 32 g/d was determined for anglers that ate fish and/or shellfish from SF Bay. This study was not corrected for avidity bias.

9.5.1.6 2010 California Central Valley Delta Fish Consumption Study

A fish consumption survey was conducted in the California Central Valley Delta (including the Sacramento-San Joaquin Rivers Delta) where a high rate of subsistence fishing of potentially mercury-contaminated fish occurs (Shilling et al., 2010). This study reflects a region where both freshwater and anadromous fish are caught. Anglers were chosen for interviews as they were encountered along the riverbank by surveyors. Shore anglers (n=373) were interviewed during biweekly to monthly site visits between September 2005 and June 2008. Anyone reporting that they had been previously interviewed was not interviewed again. Fish consumption rates (g/d) were calculated for each individual based on 30-day recall of how much and how often individual types of fish were eaten. Fish fillet models were used representing 1.5, 4.5, and 7.5 oz cooked weights of fish fillet for the estimate of actual fish consumption rates.

The arithmetic mean and median consumption rates of locally caught fish were 27.4 and 19.7 g/day, respectively, for anglers. There were no statistically significant differences in consumption rates among age groups (18-34, 35-49, and >49 years of age). The 95th percentile rate of locally caught fish (126.6 g/d) was also determined to represent the majority of the fish consuming population. Note that this distribution is not normally distributed. The arithmetic mean and median consumption rates of locally caught fish for children (n=174, age unspecified) in households of anglers were 35.3 and 22.2 g/day, respectively. This study was not corrected for avidity bias.

In addition to interviewing shore anglers, interviews were conducted with selected members of the local South East Asian community in which it was known that a member of their extended family fished. The mean corresponding consumption rate for locally-caught fish from the community member survey was 55.2 g/day, which was higher than the corresponding rate for anglers in the field. Because this portion of the study was a community-based, rather than angler-based, survey of an ethnic group known for high consumption of locally-caught fish, it does not represent an overall California fish consumption rate.

9.5.2 Freshwater Fish Consumption Studies

9.5.2.1 Washington King County Lakes Study

A survey was conducted at three Washington state freshwater lakes from June 2002 to May 2003 (Mayfield et al., 2007). A total of 212 anglers were interviewed and asked to estimate their typical meal size from a visual aid (6, 8, 10, and 12 oz. fillets) and how often they had consumed fish they caught from the lakes in the previous month. Surveyors also asked the anglers to provide the same information for any children (i.e., <18 years) who also consumed their catch. Forty-six percent of anglers reported sharing their catch with children. The mean consumption rate was 10 and 7 g/d for anglers and their children, respectively. The 95th percentiles were 42 and 29 g/d for anglers and the children of anglers, respectively. Although many anglers reported consuming fish from King County Lakes, many had not consumed any fish in the previous month. Therefore, the median consumption rate was zero.

9.5.2.2 Michigan Freshwater Fish Consumption Studies

The University of Michigan conducted a stratified random mail survey of 2600 Michigan residents with annual fishing licenses during the period of January to June 1988 (West et al., 1989a; 1989b; 1989c). Those with one day fishing licenses from both in state and out of state were excluded thus eliminating some infrequent fishers. Fish meals included self-caught, market, restaurant, and gift fish. Fish consumption information was gathered from all members of the household for a 7-day recall period and included only those individuals who responded that they ate fish. However, all responses were tabulated in one of only three meal sizes, 5, 8, and 10 oz. Because the overall response rate was only 47.3 percent, the authors adjusted the population mean value of 18.3 g/d downward by 2.2 g/d to account for nonresponse bias, thus deriving a mean rate of 16.1 g/d. Derivation of the adjustment factor was based on a follow-up telephone survey of respondents and nonrespondents (West et al., 1989b). The researchers did not generate a distribution. The probability of being contacted in this study was not dependent on the frequency of fishing; therefore, the avidity bias found in intercept surveys is not present in the data. However, the authors noted that the sampled population may not have represented subsistence fishers because it was selected from licensed anglers only.

Murray and Burmaster (1994) used the raw data of West et al. studies to generate a distribution for total fish and self-caught fish among adults only, providing 12 empirical distributions for eight population subgroups. Fish consumption rate estimates were derived for persons who consumed self-caught fish during the recall period, resulting in a consumption rate based on a population that more frequently consumes fish. This study represents the most comprehensive analysis of freshwater sport fish consumption by anglers. Table 9.3 includes empirical distribution data for average daily fish consumption rate in the four adult subgroups that are most relevant for the California “Hot Spots” program. The Great Lakes fish population groups refer to anglers and family members who only ate self-caught fish from the Great Lakes. These groups may

be analogous to sport fishers in California that fish only from one or a few lakes in a defined area that are impacted by pollutants. The self-caught fish population groups refer to groups that caught and consumed fish caught anywhere in Michigan.

Table 9.3. Average Daily Fish Consumption Rates in g/day of Adults for Four Subgroups from Murray and Burmaster (1994)

Population group[a]	Distribution for fish consumption type	N	Fraction as % of adults[b]	Mean	SD	Percentile	
						50th	95th
Anglers/ate self-caught fish	Self-caught fish	191	0.08	45.0	23.7	32.7	98.0
All/ate self-caught fish	Self-caught fish	418	0.18	42.3	22.3	32.7	98.0
Anglers/ate Great Lakes fish	Great Lakes fish	89	0.04	40.9	19.9	32.7	81.6
All/ate Great Lakes fish	Great Lakes fish	188	0.08	38.5	19.0	32.7	81.6

[a] The first two rows refer only to fish consumption of self-caught fish for anglers only (anglers) or the anglers plus adult family members (all). The last two rows refer to fish consumption of only self-caught fish from the Great Lakes for anglers only (anglers) or the anglers plus adult family members (all).
[b] This column represents the percentage of general population (i.e., Michigan adults) that ate self-caught fish.

Murray and Burmaster (1994) found that a lognormal model fit the empirical data well and provided parametric compound distributions for use in Monte Carlo simulations.

9.5.2.3 1992-1993 Freshwater Fish Consumption by Alabama Anglers

A statewide survey was conducted from August 1992 to July 1993 to estimate daily fish consumption of freshwater fish harvested by anglers fishing from 29 locations throughout Alabama, including tailwater sites, reservoirs, and river drainages (Meredith and Malvestuto, 1996). A total of 1,586 anglers were interviewed at the completion of fishing activity. Of the total anglers interviewed, 1,303 anglers reported consumption of fish from the study areas. Serving size was estimated by equating the entire surface (palm side) of the flat open hand to a single 113 g (4 ounce) serving. To estimate fish consumption rates, anglers were asked to estimate the number of fish meals eaten in the past month consisting of fish caught at the study sites ("site meals") and those caught at all lakes and rivers in Alabama, including study sites ("all meals"). Only anglers indicating they consumed fish from the study sites were included in the analysis. The mean annual consumption rate estimated by this method was 30.3 g/d for site meals and 45.8 g/d for all meals.

9.5.3 Studies of Household Members Who Eat Sport-Caught Fish

Determining the consumption rate of sport fish eaten by others in angler households was beyond the scope of most studies summarized above. Some studies have shown that people who do not go fishing eat sport-caught fish given to them by friends and family, but possibly at reduced rates compared to the anglers themselves (Toth and Brown, 1997; Burger, 2000; Nadon et al., 2002; Mayfield et al., 2007). The household members of anglers are of particular interest because the anglers are predominantly male, and may bring home fish to household members that are at higher risk from consuming contaminated sport-caught fish (i.e., pregnant and lactating women, women who are of childbearing age, and children). Table 9.4 below presents the data from studies that did estimate consumption rates for household members that eat freshwater sport-caught fish.

Table 9.4. Freshwater Sport Fish Consumption Rates by Household Members of Anglers

Group	N	Consumption rate (g/day)	Consumption rate (g/kg-day)	Reference
Children		Arithmetic Means	Arithmetic Means	
1-5 yrs 6-10 yrs 11-20 yrs	121 151 249	5.63 7.94 7.27	0.369 0.276 0.123	U.S. EPA (2002) [a]
<18 yrs	81	7	0.19	Mayfield et al. (2007)
Not Specified [b]	174	35.3	0.95 [c]	Shilling et al. (2010)
Women				
All ages (<17-50+) <17 yrs Pregnant lactating	80 5 6 11	10.5 [d] 13.9 12.8 10.2	0.14 [d]	Silver et al. (2007)
18-49 yrs	217	33.0	0.44	Shilling et al. (2010)

[a] U.S. EPA values are based on treatment of data from West et al. (1989a)
[b] Child age range not specified, but can be inferred from the study to mean <18 years of age.
[c] Based on average body weight of 37.0 kg for children 2<16 yrs of age from Table 10.1
[d] Only geometric mean consumption rates were available

9.5.3.1 U.S. EPA analysis of West et al. (1989a) child fish consumption data subset

The U.S. EPA (2002) child fish consumption rates presented in Table 9.4 were obtained from the raw data by West et al. (1989a) to estimate freshwater recreational fish consumption rates for household members of anglers, based on the 7-day recall data. The household members were divided into three age groups, age 1-5, 6-10, and 11-20 years. The analysis was restricted to individuals who ate fish and who resided in households reporting some recreational fish consumption during the previous year.

Since the study was a stratified random mail survey of Michigan residents with annual fishing licenses, the study was not dependent on the frequency of fishing and did not need to be corrected for avidity bias.

Using an average adult body weight of 80.0 kg from Table 10.1of this document, the average adult angler consumption rate on a per kg body weight basis is 0.56 g/kg-day (45.0 g/day from Table 9.1 ÷ 80.0 kg). Comparing the child consumption rates in Table 9.4 to that of adult anglers who ate self-caught fish, this study suggests that the children in households of anglers eat less on a per body weight basis than the adult anglers.

9.5.3.2 Child sport fish consumption rate for the Washington King County Lakes Study

The Washington state freshwater fish consumption study recorded a mean consumption rate of 7 g/day for children (<18 years) of anglers interviewed (Mayfield et al., 2007). However, this study was not corrected for avidity bias, and included persons who did not consume sport fish during the 30-day recall period. Not accounting for avidity may overestimate consumption, while including anglers and their children who did not consume sport fish in the last month may underestimate the consumption rate of persons who frequently consume sport fish.

Using a mean body weight of 37.0 kg for children age 2<16 years, and 80.0 kg (age 18<75) for the mean body weight of adults, the sport fish consumption rates on a per kg body weight basis are 0.19 g/kg-day for children (7 g/d ÷ 37.0 kg) and 0.13 g/kg-day for adults (10 g/d ÷ 80.0 kg). The Washington state freshwater fish consumption data suggest that, if corrected for differences in body weight, children of anglers may consume as much fish, or more, on a per kg body weight basis as the anglers themselves. However, when compared to avidity-adjusted average adult angler consumption rates corrected for body weight from the S.F. Bay study (0.36 g/kg-day, see Table 9.1), the child consumption rate from the Washington study is only about half that of the adult S.F. Bay anglers.

9.5.3.3 California sport fish consumption survey among low-income women

The only study that investigated sport-caught fish consumption rates among a California population at increased risk (and presumably household members of an angler) was a survey of low-income women at a Special Supplemental Nutrition Program for Women, Infants and Children (WIC) clinic in the California Sacramento-San Joaquin Delta region (Silver et al., 2007). Of 500 eligible women participating in the survey, 80 (16%) reported eating sport fish in the last 30 days. These participants were asked about consumption frequency, portion size of cooked meals, and source of the fish. To assist with recall of portion size, fish fillet “portion models” were shown corresponding to 1.5, 3.0, 4.5, and 7.5 oz weight. The geometric mean sport fish consumption rate among this group was 10.5 g/d. Hmong and Cambodian women consumption rates showed a higher consumption trend but were not statistically significantly different.

Comparison of this geometric mean sport fish consumption rate for women in angler households with the geometric mean sport fish consumption rate among anglers in the

SF Bay and Santa Monica Bay studies suggests household members eat less sport-caught fish than the anglers themselves. The unadjusted geometric mean sport fish consumption rate for the SF Bay study and Santa Monica Bay study were 16.5 and 23.6 g/d, respectively. However, these consumption rates did not account for gender body weight differences and the predominance of male anglers in surveys (e.g., 92% of interviewed anglers in the SF Bay study were male), which would bring sport fish consumption rates among anglers and women household members closer together. Using mean body weight data by gender summarized in Table 10.2, the SF Bay and Santa Monica Bay mean consumption rates were divided by the average body weight of adult males (88.3 kg, age 20 yrs and above) and the WIC mean consumption rate divided by the average body weight for adult females (74.7 kg, age 20 yrs and above). Consumption rates on a per body weight basis yields values of 0.19, 0.27 and 0.14 g/kg-day for the SF Bay, Santa Monica Bay and WIC fish consumption studies, respectively.

9.5.3.4 California Central Valley Delta study of household fish consumption

The household consumption rates of women and children in the study by Shilling et al. (2010) are considerably higher compared to the household members in other studies. This may be due to the high number of subsistence fishers in this study, and that a majority of the anglers reported catching fish in order to feed their families. This study did not correct the consumption rate for avidity bias, so consumption rate may be overestimated.

Comparing the anglers with their family members, the consumption rates of children and women in households of anglers were not statistically significantly greater than the anglers themselves ($P < 0.05$, *t*-test). The study reported average consumption rates of 26.4, 33.0, and 35.1 g/day for male anglers, women in households of anglers, and children in households of anglers, respectively. However, when OEHHA divided the consumption rates by average body weights for men (88.3 kg), women (74.7 kg) and children (37 kg for 2 to <16 yrs), the fish consumption on a per body weight basis was 0.30, 0.44, and 0.95 g/kg-day, respectively. The results from this study suggest that household members of anglers, many of which are subsistence fisherman that fish mainly to feed their families, have a greater fish consumption rate than the anglers themselves.

9.5.3.5 Household sport fish consumption frequency surveys

A nationwide telephone survey of fish consumption patterns found that the presence of a fishing license in the home was a significant predictor of sport-caught fish ingestion by family members, including children and their mothers (Imm et al., 2007). Families with a fishing license in the home were more likely to eat sport-caught fish than families without a fishing license in the home. Forty-seven percent of children (2-17 years of age) who lived with a licensed angler ate sport-caught fish, with an average of 16 sport-caught fish meals (median = 8 meals; maximum = 240 meals) per year. A nationwide survey of 3015 women of childbearing age (ages 18-45) reported that 29% of participants had consumed sport fish in the previous 12 months (Anderson et al., 2004). Among those reporting sport fish consumption, the median and mean number of sport-caught fish meals for the past 12 months were 6 and 16, respectively. Neither study collected data on portion sizes of fish meals to estimate consumption rate.

9.6 Comparison of Marine Fish Consumption Rates among California Studies

Fish consumption rates for four California fish consumption studies, the SF Bay study, the Santa Monica Bay study, the Save the Bay Study (Wong, 1997), and the Central Valley Delta study (Shilling et al., 2010) are shown in Table 9.5 for comparison. The data from the SF Bay and Santa Monica Bay studies are presented both adjusted and unadjusted for avidity bias as discussed under section 9.8.2.1. Differences among the consumption rates could be explained by the different study methodologies used by the studies.

For example, the unadjusted geometric mean consumption rate from the Santa Monica Bay study is about 50 percent higher than the unadjusted rate derived from the SF Bay study, and the difference was found to be statistically significant. In the Santa Monica study, the frequency of consumption was increased by one to account for consumption of any fish in hand at the time of the interview. Fish in hand at the time of interview was not included in the SF Bay consumption rate estimates. This factor was thought to explain the higher consumption rates of the Santa Monica Bay study (SFEI, 2000). Another difference between the two studies was that the Santa Monica Bay study used a 5.3 ounce (150 g) portion model while the SF Bay study used an 8 ounce (227 g) portion model. The model size appears to have influenced the responses in both studies. Whether the different model sizes would widen or narrow the consumption rate difference between the two studies is not known.

In the Save the Bay study, the median consumption rate (32 g/d) was considerably higher than the unadjusted consumption rates of the other two California studies. However, only 7-day recall of fish consumption was surveyed among interviewed anglers. This short recall period creates an even smaller subset of all anglers compared to the 4-week recall used in the California studies, and also selectively includes anglers with the highest consumption rates.

Other factors unrelated to methodologies that may contribute to consumption rate differences among studies include differences in climate, fishery production, year of

study, and demographic characteristics. As noted in Section 9.5.3.4, the California Central Valley Delta study by Shilling et al. (2010) contained a high number of subsistence anglers that reported catching fish in order to feed their families. This study also did not correct the consumption rate for avidity bias. Even so, consumption rates among the Central Valley Delta anglers are similar to avidity-adjusted rates in Table 9.3. This study suggests that a greater proportion of this population of subsistence anglers gives the fish they catch to their families, and this may account for the high consumption rate of household family members shown in Table 9.4.

Table 9.5 Comparison of Consumption Rates (in g/day) for the San Francisco Bay Seafood Consumption Study, Santa Monica Bay Study, Save the Bay Study and the Central Valley Delta Study[a]

	Adjusted SF Bay Study[b]	Adjusted Santa Monica Study[c]	Unadjusted SF Bay Study[b]	Unadjusted Santa Monica Study[c]	Save the Bay Study[d]	Central Valley Study[e]
Respondents	n=1152	[f]	n=1331	n=1244	n=222	[f]
Population used to derive consumption rate (% of respondents)	n=465 (40%)	[f]	n=501 (38%)	n=555 (45%)	n=62 (27%)	n=373 ([f])
	4-week recall	4-week recall	4-week recall	4-week recall	7-day recall	4-week recall
Mean (Standard Deviation)	23.0 (32.1)	30.5 (45)	28.0 (39.5)	49.6 (111.1)	[f]	27.4 ([f])
Geometric Mean	14.0	[f]	16.5	23.6	[f]	[f]
50th Percentile	16.0	15.0	16.0	21.4	32	19.7
90th Percentile	48.0	62.4	56.0	107.1	[f]	[f]
95th Percentile	80.0	85.2	108.0	161	[f]	126.6

[a] Table modified from SFEI (2000)
[b] SFEI, 2000; [c] Allen et al. (1996); [d] Wong, 1997; [e] Shilling et al. (2010)
[f] Not reported

9.7 Comparison of Freshwater and Marine Fish Consumption Rate Studies

Although the California fish consumption rate studies are derived from a population fishing from marine water bodies, a similar distribution of consumption rates also occurred from data obtained of populations fishing from freshwater bodies. For example, Murray and Burmaster (1994) calculated mean rates for non-avidity-biased consumption of Michigan sport-caught freshwater fish by anglers as 45.0 g/d for self-caught fish in general, and 40.9 g/d for anglers consuming fish from the Great Lakes, in particular. Meredith and Malvestuto (1996) reported an avidity-biased consumption rate of 30.3 g/d for specific study sites in Alabama, and 45.8 g/d for all sport-caught meals

caught in the state. These mean values fall between the adjusted mean for the SF Bay study (23.0 g/d) and the unadjusted mean for the Santa Monica Bay study (49.6 g/d) shown in Table 9.5. These saltwater and freshwater studies were comparable in many study parameters and in analytical evaluation and, thus, can be reasonably used to support angler-caught freshwater fish consumption estimates in California.

The Washington King County Lakes study (Mayfield et al., 2007) exhibited a lower mean angler consumption rate of 10 g/day for freshwater fish compared to the Alabama and Michigan studies. The lower consumption rate in the Washington study is likely due to differences in methodology. Anglers that had not eaten sport fish in the previous month were included in the consumption rate analysis, whereas the Alabama and Michigan studies excluded anglers who had not eaten sport fish in the previous month. Thus, the Alabama and Michigan studies target the angler population that are the most frequent consumers of sport fish.

A more analogous comparison to the Washington King County Lakes study might be made with the unadjusted mean fish consumption rate based on 12-month recall in the SF Bay study. A lower mean consumption rate of 11.0 g/d was recorded for this group, which includes frequent (i.e., consumed sport fish in the last 4 weeks) and infrequent (i.e., consumed sport fish in the previous year, but not in the previous 4 weeks) anglers. The Washington King County Lakes mean consumption rate of 10 g/d is similar, using the assumption that this consumption rate includes both frequent and infrequent anglers that probably consumed sport fish in the previous year.

9.8 Determination of Fish Consumption Distribution

9.8.1 Choice of Study

The data from the San Francisco Bay Seafood Consumption Study (SFEI, 2000) were determined to be the most comprehensive and appropriate report for our estimation of average daily sport fish consumption in California. The SF Bay study was chosen over the other major California fish consumption studies in Table 9.5 because it represents the most recent well-conducted study of a California population. The SF Bay study applies to salt water sport-caught fish, whereas the “Hot Spots” program primarily applies to consumption of contaminated fresh water sport fish. However, as discussed above, comparable fish consumption rates have been observed for both marine and fresh water angler populations. If comprehensive and reliable data become available which describe consumption of freshwater sport fish in California, the current consumption rate values will be revisited

The Central Valley Delta fish consumption study by Shilling et al. (2010) was considered. This study contained a high number of subsistence anglers and did not correct for avidity bias. However, the mean consumption rate of 27.4 g/day for all anglers, and the body weight adjusted value of 0.33 g/kg-day compared well to the SF Bay study avidity-corrected average consumption rates of 28.8 g/day and 0.36 g/kg-day, respectively, for adults (see Table 9.1).

9.8.2 Statistical Correction for Unequal Sampling Probabilities

Samples obtained from on-site surveys, such as the SF Bay and Santa Monica Bay fish consumption rate studies, can provide estimates of the distribution of fish consumption rates for the total angler population being sampled. In order to obtain unbiased estimates for the total angler population in the SF Bay study, the estimates were (1) adjusted for sources of unequal sampling probabilities in fishing frequency, leading to avidity bias, and (2) examined for the effect of interview decliners on the consumption rate estimate.

9.8.2.1 Avidity Bias

How frequently anglers go fishing (i.e., their avidity) can vary widely among anglers. Some may fish daily while others may fish only once per year. In on-site surveys, how often an angler goes fishing determines how likely he or she will be included in the survey. Generally, avid anglers will be over represented in the sample and infrequent anglers will be under represented, resulting in avidity bias (Price et al., 1994; U.S. EPA, 1997; OEHHA, 2001).

Avidity bias presents a concern when an angler's avidity is correlated with important parameters that are being studied, such as consumption rate. If no correlation exists, there is no bias and data adjustments will not change the results. However, if correlation exists, the sample will not accurately reflect the overall angler population. Adjusting for avidity bias allows for the results to more closely reflect general exposure of the target population of the study (i.e., San Francisco Bay anglers), and to determine a point estimate for the California fish consumption rate.

In the SF Bay study, sample data were adjusted for avidity bias by weighting the respondents in proportion to the inverse of their sampling probability during the four weeks prior to the interview. The algorithm for the statistical adjustment for avidity bias can be found in the report. For cases where the population of concern for risk assessment is the general fishing population and fish is not a major exposure pathway, as can be expected in most cases under the "Hot Spots" program, the adjusted (weighted) results that correct for avidity bias are recommended. However, if the fishing population of concern are fishers that consume sport fish on a regular and frequent basis (i.e., at least once per month), the unadjusted values are considered most relevant (OEHHA, 2001). For risks associated with a single fish species from a water body (i.e., single pathway exposures where fish consumption is a major pathway), it has been recommended that the unadjusted values representing the median and the 90^{th} percentile be used to characterize the population at risk (SCCWRP and MBC, 1994; OEHHA, 2001)

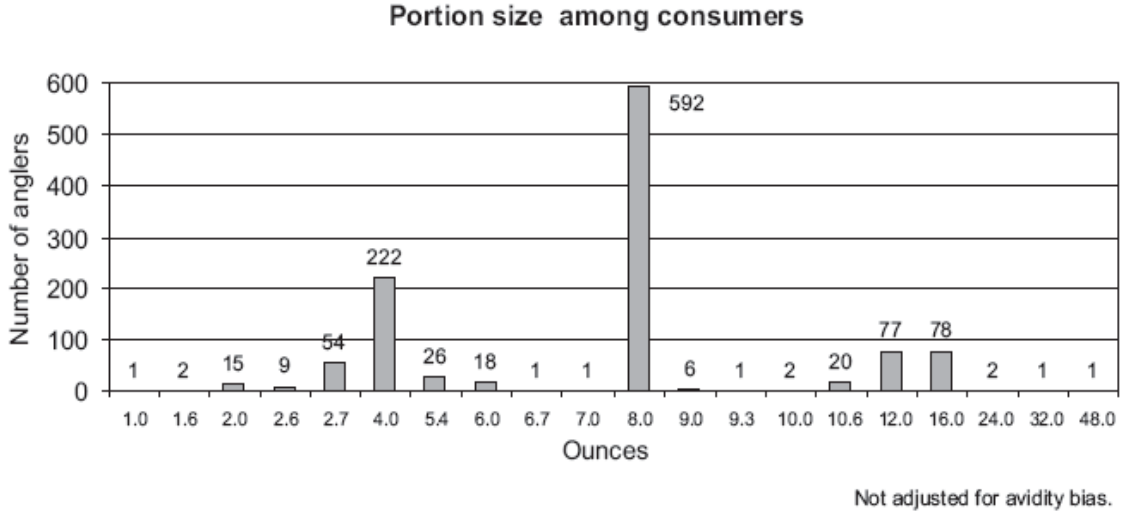
9.8.2.2 Influence of Interview Decliners on the Fish Consumption Rate

Anglers who declined to be interviewed for the SF Bay study represented 23% (n=407) of the net attempted interviews. Lacking data on nearly one fourth of the sample may have introduced some bias. As a worst-case scenario, it was assumed that all decliners had recent consumption (in the last four weeks) of Bay fish, to ensure that the influence of decliners did not result in an underestimation of overall consumption rates of recent consumers. Because ethnicity was the only demographic variable that showed a significant influence on consumption rate, the sample was adjusted to account for ethnic differences between the decliners and interviewed anglers. This was done by assuming that decliners of a certain ethnic group had the same consumption rate as recent consumers interviewed in the same ethnic group. Although any bias associated with anglers who declined to be interviewed is not quantifiable, the analysis using reasonable assumptions about this group revealed that the 23% of anglers from whom the researchers could not directly obtain consumption data were unlikely to influence the overall derived consumption estimates.

9.8.3 Graphical and Statistical Presentation of Consumption Rate Distributions

Figure 9-1 shows the portion size responses among consumers from the SF Bay study (SFEI, 2000) as a distribution. Portion size responses for consumers and recent consumers (i.e., anglers who reported consuming SF Bay fish in the last four weeks) were similar. In general, anglers gave portion size responses in multiples or fractions of the 8-ounce fish fillet model they were shown during the interview. Just over half of consumers reported that the 8-ounce model was equal to the amount they eat at one time, and the overall mean portion size for consumers was 7.7 ounces.

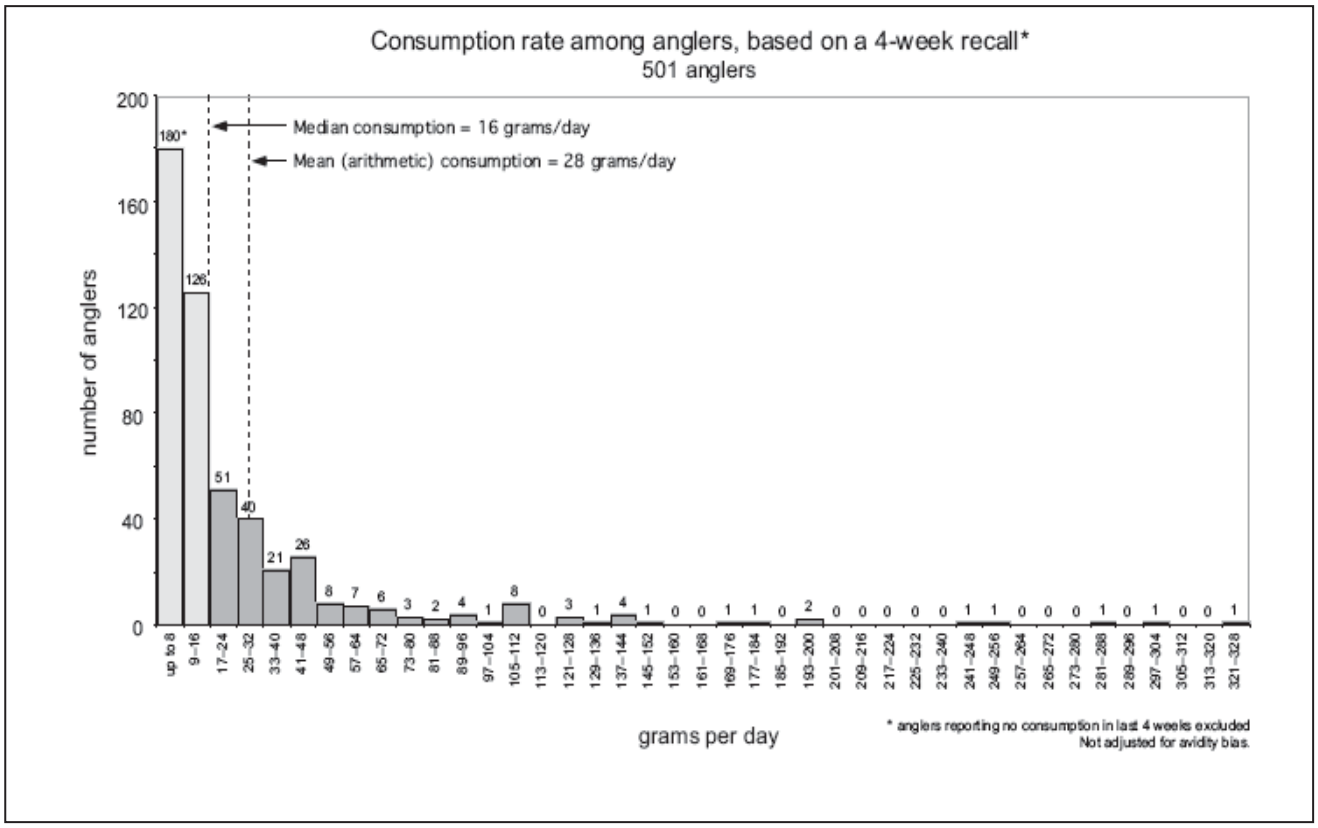
Figure 9



(Reprinted from SFEI, 2000)

Multiplying portion size by meal frequency responses provided by the anglers during the interview gives the consumption rate. Figure 9-2 shows the raw (untransformed) data for consumption rate distribution for recent consumers.

Figure 9-2



(Reprinted from SFEI, 2000)

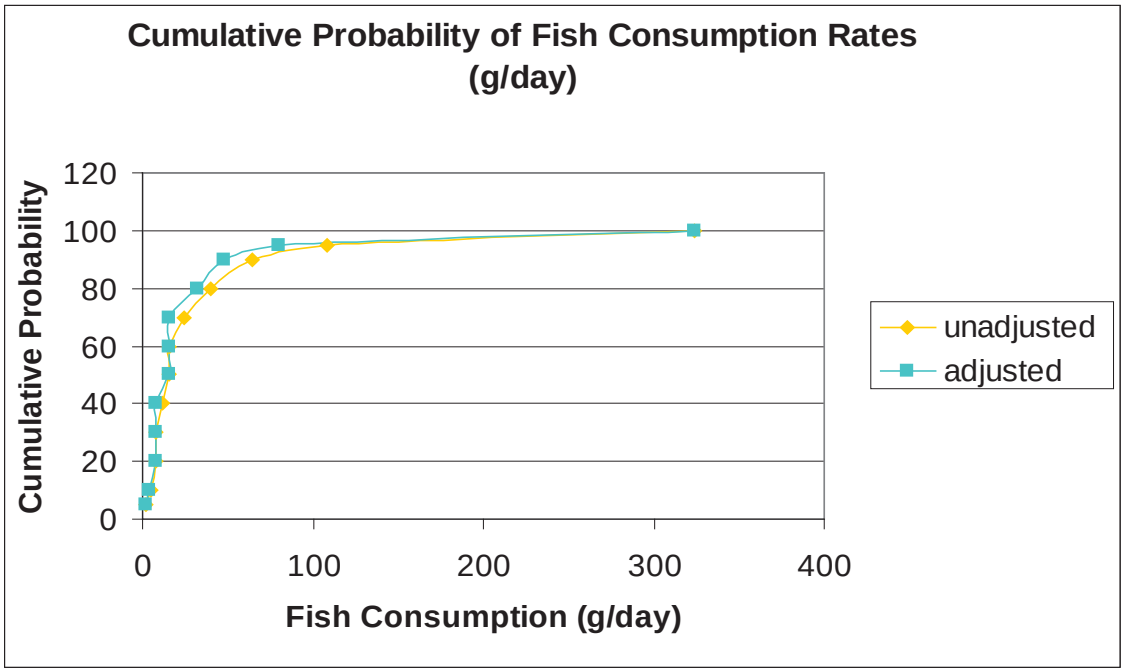
The cumulative empirical distribution curves for the rate of fish consumption for all anglers who caught Bay fish in the SF Bay survey, both unadjusted and adjusted for avidity bias, are shown in Fig. 9-3. The fish consumption rate distribution is highly skewed to the right with a long upper tail, characteristic of a lognormal distribution. The skewness and kurtosis, shown in Table 9.6, are positive. A positive skewness indicates a distribution with a tail to the right. In other words, skewness is an indicator of the lack of symmetry of the distribution. The kurtosis indicates heaviness of the tails. Kurtosis is a measure of whether the data are peaked or flat relative to a normal distribution. That is, data sets with high kurtosis tend to have a distinct peak near the mean, decline rather rapidly, and have heavy tails. Data sets with low kurtosis tend to have a flat top near the mean rather than a sharp peak.

The best fit for the empirical distribution of avidity adjusted fish consumption rates was checked using Crystal Ball (Decisioneering, 2008). The best fit was the lognormal distribution based on the Anderson-Darling, Chi-square, and Kolgomorov-Smirnov goodness of fit tests. The Anderson-Darling test was the most important for our purposes because it gave greater weight to the tails of the distribution. The right tail

represents the most highly exposed in the population so it is important to properly characterize this region of the distribution. Because the lognormal distribution was found to be the best fit, Crystal Ball was also used to fit a lognormal parametric model to the avidity-adjusted data.

Moments and percentiles of the empirical distributions (unadjusted and adjusted for avidity) and of the lognormal fitted avidity adjusted fish consumption rates are presented in Table 9.6. Figure 9-4 depicts the cumulative probability distribution of the lognormal fitted data. The lognormally fit distribution is slightly more skewed to the right than the original empirical distribution. Nonetheless, the empirical avidity adjusted distribution was non-continuous, as evidenced by the somewhat staircase appearance of its graphs (Figs 9-2 and 9-3). The 20th, 30th, and 40th cumulative percentiles all had the same consumption rate value (i.e., 8 g/day) (Table 9.6). Likewise, the 50th, 60th, and 70th percentiles had a 16 g/day value. Fitting a lognormal distribution to the empirical data smoothes the choppy empirical distribution. Though the empirical distribution was appropriate for the sample, the lognormally fit distribution is likely more realistic for the population. For the empirical data, the unadjusted values are higher than the adjusted values because the correction for avidity bias is crucial to compensate for the increase of fish consumption rates with increased frequency (i.e., avidity) of fishing.

Figure 9-3



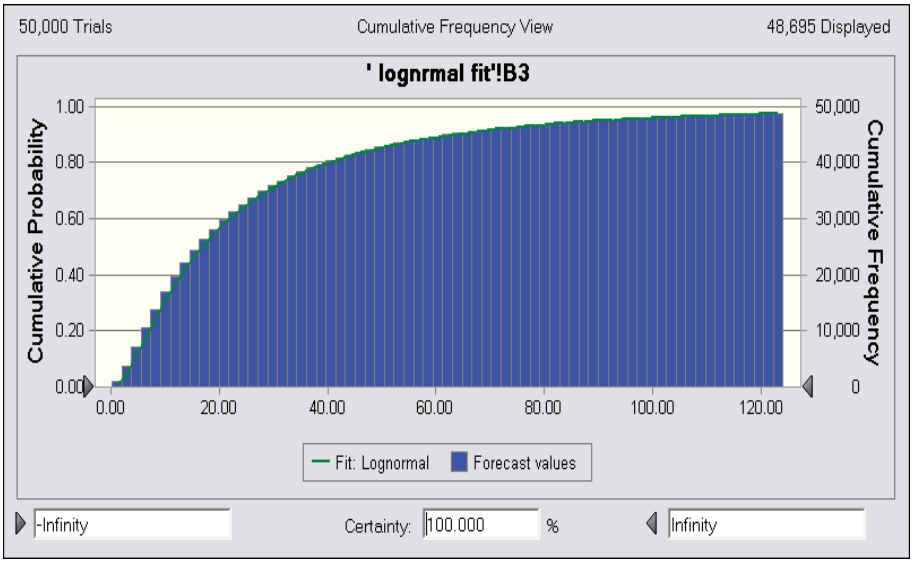
Data source: SFEI (2000)

Table 9.6 Comparison of Empirical Distributions and the Recommended Lognormal Model of Fish Consumption Rates for Stochastic Analysis

	Moments and Percentiles (g/day)		
	Empirical Distribution Unadjusted[a]	**Empirical Distribution Avidity-Bias Adjusted[a]**	**Lognormal Parametric Model Fit to Avidity-Bias Adjusted Data**
Geometric Mean	16.55	13.97	[b]
Arithmetic Mean	28.08	23.02	28.8
Standard Deviation	39.63	32.05	39.6
Skewness	3.9	[b]	6.7
Kurtosis	19.9	[b]	140.3
PERCENTILES			
Sample Minimum	2.00	2.00	0.0
10	5.33	4.00	4.5
20	8.00	8.00	7.1
30	8.00	8.00	9.9
40	12.00	8.00	13.0
50	16.00	16.00	16.9
60	16.00	16.00	22.0
70	24.00	16.00	29.0
80	36.00	32.00	40.3
90	56.00	48.00	63.4
95	108.00	80.00	92.4
99	[b]	[b]	177.0
Sample Maximum	324.00	324.00	[c]

[a] Data from SFEI (2000), Appendix K, Table K29
[b] Not Reported
[c] Not Applicable

Figure 9-4 Cumulative Probability of Avidity Adjusted Fish Consumption Rates (g/day) fit to a Lognormal Distribution



9.9 References

Allen MJ, Velez PV, Diehl DW, McFadden SE and Kelsh M (1996). Demographic variability in seafood consumption rates among recreational anglers of Santa Monica Bay, California, in 1991-1992. Fishery Bulletin 94(4): 597-610.

Anderson HA, Hanrahan LP, Smith A, Draheim L, Kanarek M and Olsen J (2004). The role of sport-fish consumption advisories in mercury risk communication: a 1998-1999 12-state survey of women age 18-45. Environ Res 95(3): 315-24.

Burger J (2000). Gender differences in meal patterns: role of self-caught fish and wild game in meat and fish diets. Environ Res 83(2): 140-9.

Decisioneering (2008). Crystal Ball, Version 11, Fusion Edition, Oracle Corporation, Redwood Shores, CA.

Harnly M, Seidel S, Rojas P, Fornes R, Flessel P, Smith D, Kreutzer R and Goldman L (1997). Biological monitoring for mercury within a community with soil and fish contamination. Environ Health Perspect 105(4): 424-9.

Imm P, Knobeloch L and Anderson HA (2007). Maternal recall of children's consumption of commercial and sport-caught fish: findings from a multi-state study. Environ Res 103(2): 198-204.

Mayfield DB, Robinson S and Simmonds J (2007). Survey of fish consumption patterns of King County (Washington) recreational anglers. J Expo Sci Environ Epidemiol 17(7): 604-12.

Meredith EK and Malvestuto SP (1996). Evaluation of two on-site survey methods for determining daily per capita freshwater fish consumption by anglers. Am Fisheries Soc Symp 16: 271-6.

Murray DM and Burmaster DE (1994). Estimated distribution for average daily consumption of total and self-caught fish for adults in Michigan angler households. Risk Analysis 14(4): 513-9.

Nadon S, Kosatsky T and Przybysz R (2002). Contaminant exposure among women of childbearing age who eat St. Lawrence River sport fish. Arch Environ Health 57(5): 473-81.

OEHHA (2000). Air Toxics Hot Spots Program Risk Assessment Guidelines. Part IV. Exposure Assessment and Stochastic Technical Support Document. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA. Available online at: http://www.oehha.ca.gov.

Technical Support Document for Exposure Assessment and Stochastic Analysis, FINAL, August, 2012

OEHHA (2001). Chemicals in Fish: Consumption of Fish and Shellfish in California and the United States. Final Report. Pesticide and Environmental Toxicology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Available online at: www.oehha.ca.gov/fish/pdf/Fishconsumptionrpt.pdf.

OEHHA (2009). Technical Support Document for Cancer Potency Factors:Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environrmental Health Hazard Assessment. Online at:http://www.oehha.ca.gov/air/hot_spots/2009/TSDCancerPotency.pdf.

Price PS, Su HS and Gray MN (1994). The effect of sampling bias on estimates of angler consumption rates in creel surveys. J Expo Anal Environ Epidemiol 4(3): 355-71.

Puffer HW, Azen SP, Duda MJ and Young DR (1982b). Consumption Rates of Potentially Hazardous Marine Fish Caught in the Metropolitan Los Angeles Area. Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Corvallis, Oregon. Report No. EPA-600/3-82-070.

Puffer HW, Azen SP and Young DR (1982a). Potential health hazards from consumption of fish caught in polluted coastal waters of Los Angeles County. N Am J Fish Management 2: 74-9.

SCCWRP and MBC. (1994). *Santa Monica Bay Seafood Consumption Study. Final Report*. Santa Monica Restoration Project, Monterey Park, CA. Prepared by Southern California Coastal Water Research Project and MBC Applied Environmental Sciences.

SDCDHS. (1990). *San Diego Bay Health Risk Study*. June 12, 1990. San Diego County Department of Health Services. Prepared for Port of San Diego, San Diego, CA. Doc No 25467.

SFEI. (2000). *San Francisco Bay Seafood Consumption Report*. San Francisco Estuary Institute, Richmond CA. Available online at: http://www.sfei.org/node/2022.

Shilling F, White A, Lippert L and Lubell M (2010). Contaminated fish consumption in California's Central Valley Delta. Environ Res 110(4): 334-44.

Silver E, Kaslow J, Lee D, Lee S, Lynn Tan M, Weis E and Ujihara A (2007). Fish consumption and advisory awareness among low-income women in California's Sacramento-San Joaquin Delta. Environ Res 104(3): 410-9.

Toth JF and Brown RB (1997). Racial and gender meanings of why people participate in recreational fishing. Leisure Sci 19: 129-46.

U.S. EPA (1991). OSWER Directive 9285.6-03 Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors". PB91-921314.

Technical Support Document for Exposure Assessment and Stochastic Analysis, FINAL, August, 2012

U.S. EPA. (1997). *Exposure Factors Handbook*. Volume II - Food Ingestion Factors, Chapter 10 - Intake of Fish and Shellfish. United States Environmental Protection Agency, Washington DC, Doc No. EPA/600/P-95/002Fb. Available Online at: www.epa.gov/ncea/efh/pdfs/efh-chapter10.pdf.

U.S. EPA. (2000). *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories. Volume 2, Risk Assessment and Fish Consumption Limits, Third Edition*. United States Environmental Protection Agency, Washington DC. EPA823-B-00-008.

U.S. EPA. (2002). *Child-Specific Exposure Factors Handbook*. National Center for Environmental Assessment, United States Environmental Protection Agency, Washington DC, EPA/600/P-00/002B. Available online at: www.epa.gov/ncea.

West PC, Fly JM, Marnas R and Larkin F. (1989b). *Michigan Sport Anglers Fish Consumption Survey Supplement I, Non-Response Bias and Consumption Suppression Effect Adjustments: A Report to the Michigan Toxic Substance Control Commission*. September, 1989. University of Michigan, School of Natural Resources, Natural Resource Sociology Research Lab, Technical Report 2. Ann Arbor, MI.

West PC, Fly JM, Marnas R and Larkin F. (1989c). *Michigan Sport Anglers Fish Consumption Survey, Supplement II, Test for Stability of Consumption Rates Over Time: A Report to the Michigan Toxic Substance Control Commission.* . October, 1989. University of Michigan, School of Natural Resources, Natural Resource Sociology Research Laboratory Technical Report 3, Ann Arbor, MI

West PC, Fly JM, R. M and Larkin F. (1989a). *Michigan Sport Anglers Fish Consumption Survey: A Report to the Michigan Toxic Substance Control Commission.* May, 1989. University of Michigan, School of Natural Resources, Natural Resource Sociology Research Lab, Technical Report #1, Ann Arbor, MI

Wong K. (1997). Fishing for Food in San Francisco Bay: Part II. An Environmental Health and Safety Report from Save San Francisco Bay Association. Oakland, CA

10 Body Weight

10.1 Introduction

Body weight is an important variate in risk assessment that is used in calculating dose (mg/kg body wt). Many of the point estimates and distributions of exposure variates are based on studies that collected body weight data on individual subjects. For example, the food consumption rate data for each subject collected in the Continuing Survey of Food Intake Among Individuals (USDA, 2000) was divided by the body weight of that subject, and distributions of consumption per unit body weight per day were generated. However, a few variates (i.e., fish consumption and soil ingestion) are based on studies that did not collect body weight information on the individual subjects. Therefore a review of the body weight literature was conducted and appropriate body weight defaults were selected to use to calculate the dose in mg/kg body weight in risk assessments for exposure via fish consumption and soil ingestion. Note that the fish consumption pathway has been very rarely invoked in the Hot Spots program.

10.2 Recommended Point Estimates for Body Weights

Recommended body weight point estimates in Table 10.1 for specific age groupings are based on raw data for age-specific body weights of U.S. residents collected in the National Health and Nutrition Examination Surveys (NHANES) discussed below in Section 10.3. The measured NHANES-derived body weight data likely represent accurate estimates of body weight for Californians and U.S. citizens.

In the interest of simplicity males and females are averaged. Little gender-based data is available for the two variates in which this body weight information is used, namely soil ingestion and angler-caught fish consumption. OEHHA concluded that the additional level of refinement by gender for body weight to use in these two exposure pathways does not add enough useful information to a risk assessment to warrant the increased complexity of the assessment. If a toxicant affects only one or predominantly one gender, the assessor may want to adjust point estimates and distributions of intake parameters to reflect body weight of the gender in question. However, such an adjustment will not result in a significant change in the results of the risk assessment.

Table 10.1. Mean Point Estimates for Body Weight (Kg)

Age Range (years)	Mean
0<2	9.7
2<9	21.9
2<16	37.0
16<30	75.9
16-70	80.0

Although body weight data of Californians are available, the data are self-reported (See Section 10.4, The California Health Interview Survey). Comparison of the NHANES and California Health Interview Survey datasets presented in Tables 10.4 and 10.7, respectively, shows that California body weight values are similar to the NHANES body weights, but consistently lower in most age groups by <1 to 12%. These generally small differences could mean that self-reported body weights are often underestimated by the CHIS participants. Another possibility is that Californians have body weights that are lower compared to the rest of the U.S. Obesity trends in the U.S. show a lower prevalence for obesity in California compared to many other states (CDC, 2009). However, because the California body weight data was self-reported and NHANES body weight data was not, we chose to utilize the NHANES data.

OEHHA is not recommending body weight distributions for a stochastic approach because most of the consumption rate distributions that we derive from raw data, or recommend from the literature already incorporate subject body weight. It may be appropriate to use body weight distributions when the correlation between body weight and the consumption rate of interest is known. For the fish consumption distribution we have chosen to divide the consumption distribution by a point estimate of body weight because the correlation is not known. If body weight distributions are used without the appropriate correlation, broad distributions are generated that may overestimate the variability in the parameter of interest. We do not have enough information to derive appropriate soil ingestion distributions; thus, use of a point estimate for body weight is appropriate.

10.3 Body Weights Derived from the National Health and Nutrition Examination Surveys (NHANES)

The data collected by NHANES includes detailed anthropometric measurements such as body weight for assessments on the health and nutrition status of U.S. residents (CDC, 2006). The most comprehensive surveys (NHANES II, and III) for body weight were conducted periodically by the National Center for Health Statistics (NCHS) since the 1970s. However, NHANES became a continuous survey in 1999. As anthropometric reference data collection for children and adults is ongoing, 2-year data sets are released as more data become available. The survey samples are nationally representative, from birth to 80+ years of age, from the civilian, non-institutionalized population of the United States. Body weights were recorded for individuals wearing disposable gowns and socks to the nearest 0.1 kg. Some subpopulation subgroups (low income, preschool children, elderly) were oversampled to ensure that sufficient numbers of subjects are available to support estimation to the specified level of precision.

NHANES body weight data represent the most current information on body weight of the U.S. population. NHANES has a large sample size and provides raw data from which interindividual variability can be assessed and categorized by specific age groupings. The body weights recorded for the NHANES reports also have the advantage of being directly measured rather than self-reported.

The most current information on body weights is preferred and summarized in this document because of the rapid increase in obesity incidence in U.S. residents over the last 30 years (Portier et al., 2007). Thus, earlier studies of body weight distributions derived from the NHANES II, including Brainard and Burmaster (1992), Burmaster and Hull (1997), Burmaster and Crouch (1997), and Finley et al. (1994), are not summarized here but can be found in the first edition of this document (OEHHA, 2000).

10.3.1 NCHS Analysis of NHANES 2003-2006 body weight data

The most recently published study by the NCHS that presented NHANES-generated body weight distributions used a combined 4-year dataset based on 2003-2004 and 2005-2006 data (McDowell et al., 2008). A 4-year dataset improves the stability and reliability of the statistical estimates for subgroup analysis. Adolescents 12-19 years of age, persons 60 years of age or older, Mexican Americans, black persons, and low-income persons were oversampled to improve the precision of the statistical estimates for these groups. The 2003-2006 analytic sample was based on 19,593 persons and excluded pregnant females from body weight tabulations. Mean, standard error, and selected percentiles by age group and sex are shown in Table 10.2.

In Table 10.2, estimation of some of the higher percentiles (90^{th} and 95^{th}) did not meet standards of reliability or precision. The reliability of the estimates was evaluated using the relative standard error (RSE), which is calculated by dividing the standard error by the estimate, and the minimum sample size criterion. NCHS recommends that an estimate with an RSE greater than 30 percent be considered unreliable.

Table 10.2. Body Weight in Kg for Children and Adults Derived by NCHS From NHANES 2003-2006

Age Category	Body Weight Means and Percentiles in Kg									
	Males [a]					Females [b]				
	Mean	SE	50th	90th	95th	Mean	SE	50th	90th	95th
0-2 mo	5.2	0.12	5.2	[c]	[c]	4.9	0.10	4.9	[c]	[c]
3-5 mo	7.3	0.08	7.2	8.2	[c]	6.8	0.10	6.6	[c]	[c]
6-8 mo	8.4	0.13	8.4	9.9	[c]	8.1	0.13	8.0	[c]	[c]
9-11 mo	9.7	0.15	9.7	[c]	[c]	9.2	0.11	9.0	[c]	[c]
1 yr	11.6	0.12	11.5	13.8	14.4	10.9	0.11	10.9	13.0	13.4
2 yr	14.1	0.14	13.9	16.4	16.9	13.4	0.13	13.1	16.1	16.8
3 yr	15.8	0.16	15.3	18.7	[c]	15.8	0.20	15.5	18.5	[c]
4 yr	18.6	0.31	18.1	22.7	[c]	17.9	0.21	17.5	20.8	[c]
5 yr	22.1	0.49	21.0	26.9	[c]	20.5	0.37	19.6	25.5	[c]
6 yr	24.2	0.33	23.7	29.5	[c]	23.4	0.49	22.1	29.7	[c]
7 yr	26.6	0.58	25.6	33.9	[c]	27.3	0.62	25.7	35.5	[c]
8 yr	31.4	0.90	29.0	41.9	[c]	30.7	0.94	28.2	42.1	[c]
9 yr	34.6	0.71	32.3	44.1	[c]	36.7	0.99	34.0	50.7	[c]
10 yr	40.1	0.86	37.3	56.8	[c]	42.4	1.07	40.5	58.5	[c]
11 yr	46.8	1.62	44.2	67.0	[c]	49.2	1.31	47.3	68.2	[c]
12 yr	50.8	1.23	46.9	72.8	82.9	52.9	1.31	49.5	76.2	[c]
13 yr	57.8	1.37	55.6	81.0	90.9	57.4	0.98	54.4	76.0	88.5
14 yr	63.1	1.73	59.8	84.3	99.1	58.8	1.75	54.4	81.0	[c]
15 yr	70.2	1.36	66.3	89.9	100.4	60.9	0.76	57.6	81.0	[c]
16 yr	76.1	1.50	70.7	101.9	116.1	61.5	0.95	58.8	79.6	[c]
17 yr	75.0	1.30	70.6	101.3	111.0	66.0	1.66	60.6	87.3	[c]
18 yr	77.2	1.67	72.7	105.8	110.4	67.6	2.15	63.0	92.1	[c]
19 yr	80.2	1.69	76.5	107.3	117.3	67.4	1.79	63.0	92.7	[c]
20-29 yr	85.4	1.06	81.1	111.5	122.6	70.7	1.03	65.3	98.6	110.7
30-39 yr	88.1	0.80	85.9	109.6	120.8	74.7	1.06	70.2	101.7	114.2
40-49 yr	91.8	0.83	88.9	114.0	124.7	77.7	1.03	72.9	106.6	116.9
50-59 yr	90.2	0.95	88.7	113.1	124.4	78.0	1.15	73.7	106.3	117.8
60-69 yr	90.0	0.98	88.0	112.9	121.3	77.3	0.91	74.0	102.0	112.9
70-79 yr	85.0	0.92	83.8	104.5	116.7	70.6	1.07	68.3	91.2	98.9
20 yrs and over	88.3	0.46	85.6	111.5	122.6	74.7	0.53	70.7	101.8	113.6

[a] For male children age groups, n ranged from 101 to 360; for male adult 10-year age groups, n ranged from 555 to 811.
[b] For female children age groups, n ranged from 81 to 335; for female adult 10-year age groups, n ranged from 468 to 779.
[c] Figure does not meet standards of reliability or precision.

10.3.2 U.S. EPA Analysis of NHANES 1999-2006 body weight data

The U.S. EPA analyzed data from the 1999-2006 NHANES to generate distributions of body weight for various age ranges of children in their Child-Specific Exposure Factors Handbook (U.S. EPA, 2008). Because four NHANES datasets were utilized in the analysis (NHANES 1999-2000, 2001-2002, 2003-2004, and 2005-2006) containing approximately 20,000 children, sample weights were developed for the combined dataset in accordance with CDC guidance. Mean and selected percentile body weights for specified age groups derived from NHANES are presented in Table 10.3 for males and females combined.

Table 10.3. Body Weight For Children in Kg Derived by U.S. EPA (2008) From NHANES 1999-2006, Males and Females Combined

Age Group	N	Body Weight Means and Percentiles in Kg				
		Mean	50th	75th	90th	95th
Birth to < 1 mo	158	4.8	4.8	5.1	5.8	6.2
1 to <3 mo	284	5.9	5.9	6.6	7.1	7.3
3 to <6 mo	489	7.4	7.3	8.0	8.7	9.1
6 to <12 mo	927	9.2	9.1	10.1	10.8	11.3
1 to <2 yr	1176	11.4	11.3	12.4	13.4	14.0
2 to <3 yr	1144	13.8	13.6	14.9	16.3	17.1
3 to <6 yr	2318	18.6	17.8	20.3	23.6	26.2
6 to <11 yr	3593	31.8	29.3	36.8	45.6	52.5
11 to <16 yr	5297	56.8	54.2	65.0	79.3	88.8
16 to <21 yr	4851	71.6	67.6	80.6	97.7	108.0

For our objectives, the OEHHA stochastic risk assessment approach is focused on chronic exposure and on deriving parameter distributions for use in assessing cancer risk weighted by age-at-exposure. Thus, we need age groupings that represent 0<2, 2<9, 2<16, 16<30, and 16-70 yrs. The U.S. EPA's body weight data for specified age groups would be useful for assessing hazard for acute and subchronic exposures.

10.3.3 OEHHA Analysis of NHANES 1999-2006 body weight data

The body weight estimates derived by OEHHA in this document consist of a combined 8-year NHANES dataset from 1999 to 2006, each one spanning 2 years (1999-2000, 2001-2002, 2003-2004, and 2005-2006) (NCHS, 2005; 2006; 2007). As of this writing, the 2007-2008 NHANES dataset results had not been finalized. The NHANES body weight data represent the most current information on body weight. NHANES has a large sample size and provides raw data from which OEHHA can assess interindividual variability and categorize by specific age groupings for the purposes of the "Hot Spots" program. Since the survey was meant to be representative of the U.S. population, the

raw data were weighted to reflect the age structure, sex and race of the population at the time of the survey.

The NHANES data included the body weight and age for each participant, so participants were placed into the age groupings consistent with OEHHA's "Hot Spots" program. The body weights for each age group were fit to a lognormal distribution using Crystal Ball® (Decisioneering, 2009). Crystal Ball® was also used to determine the best parametric model fit for the distribution of body weights for each age group. The Anderson-Darling goodness-of-fit test was chosen to determine the best fit distribution because this test specifically gives greater weight to the tails than to the center of the distribution. OEHHA is interested in the tails since the right tail represents the high-end (e.g., 95th percentile) body weights.

For each age group, males and females combined, the mean, and percentiles (50th, 75th, 90th, and 95th) of the body weight distributions are presented in Table 10.4.

Table 10.4. OEHHA-Derived Body Weight Distributional Results Based on the NHANES IV 1999-2006 Surveys, Males and Females Combined

Age Range (years)	N	Body Weight Mean and Percentiles (in kg)				
		Mean	50th	75th	90th	95th
0<2	3034	9.7	9.9	11.5	12.7	13.4
2<9	5626	21.9	20.3	25.5	32.7	36.8
2<16	12,352	37.0	32.1	50.1	64.3	74.8
16<30	8083	75.9	72.1	85.9	102.8	114.9
16-70	32,012	80.0	77.4	91.5	106.6	116.8

Directly measured body weights that are representative of the U.S. population and the large sample sizes are clear advantages for using these body weight distributions. The limitation for using NHANES body weight data is that it is not California-specific; the body weights collected from California participants could not be removed from the report and analyzed separately.

10.3.4 Analysis of NHANES data for body weight changes over time

Distributional changes in body weight over a 24-year period were investigated by Portier et al. (2007) based on NHANES data from three different surveys (II, 1976-1980; III, 1988-1994; IV 1999-2002). For each of the three body weight data sets, the weighted mean and standard deviation of natural log-transformed body weights were computed for single-year age groups and population-specific weight patterns further described using piece-wise polynomial spline functions and nonparametric age-smoothed trend lines.

The analysis demonstrated that there were changes in body weight as well as changes in age-specific distributions over the 24-year time period (Table 10.5). However, the

changes were not constant for all ages. For the most part, mean body weights of children (1-6 yrs) did not change for males, and there was only about a 1 kg change in females from NHANES III to IV. Similarly, there was no change for adolescent males (7-16 years), but there was an upward change in female adolescent average body weight of about 4 kg from the NHANES II to IV surveys. The major differences occurred among adults, where mean body weight for males (18-65 yrs) showed an upward trend of about 3.5 to 4 kg between each survey with about a 4 to 5 kg increase for females (18-65 yrs). Percentile distributions by age group were not provided. This study demonstrates the changing nature of body weights in the U.S. population and the value of using more recent data for risk assessment purposes.

Table 10.5. Comparison of Body Weights in Kg for Selected Age Groupings from NHANES II, III AND IV Surveys

Age Range (years)	NHANES	Male		Female		Overall Male and Female	
		Mean	Std Dev	Mean	Std Dev	Mean	Std Dev
1-6	II	17.04	4.58	16.34	4.70	16.66	4.47
	III	16.88	4.70	16.52	4.91	16.75	4.98
	IV	17.10	4.86	17.46	5.02	17.27	4.97
7-16	II	45.15	17.64	43.93	15.91	44.75	17.49
	III	49.34	20.94	46.77	18.02	47.76	18.40
	IV	47.86	20.10	47.87	19.19	47.73	19.13
18-65	II	78.65	13.23	65.47	13.77	71.23	11.97
	III	82.19	16.18	69.45	16.55	75.61	18.02
	IV	85.47	19.03	74.55	19.32	79.96	20.73
65+	II	74.45	13.05	66.26	13.25	69.56	12.20
	III	79.42	14.66	66.76	14.52	72.25	15.71
	IV	83.50	16.35	69.59	14.63	75.54	15.88

10.3.5 Child Growth Charts Derived from NHANES data

Child growth charts, including weight-for-age data, were published by the Centers for Disease Control (Kuczmarski et al., 2002) using improved statistical smoothing procedures in conjunction with several national surveys (NHANES II and III, NHANES I, II and III). Growth charts and percentile distributions for weight by sex and age were presented in two sets of data: Birth to 36 months (infants) and 2 to 20 years (children and adolescents). The surveys were pooled because no single survey in the NHANES series had enough observations to construct growth charts. Sample sizes from 400 to 500 were required to achieve precision of the empirical percentiles at the specific ages for the curve fitting. The weight-for-age curves were smoothed using a 3-parameter linear model and locally weighted regression.

The evaluation of the growth charts found no large or systematic differences between the smoothed percentiles and the empirical data. Very low birth weight (VLBW) infants

were excluded from the infant percentiles, but included in the older child percentile where the effect of VLBW is diminished. The observed mean, standard deviation, and selected percentiles were presented in one month age intervals for infants (birth to 36 months), and 0.5-year intervals for children and adolescents ages 2-20 years.

More recent children body weight results derived from NHANES data have been published and presented above (McDowell et al., 2008; U.S. EPA, 2008), so the CDC growth charts are not reprinted here in this document. However, the growth charts can be downloaded from the website in the listed citation by Kuczmarski et al. (2002) below. The report did not address the upward trend in weight of female children over time noted by Portier et al. (2007), possibly because the later release of NHANES IV survey data (1999-2002) strengthened the observed trend that was not yet firmly established by the earlier surveys used in the CDC report.

10.4 California Health Interview Survey

The California Health Interview Survey (CHIS) is conducted by the California Department of Health Services every two years, with the most recent published survey data collected in 2005 (CHIS, 2006). CHIS is the largest population-based state health survey including individual health information such as health conditions and limitations, health behaviors, and health care access and health insurance coverage information. The report used the same method to adjust for non-response as that used by NHANES, correcting for several factors (e.g., race, ethnicity, household income, etc.) in order to make the body weights more representative of the California population. The individual self-reported body weight information is available to researchers in a statistical program format.

Because body weight and age information was collected for each participant, OEHHA combined the data into the specified age groups and fit a lognormal distribution to their body weights using Crystal Ball® (Decisioneering, 2009), as similarly performed for the NHANES body weight data. The best parametric model fit for the distribution of body weights was determined for each age group and the Anderson-Darling test was used for goodness-of-fit. For each age group, males and females combined, minimum and maximum values, mean, standard error of the mean, and percentiles of the body weight distributions are presented in Table 10.6.

Table 10.6. Body Weight Distributional Data from the California Health Interview Survey, Males and Females Combined

Age Group (years)	N	Body Weight Mean and Percentiles (in kg)						
		Min	Max	Mean	SEM	50th	90th	95th
0<2	1,927	3	32	9.4	0.07	10	13	14
2<9	6,022	9	79	21.4	0.095	20	31	36
2<16	11,719	9	145	36.6	0.176	32	62	71
16<30	6,367	41	150	72.1	0.22	68	95	107
16<70	37,108	41	150	76.0	0.095	73	100	109

Although the state-wide body weight database is specific for Californians, it is self-reported. Self-reported body weights are often underestimated by the participants. The survey, which was conducted by phone, reported a relatively low response rate of 29.2%. However, the report noted that this nonresponse rate was similar to the rate for other phone surveys, and the sampling weights used in the analysis would be expected to adjust much of the bias associated with the high nonresponse rate.

10.5 Analysis of CSFII body weight data

The U.S. Department of Agriculture (USDA) conducts a continuing survey of the food intakes by individuals. Self-reported body weight data were collected during the USDA's 1994-1996 and 1998 Continuing Survey of Food Intake by Individuals (CSFII), which was a multistage probability sample survey of individuals within U.S. households. Distributions of body weights by different age categories from this survey were calculated by Kahn and Stralka (2009) and are shown in Table 10.7.

Table 10.7. Body Weight Distributions from the CSFII, Males and Females Combined

Age Group	N	Body Weight Mean and Percentiles (in kg)				
		Mean	50th	75th	90th	95th
<1 mo	88	4	3	4	4[a]	5[a]
1 to <3 mo	245	5	5	6	6	7[a]
3 to <6 mo	411	7	7	8	9	10
6 to <12 mo	678	9	9	10	11	12
1 to <2 yr	1002	12	11	13	14	15
2 to <3 yr	994	14	14	16	18	19
3 to <6 yr	4112	18	18	20	23	25
6 to <11 yr	1553	30	27	35	41	45
11 to <16 yr	975	54	52	61	72	82
16 to <18 yr	360	67	63	73	86	100[a]
18 to <21 yr	383	69	66	77	89	100[a]
≥21 yr	9049	76	74	86	99	107
≥65 yr	2139	72	71	81	93	100

[a] The sample size did not meet minimum reporting requirements

The CSFII body weight results have the same limitation as the CHIS body weight data, in that self-reported body weights are often underestimated by the participants. Also, more recent and comprehensive national body weight data are available from NHANES.

10.6 International Commission on Radiological Protection

The International Commission on Radiological Protection (ICRP) reviewed and compiled extensive data on anatomical measurements, elemental composition, and physiological values for the human body (ICRP, 2003). Weight (W), length (L), and surface area (SA) during prenatal life are presented as means +/- standard deviation (SD) as a function of gestational age. From the data, a number of allometric relations were derived which relate gestational age to average length, and length to surface area and weight. Postnatal life data from a number of sources were reviewed. Charts presented in the report show mean body weight $\pm$ one SD from 0 to 15 years and adults by sex. However, the bulk of the body weight information is based on Western European data, and it was noted that in some age groupings, differences exist in body weight between North Americans and Europeans.

10.7 References

Brainard J and Burmaster DE (1992). Bivariate distributions for height and weight of men and women in the United States. Risk Anal 12(2): 267-75.

Burmaster DE and Crouch EA (1997). Lognormal distributions for body weight as a function of age for males and females in the United States, 1976-1980. Risk Anal 17(4): 499-505.

Burmaster DE and Hull AH (1997). Using lognormal distributions and lognormal probability plots in probabilistic risk assessments. Hum Ecol Risk Assess 3(2): 235-55.

CDC. (2006). *Analytic Guidelines*. Centers for Disease Control and Prevention. National Center for Health Statistics. Hyattsville, MD. Available online at: http://www.cdc.gov/nchs/data/nhanes/nhanes_03_04/nhanes_analytic_guidelines_dec_2005.pdf.

CDC. (2009). *U.S. Obesity Trends*. Centers for Disease Control and Prevention. Available online at: http://www.cdc.gov/obesity/data/trends.html.

CHIS. (2006). California Health Interview Survey. The Regents of the University of California. Available online at: http://www.chis.ucla.edu.

Decisioneering (2009). Crystal Ball, Version 11, Fusion Edition, Oracle Corporation, Redwood Shores, CA.

Finley B, Proctor D, Scott P, Harrington N, Paustenbach D and Price P (1994). Recommended distributions for exposure factors frequently used in health risk assessment. Risk Anal 14(4): 533-53.

ICRP (2003). Basic anatomical and physiological data for use in radiological protection: reference values. International Commission on Radiological Protection, ICRP Publication 89. Elsevier Science Ltd.

Kahn HD and Stralka K (2009). Estimated daily average per capita water ingestion by child and adult age categories based on USDA's 1994-1996 and 1998 continuing survey of food intakes by individuals. J Expo Sci Environ Epidemiol 19(4): 396-404.

Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, Wei R, Curtin LR, Roche AF and Johnson CL (2002). 2000 CDC Growth Charts for the United States: methods and development. Vital Health Stat 11(246): 1-190, Available online at: http://www.cdc.gov/nchs/data/series/sr_11/sr11_246.pdf.

McDowell MA, Fryar CD, Ogden CL and Flegal KM. (2008). *Anthropometric reference data for children and adults: United States, 2003-2006*. no 10. National Center for Health Statisctics, Hyattsville, MD.

NCHS. (2005). *National Health and Nutrition Examination Surveys 2003-2004*. National Center for Health Statistics. Available online at: http://www.cdc.gov/nchs/about/major/nhanes/nhanes2003-2004/exam03_04.htm.

NCHS. (2006). *National Health and Nutrition Examination Surveys 1999-2006* National Center for Health Statistics. Available online at: http://www.cdc.gov/nchs/about/major/nhanes/datalink.htm.

NCHS. (2007). *National Health and Nutrition Examination Survey, 2005-2006 examination files.*: National Center for Health Statistics. Available online at: http://www.cdc.gov/nchs/about/major/nhanes/nhanes2005-2006/exam05_06.htm.

OEHHA (2000). Air Toxics Hot Spots Program Risk Assessment Guidelines. Part IV. Exposure Assessment and Stochastic Technical Support Document. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA. Available online at: http://www.oehha.ca.gov.

Portier K, Tolson JK and Roberts SM (2007). Body weight distributions for risk assessment. Risk Anal 27(1): 11-26.

U.S. EPA (2008). Child-Specific Exposure Factors Handbook (Final Report). Chapter 8 - Body Weight. U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-06/096F, 2008. Available online at: http://cfpub.epa.gov/ncea/CFM/recordisplay.cfm?deid=199243.

USDA. (2000). *Continuing Survey of Food Intake by Individuals (CSFII) 1994-96, 1998. CD-ROM*. U. S. Department of Agriculture, Agricultural Research Service.

11 Residential and Worker Exposure Duration, Individual vs. Population Cancer Risk, and Evaluation of Short Term Projects

11.1 Introduction

This chapter covers topics related to estimating cancer risk for facility-specific emissions under the Air Toxics Hot Spots program. The Hot spots statute mandates the assessment of cancer risks from airborne emissions of stationary sources to people living or working near a specific facility. The duration of exposure for residential and offsite worker receptors influences the estimate of cancer risk from a specific facility. In the past, cancer risk was estimated for the maximally exposed individual resident who was assumed to be at the point of highest exposure to emitted carcinogens 24 hours per day, 7 days per week for a lifetime. This is a health protective but not particularly realistic assumption. To address this problem, ARB and OEHHA evaluated information available on length of residence at a specific address to develop guidance on the duration of exposure for the residential exposure scenario.

Past risk assessments assumed a 40 year exposure duration for offsite workers based on little data. For the offsite worker exposure scenario, ARB and OEHHA evaluated information available on the length of time people work at the same location. Information on the percentage of time people are at home was also evaluated to provide an adjustment based on activity patterns for time away from home.

This chapter also discusses reporting and more explicitly considering population wide cancer risks separately from the traditional maximally exposed individual cancer risk estimate.

Finally, the chapter presents guidance to the Air Districts for evaluating cancer risks from short-term projects in their purview that are not Hot Spots facilities.

11.1.1 Residential Exposure Duration for Cancer Risk Assessment

An assumption of lifetime exposure duration (70 years) for the calculation of cancer risk is incorporated into the unit risk factors, inhalation cancer potency factors and oral cancer potency factors. The cancer potency factors and unit risk factors are estimated from data from long-term worker epidemiological studies or lifetime rodent studies. A lifetime cancer risk of 5×10^{-5} means that in a population of a million chronically exposed individuals, 50 excess cancer cases would be predicted. Since the cancer potency factors and unit risk factors are based on lifetime or very long-term studies, there are uncertainties in calculating less than lifetime risk.

A complicating factor in estimating cancer risk is the greater impact of early-in-life exposure. Analyses of available data on the influence of age-at-exposure on potency of carcinogens by OEHHA (OEHHA, 2009) and U.S.EPA (U.S.EPA, 2005, Barton et al.,

2005) indicate that early in life exposures to carcinogens are more potent than later in life exposures. This is discussed in detail in OEHHA (2009).

In order to address the issue of early-in life exposures, OEHHA has adopted a policy, based on the available scientific data, of weighting cancer risk from exposures from the third trimester to <2 yrs of age by a factor of ten, and exposures from age two to less than sixteen years by a factor of three (OEHHA, 2009). In addition to innate sensitivities to some carcinogens, children have greater exposures due to physiological and behavioral factors. As a result, a greater proportion of total lifetime risk is accrued by age 16 with lifetime exposure to a constant air concentration than was previously recognized.

Accumulation of risk over a lifetime is thus no longer assumed linear with increasing length of exposure to a constant dose, but depends on the age at exposure. To further complicate estimation of risk, exposure to a constant air contaminant concentration or soil contaminant concentration over time is also not linear. There are physiological and behavioral differences between adults and children, which results in children's doses (mg/kg body weight) being greater than adults at the same environmental contaminant concentration.

When estimating cancer risk from individual stationary facilities to nearby residents, exposure duration is an important determinant of cancer risk. Cancer risk for residents is also influenced by activity patterns. Exposure duration for the resident near a facility amounts to the time that resident lives in his or her house. Another important factor is the number of hours that the resident spends at his or her residence. This factor varies with age. Section 11.5 discusses available information to use in estimating exposure duration for residential exposure scenarios.

11.1.2 Offsite Worker Exposure Duration for Cancer Risk Assessment

Offsite workers near a stationary source of airborne emissions are treated as members of the public in the Hot Spots program. The length of time that a worker is on the job at a specific location determines the exposure duration and is directly proportional to the cancer risks estimated from a specific stationary source. In the past, OEHHA recommended a default of 40 years for employment tenure. OEHHA has examined the data on job tenure in the United States in order to develop a new data-derived high-end estimate of job tenure that would be public health protective without being unnecessarily conservative. These data are not perfect for this purpose but provide a useful basis for our new recommendation. Section 11.6 discusses available information to use in estimating exposure duration for offsite worker exposure scenarios.

The point estimate risk assessment approach (Tier 1 and 2) can be used with more than one estimate of resident chronic exposure duration to give multiple point estimates of cancer risk. For stochastic risk assessment (Tier 3 and 4), OEHHA recommends calculating separate cancer risk distributions for each fixed chronic exposure duration. An alternative approach would be to express the variability in exposure duration as a distribution of residency times and equate residency time to exposure duration. The

variance in residency times would be propagated through the model and contribute to the variance in the cancer risk.

OEHHA does not recommend a distribution of residence times for our model (Tier III). Since each individual knows the length of time that he or she has resided near the facility, if the 9, 30 and 70-year cancer risks are presented the residents should have a better idea of his or her risk.

11.2 Recommendations

11.2.1 Exposure Duration for Estimating Cancer Risk in the Residential and Offsite Worker Exposure Scenarios

OEHHA is recommending that an exposure duration (residency time) of 30 years be used for individual cancer risk determination for the maximally exposed individual resident (MEIR) (Table 11.1). This should provide adequate public health protection against individual risk. Note that the 30 year exposure duration starts in the third trimester to accommodate the increased susceptibility of exposures in early life (OEHHA, 2009), and would apply to both the point estimate and stochastic approaches. Reducing the residency time assumption from 70 years to 30 years will however reduce the protection for the population. Thus, we have recommendations below (Section 11.1.3) for specifically evaluating population cancer risk from facility emissions.

As supplemental information in the risk assessment for the MEIR scenario, OEHHA is recommending that point estimate and stochastic risk estimates also be presented for 9 and 70-year exposure durations, both starting in the third trimester. This will help convey the message to the public that cancer risk is proportional to the duration of exposure (i.e., length of residency near the facility). Different communities may have different patterns of residency duration and the pattern within the community may need to be considered by the risk manager.

Although the data for determining residency duration is less than perfect, it is likely that 30 years is a reasonable estimate of the 90^{th} or 95^{th} percentile of residency duration in a population. Thus, a 30-year residency time is consistent with recommendations for other risk assessment variates in our model. In addition, it should be noted that accounting for the greater potency of early-in-life exposure using the Age Sensitivity Factors (OEHHA, 2009) means that a smaller fraction of lifetime risk is incurred after age 30.

Note that there is an assumption that after the person moves, he or she is no longer significantly exposed to the emissions from the facility in question. However the larger the isopleths of cancer risks, the greater the probability that the person could be moving into a residence still impacted by the facility. As the size of the cancer risk isopleths increases, the probability that population risk will be more important in terms of public health increases (see discussion in Section 11.7).

OEHHA recommends, based on the available data, that 25 years be used as a reasonable estimate of the 95^{th} percentile of employment duration for the Hot Spots

program. Thus, for estimating cancer risk for the offsite worker scenario, a 25 year exposure duration should be used.

The time that a person is away from his or her residence can mean either no exposure to a small facility's emissions, or in the case of a facility with a large isopleth footprint, continuing significant exposure. The available California data do not determine distance from residence during time away from residence (Appendix L). This makes it difficult to come up with a general recommendation, protective of public health, for evaluating risk to the residential MEI during the time that a person is away from the residence. However, OEHHA notes it is appropriate to consider the fraction of time people spend at home as an adjustment for exposure to carcinogens (Table 11.2)

A large fraction of lifetime (70-year) cancer risk and an even larger fraction of the cancer risk for the first 30 years in life is incurred during the first 16 years of life because of the higher risk of early in life exposure. A good fraction of the time away from residence will be spent at school for the first sixteen years of life. Many California schoolchildren attend a local neighborhood school. Therefore, OEHHA is recommending that time away from residence be considered as away from facility emissions (no facility cancer risk) for facilities that do not have a school within the 1×10^{-6} or greater cancer risk isopleth. We recommend no adjustment for time away from residence when there are schools inside the 1×10^{-6} (or greater) cancer risk isopleth. The larger facilities with multiple emissions sources are most likely to have schools within the 1×10^{-6} isopleth and are more likely to cause significant exposure to people while they are away from their residences.

11.2.2 Activity Patterns and Time Spent at Home

OEHHA and ARB evaluated information from activity patterns databases to estimate the percentage of the day that people are home (discussed in Appendix L). This information can be used to adjust exposure duration and risk from a specific facility's emissions, based on the assumption that exposure to the facility's emissions are not occurring away from home. Table L.6 in Appendix L shows the number of minutes spent at home, statewide in California, and the percentage of total time spent at home as well. Ages 0 to 2 spend 85% of their time at home, ages 2 through 15 spend 72% of the their time at home, and ages greater than 15 spend 73% of their time at home (Table 11.2). The data used to determine these percentages were collected by the California Department of Transportation in 2000 and 2001 (Cal Trans, 2001). The time away from the home includes vacations.

11.2.3 Recommendations for Presenting Population Risks

Clear separation of individual risk and population risk and their separate evaluation will be helpful in risk communication and could result in better public health protection and more equitable risk management decisions (further discussed in Section 11.7). The cancer risk estimate based on a 70-year residential exposure does not account for an important aspect of population risk. In particular, large facilities with multiple stacks can dilute emissions over a large area that impact thousands of individuals and theoretically

cause a large number of cancer cases, but because of the dilution, the cancer risk estimate for the maximally exposed individual resident, which is what most risk management decisions are based upon, is below a level of concern. A small facility with a single stack, impacting very few individuals due to more concentrated emissions can exceed individual risk limits set by the air districts, thus triggering notification and other measures. The large facility may in fact have a much greater public health impact (greater number of cancer cases) when population risk is considered. There are different methods that can be used as measure of population burden, based on a lifetime (70 year) cancer risk estimate. Calculating cancer burden as described below is one method. The number of individuals residing within a 1 X 10^{-6}, 1 X 10^{-5}, and/or 1 X 10^{-4} isopleth is another potential measure of population burden (OEHHA, 2003). OEHHA recommends this latter approach for the Hot Spots risk assessments to more explicitly consider population-wide cancer risks from facility emissions. This metric is more easily understood, and provides a metric for population-wide cancer risks that can inform risk management decisions. Cancer burden can also be presented, based on a 70 year lifetime risk estimate.

11.2.4 Recommendations for Exposure Duration for Short-term projects

We recommend that exposure from projects less than 6 months be assumed to last 6 months (e.g., a 2-month project would be evaluated as if it lasted 6 months). Exposure from projects lasting less than two months would not be evaluated for cancer risk. We recommend that exposure from projects lasting more than 6 months be evaluated for the duration of the project. In all cases the exposure should be assumed to start in the third trimester to allow for the use of the Age Sensitivity Factors (OEHHA, 2009). Thus, if the District is evaluating a proposed 5-year mitigation project at a hazardous waste site, the exposure duration for the residents would be from the third trimester through the first five years of life. The exposure duration for the offsite worker scenario would be five years in this case.

Table 11.1 Summary of Recommendations for Exposure Duration Receptor Recommendation

Resident	30 years[a]
Resident (supplemental Information)	9 years for central tendency; 70 years for maximum
Worker	25 years

[a] All durations start with exposure in the third trimester to accommodate use of the Age Sensitivity Factors for early life exposure to carcinogens

Table 11.2 Recommendations for Time Away from Residence for Evaluating Cancer Risk for Facilities Without a School Within the 1x10^{-6} (or greater) Cancer Risk Isopleth[1]

Age Range	Fraction of Time at Residence
3rd Trimester<2	0.85
2<16	0.72
16-30	0.73

[1] Facilities with a school within the 1 X10^{-6} (or greater) cancer risk isopleth should use 1 as the fraction of time at the residence for ages 3rd trimester to less than age 16.

11.3 Cancer Risk Algorithm and Exposure Duration

The following equations for cancer risk can accommodate different exposure durations:

9-year exposure duration - Calculation of Cancer Risk from the Third Trimester to Age Nine:

$$\text{Cancer Risk} = [(ADD_{\text{third trimester}} \times CPF \times 10) \times 0.3\ \text{yrs}/70\ \text{yrs}] + [(ADD_{0\ \text{to}\ <2\text{yrs}} \times CPF \times 10) \times 2\ \text{yrs}/70\ \text{yrs}] + [(ADD_{2 < 9\text{yrs}} \times CPF \times 3) \times 7\ \text{yrs}/70\ \text{yrs}] \times FAH$$

30-year exposure duration - Calculation of Cancer Risk from Third Trimester to Age 30:

$$\text{Cancer Risk} = [(ADD_{\text{third trimester}} \times CPF \times 10) \times 0.3\ \text{yrs}/70\ \text{yrs}] + [(ADD_{0\ \text{to}\ <2\text{yrs}} \times CPF \times 10) \times 2\ \text{yrs}/70\ \text{yrs}] + [(ADD_{2 < 16\text{yrs}} \times CPF \times 3) \times 14\ \text{yrs}/70\ \text{yrs}] + [(ADD_{16 < 30\text{yrs}} \times CPF \times 1) \times 14\text{yrs}/70\ \text{yrs}] \times FAH$$

Lifetime (70 year) exposure duration - Calculation of Cancer Risk from Third Trimester to Age 70:

$$\text{Cancer Risk} = [(ADD_{\text{third trimester}} \times CPF \times 10) \times 0.3\ \text{yrs}/70\ \text{yrs}] + [(ADD_{0\ \text{to}\ <2\text{yrs}} \times CPF \times 10) \times 2\ \text{yrs}/70\ \text{yrs}] + [(ADD_{2 < 16\text{yrs}} \times CPF \times 3) \times 14\ \text{yrs}/70\ \text{yrs}] + [(ADD_{16 < 70\text{yrs}} \times CPF \times 1) \times 54\ \text{yrs}/70\ \text{yrs}] \times FAH$$

where: ADD = Average Daily Dose, mg/kg-d, for the specified time period (estimated using the exposure variates presented in the TSD)

CPF = Cancer Potency Factor $(\text{mg/kg-d})^{-1}$

Age Sensitivity Factor third trimester to less than 2 years = 10

Age Sensitivity Factor age 2 to less than 16 years = 3

Age Sensitivity Factor age 16 to less than 70 years = 1

FAH = Fraction of time at home

ED = Exposure duration, in years

1×10^{-6} = Conversion factor ($\mu g/m^3$) to (mg/L)

AT = Averaging time (period over which exposure is averaged, in years); for carcinogenic effects, the averaging time is 70 years = 25,500 days

Adjustment for exposure less than 365 days/year (e.g., 350 out of 365 days a year to allow for a two week period away from home each year for the residential exposure scenario, or worker exposures of eight hours per day, 5 d/week for the offsite worker exposure scenario) can be factored into the equation using the EF term.

11.4 Available Studies for Evaluating Residency Time and Exposure Duration for the Residential Exposure Scenario

11.4.1 National Studies

Israeli and Nelson (1992) used information from the American Housing Survey (AHS) for the United States for 1985 and 1987 (Bureau of the Census, 1987; 1989) to develop a distribution of average total residence time for all U.S. residents. Finley et al. (1994) calculated more of the percentiles for the data presented by Israeli and Nelson (1992). The mean of the distribution presented by Israeli and Nelson (1992) is 4.6 years. In addition, distributions are presented for subpopulations such as renters and owners, and for regions of the country. The study clearly shows that homeowners have a much greater average residency time than renters and therefore may be a more at risk population from exposure to emissions of a nearby facility. The average residency time for the Western region was lower than for the entire U.S. population.

The authors note that with the methodology they used, there could be repeated sampling or over-sampling of a population of frequent movers. This methodology would also tend to overemphasize the more frequent short duration residency periods that have been found to occur from approximately age twenty to thirty by the Bureau of Census (1988). The Israeli and Nelson (1992) study has information on various categories such as renters, homeowners, farm, urban and rural populations, and large geographic regions such as the West. OEHHA staff did not consider the Israeli and Nelson (1992) study to be appropriate for determining an appropriate residency time to use in less-than-lifetime exposure scenarios in the Air Toxics “Hot Spots” program.

The Israeli and Nelson (1992) study does not examine the effect of socio-economic status on residency times. Many facilities in the Air Toxics “Hot Spots” program are located in areas surrounded by low socioeconomic status populations. OEHHA has published a framework for assessing cumulative impacts, *Cumulative Impacts - Building a Scientific Foundation* (2010), which established the need to take into account socioeconomic factors in risk assessment. As the methodology for doing so evolves, OEHHA will update the Exposure Assessment and Stochastic Analysis Technical Support Document as appropriate.

Johnson and Capel (1992) used a Monte Carlo approach for determining residency occupancy periods. Their methodology can incorporate population information about location, gender, age, and race to develop a mobility table based on US Census data. The mobility table contains the probability that a person with the demographic characteristics considered would not move. A mortality table is also used which determines the probability that a person with the demographic characteristics considered would die. Some of the results from this study are presented in Table 11.3.

Although the published methodology can be used to determine mobility for different income groups, the published tables are for the entire U.S. population. In addition, as is pointed out in the study, the Monte Carlo methodology employed in the study uses the same probability of moving for persons who have resided in their current residence for extended periods as for those who have recently moved in. The data collected by the U.S. Census does not indicate where the individuals queried move to, other than broad descriptions such as “in county”, “out of county”, “within metropolitan area”, and so forth. This problem is common to all of the studies discussed. As a result, it is difficult to define residence time within a zone of impact for those who do not move very far (e.g., within the same apartment complex, neighborhood, or town). The conclusions of this study are similar to the results that the U.S. EPA (1997) reached using the AHS study (Bureau of the Census, 1993) (Table 11.3).

The U.S. EPA (1997) has reviewed the studies presented above. In addition, the U.S. EPA (1997) reviewed the results of the 1991 AHS (Bureau of the Census, 1993). The U.S. Bureau of the Census (1993) conducted a survey using 55,000 interviews, which covered homeowners and renters. Black, white and Hispanic ethnic groups were represented in this study. The U.S. EPA used the information available in this study to determine a distribution of the percent of households who have lived at their current address for several ranges of years. The median and 90th percentiles of this distribution are 9.1 and 32.7 years, respectively. The methodology used to derive the distribution was not specified in the report (U.S. EPA, 1995). Based on the studies by Israeli and Nelson (1992), Johnson and Capel (1992), and their analysis of the U.S. Bureau of the Census (1993), U.S. EPA recommends a central tendency estimate of 9 years, and a high-end estimate of 30 years for residency time.

11.4.2 California-Specific Data on Residency Time

Appendix L used data from The Integrated Public Use Microdata Series (IPUMS-USA) to evaluate residency time. IPUMS-USA consists of more than fifty samples of the American population drawn from fifteen federal censuses and from the American Community Surveys (ACS). ACS is a nationwide survey that collects and produces population and housing information every year from about three million selected housing unit addresses across every county in the nation (ACS). IPUMS-USA samples, which draw on every surviving census from 1850-2000 and the 2000-2009 ACS samples, collectively constitute the quantitative information on long-term changes in the American population. These records for the period since 1940 only identify geographic areas with equal or larger than 100,000 residents (250,000 in 1960 and 1970) (IPUMS-USA).The IPUMS-USA identifies the date moved into the residence and therefore a cumulative distribution of length of time that population has lived in the current residence can be constructed from these data. Figure L2 shows that 91% of the population has lived in their current residence for 29 years or less. This means that only 9% of the population has lived more than 29 years in his or her current residence.

Table 11.3 Summary of Studies of United States Residency Times (in Years)

Israeli and Nelson (1992)	1.4, 23.1 (50th and 95th percentile)
Johnson and Capel (1992)	2.0, 9.0, 33 (5th, 50th and 95th percentile)
U.S. EPA (1997); evaluation of BOC (1993) data	9.1, 32.7 (50th, 90th percentile)
CARB Analysis of IPUMS data (Appendix L)	29 (91st percentile)

11.5 Available Studies for Assessing Job Tenure and Exposure Duration for the Offsite Worker Exposure Scenario

11.5.1 Key National Studies on Job Tenure

The data with respect to job tenure in the United States are mainly cross sectional for determining a Tier 1 default. However, there are some longitudinal data. The purpose of the Census Bureau's Survey of Income and Program Participation (SIPP) is to collect information on source and amount of income, labor force participation, program participation and eligibility, and general demographic characteristics, to measure the effectiveness of existing federal, state, and local programs. The data were collected to estimate future costs and coverage for government programs, such as food stamps, to provide improved statistics on the distribution of income and measures of economic well-being; and to evaluate the effectiveness of federal, state, and local programs.

Like NHANES, the SIPP sample is a multistage-stratified sample of the U.S. civilian non-institutionalized population. Individuals selected for the survey, along with others who live with them, are interviewed once every 4 months over a 48-month period. To spread the work evenly over the 4-month reference period for the interviewers, the Census Bureau randomly divides each panel into four rotation groups. Each rotation group is interviewed in a separate month. Four rotation groups constitute one cycle, or wave, of interviewing, for the entire panel.

The first SIPP panel began interviews in 1983. During the period 1984-1993, a new panel of households was introduced each year in February. In 1990, the Committee on National Statistics (CNSTAT) at the National Research Council reviewed SIPP protocols and made recommendations, many of which were implemented in 1996 and continue to be followed today. In the current version, SIPP is a longitudinal survey that consists of 12 waves of 4 months (4 rotations) each, resulting in a 4-year non-overlapping, continuous cycle, with sample size ranging from approximately 14,000 to 36,700 interviewed households. Included in the SIPP database is information about employment, such as number of concurrent jobs, starting and ending dates of jobs, types of employment, employment income and unemployment compensation, and reasons for leaving a job.

OEHHA analyzed the most recent set of SIPP job data from Wave 1 of the 2008 SIPP survey to evaluate the distribution of employment tenure among employed people in a nationally representative sample. SIPP participants were asked when they started working for a current or most recent past employer, and when they stopped working for that same employer. We disregarded data pertaining to second jobs for individuals who had more than one job at a time. We calculated job duration using job start and end dates, and used an end date of December 31, 2008 for those who were still employed at the same job. We ran frequency distributions of years on the job and years on the job by age using the FREQUENCY and SURVEYFREQ procedures in SAS version 9.1.3 (Table 11.4).

Table 11.4 Employment Tenure by Years on the Job from the Survey of Income and Program Participation (SIPP), 1996-2008

Years on the Job	Percent of Total					
	1996-2008	1996-2008 Summary	2008 Only	2008 Summary	2008 Cumulative Total 0 to 100%	2008 Cumulative Total 100 to 0%
N	**150,017**	**150,017**	**45,363**	**45,363**	**-**	**-**
0	12.67		19.42			100
1	17.87		13.15			
2	10.34		9.87			
3	7.86		7.53			
4	6.06	54.79	5.41	55.38	55.38	44.62
5	5.09		4.58			
6	4.34		3.62			
7	3.48		3.72			
8	3.30		3.87			
9	2.47	18.67	2.59	18.39	73.77	26.23
10	2.82		3.20			
11	2.08		1.93			
12	1.84		1.75			
13	1.59		1.70			
14	1.52	9.84	1.33	9.91	83.68	16.32
15	1.59		1.40			
16	1.45		1.12			
17	1.22		0.94			
18	1.30		1.27			
19	1.05	6.61	1.05	5.78	89.46	10.54
20	1.23		1.34			
21	0.86		0.90			
22	0.82		0.91			
23	0.83		0.84			
24	0.75	4.48	0.63	4.62	94.08	5.92

Table 11.4 Employment Tenure by Years on the Job from the Survey of Income and Program Participation (SIPP), 1996-2008

Years on the Job	Percent of Total					
	1996-2008	1996-2008 Summary	2008 Only	2008 Summary	2008 Cumulative Total 0 to 100%	2008 Cumulative Total 100 to 0%
25	0.70		0.62			
26	0.64		0.47			
27	0.53		0.50			
28	0.57		0.72			
29	0.43	2.87	0.45	2.75	96.83	3.17
30	0.51		0.62			
31	0.37		0.38			
32	0.30		0.30			
33	0.23		0.26			
34	0.23	1.65	0.30	1.87	98.7	1.3
35	0.22		0.26			
36	0.17		0.17			
37	0.13		0.16			
38	0.11		0.17			
39	0.09	0.72	0.12	0.88	99.58	0.42
40	0.08		0.12			
41	0.07		0.06			
42	0.04		0.05			
43	0.04		0.06			
44	0.03	0.25	0.02	0.31	99.89	0.11
45	0.02		0.03			
46	0.01		0.01			
47	0.01		0.01			
48	0.02		0.03			
49	0.01	0.08	0.01	0.09	99.98	0.02
50	0.01		0.01			
51-70	0.044	0.044	0.02	0.02	100	

11.5.2 Supporting Studies

11.5.2.1 Current Population Survey

The Bureau of Labor Statistics (BLS) collects extensive information on the U.S. labor force through the ongoing Current Population Survey (CPS). The CPS is a monthly survey of about 60,000 households that provides data on the labor force status, demographics, and other characteristics of the civilian noninstitutional population ≥16 years of age. One part of the survey includes questions about employee tenure, which is a measure of how long workers had been with their current employer at the time of the survey (BLS, 2008a). Information on employee tenure has been obtained from supplemental questions to the current CPS every two years since 1996. The percent distribution by tenure with current employer is shown in Table 11.5. The data refer to the sole or principal job of full- and part-time workers. All data exclude the incorporated and unincorporated self-employed.

Table 11.5 Distribution of Employed Wage and Salary Workers by Tenure with Current Employer and Age, Males and Females Combined, January 2008 From BLS CPS

Age Group (yrs)	Number employed (in thousands)	Percent Distribution by Tenure with Current Employer							
		≤12 mo	13 to 23 mo	2 yrs	3 to 4 yrs	5 to 9 yrs	10 to 14 yrs	15 to 19 yrs	≥20 yrs
≥16	129,276	22.9	7.4	5.6	16.9	20.2	10.6	6.2	10.3
16-19	5,200	73.8	11.5	7.5	7.0	0.3	- [a]	-	-
≥20	124,076	20.8	7.2	5.5	17.3	21.0	11.0	6.4	10.7
20 - 24	13,139	49.9	13.2	10.2	20.4	6.4	<0.05	-	-
25 - 34	29,097	28.2	10.4	8.5	23.4	23.5	5.4	0.6	<0.05
35 - 44	30,150	17.1	6.6	4.8	18.1	25.5	15.3	8.2	4.5
45 - 54	30,151	12.9	4.4	3.5	13.7	21.6	14.4	9.9	19.4
55 - 64	17,242	9.4	4.3	2.6	11.2	19.7	14.1	10.9	27.8
≥65	4,297	8.9	2.5	2.8	10.6	18.9	16.6	10.4	29.2

[a] Dash represents zero or rounds to zero.

The tenure question in the CPS was designed specifically as a gauge of employment security. Tenure durations beyond 20 years were not computed for Table 11.5, possibly due to the definition of a “lifetime” job lasting at least 20 years by Hall (1982). Thus, longer tenure employment statistical analysis was not considered necessary.

The BLS also presented longitudinal data for median employee tenure by age over the years 1996 to 2008 (Table 11.6). Other distributional percentiles for this tenure data were not presented in the report.

Table 11.6 Median (50th Percentile) Years of Tenure with Current Employer for Employed Wage and Salary Workers by Age 1996 to 2008, Males and Females Combined, from BLS

Age Group (yrs)	1996	1998	2000	2002	2004	2006	2008
≥16	3.8	3.6	3.5	3.7	4.0	4.0	4.1
16 - 17	0.7	0.6	0.6	0.7	0.7	0.6	0.7
18 - 19	0.7	0.7	0.7	0.8	0.8	0.7	0.8
20 - 24	1.2	1.1	1.1	1.2	1.3	1.3	1.3
≥25	5.0	4.7	4.7	4.7	4.9	4.9	5.1
25 - 34	2.8	2.7	2.6	2.7	2.9	2.9	2.7
35 - 44	5.3	5.0	4.8	4.6	4.9	4.9	4.9
45 - 54	8.3	8.1	8.2	7.6	7.7	7.3	7.6
55 - 64	10.2	10.1	10.0	9.9	9.6	9.3	9.9
≥65	8.4	7.8	9.4	8.6	9.0	8.8	10.2

A number of factors can affect employee tenure, including the age profile among workers, type of occupation, and changes in the number of hires and separations with time. The most apparent effect on employee tenure is the age of the worker. As expected, length of tenure to one's employer is strongly related to the age of the worker. For example, in Table 11.6 the median tenure for employees age 55 to 64 in 2008 was 9.9 years, almost four times the tenure (2.7 years) for workers age 25 to 34. Younger working age participants tend to be a more mobile work force. Younger participants also have not accumulated enough working years with any one employer to be considered long-term tenured workers. As workers age, both job stability increases and the number of years since the worker initially began working increases resulting in more workers with jobs that will last 20 years or more.

An earlier study by Farber (1995) used the raw data from the CPS to calculate a distribution of employment-based job duration. Table 11.7 presents the median (50th percentile) and 0.9 quantile (90th percentile) results based on the 1993 CPS findings for tenure with current employer. Although the quantile job tenure results were generated in 1993, the longitudinal median tenure findings in Table 11.6 suggest there has been little change in the numbers since the 1990s.

Table 11.7 Median (50th Percentile) and 0.9 Quantile Job Tenure (in Years) with Current Employer in 1993, Males and Females Combined

Job Tenure Quantiles	Age Category (Years)			
	25-34	35-44	45-54	55-64
Median	3.2	5.8	9.5	12.4
0.9	9.7	17.5	25.2	31.5

The main limitation using the CPS to estimate occupational duration at a single location is that the job tenure question asks for years spent with current employer (i.e., the job is still in progress), rather than completed job duration where there is a start and end date. However, the survey covers the entire span of working years from age 16 to 70+ years. In particular, the oldest groups of participants represent those workers at or near retirement age with a full work history. In addition, Nardone et al. (1997) observed that similar job tenure percentiles were obtained when comparing young workers from both the CPS and NLSY79 surveys (see below).

Comparison of this survey with the SIPP shows that for the first 20 years of employment beginning at age 15 or 16 years, the tenure percentages are almost identical. The CPS shows that 10.3 percent of participants beginning at age 16 are still with their current employer after 20 years. The SIPP (Table 11.4) estimates 10.54 percent of participants are still with their current employer after 20 years.

11.5.2.2 National Survey of Youth 1979

The BLS also collects employment duration data from a separate survey called the National Survey of Youth 1979 (NLSY79). A unique feature of this survey is that it collects the beginning and ending dates of all jobs held by a respondent so that a longitudinal history can be constructed of each respondent's work experience. The NLSY79 is a nationally representative sample of 12,686 young men and women who were 14 to 22 years of age when first surveyed in 1979. The estimates in the current release of data for 2006-2007 contain the first 22 rounds of the survey since 1979 (BLS, 2008b).

The respondents in the NLSY79 are still relatively young, ages 41 to 50 in 2006-07. As the cohort continues to age, information that is more complete will become available. Thus, the current release covers only the period while the respondents were ages 18 to 42; older participants in the study are not included because sample sizes were still too small to provide statistically reliable estimates for age groups >42.

As part of the NLSY79, the duration of employment with a single employer for all jobs started from age 18 to 42 in 1978-2006 is estimated. A job is defined in the survey as an uninterrupted period of work with a particular employer. Jobs are therefore employer-based, not position-based. However, if a respondent indicates that he or she left a job but in a subsequent survey returned to the same job, it is counted as a new job.

Individuals were surveyed annually from 1979 to 1994 and biennially since 1994. In 2006-07, 7,654 individuals responded to the survey, for a retention rate of 77 percent. Only these individuals are included in the estimates in this release. All results are weighted using the 2006-07 survey weights that correct for the oversampling, interview nonresponse, and permanent attrition from the survey. When weighted, the estimates represent all persons born in the years 1957 to 1964 and living in the U.S. when the survey began in 1979 (Table 11.8). Not represented are U.S. immigrants who were born from 1957 to 1964 and moved to the United States after 1979.

Table 11.8 Duration of Employment Relationships with a Single Employer for All Jobs Started from Age 18 to Age 42 in 1978-2006 by Age at Start of Job

Age Group (yrs)	Cumulative Percent Distribution of Duration of Completed Employment Relationships					Percent of jobs ongoing in 2006
	<1 yr	<2 yrs	<5 yrs	<10 yrs	<15 yrs	
18 - 22	72.3	85.2	94.1	97.1	98.0	1.3
23 - 27	59.2	75.9	88.8	94.0	95.7	3.5
28 - 32	52.5	69.7	85.5	91.6	93.6	6.2
33 - 37	42.8	60.7	80.6	88.2	88.9	11.1
38 - 42	30.5	46.6	65.1	ND	ND	30.2

ND - No data. Estimates are not presented for these categories because most sample members were not yet old enough at the time of the 2006-07 survey to have completed jobs of these durations.

Unlike the CPS results, the job duration data in the NLSY79 report are based on starting and ending dates for jobs with a single employer. A limitation of the data is that the survey is still ongoing. Hence, some of the numbers in Table 11.8 will change as the survey is periodically updated, particularly for the most recent findings. Presumably, additional information will also be available for long-term employment in future surveys (i.e., duration of completed employment 15 to <20 yrs).

11.5.2.3 Comparison of the CPS and the NLSY79

Job durations the CPS report were compared by Nardone *et al.* (1997) with a similar cohort of individuals from the NLSY79 data as a yardstick to examine the quality of the CPS data. Specifically, the most recent job tenure data from the NLSY79 28- to 36-year old workers collected in 1993 were compared to the CPS findings for the same age group. Despite the differences in data collection methods between the CPS and NLSY79, the differences in the job tenure distributions were quite small (Table 11.9). Little difference is found at the 90th percentile, with CPS job tenure registering 11.22 years and that of the NLSY79 11.13 years. Overall, Nardone et al. (1997) concluded that the CPS data appear to provide an adequate approximation of the tenure distribution among young workers.

Table 11.9 Distribution of Years of Tenure Among 28- to 35-year old Workers, Current Population Survey (CPS) and National Longitudinal Survey of Youth 1979 (NLSY79), Males and Females Combined

Job Tenure Quantiles	Percentile				
	10th	25th	50th	75th	90th
CPS	0.04	1.04	3.34	7.00	11.22
NLSY79	0.37	1.13	3.46	7.03	11.13

11.6 Individual Resident Cancer Risk vs. Residential Population Risk

A threshold dose for cancer risk for almost all carcinogens cannot be established. Therefore, risk managers must establish a cancer risk that is considered acceptable or de minimus through the political process. Most risk assessments estimate cancer risk at the worker point of maximum exposure (Maximum Exposed Individual Worker or MEIW) and the residential point of maximum exposure (MEIR). This ensures that individual risk is measured at the point with the estimated highest air concentrations of cancer-causing chemicals. The acceptable risk level for individual cancer risk varies in different Federal and State programs from 1×10^{-6} to 1×10^{-4}. In the Hot Spots program, a 1×10^{-5} level for notification is a common standard for the Air Districts. The District may have different levels for permitting, or requiring additional pollution control devices for existing facilities.

The previous OEHHA recommendation of estimating cancer risk for a 70-year residency as a default is health protective for individual risk and provides a degree of population risk public health protection as well. Basing risk management on the cancer risk estimated for a 70 year exposure duration helps reduce the chances a person will experience a cancer risk greater than the acceptable limit (e.g., 10^{-5}) if he or she moves within the isopleths of another similar-risk facility. However, a 70-year residency default also confuses the two concepts of individual risk and population risk. The cancer potency factors are based on the risk to a population, either the population of workers in an occupational study or a population of animals. Yet it is applied to a person or a few people living at the estimated point of maximum impact (the MEI). On the other hand, whether or not a single person is residing at the MEI location over 70 years, there is an assumption in considering population risk that someone will always be living at the MEI location. Thus, in terms of population risk it is irrelevant that the risk at that location is spread over different individuals over time (see discussion below of population versus maximally exposed individual risk).

The individual cancer risk approach has some inherent limitations in terms of protecting public health. A small facility with a single stack can impact a few individuals with an individual cancer risk that is unacceptable, whereas a large facility may have an individual cancer risk that is below the acceptable limit for individual risk but exposes many more people. This large facility can cause more potential cancer cases than the smaller facility and thus have a greater public health impact.

For large facilities with multiple sources such as refineries, ports or rail yards, the population impacts are the primary public health concern. A population risk metric is a better measure of the public health impact and efficacy of proposed control measures. For example, dispersal of repair operations with high diesel emissions in a rail yard will lower individual risk but will not impact population risk. Such a dispersal of operations would not affect the number of cancer cases that would be predicted, but would spread the risk over a larger number of people. Individual risk is a poor metric for progress in public health protection in this example.

To evaluate population risk, regulatory agencies have used the cancer burden as a method to account for the number of excess cancer cases that could occur in a population. The population burden can be calculated by multiplying the cancer risk at a census block centroid times the number of people who live in the census block, and adding up the cancer cases across the zone of impact. A census block is defined as the smallest entity for which the Census Bureau collects and tabulates decennial census information; it is bounded on all sides by visible and nonvisible features shown on Census Bureau maps. The centroid is defined as the central location within a specified geographic area (U.S. Department of Commerce, 1994).

The cancer burden is calculated on the basis of lifetime (70 year) risks. It is independent of how many people move in or out of the vicinity of an individual facility. The number of cancer cases is considered independent of the number of people exposed, within some lower limits of exposed population size, and the length of exposure (within reason). If 10,000 people are exposed to a carcinogen at a concentration with a $1X10^{-5}$ cancer risk for a lifetime the cancer burden is 0.1, and if 100,000 people are exposed to a $1 X 10^{-5}$ risk the cancer burden is 1.

There are different methods that can be used as measure of population burden. The number of individuals residing within a $1 X 10^{-6}$, $1 X 10^{-5}$, and/or $1 X 10^{-4}$ isopleth is another potential measure of population burden (OEHHA, 2003).

11.7 Factors That Can Impact Population Risk – Cumulative Impacts

Although the Hot Spots program is designed to address the impacts of single facilities and not aggregate or cumulative impacts, there are a number of known factors that influence the susceptibility of the exposed population and thus may influence population risk. Socioeconomic status influences access to health care, nutrition, and outcome after cancer diagnosis. Community unemployment can affect exposure and residency time near a facility. Factors that affect the vulnerability of the population are discussed in the report *Cumulative Impacts Building a Scientific Foundation* (OEHHA, 2010). Information on many of these factors is relatively easy to obtain on a census tract level. The OEHHA recommends that these types of factors be considered by the risk manager, along with the quantitative measures of population risk. OEHHA is in the process of developing guidance on quantification of the impact of these factors.

11.8 Cancer Risk Evaluation of Short Term Projects

The local air pollution control districts sometimes use the risk assessment guidelines for the Hot Spots program in permitting decisions. Frequently, the issue of how to address cancer risks from short term projects arises.

Cancer potency factors are based on animal lifetime studies or worker studies where there is long-term exposure to the carcinogenic agent. There is considerable uncertainty in trying to evaluate the cancer risk from projects that will only last a small fraction of a lifetime. There are some studies indicating that dose rate changes the potency of a given dose of a carcinogenic chemical. In others words, a dose delivered

over a short time period may have a different potency than the same dose delivered over a lifetime.

The OEHHA's evaluation of the impact of early-in-life exposure has likely reduced some of the uncertainty in evaluating the cancer risk to the general population for shorter-term exposures, as it helps account for susceptibility to carcinogens by age at exposure (OEHHA, 2009). Thus, we have recommended for short term exposures that the risk assessment start at the third trimester for cancer risk calculation.

11.9 References

(ACS) American Community Survey
http://factfinder2.census.gov/faces/nav/jsf/pages/wc_acs.xhtml

Barton HA, Cogliano VJ, Flowers L, Valcovic L, Setzer RW, Woodruff TJ. (2005) Assessing susceptibility to early life exposure to carcinogens. Environ Health Perspect. Sep;113(9):1125-33.

Bureau of the Census (1988). U.S. Department of Commerce, Bureau of Census and U.S. Department of Housing and Urban Development, American Housing Survey for the United States 1985, Current Housing Reports (H-150-85, 28-29, 1988)

Bureau of the Census (1988). U.S. Department of Commerce, Bureau of the Census, 1980-1985. Mobility Patterns by Age.

Bureau of the Census (1989). U.S. Department of Commerce, Bureau of Census and U.S. Department of Housing and Urban Development, American Housing Survey for the United States 1987, Current Housing Reports (H-150-87, 52-53, 1989).

Bureau of the Census (1993). U.S. Department of Commerce, Bureau of Census and U.S. Department of Housing and Urban Development, American Housing Survey for the United States 1991, Current Housing Reports (H-150-93, 1993).

(CHTS, 2003) 2000-2001 California Statewide Travel Survey Weekday Travel Report. Caltrans, June 2003.
http://www.dot.ca.gov/hq/tsip/tab/documents/travelsurveys/Final2001_StwTravelSurveyWkdayRpt.pdf

Finley B, Proctor D, Scott P, Harrington N, Paustenbach D, and Price P (1994). Recommended distributions for exposure factors frequently used in health risk assessment. Risk Anal 14:533-553.

(IPUMS-USA) Steven Ruggles, J. Trent Alexander, Katie Genadek, Ronald Goeken, Matthew B. Schroeder, and Matthew Sobek. *Integrated Public Use Microdata Series: Version 5.0* [Machine-readable database]. Minneapolis: University of Minnesota, 2010.

Israeli M, and Nelson C (1992). Distribution and expected time of residence of U.S. households. Risk Anal 12:65-72.

Johnson T, and Capel JA (1992). Monte Carlo approach to simulating residential occupancy periods and its application to the general U.S. population. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality and Standards.

OEHHA (2009) Air Toxics Hot Spots Risk Assessment Guidelines. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available

values, and adjustments to allow for early life stage exposures. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots/2009/TSDCancerPotency.pdf

OEHHA (2010) Cumulative Impacts: Building a Scientific Foundation. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Available at: http://www.oehha.ca.gov/ej/cipa123110.html

U.S. Department of Commerce, (1994) Geographic Areas Reference Manual, U. S. Department of Commerce, November, 1994 http://www.census.gov/geo/www/GARM/GARMcont.pdf

U.S. EPA (1997). Exposure Factors Handbook, August 1997. Volume III. Activity Factors. EPA/600/P-95/002Fc.

U.S. EPA (2005) Supplemental Guidelines for Assessing Susceptability from Early Life Exposure to Carcinogens. U.S. Environmental Protection Agency, Washington D.C. EPA/630/R-03/003F; Available at : http://www.epa.gov/cancerguidelines/guidelines-carcinogen-supplement.htm

Appendix A

Substances for which Emissions Must Be Quantified

(as of August, 2007)

SUBSTANCES FOR WHICH EMISSIONS MUST BE QUANTIFIED

CAS number	Substance name
75070	Acetaldehyde
60355	Acetamide
75058	Acetonitrile
98862	Acetophenone
53963	2-Acetylaminofluorene [PAH-Derivative, POM]
107028	Acrolein
79061	Acrylamide
79107	Acrylic acid
107131	Acrylonitrile
107051	Allyl chloride
7429905	Aluminum
1344281	Aluminum oxide (fibrous forms)
117793	2-Aminoanthraquinone [PAH-Derivative, POM]
92671	4-Aminobiphenyl [POM]
61825	Amitrole
7664417	Ammonia
6484522	Ammonium nitrate
7783202	Ammonium sulfate
62533	Aniline
90040	o-Anisidine
-	Anthracene [PAH, POM], (see PAH)
7440360	Antimony
*	Antimony compounds including but not limited to:
1309644	Antimony trioxide
7440382	Arsenic
1016	Arsenic compounds (inorganic) including but not limited to:
7784421	Arsine
1017	Arsenic compounds (other than inorganic)
-	Asbestos (see Mineral fibers)
7440393	Barium
*	Barium Compounds
-	Benz[a]anthracene [PAH, POM], (see PAH)
71432	Benzene
92875	Benzidine (and its salts) [POM]
1020	Benzidine-based dyes [POM] including but not limited to:
1937377	Direct Black 38 [PAH-Derivative, POM]
2602462	Direct Blue 6 [PAH-Derivative, POM]
16071866	Direct Brown 95 (technical grade) [POM]

CAS number	Substance name
-	Benzo[a]pyrene [PAH, POM], (see PAH)
-	Benzo[b]fluoranthene [PAH, POM], (see PAH)
271896	Benzofuran
98077	Benzoic trichloride {Benzotrichloride}
-	Benzo[j]fluoranthene [PAH, POM] (see PAH)
-	Benzo[k]fluoranthene [PAH, POM] (see PAH)
98884	Benzoyl chloride
94360	Benzoyl peroxide
100447	Benzyl chloride
7440417	Beryllium
*	Beryllium compounds
92524	Biphenyl [POM]
111444	Bis(2-chloroethyl) ether {DCEE}
542881	Bis(chloromethyl) ether
103231	Bis(2-ethylhexyl) adipate
7726956	Bromine
*	Bromine compounds (inorganic) including but not limited to:
7789302	Bromine pentafluoride
10035106	Hydrogen bromide
7758012	Potassium bromate
75252	Bromoform
106990	1,3-Butadiene
540885	t-Butyl acetate
141322	Butyl acrylate
71363	n-Butyl alcohol
78922	sec-Butyl alcohol
75650	tert-Butyl alcohol
85687	Butyl benzyl phthalate
7440439	Cadmium
*	Cadmium compounds
156627	Calcium cyanamide
105602	Caprolactam
2425061	Captafol
133062	Captan
63252	Carbaryl [PAH-Derivative, POM]
1050	Carbon black extracts
75150	Carbon disulfide
56235	Carbon tetrachloride
463581	Carbonyl sulfide
1055	Carrageenan (degraded)
120809	Catechol
133904	Chloramben
57749	Chlordane
108171262	Chlorinated paraffins (average chain length, C12; approximately 60% Chlorine by weight)
7782505	Chlorine
10049044	Chlorine dioxide
79118	Chloroacetic acid

CAS number	Substance name
532274	2-Chloroacetophenone
106478	p-Chloroaniline
1058	Chlorobenzenes including but not limited to:
108907	Chlorobenzene
25321226	Dichlorobenzenes (mixed isomers) including:
95501	1,2-Dichlorobenzene
541731	1,3-Dichlorobenzene
106467	p-Dichlorobenzene {1,4-Dichlorobenzene}
120821	1,2,4-Trichlorobenzene
510156	Chlorobenzilate [POM] {Ethyl-4,4'-dichlorobenzilate}
67663	Chloroform
107302	Chloromethyl methyl ether (technical grade)
1060	Chlorophenols including but not limited to:
95578	2-Chlorophenol
120832	2,4-Dichlorophenol
87865	Pentachlorophenol
25167833	Tetrachlorophenols including but not limited to:
58902	2,3,4,6-Tetrachlorophenol
95954	2,4,5-Trichlorophenol
88062	2,4,6-Trichlorophenol
95830	4-Chloro-o-phenylenediamine
76062	Chloropicrin
126998	Chloroprene
95692	p-Chloro-o-toluidine
7440473	Chromium
*	Chromium compounds (other than hexavalent)
18540299	Chromium, hexavalent (and compounds) including but not limited to:
10294403	Barium chromate
13765190	Calcium chromate
1333820	Chromium trioxide
7758976	Lead chromate
10588019	Sodium dichromate
7789062	Strontium chromate
-	Chrysene [PAH, POM], (see PAH)
7440484	Cobalt
*	Cobalt compounds
1066	Coke oven emissions
7440508	Copper
*	Copper compounds
1070	Creosotes
120718	p-Cresidine
1319773	Cresols (mixtures of) {Cresylic acid} including:
108394	m-Cresol
95487	o-Cresol
106445	p-Cresol
4170303	Crotonaldehyde
98828	Cumene
80159	Cumene hydroperoxide

CAS number	Substance name
135206	Cupferron
1073	Cyanide compounds (inorganic) including but not limited to:
74908	Hydrocyanic acid
110827	Cyclohexane
108930	Cyclohexanol
66819	Cycloheximide
	Decabromodiphenyl oxide [POM] (see Polybrominated diphenyl ethers)
1075	Dialkylnitrosamines including but not limited to:
924163	N-Nitrosodi-n-butylamine
1116547	N-Nitrosodiethanolamine
55185	N-Nitrosodiethylamine
62759	N-Nitrosodimethylamine
621647	N-Nitrosodi-n-propylamine
10595956	N-Nitrosomethylethylamine
615054	2,4-Diaminoanisole
1078	Diaminotoluenes (mixed isomers) including but not limited to:
95807	2,4-Diaminotoluene {2,4-Toluene diamine}
334883	Diazomethane
226368	Dibenz[a,h]acridine [POM]
224420	Dibenz[a,j]acridine [POM]
-	Dibenz[a,h]anthracene [PAH, POM], (see PAH)
194592	7H-Dibenzo[c,g]carbazole
-	Dibenzo[a,e]pyrene [PAH, POM], (see PAH)
-	Dibenzo[a,h]pyrene [PAH, POM], (see PAH)
-	Dibenzo[a,i]pyrene [PAH, POM], (see PAH)
-	Dibenzo[a,l]pyrene [PAH, POM], (see PAH)
132649	Dibenzofuran [POM]
96128	1,2-Dibromo-3-chloropropane {DBCP}
96139	2,3-Dibromo-1-propanol
84742	Dibutyl phthalate
-	p-Dichlorobenzene (1,4-Dichlorobenzene) (see Chlorobenzenes)
91941	3,3'-Dichlorobenzidine [POM]
72559	Dichlorodiphenyldichloroethylene {DDE} [POM]
75343	1,1-Dichloroethane {Ethylidene dichloride}
94757	Dichlorophenoxyacetic acid, salts and esters {2,4-D}
78875	1,2-Dichloropropane {Propylene dichloride}
542756	1,3-Dichloropropene
62737	Dichlorovos {DDVP}
115322	Dicofol [POM]
--	Diesel engine exhaust
9901	Diesel engine exhaust, particulate matter **{**Diesel PM**}**
9902	Diesel engine exhaust, total organic gas
#	Diesel fuel (marine)
111422	Diethanolamine
117817	Di(2-ethylhexyl) phthalate {DEHP}
64675	Diethyl sulfate
119904	3,3'-Dimethoxybenzidine [POM]

CAS number	Substance name
60117	4-Dimethylaminoazobenzene [POM]
121697	N,N-Dimethylaniline
57976	7,12-Dimethylbenz[a]anthracene [PAH-Derivative, POM]
119937	3,3'-Dimethylbenzidine {o-Tolidine} [POM]
79447	Dimethyl carbamoyl chloride
68122	Dimethyl formamide
57147	1,1-Dimethylhydrazine
131113	Dimethyl phthalate
77781	Dimethyl sulfate
534521	4,6-Dinitro-o-cresol (and salts)
51285	2,4-Dinitrophenol
42397648	1,6-Dinitropyrene [PAH-Derivative, POM]
42397659	1,8-Dinitropyrene [PAH-Derivative, POM]
25321146	Dinitrotoluenes (mixed isomers) including but not limited to:
121142	2,4-Dinitrotoluene
606202	2,6-Dinitrotoluene
123911	1,4-Dioxane
-	Dioxins (Chlorinated dibenzodioxins) (see Polychlorinated dibenzo-p-dioxins) [POM]
630933	Diphenylhydantoin [POM]
122667	1,2-Diphenylhydrazine {Hydrazobenzene} [POM]
1090	Environmental Tobacco Smoke
106898	Epichlorohydrin
106887	1,2-Epoxybutane
1091	Epoxy resins
140885	Ethyl acrylate
100414	Ethyl benzene
75003	Ethyl chloride {Chloroethane}
-	Ethyl-4,4'-dichlorobenzilate (see Chlorobenzilate)
74851	Ethylene
106934	Ethylene dibromide {EDB, 1,2-Dibromoethane}
107062	Ethylene dichloride {EDC, 1,2-Dichloroethane}
107211	Ethylene glycol
151564	Ethyleneimine {Aziridine}
75218	Ethylene oxide
96457	Ethylene thiourea
1101	Fluorides and compounds including but not limited to:
7664393	Hydrogen fluoride
1103	Fluorocarbons (brominated)
1104	Fluorocarbons (chlorinated) including but not limited to:
76131	Chlorinated fluorocarbon {CFC-113} {1,1,2-Trichloro-1,2,2-trifluoroethane}
75456	Chlorodifluoromethane {Freon 22}
75718	Dichlorodifluoromethane {Freon 12}
75434	Dichlorofluoromethane {Freon 21}
75694	Trichlorofluoromethane {Freon 11}
50000	Formaldehyde
110009	Furan

CAS number	Substance name
--	Gasoline engine exhaust including but not limited to:
--	Gasoline engine exhaust (condensates & extracts)
9910	Gasoline engine exhaust, particulate matter
9911	Gasoline engine exhaust, total organic gas
1110	Gasoline vapors
111308	Glutaraldehyde
1115	Glycol ethers and their acetates including but not limited to:
111466	Diethylene glycol
111966	Diethylene glycol dimethyl ether
112345	Diethylene glycol monobutyl ether
111900	Diethylene glycol monoethyl ether
111773	Diethylene glycol monomethyl ether
25265718	Dipropylene glycol
34590948	Dipropylene glycol monomethyl ether
629141	Ethylene glycol diethyl ether
110714	Ethylene glycol dimethyl ether
111762	Ethylene glycol monobutyl ether
110805	Ethylene glycol monoethyl ether
111159	Ethylene glycol monoethyl ether acetate
109864	Ethylene glycol monomethyl ether
110496	Ethylene glycol monomethyl ether acetate
2807309	Ethylene glycol monopropyl ether
107982	Propylene glycol monomethyl ether
108656	Propylene glycol monomethyl ether acetate
112492	Triethylene glycol dimethyl ether
76448	Heptachlor
118741	Hexachlorobenzene
87683	Hexachlorobutadiene
608731	Hexachlorocyclohexanes (mixed or technical grade) including but not limited to:
319846	alpha-Hexachlorocyclohexane
319857	beta-Hexachlorocyclohexane
58899	Lindane {gamma-Hexachlorocyclohexane}
77474	Hexachlorocyclopentadiene
67721	Hexachloroethane
680319	Hexamethylphosphoramide
110543	Hexane
302012	Hydrazine
7647010	Hydrochloric acid
-	Hydrocyanic acid (see Cyanide compounds)
7783064	Hydrogen sulfide
123319	Hydroquinone
-	Indeno[1,2,3-cd]pyrene [PAH, POM], (see PAH)
13463406	Iron pentacarbonyl
1125	Isocyanates including but not limited to:
822060	Hexamethylene-1,6-diisocyanate
101688	Methylene diphenyl diisocyanate {MDI} [POM]
624839	Methyl isocyanate

CAS number	Substance name
-	Toluene-2,4-diisocyanate (see Toluene diisocyanates)
-	Toluene-2,6-diisocyanate (see Toluene diisocyanates)
78591	Isophorone
78795	Isoprene, except from vegetative emission sources
67630	Isopropyl alcohol
80057	4,4'-Isopropylidenediphenol [POM]
7439921	Lead
1128	Lead compounds (inorganic) including but not limited to:
301042	Lead acetate
-	Lead chromate (see Chromium, hexalent)
7446277	Lead phosphate
1335326	Lead subacetate
1129	Lead compounds (other than inorganic)
108316	Maleic anhydride
7439965	Manganese
*	Manganese compounds
7439976	Mercury
*	Mercury compounds including but not limited to:
7487947	Mercuric chloride
593748	Methyl mercury {Dimethylmercury}
67561	Methanol
72435	Methoxychlor [POM]
75558	2-Methylaziridine {1,2-Propyleneimine}
74839	Methyl bromide {Bromomethane}
74873	Methyl chloride {Chloromethane}
71556	Methyl chloroform {1,1,1-Trichloroethane}
56495	3-Methylcholanthrene [PAH-Derivative, POM]
3697243	5-Methylchrysene [PAH-Derivative, POM]
101144	4,4'-Methylene bis(2-chloroaniline) {MOCA} [POM]
75092	Methylene chloride {Dichloromethane}
101779	4,4'-Methylenedianiline (and its dichloride) [POM]
78933	Methyl ethyl ketone {2-Butanone}
60344	Methyl hydrazine
74884	Methyl iodide {Iodomethane}
108101	Methyl isobutyl ketone {Hexone}
75865	2-Methyllactonitrile {Acetone cyanohydrin}
80626	Methyl methacrylate
109068	2-Methylpyridine
1634044	Methyl tert-butyl ether
90948	Michler's ketone [POM]
1136	Mineral fibers (fine mineral fibers which are man-made, and are airborne particles of a respirable size greater than 5 microns in length, less than or equal to 3.5 microns in diameter, with a length to diameter ratio of 3:1) including but not limited to:
1056	Ceramic fibers
1111	Glasswool fibers
1168	Rockwool
1181	Slagwool

CAS number	Substance name
1135	Mineral fibers (other than man-made) including but not limited to:
1332214	Asbestos
12510428	Erionite
1190	Talc containing asbestiform fibers
1313275	Molybdenum trioxide
-	Naphthalene [PAH, POM], (see PAH)
7440020	Nickel
*	Nickel compounds including but not limited to:
373024	Nickel acetate
3333673	Nickel carbonate
13463393	Nickel carbonyl
12054487	Nickel hydroxide
1271289	Nickelocene
1313991	Nickel oxide
12035722	Nickel subsulfide
1146	Nickel refinery dust from the pyrometallurgical process
7697372	Nitric acid
139139	Nitrilotriacetic acid
602879	5-Nitroacenaphthene [PAH-Derivative, POM]
98953	Nitrobenzene
92933	4-Nitrobiphenyl [POM]
7496028	6-Nitrochrysene [PAH-Derivative, POM]
607578	2-Nitrofluorene [PAH-Derivative, POM]
302705	Nitrogen mustard N-oxide
100027	4-Nitrophenol
79469	2-Nitropropane
5522430	1-Nitropyrene [PAH-Derivative, POM]
57835924	4-Nitropyrene [PAH-Derivative, POM]
86306	N-Nitrosodiphenylamine
156105	p-Nitrosodiphenylamine [POM]
684935	N-Nitroso-N-methylurea
59892	N-Nitrosomorpholine
100754	N-Nitrosopiperidine
930552	N-Nitrosopyrrolidine
*	Oleum (see Sulfuric acid and oleum)
--	PAHs (Polycyclic aromatic hydrocarbons) [POM] including but not limited to:
1151	PAHs, total, w/o individ. components reported [PAH, POM]
1150	PAHs, total, with individ. components also reported [PAH, POM]
83329	Acenaphthene [PAH, POM]
208968	Acenaphthylene [PAH, POM]
120127	Anthracene [PAH, POM]
56553	Benz[a]anthracene [PAH, POM]
50328	Benzo[a]pyrene [PAH, POM]
205992	Benzo[b]fluoranthene
192972	Benzo[e]pyrene [PAH, POM]
191242	Benzo[g,h,i]perylene [PAH, POM]
205823	Benzo[j]fluoranthene [PAH, POM]

CAS number	Substance name
207089	Benzo[k]fluoranthene [PAH, POM]
218019	Chrysene [PAH, POM]
53703	Dibenz[a,h]anthracene [PAH, POM]
192654	Dibenzo[a,e]pyrene [PAH, POM]
189640	Dibenzo[a,h]pyrene [PAH, POM]
189559	Dibenzo[a,i]pyrene [PAH, POM]
191300	Dibenzo[a,l]pyrene [PAH, POM]
206440	Fluoranthene [PAH, POM]
86737	Fluorene [PAH, POM]
193395	Indeno[1,2,3-cd]pyrene [PAH, POM]
91576	2-Methyl naphthalene [PAH, POM]
91203	Naphthalene [PAH, POM]
198550	Perylene [PAH, POM]
85018	Phenanthrene [PAH, POM]
129000	Pyrene [PAH, POM]
#	PAH-Derivatives (Polycyclic aromatic hydrocarbon derivatives) [POM] (including but not limited to those substances listed in Appendix A with the bracketed designation [PAH-Derivative, POM])
56382	Parathion
1336363	PCBs (Polychlorinated biphenyls), total [POM] including but not limited to:
32598133	3,3',4,4'-Tetrachlorobiphenyl (PCB 77)
70362504	3,4,4',5-Tetrachlorobiphenyl (PCB 81)
32598144	2,3,3',4,4'-Pentachlorobiphenyl (PCB 105)
74472370	2,3,4,4',5-Pentachlorobiphenyl (PCB 114)
31508006	2,3',4,4',5-Pentachlorobiphenyl (PCB 118)
65510443	2,3',4,4',5'-Pentachlorobiphenyl (PCB 123)
57465288	3,3',4,4',5-Pentachlorobiphenyl (PCB 126)
38380084	2,3,3',4,4',5-Hexachlorobiphenyl (PCB 156)
69782907	2,3,3',4,4',5'-Hexachlorobiphenyl (PCB 157)
52663726	2,3',4,4',5,5'-Hexachlorobiphenyl (PCB 167)
32774166	3,3',4,4',5,5'-Hexachlorobiphenyl (PCB 169)
39635319	2,3,3',4,4',5,5'-Heptachlorobiphenyl (PCB 189)
82688	Pentachloronitrobenzene {Quintobenzene}
79210	Peracetic acid
127184	Perchloroethylene {Tetrachloroethene}
2795393	Perfluorooctanoic acid {PFOA} and its salts, esters, and sulfonates
108952	Phenol
106503	p-Phenylenediamine
90437	2-Phenylphenol [POM]
75445	Phosgene
7723140	Phosphorus
--	Phosphorus compounds:
7803512	Phosphine
7664382	Phosphoric acid
10025873	Phosphorus oxychloride
10026138	Phosphorus pentachloride
1314563	Phosphorus pentoxide

CAS number	Substance name
7719122	Phosphorus trichloride
126738	Tributyl phosphate
78400	Triethyl phosphine
512561	Trimethyl phosphate
78308	Triorthocresyl phosphate [POM]
115866	Triphenyl phosphate [POM]
101020	Triphenyl phosphite [POM]
85449	Phthalic anhydride
2222	Polybrominated diphenyl ethers {PBDEs}, including but not limited to:
1163195	Decabromodiphenyl oxide [POM]
--	Polychlorinated dibenzo-p-dioxins {PCDDs or Dioxins} [POM including but not limited to:
1086	Dioxins, total, w/o individ. isomers reported {PCDDs} [POM]
1085	Dioxins, total, with individ. isomers also reported {PCDDs} [POM]
1746016	2,3,7,8-Tetrachlorodibenzo-p-dioxin {TCDD} [POM]
40321764	1,2,3,7,8-Pentachlorodibenzo-p-dioxin [POM]
39227286	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin [POM]
57653857	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin [POM]
19408743	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin [POM]
35822469	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin [POM]
3268879	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin [POM]
41903575	Total Tetrachlorodibenzo-p-dioxin [POM]
36088229	Total Pentachlorodibenzo-p-dioxin [POM]
34465468	Total Hexachlorodibenzo-p-dioxin [POM]
37871004	Total Heptachlorodibenzo-p-dioxin [POM]
--	Polychlorinated dibenzofurans {PCDFs or Dibenzofurans} [POM] including but not limited to:
1080	Dibenzofurans (Polychlorinated dibenzofurans) {PCDFs} [POM]
51207319	2,3,7,8-Tetrachlorodibenzofuran [POM]
57117416	1,2,3,7,8-Pentachlorodibenzofuran [POM]
57117314	2,3,4,7,8-Pentachlorodibenzofuran [POM]
70648269	1,2,3,4,7,8-Hexachlorodibenzofuran [POM]
57117449	1,2,3,6,7,8-Hexachlorodibenzofuran [POM]
72918219	1,2,3,7,8,9-Hexachlorodibenzofuran [POM]
60851345	2,3,4,6,7,8-Hexachlorodibenzofuran [POM]
67562394	1,2,3,4,6,7,8-Heptachlorodibenzofuran [POM]
55673897	1,2,3,4,7,8,9-Heptachlorodibenzofuran [POM]
39001020	1,2,3,4,6,7,8,9-Octachlorodibenzofuran [POM]
55722275	Total Tetrachlorodibenzofuran [POM]
30402154	Total Pentachlorodibenzofuran [POM]
55684941	Total Hexachlorodibenzofuran [POM]
38998753	Total Heptachlorodibenzofuran [POM]
#	POM (Polycyclic organic matter) (including but not limited to those substances listed in Appendix A with the bracketed designation of [POM], [PAH, POM], or [PAH-Derivative, POM])
1120714	1,3-Propane sultone
57578	beta-Propiolactone
123386	Propionaldehyde

CAS number	Substance name
114261	Propoxur {Baygon}
115071	Propylene
75569	Propylene oxide
-	1,2-Propyleneimine (see 2-Methylaziridine)
110861	Pyridine
91225	Quinoline
106514	Quinone
1165	Radionuclides including but not limited to:
24267569	Iodine-131
1166	Radon and its decay products
50555	Reserpine [POM]
#	Residual (heavy) fuel oils
7782492	Selenium
*	Selenium compounds including but not limited to:
7783075	Hydrogen selenide
7446346	Selenium sulfide
1175	Silica, crystalline (respirable)
7440224	Silver
*	Silver compounds
1310732	Sodium hydroxide
100425	Styrene
96093	Styrene oxide
*	Sulfuric acid and oleum
8014957	Oleum
7446719	Sulfur trioxide
7664939	Sulfuric acid
100210	Terephthalic acid
79345	1,1,2,2-Tetrachloroethane
-	Tetrachlorophenols (see Chlorophenols)
7440280	Thallium
*	Thallium compounds
62555	Thioacetamide
62566	Thiourea
7550450	Titanium tetrachloride
108883	Toluene
-	2,4-Toluenediamine (see 2,4-Diaminotoluene)
26471625	Toluene diisocyanates including but not limited to:
584849	Toluene-2,4-diisocyanate
91087	Toluene-2,6-diisocyanate
95534	o-Toluidine
8001352	Toxaphene {Polychlorinated camphenes}
-	1,1,1-Trchloroethane (see Methyl chloroform)
79005	1,1,2-Trichloroethane {Vinyl trichloride}
79016	Trichloroethylene
-	2,4,6-Trichlorophenol (see Chlorophenols)
96184	1,2,3-Trichloropropane
121448	Triethylamine
1582098	Trifluralin

CAS number	Substance name
25551137	Trimethylbenzenes including but not limited to:
95636	1,2,4-Trimethylbenzene
540841	2,2,4-Trimethylpentane
51796	Urethane {Ethyl carbamate}
7440622	Vanadium (fume or dust)
1314621	Vanadium pentoxide
108054	Vinyl acetate
593602	Vinyl bromide
75014	Vinyl chloride
100403	4-Vinylcyclohexene
75025	Vinyl fluoride
75354	Vinylidene chloride
1206	Wood preservatives (containing arsenic and chromate)
1330207	Xylenes (mixed**)** including:
108383	m-Xylene
95476	o-Xylene
106423	p-Xylene
7440666	Zinc
*	Zinc compounds including but not limited to:
1314132	Zinc oxide

Appendix B: Regulations and Legislation

B.1. Air Toxics Hot Spots Program Overview

(Air resources Board, 2011: see http://www.arb.ca.gov/ab2588/overview.htm)

INTRODUCTION

The Air Toxics "Hot Spots" Information and Assessment Act (AB 2588, 1987, Connelly) was enacted in September 1987. Under this, stationary sources are required to report the types and quantities of certain substances their facilities routinely release into the air. Emissions of interest are those that result from the routine operation of a facility or that are predictable, including but not limited to continuous and intermittent releases and process upsets or leaks.

The goals of the Air Toxics "Hot Spots" Act are to collect emission data, to identify facilities having localized impacts, to ascertain health risks, and to notify nearby residents of significant risks. In September 1992, the "Hot Spots" Act was amended by Senate Bill (SB) 1731 (Calderon) to address the reduction of significant risks. The bill requires that owners of significant-risk facilities reduce their risks below the level of significance.

The Act requires that toxic air emissions from stationary sources (facilities) be quantified and compiled into an inventory according to criteria and guidelines developed by the ARB, that each facility be prioritized to determine whether a risk assessment must be conducted, that the risk assessments be conducted according to methods developed by the Office of Environmental Health Hazard Assessment (OEHHA), that the public be notified of significant risks posed by nearby facilities, and that emissions which result in a significant risk be reduced. Since the amendment of the statute in 1992 by enactment of SB 1731, facilities that pose a potentially significant health risks to the public are required to reduce their risks, thereby reducing the near-source exposure of Californians to toxic air pollutants. Owners of facilities found to pose significant risks by a district must prepare and implement risk reduction audit and plans within 6 months of the determination.

The Air Resources Board (ARB) is required to develop a program to make the emission data collected under the "Hot Spots" Program available to the public. If requested, districts must make health risk assessments available for public review. Districts must also publish annual reports which summarize the health risk assessment program, rank facilities according to the cancer risk posed, identify the facilities posing non-cancer health risks, and describe the status of the development of control measures.

The "Hot Spots" Program has complemented the ARB's existing air toxics identification and control programs. It has located sources of substances not previously under evaluation, and it has provided exposure information necessary to prioritize substances for control measures and develop regulatory action. Also, the preparation of the "Hot Spots" emission inventory made facility owners aware of their toxics problems. As a

result, facilities have taken voluntary steps to reduce emissions of air toxics. Limited district and facility surveys have identified voluntary reductions of over 1.9 million pounds per year in the emission of air toxics from just 21 facilities in California. The benefits that come from this type of action are less risk to workers and to the public, reduced operation costs, demonstration of emission reduction options for other sources, and improved community relations.

The Act was further modified by AB 564, chaptered on September 19, 1996. The passage of AB 564 amended the Hot Spots statute in several ways, including adding provisions that: exempt specified low priority facilities from further compliance with the Hot Spots program; reinstate exempted facilities if specified criteria are met; specify an alternative evaluation process for facilities subject to district permit programs; and other changes to exempt specified facilities from further compliance with the Hot Spots Program.

B.2. Health and Safety Code Related to Air Toxics Hot Spots.

PART 6. AIR TOXICS "HOT SPOTS" INFORMATION AND ASSESSMENT
(Part 6 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384. Note: Sections 44380 and 44384 became operative Jan. 1, 1988.)

CHAPTER 1: LEGISLATIVE FINDINGS AND DEFINITIONS

44300. This part shall be known and may be cited as the Air Toxics "Hot Spots" Information and Assessment Act of 1987.

44301. The Legislature finds and declares all of the following:

(a) In the wake of recent publicity surrounding planned and unplanned releases of toxic chemicals into the atmosphere, the public has become increasingly concerned about toxics in the air.

(b) The Congressional Research Service of the Library of Congress has concluded that 75 percent of the United States population lives in proximity to at least one facility that manufactures chemicals. An incomplete 1985 survey of large chemical companies conducted by the Congressional Research Service documented that nearly every chemical plant studied routinely releases into the surrounding air significant levels of substances proven to be or potentially hazardous to public health.

(c) Generalized emissions inventories compiled by air pollution control districts and air quality management districts in California confirm the findings of the Congressional Research Service survey as well as reveal that many other facilities and businesses which do not actually manufacture chemicals do use hazardous substances in sufficient quantities to expose, or in a manner that exposes, surrounding populations to toxic air releases.

(d) These releases may create localized concentrations or air toxics "hot spots" where emissions from specific sources may expose individuals and population groups to elevated risks of adverse health effects, including, but not limited to, cancer and contribute to the cumulative health risks of emissions from other sources in the area. In some cases where large populations may not be significantly affected by adverse health risks, individuals may be exposed to significant risks.

(e) Little data is currently available to accurately assess the amounts, types, and health impacts of routine toxic chemical releases into the air. As a result, there exists significant uncertainty about the amounts of potentially hazardous air pollutants which are released, the location of those releases, and the concentrations to which the public is exposed.

(f) The State of California has begun to implement a long-term program to identify, assess, and control ambient levels of hazardous air pollutants, but additional legislation is needed to provide for the collection and evaluation of information concerning the amounts, exposures, and short- and long-term health effects of

hazardous substances regularly released to the surrounding atmosphere from specific sources of hazardous releases.

(g) In order to more effectively implement control strategies for those materials posing an unacceptable risk to the public health, additional information on the sources of potentially hazardous air pollutants is necessary.

(h) It is in the public interest to ascertain and measure the amounts and types of hazardous releases and potentially hazardous releases from specific sources that may be exposing people to those releases, and to assess the health risks to those who are exposed.

44302. The definitions set forth in this chapter govern the construction of this part.

44303. "Air release" or "release" means any activity that may cause the issuance of air contaminants, including the actual or potential spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing of a substance into the ambient air and that results from the routine operation of a facility or that is predictable, including, but not limited to, continuous and intermittent releases and predictable process upsets or leaks.

44304. "Facility" means every structure, appurtenance, installation, and improvement on land which is associated with a source of air releases or potential air releases of a hazardous material.

44306. "Health risk assessment" means a detailed comprehensive analysis prepared pursuant to Section 44361 to evaluate and predict the dispersion of hazardous substances in the environment and the potential for exposure of human populations and to assess and quantify both the individual and populationwide health risks associated with those levels of exposure.

44307. "Operator" means the person who owns or operates a facility or part of a facility.

44308. "Plan" means the emissions inventory plan which meets the conditions specified in Section 44342.

44309. "Report" means the emissions inventory report specified in Section 44341.

CHAPTER 2: FACILITIES SUBJECT TO THIS PART

44320. This part applies to the following:

(a) Any facility which manufactures, formulates, uses, or releases any of the substances listed pursuant to Section 44321 or any other substance which reacts to form a substance listed in Section 44321 and which releases or has the potential to release total organic gases, particulates, or oxides of nitrogen or sulfur in the amounts specified in Section 44322.

(b) Except as provided in Section 44323, any facility which is listed in any current toxics use or toxics air emission survey, inventory, or report released or

compiled by a district. A district may, with the concurrence of the state board, waive the application of this part pursuant to this subdivision for any facility which the district determines will not release any substance listed pursuant to Section 44321 due to a shutdown or a process change.

44321. For the purposes of Section 44320, the state board shall compile and maintain a list of substances that contains, but is not limited to, all of the following:

(a) Substances identified by reference in paragraph (1) of subdivision (b) of Section 6382 of the Labor Code and substances placed on the list prepared by the National Toxicology Program and issued by the United States Secretary of Health and Human Services pursuant to paragraph (4) of subsection (b) of Section 241 of Title 42 of the United States Code. For the purposes of this subdivision, the state board may remove from the list any substance which meets both of the following criteria:
  (1) No evidence exists that it has been detected in air.
  (2) The substance is not manufactured or used in California, or, if manufactured or used in California, because of the physical or chemical characteristics of the substance or the manner in which it is manufactured or used, there is no possibility that it will become airborne.

(b) Carcinogens and reproductive toxins referenced in or compiled pursuant to Section 25249.8, except those which meet both of the criteria identified in subdivision (a).

(c) Substances designated by the state board as toxic air contaminants pursuant to subdivision (b) of Section 39657 and substances on the candidate list of potential toxic air contaminants and the list of designated toxic air contaminants prepared by the state board pursuant to Article 3 (commencing with Section 39660) of Chapter 3.5 of Part 2, including, but not limited to, all substances currently under review and scheduled or nominated for review and substances identified and listed for which health effects information is limited.

(d) Substances for which an information or hazard alert has been issued by the repository of current data established pursuant to Section 147.2 of the Labor Code.

(e) Substances reviewed, under review, or scheduled for review as air toxics or potential air toxics by the Office of Air Quality Planning and Standards of the Environmental Protection Agency, including substances evaluated in all of the following categories or their equivalent: preliminary health and source screening, detailed assessment, intent to list, decision not to regulate, listed, standard proposed, and standard promulgated.

(f) Any additional substances recognized by the state board as presenting a chronic or acute threat to public health when present in the ambient air, including, but not limited to, any neurotoxicants or chronic respiratory toxicants not included within subdivision (a), (b), (c), (d), or (e).

44322. This part applies to facilities specified in subdivision (a) of Section 44320 in accordance with the following schedule:

(a) For those facilities that release, or have the potential to release, 25 tons per year or greater of total organic gases, particulates, or oxides of nitrogen or sulfur, this part becomes effective on July 1, 1988.
(b) For those facilities that release, or have the potential to release, more than 10 but less than 25 tons per year of total organic gases, particulates, or oxides of nitrogen or sulfur, this part becomes effective July 1, 1989.
(c) For those facilities that release, or have the potential to release, less than 10 tons per year of total organic gases, particulates, or oxides of nitrogen or sulfur, the state board shall, on or before July 1, 1990, prepare and submit a report to the Legislature identifying the classes of those facilities to be included in this part and specifying a timetable for their inclusion.

44323. A district may prepare an industrywide emissions inventory and health risk assessment for facilities specified in subdivision (b) of Section 44320 and subdivisions (a) and (b) of Section 44322, and shall prepare an industrywide emissions inventory for the facilities specified in subdivision (c) of Section 44322, in compliance with this part for any class of facilities that the district finds and determines meets all of the following conditions:
(a) All facilities in the class fall within one four-digit Standard Industrial Classification Code.
(b) Individual compliance with this part would impose severe economic hardships on the majority of the facilities within the class.
(c) The majority of the class is composed of small businesses.
(d) Releases from individual facilities in the class can easily and generically be characterized and calculated.

44324. This part does not apply to any facility where economic poisons are employed in their pesticidal use, unless that facility was subject to district permit requirements on or before August 1, 1987. As used in this section, "pesticidal use" does not include the manufacture or formulation of pesticides.

44325. Any solid waste disposal facility in compliance with Section 41805.5 is in compliance with the emissions inventory requirements of this part.

CHAPTER 3: AIR TOXICS EMISSION INVENTORIES

44340.
(a) The operator of each facility subject to this part shall prepare and submit to the district a proposed comprehensive emissions inventory plan in accordance with the criteria and guidelines adopted by the state board pursuant to Section 44342.
(b) The proposed plan shall be submitted to the district on or before August 1, 1989, except that, for any facility to which subdivision (b) of Section 44322 applies, the proposed plan shall be submitted to the district on or before August 1, 1990. The district shall approve, modify, and approve as modified, or return for revision and resubmission, the plan within 120 days of receipt.
(c) The district shall not approve a plan unless all of the following conditions are met:

(1) The plan meets the requirements established by the state board pursuant to Section 44342.
(2) The plan is designed to produce, from the list compiled and maintained pursuant to Section 44321, a comprehensive characterization of the full range of hazardous materials that are released, or that may be released, to the surrounding air from the facility. Air release data shall be collected at, or calculated for, the primary locations of actual and potential release for each hazardous material. Data shall be collected or calculated for all continuous, intermittent, and predictable air releases.
(3) The measurement technologies and estimation methods proposed provide state-of-the-art effectiveness and are sufficient to produce a true representation of the types and quantities of air releases from the facility.
(4) Source testing or other measurement techniques are employed wherever necessary to verify emission estimates, as determined by the state board and to the extent technologically feasible. All testing devices shall be appropriately located, as determined by the state board.
(5) Data are collected or calculated for the relevant exposure rate or rates of each hazardous material according to its characteristic toxicity and for the emission rate necessary to ensure a characterization of risk associated with exposure to releases of the hazardous material that meets the requirements of Section 44361. The source of all emissions shall be displayed or described.

44341. Within 180 days after approval of a plan by the district, the operator shall implement the plan and prepare and submit a report to the district in accordance with the plan. The district shall transmit all monitoring data contained in the approved report to the state board.

44342. The state board shall, on or before May 1, 1989, in consultation with the districts, develop criteria and guidelines for site-specific air toxics emissions inventory plans which shall be designed to comply with the conditions specified in Section 44340 and which shall include at least all of the following:

(a) For each class of facility, a designation of the hazardous materials for which emissions are to be quantified and an identification of the likely source types within that class of facility. The hazardous materials for quantification shall be chosen from among, and may include all or part of, the list specified in Section 44321.
(b) Requirements for a facility diagram identifying each actual or potential discrete emission point and the general locations where fugitive emissions may occur. The facility diagram shall include any nonpermitted and nonprocess sources of emissions and shall provide the necessary data to identify emission characteristics. An existing facility diagram which meets the requirements of this section may be submitted.
(c) Requirements for source testing and measurement. The guidelines may specify appropriate uses of estimation techniques including, but not limited to, emissions factors, modeling, mass balance analysis, and projections, except that source

testing shall be required wherever necessary to verify emission estimates to the extent technologically feasible. The guidelines shall specify conditions and locations where source testing, fence-line monitoring, or other measurement techniques are to be required and the frequency of that testing and measurement.

(d) Appropriate testing methods, equipment, and procedures, including quality assurance criteria.

(e) Specifications for acceptable emissions factors, including, but not limited to, those which are acceptable for substantially similar facilities or equipment, and specification of procedures for other estimation techniques and for the appropriate use of available data.

(f) Specification of the reporting period required for each hazardous material for which emissions will be inventoried.

(g) Specifications for the collection of useful data to identify toxic air contaminants pursuant to Article 2 (commencing with Section 39660) of Chapter 3.5 of Part 2.

(h) Standardized format for preparation of reports and presentation of data.

(i) A program to coordinate and eliminate any possible overlap between the requirements of this chapter and the requirements of Section 313 of the Superfund Amendment and Reauthorization Act of 1986 (Public Law 99-499). The state board shall design the guidelines and criteria to ensure that, in collecting data to be used for emissions inventories, actual measurement is utilized whenever necessary to verify the accuracy of emission estimates, to the extent technologically feasible.

44343. The district shall review the reports submitted pursuant to Section 44341 and shall, within 90 days, review each report, obtain corrections and clarifications of the data, and notify the Office of Environmental Health Hazard Assessment, the Department of Industrial Relations, and the city or county health department of its findings and determinations as a result of its review of the report.

44344. Except as provided in Section 44391, emissions inventories developed pursuant to this chapter shall be updated every four years, in accordance with the procedures established by the state board. Those updates shall take into consideration improvements in measurement techniques and advancing knowledge concerning the types and toxicity of hazardous material released or potentially released.

44344.4.

(a) Except as provided in subdivision (d) and in Section 44344.7, a facility shall be exempt from further compliance with this part if the facility's prioritization scores for cancer and noncancer health effects are both equal to or less than one, based on the results of the most recent emissions inventory or emissions inventory update. An exempt facility shall no longer be required to pay any fee or submit any report to the district or the state board pursuant to this part.

(b) Except for facilities that are exempt from this part pursuant to subdivision (a), a facility for which the prioritization scores for cancer and noncancer health effects are both equal to or less than 10, based on the results of the most recent

emissions inventory or emissions inventory update, shall not be required to pay any fee or submit any report to the district or the state board pursuant to this part, except for the quadrennial emissions inventory update required pursuant to Section 44344. A district may, by regulation, establish a fee to be paid by a facility operator in connection with the operator's submission to the district of a quadrennial emissions inventory update pursuant to this subdivision. The fee shall not be greater than one hundred twenty-five dollars ($125). A district may increase the fee above that amount upon the adoption of written findings that the costs of processing the emission inventory update exceed one hundred twenty-five dollars ($125). However, the district shall not adopt a fee greater than that supported by the written findings.

(c) For the purposes of this part, "prioritization score" means a facility's numerical score for cancer health effects or noncancer health effects, as determined by the district pursuant to Section 44360 in a manner consistent with facility prioritization guidelines prepared by the California Air Pollution Control Officers Association and approved by the state board.

(d) Notwithstanding subdivision (a) and Section 44344.7, if a district has good cause to believe that a facility may pose a potential threat to public health and that the facility therefore does not qualify for an exemption claimed by the facility pursuant to subdivision (a), the district may require the facility to document the facility's emissions and health impacts, or the changes in emissions expected to occur as a result of a particular physical change, a change in activities or operations at the facility, or a change in other factors. The district may deny the exemption if the documentation does not support the claim for the exemption.

44344.5.

(a) The operator of any new facility that previously has not been subject to this part shall prepare and submit an emissions inventory plan and report.

(b) Notwithstanding subdivision (a), a new facility shall not be required to submit an emissions inventory plan and report if all of the following conditions are met:

(1) The facility is subject to a district permit program established pursuant to Section 42300.

(2) The district conducts an assessment of the potential emissions or their associated risks, whichever the district determines to be appropriate, attributable to the new facility and finds that the emissions will not result in a significant risk. A risk assessment conducted pursuant to this paragraph shall comply with paragraph (2) of subdivision (b) of Section 44360.

(3) The district issues a permit authorizing construction or operation of the new facility.

44344.6. A district shall redetermine a facility's prioritization score, or evaluate the prioritization score as calculated and submitted by the facility, within 90 days from the date of receipt of a quadrennial emissions inventory update pursuant to Section 44344 or subdivision (b) of Section 44344.4, within 90 days from the date of receipt of an emissions inventory update submitted pursuant to Section 44344.7, or within 90 days

from the date of receiving notice that a facility has completed the implementation of a plan prepared pursuant to Section 44392.

44344.7.

(a) A facility exempted from this part pursuant to subdivision (a) of Section 44344.4 shall, upon receipt of a notice from the district, again be subject to this part and the operator shall submit an emissions inventory update for those sources and substances for which a physical change in the facility or a change in activities or operations has occurred, as follows:
  (1) The facility emits a substance newly listed pursuant to Section 44321.
  (2) A sensitive receptor has been established or constructed within 500 meters of the facility after the facility became exempt.
  (3) The facility emits a substance for which the potency factor has increased.

(b) The operator of a facility exempted from this part pursuant to subdivision (a) of Section 44344.4 shall submit an emissions inventory update for those sources and substances for which a particular physical change in the facility or a change in activities or operations occurs if, as a result of the particular change, either of the following has occurred:
  (1) The facility has begun emitting a listed substance not included in the previous emissions inventory.
  (2) The facility has increased its emissions of a listed substance to a level greater than the level previously reported for that substance, and the increase in emissions exceeds 100 percent of the previously reported level.

(c) Notwithstanding subdivision (b), a physical change or change in activities or operations at a facility shall not cause the facility to again be subject to this part if all of the following conditions are met:
  (1) The physical change or change in activities or operations is subject to a district permit program established pursuant to Section 42300.
  (2) The district conducts an assessment of the potential changes in emissions or their associated risks, whichever the district determines to be appropriate, attributable to the physical change or change in activities or operations and finds that the changes in emissions will not result in a significant risk. A risk assessment conducted pursuant to this paragraph shall comply with paragraph (2) of subdivision (b) of Section 44360.
  (3) The district issues a permit for the physical change or change in activities or operations.

44345.

(a) On or before July 1, 1989, the state board shall develop a program to compile and make available to other state and local public agencies and the public all data collected pursuant to this chapter.

(b) In addition, the state board, on or before March 1, 1990, shall compile, by district, emissions inventory data for mobile sources and area sources not subject to district permit requirements, and data on natural source emissions, and shall incorporate these data into data compiled and released pursuant to this chapter.

44346.

(a) If an operator believes that any information required in the facility diagram specified pursuant to subdivision (b) of Section 44342 involves the release of a trade secret, the operator shall nevertheless make the disclosure to the district, and shall notify the district in writing of that belief in the report.

(b) Subject to this section, the district shall protect from disclosure any trade secret designated as such by the operator, if that trade secret is not a public record.

(c) Upon receipt of a request for the release of information to the public which includes information which the operator has notified the district is a trade secret and which is not a public record, the following procedure applies:

(1) The district shall notify the operator of the request in writing by certified mail, return receipt requested.

(2) The district shall release the information to the public, but not earlier than 30 days after the date of mailing the notice of the request for information, unless, prior to the expiration of the 30-day period, the operator obtains an action in an appropriate court for a declaratory judgment that the information is subject to protection under this section or for a preliminary injunction prohibiting disclosure of the information to the public and promptly notifies the district of that action.

(d) This section does not permit an operator to refuse to disclose the information required pursuant to this part to the district.

(e) Any information determined by a court to be a trade secret, and not a public record pursuant to this section, shall not be disclosed to anyone except an officer or employee of the district, the state, or the United States, in connection with the official duties of that officer or employee under any law for the protection of health, or to contractors with the district or the state and its employees if, in the opinion of the district or the state, disclosure is necessary and required for the satisfactory performance of a contract, for performance of work, or to protect the health and safety of the employees of the contractor.

(f) Any officer or employee of the district or former officer or employee who, by virtue of that employment or official position, has possession of, or has access to, any trade secret subject to this section, and who, knowing that disclosure of the information to the general public is prohibited by this section, knowingly and willfully discloses the information in any manner to any person not entitled to receive it is guilty of a misdemeanor. Any contractor of the district and any employee of the contractor, who has been furnished information as authorized by this section, shall be considered an employee of the district for purposes of this section.

(g) Information certified by appropriate officials of the United States as necessary to be kept secret for national defense purposes shall be accorded the full protections against disclosure as specified by those officials or in accordance with the laws of the United States.

(h) As used in this section, "trade secret" and "public record" have the meanings and protections given to them by Section 6254.7 of the Government Code and Section 1060 of the Evidence Code. All information collected pursuant to this chapter, except for data used to calculate emissions data required in the facility

diagram, shall be considered "air pollution emission data," for the purposes of this section.

CHAPTER 4: RISK ASSESSMENT

44360.

(a) Within 90 days of completion of the review of all emissions inventory data for facilities specified in subdivision (a) of Section 44322, but not later than December 1, 1990, the district shall, based on examination of the emissions inventory data and in consultation with the state board and the State Department of Health Services, prioritize and then categorize those facilities for the purposes of health risk assessment. The district shall designate high, intermediate, and low priority categories and shall include each facility within the appropriate category based on its individual priority. In establishing priorities pursuant to this section, the district shall consider the potency, toxicity, quantity, and volume of hazardous materials released from the facility, the proximity of the facility to potential receptors, including, but not limited to, hospitals, schools, day care centers, worksites, and residences, and any other factors that the district finds and determines may indicate that the facility may pose a significant risk to receptors. The district shall hold a public hearing prior to the final establishment of priorities and categories pursuant to this section.

(b)

(1) Within 150 days of the designation of priorities and categories pursuant to subdivision (a), the operator of every facility that has been included within the highest priority category shall prepare and submit to the district a health risk assessment pursuant to Section 44361. The district may, at its discretion, grant a 30-day extension for submittal of the health risk assessment.

(2) Health risk assessments required by this chapter shall be prepared in accordance with guidelines established by the Office of Environmental Health Hazard Assessment. The office shall prepare draft guidelines which shall be circulated to the public and the regulated community and shall adopt risk assessment guidelines after consulting with the state board and the Risk Assessment Committee of the California Air Pollution Control Officers Association and after conducting at least two public workshops, one in the northern and one in the southern part of the state. The adoption of the guidelines is not subject to Chapter 3.5 (commencing with Section 11340) of Part 1 of Division 3 of Title 2 of the Government Code. The scientific review panel established pursuant to Section 39670 shall evaluate the guidelines adopted under this paragraph and shall recommend changes and additional criteria to reflect new scientific data or empirical studies.

(3) The guidelines established pursuant to paragraph (2) shall impose only those requirements on facilities subject to this subdivision that are necessary to ensure that a required risk assessment is accurate and complete and shall specify the type of site-specific factors that districts may take into account in determining when a single health risk assessment may be allowed under subdivision (d). The guidelines shall, in addition, allow the operator of a

facility, at the operator's option, and to the extent that valid and reliable data are available, to include for consideration by the district in the health risk assessment any or all of the following supplemental information:

(A) Information concerning the scientific basis for selecting risk parameter values that are different than those required by the guidelines and the likelihood distributions that result when alternative values are used.

(B) Data from dispersion models, microenvironment characteristics, and population distributions that may be used to estimate maximum actual exposure.

(C) Risk expressions that show the likelihood that any given risk estimate is the correct risk value.

(D) A description of the incremental reductions in risk that occur when exposure is reduced.

(4) To ensure consistency in the use of the supplemental information authorized by subparagraphs (A), (B), (C), and (D) of paragraph (3), the guidelines established pursuant to paragraph (2) shall include guidance for use by the districts in considering the supplemental information when it is included in the health risk assessment.

(c) Upon submission of emissions inventory data for facilities specified in subdivisions (b) and (c) of Section 44322, the district shall designate facilities for inclusion within the highest priority category, as appropriate, and any facility so designated shall be subject to subdivision (b). In addition, the district may require the operator of any facility to prepare and submit health risk assessments, in accordance with the priorities developed pursuant to subdivision (a).

(d) The district shall, except where site specific factors may affect the results, allow the use of a single health risk assessment for two or more substantially identical facilities operated by the same person.

(e) Nothing contained in this section, Section 44380.5, or Chapter 6 (commencing with Section 44390) shall be interpreted as requiring a facility operator to prepare a new or revised health risk assessment using the guidelines established pursuant to paragraph (2) of subdivision (a) of this section if the facility operator is required by the district to begin the preparation of a health risk assessment before those guidelines are established.

44361.

(a) Each health risk assessment shall be submitted to the district. The district shall make the health risk assessment available for public review, upon request. After preliminary review of the emissions impact and modeling data, the district shall submit the health risk assessment to the Office of Environmental Health Hazard Assessment for review and, within 180 days of receiving the health risk assessment, the State office shall submit to the district its comments on the data and findings relating to health effects. The district shall consult with the state board as necessary to adequately evaluate the emissions impact and modeling data contained within the risk assessment.

(b) For the purposes of complying with this section, the Office of Environmental Health Hazard Assessment may select a qualified independent contractor to review the data and findings relating to health effects. The office shall not select an independent contractor to review a specific health risk assessment who may have a conflict of interest with regard to the review of that health risk assessment. Any review by an independent contractor shall comply with the following requirements:
(1) Be performed in a manner consistent with guidelines provided by the office.
(2) Be reviewed by the office for accuracy and completeness.
(3) Be submitted by the office to the district in accordance with this section.

(c) The district shall reimburse the Office of Environmental Health Hazard Assessment or the qualified independent contractor designated by the office pursuant to subdivision (b), within 45 days of its request, for its actual costs incurred in reviewing a health risk assessment pursuant to this section.

(d) If a district requests the Office of Environmental Health Hazard Assessment to consult with the district concerning any requirement of this part, the district shall reimburse the office, within 45 days of its request, for the costs incurred in the consultation.

(e) Upon designation of the high priority facilities, as specified in subdivision (a) of Section 44360, the Office of Environmental Health Hazard Assessment shall evaluate the staffing requirements of this section and may submit recommendations to the Legislature, as appropriate, concerning the maximum number of health risk assessments to be reviewed each year pursuant to this section.

44362.

(a) Taking the comments of the Office of Environmental Health Hazard Assessment into account, the district shall approve or return for revision and resubmission and then approve, the health risk assessment within one year of receipt. If the health risk assessment has not been revised and resubmitted within 60 days of the district's request of the operator to do so, the district may modify the health risk assessment and approve it as modified.

(b) Upon approval of the health risk assessment, the operator of the facility shall provide notice to all exposed persons regarding the results of the health risk assessment prepared pursuant to Section 44361 if, in the judgment of the district, the health risk assessment indicates there is a significant health risk associated with emissions from the facility. If notice is required under this subdivision, the notice shall include only information concerning significant health risks attributable to the specific facility for which the notice is required. Any notice shall be made in accordance with procedures specified by the district.

44363.

(a) Commencing July 1, 1991, each district shall prepare and publish an annual report which does all of the following:
  (1) Describes the priorities and categories designated pursuant to Section 44360 and summarizes the results and progress of the health risk assessment program undertaken pursuant to this part.
  (2) Ranks and identifies facilities according to the degree of cancer risk posed both to individuals and to the exposed population.
  (3) Identifies facilities which expose individuals or populations to any noncancer health risks.
  (4) Describes the status of the development of control measures to reduce emissions of toxic air contaminants, if any.

(b) The district shall disseminate the annual report to county boards of supervisors, city councils, and local health officers and the district board shall hold one or more public hearings to present the report and discuss its content and significance.

44364. The state board shall utilize the reports and assessments developed pursuant to this part for the purposes of identifying, establishing priorities for, and controlling toxic air contaminants pursuant to Chapter 3.5 (commencing with Section 39650) of Part 2.

44365.

(a) If the state board finds and determines that a district's actions pursuant to this part do not meet the requirements of this part, the state board may exercise the authority of the district pursuant to this part to approve emissions inventory plans and require the preparation of health risk assessments.

(b) This part does not prevent any district from establishing more stringent criteria and requirements than are specified in this part for approval of emissions inventories and requiring the preparation and submission of health risk assessments. Nothing in this part limits the authority of a district under any other provision of law to assess and regulate releases of hazardous substances.

44366.

(a) In order to verify the accuracy of any information submitted by facilities pursuant to this part, a district or the state board may proceed in accordance with Section 41510.

CHAPTER 5: FEES AND REGULATIONS

44380.

(a) The state board shall adopt a regulation which does all of the following:
  (1) Sets forth the amount of revenue which the district must collect to recover the reasonable anticipated cost which will be incurred by the state board and the Office of Environmental Health Hazard Assessment to implement and administer this part.

(2) Requires each district to adopt a fee schedule which recovers the costs of the district and which assesses a fee upon the operator of every facility subject to this part, except as specified in subdivision (b) of Section 44344.4. A district may request the state board to adopt a fee schedule for the district if the district's program costs are approved by the district board and transmitted to the state board by April 1 of the year in which the request is made.

(3) Requires any district that has an approved toxics emissions inventory compiled pursuant to this part by August 1 of the preceding year to adopt a fee schedule, as described in paragraph (2), which imposes on facility operators fees which are, to the maximum extent practicable, proportionate to the extent of the releases identified in the toxics emissions inventory and the level of priority assigned to that source by the district pursuant to Section 44360.

(b) Commencing August 1, 1992, and annually thereafter, the state board shall review and may amend the fee regulation.

(c) The district shall notify each person who is subject to the fee of the obligation to pay the fee. If a person fails to pay the fee within 60 days after receipt of this notice, the district, unless otherwise provided by district rules, shall require the person to pay an additional administrative civil penalty. The district shall fix the penalty at not more than 100 percent of the assessed fee, but in an amount sufficient in its determination, to pay the district's additional expenses incurred by the person's noncompliance. If a person fails to pay the fee within 120 days after receipt of this notice, the district may initiate permit revocation proceedings. If any permit is revoked, it shall be reinstated only upon full payment of the overdue fee plus any late penalty, and a reinstatement fee to cover administrative costs of reinstating the permit.

(d) Each district shall collect the fees assessed pursuant to subdivision (a). After deducting the costs to the district to implement and administer this part, the district shall transmit the remainder to the Controller for deposit in the Air Toxics Inventory and Assessment Account, which is hereby created in the General Fund. The money in the account is available, upon appropriation by the Legislature, to the state board and the Office of Environmental Health Hazard Assessment for the purposes of administering this part.

(e) For the 1997-98 fiscal year, air toxics program revenues for the state board and the Office of Environmental Health Hazard Assessment shall not exceed two million dollars ($2,000,000), and for each fiscal year thereafter, shall not exceed one million three hundred fifty thousand dollars ($1,350,000). Funding for the Office of Environmental Health Hazard Assessment for conducting risk assessment reviews shall be on a fee-for-service basis.

44380.1. A facility shall be granted an exemption by a district from paying a fee in accordance with Section 44380 if all of the following criteria are met:

(a) The facility primarily handles, processes, stores, or distributes bulk agricultural commodities or handles, feeds, or rears livestock.

(b) The facility was required to comply with this part only as a result of its particulate matter emissions.

(c) The fee schedule adopted by the district or the state board for these types of facilities is not solely based on toxic emissions weighted for potency or toxicity.

44380.5. In addition to the fee assessed pursuant to Section 44380, a supplemental fee may be assessed by the district, the state board, or the Office of Environmental Health Hazard Assessment upon the operator of a facility that, at the operator's option, includes supplemental information authorized by paragraph (3) of subdivision (b) of Section 44360 in a health risk assessment, if the review of that supplemental information substantially increases the costs of reviewing the health risk assessment by the district, the state board, or the office. The supplemental fee shall be set by the state board in the regulation required by subdivision (a) of Section 44380 and shall be set in an amount sufficient to cover the direct costs to review the information supplied by an operator pursuant to paragraph (3) of subdivision (b) of Section 44360.

44381.

(a) Any person who fails to submit any information, reports, or statements required by this part, or who fails to comply with this part or with any permit, rule, regulation, or requirement issued or adopted pursuant to this part, is subject to a civil penalty of not less than five hundred dollars ($500) or more than ten thousand dollars ($10,000) for each day that the information, report, or statement is not submitted, or that the violation continues.

(b) Any person who knowingly submits any false statement or representation in any application, report, statement, or other document filed, maintained, or used for the purposes of compliance with this part is subject to a civil penalty of not less than one thousand dollars ($1,000) or more than twenty-five thousand dollars ($25,000) per day for each day that the information remains uncorrected.

44382. Every district shall, by regulation, adopt the requirements of this part as a condition of every permit issued pursuant to Chapter 4 (commencing with Section 42300) of Part 4 for all new and modified facilities.

44384. Except for Section 44380 and this section, all provisions of this part shall become operative on July 1, 1988.

CHAPTER 6: FACILITY RISK REDUCTION AUDIT AND PLAN

44390. For purposes of this chapter, the following definitions apply:

(a) "Airborne toxic risk reduction measure" or "ATRRM" means those in-plant changes in production processes or feedstocks that reduce or eliminate toxic air emissions subject to this part. ATRRM's may include:
  (1) Feedstock modification.
  (2) Product reformulations.
  (3) Production system modifications.
  (4) System enclosure, emissions control, capture, or conversion.
  (5) Operational standards and practices modification.

(b) Airborne toxic risk reduction measures do not include measures that will increase risk from exposure to the chemical in another media or that increase the risk to workers or consumers.
(c) "Airborne toxic risk reduction audit and plan" or "audit and plan" means the audit and plan specified in Section 44392.

44391.
(a) Whenever a health risk assessment approved pursuant to Chapter 4 (commencing with Section 44360) indicates, in the judgment of the district, that there is a significant risk associated with the emissions from a facility, the facility operator shall conduct an airborne toxic risk reduction audit and develop a plan to implement airborne toxic risk reduction measures that will result in the reduction of emissions from the facility to a level below the significant risk level within five years of the date the plan is submitted to the district. The facility operator shall implement measures set forth in the plan in accordance with this chapter.
(b) The period to implement the plan required by subdivision (a) may be shortened by the district if it finds that it is technically feasible and economically practicable to implement the plan to reduce emissions below the significant risk level more quickly or if it finds that the emissions from the facility pose an unreasonable health risk.
(c) A district may lengthen the period to implement the plan required by subdivision (a) by up to an additional five years if it finds that a period longer than five years will not result in an unreasonable risk to public health and that requiring implementation of the plan within five years places an unreasonable economic burden on the facility operator or is not technically feasible.
(d)
(1) The state board and districts shall provide assistance to smaller businesses that have inadequate technical and financial resources for obtaining information, assessing risk reduction methods, and developing and applying risk reduction techniques.
(2) Risk reduction audits and plans for any industry subject to this chapter which is comprised mainly of small businesses using substantially similar technology may be completed by a self-conducted audit and checklist developed by the state board. The state board, in coordination with the districts, shall provide a copy of the audit and checklist to small businesses within those industries to assist them to meet the requirements of this chapter.
(e) The audit and plan shall contain all the information required by Section 44392.
(f) The plan shall be submitted to the district, within six months of a district's determination of significant risk, for review of completeness. Operators of facilities that have been notified prior to January 1, 1993, that there is a significant risk associated with emissions from the facility shall submit the plan by July 1, 1993. The district's review of completeness shall include a substantive analysis of the emission reduction measures included in the plan, and the ability

of those measures to achieve emission reduction goals as quickly as feasible as provided in subdivisions (a) and (b).

(g) The district shall find the audit and plan to be satisfactory within three months if it meets the requirements of this chapter, including, but not limited to, subdivision (f). If the district determines that the audit and plan does not meet those requirements, the district shall remand the audit and plan to the facility specifying the deficiencies identified by the district. A facility operator shall submit a revised audit and plan addressing the deficiencies identified by the district within 90 days of receipt of a deficiency notice.

(h) Progress on the emission reductions achieved by the plan shall be reported to the district in emissions inventory updates. Emissions inventory updates shall be prepared as required by the audit and plan found to be satisfactory by the district pursuant to subdivision (g).

(i) If new information becomes available after the initial risk reduction audit and plan, on air toxics risks posed by a facility, or emission reduction technologies that may be used by a facility that would significantly impact risks to exposed persons, the district may require the plan to be updated and resubmitted to the district.

(j) This section does not authorize the emission of a toxic air contaminant in violation of an airborne toxic control measure adopted pursuant to Chapter 3.5 (commencing with Section 39650) or in violation of Section 41700.

44392. A facility operator subject to this chapter shall conduct an airborne toxic risk reduction audit and develop a plan which shall include at a minimum all of the following:

(a) The name and location of the facility.

(b) The SIC code for the facility.

(c) The chemical name and the generic classification of the chemical.

(d) An evaluation of the ATRRM's available to the operator.

(e) The specification of, and rationale for, the ATRRMs that will be implemented by the operator. The audit and plan shall document the rationale for rejecting ATRRMs that are identified as infeasible or too costly.

(f) A schedule for implementing the ATRRMs. The schedule shall meet the time requirements of subdivision (a) of Section 44391 or the time period for implementing the plan set by the district pursuant to subdivision (b) or (c) of Section 44391, whichever is applicable.

(g) The audit and plan shall be reviewed and certified as meeting this chapter by an engineer who is registered as a professional engineer pursuant to Section 6762 of the Business and Professions Code, by an individual who is responsible for the processes and operations of the site, or by an environmental assessor registered pursuant to Section 25570.3.

44393. The plan prepared pursuant to Section 44391 shall not be considered to be the equivalent of a pollution prevention program or a source reduction program, except insofar as the audit and plan elements are consistent with source reduction, as defined in Section 25244.14, or subsequent statutory definitions of pollution prevention.

44394. Any facility operator who does not submit a complete airborne toxic risk reduction audit and plan or fails to implement the measures set forth in the plan as set forth in this chapter is subject to the civil penalty specified in subdivision (a) of Section 44381, and any facility operator who, in connection with the audit or plan, knowingly submits any false statement or representation is subject to the civil penalty specified in subdivision (b) of Section 44381.

B.3. Toxic Air Contaminants Program Overview

(Air resources Board, 2011: see http://www.arb.ca.gov/toxics/background.htm)

AB 1807 Program

In 1983, the California Legislature established a two-step process of risk identification and risk management to address the potential health effects from air toxic substances and protect the public health of Californians. During the first step (identification), the ARB and the Office of Environmental Health Hazard Assessment (OEHHA) determines if a substance should be formally identified as a toxic air contaminant (TAC) in California. During this process, the ARB and the OEHHA staff draft a report that serves as the basis for this determination. The ARB staff assesses the potential for human exposure to a substance and the OEHHA staff evaluates the health effects. A thorough public process assures accountability and public input. Public workshops are conducted to allow for direct exchanges of information with interested constituencies. The draft risk assessments themselves are published and widely distributed with a public notice requesting comment to further assure involvement. The final risk assessment (identification) report includes a record of the public comments and how they were addressed. After the ARB and the OEHHA staff hold several comment periods and workshops, the report is then submitted to an independent, nine member, Scientific Review Panel (SRP), who review the report for its scientific accuracy. If the SRP approves the report, they develop specific scientific findings which are officially submitted to the ARB. The ARB staff then prepares a hearing notice and draft regulation to formally identify the substance as a TAC. Based on the input from the public and the information gathered from the report, the Board will decide whether to identify a substance as a TAC. Any person may petition the Board to review a previous determination by providing new evidence.

In the second step (risk management), the ARB reviews the emission sources of an identified TAC to determine if any regulatory action is necessary to reduce the risk. The analysis includes a review of controls already in place, the available technologies and associated costs for reducing emissions, and the associated risk. Public outreach is an essential element in the development of a control plan and any control measure to ensure that the ARB efforts are cost-effective and appropriately balance public health protection and economic growth.

In 1993, the California Legislature amended the AB 1807 program for the identification and control of TACs (AB 2728). Specifically, AB 2728 required the ARB to identify the 189 federal hazardous air pollutants as TACs. For those substances that have not previously been identified under AB 1807 and identified under AB 2728, health effects values will need to be developed. This report will serve as a basis for that evaluation. For substances that were not identified as TACs and are on the TAC Identification List, this report will provide information to evaluate which substances may be entered into the air toxics identification process.

B.4. Senate Bill 352. Schoolsites: sources of pollution

CHAPTER 668

FILED WITH SECRETARY OF STATE OCTOBER 3, 2003
APPROVED BY GOVERNOR OCTOBER 2, 2003
PASSED THE SENATE SEPTEMBER 11, 2003
PASSED THE ASSEMBLY SEPTEMBER 8, 2003
AMENDED IN ASSEMBLY SEPTEMBER 4, 2003
AMENDED IN ASSEMBLY AUGUST 18, 2003
AMENDED IN ASSEMBLY JULY 16, 2003
AMENDED IN SENATE JUNE 3, 2003
AMENDED IN SENATE MAY 19, 2003
AMENDED IN SENATE MAY 8, 2003
AMENDED IN SENATE MARCH 24, 2003

INTRODUCED BY Senator Escutia

FEBRUARY 19, 2003

An act to amend Section 17213 of the Education Code, and to amend Section 21151.8 of the Public Resources Code, relating to public schools.

LEGISLATIVE COUNSEL'S DIGEST

SB 352, Escutia. Schoolsites: sources of pollution.

Existing law sets forth various requirements regarding the siting, structural integrity, safety, and fitness-for-occupancy of school buildings, including, but not limited to, a prohibition of the approval by the governing board of a school district of the acquisition of a schoolsite by a school district, unless prescribed conditions relating to possible exposure to hazardous substances are satisfied, and a prohibition on the approval of a related environmental impact report or negative declaration.

This bill would, in addition, prohibit the approval by the governing board of a school district of a schoolsite that is within 500 feet from the edge of the closest traffic lane of a freeway or other busy traffic corridor, unless prescribed conditions are met and would make conforming and other technical, nonsubstantive changes.

Existing law requires the lead agency to consult with prescribed agencies to identify facilities that might reasonably be anticipated to emit hazardous materials, within 1/4 of a mile of the schoolsite.

This bill would define "facility" for this purpose and would require the lead agency to consult to identify freeways and other busy traffic corridors, as defined, large agricultural operations, and railyards, within 1/4 of a mile of the schoolsite, and would make conforming and other technical, nonsubstantive changes.

THE PEOPLE OF THE STATE OF CALIFORNIA DO ENACT AS FOLLOWS:

SECTION 1.
The Legislature finds and declares all of the following:

(a) Many studies have shown significantly increased levels of pollutants, particularly diesel particulates, in close proximity to freeways and other major diesel sources. A recent study of Los Angeles area freeways measured diesel particulate levels up to 25 times higher near freeways than those levels elsewhere. Much of the pollution from freeways is associated with acute health effects, exacerbating asthma and negatively impacting the ability of children to learn.
(b) Cars and trucks release at least forty different toxic air contaminants, including, but not limited to, diesel particulate, benzene, formaldehyde, 1,3-butadiene and acetaldehyde. Levels of these pollutants are generally concentrated within 500 feet of freeways and very busy roadways.
(c) Current state law governing the siting of schools does not specify whether busy freeways should be included in environmental impact reports of nearby "facilities." Over 150 schools are already estimated to be within 500 feet of extremely high traffic roadways.
(d) A disproportionate number of economically disadvantaged pupils may be attending schools that are close to busy roads, putting them at an increased risk of developing bronchitis from elevated levels of several pollutants associated with traffic. Many studies have confirmed that increased wheezing and bronchitis occurs among children living in high traffic areas.
(e) It is therefore the intent of the Legislature to protect school children from the health risks posed by pollution from heavy freeway traffic and other nonstationary sources in the same way that they are protected from industrial pollution.

SECTION 2.

Section 17213 of the Education Code is amended to read:
17213. The governing board of a school district may not approve a project involving the acquisition of a schoolsite by a school district, unless all of the following occur:
(a) The school district, as the lead agency, as defined in Section 21067 of the Public Resources Code, determines that the property purchased or to be built upon is not any of the following:
(1) The site of a current or former hazardous waste disposal site or solid waste disposal site, unless if the site was a former solid waste disposal site, the governing board of the school district concludes that the wastes have been removed.
(2) A hazardous substance release site identified by the Department of Toxic Substances Control in a current list adopted pursuant to Section 25356 of the Health and Safety Code for removal or remedial action pursuant to Chapter 6.8 (commencing with Section 25300) of Division 20 of the Health and Safety Code.

(3) A site that contains one or more pipelines, situated underground or aboveground, that carries hazardous substances, acutely hazardous materials, or hazardous wastes, unless the pipeline is a natural gas line that is used only to supply natural gas to that school or neighborhood.

(b) The school district, as the lead agency, as defined in Section 21067 of the Public Resources Code, in preparing the environmental impact report or negative declaration has consulted with the administering agency in which the proposed schoolsite is located, pursuant to Section 2735.3 of Title 19 of the California Code of Regulations, and with any air pollution control district or air quality management district having jurisdiction in the area, to identify both permitted and nonpermitted facilities within that district's authority, including, but not limited to, freeways and other busy traffic corridors, large agricultural operations, and railyards, within one-fourth of a mile of the proposed schoolsite, that might reasonably be anticipated to emit hazardous air emissions, or to handle hazardous or acutely hazardous materials, substances, or waste. The school district, as the lead agency, shall include a list of the locations for which information is sought.

(c) The governing board of the school district makes one of the following written findings:

(1) Consultation identified none of the facilities or significant pollution sources specified in subdivision (b).

(2) The facilities or other pollution sources specified in subdivision (b) exist, but one of the following conditions applies:

(A) The health risks from the facilities or other pollution sources do not and will not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the school.

(B) The governing board finds that corrective measures required under an existing order by another governmental entity that has jurisdiction over the facilities or other pollution sources will, before the school is occupied, result in the mitigation of all chronic or accidental hazardous air emissions to levels that do not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school. If the governing board makes this finding, the governing board shall also make a subsequent finding, prior to the occupancy of the school, that the emissions have been mitigated to these levels.

(C) For a schoolsite with a boundary that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor, the governing board of the school district determines, through analysis pursuant to paragraph (2) of subdivision (b) of Section 44360 of the Health and Safety Code, based on appropriate air dispersion modeling, and after considering any potential mitigation measures, that the air quality at the proposed site is such that neither short-term nor long-term exposure poses significant health risks to pupils.

(D) The governing board finds that neither of the conditions set forth in subparagraph (B) or (C) can be met, and the school district is unable to locate an alternative site that is suitable due to a severe shortage of sites

that meet the requirements in subdivision (a) of Section 17213. If the governing board makes this finding, the governing board shall adopt a statement of Overriding Considerations pursuant to Section 15093 of Title 14 of the California Code of Regulations.

(d) As used in this section:

(1) "Hazardous air emissions" means emissions into the ambient air of air contaminants that have been identified as a toxic air contaminant by the State Air Resources Board or by the air pollution control officer for the jurisdiction in which the project is located. As determined by the air pollution control officer, hazardous air emissions also means emissions into the ambient air from any substance identified in subdivisions (a) to (f), inclusive, of Section 44321 of the Health and Safety Code.

(2) "Hazardous substance" means any substance defined in Section 25316 of the Health and Safety Code.

(3) "Acutely hazardous material" means any material defined pursuant to subdivision (a) of Section 25532 of the Health and Safety Code.

(4) "Hazardous waste" means any waste defined in Section 25117 of the Health and Safety Code.

(5) "Hazardous waste disposal site" means any site defined in Section 25114 of the Health and Safety Code.

(6) "Administering agency" means any agency designated pursuant to Section 25502 of the Health and Safety Code.

(7) "Handle" means handle as defined in Article 1 (commencing with Section 25500) of Chapter 6.95 of Division 20 of the Health and Safety Code.

(8) "Facilities" means any source with a potential to use, generate, emit or discharge hazardous air pollutants, including, but not limited to, pollutants that meet the definition of a hazardous substance, and whose process or operation is identified as an emission source pursuant to the most recent list of source categories published by the California Air Resources Board.

(9) "Freeway or other busy traffic corridors" means those roadways that, on an average day, have traffic in excess of 50,000 vehicles in a rural area as defined in Section 50101 of the Health and Safety Code, and 100,000 vehicles in an urban area, as defined in Section 50104.7 of the Health and Safety Code.

SECTION 3.

Section 21151.8 of the Public Resources Code is amended to read:

21151.8.

(a) An environmental impact report or negative declaration may not be approved for any project involving the purchase of a schoolsite or the construction of a new elementary or secondary school by a school district unless all of the following occur:

(1) The environmental impact report or negative declaration includes information that is needed to determine if the property proposed to be purchased, or to be constructed upon, is any of the following:

(A) The site of a current or former hazardous waste disposal site or solid waste disposal site and, if so, whether the wastes have been removed.

(B) A hazardous substance release site identified by the Department of Toxic Substances Control in a current list adopted pursuant to Section 25356 of the Health and Safety Code for removal or remedial action pursuant to Chapter 6.8 (commencing with Section 25300) of Division 20 of the Health and Safety Code.

(C) A site that contains one or more pipelines, situated underground or aboveground, that carries hazardous substances, acutely hazardous materials, or hazardous wastes, unless the pipeline is a natural gas line that is used only to supply natural gas to that school or neighborhood, or other nearby schools.

(D) A site that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor.

(2) The school district, as the lead agency, in preparing the environmental impact report or negative declaration has notified in writing and consulted with the administering agency in which the proposed schoolsite is located, pursuant to Section 2735.3 of Title 19 of the California Code of Regulations, and with any air pollution control district or air quality management district having jurisdiction in the area, to identify both permitted and nonpermitted facilities within that district's authority, including, but not limited to, freeways and busy traffic corridors, large agricultural operations, and railyards, within one-fourth of a mile of the proposed schoolsite, that might reasonably be anticipated to emit hazardous emissions or handle hazardous or acutely hazardous materials, substances, or waste. The notification by the school district, as the lead agency, shall include a list of the locations for which information is sought.

(3) The governing board of the school district makes one of the following written findings:

(A) Consultation identified no facilities of this type or other significant pollution sources specified in paragraph (2).

(B) The facilities or other pollution sources specified in paragraph (2) exist, but one of the following conditions applies:

(i) The health risks from the facilities or other pollution sources do not and will not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school.

(ii) Corrective measures required under an existing order by another agency having jurisdiction over the facilities or other pollution sources will, before the school is occupied, result in the mitigation of all chronic or accidental hazardous air emissions to levels that do not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school. If the governing board makes a finding pursuant to this clause, it shall also make a subsequent finding, prior to occupancy of the school, that the emissions have been so mitigated.

(iii) For a schoolsite with a boundary that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor, the governing board of the school district determines, through analysis pursuant to paragraph (2) of subdivision (b) of Section 44360 of the Health and Safety Code, based on appropriate air dispersion modeling, and after considering any potential mitigation measures, that the air quality at the proposed site is such that neither short-term nor long-term exposure poses significant health risks to pupils.

(C) The facilities or other pollution sources specified in paragraph (2) exist, but conditions in clause (i), (ii) or (iii) of subparagraph (B) cannot be met, and the school district is unable to locate an alternative site that is suitable due to a severe shortage of sites that meet the requirements in subdivision (a) of Section 17213 of the Education Code. If the governing board makes this finding, the governing board shall adopt a statement of Overriding Considerations pursuant to Section 15093 of Title 14 of the California Code of Regulations.

(4) Each administering agency, air pollution control district, or air quality management district receiving written notification from a lead agency to identify facilities pursuant to paragraph (2) shall provide the requested information and provide a written response to the lead agency within 30 days of receiving the notification. The environmental impact report or negative declaration shall be conclusively presumed to comply with this section as to the area of responsibility of any agency that does not respond within 30 days.

(b) If a school district, as a lead agency, has carried out the consultation required by paragraph (2) of subdivision (a), the environmental impact report or the negative declaration shall be conclusively presumed to comply with this section, notwithstanding any failure of the consultation to identify an existing facility or other pollution source specified in paragraph (2) of subdivision (a).

(c) As used in this section and Section 21151.4, the following definitions shall apply:

(1) "Hazardous substance" means any substance defined in Section 25316 of the Health and Safety Code.

(2) "Acutely hazardous material" means any material defined pursuant to subdivision (a) of Section 25532 of the Health and Safety Code.

(3) "Hazardous waste" means any waste defined in Section 25117 of the Health and Safety Code.

(4) "Hazardous waste disposal site" means any site defined in Section 25114 of the Health and Safety Code.

(5) "Hazardous air emissions" means emissions into the ambient air of air contaminants that have been identified as a toxic air contaminant by the State Air Resources Board or by the air pollution control officer for the jurisdiction in which the project is located. As determined by the air pollution control officer, hazardous air emissions also means emissions into the ambient air from any substances identified in subdivisions (a) to (f), inclusive, of Section 44321 of the Health and Safety Code.

(6) "Administering agency" means an agency designated pursuant to Section 25502 of the Health and Safety Code.

(7) "Handle" means handle as defined in Article 1 (commencing with Section 25500) of Chapter 6.95 of Division 20 of the Health and Safety Code.
(8) "Facilities" means any source with a potential to use, generate, emit or discharge hazardous air pollutants, including, but not limited to, pollutants that meet the definition of a hazardous substance, and whose process or operation is identified as an emission source pursuant to the most recent list of source categories published by the California Air Resources Board.
(9) "Freeway or other busy traffic corridors" means those roadways that, on an average day, have traffic in excess of 50,000 vehicles in a rural area, as defined in Section 50101 of the Health and Safety Code, and 100,000 vehicles in an urban area, as defined in Section 50104.7 of the Health and Safety Code.

B.5. Senate Bill 25, Children's Environmental Health Protection.

CHAPTER 731
FILED WITH SECRETARY OF STATE OCTOBER 10, 1999
APPROVED BY GOVERNOR OCTOBER 7, 1999
PASSED THE SENATE SEPTEMBER 8, 1999
PASSED THE ASSEMBLY SEPTEMBER 7, 1999
AMENDED IN ASSEMBLY SEPTEMBER 2, 1999
AMENDED IN ASSEMBLY AUGUST 16, 1999
AMENDED IN ASSEMBLY JULY 8, 1999
AMENDED IN SENATE JUNE 1, 1999
AMENDED IN SENATE APRIL 28, 1999
AMENDED IN SENATE MARCH 22, 1999
INTRODUCED BY Senator Escutia
(Principal coauthors: Assembly Members Kuehl and Villaraigosa)
(Coauthors: Senators Alarcon, Figueroa, Ortiz, Perata, Polanco, Sher, Solis, and Speier)
(Coauthors: Assembly Members Alquist, Aroner, Firebaugh, Honda, Jackson, Knox, Lempert, Mazzoni, Romero, Shelley, Steinberg, Thomson, Vincent, Washington, and Wildman)
DECEMBER 7, 1998
An act to amend Sections 39606, 39660, and 40451 of, to add Section 39617.5 to, to add Part 3 (commencing with Section 900) to Division 1 of, and to add Article 4.5 (commencing with Section 39669.5) to Chapter 3.5 of Part 2 of Division 26 of, the Health and Safety Code, relating to environmental health protection.

LEGISLATIVE COUNSEL'S DIGEST

SB 25, Escutia. Environmental health protection: children.

(1) Existing law requires the State Air Resources Board to adopt ambient air quality standards in consideration of specified factors, including public health effects, as provided, and to specify threshold levels for health effects in listing substances determined to be toxic air contaminants. Existing law requires the Office of Environmental Health Hazard Assessment, upon request of the state board, to evaluate the health effects of and prepare recommendations regarding specified substances which may be or are emitted into the ambient air and that may be determined to be toxic air contaminants. Under existing law, the state board's request is required to be in accordance with an agreement that ensures that the office's workload in implementing these provisions will not be increased over that budgeted for the 1991-92 fiscal year, as provided.

This bill would eliminate the requirement for that agreement, and would impose specified requirements on the state board and the office generally relating to the protection of infants and children from environmental health hazards. The bill would require the state board, not later than December 31, 2000, to review all existing

health-based ambient air quality standards to determine whether the standards adequately protect the health of the public, including infants and children, and to revise the highest priority air quality standard determined to be inadequate, not later than December 31, 2002. The bill would require the office, by July 1, 2001, to establish a list of up to 5 specified toxic air contaminants that may cause infants and children to be especially susceptible to illness. The bill would require the state board to review and, as appropriate, revise any control measures adopted for those toxic air contaminants, to reduce exposure to those toxic air contaminants, as provided.

(2) Existing law requires the South Coast Air Quality Management District to notify all schools in the South Coast Air Basin whenever any federal primary ambient air quality standard is predicted to be exceeded. This bill would also require the south coast district to notify day care centers in that basin, to the extent feasible and upon request. The bill would create a state-mandated local program by imposing new duties on the south coast district.

(3) The bill would create the Children's Environmental Health Center within the Environmental Protection Agency to, among other things, serve as chief advisor to the Secretary for Environmental Protection and to the Governor on matters within the jurisdiction of the agency relating to environmental health and environmental protection as it relates to children.

(4) This bill would incorporate additional changes to Section 40451 of the Health and Safety Code, proposed by SB 1195, to be operative only if SB 1195 and this bill are both chaptered on or before January 1, 2000, and this bill is chaptered last. (5) The California Constitution requires the state to reimburse local agencies and school districts for certain costs mandated by the state. Statutory provisions establish procedures for making that reimbursement, including the creation of a State Mandates Claims Fund to pay the costs of mandates that do not exceed $1,000,000 statewide and other procedures for claims whose statewide costs exceed $1,000,000.

This bill would provide that, if the Commission on State Mandates determines that the bill contains costs mandated by the state, reimbursement for those costs shall be made pursuant to these statutory provisions.

THE PEOPLE OF THE STATE OF CALIFORNIA DO ENACT AS FOLLOWS:

SECTION 1.
The Legislature finds and declares all of the following:

(a) Infants and children have a higher ventilation rate than adults relative to their body weight and lung surface area, resulting in a greater dose of pollution delivered to their lungs.

(b) Children have narrower airways than adults. Thus, irritation or inflammation caused by air pollution that would produce only a slight response in an adult can result in a potentially significant obstruction of the airway in a young child.

(c) Children spend significantly more time outdoors, especially in the summer, when ozone air pollution levels are typically highest. National statistics show that children spend an average of 50 percent more time outdoors than adults.
(d) Air pollution is known to exacerbate asthma and be a trigger for asthma attacks in infants and children, 500,000 of whom are afflicted with this chronic lung disease in California.
(e) Infant's and children's developing organs and tissues are more susceptible to damage from some environmental contaminants than are adult organs and tissues.
(f) It is the intent of the Legislature in enacting this act, to require that the state's air quality standards and airborne toxic control measures be reviewed to determine if they adequately protect the health of infants and children, and that these standards and measures be revised if they are determined to be inadequate.
(g) It is also the intent of the Legislature in enacting this act to require the State Air Resources Board and the Office of Environmental Health Hazard Assessment to consider the health impacts to all populations of children, including special subpopulations of infants and children that comprise a meaningful portion of the general population, such as children with asthma, cystic fibrosis, or other respiratory conditions or diseases, in setting or revising standards pursuant to this act.

SECTION 2.
Part 3 (commencing with Section 900) is added to Division 1 of the Health and Safety Code, to read:
PART 3. CHILDREN'S ENVIRONMENTAL HEALTH CENTER 900. There is hereby created the Children's Environmental Health Center within the Environmental Protection Agency. The primary purposes of the center shall include all of the following:
(a) To serve as the chief advisor to the Secretary for Environmental Protection and to the Governor on matters within the jurisdiction of the Environmental Protection Agency relating to environmental health and environmental protection as each of those matters relates to children.
(b) To assist the boards, departments, and offices within the Environmental Protection Agency to assess the effectiveness of statutes, regulations, and programs designed to protect children from environmental hazards.
(c) To coordinate within the Environmental Protection Agency and with other state agencies, regulatory efforts, research and data collection, and other programs and services that impact the environmental health of children, and coordinate with appropriate federal agencies conducting related regulatory efforts and research and data collection.
(d) In consultation with the State Air Resources Board and the Office of Environmental Health Hazard Assessment, and notwithstanding Section 7550.5 of the Government Code, to report to the Legislature and the Governor no later than December 31, 2001, on the progress of the state board and the office toward implementing the act that added this part during the 1999-2000 Regular Session and to make recommendations for any statutory or regulatory changes that may be necessary to carry out the intent of that act to protect the public

health, including infants and children, from air pollutants and toxic air contaminants.

SECTION 3.
Section 39606 of the Health and Safety Code is amended to read:
39606.

(a) The state board shall do both of the following:
  (1) Based upon similar meteorological and geographic conditions and consideration for political boundary lines whenever practicable,divide the state into air basins to fulfill the purposes of this division.
  (2) Adopt standards of ambient air quality for each air basin in consideration of the public health, safety, and welfare, including,but not limited to, health, illness, irritation to the senses, aesthetic value, interference with visibility, and effects on the economy. These standards may vary from one air basin to another. Standards relating to health effects shall be based upon the recommendations of the Office of Environmental Health Hazard Assessment.

(b) In its recommendations for submission to the state board pursuant to paragraph (2) of subdivision (a), the Office of Environmental Health Hazard Assessment, to the extent that information is available, shall assess the following:
  (1) Exposure patterns, including, but not limited to, patterns determined by relevant data supplied by the state board, among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.
  (2) Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.
  (3) The effects on infants and children of exposure to ambient air pollutants and other substances that have a common mechanism of toxicity.
  (4) The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

(c) In assessing the factors specified in subdivision (b), the office shall use current principles, practices, and methods used by public health professionals who are experienced practitioners in the field of human health effects assessment. The scientific basis or scientific portion of the method used by the office to assess the factors set forth in subdivision (b) shall be subject to peer review as described in Section 57004 or in a manner consistent with the peer review requirements of Section 57004. Any person may submit any information for consideration by the entity conducting the peer review, which may receive oral testimony.

(d)
  (1) No later than December 31, 2000, the state board in consultation with the office, shall review all existing health-based ambient air quality standards to determine whether, based on public health, scientific literature, and exposure pattern data, the standards adequately protect the health of the public, including infants and children, with an adequate margin of safety. The state board shall publish a report summarizing these findings.
  (2) The state board shall revise the highest priority ambient air quality standard determined to be inadequate to protect infants and children with an adequate

margin of safety, based on its report, no later than December 31, 2002. Following the revision of the highest priority standard, the state board shall revise any additional standards determined to be inadequate to protect infants and children with an adequate margin of safety, at the rate of at least one per year. The standards shall be established at levels that adequately protect the health of the public, including infants and children, with an adequate margin of safety (e) Nothing in this section shall restrict the authority of the state board to consider additional information in establishing ambient air quality standards or to adopt an ambient air quality standard designed to protect vulnerable populations other than infants and children.

SECTION 4.
Section 39617.5 is added to the Health and Safety Code, to read:
39617.5.

(a) Not later than January 1, 2003, the state board shall do all of the following:
  (1) Evaluate the adequacy of the current monitoring network for its ability to gather the data necessary to determine the exposure of infants and children to air pollutants including criteria air pollutants and toxic air contaminants.
  (2) Identify areas where the exposure of infants and children to air pollutants is not adequately measured by the current monitoring network.
  (3) Recommend changes to improve air pollution monitoring networks and data collection to more accurately reflect the exposure of infants and children to air pollutants.

(b) In carrying out this section, the state board, in cooperation with the districts, shall expand its existing monitoring program in six communities around the state in nonattainment areas, as selected by the state board, to include special monitoring of children's exposure to air pollutants and toxic contaminants. The expanded program shall include placing air pollution monitors near schools, day care centers, and outdoor recreational facilities that are in close proximity to, or downwind from, major industrial sources of air pollutants and toxic air contaminants, including, freeways and major traffic areas. The purpose of the air pollution monitors shall be to conduct sampling of air pollution levels affecting children. Monitoring may include the use of fixed, mobile, and other monitoring devices, as appropriate.

(c) The expanded monitoring program shall include the following:
  (1) Monitoring during multiple seasons and at multiple locations within each community at schools, day care centers, recreational facilities, and other locations where children spend most of their time.
  (2) A combination of upgrading existing fixed monitoring sites, establishing new fixed monitoring sites, and conducting indoor and outdoor sampling and personal exposure measurements in each community to provide the most comprehensive data possible on the levels of children's exposure to air pollutants and toxic air contaminants.

(d) Data collected from expanded air quality monitoring activities conducted pursuant to this section may be used for any purpose authorized by law, including, but not limited to, determinations as to whether an area has attained or has not attained

the state and national ambient air quality standards, if the monitoring devices from which the data was collected meet the monitoring requirements specified in Section 58.14 of Title 40 of the Code of Federal Regulations for special purpose monitors, all other monitoring requirements of Part 58 of Title 40 of the Code of Federal Regulations, and all applicable requirements specified in regulations adopted by the state board.

SECTION 5.
Section 39660 of the Health and Safety Code is amended to read:
39660.

(a) Upon the request of the state board, the office, in consultation with and with the participation of the state board, shall evaluate the health effects of and prepare recommendations regarding substances, other than pesticides in their pesticidal use, which may be or are emitted into the ambient air of California and that may be determined to be toxic air contaminants.

(b) In conducting this evaluation, the office shall consider all available scientific data, including, but not limited to, relevant data provided by the state board, the State Department of Health Services, the Occupational Safety and Health Division of the Department of Industrial Relations, the Department of Pesticide Regulation, international and federal health agencies, private industry, academic researchers, and public health and environmental organizations. The evaluation shall be performed using current principles, practices, and methods used by public health professionals who are experienced practitioners in the fields of epidemiology, human health effects assessment, risk assessment, and toxicity.

(c)

(1) The evaluation shall assess the availability and quality of data on health effects, including potency, mode of action, and other relevant biological factors, of the substance, and shall, to the extent that information is available, assess all of the following:

(A) Exposure patterns among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.

(B) Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.

(C) The effects on infants and children of exposure to toxic air contaminants and other substances that have a common mechanism of toxicity.

(D) The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

(2) The evaluation shall also contain an estimate of the levels of exposure that may cause or contribute to adverse health effects. If it can be established that a threshold of adverse health effects exists, the estimate shall include both of the following factors:

(A) The exposure level below which no adverse health effects are anticipated.

(B) An ample margin of safety that accounts for the variable effects that heterogeneous human populations exposed to the substance under evaluation may experience, the uncertainties associated with the

applicability of the data to human beings, and the completeness and quality of the information available on potential human exposure to the substance. In cases in which there is no threshold of significant adverse health effects, the office shall determine the range of risk to humans resulting from current or anticipated exposure to the substance.

(3) The scientific basis or scientific portion of the method used by the office to assess the factors set forth in this subdivision shall be reviewed in a manner consistent with this chapter by the Scientific Review Panel on Toxic Air Contaminants established pursuant to Article 5 (commencing with Section 39670). Any person may submit any information for consideration by the panel, which may receive oral testimony.

(d) The office shall submit its written evaluation and recommendations to the state board within 90 days after receiving the request of the state board pursuant to subdivision (a). The office may, however, petition the state board for an extension of the deadline, not to exceed 30 days, setting forth its statement of the reasons that prevent the office from completing its evaluation and recommendations within 90 days. Upon receipt of a request for extension of, or noncompliance with, the deadline contained in this section, the state board shall immediately transmit to the Assembly Committee on Rules and the Senate Committee on Rules, for transmittal to the appropriate standing, select, or joint committee of the Legislature, a statement of reasons for extension of the deadline, along with copies of the office's statement of reasons that prevent it from completing its evaluation and recommendations in a timely manner.

(e)

(1) The state board or a district may request, and any person shall provide, information on any substance that is or may be under evaluation and that is manufactured, distributed, emitted, or used by the person of whom the request is made, in order to carry out its responsibilities pursuant to this chapter. To the extent practical, the state board or a district may collect the information in aggregate form or in any other manner designed to protect trade secrets.

(2) Any person providing information pursuant to this subdivision may, at the time of submission, identify a portion of the information submitted to the state board or a district as a trade secret and shall support the claim of a trade secret, upon the written request of the state board or district board. Subject to Section 1060 of the Evidence Code, information supplied that is a trade secret, as specified in Section 6254.7 of the Government Code, and that is so marked at the time of submission, shall not be released to any member of the public. This section does not prohibit the exchange of properly designated trade secrets between public agencies when those trade secrets are relevant and necessary to the exercise of their jurisdiction if the public agencies exchanging those trade secrets preserve the protections afforded that information by this paragraph.

(3) Any information not identified as a trade secret shall be available to the public unless exempted from disclosure by other provisions of law. The fact that information is claimed to be a trade secret is public information. Upon receipt

of a request for the release of information that has been claimed to be a trade secret, the state board or district shall immediately notify the person who submitted the information, and shall determine whether or not the information claimed to be a trade secret is to be released to the public. The state board or district board, as the case may be, shall make its determination within 60 days after receiving the request for disclosure, but not before 30 days following the notification of the person who submitted the information. If the state board or district decides to make the information public, it shall provide the person who submitted the information 10 days' notice prior to public disclosure of the information.

(f) The office and the state board shall give priority to the evaluation and regulation of substances based on factors related to the risk of harm to public health, amount or potential amount of emissions, manner of, and exposure to, usage of the substance in California, persistence in the atmosphere, and ambient concentrations in the community. In determining the importance of these factors, the office and the state board shall consider all of the following information, to the extent that it is available:

(1) Research and monitoring data collected by the state board and the districts pursuant to Sections 39607, 39617.5, 39701, and 40715, and by the United States Environmental Protection Agency pursuant to paragraph (2) of subsection (k) of Section 112 of the federal act (42 U.S.C. Sec. 7412(k)(2)).

(2) Emissions inventory data reported for substances subject to Part 6 (commencing with Section 44300) and the risk assessments prepared for those substances.

(3) Toxic chemical release data reported to the state emergency response commission pursuant to Section 313 of the Emergency Planning and Community Right-To-Know Act of 1986 (42 U.S.C. Sec. 11023) and Section 6607 of the Pollution Prevention Act of 1990 (42 U.S.C. Sec. 13106).

(4) Information on estimated actual exposures to substances based on geographic and demographic data and on data derived from analytical methods that measure the dispersion and concentrations of substances in ambient air.

SECTION 6.

Article 4.5 (commencing with Section 39669.5) is added to Chapter 3.5 of Part 2 of Division 26 of the Health and Safety Code, to read:

Article 4.5. Special Provisions For Infants And Children

39669.5. The Legislature finds and declares that certain toxic air contaminants may pose risks that cause infants and children to be especially susceptible to illness and that certain actions are necessary to ensure their safety from toxic air contaminants.

(a) By July 1, 2001, the following shall occur

(1) The office, in consultation with the state board, shall establish a list of up to five toxic air contaminants identified or designated by the state board pursuant to Section 39657 that may cause infants and children to be especially susceptible to illness. In developing the list, the office shall take into account public exposures to toxic air contaminants, whether by

themselves or interacting with other toxic air contaminants or criteria pollutants, and the factors listed in subdivision (c) of Section 39660. The office shall submit a report containing the list and its reasons for including the toxic air contaminants on the list to the Scientific Review Panel on Toxic Air Contaminants established pursuant to Article 5 (commencing with Section 39670).

(2) The scientific review panel, in a manner consistent with this chapter, shall review the list of toxic air contaminants submitted by the office pursuant to paragraph (1). As part of the review, any person may submit any information for consideration by the panel, which may receive oral testimony.

(b)

(1) Within two years of the establishment of the list required pursuant to subdivision (a), the state board shall review and, as appropriate, revise any control measures adopted for the toxic air contaminants identified on the list, to reduce exposure to those toxic air contaminants pursuant to Article 4 (commencing with Section 39665), to protect public health, and particularly infants and children.

(2) Within three years of the establishment of the list required pursuant to subdivision (a), for up to five of those toxic air contaminants for which no control measures have been previously adopted, the state board shall prepare a report on the need for regulations, following the procedure specified in Section 39665. The state board shall adopt within that same three-year timeframe, as appropriate, any new control measures to reduce exposure to those toxic air contaminants pursuant to Article 4 (commencing with Section 39665), to protect public health, particularly infants and children.

(c) Beginning July 1, 2004, the office shall annually evaluate at least 15 toxic air contaminants identified or designated by the state board pursuant to Section 39657, and provide threshold exposure levels and nonthreshold health values, as appropriate, for those toxic air contaminants. The activities required pursuant to this subdivision shall continue until all toxic air contaminants are evaluated. The levels shall be established pursuant to the procedures adopted for health and risk assessments pursuant to paragraph (2) of subdivision (b) of Section 44360, and taking into account the factors listed in subdivision (c) of Section 39660. Based on this evaluation, and after review by the scientific review panel as prescribed in paragraph (2) of subdivision (a), the office shall update the list established pursuant to subdivision (a), by July 1, 2005, and each year thereafter. Within three years of the initial or subsequent listing update, for up to five of the toxic air contaminants contained on that list for which no control measures have been previously adopted, or for at least five of the toxic air contaminants if more than five toxic air contaminants have been identified, the state board shall prepare a report on the need for regulation, following the procedure specified in Section 39665. The state board shall adopt within that three-year timeframe, as appropriate, new control measures, pursuant to Article 4 (commencing with Section 39665), to reduce exposure to those toxic air contaminants, to protect public health, and particularly infants and children.

(d) Toxic air contaminants evaluated and listed pursuant to this section shall not include substances in those uses that are not subject to regulation by the state board pursuant to this chapter.

SECTION 7.
Section 40451 of the Health and Safety Code is amended to read:
40451.

(a) The south coast district shall use the Pollutant Standards Index developed by the Environmental Protection Agency and shall report and forecast pollutant levels daily for dissemination in the print and electronic media.
(b) Using existing communication facilities available to it, the south coast district shall notify all schools and, to the extent feasible and upon request, daycare centers in the South Coast Air Basin whenever any federal primary ambient air quality standard is predicted to be exceeded.
(c) Whenever it becomes available, the south coast district shall disseminate to schools, amateur adult and youth athletic organizations, and all public agencies operating parks and recreational facilities in the south coast district the latest scientific information and evidence regarding the need to restrict exercise and other outdoor activities during periods when federal primary air quality standards are exceeded.
(d) Once every two months and annually, the south coast district shall report on the number of days and locations that federal and state ambient air quality standards were exceeded and the number of days and locations of these occurrences.

SECTION 7.5.
Section 40451 of the Health and Safety Code is amended to read:
40451.

(a) The south coast district shall use the Pollutant Standards Index developed by the United States Environmental Protection Agency and shall report and forecast pollutant levels daily for dissemination in the print and electronic media. Commencing July 1, 2001, the south coast district shall also include in its report and forecast levels of PM2.5 in excess of the 24-hour federal ambient air standard, as adopted in July 1997, or any standard adopted by the United States Environmental Protection Agency that succeeds that standard.
(b) Using existing communication facilities available to it, the south coast district shall notify all schools and, to the extent feasible and upon request, daycare centers in the South Coast Air Basin whenever any federal primary ambient air quality standard is predicted to be exceeded. Commencing July 1, 2001, using communication facilities available to it, the south coast district shall also notify all schools in the South Coast Air Basin when the ambient level of PM2.5 is predicted to exceed the 24-hour federal ambient air standard, as adopted in July 1997, or any standard adopted by the United States Environmental Protection Agency that succeeds that standard.
(c) Whenever it becomes available, the south coast district shall disseminate to schools, amateur adult and youth athletic organizations, and all public agencies operating parks and recreational facilities in the south coast district the latest

scientific information and evidence regarding the need to restrict exercise and other outdoor activities during periods when federal primary air quality standards and the 24-hour federal ambient air standard for PM2.5, as adopted in July 1997, or any standards adopted by the United States Environmental Protection Agency that succeed those standards, are exceeded.

(d) Once every two months and annually, the south coast district shall report on the number of days and locations that federal and state ambient air quality standards were exceeded. Commencing July 1, 2001, the south coast district shall also include in that report the number of days and locations on and at which the 24-hour federal ambient air standard for PM2.5, as adopted in July 1997, or any standard adopted by the United States Environmental Protection Agency that succeeds that standard, is exceeded.

SECTION 8.

Section 7.5 of this bill incorporates amendments to Section 40451 of the Health and Safety Code proposed by both this bill and SB 1195. It shall only become operative if

(1) both bills are enacted and become effective on or before January 1, 2000,

(2) each bill amends Section 40451 of the Health and Safety Code, and

(3) this bill is enacted after SB 1195, in which case Section 7 of this bill shall not become operative.

SECTION 9.

Notwithstanding Section 17610 of the Government Code, if the Commission on State Mandates determines that this act contains costs mandated by the state, reimbursement to local agencies and school districts for those costs shall be made pursuant to Part 7 (commencing with Section 17500) of Division 4 of Title 2 of the Government Code. If the statewide cost of the claim for reimbursement does not exceed one million dollars ($1,000,000), reimbursement shall be made from the State Mandates Claims Fund.

Appendix C

Spatial Averaging of Receptors for Toxics Risk Assessments

C.1 Summary

Air dispersion modeling for long term averages for risk assessments typically include the single receptor at the highest concentration (i.e., the Point of Maximum Impact, or PMI), the maximally exposed individual resident (MEIR), and the maximally exposed individual worker (MEIW). Because individuals at a residence or a workplace may tend to move around and not remain at a single point, it seemed reasonable to the ARB and OEHHA to compare modeled air concentrations at a single point with the air concentrations averaged over an area where exposure might more realistically occur. Appendix C compares modeled average air concentrations of several sized averaging domains with the estimate at the PMI. It also looks at area, volume, point and line sources to determine the impact of source type and size of source on the ratio of the PMI to averaged domain. The analysis presented in this document shows how the spatial average of the collective nearby receptors can be approximately 45% to 80% of the highest concentration depending on the source type. The spatial averaging of air concentrations at receptors is more sensitive to emissions from small sources vs. large sources. The spatial averages for nearby areas as small as (10m x 10m) up to (100m x 100m) are shown.

C.2 Introduction

Since the inception of the “Hot Spots” and the air toxics programs in California, health risk assessment (HRA) results for an individual have typically been based on air dispersion modeling results at a single point or location. This method has been traditionally used for all types of receptors (e.g., PMI, MEIR, and MEIW, pathway receptors, etc.). The assumptions used in a risk assessment are designed to err on the side of overestimation rather than underestimation of health impacts to the public – a health protective approach.

Air pollutant concentrations are estimated at receptors which are distributed in a grid pattern of sufficient size and density to capture the maximum concentration (e.g., at the Point of Maximum Impact (PMI)). Under some conditions, the PMI may be significantly higher than receptors only a few meters away. A more refined inhalation exposure estimate in such situations can be obtained by estimating an average concentration in a small area where the receptor might be moving about.

The Air Resources Board (ARB), in conjunction with the Office of Environmental Health Hazard Assessment (OEHHA), performed sensitivity analyses to evaluate the impacts of spatially averaging air dispersion modeling results. In this appendix, we study the sensitivity of spatially averaging the concentration of a group of receptors in the vicinity of the PMI in order to obtain an average concentration that better represents the long-term average over space and time. That information is presented below.

C.3 Source Types

Air quality modeling of facility emissions are normally carried out with a Gaussian plume model such as US-EPA's AERMOD[1]. The AERMOD algorithms include features that allow for the modeling of point, volume, and area sources. Line sources can be a special case of a series of volume or area sources.

For this analysis, we categorize each of the four source types (point, volume, area, and line) into three sizes; small, medium, and large. (Line sources are only treated as small and large.) The release parameters for input to the dispersion model are summarized in Tables 1, 2, 3, and 4. These sources are depicted schematically in Figures 1, 2, 3, and 4.

Air dispersion modeling for line sources is completed with the CAL3QHCR[2] model. CAL3QHCR is a roadway line source model. The line sources represented in this sensitivity analysis are roadway motor vehicle emissions. Roadways are not part of the Hot Spots program because the program only addresses stationary sources. However, roadways need to be modeled for proposed school sites within 500 feet of a busy roadway under SB-352. SB-352 specifies that the Hot Spots risk assessment guidance is used for the risk assessment. Differences between AERMOD and CAL3QHCR are beyond the scope of this appendix. The concepts of spatial averaging with CAL3QHCR results could be extended to AERMOD line source studies.

[1] AERMOD – A steady-state plume model that incorporates air dispersion based on planetary boundary layer turbulence structure and scaling concepts, including treatment of both surface and elevated sources, and both simple and complex terrain. U.S. EPA (2004). User's Guide for the AMS/EPA Regulatory Model - AERMOD. EPA-454/B-03-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

[2] CAL3QHCR – Line Source Model – Environmental Protection Agency, 1992. User's Guide for CAL3QHC Version 2: A Modeling Methodology for Predicting Pollutant Concentrations near Roadway Intersections. Publication No. EPA–454/R– 92–006. Office of Air Quality Planning & Standards, Research Triangle Park, NC. (NTIS No. PB 93–210250)

Figure 1 – Point Sources

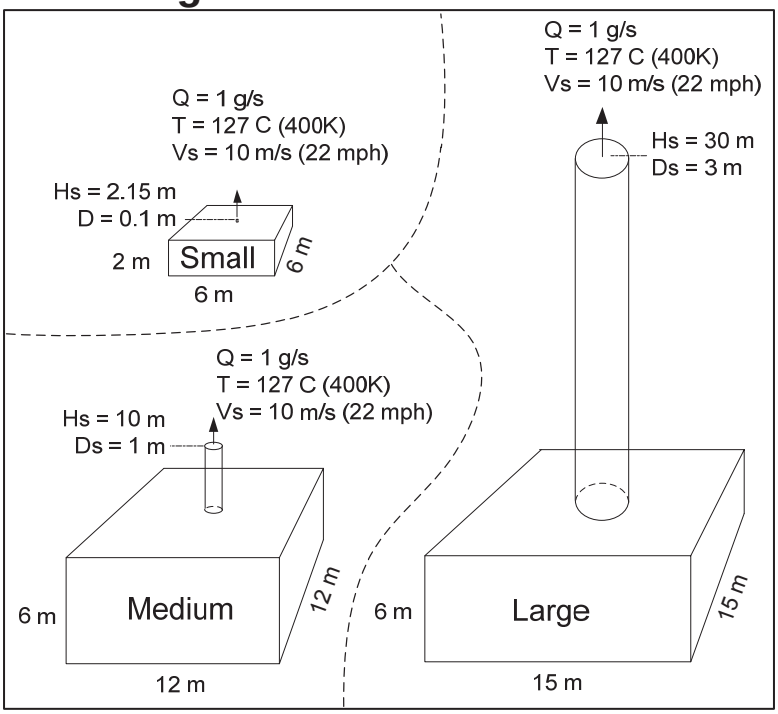


Table 1 – Point Source (Stack) Modeling Parameters

Source Size	Qs[a] (g/s)	Hs[b] (m)	Ds[c] (m)	Ts[d] (K)	Vs[e] (m/s)	FPH[f] (m)	Bh[g] (m)	Bl[h] (m)	Xadj Yadj (m)[i]	Similar Sources
Large	1	30	3	400	10	370.	6	15	7.5	Power Plant / Boiler
Medium	1	10	1	400	10	97.8	6	12	6	Asphalt Batch Plant
Small	1	2.15	0.1	400	10	5.15	2	6	3	Truck Engine

a) Emission rate
b) Release height above ground
c) Stack inside diameter
d) Stack exit temp, 400 K (260 F) is at the lower end of the combustion exhaust temperature range.
e) Stack exit velocity
f) FPH (Final Plume Height) varies with atmospheric conditions and is calculated hourly by the air quality model. For this table we calculated the FPH with US-EPA's SCREEN3 model under neutral atmospheric stability (D) and low wind speed (1m/s) for comparative purposes.
g) Building height
h) Building length
i) Along-flow (Xadj) and across-flow (Yadj) distances from the stack to the center of the upwind face of the projected building.

Figure 2 – Volume Sources

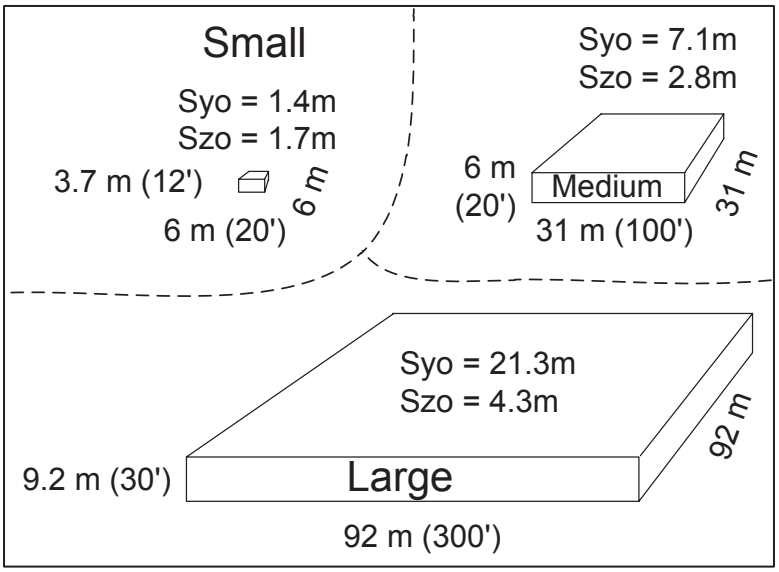


Table 2 – Volume Source Modeling Parameters					
Source Size	Qs (g/s)	Hs (m)	Syo (m)	Szo (m)	Similar Sources
Large	1	4.6	21.3 (L=92m)	4.3	Fleet Facility (300'x300'x30')
Medium	1	3.0	7.1 (L=31m)	2.8	(100'x100'x20')
Small	1	1.8	1.4 (L=6m)	1.7	Dry Cleaner (20'x20'x12')

H: Volume source height
Hs: Plume centerline release height (H = 2 Hs)
Syo: Initial plume dispersion in the horizontal (Syo = L / 4.3)
Szo: Initial plume dispersion in the vertical (Szo = H / 2.15)

Figure 3 – Area Sources

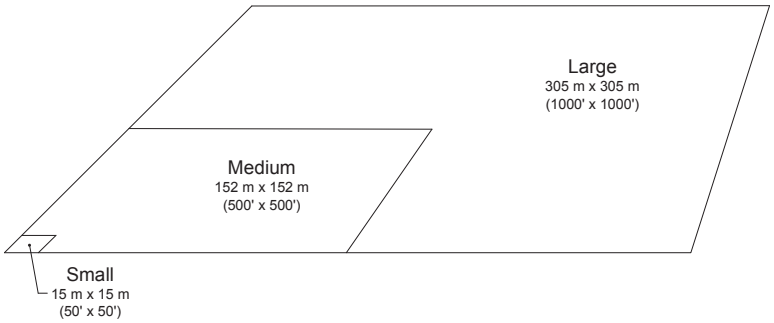


Table 3 – Area Source Modeling Parameters				
Source Size	Qs (g/s)	Hs (m)	Ls (m)	Similar Sources
Large	1	3.0	305	Rail Facility (1000'x1000')
Medium	1	3.0	152	Industrial Loading Facility (500'x500')
Small	1	2.0	15	Pile (50'x50')

Figure 4 – Line Source – Large and Small

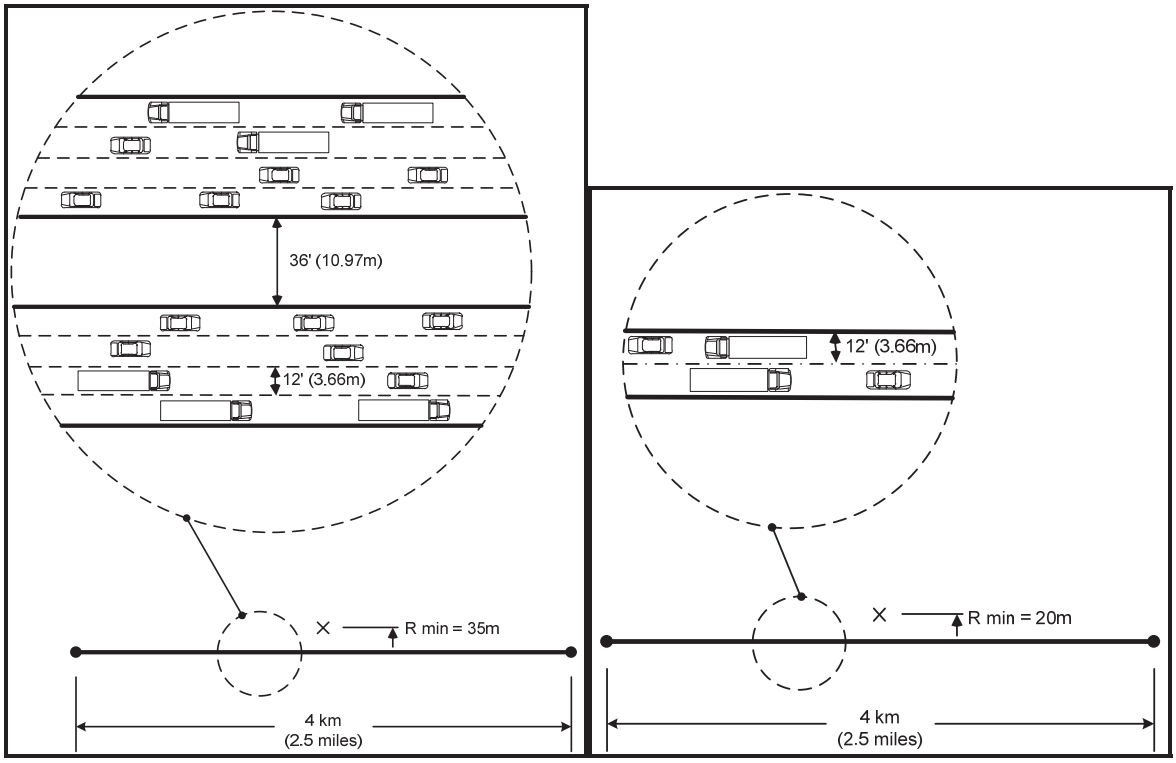


Table 4 – Line Source Modeling Parameters					
Source Size	Qs (g/s)	Vehicles per Day	Lanes	Ls (m)	Min Receptor Placement (m)
Large	1	250,000	8	4000	35
Small	1	5,000	2	4000	20

The roadway line source is simulated as four kilometers of straight roadway. The large source is an eight lane roadway where the first receptor is located 35 m from the edge of the roadway. The small source is a two lane roadway where the first receptor is located 20 meters from the edge of the roadway. Hourly variations in traffic flow are shown in the Appendix C-1.

US-EPA Guidelines[3] accept the CALINE3 and CAL3QHCR models to simulate emissions from roadways. Algorithms to simulate the enhanced mechanical turbulence and thermal buoyancy associated with motor vehicles are included in the CALINE series of models. CALINE is formulated with the Pasquill-Gifford plume distributions to simulate downwind dispersion. AERMOD is US-EPA's state-of-science dispersion model. AERMOD does not use the Pasquill-Gifford step functions of dispersion curves for estimating atmospheric stability, but rather a continuum of atmospheric dispersion is

[3] U.S. EPA (2005). Federal Register / Volume 70, Number 216 / November 9, 2005 / Rules and Regulations, 40 CFR Part 51 Appendix W, Revision to the Guideline on Air Quality Models, U.S. Environmental Protection Agency

simulated. However, AERMOD does not facilitate the hourly mechanical turbulence or thermal buoyancy associated with motor vehicles.

CAL3QHCR is used for the roadway motor vehicle emissions. Although there is potential to carefully apply AERMOD to line sources, comparing the results from these two models is beyond the scope of this sensitivity study.

C.4 Meteorological Data

AERMET is the computer program that processes and prepares meteorological data for use in AERMOD. Meteorological data that have been processed with the AERMET processor are obtained from various Districts. The latest consecutive years (up to five) were obtained. We selected the following stations for this analysis. Also see Figure 5.

- Costa Mesa (2005-2007)
- Fresno Air Terminal (FAT) (2004-2008)
- Kearny Mesa (2003-2005)
- Lynwood (2005-2007)
- San Bernardino (SBO) (2005-2007)

Figure 5 – Meteorological Station Locations

Wind rose summaries for each meteorological station are available in Appendix C- 2. The data for Costa Mesa, Lynwood, and San Bernardino are provided by the South Coast Air Quality Management District. Fresno Air Terminal (FAT) data are provided by the San Joaquin Valley Air Pollution Control District. Kearny Mesa data are provided by the San Diego Air Pollution Control District.

CAL3QHCR is a version of CALINE that can be used to simulate roadway emissions and also accepts a complete year of hourly meteorological data. CAL3QHCR requires meteorological data with Pasquill-Gifford (PG) classifications for stability. The meteorological data provided for AERMOD as discussed above do not include PG stability. Rather a continuum of stability is represented.

For the purpose of using CAL3QHCR in this sensitivity study, the PG stability class is estimated from the Monin-Obukhov length available in the AERMET processed meteorological data. As suggested by Sykes and Lewellen 1992[4], the relationship between Monin-Obukhov length and PG stability class is shown in Table 5.

Table 5 – Stability Estimates

PG Stability Class	Monin-Obukhov Length (m)
A	-5
B	-12.5
C	-50
D	-1000
E	25
F	13

As suggested by Sykes, R.I. and W.S. Lewellen (1992), "Review of potential models for UF_6 dispersion," Martin Marietta Energy Systems, Inc., Safety and Analysis Report-19 (SAR-19)

For regulatory purposes, we recommend that the stability class be determined with standard procedures for processing meteorological data with PG stability such as those available for the Industrial Source Complex – Short Term dispersion model.

The mixing height is constant at 500 meters for the CAL3QHCR simulations.

[4] Sykes, R.I. and W.S. Lewellen (1992), "Review of potential models for UF_6 dispersion," Martin Marietta Energy Systems, Inc., Safety and Analysis Report-19 (SAR-19).

C.5 Receptors

Receptors are set as flagpoles 1.2 meters above ground. A coarse receptor grid with 20 meters spacing is used to locate and center a nested grid with five meter spacing on the point of maximum impact (PMI). We selected the PMI no closer than 20 meters to a point source; 20 meters to the virtual edge of a volume source; or zero meters to the edge of an area source. AERMOD limitations on receptor placement are that no receptors be located within one meter of the point source and no receptors within a volume source. Receptors within an area source are still valid.

The nested grid was centered on the PMI for the large and medium point source receptors. For the small point source, volume sources, area sources, and line sources, the near edge of the grid was centered on the PMI in order to keep nested receptors off of the source. Simple arithmetic averaging was used to average the nested grid over the PMI with various nesting domain sizes. Figure 6 shows the PMI and two nested grids for the large point source.

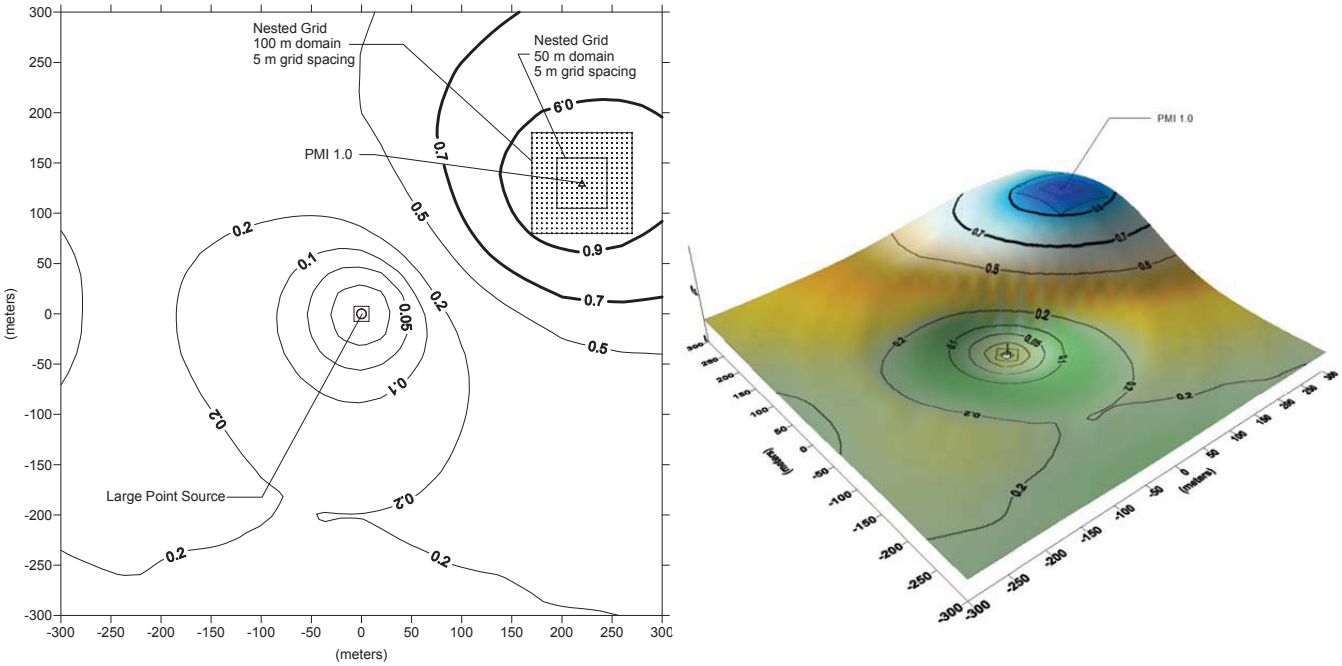
Appendix C-3 shows the PMI and two nested grids for each source (point, volume, area, and line) and for all sizes.

The spatial average was calculated for nested grids at ten different domains; 10m x 10m up to 100m x 100m, even though only two nested grids are shown on each plot.

An emission rate of 1 g/s was used for each source type. The resulting concentration field output was normalized to the offsite PMI. Therefore, the offsite receptor concentrations have a maximum value of 1.00 $\mu g/m^3$.

Figure 6
Concentration Distribution (Normalized to PMI)
Large Point Source

Isopleths ***Surface***



C.6 Results

The graphical displays of the concentration fields from the multitude of source types and meteorological representation are available in Appendix C-3. It is evident from these figures that estimated ground level concentrations fall off most steeply from the PMI with smaller source types with a low plume rise where the PMI is located at the property fence line. This is to say that the spatial average is lowest relative to the PMI with this type of small source. Source types with high plume rise (e.g., tall stacks in Figures AP C-3.1.1 – 1.5) show a PMI far downwind where the concentration gradient is more gradual and therefore the difference between the estimated air concentration with the spatial average and the PMI is less.

The results of the spatial averaging are summarized in Figures 7 – 10. Supporting tables are available in Appendix C-4.

The spatial averaging for a 10m x 10m receptor field can be as low as 65% of the PMI value as seen in Table AP C-4.3.3 and Figure 9.3.

In addition, the graphical displays in Appendix C-3 show that the dominant plume centerline is sometimes tilted from the cardinal directions. Since the nested grids for spatial averaging were placed along the cardinal directions, the results in Appendix C- 4

may underestimate a spatial average centered on the dominate plume centerline. Appendix C-5 shows how tilting the nested grid to coincide with the dominat plume centerline can increase the value of the spatial average. The value of the spatial averaged tilted grid may be higher than the non-tilted counterpart (e.g., 0.69 vs. 0.59). Whether or not to tilt the grid is a subjective decision and should be considered on a case-by-case basis.

C.7 Recommendations

Spatial averaging may be used to estimate a long term concentration over a small nested grid of receptors to represent an area vs. a single location as determined by the Point of Maximum Impact (PMI). Spatial averaging is most applicable for the following conditions.

- Long term averages are being calculated to represent multi-year impacts.
- The Point of Maximum Impact (PMI) is located at the fence line and close to the emission source.
- The concentration gradient is high near the PMI. This is most often associated with low level plumes such as fugitive, volume, or area sources.

The following are recommendations for calculating the spatial average.

1. Spatial averaging should not be used for maximum one hour air concentration estimation.
2. Locate the off-site PMI with a nested grid resolution spacing of no greater than five meters. Two or more model runs with successively finer grid resolutions centered on the new PMI may be required to locate the final PMI.
3. Center the nested grid on the off-site receptors about the PMI. Limit the nested grid to 20m x 20m. The grid resolution spacing should be no greater than five meters. With a 5m grid resolution, the 20m x 20m nest will result in 25 receptors.
4. If necessary, tilt the nested grid to coincide with the dominant plume centerline. Polar receptors are easier to implement than a tilted rectangular grid. The domain of the polar receptor field should be limited to a 15 meter polar radius.

Although this sensitivity study evaluated nested grids up to 100m x 100m, the above recommendation is to limit the nested grid domain to 20m x 20m if rectangular and a radius of 15m if polar. (A 20m x 20m square area is equivalent to a 16m radius half circle. Therefore we rounded down to 15m radius for convenience.)

As a frame of reference, low density single family detached dwellings have been described in some city municipal codes as RD4 – RD7 zoning. RD4 allows four units per acre of land and RD7 allows seven units per acre of land. Table 6 shows the equivalent acreage and size in meters of RD4 – RD7 lots assuming uniformly distributed and square lots.

Table 6 – Residential Zoning vs Lot Size

Zone	Lot Size (acres)	Lot Size Square Meter
RD4	0.250	32m x 32m
RD5	0.200	28m x 28m
RD7	0.143	24m x 24m
-	*0.099*	*20m x 20m*

Figure 7.1

Large Point Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

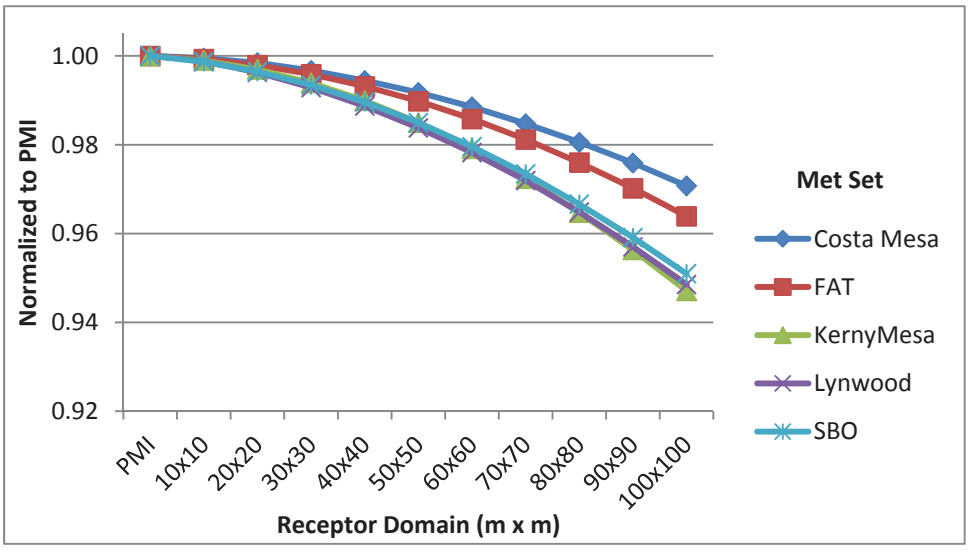


Figure 7.2
Medium Point Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

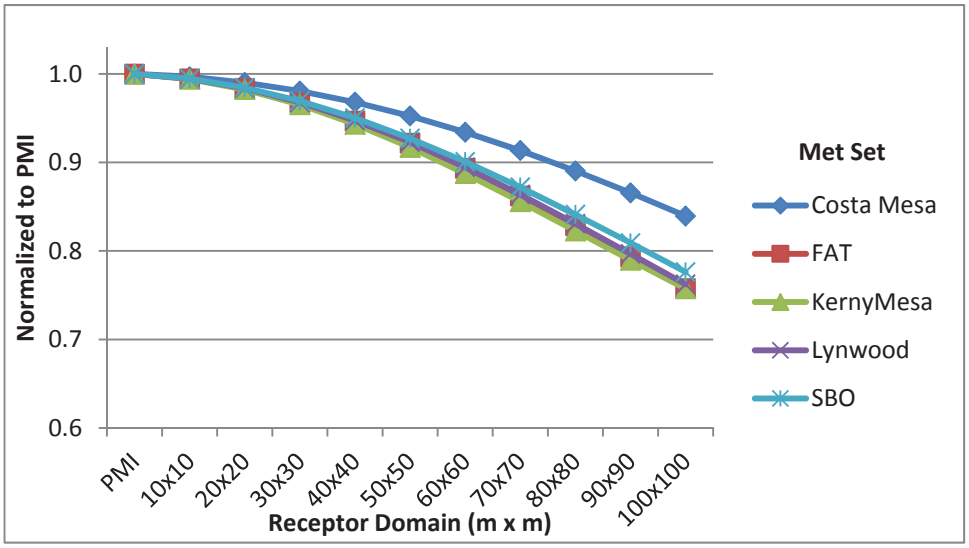


Figure 7.3
Small Point Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

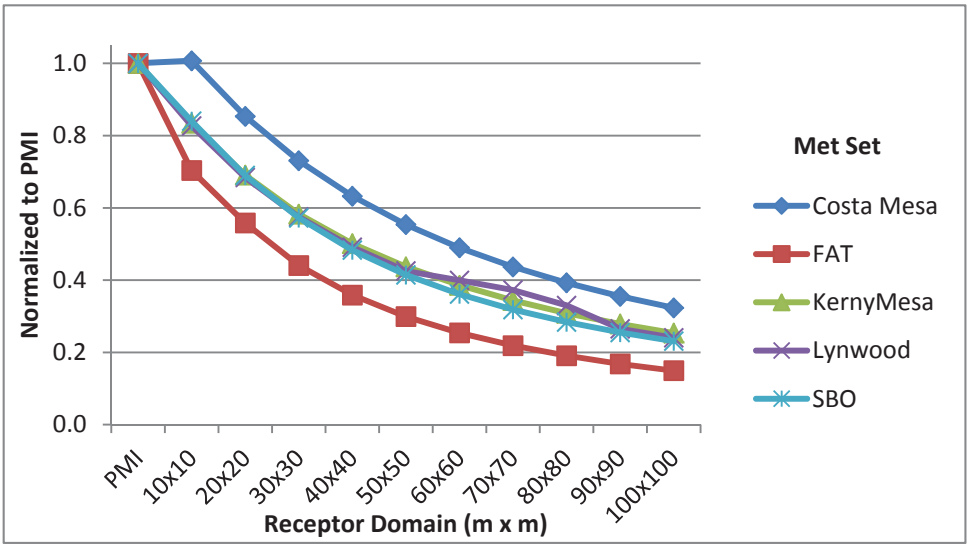


Figure 8.1
Large Volume Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

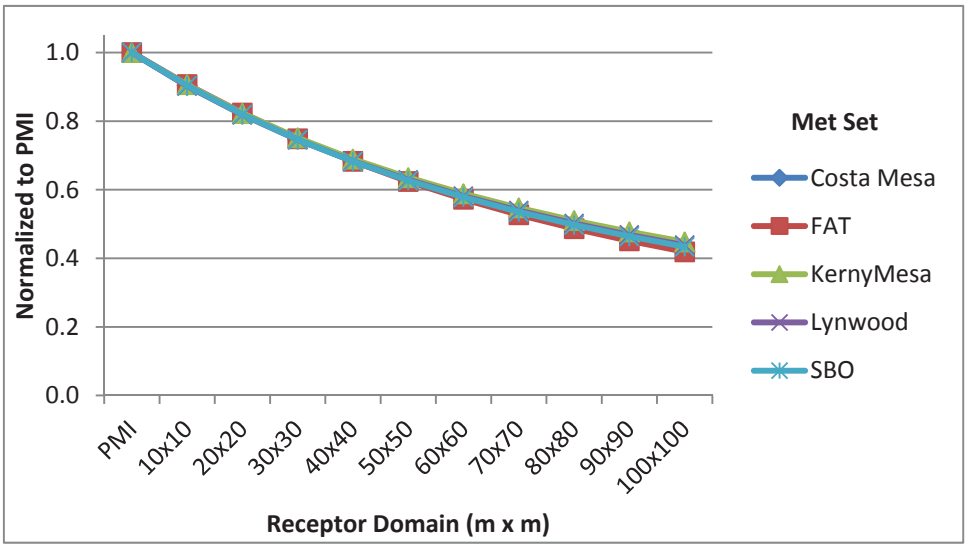


Figure 8.2
Medium Volume Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

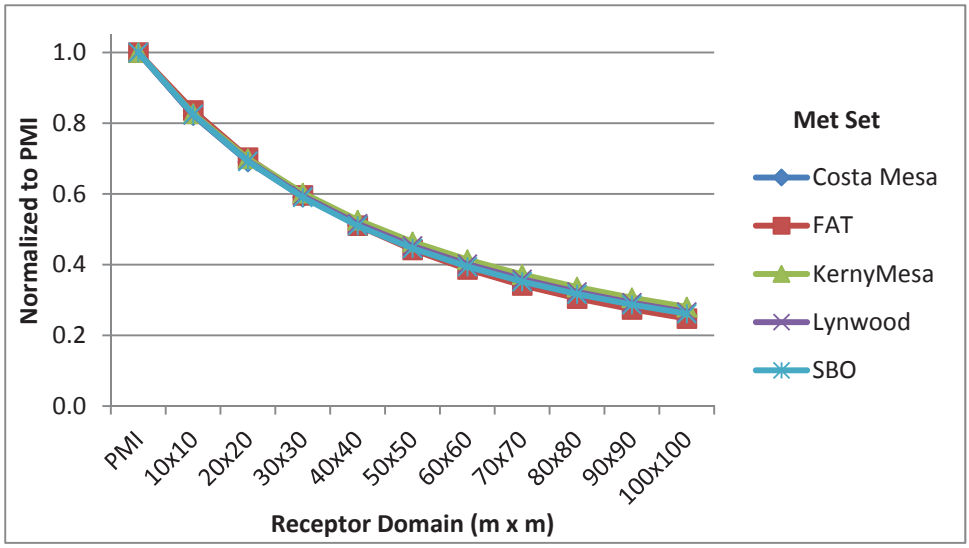


Figure 8.3
Small Volume Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

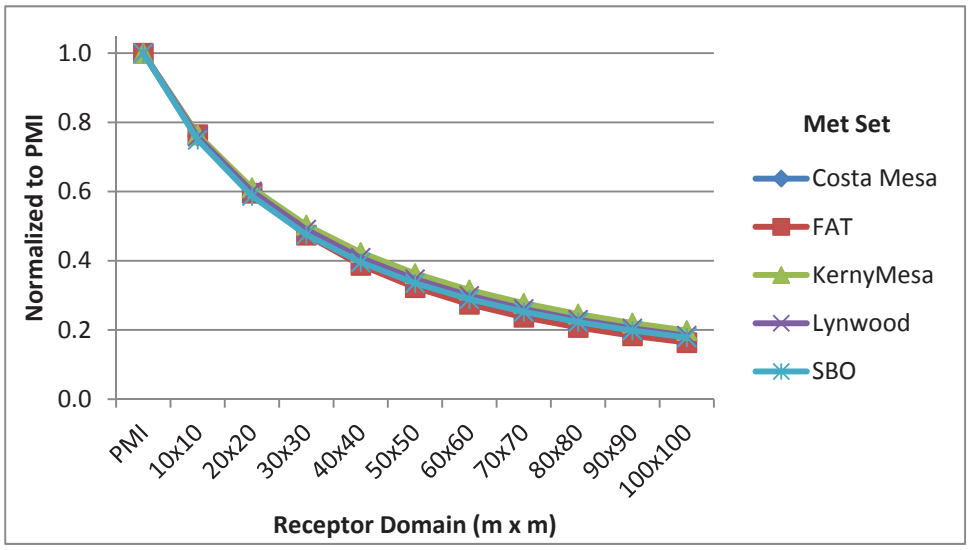


Figure 9.1
Large Area Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

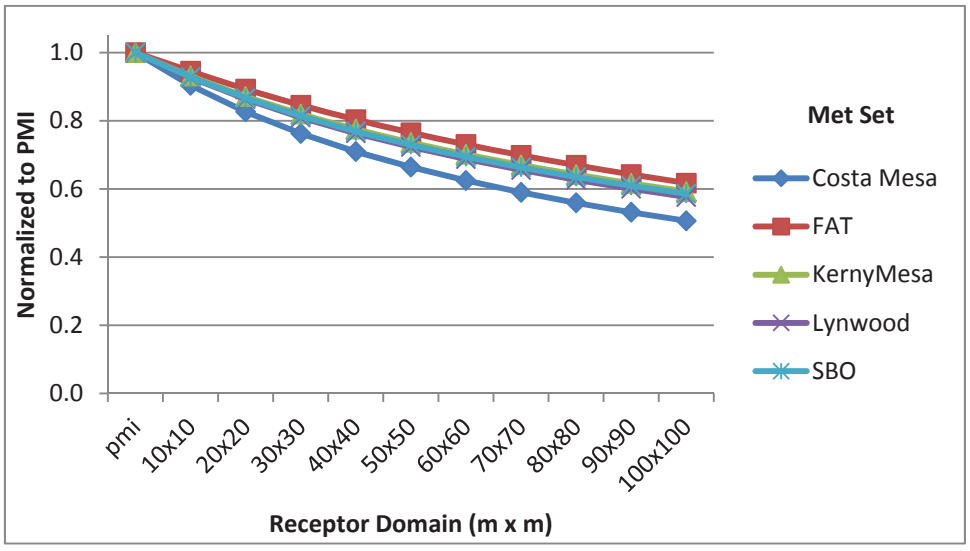


Figure 9.2
Medium Area Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

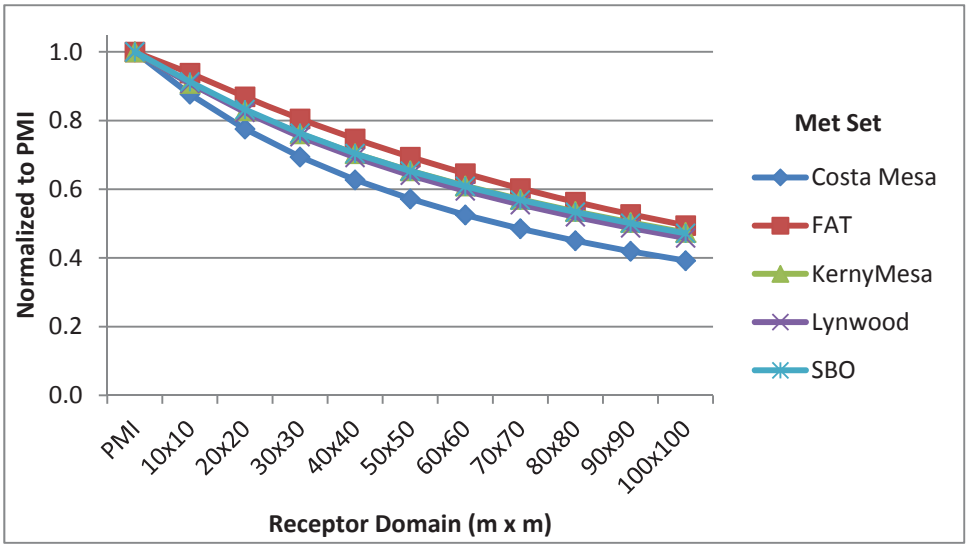


Figure 9.3
Small Area Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

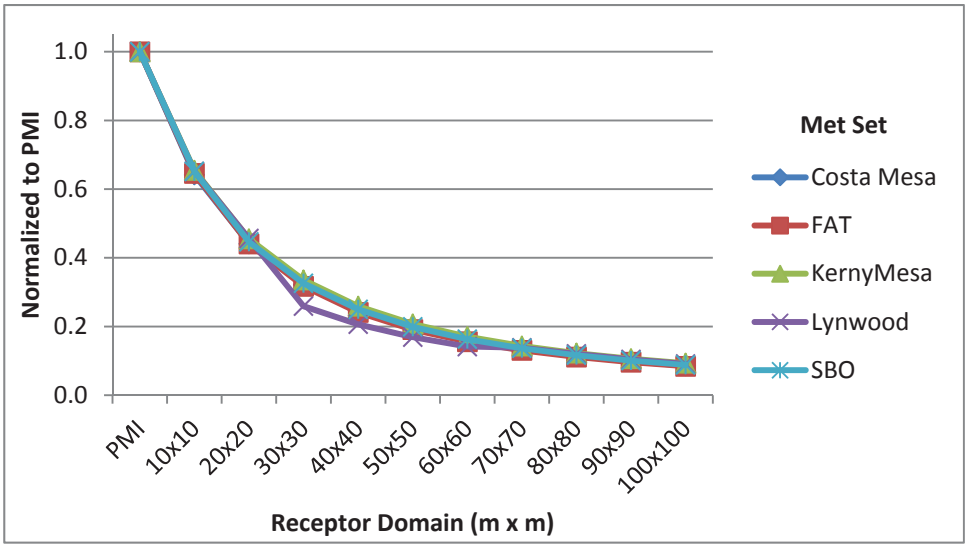


Figure 10.1
Large Line Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

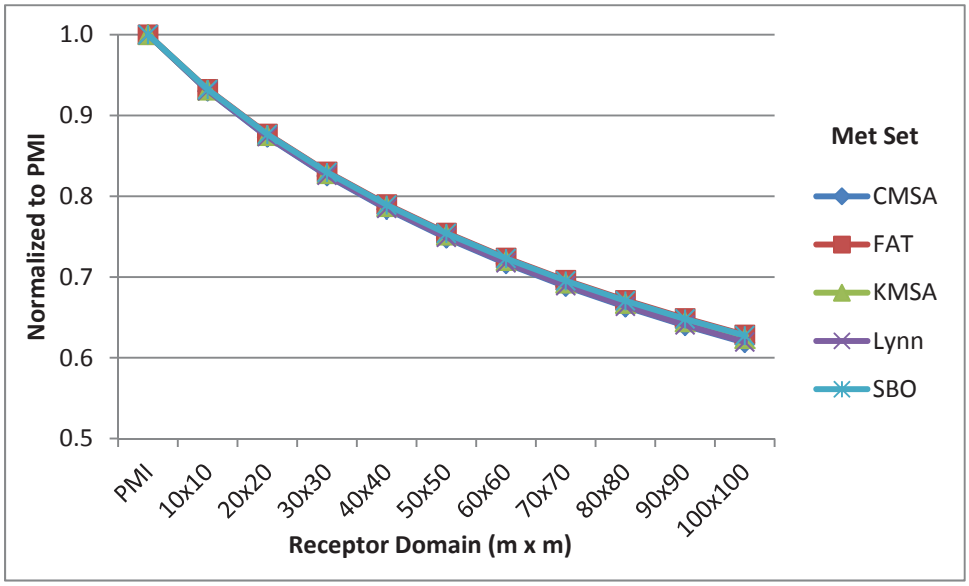
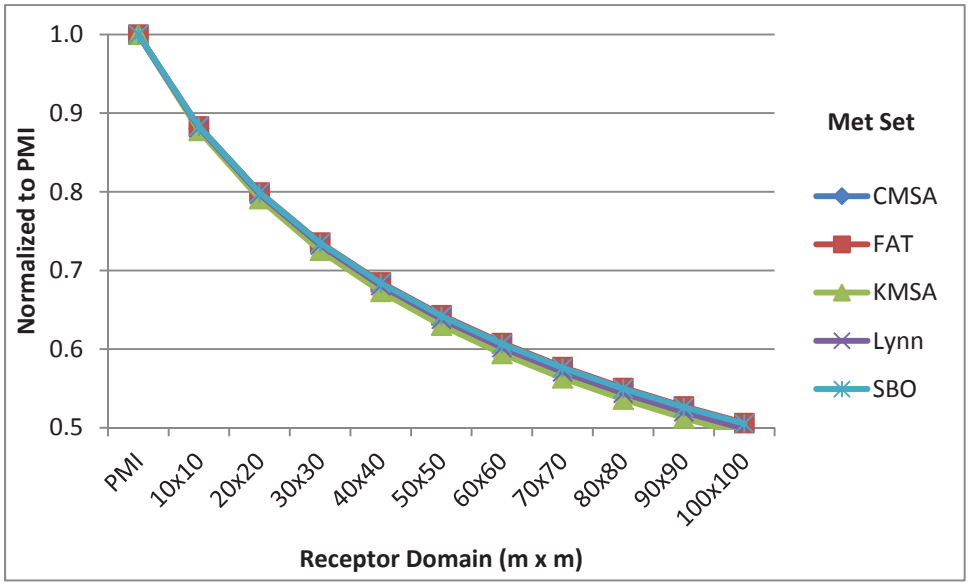
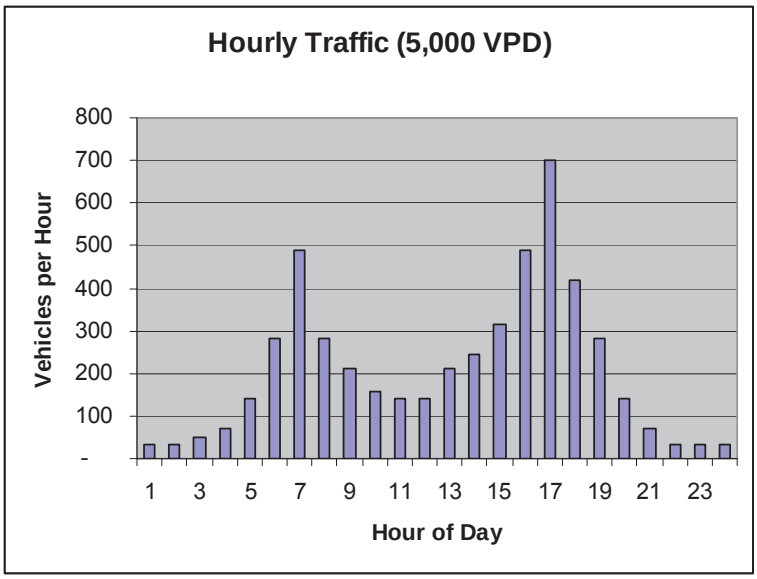


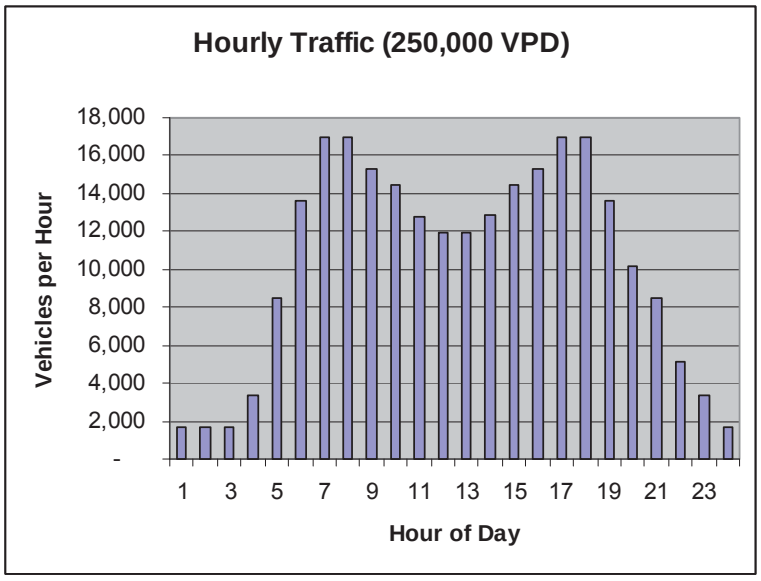
Figure 10.2
Small Line Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets



Appendix C-1 – Hourly Variation for Traffic Line Source

Hour	5K VPD	250K VPD
1	35	1,700
2	35	1,700
3	49	1,700
4	70	3,400
5	140	8,500
6	280	13,600
7	490	17,000
8	280	17,000
9	210	15,300
10	156	14,450
11	140	12,750
12	140	11,900
13	210	11,900
14	245	12,850
15	315	14,450
16	490	15,300
17	700	17,000
18	420	17,000
19	280	13,600
20	140	10,200
21	70	8,500
22	35	5,100
23	35	3,400
24	35	1,700
Sum	5,000	250,000
Peak Hour	700	17,000





Appendix C-2 – Meteorological Data

Figure ApC-2.1
AERMET Data from Districts

The above figure shows the locations where AERMET data are available from Districts. We selected the following stations for this analysis which include stations that are near the ocean and inland – Costa Mesa, Fresno Air Terminal (FAT), Kearny Mesa, Lynwood, and San Bernardino.

Figure AP C-2.2 – Costa Mesa – Wind Rose Summary

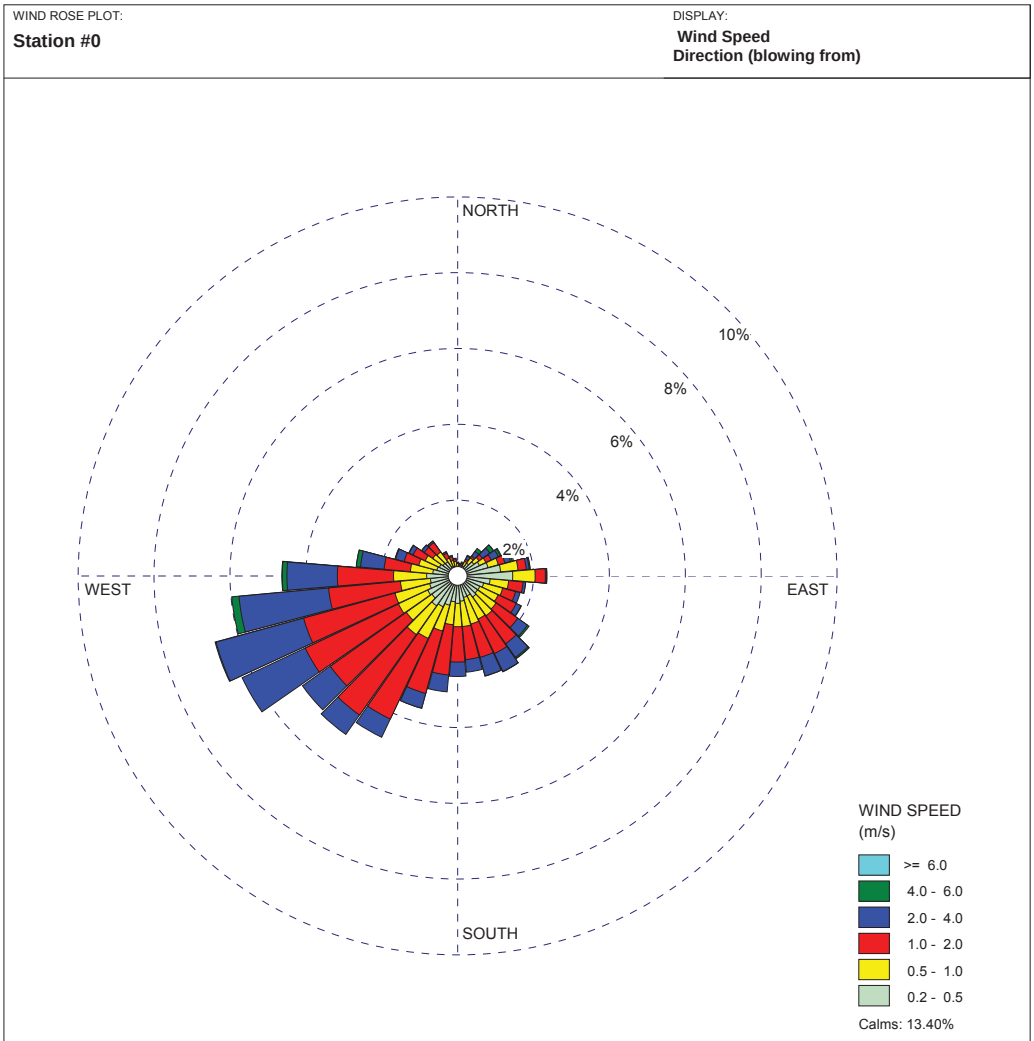


Figure AP C-2.3 – Fresno Air Terminal – Wind Rose Summary

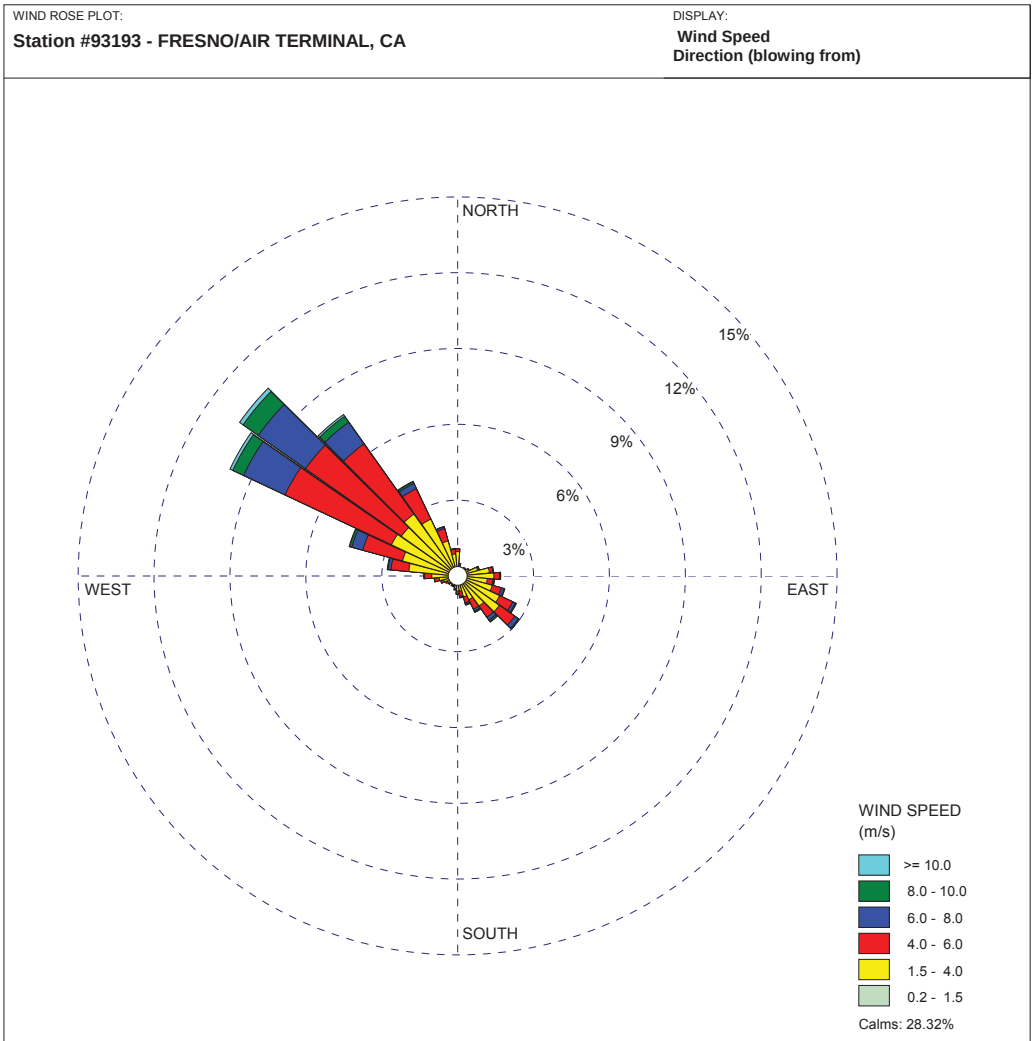


Figure AP C-2.4 – Kearny Mesa – Wind Rose Summary

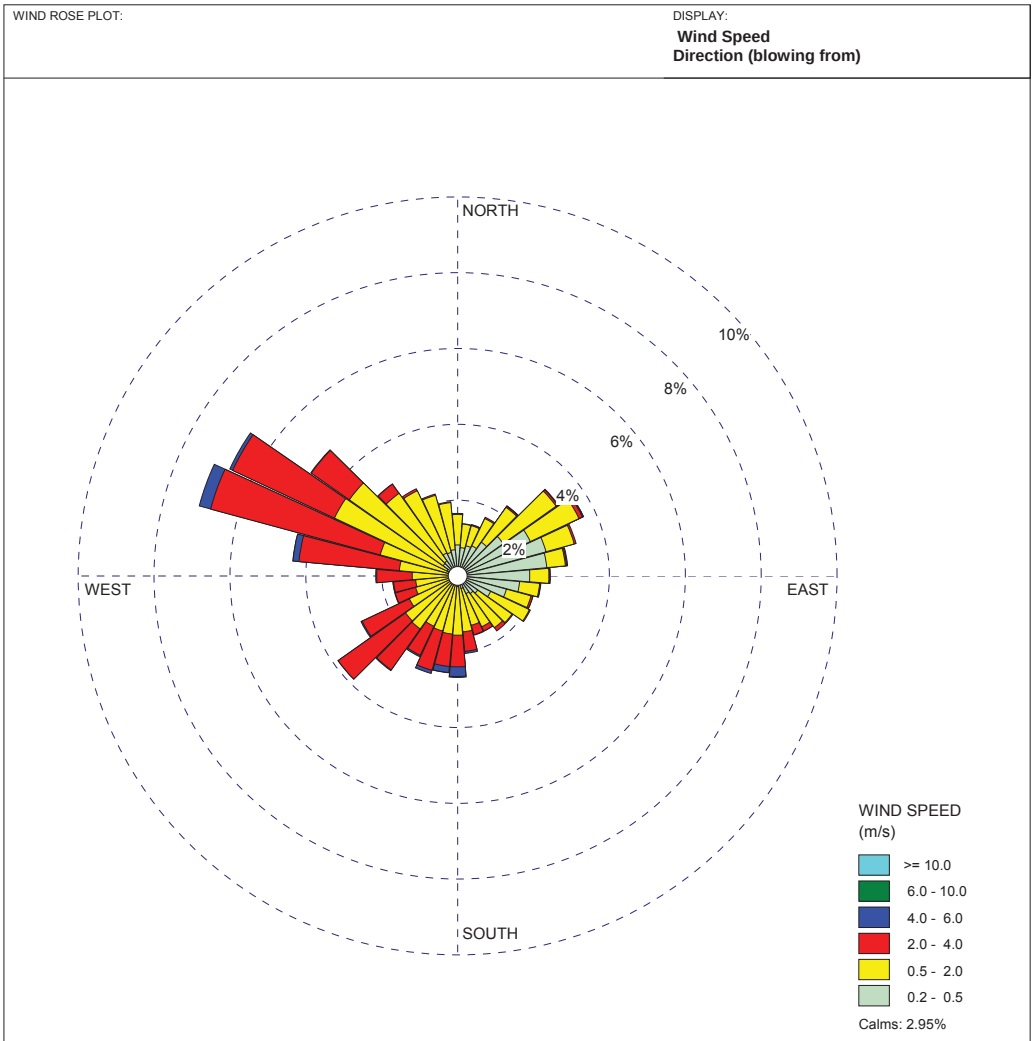


Figure AP C-2.5 – Lynwood – Wind Rose Summary

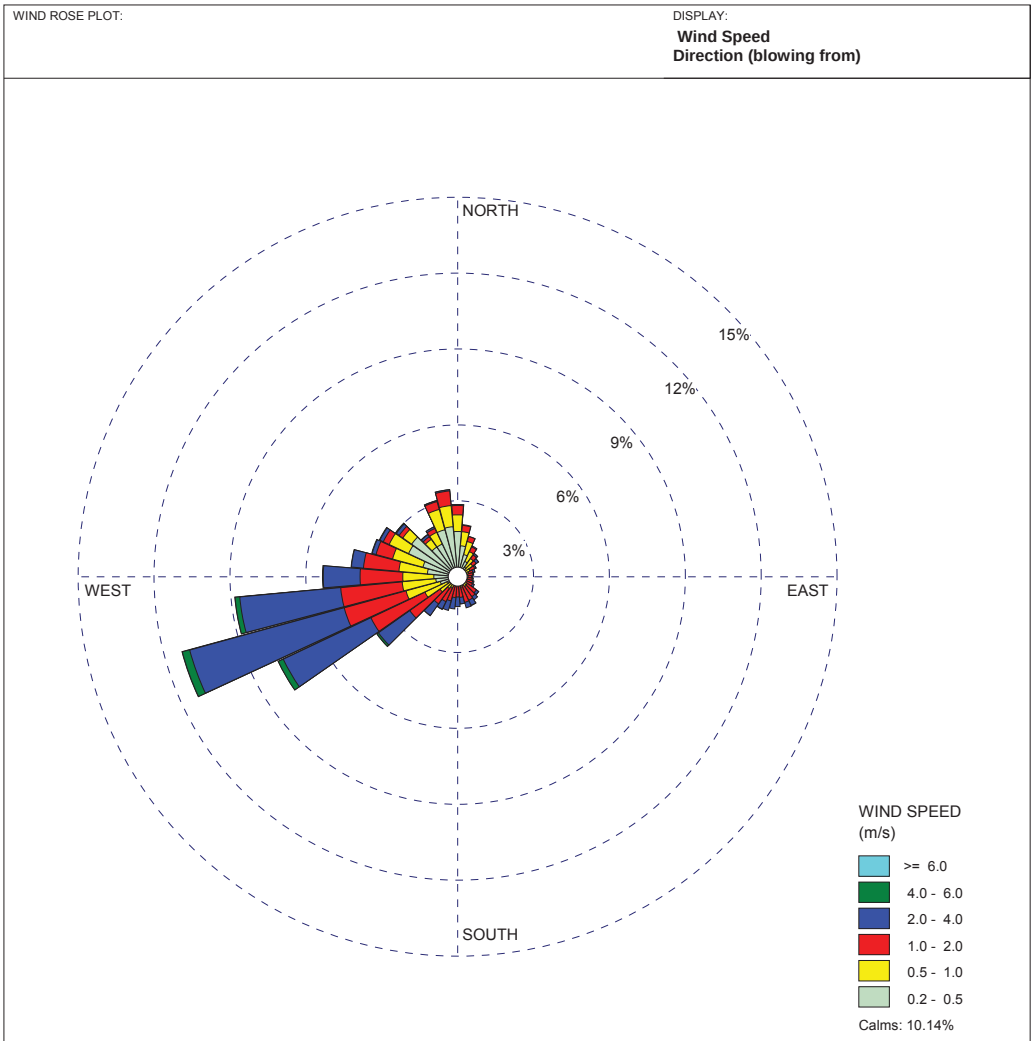
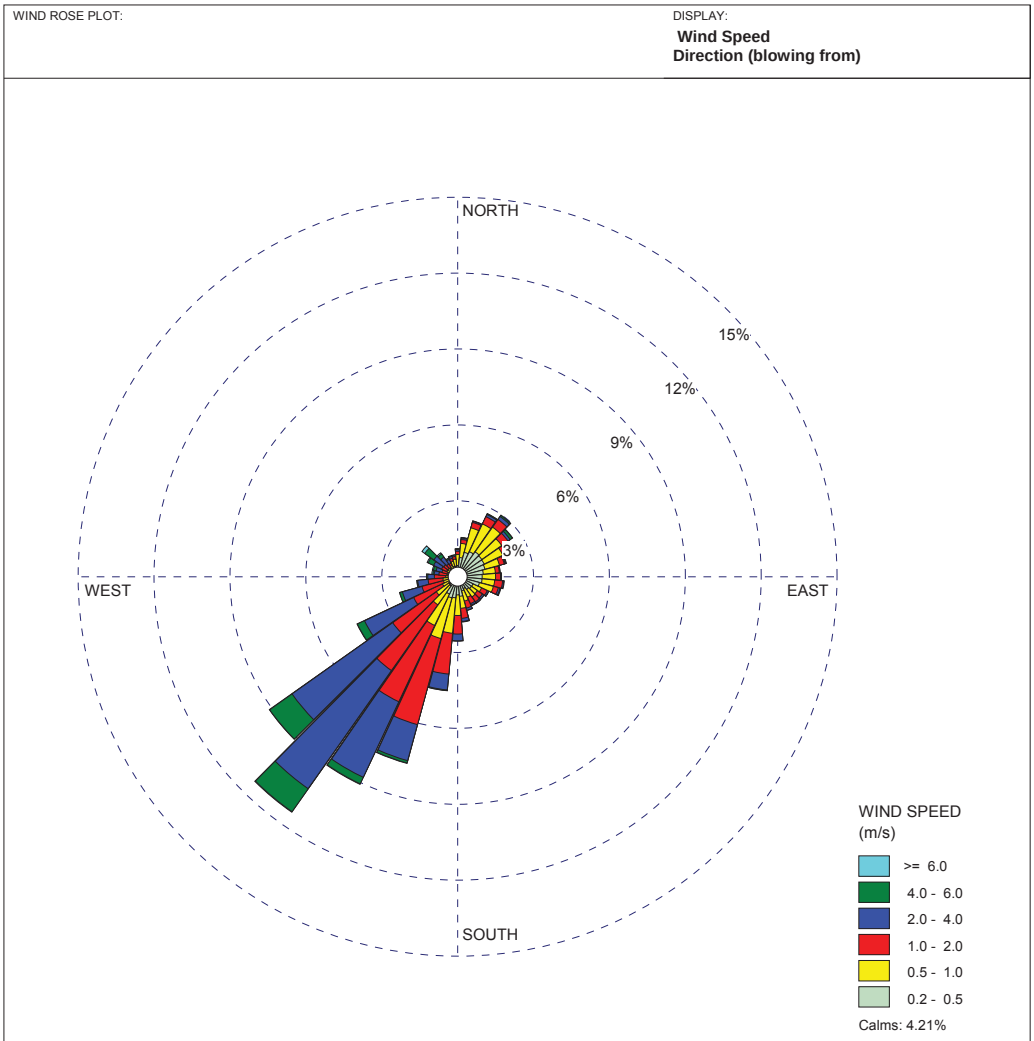


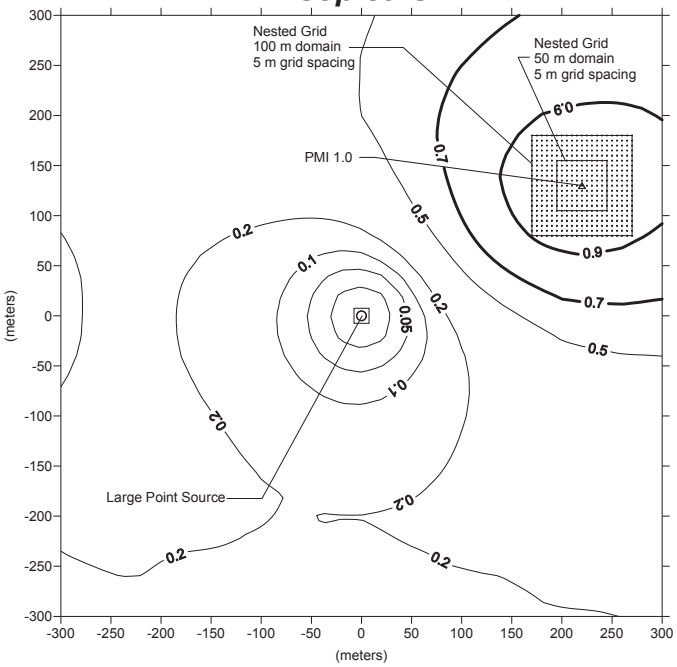
Figure AP C-2.6 – San Bernardino – Wind Rose Summary



Appendix C-3 – Sources, Receptors, Concentrations

Figure AP C-3.1.1 – Large Point Source – Costa Mesa

Isopleths



Surface

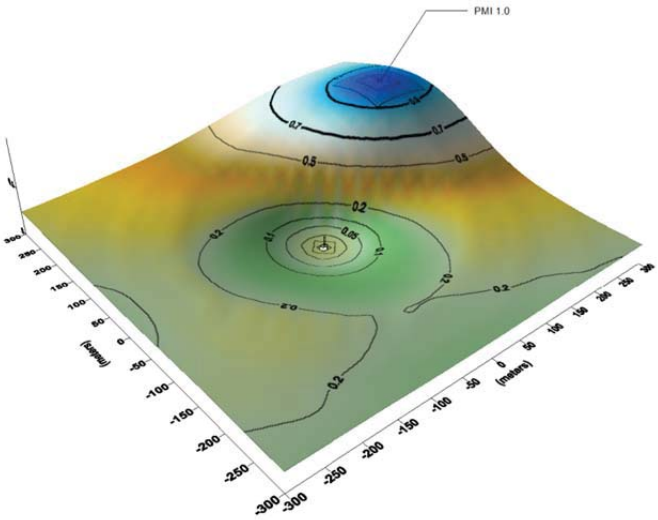
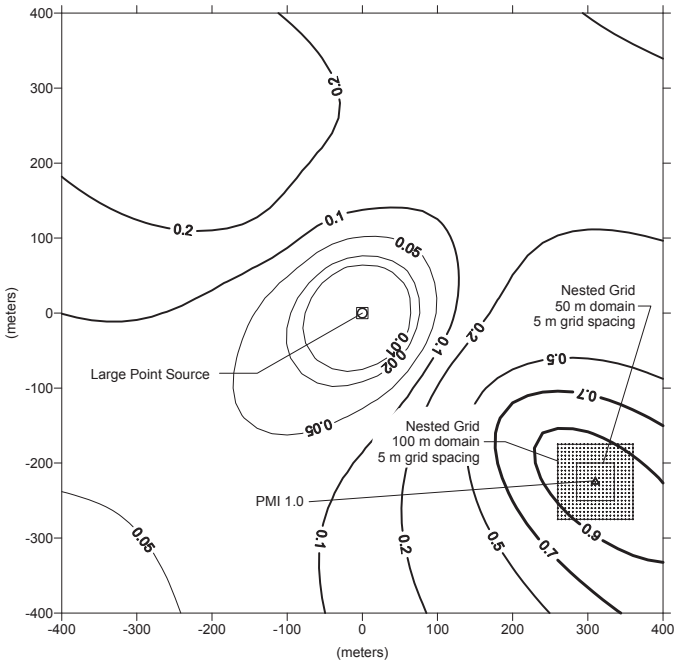


Figure AP C-3.1.2 – Large Point Source – Fresno Air Terminal

Isopleths



Surface

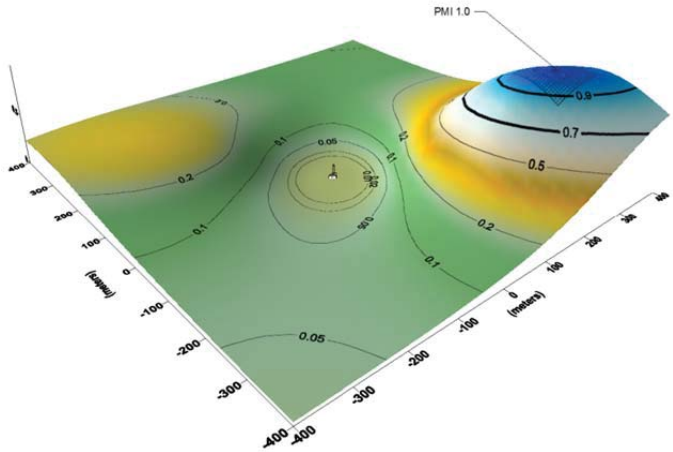


Figure AP C-3.1.3 – Large Point Source – Kearny Mesa

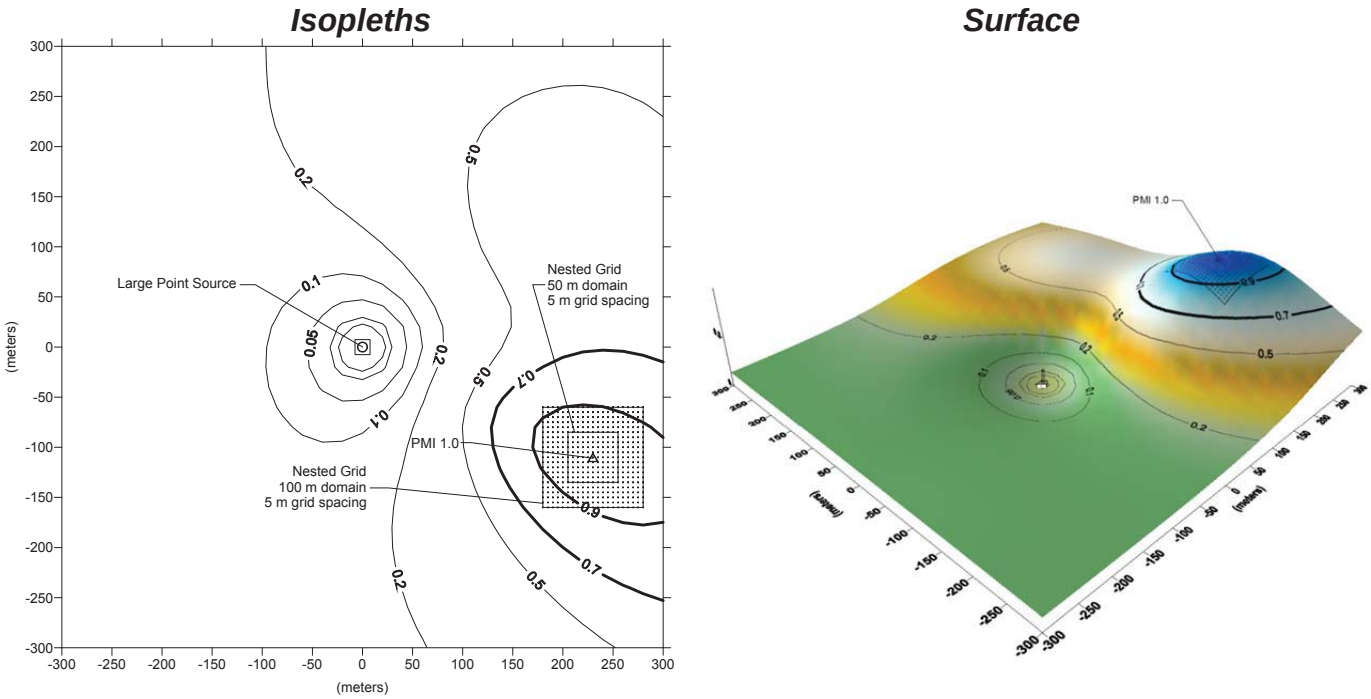


Figure AP C-3.1.4 – Large Point Source – Lynwood

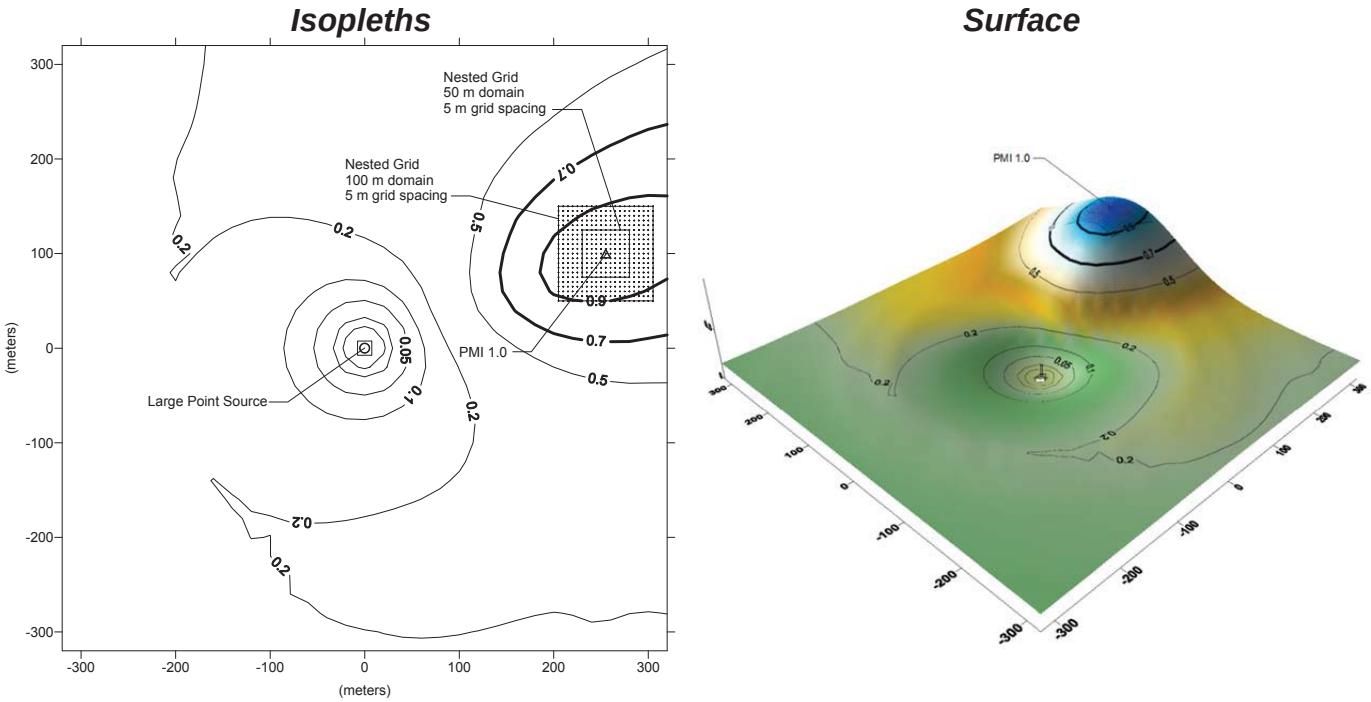
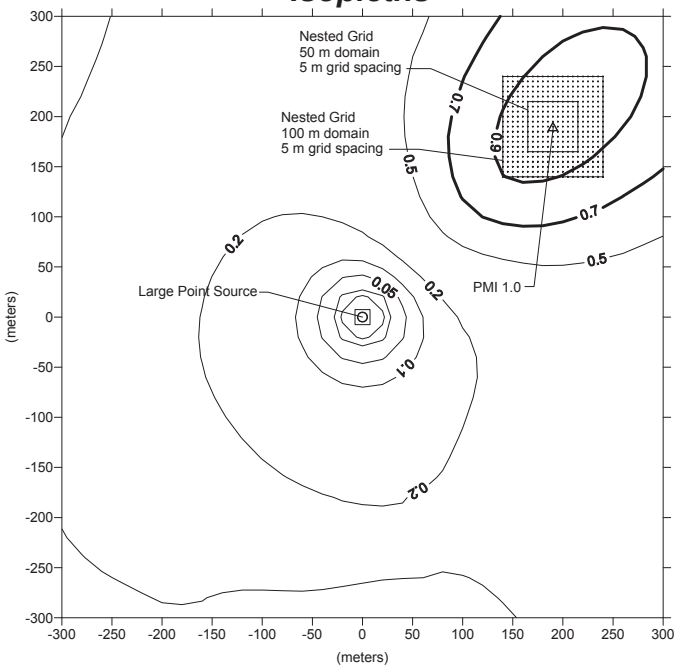


Figure AP C-3.1.5 – Large Point Source – San Bernardino

Isopleths



Surface

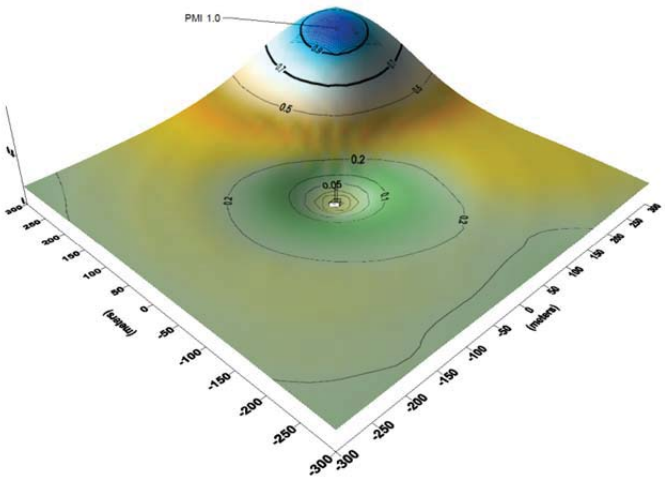
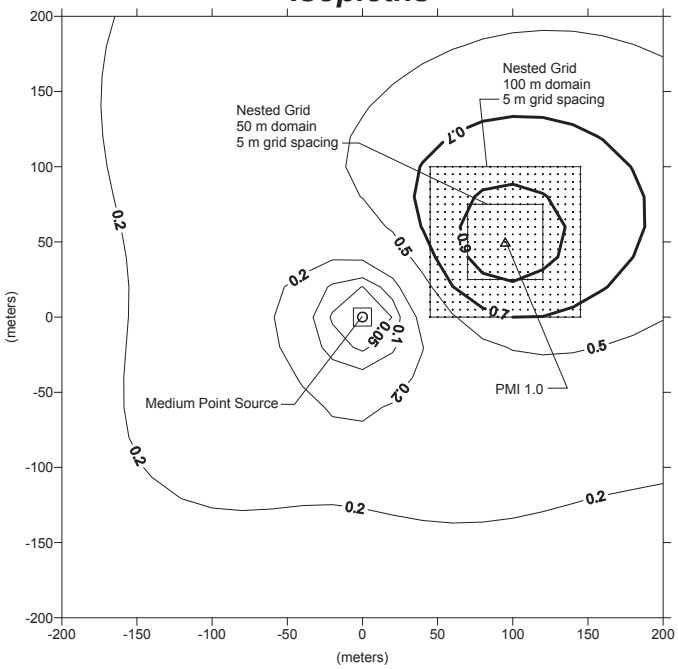


Figure AP C-3.2.1 – Medium Point Source – Costa Mesa

Isopleths



Surface

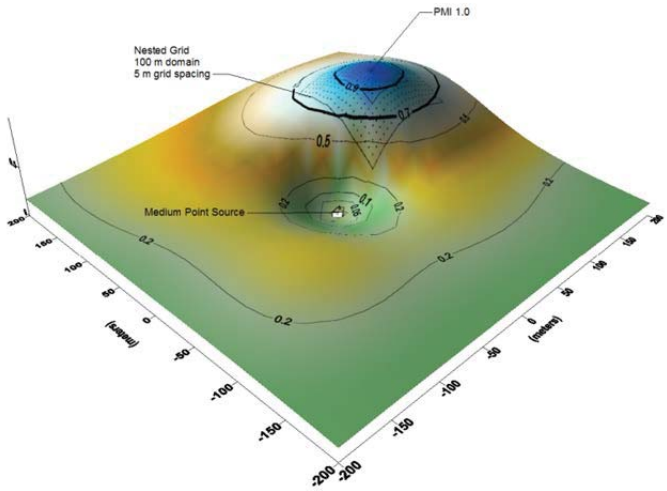
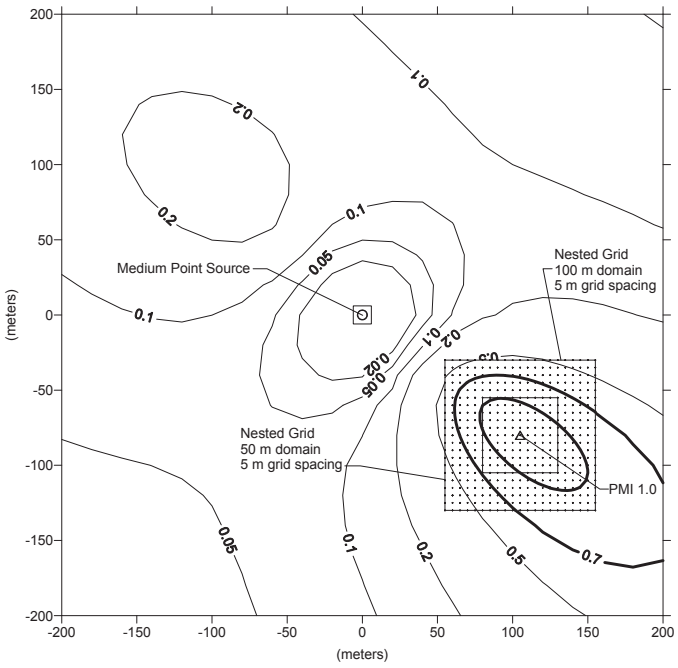


Figure AP C-3.2.2 – Medium Point Source – Fresno Air Terminal

Isopleths



Surface

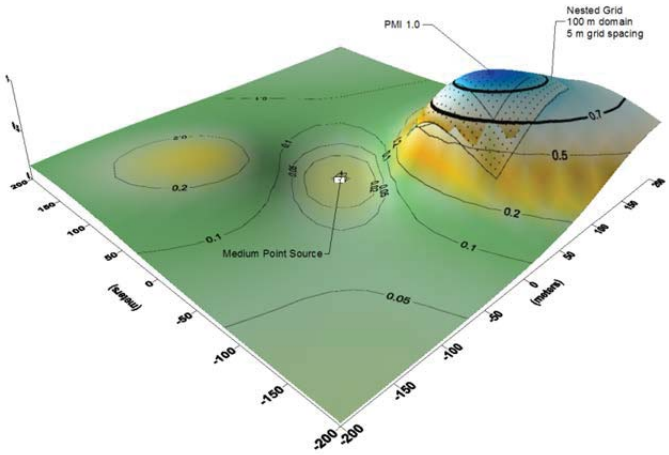


Figure AP C-3.2.3 – Medium Point Source – Kearny Mesa

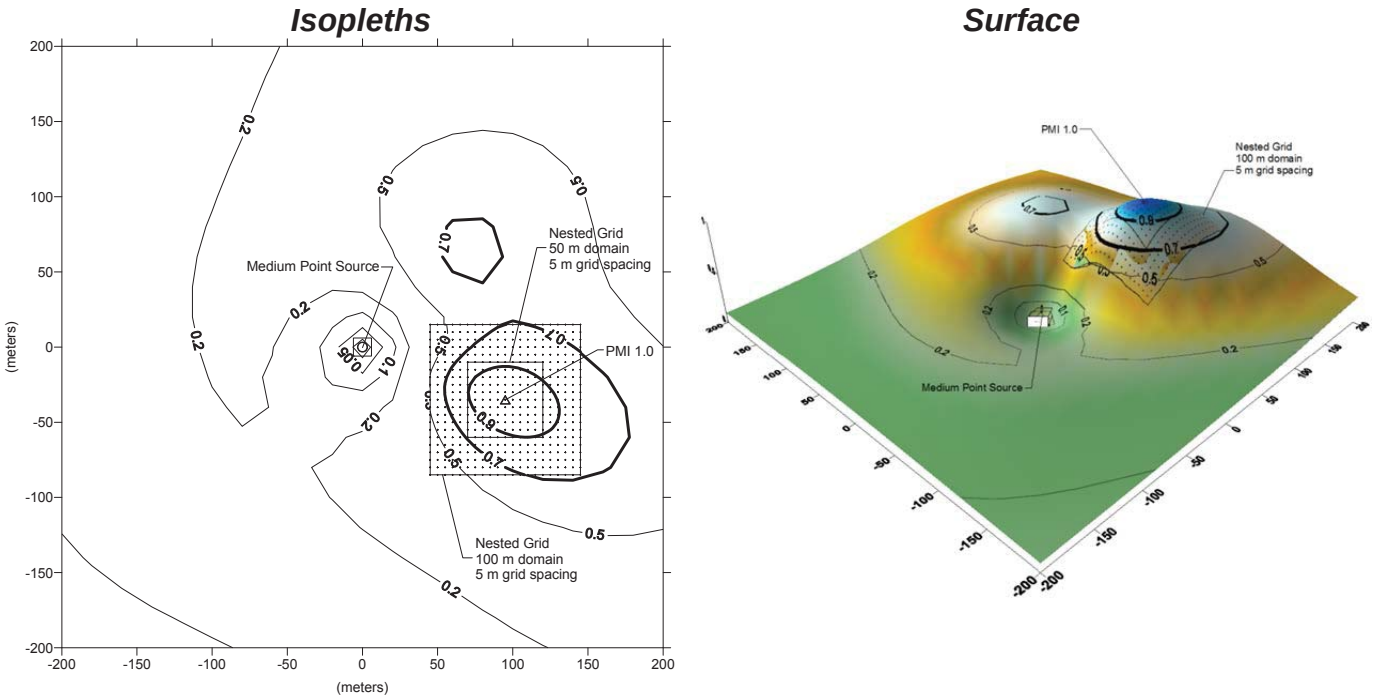


Figure AP C-3.2.4 – Medium Point Source – Lynwood

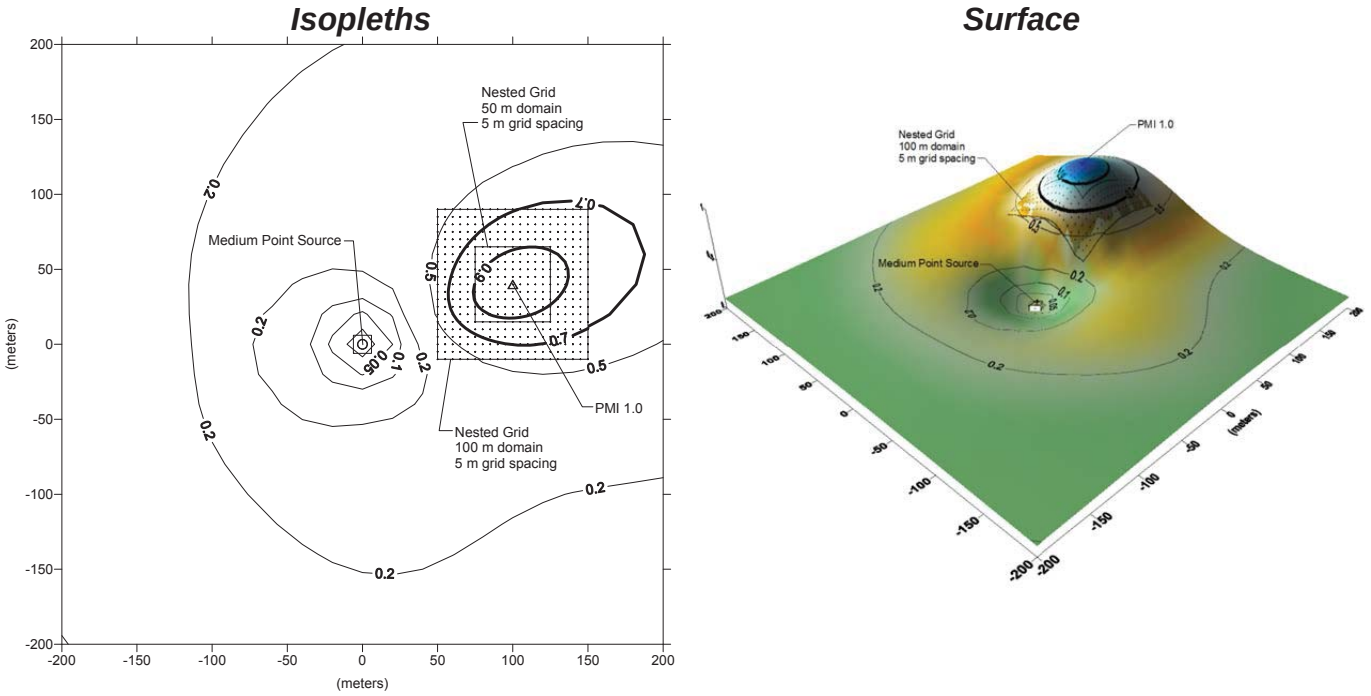
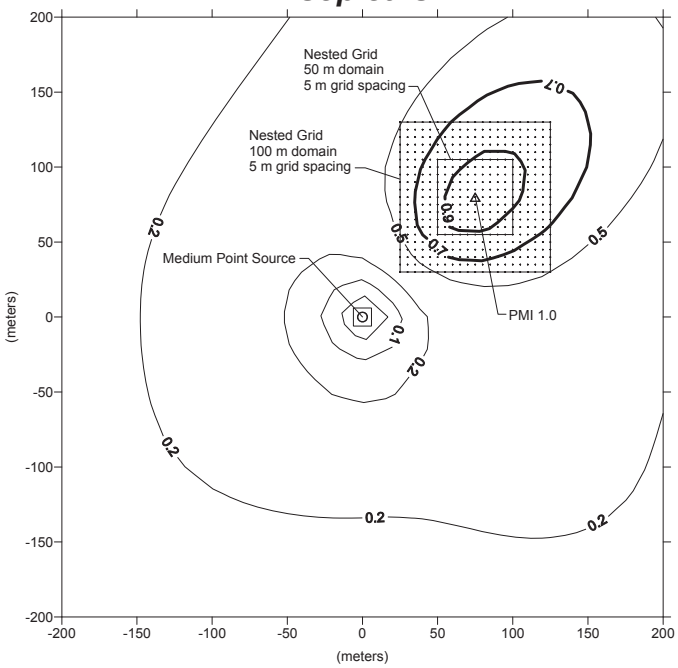


Figure AP C-3.2.5 – Medium Point Source – San Bernardino

Isopleths



Surface

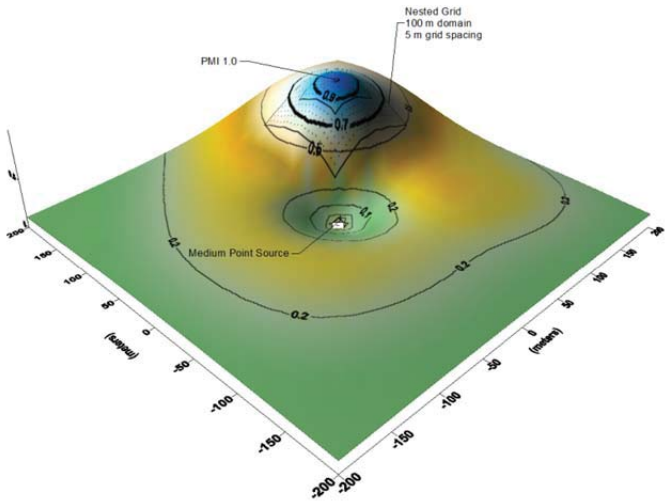
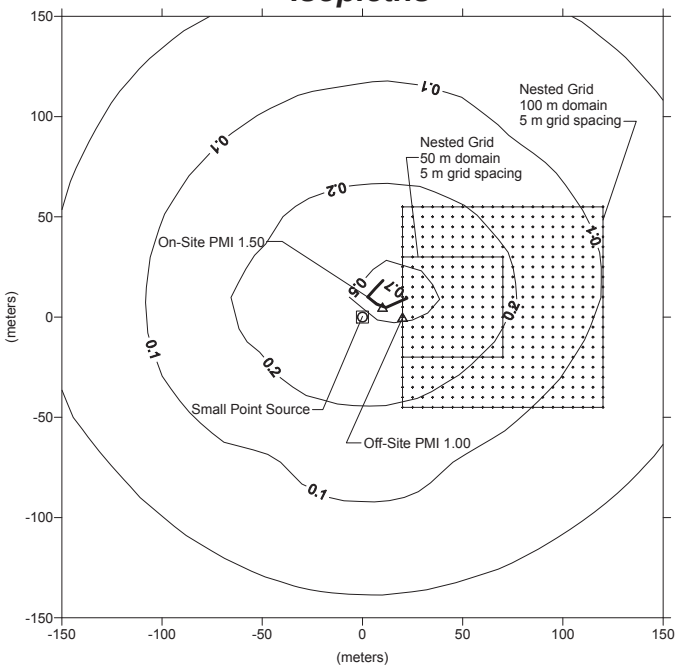


Figure AP C-3.3.1 – Small Point Source – Costa Mesa

Isopleths



Surface

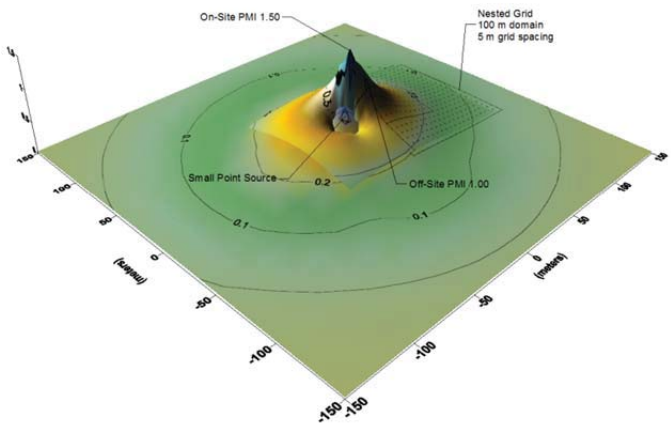
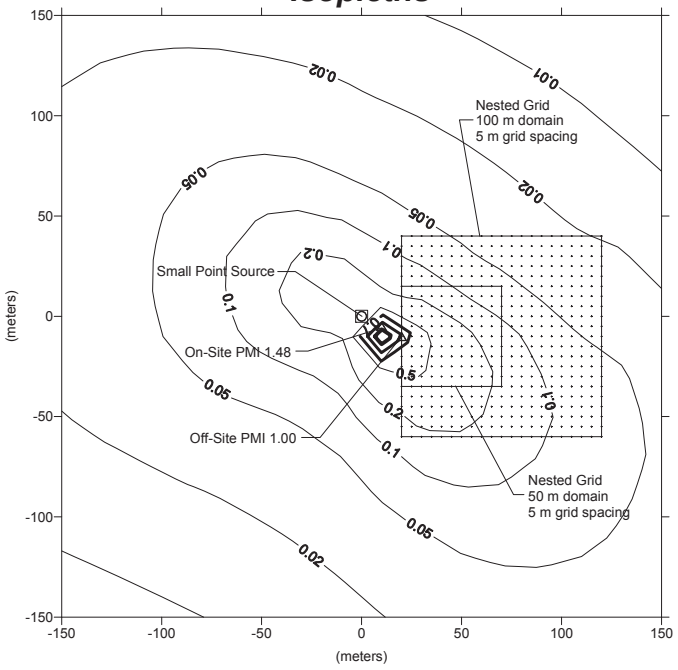


Figure AP C-3.3.2 – Small Point Source – Fresno Air Terminal

Isopleths



Surface

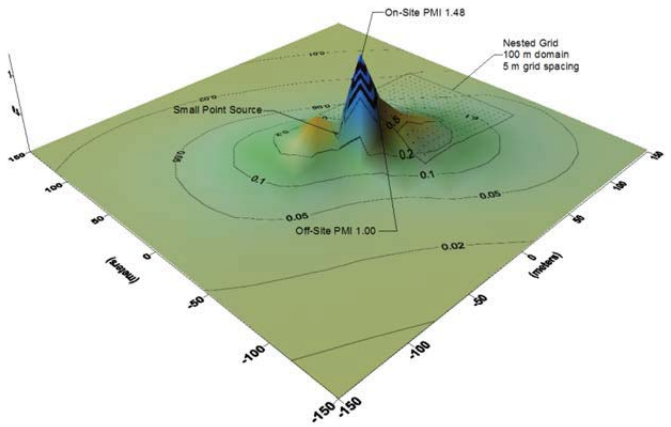
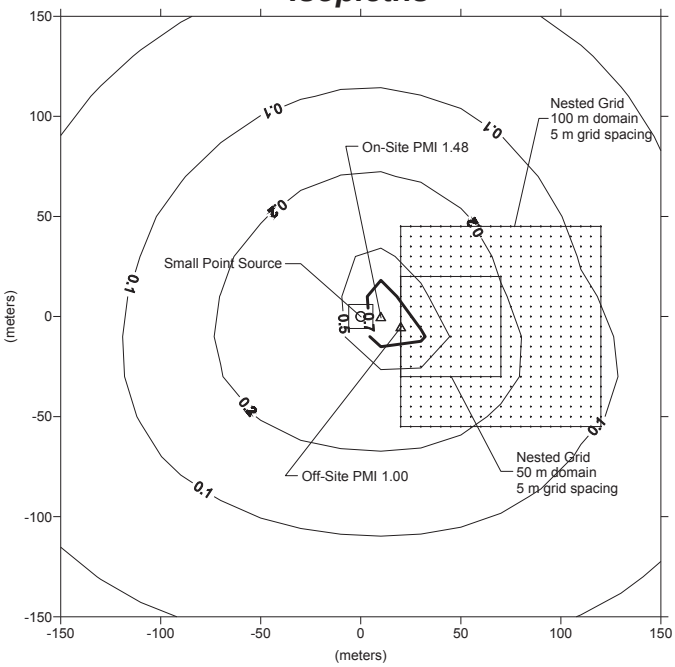


Figure AP C-3.3.3 – Small Point Source – Kearny Mesa

Isopleths



Surface

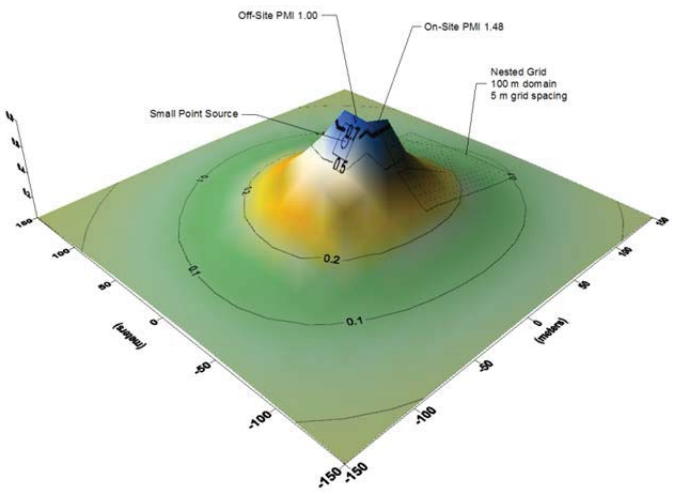
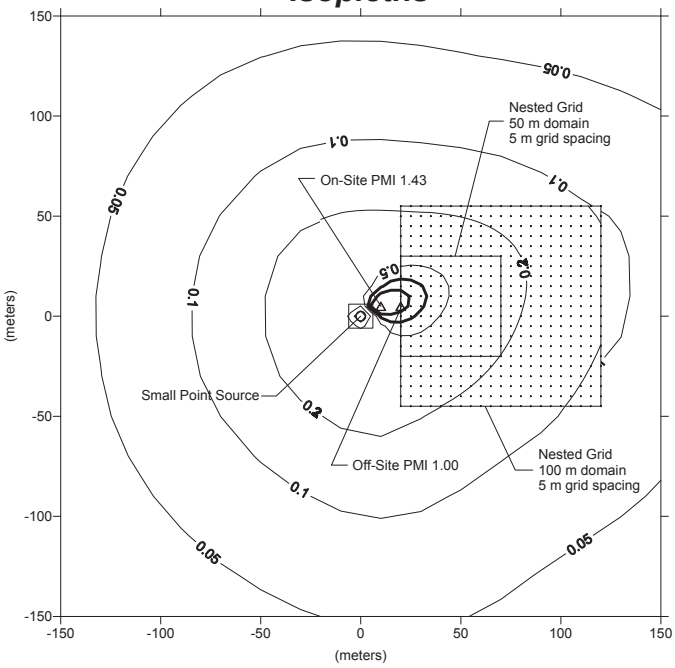


Figure AP C-3.3.4 – Small Point Source – Lynwood

Isopleths



Surface

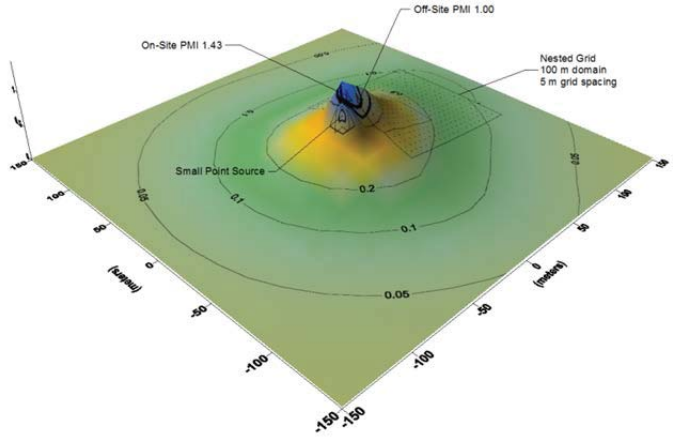
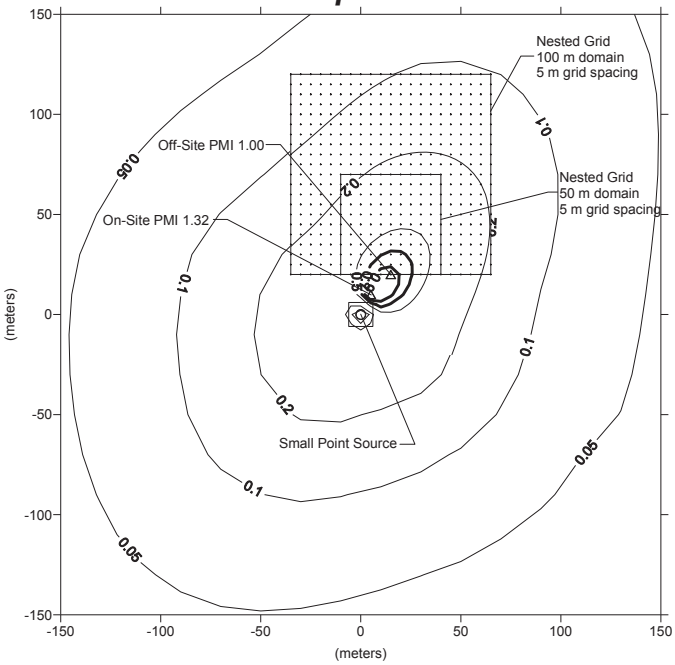


Figure AP C-3.3.5 – Small Point Source – San Bernardino

Isopleths



Surface

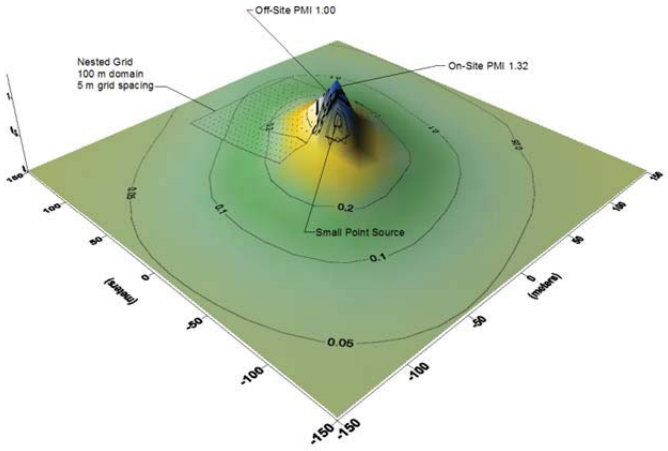
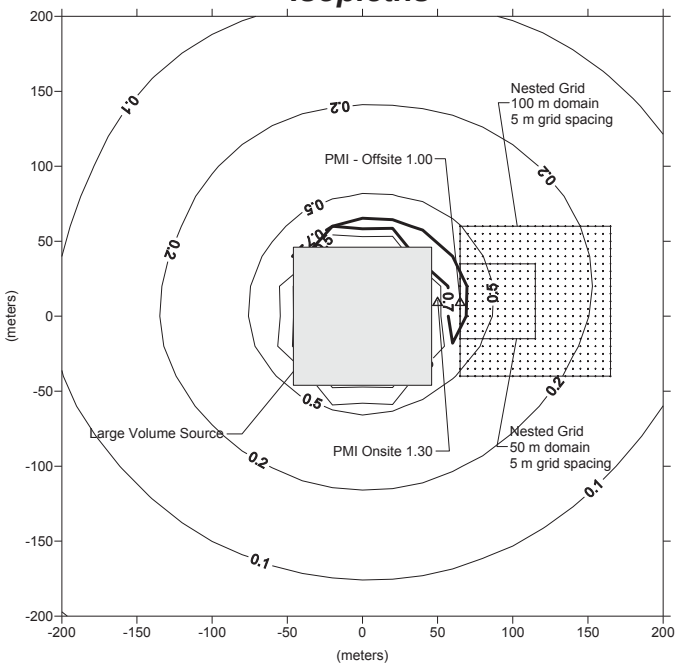


Figure AP C-3.4.1 – Large Volume Source – Costa Mesa

Isopleths



Surface

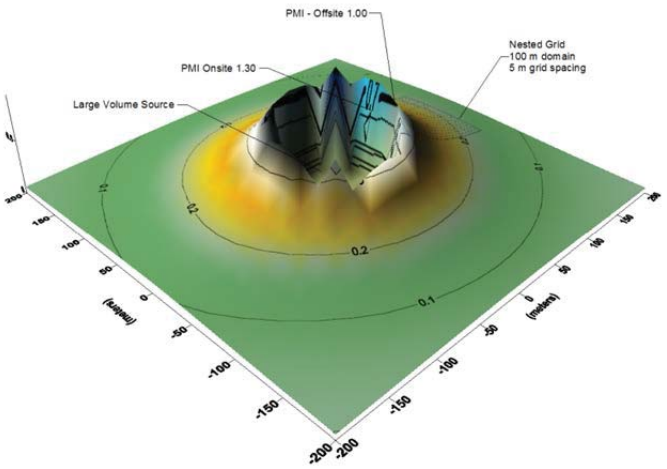
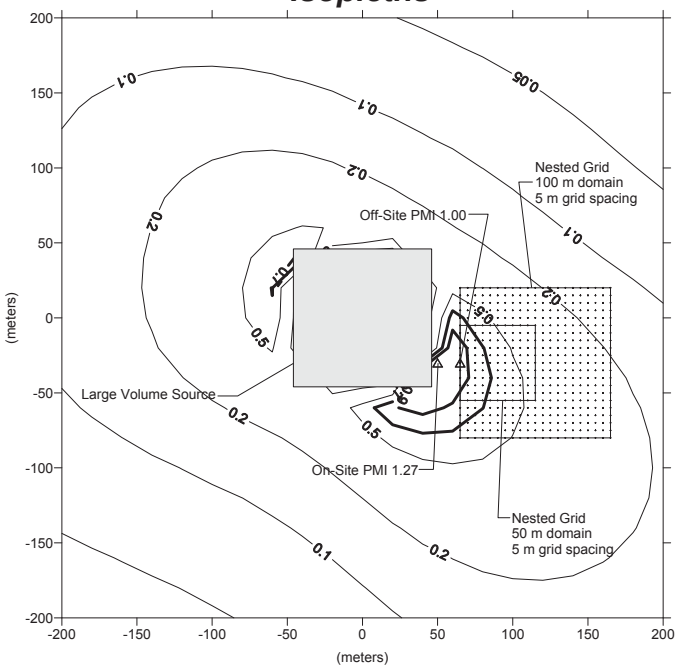


Figure AP C-3.4.2 – Large Volume Source – Fresno Air Terminal

Isopleths



Surface

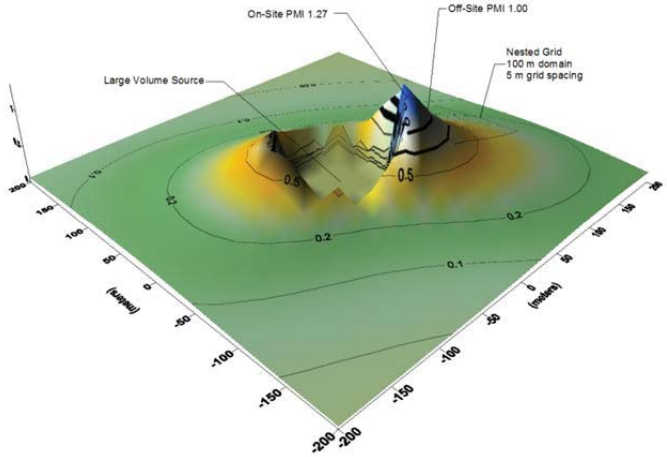
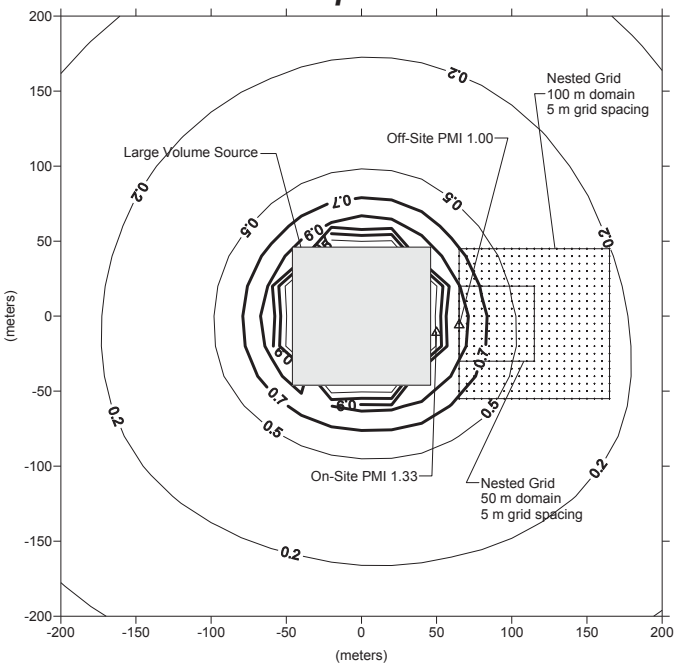


Figure AP C-3.4.3 – Large Volume Source – Kearny Mesa

Isopleths



Surface

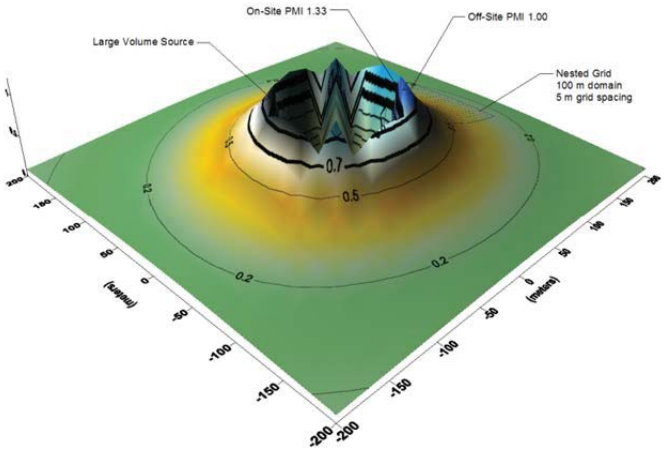
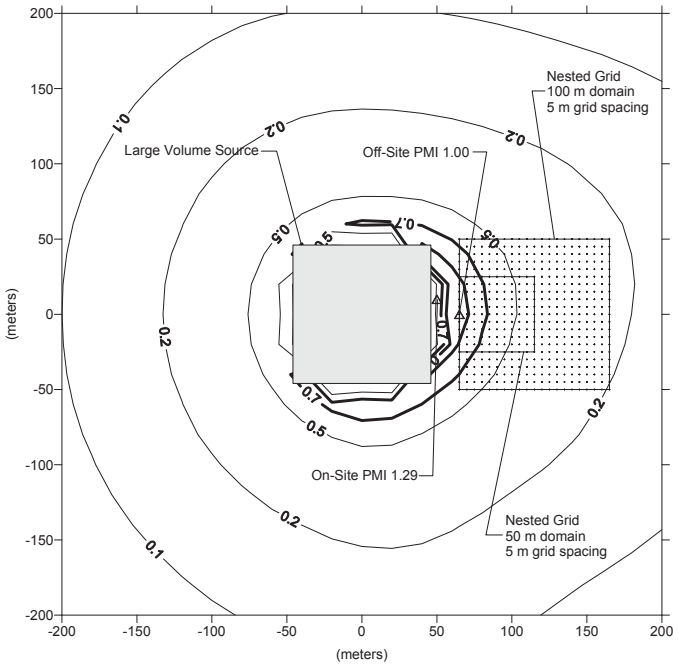


Figure AP C-3.4.4 – Large Volume Source – Lynwood

Isopleths



Surface

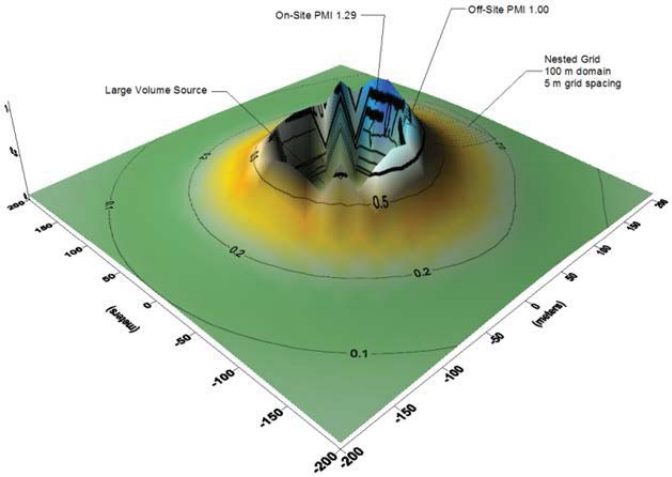
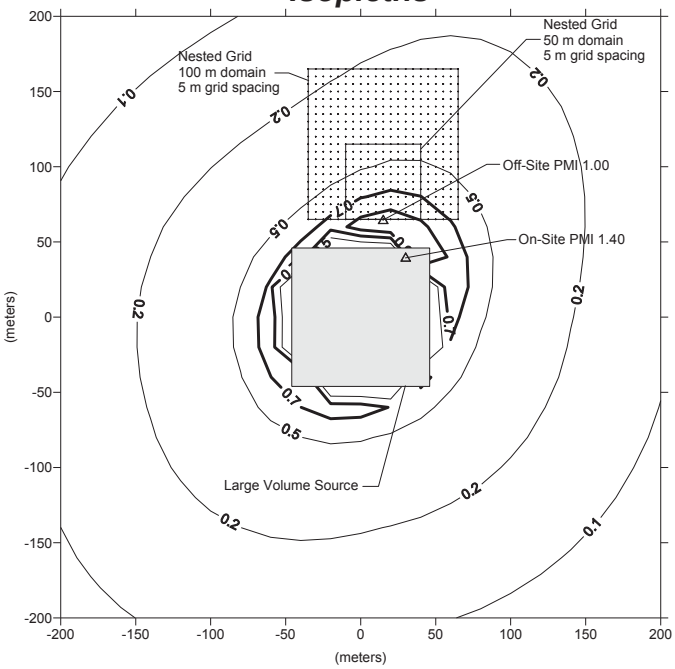


Figure AP C-3.4.5 – Large Volume Source – San Bernardino

Isopleths



Surface

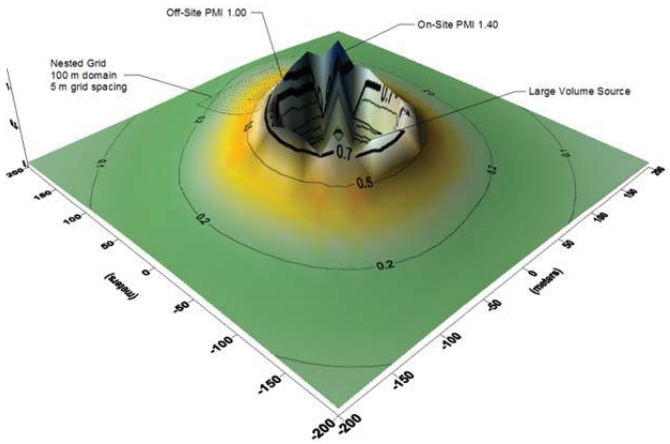


Figure AP C-3.5.1 – Medium Volume Source – Costa Mesa

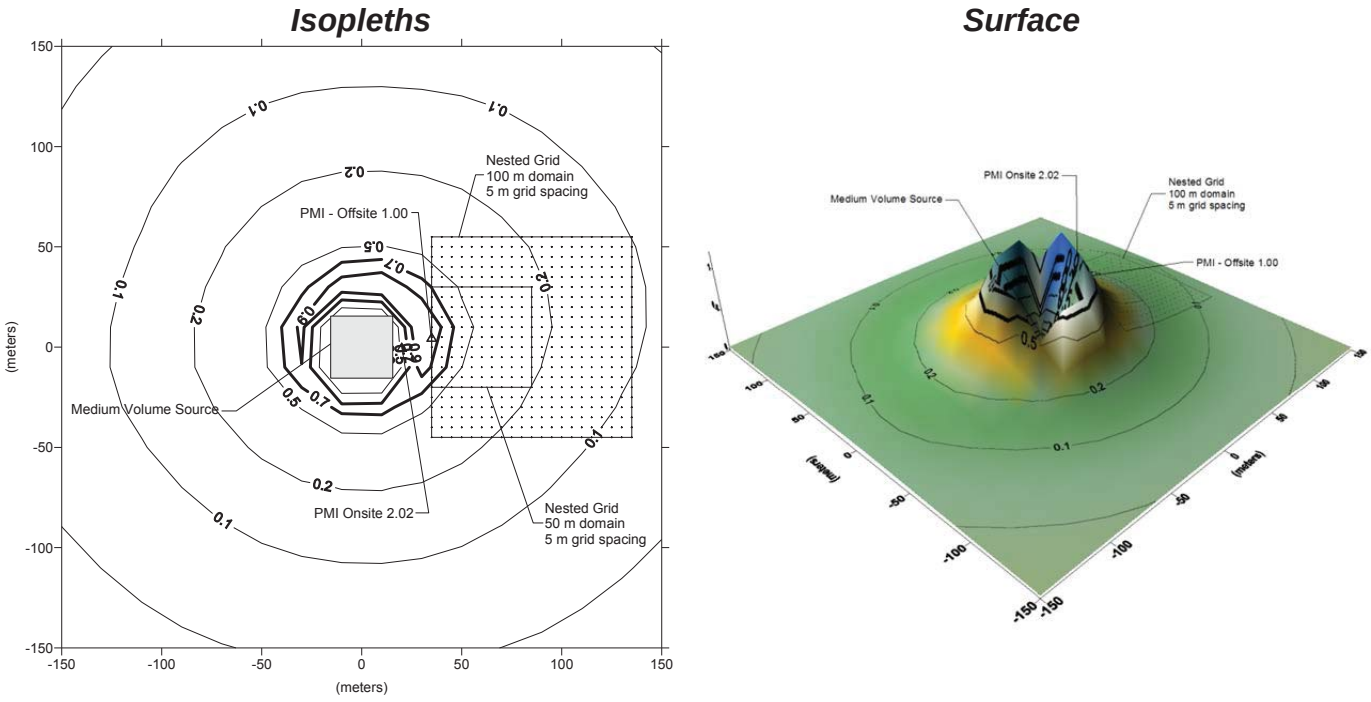


Figure AP C-3.5.2 – Medium Volume Source – Fresno Air Terminal

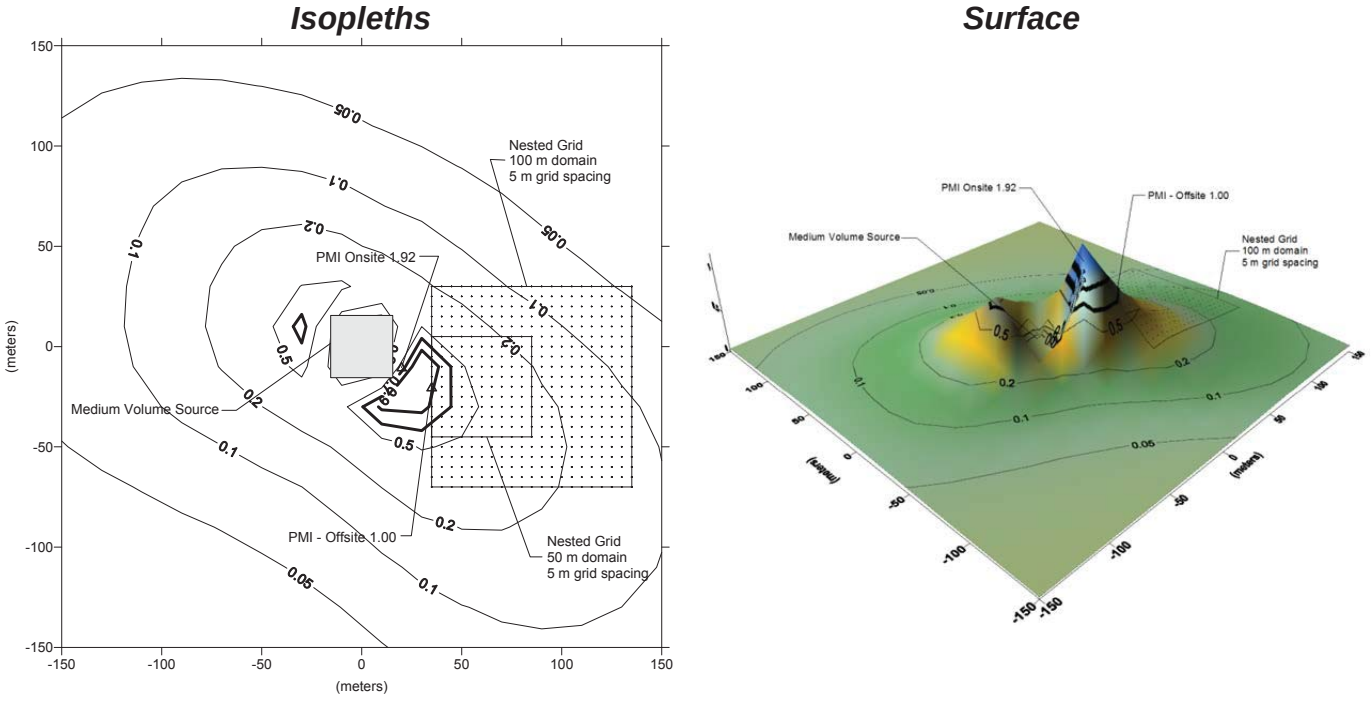


Figure AP C-3.5.3 – Medium Volume Source – Kearny Mesa

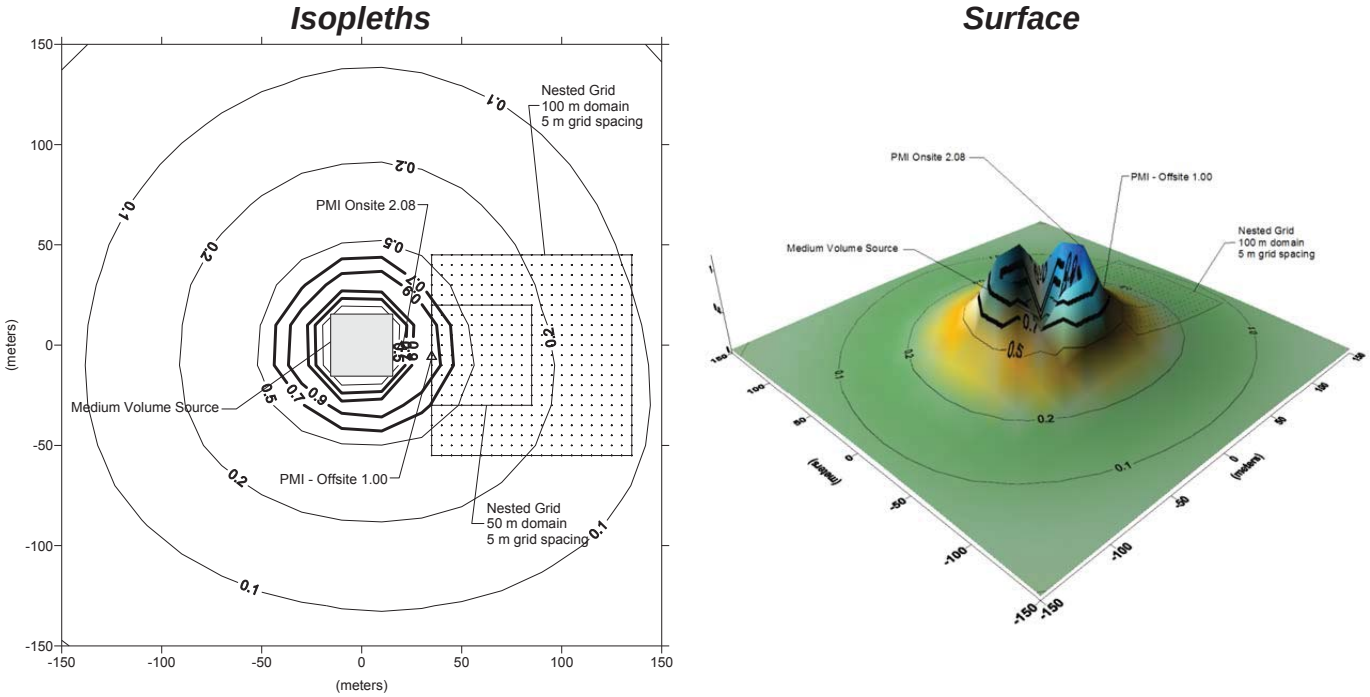


Figure AP C-3.5.4 – Medium Volume Source – Lynnwood

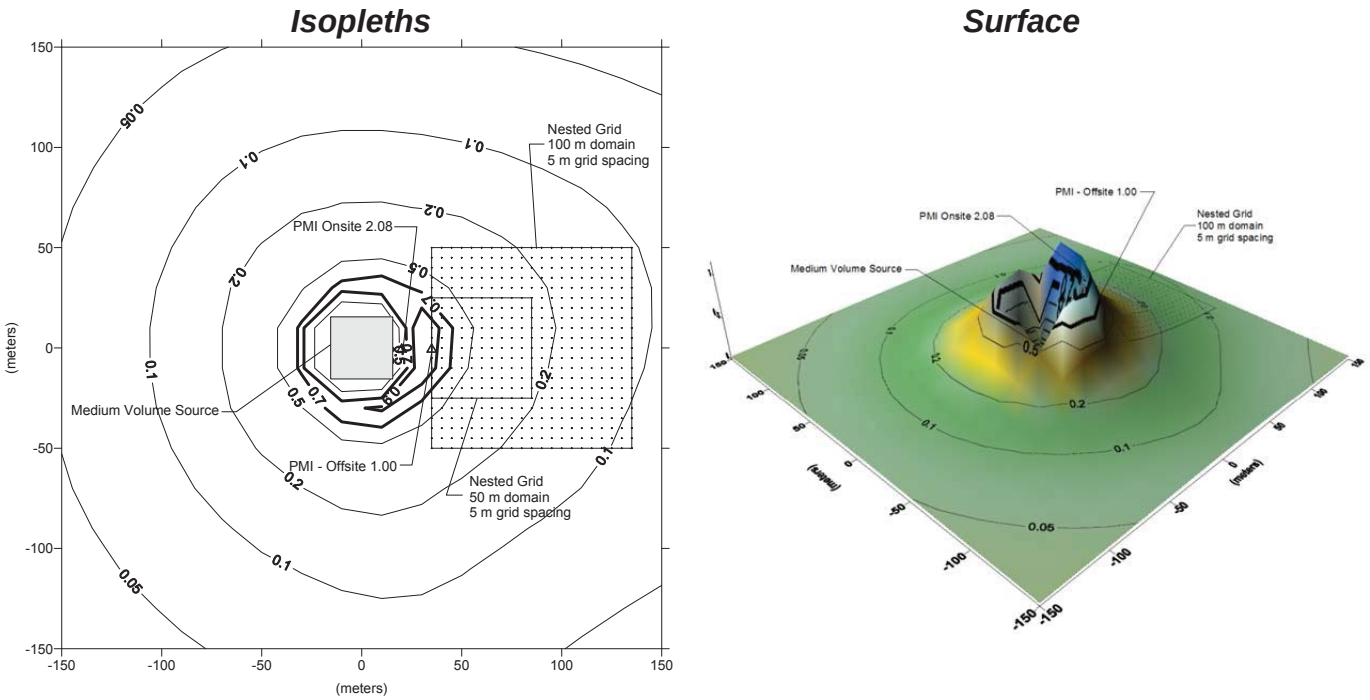
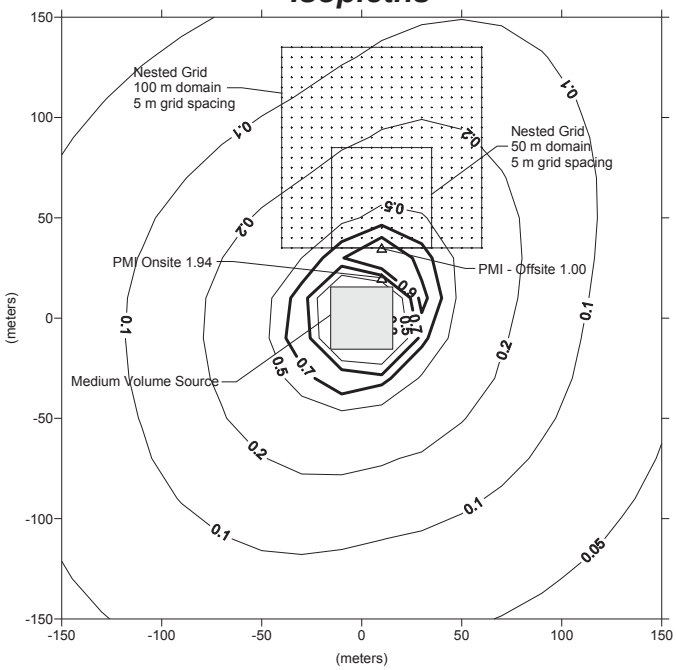


Figure AP C-3.5.5 – Medium Volume Source – San Bernardino

Isopleths



Surface

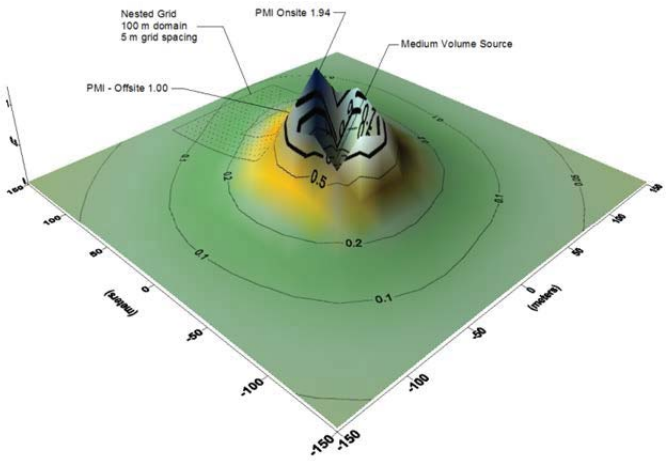
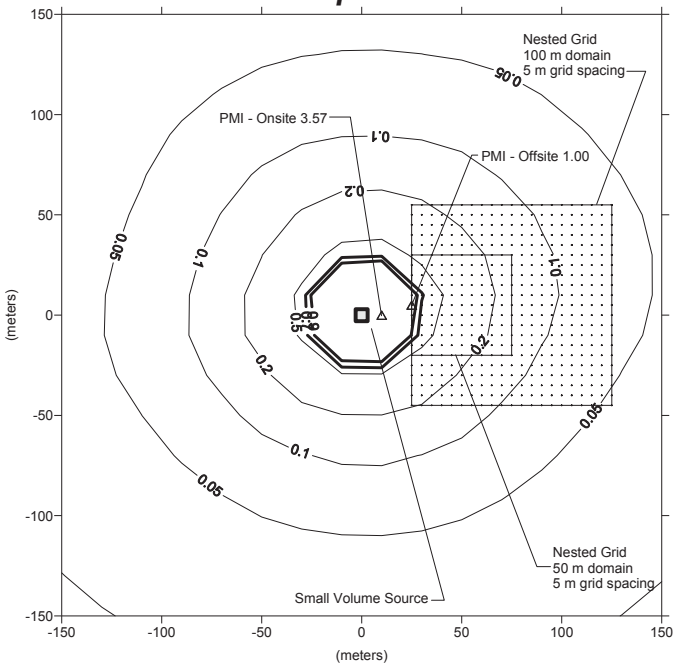


Figure AP C-3.6.1 – Small Volume Source – Costa Mesa

Isopleths



Surface

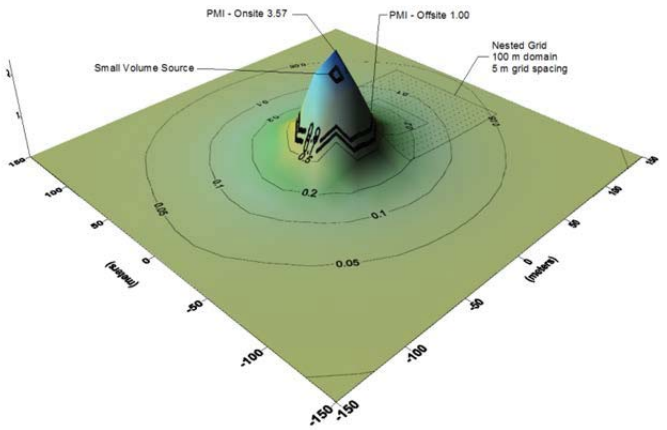
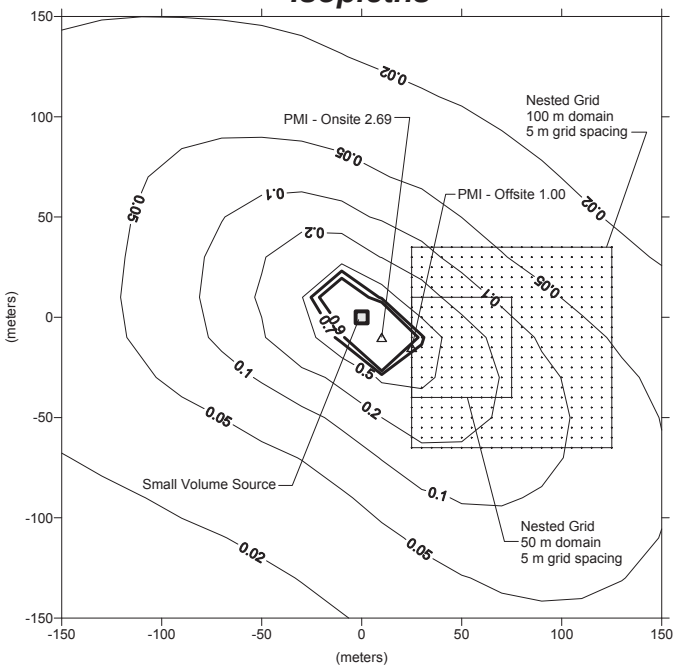


Figure AP C-3.6.2 – Small Volume Source – Fresno Air Terminal

Isopleths



Surface

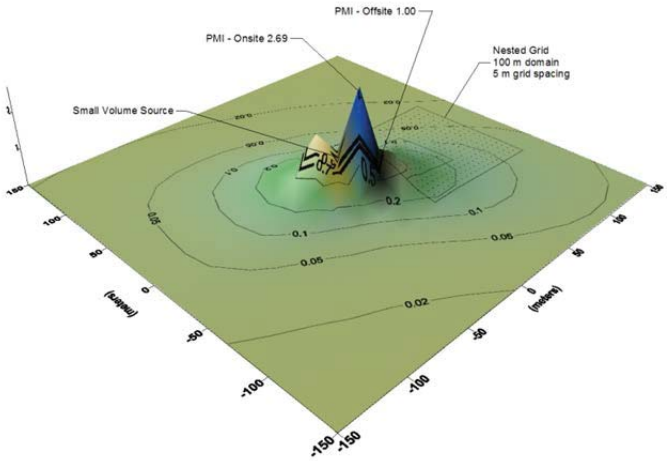
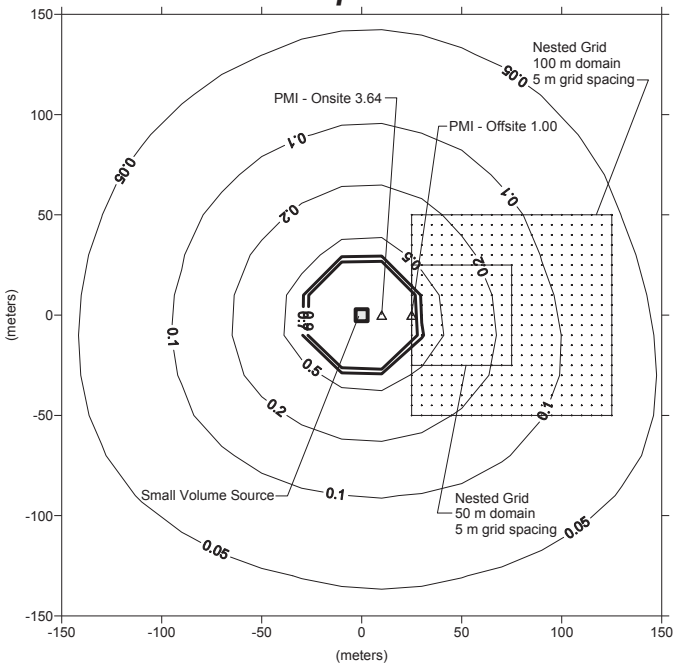


Figure AP C-3.6.3 – Small Volume Source – Kearny Mesa

Isopleths



Surface

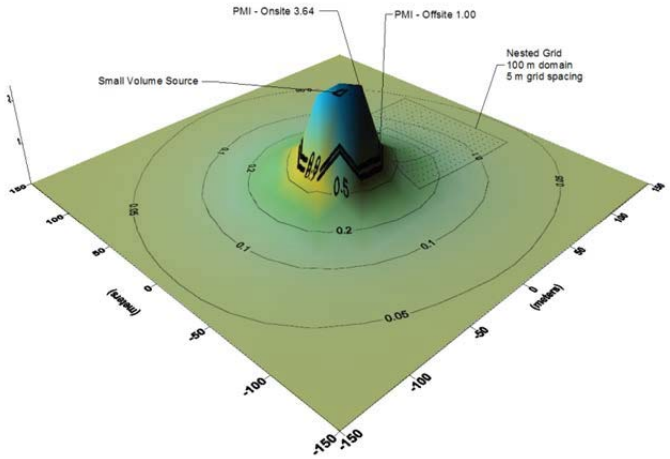
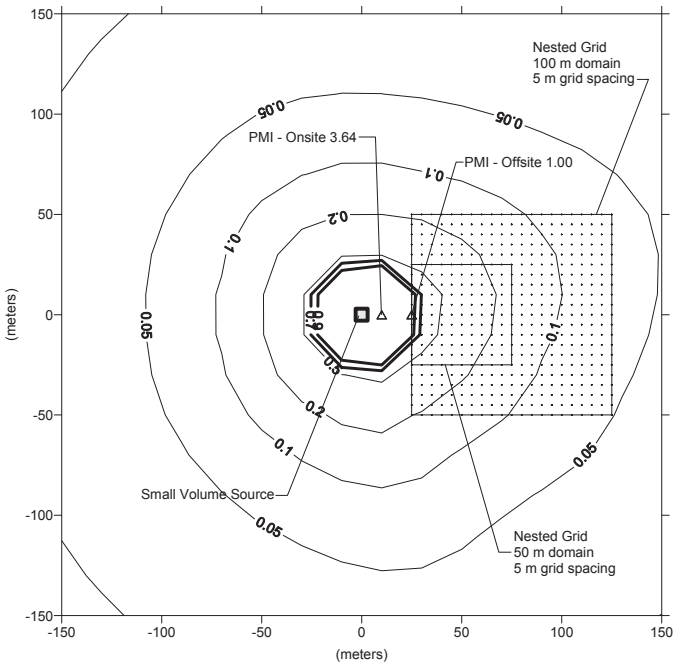


Figure AP C-3.6.4 – Small Volume Source – Lynnwood

Isopleths



Surface

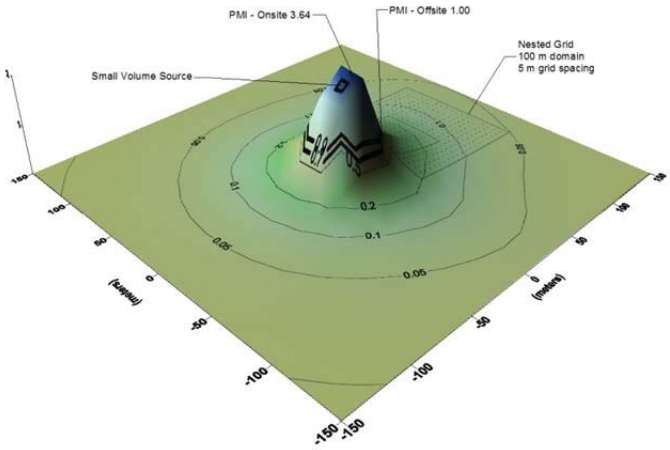
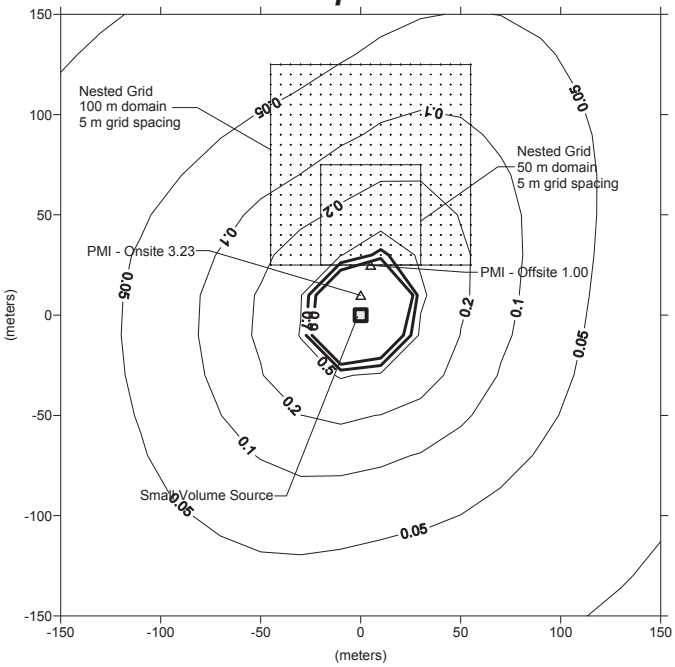


Figure AP C-3.6.5 – Small Volume Source – San Bernardino

Isopleths



Surface

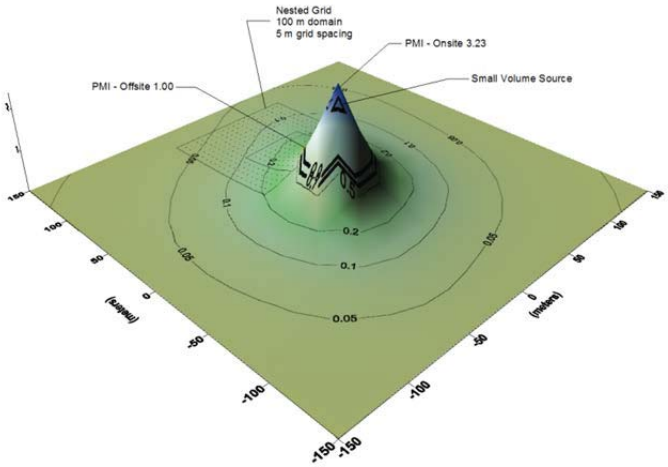
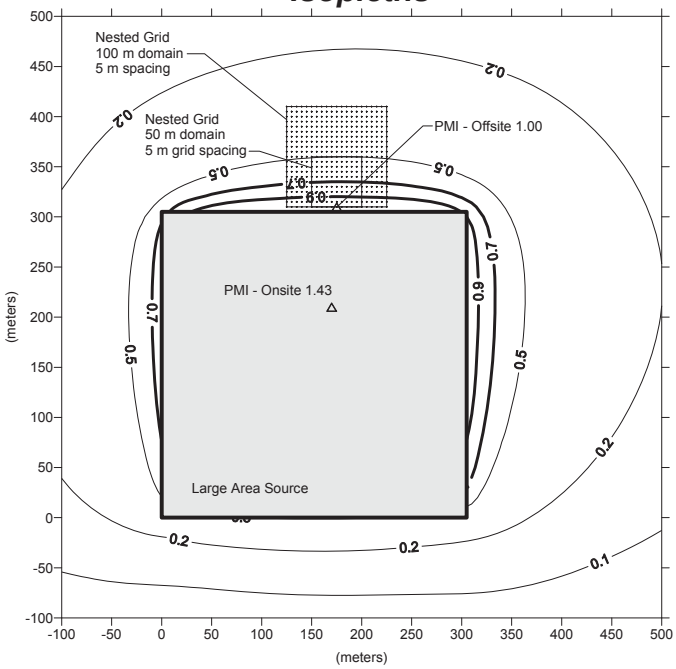


Figure AP C-3.7.1 – Large Area Source – Costa Mesa

Isopleths



Surface

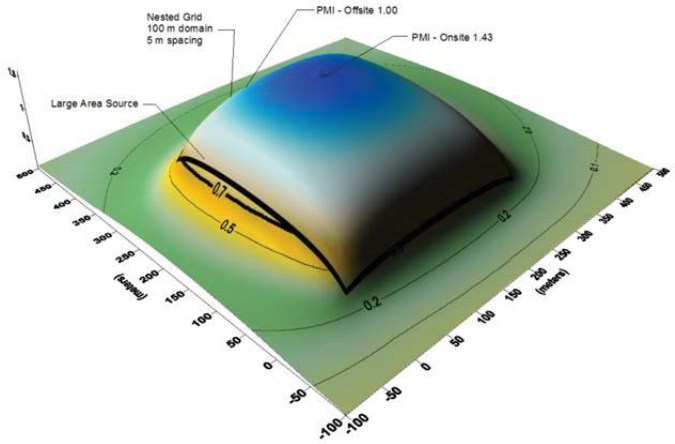
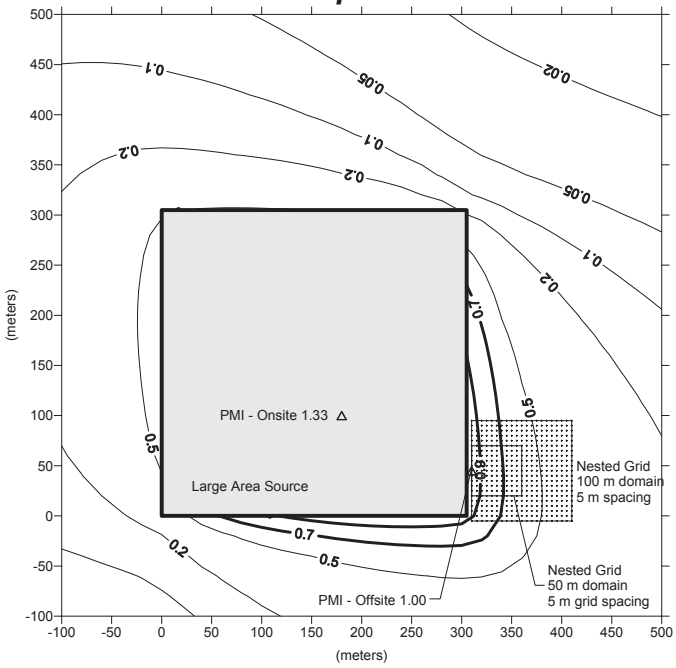


Figure AP C-3.7.2 – Large Area Source – Fresno Air Terminal

Isopleths



Surface

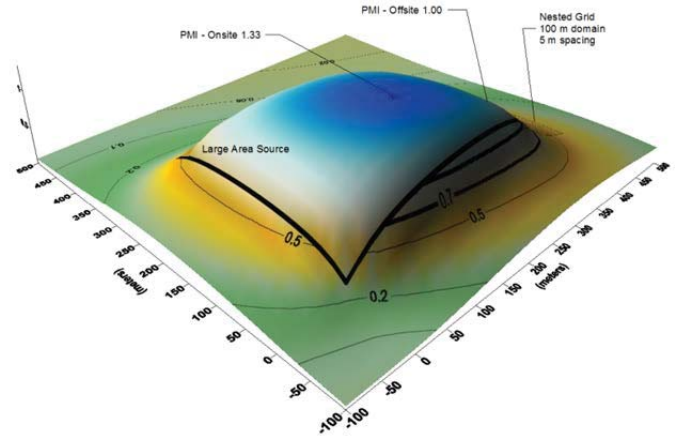
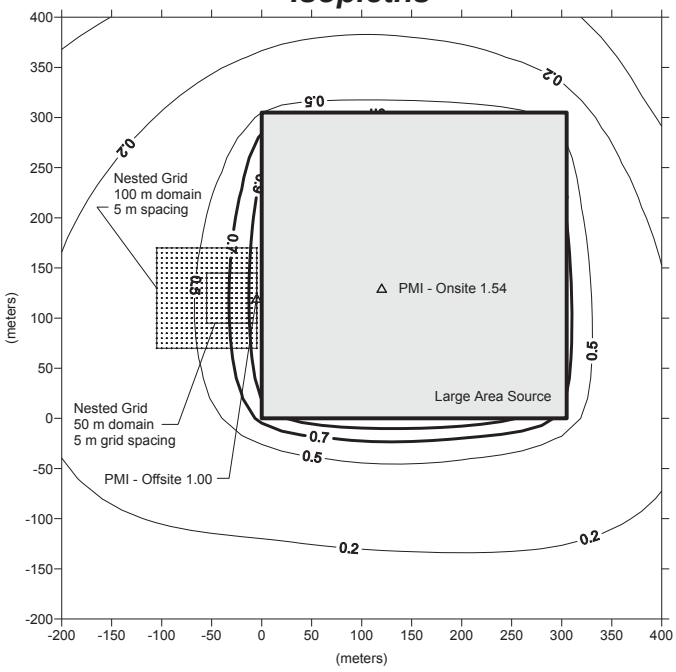


Figure AP C-3.7.3 – Large Area Source – Kearny Mesa

Isopleths



Surface

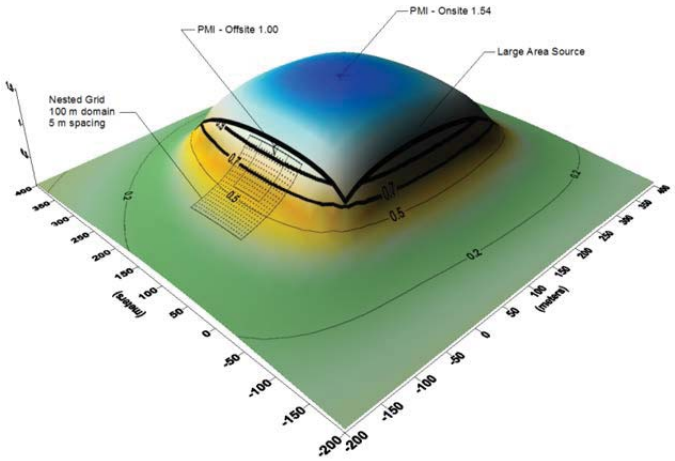
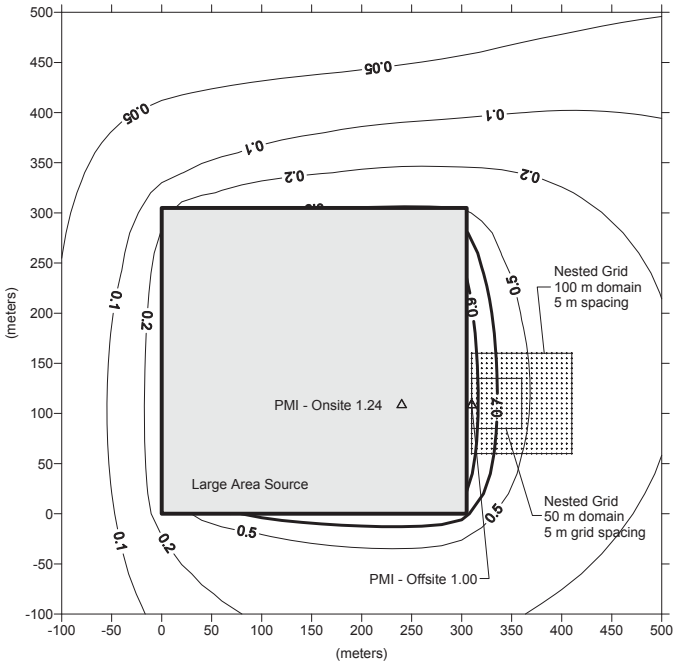


Figure AP C-3.7.4 – Large Area Source – Lynwood

Isopleths



Surface

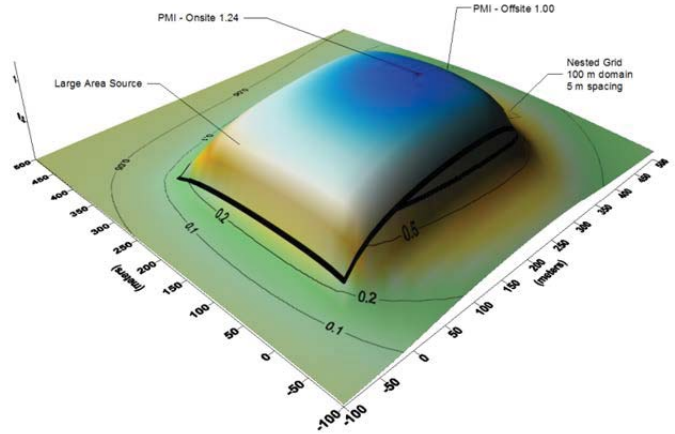
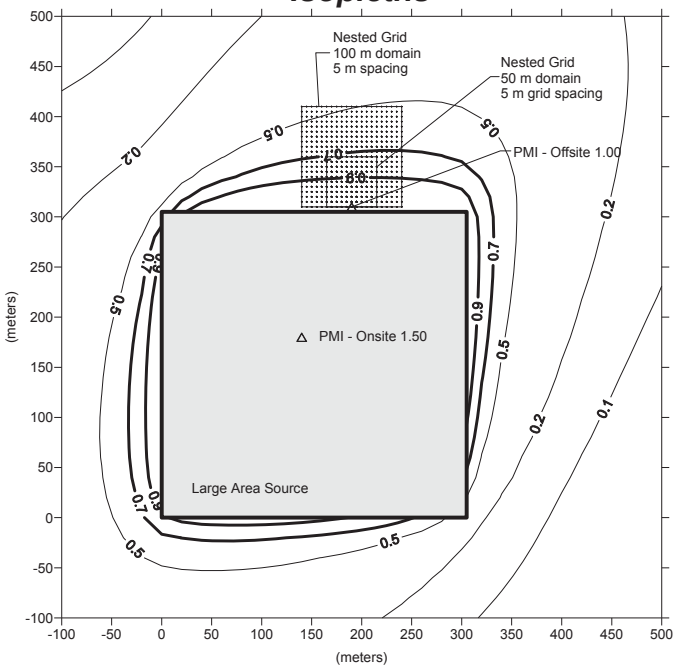


Figure AP C-3.7.5 – Large Area Source – San Bernardino

Isopleths



Surface

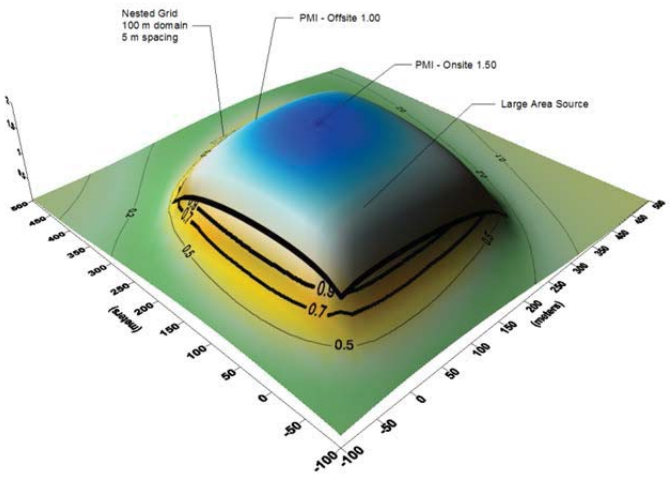


Figure AP C-3.8.1 – Medium Area Source – Costa Mesa

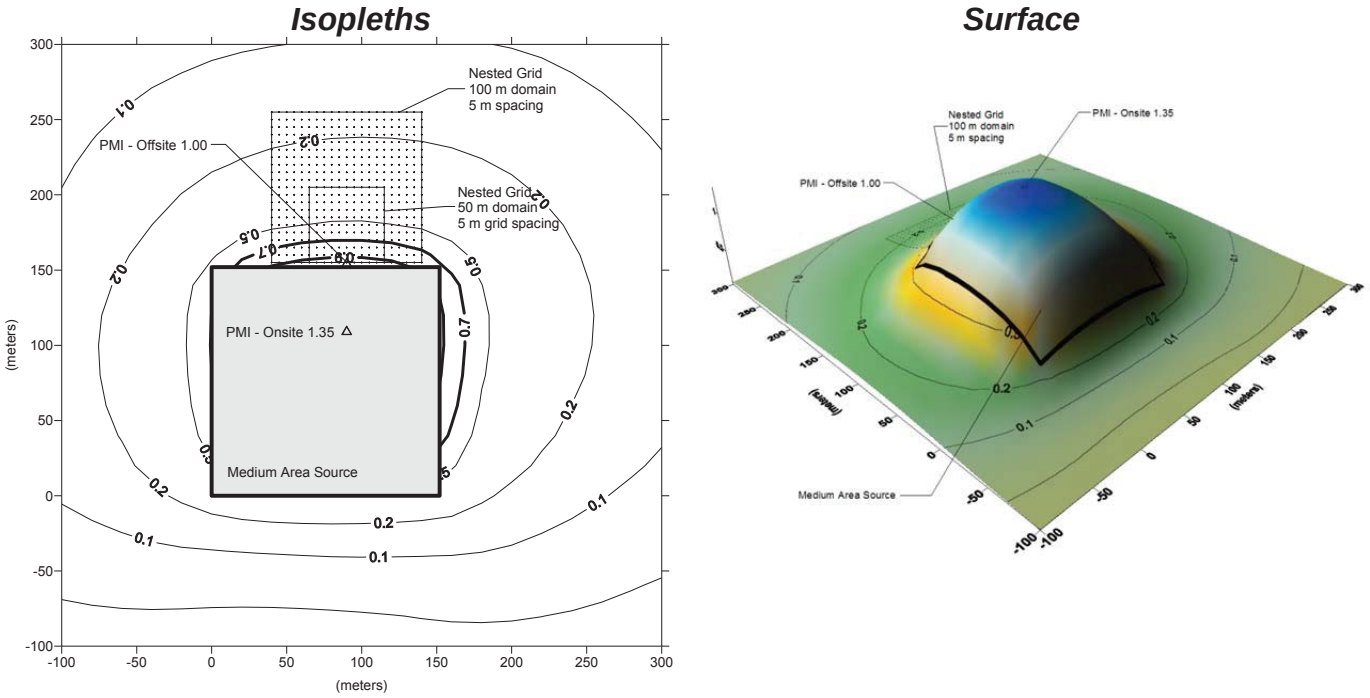


Figure AP C-3.8.2 – Medium Area Source – Fresno Air Terminal

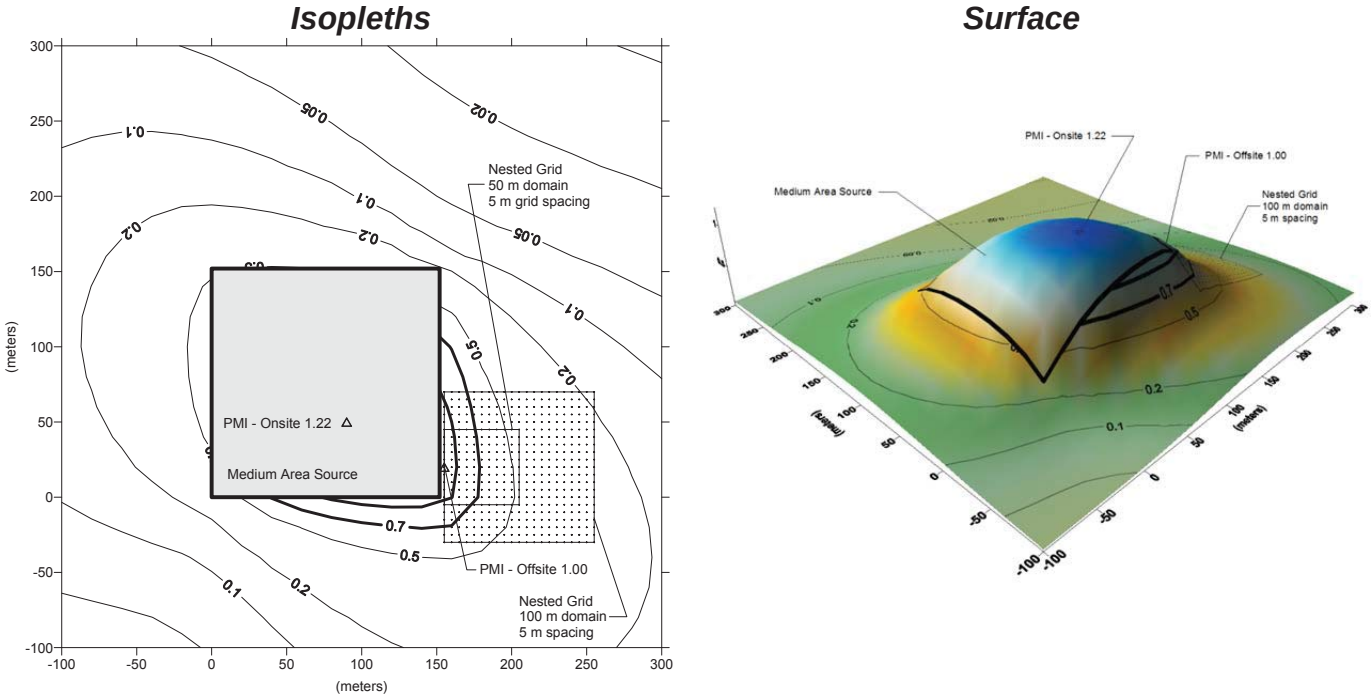
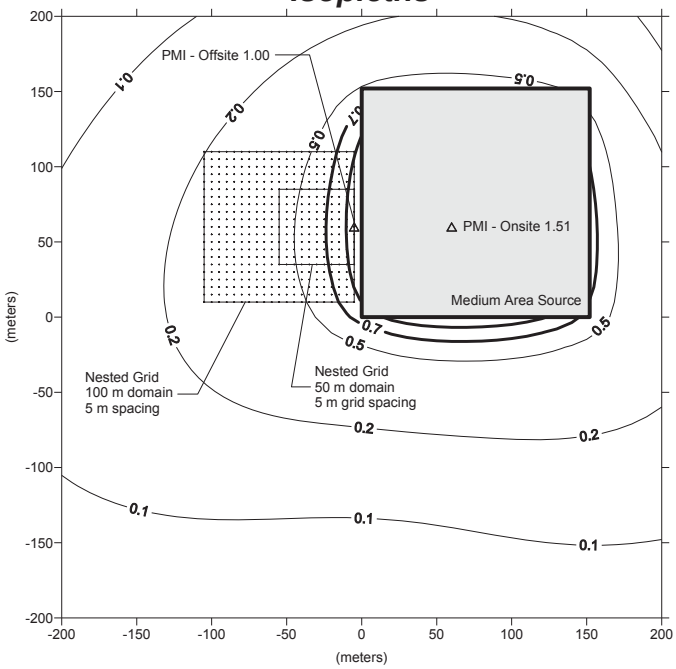


Figure AP C-3.8.3 – Medium Area Source – Kearny Mesa

Isopleths



Surface

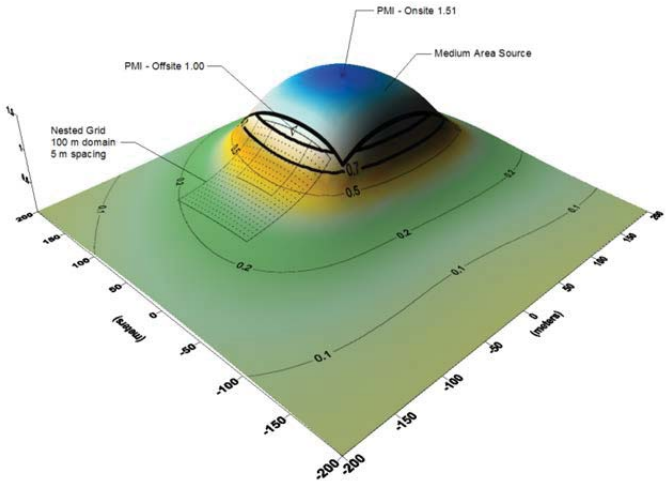
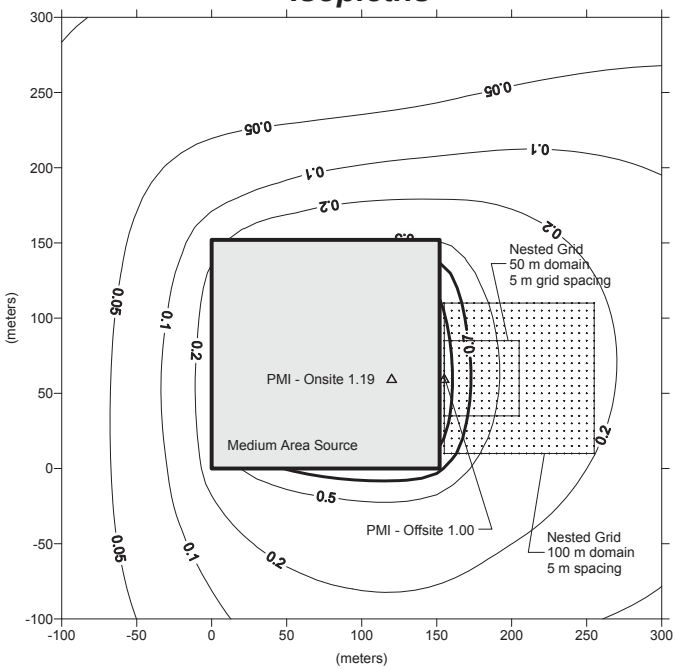


Figure AP C-3.8.4 – Medium Area Source – Lynwood

Isopleths



Surface

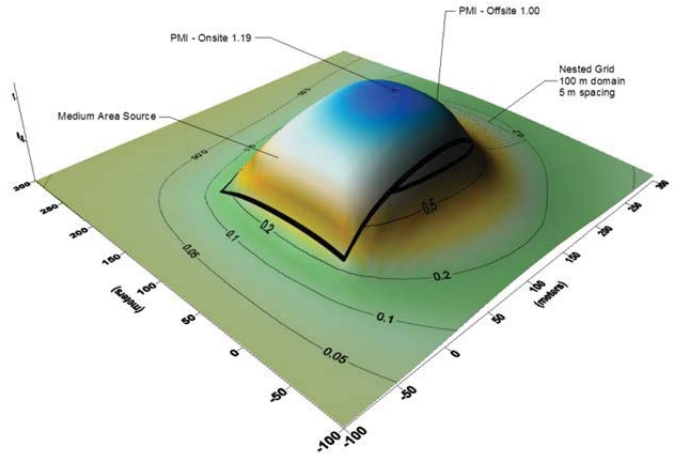
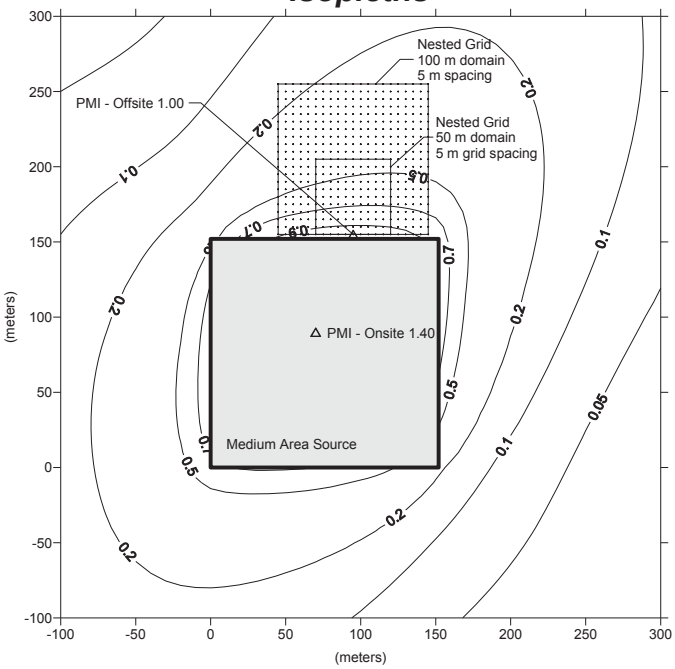


Figure AP C-3.8.5 – Medium Area Source – San Bernardino

Isopleths



Surface

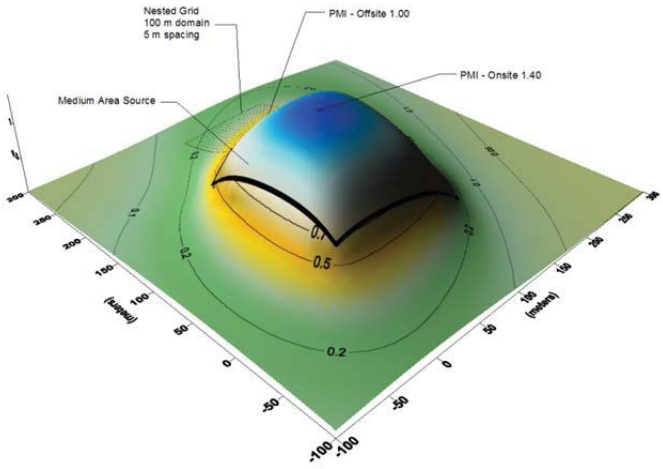
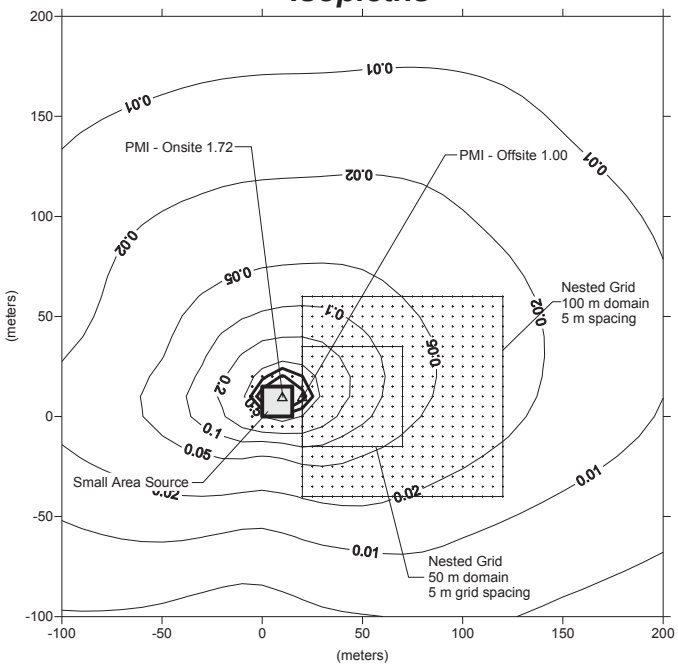


Figure AP C-3.9.1 – Small Area Source – Costa Mesa

Isopleths



Surface

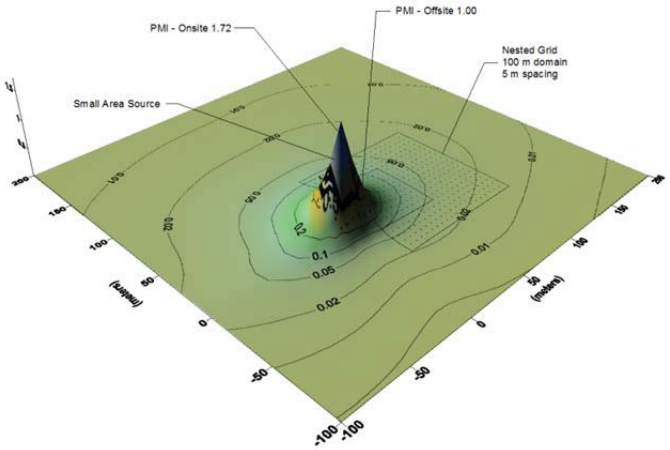
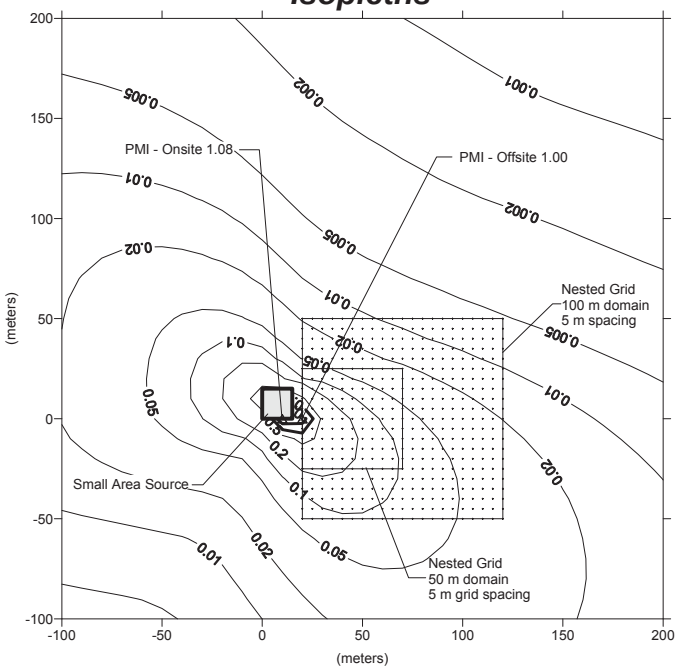


Figure AP C-3.9.2 – Small Area Source – Fresno Air Terminal

Isopleths



Surface

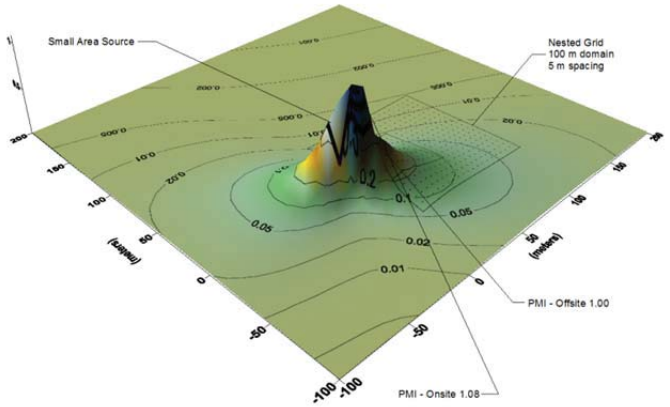
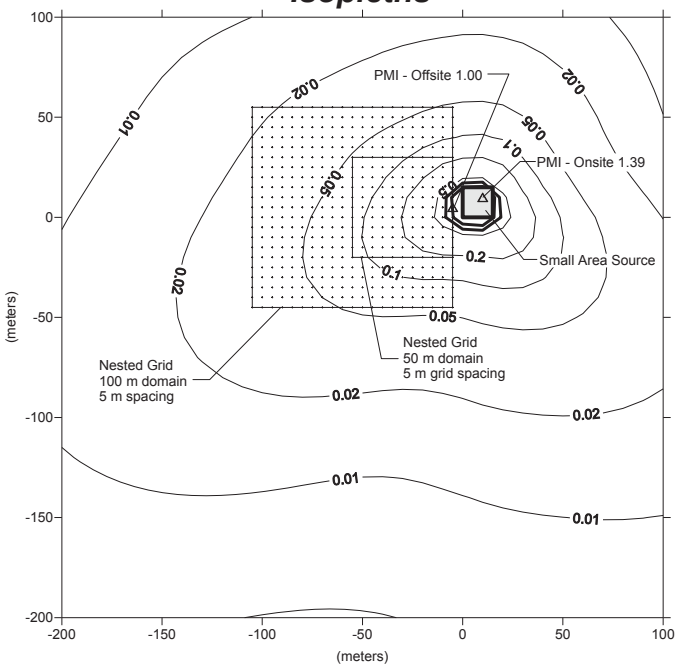


Figure AP C-3.9.3 – Small Area Source – Kearny Mesa

Isopleths



Surface

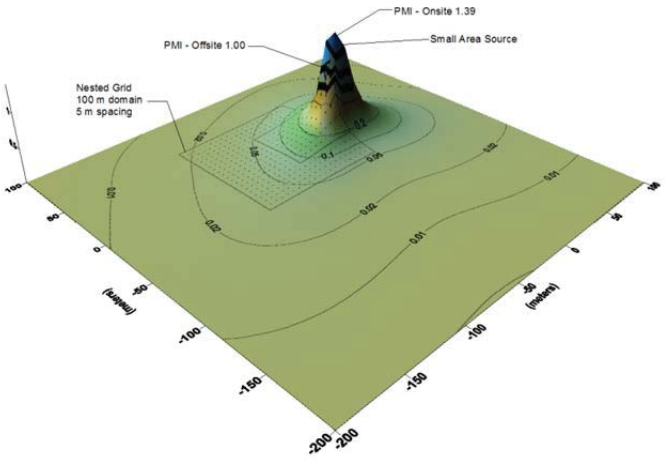
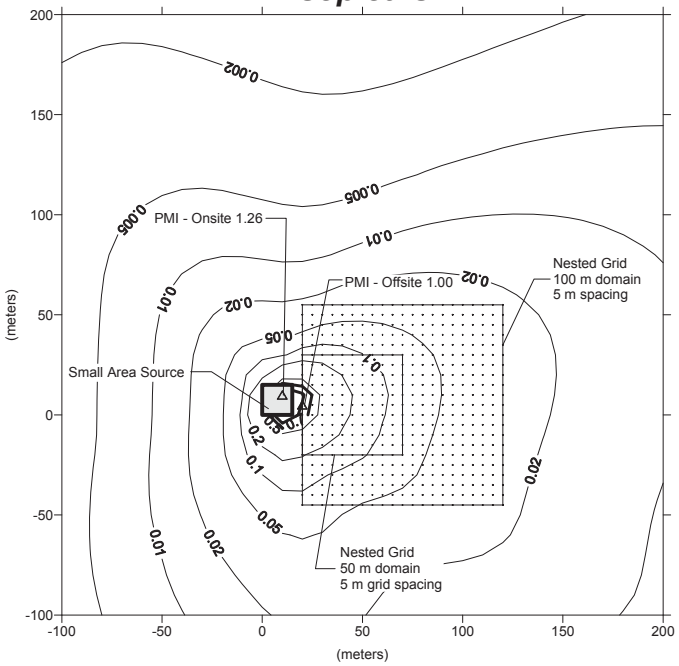


Figure AP C-3.9.4 – Small Area Source – Lynwood

Isopleths



Surface

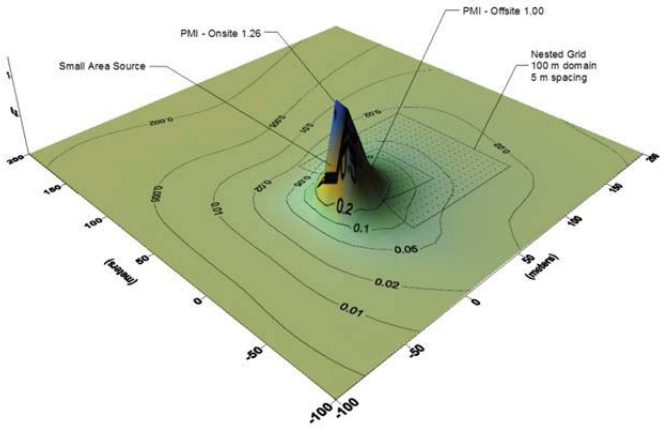
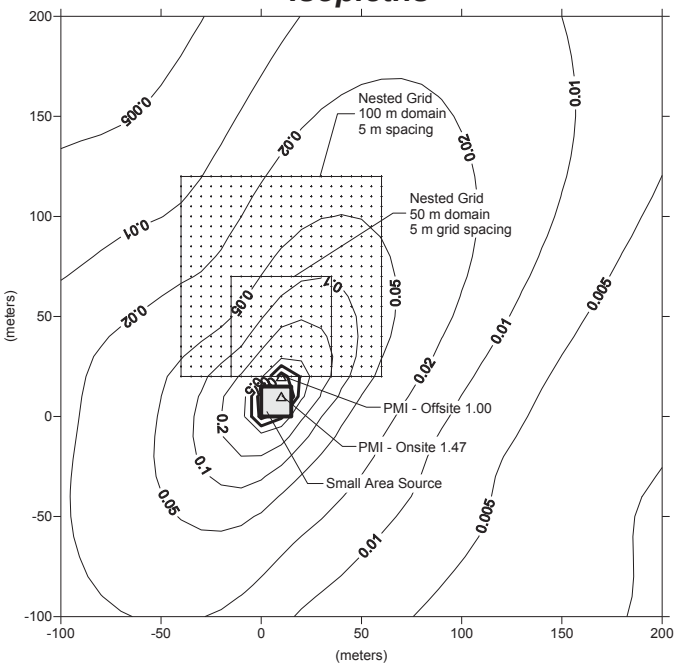


Figure AP C-3.9.5 – Small Area Source – San Bernardino

Isopleths



Surface

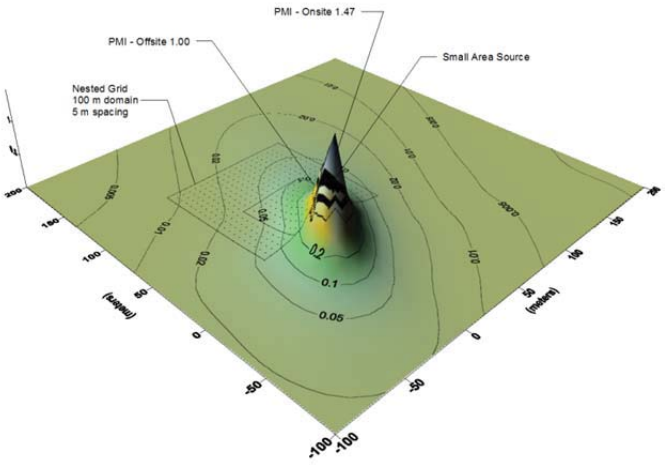


Figure AP C-3.10.1 – Large Line Source, CALINE – Costa Mesa

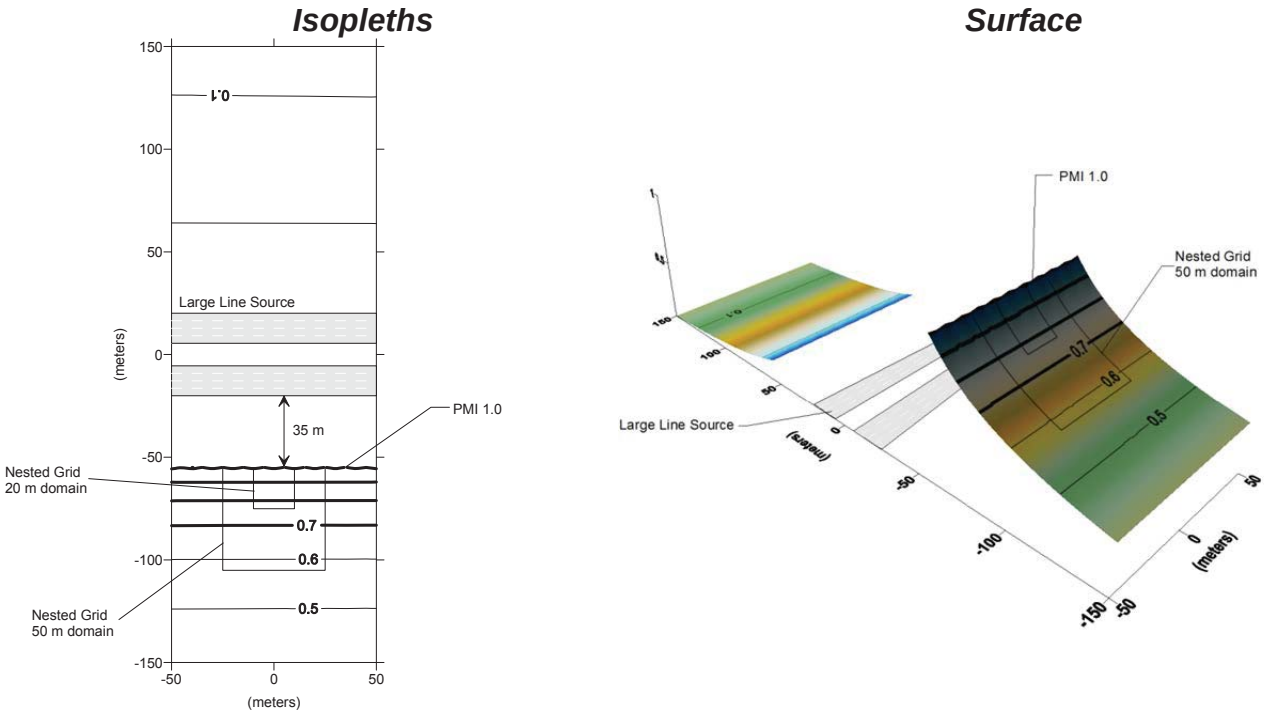


Figure AP C-3.10.2 – Large Line Source, CALINE – Fresno Air Terminal

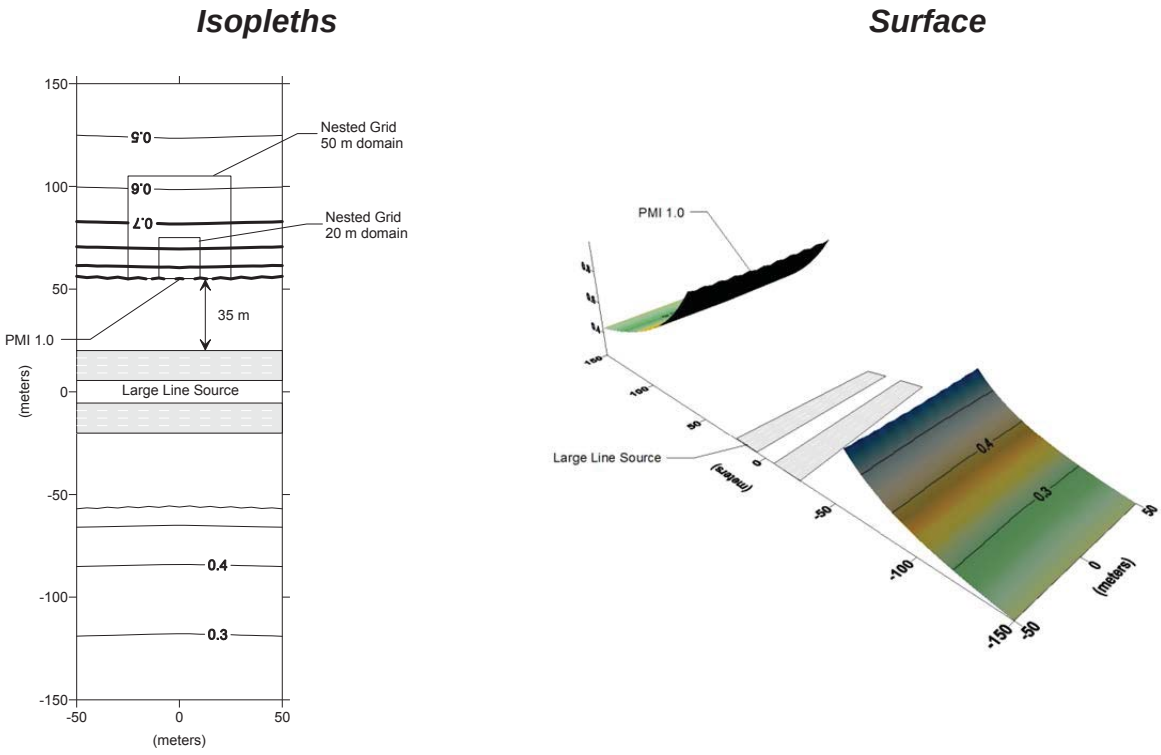


Figure AP C-3.10.3 – Large Line Source, CALINE – Kearny Mesa

Isopleths ***Surface***

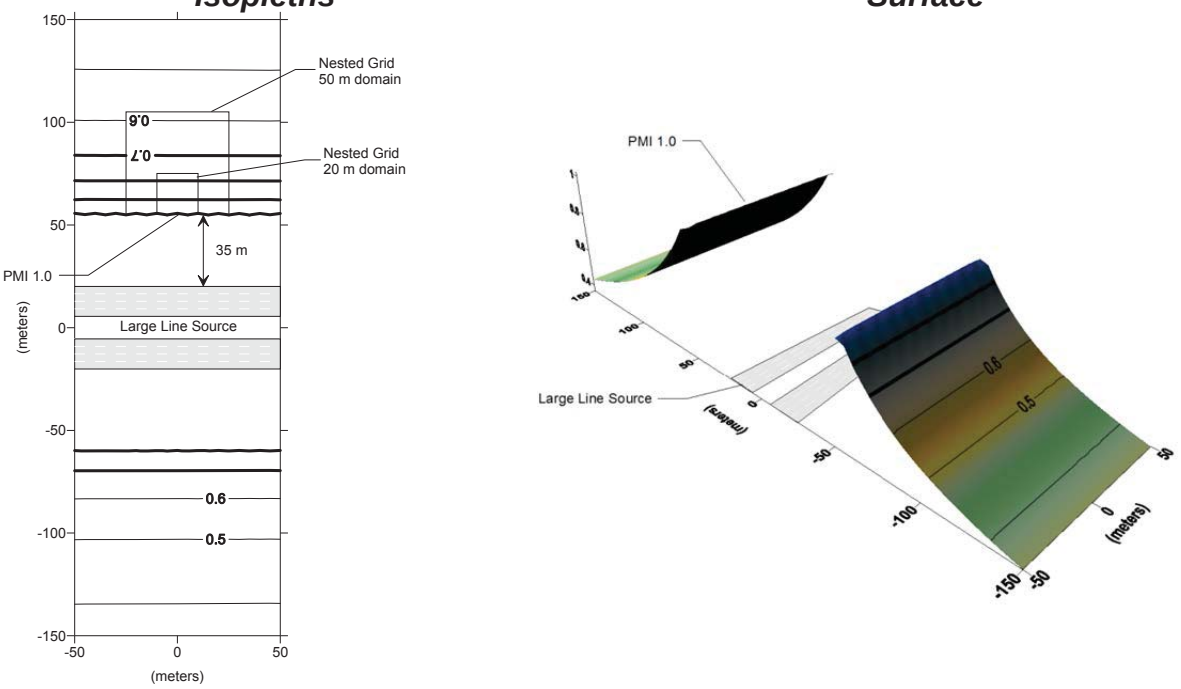


Figure AP C-3.10.4 – Large Line Source, CALINE – Lynwood

Isopleths ***Surface***

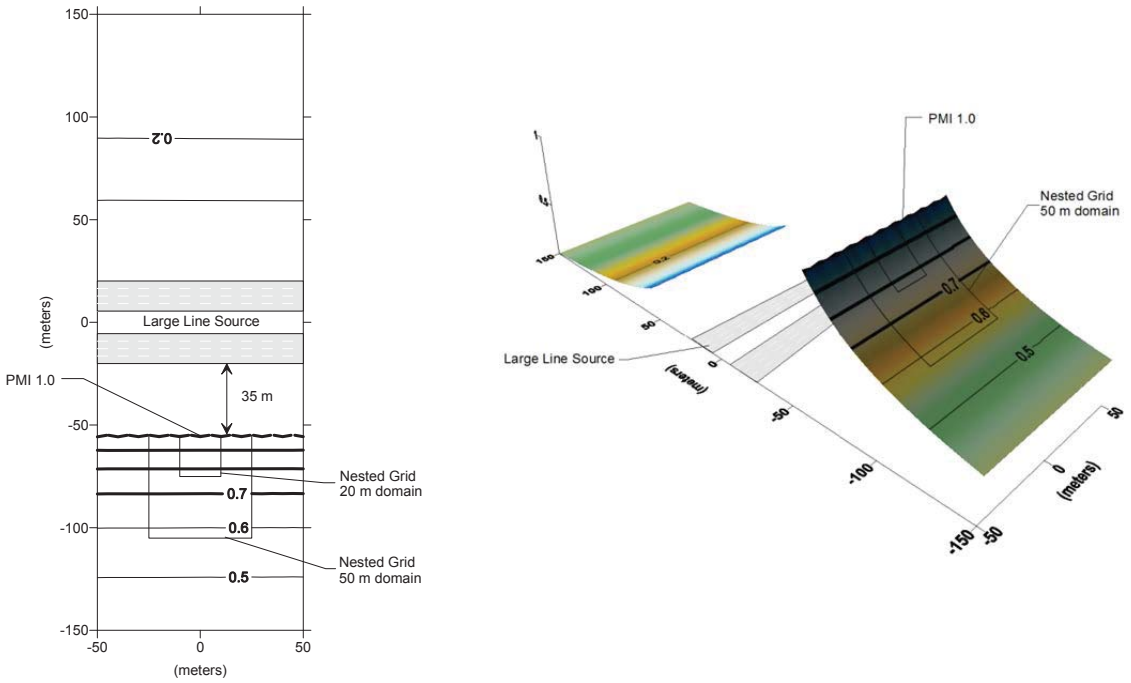
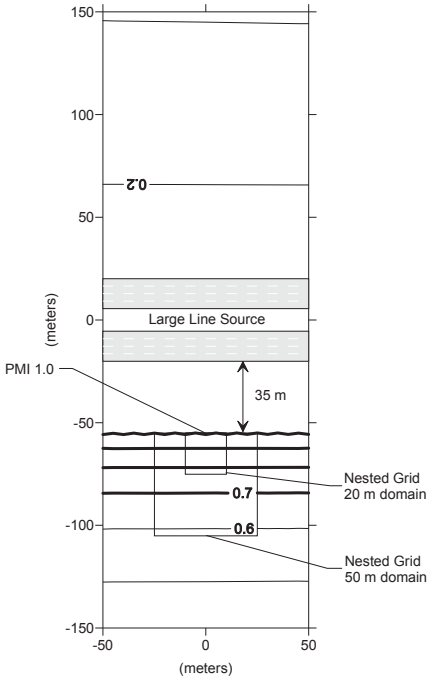


Figure AP C-3.10.5 – Large Line Source, CALINE – San Bernardino

Isopleths ***Surface***



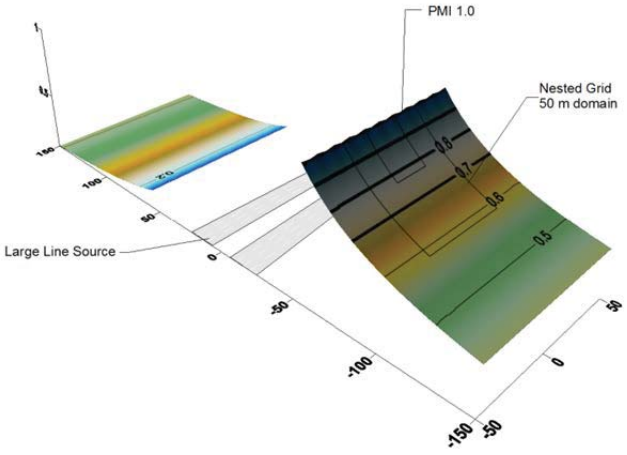
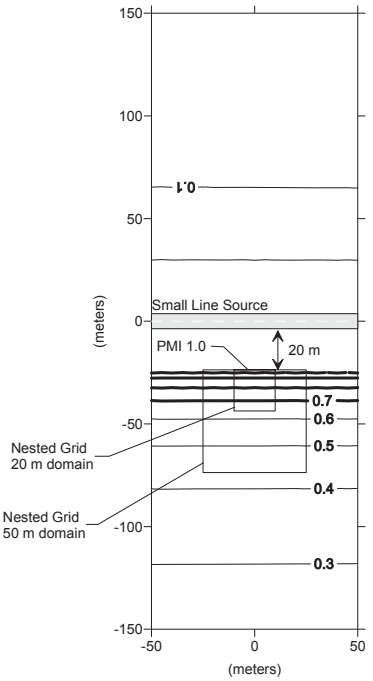


Figure AP C-3.11.1 – Small Line Source, CALINE – Costa Mesa

Isopleths ***Surface***



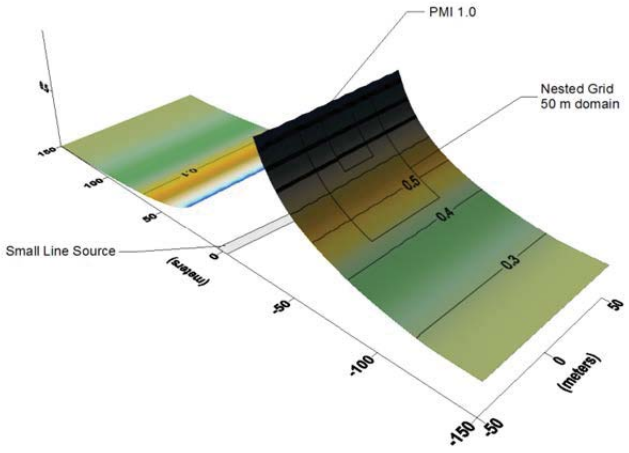
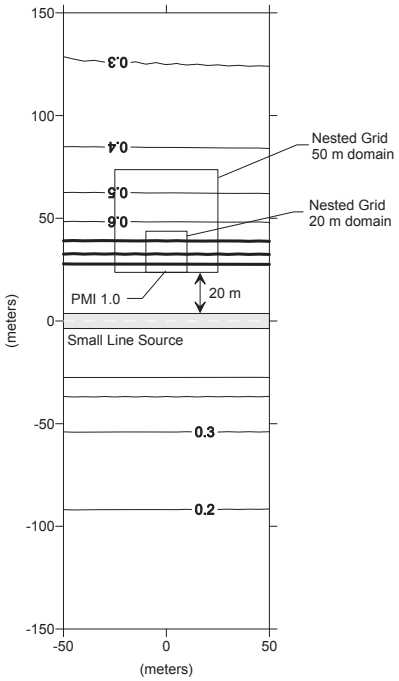


Figure AP C-3.11.2 – Small Line Source, CALINE – Fresno Air Terminal

Isopleths ***Surface***



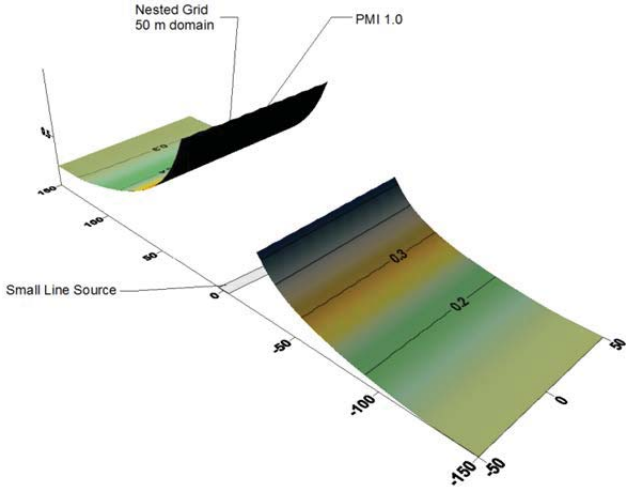
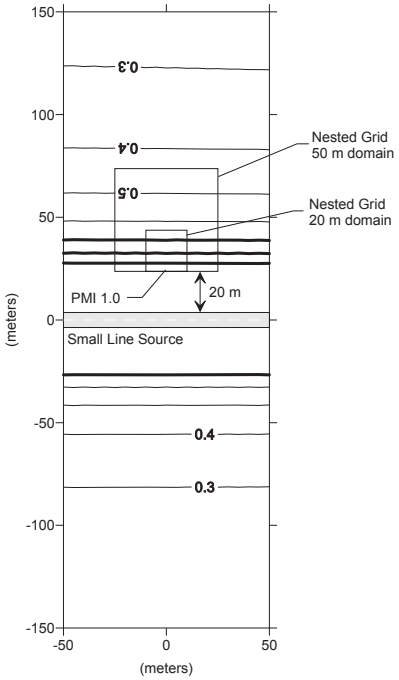


Figure AP C-3.11.3 – Small Line Source, CALINE – Kearny Mesa

Isopleths ***Surface***



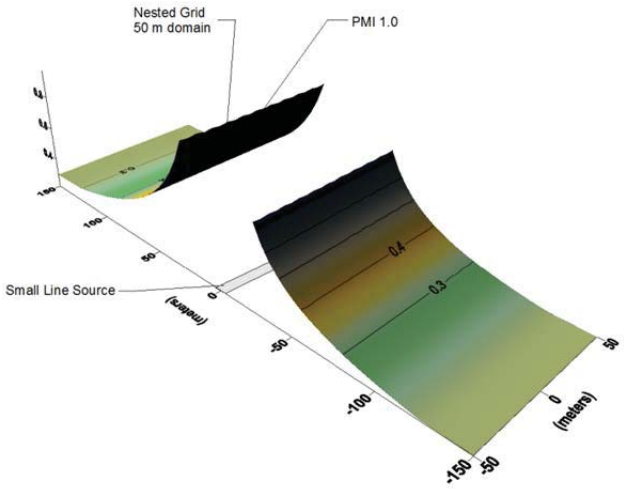
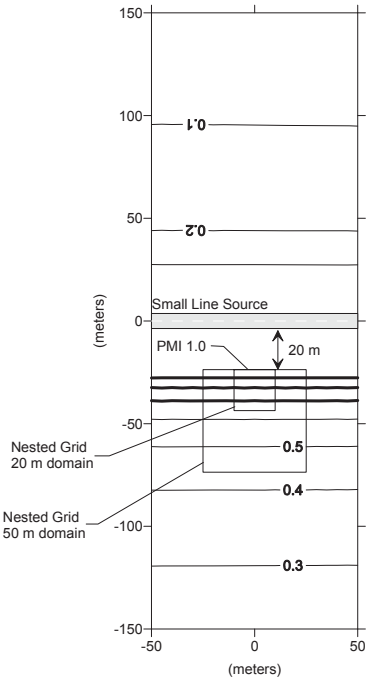


Figure AP C-3.11.4 – Small Line Source, CALINE – Lynwood

Isopleths ***Surface***



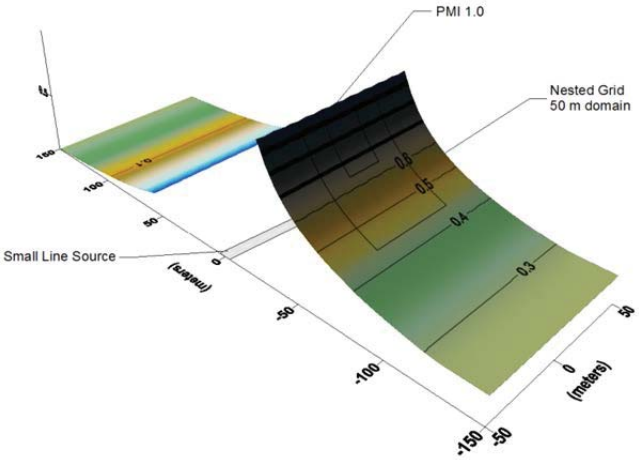
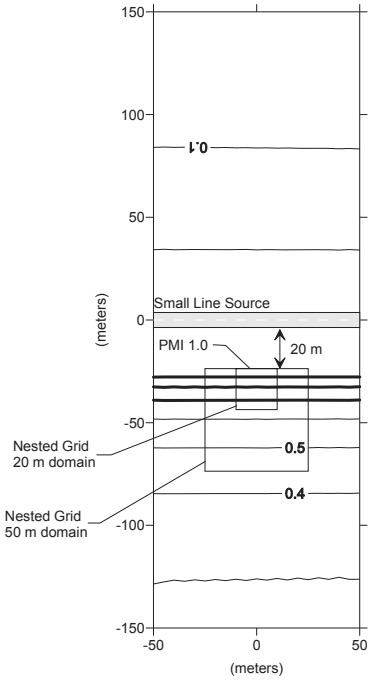
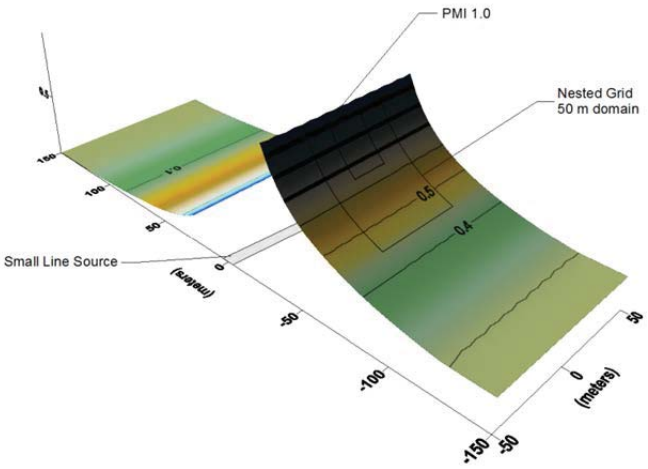


Figure AP C-3.11.5 – Small Line Source, CALINE – San Bernardino

Isopleths ***Surface***





Appendix C-4 – Spatial Average Tables

Table AP **C-**4.1.1 – Spatial Average – Point Source, Large

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.000	1.000	1.000	1.000	1.000
10x10	0.999	0.999	0.999	0.999	0.999
20x20	0.998	0.998	0.997	0.996	0.996
30x30	0.997	0.996	0.994	0.993	0.993
40x40	0.994	0.993	0.990	0.989	0.990
50x50	0.992	0.990	0.985	0.984	0.985
60x60	0.989	0.986	0.979	0.978	0.980
70x70	0.985	0.981	0.972	0.972	0.973
80x80	0.981	0.976	0.965	0.965	0.967
90x90	0.976	0.970	0.956	0.957	0.959
100x100	0.971	0.964	0.947	0.949	0.951

Table AP **C-**4.1.2 – Spatial Average – Point Source, Medium

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	1.00	0.99	0.99	0.99	0.99
20x20	0.99	0.98	0.98	0.98	0.98
30x30	0.98	0.97	0.97	0.97	0.97
40x40	0.97	0.95	0.94	0.95	0.95
50x50	0.95	0.92	0.92	0.92	0.93
60x60	0.93	0.89	0.89	0.89	0.90
70x70	0.91	0.86	0.86	0.86	0.87
80x80	0.89	0.83	0.82	0.83	0.84
90x90	0.87	0.79	0.79	0.80	0.81
100x100	0.84	0.76	0.76	0.76	0.78

Table AP **C-**4.1.3 – Spatial Average – Point Source, Small

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	1.01	0.70	0.83	0.83	0.84
20x20	0.85	0.56	0.69	0.68	0.69
30x30	0.73	0.44	0.58	0.58	0.57
40x40	0.63	0.36	0.50	0.49	0.48
50x50	0.55	0.30	0.44	0.43	0.41
60x60	0.49	0.25	0.39	0.40	0.36
70x70	0.44	0.22	0.34	0.37	0.32
80x80	0.39	0.19	0.31	0.33	0.28
90x90	0.36	0.17	0.28	0.27	0.26
100x100	0.32	0.15	0.25	0.24	0.23

Table AP **C-**4.2.1 – Spatial Average – Volume Source, Large

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.90	0.91	0.91	0.90	0.90
20x20	0.82	0.82	0.82	0.82	0.82
30x30	0.75	0.75	0.75	0.75	0.75
40x40	0.68	0.68	0.69	0.68	0.68
50x50	0.63	0.62	0.64	0.63	0.63
60x60	0.58	0.57	0.59	0.58	0.58
70x70	0.54	0.53	0.55	0.54	0.54
80x80	0.50	0.49	0.51	0.50	0.50
90x90	0.47	0.45	0.48	0.47	0.46
100x100	0.44	0.42	0.45	0.44	0.43

Table AP **C-**4.2.2 – Spatial Average – Volume Source, Medium

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.82	0.84	0.83	0.82	0.82
20x20	0.69	0.70	0.70	0.69	0.69
30x30	0.59	0.60	0.60	0.59	0.59
40x40	0.51	0.51	0.53	0.52	0.51
50x50	0.45	0.44	0.46	0.45	0.45
60x60	0.40	0.39	0.41	0.40	0.39
70x70	0.35	0.34	0.37	0.36	0.35
80x80	0.32	0.30	0.34	0.32	0.32
90x90	0.29	0.27	0.31	0.29	0.29
100x100	0.26	0.25	0.28	0.27	0.26

Table AP **C-**4.2.3 – Spatial Average – Volume Source, Small

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.76	0.76	0.76	0.76	0.75
20x20	0.60	0.60	0.61	0.60	0.59
30x30	0.49	0.47	0.50	0.49	0.48
40x40	0.41	0.39	0.42	0.41	0.40
50x50	0.35	0.32	0.36	0.35	0.34
60x60	0.30	0.27	0.32	0.30	0.29
70x70	0.26	0.24	0.28	0.26	0.25
80x80	0.23	0.21	0.25	0.23	0.22
90x90	0.20	0.18	0.22	0.20	0.20
100x100	0.18	0.16	0.20	0.18	0.18

Table AP **C-**4.3.1 – Spatial Average – Area Source, Large

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.90	0.95	0.93	0.93	0.93
20x20	0.83	0.89	0.87	0.86	0.87
30x30	0.76	0.85	0.82	0.81	0.81
40x40	0.71	0.80	0.78	0.76	0.77
50x50	0.66	0.77	0.74	0.72	0.73
60x60	0.62	0.73	0.70	0.69	0.69
70x70	0.59	0.70	0.67	0.66	0.66
80x80	0.56	0.67	0.64	0.63	0.64
90x90	0.53	0.64	0.62	0.60	0.61
100x100	0.51	0.62	0.59	0.58	0.59

Table AP **C-**4.3.2 – Spatial Average – Area Source, Medium

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1	1	1	1	1
10x10	0.88	0.94	0.91	0.91	0.91
20x20	0.78	0.87	0.83	0.82	0.83
30x30	0.69	0.81	0.76	0.75	0.76
40x40	0.63	0.75	0.70	0.69	0.70
50x50	0.57	0.69	0.65	0.64	0.65
60x60	0.53	0.65	0.61	0.60	0.61
70x70	0.49	0.60	0.57	0.56	0.57
80x80	0.45	0.56	0.54	0.52	0.53
90x90	0.42	0.53	0.50	0.49	0.50
100x100	0.39	0.49	0.47	0.46	0.47

Table AP **C-**4.3.3 – Spatial Average – Area Source, Small

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.64	0.65	0.65	0.65	0.65
20x20	0.44	0.44	0.46	0.46	0.45
30x30	0.32	0.32	0.34	0.26	0.33
40x40	0.25	0.24	0.26	0.21	0.25
50x50	0.20	0.19	0.21	0.17	0.20
60x60	0.16	0.16	0.17	0.14	0.16
70x70	0.13	0.13	0.14	0.14	0.14
80x80	0.11	0.11	0.12	0.12	0.12
90x90	0.10	0.10	0.11	0.10	0.10
100x100	0.09	0.08	0.09	0.09	0.09

Table AP **C-**4.4.1 – Spatial Average – Line Source, Large

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.93	0.93	0.93	0.93	0.93
20x20	0.87	0.88	0.88	0.87	0.88
30x30	0.83	0.83	0.83	0.83	0.83
40x40	0.78	0.79	0.79	0.79	0.79
50x50	0.75	0.75	0.75	0.75	0.75
60x60	0.72	0.72	0.72	0.72	0.72
70x70	0.69	0.70	0.69	0.69	0.70
80x80	0.66	0.67	0.67	0.66	0.67
90x90	0.64	0.65	0.64	0.64	0.65
100x100	0.62	0.63	0.62	0.62	0.63

Table AP **C-**4.4.2 – Spatial Average – Line Source, Small

Domain	CMSA	FAT	KMSA	Lynn	SBO
PMI	1.00	1.00	1.00	1.00	1.00
10x10	0.88	0.88	0.88	0.88	0.88
20x20	0.80	0.80	0.79	0.80	0.80
30x30	0.73	0.74	0.73	0.73	0.73
40x40	0.68	0.69	0.67	0.68	0.68
50x50	0.64	0.64	0.63	0.64	0.64
60x60	0.60	0.61	0.59	0.60	0.61
70x70	0.57	0.58	0.56	0.57	0.58
80x80	0.54	0.55	0.54	0.54	0.55
90x90	0.52	0.53	0.51	0.52	0.53
100x100	0.50	0.51	0.49	0.50	0.51

Appendix C-5 – Tilted Spatial Averaging

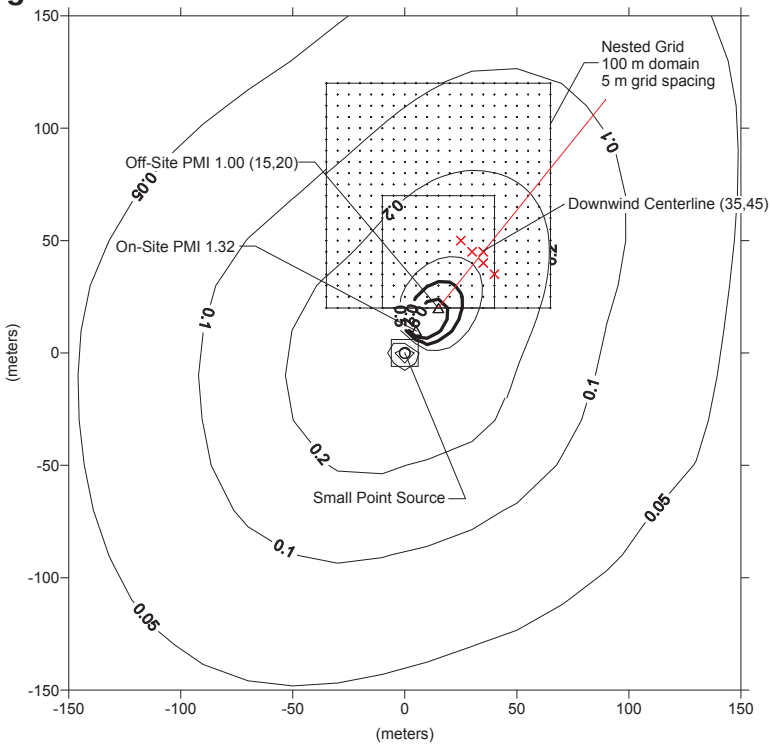
Tilted Spatial Averaging

Small sources tend to show an offsite PMI located at the fence line. It may be necessary to tilt the spatial averaging receptor field when the predominate wind direction carries the average plume centerline askew from the cardinal directions.

The first step in tilting the receptor field is to determine the centerline of the tilted receptor field. The centerline intersects the offsite PMI in the near field. We recommend locating the far end of the centerline by selecting receptors from the 5m spaced grid with the highest concentrations located approximately 30 meters from the offsite PMI.

For example, in the case of San Bernardino meteorology and a small point source, the offsite PMI is located at (15, 20). The dominant plume centerline can be determined from the existing set of receptors spaced at a 5 m grid cell resolution. The maximum concentration located approximately 30 meters from the offsite PMI can be used for the centerline. In this case the plume centerline was determined by plotting the receptors with the five highest concentrations and making a subjective selection of the centerline receptor at (35, 45). See red "x" receptors in Figure AP C-5.1.

Figure AP C-5.1 – San Bernardino Small Point Source



Polar coordinates can be easily calculated from the two points, (15, 20) and (35, 45), with basic trigonometry. In this case, dy/dx = 1.250, and the centerline tilted angle is 38.660 degrees from vertical (51.340 degrees from horizontal).

$$\tan\theta = \frac{dy}{dx} = \frac{45-20}{35-15} = \frac{25}{20} = 1.250$$

Therefore, $\theta = 38.660°$

We recommend that the polar receptor field cover half of a circular area, a 180 degree arc. So for our example the polar receptors centered on 38.660 degrees will sweep an arc from 308.660 degrees to 128.660 degrees (i.e., 38.660º ± 90º).

Polar receptors in AERMOD are easy to specify. Receptors should be placed on radials incremented every five meters. The polar angle of the radials should be placed to closely represent 5 meter grid spacing. For example, Table AP C-5.1 below shows the angular increment of radials for receptor placement out to 25m from the offsite PMI.

Table AP C-5.1 – Recommended Spacing for Tilted Polar Nested Grid

Radial Distance from PMI	0m	5m	10m	15m	20m	25m
Angle Increment (deg)	PMI	60.000	30.000	18.000	13.846	11.250
Resultant spacing along arc	PMI	5.24m	5.24m	4.71m	4.83m	4.91m

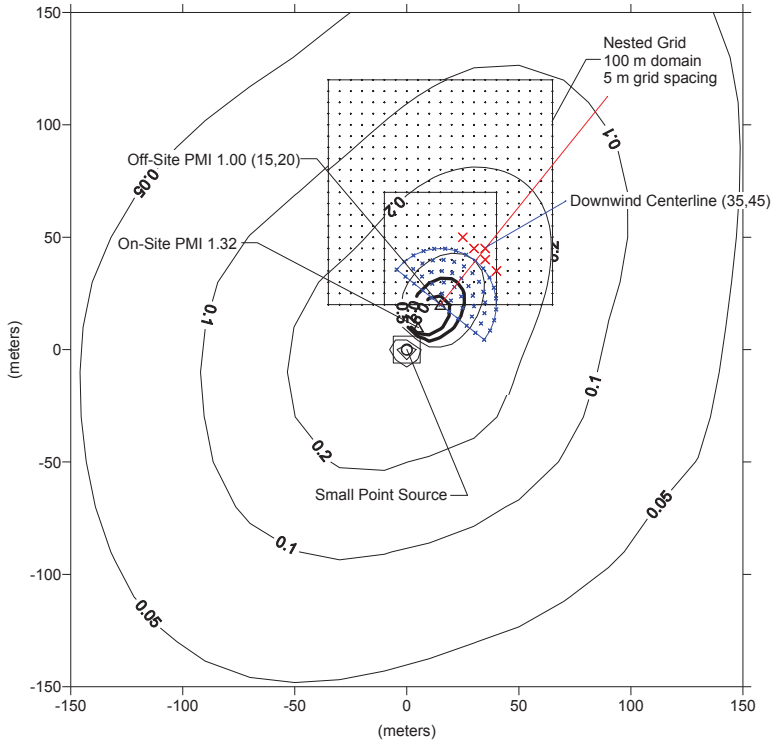
As a result of the above receptor spacing, the following field of polar receptors in Table AP **C-5.2** is needed for the San Bernardino example.

Table AP C-5.2 – Tilted Nested Grid for San Bernardino Example

Radial Distance →	5m	10m	15m	20m	25m
Radial Direction (degrees)					
1	308.660	308.660	308.660	308.660	308.660
2	8.660	338.660	326.660	322.506	319.910
3	68.660	8.660	344.660	336.352	331.160
4	128.660	38.660	2.660	350.198	342.410
5	-	68.660	20.660	4.044	353.660
6	-	98.660	38.660	17.891	4.910
7	-	128.660	56.660	31.737	16.160
8	-	-	74.660	45.583	27.410
9	-	-	92.660	59.429	38.660
10	-	-	110.660	73.275	49.910
11	-	-	128.660	87.121	61.160
12	-	-	-	100.968	72.410
13	-	-	-	114.814	83.660
14	-	-	-	128.660	94.910
15	-	-	-	-	106.160
16	-	-	-	-	117.410
17	-	-	-	-	128.660
Note: Be sure to include the offsite PMI in the polar spatial average.					

Figure AP **C-5.2** shows the resulting receptors for the above field as blue “x”s.

Figure AP C-5.2 – Tilted Nested Polar Grid for San Bernardino Point – Small



As an alternative, a rectangular tilted receptor field can also be created as shown in Figure **AP C-5.3**, below. The tilted rectangular field shown below requires more calculations than the tilted polar field above because discrete receptors must be generated outside of AERMOD. We recommend the tilted polar field approach because of the simplicity of inputting polar receptors into AERMOD.

Table AP C-5.3.1shows a summary of the spatial averaging of tilted nested grids for the San Bernardino meteorological data. In this example, there is little difference between the regular rectangular grid and the tilted rectangular grid.

Figures AP C-5.3.2 and E3.3 show the tilted grids for the volume and area sources examples. In these cases, the tilted grid spatial average is higher than the non-tilted grid. Table APC 5.3.2 shows the spatial average increases from 0.59 to 0.69 for the 20m x 20m nested grid.

Figures APC 5.4.1- APC 5.4.3 show similar trends for nested grids, in this case with meteorological data from the Fresno Air Terminal.

Figure AP C-5.3.1 – Tilted Nested Rectangular Grid for San Bernardino Point – Small

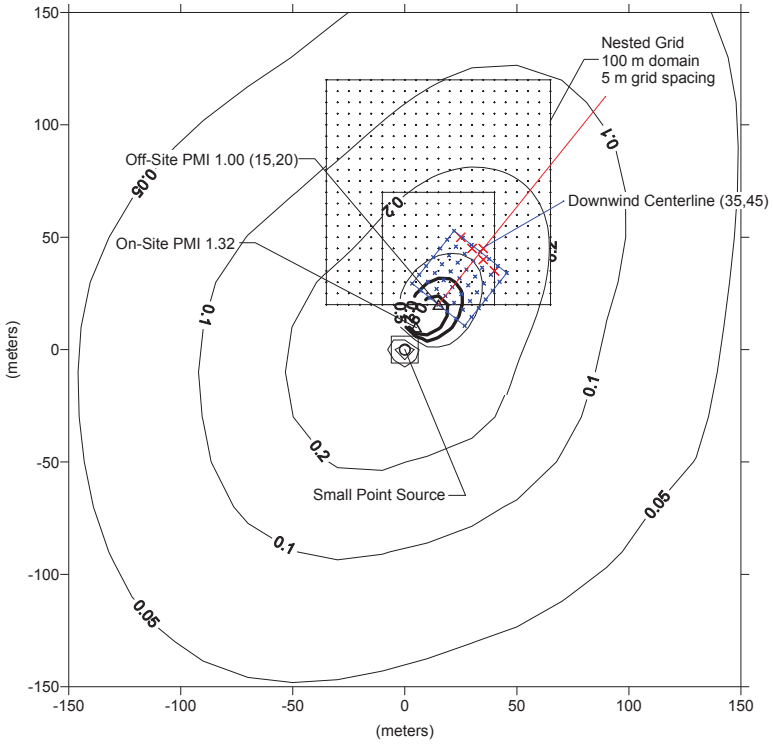


Table AP C-5.3.1 – Spatial Average – San Bernardino – Small Point Source

Nested Grid Domain in m^2	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.91	Polar, R = 5m
100	0.84	0.84	-	Rectangular, 10m x 10m
157	-	-	0.81	Polar, R = 10m
353	-	-	0.71	Polar, R = 15m
400	0.69	0.68	-	Rectangular, 20m x 20m
628	-	-	0.63	Polar, R = 20m
900	0.57	0.58	-	Rectangular, 30m x 30m
982	-	-	0.56	Polar, R = 25m

Figure AP C-5.3.2 – Tilted Nested Grid for San Bernardino Volume – Small

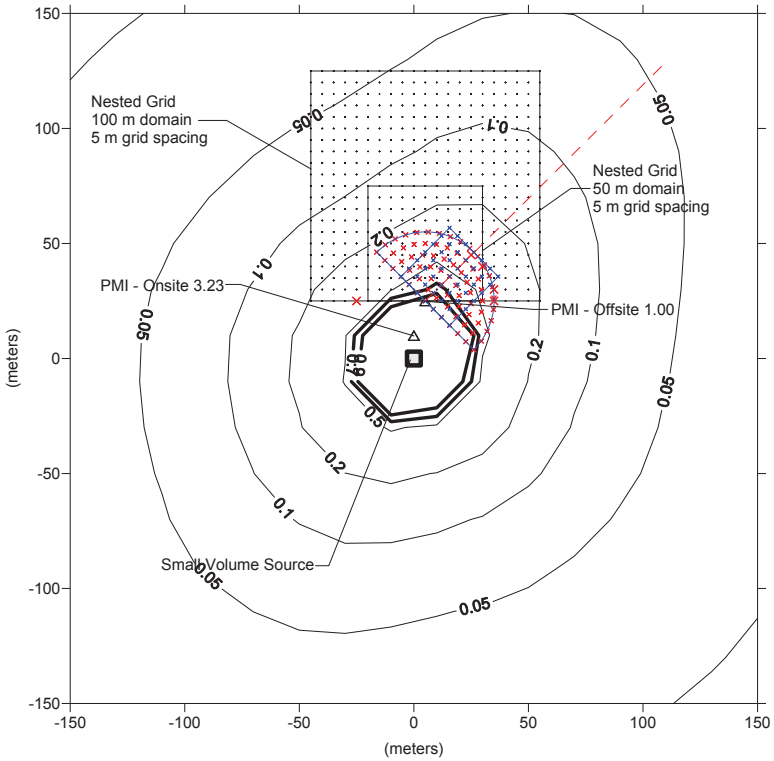


Table AP C-5.3.2 – Spatial Average – San Bernardino – Small Volume Source

Nested Grid Domain in m^2	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.94	Polar, R = 5m
100	0.75	0.83	-	Rectangular, 10m x 10m
157	-	-	0.86	Polar, R = 10m
353	-	-	0.77	Polar, R = 15m
400	0.59	0.69	-	Rectangular, 20m x 20m
628	-	-	0.68	Polar, R = 20m
900	0.48	0.57	-	Rectangular, 30m x 30m
982	-	-	0.56	Polar, R = 25m

Figure AP C-5.3.3 – Tilted Nested Grid for San Bernardino Area – Small

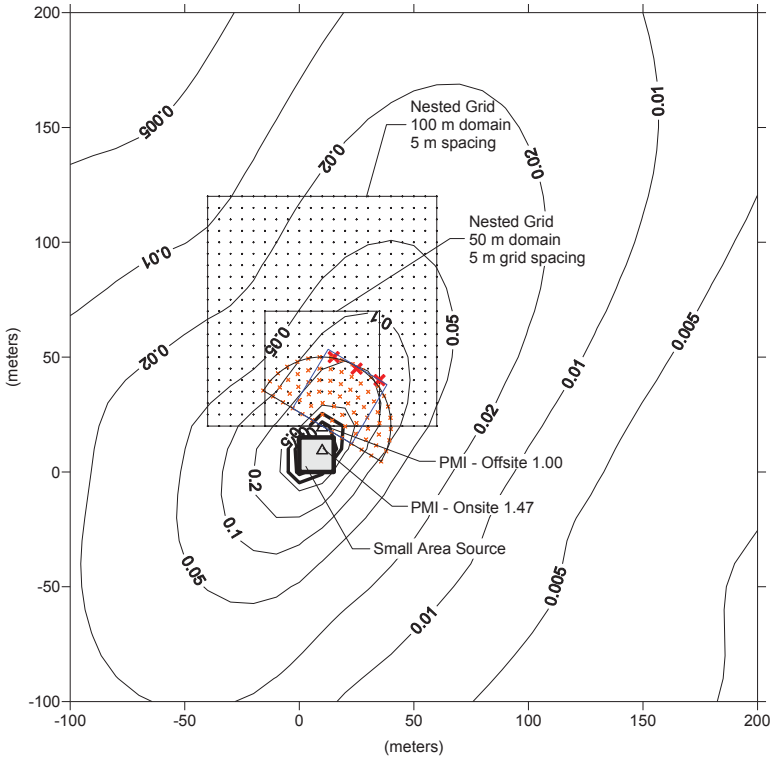


Table AP C-5.3.3 – Spatial Average – San Bernardino – Small Area Source

Nested Grid Domain in m^2	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.86	Polar, R = 5m
100	0.65	0.71	-	Rectangular, 10m x 10m
157	-	-	0.68	Polar, R = 10m
353	-	-	0.52	Polar, R = 15m
400	0.45	0.50	-	Rectangular, 20m x 20m
628	-	-	0.42	Polar, R = 20m
900	0.33	0.36	-	Rectangular, 30m x 30m
982	-	-	0.34	Polar, R = 25m

Figure AP C-5.4.1 – Tilted Nested Rectangular Grid for Fresno Air Terminal Point – Small

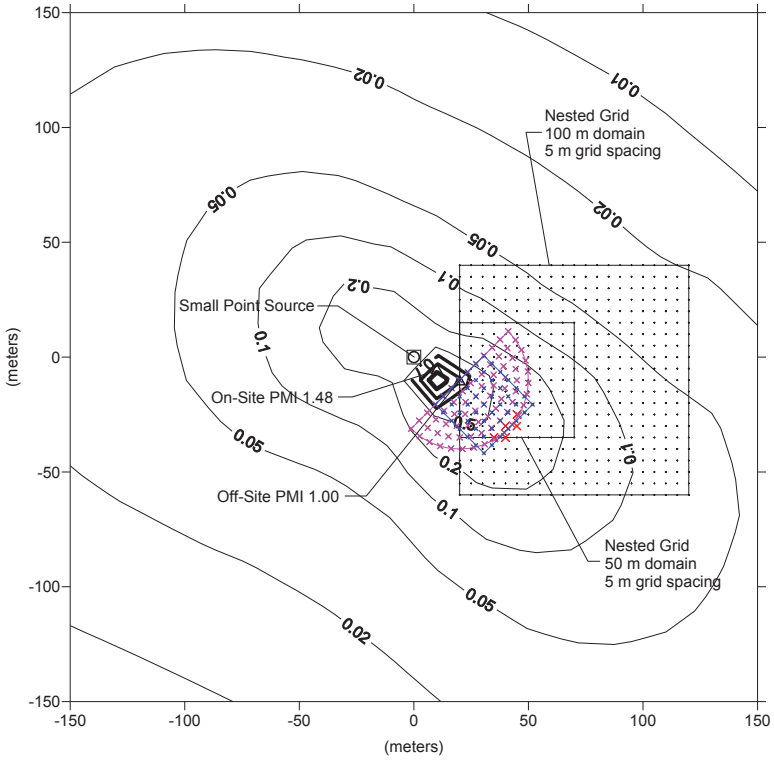


Table AP C-5.4.1 – Spatial Average – Fresno Air Terminal – Small Point Source

Nested Grid Domain in m^2	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.92	Polar, R = 5m
100	0.70	0.83	-	Rectangular, 10m x 10m
157	-	-	0.79	Polar, R = 10m
353	-	-	0.67	Polar, R = 15m
400	0.56	0.67	-	Rectangular, 20m x 20m
628	-	-	0.58	Polar, R = 20m
900	0.44	0.54	-	Rectangular, 30m x 30m
982	-	-	0.50	Polar, R = 25m

Figure AP C-5.4.2 – Tilted Nested Rectangular Grid for Fresno Air Terminal Volume – Small

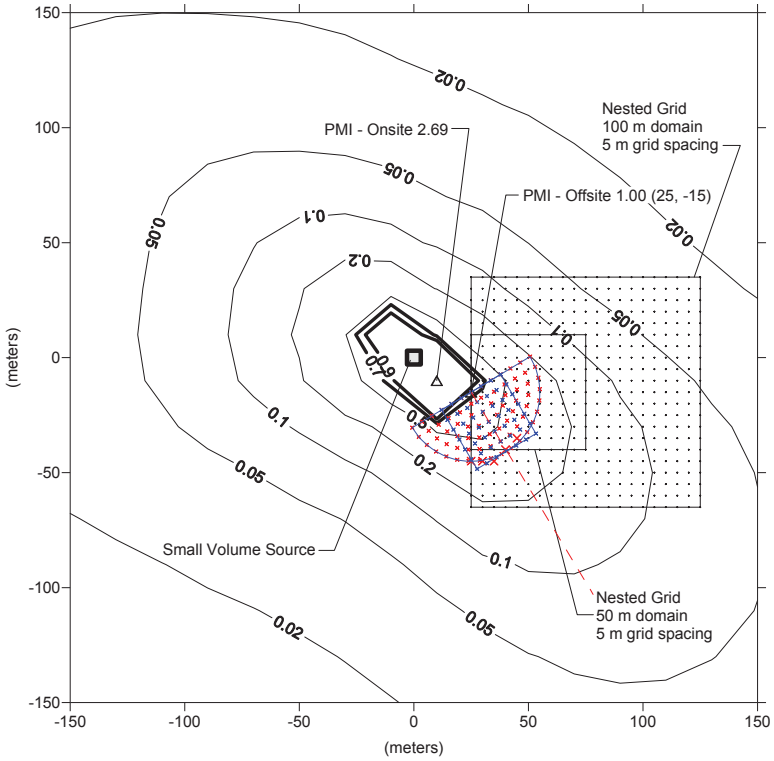


Table AP C-54.2 – Spatial Average – Fresno Air Terminal – Small Volume Source

Nested Grid Domain in m^2	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.93	Polar, R = 5m
100	0.76	0.82	-	Rectangular, 10m x 10m
157	-	-	0.83	Polar, R = 10m
353	-	-	0.73	Polar, R = 15m
400	0.60	0.67	-	Rectangular, 20m x 20m
628	-	-	0.63	Polar, R = 20m
900	0.47	0.55	-	Rectangular, 30m x 30m
982	-	-	0.55	Polar, R = 25m

Figure AP C-5.4.3 – Tilted Nested Rectangular Grid for Fresno Air Terminal Area – Small

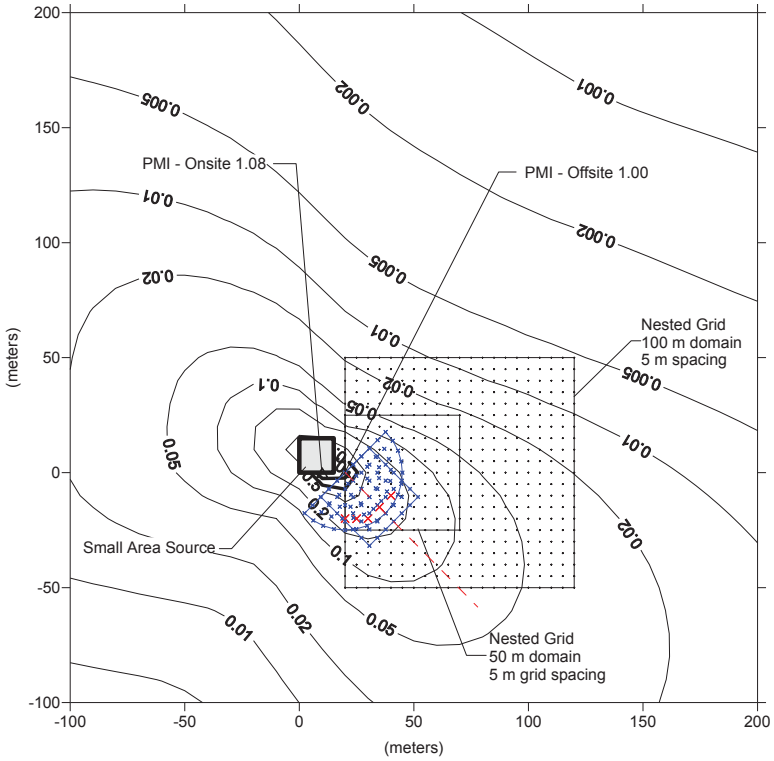


Table AP C-5.4.3 – Spatial Average – Fresno Air Terminal – Small Area Source

Nested Grid Domain in m^2	Cartesian Rectangular	Tilted Rectangular	Tilted Polar	Notes
0	1	1	1	PMI
39	-	-	0.83	Polar, R = 5m
100	0.65	0.69	-	Rectangular, 10m x 10m
157	-	-	0.65	Polar, R = 10m
353	-	-	0.51	Polar, R = 15m
400	0.44	0.49	-	Rectangular, 20m x 20m
628	-	-	0.41	Polar, R = 20m
900	0.32	0.37	-	Rectangular, 30m x 30m
982	-	-	0.34	Polar, R = 25m

Appendix D

Food Codes for NHANES

Table D.1 Food Codes for Leafy Produce

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
25	Spinach souffle	72125240
25	Broccoli casserole (broccoli, noodles, and cream sauce)	72202010
25	Broccoli casserole (broccoli, rice, cheese, and mushroom sau	72202020
25	Broccoli, batter-dipped and fried	72202030
25	Broccoli soup	72302000
25	Broccoli cheese soup, prepared with milk	72302100
25	Spinach soup	72307000
25	Dark-green leafy vegetable soup with meat, Oriental style	72308000
25	Dark-green leafy vegetable soup, meatless, Oriental style	72308500
25	Raw vegetable, NFS	75100250
25	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25	Vegetable combination (including carrots, broccoli, and/or d	75440100
25	Vegetable tempura	75440200
25	Vegetables, dipped in chick-pea flour batter, (pakora), frie	75440400
25	Vegetable combinations (including carrots, broccoli, and/or	75440500
25	Vegetable combination (including carrots, broccoli, and/or d	75450500
25	Vegetable combinations (including carrots, broccoli, and/or	75460800
25	Vegetable soup, home recipe	75649110
25	Vegetable noodle soup, home recipe	75649150
25	Vegetable beef soup, home recipe	75652010
25	Vegetable beef soup with noodles or pasta, home recipe	75652040
25	Vegetable beef soup with rice, home recipe	75652050
33	Seven-layer salad (lettuce salad made with a combination of	75145000
33	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340110
33	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340120
50	Cabbage soup	75601200
50	Cabbage with meat soup	75601210
50	Broccoli and chicken, baby food, strained	76604000
75	Spinach, cooked, NS as to form, with cheese sauce	72125250
75	Turnip greens with roots, cooked, NS as to form, fat not add	72128410

Table D.1 Food Codes for Leafy Produce

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
75	Broccoli, cooked, NS as to form, with cheese sauce	72201230
75	Broccoli, cooked, from fresh, with cheese sauce	72201231
75	Broccoli, cooked, from frozen, with cheese sauce	72201232
75	Broccoli, cooked, NS as to form, with cream sauce	72201250
75	Broccoli, cooked, from fresh, with cream sauce	72201251
75	Cab age salad or coleslaw with apples and/or raisins, with dressing	75141100
75	Cabbage salad or coleslaw with pineapple, with dressing	75141200
75	Lettuce, salad with assorted vegetables including tomatoes a	75143000
75	Lettuce, salad with cheese, tomato and/or carrots, with or w	75143200
75	Lettuce salad with egg, cheese, tomato, and/or carrots, with	75143350
75	Spinach, creamed, baby food, strained	76102010
100	Beet greens, cooked, fat not added in cooking	72101210
100	Chard, cooked, fat not added in cooking	72104210
100	Chard, cooked, fat added in cooking	72104220
100	Collards, raw	72107100
100	Collards, cooked, NS as to form, NS as to fat added in cooki	72107200
100	Collards, cooked, from fresh, NS as to fat added in cooking	72107201
100	Collards, cooked, from fresh, fat not added in cooking	72107211
100	Collards, cooked, NS as to form, fat added in cooking	72107220
100	Collards, cooked, from fresh, fat added in cooking	72107221
100	Collards, cooked, from frozen, fat added in cooking	72107222
100	Greens, cooked, from fresh, fat not added in cooking	72118211
100	Greens, cooked, NS as to form, fat added in cooking	72118220
100	Greens, cooked, from fresh, fat added in cooking	72118221
100	Kale, cooked, NS as to form, NS as to fat added in cooking	72119200
100	Kale, cooked, from fresh, fat not added in cooking	72119211
100	Kale, cooked, NS as to form, fat added in cooking	72119220
100	Kale, cooked, from fresh, fat added in cooking	72119221
100	Mustard greens, cooked, NS as to form, NS as to fat added in	72122200
100	Mustard greens, cooked, from fresh, NS as to fat added in co	72122201
100	Mustard greens, cooked, from fresh, fat not added in cooking	72122211

Table D.1 Food Codes for Leafy Produce

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
100	Mustard greens, cooked, from canned, fat not added in cookin	72122213
100	Mustard greens, cooked, from fresh, fat added in cooking	72122221
100	Mustard greens, cooked, from frozen, fat added in cooking	72122222
100	Mustard greens, cooked, from canned, fat added in cooking	72122223
100	Poke greens, cooked, fat not added in cooking	72123010
100	Poke greens, cooked, fat added in cooking	72123020
100	Radicchio, raw	72124100
100	Spinach, raw	72125100
100	Spinach, cooked, NS as to form, NS as to fat added in cookin	72125200
100	Spinach, cooked, from fresh, NS as to fat added in cooking	72125201
100	Spinach, cooked, from frozen, NS as to fat added in cooking	72125202
100	Spinach, cooked, NS as to form, fat not added in cooking	72125210
100	Spinach, cooked, from fresh, fat not added in cooking	72125211
100	Spinach, cooked, from frozen, fat not added in cooking	72125212
100	Spinach, cooked, NS as to form, fat added in cooking	72125220
100	Spinach, cooked, from fresh, fat added in cooking	72125221
100	Spinach, cooked, from frozen, fat added in cooking	72125222
100	Spinach, NS as to form, creamed	72125230
100	Turnip greens, cooked, from fresh, fat not added in cooking	72128211
100	Turnip greens, cooked, NS as to form, fat added in cooking	72128220
100	Turnip greens, cooked, from fresh, fat added in cooking	72128221
100	Turnip greens, cooked, from frozen, fat added in cooking	72128222
100	Watercress, raw	72130100
100	Broccoli, raw	72201100
100	Broccoli, cooked, NS as to form, NS as to fat added in cooki	72201200
100	Broccoli, cooked, from fresh, NS as to fat added in cooking	72201201
100	Broccoli, cooked, from frozen, NS as to fat added in cooking	72201202
100	Broccoli, cooked, NS as to form, fat not added in cooking	72201210
100	Broccoli, cooked, from fresh, fat not added in cooking	72201211
100	Broccoli, cooked, from frozen, fat not added in cooking	72201212
100	Broccoli, cooked, NS as to form, fat added in cooking	72201220

Table D.1 Food Codes for Leafy Produce

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
100	Broccoli, cooked, from fresh, fat added in cooking	72201221
100	Broccoli, cooked, from frozen, fat added in cooking	72201222
100	Sprouts, NFS	75100300
100	Alfalfa sprouts, raw	75100500
100	Artichoke, Jerusalem, raw	75100750
100	Cabbage, green, raw	75103000
100	Cabbage, Chinese, raw	75104000
100	Cabbage, red, raw	75105000
100	Cauliflower, raw	75107000
100	Celery, raw	75109000
100	Chives, raw	75109500
100	Cilantro, raw	75109550
100	Lettuce, raw	75113000
100	Lettuce, Boston, raw	75113060
100	Lettuce, arugula, raw	75113080
100	Mixed salad greens, raw	75114000
100	Parsley, raw	75119000
100	Broccoli salad with cauliflower, cheese, bacon bits, and dre	75140500
100	Cabbage salad or coleslaw, with dressing	75141000
100	Artichoke, globe (French), cooked, NS as to form, NS as to f	75201000
100	Artichoke, globe (French), cooked, NS as to form, fat not ad	75201010
100	Artichoke, globe (French), cooked, from fresh, fat not added	75201011
100	Artichoke, globe (French), cooked, from canned, fat not adde	75201013
100	Artichoke, globe (French), cooked, NS as to form, fat added	75201020
100	Artichoke, globe (French), cooked, from fresh, fat added in	75201021
100	Artichoke salad in oil	75201030
100	Brussels sprouts, cooked, NS as to form, fat not added in co	75209010
100	Brussels sprouts, cooked, from fresh, fat not added in cooki	75209011
100	Brussels sprouts, cooked, from frozen, fat not added in cook	75209012
100	Brussels sprouts, cooked, from fresh, fat added in cooking	75209021
100	Brussels sprouts, cooked, from frozen, fat added in cooking	75209022

Table D.1 Food Codes for Leafy Produce

% Leafy Produce in Food Item	Food Item Description	USDA Food Code
100	Cabbage, Chinese, cooked, NS as to fat added in cooking	75210000
100	Cabbage, Chinese, cooked, fat not added in cooking	75210010
100	Cabbage, Chinese, cooked, fat added in cooking	75210020
100	Cabbage, green, cooked, NS as to fat added in cooking	75211010
100	Cabbage, green, cooked, fat not added in cooking	75211020
100	Cabbage, green, cooked, fat added in cooking	75211030
100	Cabbage, red, cooked, fat not added in cooking	75212010
100	Cauliflower, cooked, NS as to form, NS as to fat added in co	75214000
100	Cauliflower, cooked, from fresh, NS as to fat added in cooki	75214001
100	Cauliflower, cooked, from frozen, NS as to fat added in cook	75214002
100	Cauliflower, cooked, NS as to form, fat not added in cooking	75214010
100	Cauliflower, cooked, from fresh, fat not added in cooking	75214011
100	Cauliflower, cooked, from frozen, fat not added in cooking	75214012
100	Cauliflower, cooked, NS as to form, fat added in cooking	75214020
100	Cauliflower, cooked, from fresh, fat added in cooking	75214021
100	Cauliflower, cooked, from frozen, fat added in cooking	75214022
100	Lettuce, cooked, fat not added in cooking	75220050
100	Parsley, cooked (assume fat not added in cooking)	75221210
100	Cauliflower, batter-dipped, fried	75409020
100	Cabbage, red, pickled	75502510
100	Cabbage, Kim Chee style	75502520

Table D.2 Food Codes for Exposed Produce

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
12.5	Vegetable beef soup, home recipe	75652010
12.5	Vegetable beef soup with noodles or pasta, home recipe	75652040
12.5	Vegetable beef soup with rice, home recipe	75652050
12.5	Vegetables and rice, baby food, strained	76501000
12.5	Vegetable and bacon, baby food, strained	76601010
12.5	Vegetable and beef, baby food, strained	76603010
12.5	Vegetable and beef, baby food, junior	76603020
12.5	Vegetable and chicken, baby food, strained	76605010
12.5	Vegetable and chicken, baby food, junior	76605020
12.5	Vegetable and ham, baby food, strained	76607010
12.5	Vegetable and ham, baby food, junior	76607020
12.5	Vegetable and turkey, baby food, strained	76611010
12.5	Vegetable and turkey, baby food, junior	76611020
25.0	Raw vegetable, NFS	75100250
25.0	Cabbage salad or coleslaw with apples and/or raisins, with d	75141100
25.0	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25.0	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25.0	Vegetable combination (including carrots, broccoli, and/or d	75440100
25.0	Vegetable tempura	75440200
25.0	Vegetable combinations (including carrots, broccoli, and/or	75440500
25.0	Vegetable combination (including carrots, broccoli, and/or d	75450500
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460800
25.0	Vegetable soup, home recipe	75649110
25.0	Vegetable noodle soup, home recipe	75649150
25.0	Spanish stew, Puerto Rican style (Cocido Espanol)	77513010
33.0	Grape juice	64116020
33.0	Peach juice, with sugar	64122030
33.0	Apple-banana juice, baby food	67203200
33.0	Apple-cranberry juice, baby food	67203450
33.0	Tomato soup, NFS	74601000
33.0	Tomato soup, prepared with water	74602010

Table D.2 Food Codes for Exposed Produce

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340110
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340120
33.0	Vegetable stew without meat	75439010
33.0	Mushroom soup, NFS	75607000
33.0	Mixed vegetables, garden vegetables, baby food, NS as to str	76407000
33.0	Mixed vegetables, garden vegetables, baby food, strained	76407010
33.0	Mixed vegetables, garden vegetables, baby food, junior	76407020
33.0	Jams, preserves, marmalades, dietetic, all flavors, sweetene	91406000
33.0	Jams, preserves, marmalades, sweetened with fruit juice conc	91406500
33.0	Jams, preserves, marmalades, low sugar (all flavors)	91406600
50.0	Bananas with apples and pears, baby food, strained	67106010
50.0	Pears and pineapple, baby food, strained	67114010
50.0	Pears and pineapple, baby food, junior	67114020
50.0	Tomato and corn, cooked, fat not added in cooking	74503010
50.0	Tomato and onion, cooked, NS as to fat added in cooking	74504100
50.0	Tomato and onion, cooked, fat not added in cooking	74504110
50.0	Tomato and onion, cooked, fat added in cooking	74504120
50.0	Beans, green, and potatoes, cooked, fat not added in cooking	75302050
50.0	Beans, green, with pinto beans, cooked, fat not added in coo	75302060
50.0	Beans, green, and potatoes, cooked, NS as to fat added in co	75302500
50.0	Beans, green, and potatoes, cooked, fat added in cooking	75302510
50.0	Peas with mushrooms, cooked, fat not added in cooking	75315210
50.0	Chiles rellenos, cheese-filled (stuffed chili peppers)	75410500
50.0	Chiles rellenos, filled with meat and cheese (stuffed chili	75410530
50.0	Minestrone soup, home recipe	75651000
50.0	Jelly, all flavors	91401000
50.0	Jam, preserves, all flavors	91402000
50.0	Jelly, dietetic, all flavors, sweetened with artificial swee	91405000
50.0	Jelly, reduced sugar, all flavors	91405500
66.0	Fruit juice, NFS	64100100
66.0	Apple cider	64101010

Table D.2 Food Codes for Exposed Produce

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
66.0	Apple juice	64104010
66.0	Prune juice	64132010
66.0	Prune juice, unsweetened	64132020
66.0	Strawberry juice	64132500
66.0	Apple juice, baby food	67202000
66.0	Apple with other fruit juice, baby food	67203000
66.0	Apple-cherry juice, baby food	67203400
66.0	Apple-grape juice, baby food	67203500
66.0	Apple-prune juice, baby food	67203700
66.0	Grape juice, baby food	67203800
66.0	Mixed fruit juice, not citrus, baby food	67204000
66.0	Pear juice, baby food	67212000
66.0	Tomato juice	74301100
66.0	Tomato and vegetable juice, mostly tomato	74303000
66.0	Mixed vegetable juice (vegetables other than tomato)	75132000
66.0	Celery juice	75132100
66.0	Gazpacho	75604600
100.0	Fruit, dried, NFS (assume uncooked)	62101000
100.0	Fruit mixture, dried (mixture includes three or more of the	62101050
100.0	Apple, dried, uncooked	62101100
100.0	Apple, dried, cooked, NS as to sweetened or unsweetened; swe	62101200
100.0	Apricot, dried, uncooked	62104100
100.0	Pear, dried, cooked, with sugar	62119230
100.0	Prune, dried, uncooked	62122100
100.0	Prune, dried, cooked, NS as to sweetened or unsweetened; swe	62122200
100.0	Prune, dried, cooked, unsweetened	62122220
100.0	Prune, dried, cooked, with sugar	62122230
100.0	Raisins	62125100
100.0	Raisins, cooked	62125110
100.0	Apple, raw	63101000
100.0	Applesauce, stewed apples, NS as to sweetened or unsweetened	63101110

Table D.2 Food Codes for Exposed Produce

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
100.0	Applesauce, stewed apples, unsweetened	63101120
100.0	Applesauce, stewed apples, with sugar	63101130
100.0	Applesauce, stewed apples, sweetened with low calorie sweete	63101140
100.0	Applesauce with other fruits	63101150
100.0	Apple, cooked or canned, with syrup	63101210
100.0	Apple, baked, NS as to added sweetener	63101310
100.0	Apple, baked, unsweetened	63101320
100.0	Apple, baked, with sugar	63101330
100.0	Apple, pickled	63101420
100.0	Apple, fried	63101500
100.0	Apricot, raw	63103010
100.0	Apricot, cooked or canned, NS as to sweetened or unsweetened	63103110
100.0	Apricot, cooked or canned, in light syrup	63103140
100.0	Apricot, cooked or canned, drained solids	63103150
100.0	Apricot, cooked or canned, juice pack	63103170
100.0	Cherry pie filling	63113030
100.0	Cherries, sweet, raw (Queen Anne, Bing)	63115010
100.0	Cherries, sweet, cooked or canned, drained solids	63115150
100.0	Fig, raw	63119010
100.0	Grapes, raw, NS as to type	63123000
100.0	Grapes, European type, adherent skin, raw	63123010
100.0	Grapes, seedless, cooked or canned, unsweetened, water pack	63123120
100.0	Mango, raw	63129010
100.0	Mango, cooked	63129030
100.0	Nectarine, raw	63131010
100.0	Nectarine, cooked	63131110
100.0	Peach, raw	63135010
100.0	Peach, cooked or canned, NS as to sweetened or unsweetened;	63135110
100.0	Peach, cooked or canned, in heavy syrup	63135130
100.0	Peach, cooked or canned, in light or medium syrup	63135140
100.0	Peach, cooked or canned, drained solids	63135150

Table D.2 Food Codes for Exposed Produce

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
100.0	Peach, cooked or canned, juice pack	63135170
100.0	Peach, frozen, NS as to added sweetener	63135610
100.0	Peach, frozen, unsweetened	63135620
100.0	Peach, frozen, with sugar	63135630
100.0	Pear, raw	63137010
100.0	Pear, Japanese, raw	63137050
100.0	Pear, cooked or canned, NS as to sweetened or unsweetened; s	63137110
100.0	Pear, cooked or canned, in heavy syrup	63137130
100.0	Pear, cooked or canned, in light syrup	63137140
100.0	Pear, cooked or canned, drained solids	63137150
100.0	Pear, cooked or canned, juice pack	63137170
100.0	Persimmon, raw	63139010
100.0	Plum, raw	63143010
100.0	Plum, cooked or canned, in light syrup	63143140
100.0	Plum, pickled	63143650
100.0	Rhubarb, frozen, with sugar	63147620
100.0	SUGAR APPLE, SWEETSOP (ANON), RAW	63148010
100.0	Blackberries, raw	63201010
100.0	Blackberries, cooked or canned, NS as to sweetened or unswee	63201110
100.0	Raspberries, raw, NS as to color	63219000
100.0	Raspberries, red, raw	63219020
100.0	Raspberries, cooked or canned, NS as to sweetened or unsweet	63219110
100.0	Raspberries, frozen, unsweetened	63219610
100.0	Strawberries, raw	63223020
100.0	Strawberries, raw, with sugar	63223030
100.0	Strawberries, cooked or canned, NS as to sweetened or unswee	63223110
100.0	Strawberries, cooked or canned, unsweetened, water pack	63223120
100.0	Strawberries, cooked or canned, in syrup	63223130
100.0	Strawberries, frozen, NS as to added sweetener	63223600
100.0	Strawberries, frozen, unsweetened	63223610
100.0	Strawberries, frozen, with sugar	63223620

Table D.2 Food Codes for Exposed Produce

% Exposed Produce in Food Item	Food Item Description	USDA Food Code
100.0	Fruit cocktail or mix (excluding citrus fruits), raw	63311000
100.0	Apple salad with dressing	63401010
100.0	Apple, candied	63401060
100.0	Fruit salad (excluding citrus fruits) with salad dressing or	63402950
100.0	Fruit salad (excluding citrus fruits) with cream	63402960
100.0	Fruit salad (excluding citrus fruits) with cream substitute	63402970
100.0	Fruit salad (excluding citrus fruits) with marshmallows	63402980
100.0	Fruit salad (excluding citrus fruits) with pudding	63403000
100.0	Fruit salad (including citrus fruits) with salad dressing or	63403010
100.0	Fruit salad (including citrus fruit) with cream	63403020
100.0	Fruit salad (including citrus fruits) with marshmallows	63403040
100.0	Chutney	63409020
100.0	Tomato and okra, cooked, NS as to fat added in cooking	74504000
100.0	Tomato and okra, cooked, fat not added in cooking	74504010
100.0	Tomato and okra, cooked, fat added in cooking	74504020
100.0	Tomato and celery, cooked, fat not added in cooking	74504150
100.0	Cucumber salad with creamy dressing	75142500
100.0	Cucumber salad made with cucumber, oil, and vinegar	75142550
100.0	Cucumber salad made with cucumber and vinegar	75142600
100.0	Cucumber pickles, dill	75503010
100.0	Cucumber pickles, relish	75503020
100.0	Cucumber pickles, sour	75503030
100.0	Cucumber pickles, sweet	75503040
100.0	Cucumber pickles, fresh	75503050
100.0	Mustard pickles	75503100
100.0	Cucumber pickles, dill, reduced salt	75503110

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
12.5	Vegetables and rice, baby food, strained	76501000
12.5	Vegetable and bacon, baby food, strained	76601010
12.5	Carrots and beef, baby food, strained	76602000
12.5	Vegetable and beef, baby food, strained	76603010
12.5	Vegetable and beef, baby food, junior	76603020
12.5	Vegetable and chicken, baby food, strained	76605010
12.5	Vegetable and chicken, baby food, junior	76605020
12.5	Vegetable and ham, baby food, strained	76607010
12.5	Vegetable and ham, baby food, junior	76607020
12.5	Vegetable and turkey, baby food, strained	76611010
12.5	Vegetable and turkey, baby food, junior	76611020
25.0	Lemon pie filling	61113500
25.0	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25.0	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25.0	Vegetable combination (including carrots, broccoli, and/or d	75440100
25.0	Vegetable combination (excluding carrots, broccoli, and dark	75440110
25.0	Vegetable sticks, breaded (including corn, carrots, and gree	75440170
25.0	Vegetable tempura	75440200
25.0	Vegetables, dipped in chick-pea flour batter, (pakora), frie	75440400
25.0	Vegetable combinations (including carrots, broccoli, and/or	75440500
25.0	Vegetable combination (including carrots, broccoli, and/or d	75450500
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460700
25.0	Vegetable combinations (excluding carrots, broccoli, and dar	75460710
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460800
25.0	Vegetable soup, home recipe	75649110
25.0	Vegetable noodle soup, home recipe	75649150
25.0	Vegetable beef soup, home recipe	75652010
25.0	Vegetable beef soup with noodles or pasta, home recipe	75652040
25.0	Vegetable beef soup with rice, home recipe	75652050
25.0	Fruit sauce	91361020
33.0	Strawberry-banana-orange juice	61226000
33.0	Vegetable stew without meat	75439010

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
33.0	Mixed vegetables, garden vegetables, baby food, NS as to str	76407000
33.0	Mixed vegetables, garden vegetables, baby food, strained	76407010
33.0	Mixed vegetables, garden vegetables, baby food, junior	76407020
33.0	Jams, preserves, marmalades, dietetic, all flavors, sweetene	91406000
33.0	Jams, preserves, marmalades, sweetened with fruit juice conc	91406500
33.0	Jams, preserves, marmalades, low sugar (all flavors)	91406600
50.0	Orange and banana juice	61219000
50.0	Pineapple-orange juice, NFS	61225000
50.0	Tomato and corn, cooked, fat not added in cooking	74503010
50.0	Beans, green, with pinto beans, cooked, fat not added in coo	75302060
50.0	Peas and onions, cooked, fat not added in cooking	75315110
50.0	Peas and onions, cooked, fat added in cooking	75315120
50.0	Peas with mushrooms, cooked, fat not added in cooking	75315210
50.0	Peas and potatoes, cooked, fat not added in cooking	75315300
50.0	Squash, summer, and onions, cooked, fat not added in cooking	75316000
50.0	Pinacbet (eggplant with tomatoes, bitter melon, etc.)	75340300
50.0	Eggplant, batter-dipped, fried	75412010
50.0	Eggplant dip	75412030
50.0	Eggplant parmesan casserole, regular	75412060
50.0	Pea salad	75416500
50.0	Pea salad with cheese	75416600
50.0	Squash,summer, yellow or green, breaded or battered, baked	75418000
50.0	Squash, summer, yellow or green, breaded or battered, fried	75418010
50.0	Pea soup, NFS	75609000
50.0	Carrots and peas, baby food, strained	76202000
100.0	Almonds, NFS	42100100
100.0	Almonds, unroasted	42101000
100.0	Chestnuts, roasted	42105000
100.0	Filberts, hazelnuts	42107000
100.0	Pecans	42112000
100.0	Walnuts	42116000

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Pumpkin and/or squash seeds, hulled, roasted, salted	43101100
100.0	Grapefruit, raw	61101010
100.0	Grapefruit, canned or frozen, NS as to sweetened or unsweete	61101200
100.0	Grapefruit, canned or frozen, in light syrup	61101230
100.0	Lemon, raw	61113010
100.0	Lime, raw	61116010
100.0	Orange, raw	61119010
100.0	Orange, mandarin, canned or frozen, NS as to sweetened or un	61122300
100.0	Orange, mandarin, canned or frozen, juice pack	61122320
100.0	Orange, mandarin, canned or frozen, in light syrup	61122330
100.0	Orange, mandarin, canned or frozen, drained	61122350
100.0	Tangerine, raw	61125010
100.0	Grapefruit juice, freshly squeezed	61201010
100.0	Lemon juice, NS as to form	61204000
100.0	Lemon juice, fresh	61204010
100.0	Lemon juice, frozen	61204600
100.0	Lime juice, NS as to form	61207000
100.0	Lime juice, fresh	61207010
100.0	Lime juice, frozen	61207600
100.0	Orange juice, NFS	61210000
100.0	Orange juice, freshly squeezed	61210010
100.0	Tangerine juice, NFS	61213000
100.0	Avocado, raw	63105010
100.0	Cantaloupe (muskmelon), raw	63109010
100.0	Cantaloupe, frozen (balls)	63109610
100.0	Kiwi fruit, raw	63126500
100.0	Honeydew melon, raw	63127010
100.0	Honeydew, frozen (balls)	63127610
100.0	Papaya, raw	63133010
100.0	Papaya, cooked or canned, in sugar or syrup	63133100
100.0	Pomegranate, raw	63145010

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Watermelon, raw	63149010
100.0	Guacamole with tomatoes	63408010
100.0	Guacamole with tomatoes and chili peppers	63408200
100.0	Guacamole, NFS	63409010
100.0	Pumpkin, cooked, from fresh, fat not added in cooking	73201011
100.0	Pumpkin, cooked, from canned, fat not added in cooking	73201013
100.0	Pumpkin, cooked, NS as to form, fat added in cooking	73201020
100.0	Pumpkin, cooked, from fresh, fat added in cooking	73201021
100.0	Calabaza (Spanish pumpkin), cooked	73210010
100.0	Squash, winter type, mashed, NS as to fat or sugar added in	73301000
100.0	Squash, winter type, mashed, no fat or sugar added in cookin	73301010
100.0	Squash, winter type, mashed, fat added in cooking, no sugar	73301020
100.0	Squash, winter type, baked, NS as to fat or sugar added in c	73303000
100.0	Squash, winter type, baked, no fat or sugar added in cooking	73303010
100.0	Squash, winter type, baked, fat added in cooking, no sugar a	73303020
100.0	Squash, winter, baked with cheese	73305010
100.0	Peas, green, raw	75120000
100.0	Squash, summer, yellow, raw	75128000
100.0	Squash, summer, green, raw	75128010
100.0	Beans, lima, immature, cooked, NS as to form, NS as to fat a	75204000
100.0	Beans, lima, immature, cooked, from fresh, fat not added in	75204011
100.0	Beans, lima, immature, cooked, from frozen, fat not added in	75204012
100.0	Beans, lima, immature, cooked, NS as to form, fat added in c	75204020
100.0	Beans, lima, immature, cooked, from fresh, fat added in cook	75204021
100.0	Beans, lima, immature, cooked, from frozen, fat added in coo	75204022
100.0	Bitter melon, cooked, fat added in cooking	75208310
100.0	Cactus, cooked, NS as to fat added in cooking	75213100
100.0	Cactus, cooked, fat not added in cooking	75213110
100.0	Cactus, cooked, fat added in cooking	75213120
100.0	Christophine, cooked, fat not added in cooking	75215510
100.0	Corn, cooked, NS as to form, NS as to color, NS as to fat ad	75216000

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Corn, cooked, from fresh, NS as to color, NS as to fat added	75216001
100.0	Corn, cooked, from frozen, NS as to color, NS as to fat adde	75216002
100.0	Corn, cooked, NS as to form, NS as to color, fat not added i	75216010
100.0	Corn, cooked, from fresh, NS as to color, fat not added in c	75216011
100.0	Corn, cooked, from frozen, NS as to color, fat not added in	75216012
100.0	Corn, cooked, NS as to form, NS as to color, fat added in co	75216020
100.0	Corn, cooked, from fresh, NS as to color, fat added in cooki	75216021
100.0	Corn, cooked, from frozen, NS as to color, fat added in cook	75216022
100.0	Corn, NS as to form, NS as to color, cream style	75216050
100.0	Corn, yellow, cooked, NS as to form, NS as to fat added in c	75216100
100.0	Corn, yellow, cooked, from fresh, NS as to fat added in cook	75216101
100.0	Corn, yellow, cooked, from frozen, NS as to fat added in coo	75216102
100.0	Corn, yellow, cooked, NS as to form, fat not added in cookin	75216110
100.0	Corn, yellow, cooked, from fresh, fat not added in cooking	75216111
100.0	Corn, yellow, cooked, from frozen, fat not added in cooking	75216112
100.0	Corn, yellow, cooked, NS as to form, fat added in cooking	75216120
100.0	Corn, yellow, cooked, from fresh, fat added in cooking	75216121
100.0	Corn, yellow, cooked, from frozen, fat added in cooking	75216122
100.0	Corn, yellow, NS as to form, cream style	75216150
100.0	Corn, yellow and white, cooked, NS as to form, NS as to fat	75216160
100.0	Corn, yellow and white, cooked, from fresh, NS as to fat add	75216161
100.0	Corn, yellow and white, cooked, NS as to form, fat not added	75216170
100.0	Corn, yellow and white, cooked, from fresh, fat not added in	75216171
100.0	Corn, yellow and white, cooked, from fresh, fat added in coo	75216181
100.0	Corn, white, cooked, NS as to form, NS as to fat added in co	75216200
100.0	Corn, white, cooked, from fresh, NS as to fat added in cooki	75216201
100.0	Corn, white, cooked, NS as to form, fat not added in cooking	75216210
100.0	Corn, white, cooked, from fresh, fat not added in cooking	75216211
100.0	Corn, white, cooked, from frozen, fat not added in cooking	75216212
100.0	Corn, white, cooked, from fresh, fat added in cooking	75216221
100.0	Corn, white, cooked, from frozen, fat added in cooking	75216222

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Hominy, cooked, fat not added in cooking	75217500
100.0	Hominy, cooked, fat added in cooking	75217520
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223000
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223020
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223021
100.0	Peas, cowpeas, field peas, or blackeye peas (not dried), coo	75223022
100.0	Peas, green, cooked, NS as to form, NS as to fat added in co	75224010
100.0	Peas, green, cooked, from fresh, NS as to fat added in cooki	75224011
100.0	Peas, green, cooked, from frozen, NS as to fat added in cook	75224012
100.0	Peas, green, cooked, NS as to form, fat not added in cooking	75224020
100.0	Peas, green, cooked, from fresh, fat not added in cooking	75224021
100.0	Peas, green, cooked, from frozen, fat not added in cooking	75224022
100.0	Peas, green, cooked, NS as to form, fat added in cooking	75224030
100.0	Peas, green, cooked, from fresh, fat added in cooking	75224031
100.0	Peas, green, cooked, from frozen, fat added in cooking	75224032
100.0	Pigeon peas, cooked, NS as to form, fat not added in cooking	75225010
100.0	Squash, summer, cooked, NS as to form, NS as to fat added in	75233000
100.0	Squash, summer, cooked, from fresh, NS as to fat added in co	75233001
100.0	Squash, summer, cooked, from frozen, NS as to fat added in c	75233002
100.0	Squash, summer, cooked, NS as to form, fat not added in cook	75233010
100.0	Squash, summer, cooked, from fresh, fat not added in cooking	75233011
100.0	Squash, summer, cooked, from frozen, fat not added in cookin	75233012
100.0	Squash, summer, cooked, NS as to form, fat added in cooking	75233020
100.0	Squash, summer, cooked, from fresh, fat added in cooking	75233021
100.0	Beans, lima and corn (succotash), cooked, fat not added in c	75301110
100.0	Beans, lima and corn (succotash), cooked, fat added in cooki	75301120
100.0	Peas and corn, cooked, NS as to fat added in cooking	75315000
100.0	Peas and corn, cooked, fat not added in cooking	75315010
100.0	Peas and corn, cooked, fat added in cooking	75315020
100.0	Squash, baby food, strained	76205010
100.0	Corn, creamed, baby food, strained	76405010

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Corn, creamed, baby food, junior	76405020
100.0	Peas, baby food, NS as to strained or junior	76409000
100.0	Peas, baby food, strained	76409010
100.0	Peas, baby food, junior	76409020
100.0	Marmalade, all flavors	91404000
12.5	Beet soup (borscht)	75601100
12.5	Leek soup, cream of, prepared with milk	75605010
12.5	Onion soup, French	75608100
12.5	Vegetables and rice, baby food, strained	76501000
12.5	Vegetable and bacon, baby food, strained	76601010
12.5	Vegetable and beef, baby food, strained	76603010
12.5	Vegetable and beef, baby food, junior	76603020
12.5	Vegetable and chicken, baby food, strained	76605010
12.5	Vegetable and chicken, baby food, junior	76605020
12.5	Vegetable and ham, baby food, strained	76607010
12.5	Vegetable and ham, baby food, junior	76607020
12.5	Vegetable and turkey, baby food, strained	76611010
12.5	Vegetable and turkey, baby food, junior	76611020
12.5	Puerto Rican stew (Sancocho)	77563010
25.0	Raw vegetable, NFS	75100250
25.0	Vegetables, NS as to type, cooked, NS as to fat added in coo	75200100
25.0	Vegetables, NS as to type, cooked, fat not added in cooking	75200110
25.0	Vegetable combination (including carrots, broccoli, and/or d	75440100
25.0	Vegetable combination (excluding carrots, broccoli, and dark	75440110
25.0	Vegetable tempura	75440200
25.0	Vegetables, dipped in chick-pea flour batter, (pakora), frie	75440400
25.0	Vegetable combinations (including carrots, broccoli, and/or	75440500
25.0	Vegetable combination (including carrots, broccoli, and/or d	75450500
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460700
25.0	Vegetable combinations (excluding carrots, broccoli, and dar	75460710
25.0	Vegetable combinations (including carrots, broccoli, and/or	75460800

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
25.0	Vegetable soup, home recipe	75649110
25.0	Vegetable noodle soup, home recipe	75649150
25.0	Vegetable beef soup, home recipe	75652010
25.0	Vegetable beef soup with noodles or pasta, home recipe	75652040
25.0	Vegetable beef soup with rice, home recipe	75652050
25.0	Spanish stew, Puerto Rican style (Cocido Espanol)	77513010
33.0	Mixed vegetable juice (vegetables other than tomato)	75132000
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340110
33.0	Vegetable combinations (broccoli, carrots, corn, cauliflower	75340120
33.0	Vegetable stew without meat	75439010
33.0	Mixed vegetables, garden vegetables, baby food, NS as to str	76407000

Table D.4 Food Codes for Root Vegetables

% Root Produce in Food Item	Food Item Description	USDA Food Code
33.0	Mixed vegetables, garden vegetables, baby food, strained	76407010
33.0	Mixed vegetables, garden vegetables, baby food, junior	76407020
50.0	Potato pancake	71701000
50.0	Norwegian Lefse, potato and flour pancake	71701500
50.0	Stewed potatoes, Mexican style (Papas guisadas)	71703000
50.0	Stewed potatoes with tomatoes, Mexican style (Papas guisadas	71703040
50.0	Stewed potatoes with tomatoes	71704000
50.0	Potato soup, NS as to made with milk or water	71801000
50.0	Potato soup, cream of, prepared with milk	71801010
50.0	Potato soup, prepared with water	71801020
50.0	Potato soup, instant, made from dry mix	71801040
50.0	Potato and cheese soup	71801100
50.0	Macaroni and potato soup	71802010
50.0	Potato chowder	71803010
50.0	Peas and carrots, cooked, NS as to form, NS as to fat added	73111200
50.0	Peas and carrots, cooked, from fresh, NS as to fat added in	73111201
50.0	Peas and carrots, cooked, from frozen, NS as to fat added in	73111202
50.0	Peas and carrots, cooked, NS as to form, fat not added in co	73111210
50.0	Peas and carrots, cooked, from fresh, fat not added in cooki	73111211
50.0	Peas and carrots, cooked, from frozen, fat not added in cook	73111212
50.0	Peas and carrots, cooked, NS as to form, fat added in cookin	73111220
50.0	Peas and carrots, cooked, from fresh, fat added in cooking	73111221
50.0	Peas and carrots, cooked, from frozen, fat added in cooking	73111222
50.0	Carrot soup, cream of, prepared with milk	73501000
50.0	Tomato and onion, cooked, NS as to fat added in cooking	74504100
50.0	Tomato and onion, cooked, fat not added in cooking	74504110
50.0	Tomato and onion, cooked, fat added in cooking	74504120
50.0	Beans, green, and potatoes, cooked, fat not added in cooking	75302050
50.0	Beans, green, and potatoes, cooked, NS as to fat added in co	75302500
50.0	Beans, green, and potatoes, cooked, fat added in cooking	75302510

50.0	Peas and onions, cooked, fat not added in cooking	75315110
50.0	Peas and potatoes, cooked, fat not added in cooking	75315300
50.0	Squash, summer, and onions, cooked, fat not added in cooking	75316000
50.0	Onion rings, NS as to form, batter-dipped, baked or fried	75415020
50.0	Onion rings, from fresh, batter-dipped, baked or fried	75415021
50.0	Carrots and peas, baby food, strained	76202000
50.0	Carrots and beef, baby food, strained	76602000
50.0	Sweetpotatoes and chicken, baby food, strained	76604500
75.0	White potato, cooked, with cheese	71301020
75.0	White potato, cooked, with ham and cheese	71301120
75.0	White potato, scalloped	71305010
75.0	White potato, scalloped, with ham	71305110
75.0	Carrots, cooked, from fresh, creamed	73102231
75.0	Carrots, cooked, NS as to form, glazed	73102240
75.0	Carrots, cooked, from fresh, glazed	73102241
75.0	Carrots, cooked, from frozen, glazed	73102242
75.0	Carrots, cooked, from fresh, with cheese sauce	73102251
75.0	Carrots in tomato sauce	73111400
100.0	White potato, NFS	71000100
100.0	White potato, baked, peel not eaten	71101000
100.0	White potato, baked, peel eaten, NS as to fat added in cooki	71101100
100.0	White potato, baked, peel eaten, fat not added in cooking	71101110
100.0	White potato, baked, peel eaten, fat added in cooking	71101120
100.0	White potato skins, with adhering flesh, baked	71101150
100.0	White potato, boiled, without peel, NS as to fat added in co	71103000
100.0	White potato, boiled, without peel, fat not added in cooking	71103010
100.0	White potato, boiled, without peel, fat added in cooking	71103020
100.0	White potato, boiled, with peel, NS as to fat added in cooki	71103100
100.0	White potato, boiled, with peel, fat not added in cooking	71103110
100.0	White potato, boiled, with peel, fat added in cooking	71103120
100.0	White potato, boiled, without peel, canned, low sodium, fat	71103210
100.0	White potato, roasted, NS as to fat added in cooking	71104000
100.0	White potato, roasted, fat not added in cooking	71104010
100.0	White potato, roasted, fat added in cooking	71104020
100.0	White potato, sticks	71205000

100.0	White potato skins, chips	71211000
100.0	White potato, french fries, NS as to from fresh or frozen	71401000
100.0	White potato, french fries, from fresh, deep fried	71401010
100.0	White potato, french fries, from frozen, oven baked	71401020
100.0	White potato, french fries, from frozen, deep fried	71401030
100.0	White potato, french fries, breaded or battered	71402040
100.0	White potato, home fries	71403000
100.0	White potato, home fries, with green or red peppers and onio	71403500
100.0	White potato, hash brown, NS as to from fresh, frozen, or dr	71405000
100.0	White potato, hash brown, from fresh	71405010
100.0	White potato, hash brown, from frozen	71405020
100.0	White potato, hash brown, with cheese	71405100
100.0	White potato skins, with adhering flesh, fried	71410000
100.0	White potato skins, with adhering flesh, fried, with cheese	71410500
100.0	White potato skins, with adhering flesh, fried, with cheese	71411000
100.0	White potato, mashed, NFS	71501000
100.0	White potato, from fresh, mashed, made with milk	71501010
100.0	White potato, from fresh, mashed, made with milk, sour cream	71501015
100.0	White potato, from fresh, mashed, made with milk and fat	71501020
100.0	White potato, from fresh, mashed, made with fat	71501030
100.0	White potato, from fresh, mashed, made with milk, fat and ch	71501050
100.0	White potato, from fresh, mashed, not made with milk or fat	71501080
100.0	White potato, from fresh, mashed, NS as to milk or fat	71501310
100.0	White potato, patty	71503010
100.0	White potato, puffs	71505000
100.0	White potato, stuffed, baked, peel not eaten, NS as to toppi	71507000
100.0	White potato, stuffed, baked, peel not eaten, stuffed with s	71507010
100.0	White potato, stuffed, baked, peel not eaten, stuffed with c	71507020
100.0	White potato, stuffed, baked, peel not eaten, stuffed with b	71507040
100.0	White potato, stuffed, baked, peel eaten, stuffed with sour	71508010
100.0	White potato, stuffed, baked, peel eaten, stuffed with chees	71508020
100.0	White potato, stuffed, baked, peel eaten, stuffed with chili	71508030
100.0	White potato, stuffed, baked, peel eaten, stuffed with brocc	71508040
100.0	White potato, stuffed, baked, peel eaten, stuffed with meat	71508050
100.0	White potato, stuffed, baked, peel eaten, stuffed with bacon	71508060

100.0	White potato, stuffed, baked, peel not eaten, stuffed with b	71508070
100.0	Potato salad with egg	71601010
100.0	Potato salad, German style	71602010
100.0	Potato salad	71603010
100.0	Carrots, raw	73101010
100.0	Carrots, raw, salad	73101110
100.0	Carrots, raw, salad with apples	73101210
100.0	Carrots, cooked, NS as to form, NS as to fat added in cookin	73102200
100.0	Carrots, cooked, from fresh, NS as to fat added in cooking	73102201
100.0	Carrots, cooked, from frozen, NS as to fat added in cooking	73102202
100.0	Carrots, cooked, NS as to form, fat not added in cooking	73102210
100.0	Carrots, cooked, from fresh, fat not added in cooking	73102211
100.0	Carrots, cooked, from frozen, fat not added in cooking	73102212
100.0	Carrots, cooked, NS as to form, fat added in cooking	73102220
100.0	Carrots, cooked, from fresh, fat added in cooking	73102221
100.0	Carrots, cooked, from frozen, fat added in cooking	73102222
100.0	Sweetpotato, NFS	73401000
100.0	Sweetpotato, baked, peel eaten, fat not added in cooking	73402010
100.0	Sweetpotato, baked, peel eaten, fat added in cooking	73402020
100.0	Sweetpotato, baked, peel not eaten, NS as to fat added in co	73403000
100.0	Sweetpotato, baked, peel not eaten, fat not added in cooking	73403010
100.0	Sweetpotato, baked, peel not eaten, fat added in cooking	73403020
100.0	Sweetpotato, boiled, without peel, NS as to fat added in coo	73405000
100.0	Sweetpotato, boiled, without peel, fat not added in cooking	73405010
100.0	Sweetpotato, boiled, without peel, fat added in cooking	73405020
100.0	Sweetpotato, boiled, with peel, fat not added in cooking	73405110
100.0	Sweetpotato, boiled, with peel, fat added in cooking	73405120
100.0	Sweetpotato, candied	73406000
100.0	Sweetpotato, canned, NS as to syrup	73407000
100.0	Sweetpotato, canned without syrup	73407010
100.0	Sweetpotato, canned in syrup, with fat added in cooking	73407030
100.0	Sweetpotato, casserole or mashed	73409000
100.0	Sweetpotato, fried	73410110
100.0	Beets, raw	75102500
100.0	Garlic, raw	75111500

100.0	Jicama, raw	75111800
100.0	Onions, young green, raw	75117010
100.0	Onions, mature, raw	75117020
100.0	Radish, raw	75125000
100.0	Turnip, raw	75129000
100.0	Beets, cooked, NS as to form, NS as to fat added in cooking	75208000
100.0	Beets, cooked, NS as to form, fat not added in cooking	75208010
100.0	Beets, cooked, from fresh, fat not added in cooking	75208011
100.0	Beets, cooked, NS as to form, fat added in cooking	75208020
100.0	Beets, cooked, from fresh, fat added in cooking	75208021
100.0	Garlic, cooked	75217400
100.0	Onions, mature, cooked, NS as to form, NS as to fat added in	75221000
100.0	Onions, mature, cooked, from fresh, NS as to fat added in co	75221001
100.0	Onions, mature, cooked, from frozen, NS as to fat added in c	75221002
100.0	Onions, mature, cooked, NS as to form, fat not added in cook	75221010
100.0	Onions, mature, cooked, from fresh, fat not added in cooking	75221011
100.0	Onions, mature, cooked or sauteed, NS as to form, fat added	75221020
100.0	Onions, mature, cooked or sauteed, from fresh, fat added in	75221021
100.0	Onions, mature, cooked or sauteed, from frozen, fat added in	75221022
100.0	Onions, pearl, cooked, NS as to form	75221030
100.0	Onions, pearl, cooked, from fresh	75221031
100.0	Onion, young green, cooked, NS as to form, NS as to fat adde	75221040
100.0	Onions, young green, cooked, NS as to form, fat not added in	75221050
100.0	Onions, young green, cooked, from fresh, fat not added in co	75221051
100.0	Onion, young green, cooked, from fresh, fat added in cooking	75221061
100.0	Parsnips, cooked, fat not added in cooking	75222010
100.0	Parsnips, cooked, fat added in cooking	75222020
100.0	Radish, Japanese (daikon), cooked, fat added in cooking	75227110
100.0	Turnip, cooked, from fresh, NS as to fat added in cooking	75234001
100.0	Turnip, cooked, NS as to form, fat not added in cooking	75234010
100.0	Turnip, cooked, from fresh, fat not added in cooking	75234011
100.0	Turnip, cooked, from fresh, fat added in cooking	75234021
100.0	Vegetables, stew type (including potatoes, carrots, onions,	75317000
100.0	Vegetables, stew type (including potatoes, carrots, onions,	75317010
100.0	Vegetables, stew type (including potatoes, carrots, onions,	75317020

100.0	Beets with Harvard sauce	75405010
100.0	Beets, pickled	75500210
100.0	Carrots, baby food, NS as to strained or junior	76201000
100.0	Carrots, baby food, strained	76201010
100.0	Carrots, baby food, junior	76201020
100.0	Carrots, baby food, toddler	76201030
100.0	Sweetpotatoes, baby food, NS as to strained or junior	76209000
100.0	Sweetpotatoes, baby food, strained	76209010

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
12.5	Meat loaf, NS as to type of meat	27260010
12.5	Meatballs, with breading, NS as to type of meat, with gravy	27260050
12.5	Gumbo, no rice (New Orleans type with shellfish, pork, and/o	27464000
12.5	Meat loaf dinner, NFS (frozen meal)	28160300
12.5	Meat loaf with potatoes, vegetable (frozen meal)	28160310
12.5	Meatball soup, Mexican style (Sopa de Albondigas)	28310230
12.5	Chicken soup with noodles and potatoes, Puerto Rican style	28340220
12.5	Chicken gumbo soup	28340310
12.5	Chicken noodle soup, chunky style	28340510
12.5	Chicken soup, canned, undiluted	28340520
12.5	Chicken soup	28340530
12.5	Sweet and sour soup	28340550
12.5	Chicken soup with vegetables (broccoli, carrots, celery, pot	28340580
12.5	Chicken corn soup with noodles, home recipe	28340590
12.5	Chicken or turkey vegetable soup, stew type	28340610
12.5	Chicken vegetable soup with rice, stew type, chunky style	28340630
12.5	Chicken vegetable soup with noodles, stew type, chunky style	28340640
12.5	Chicken or turkey vegetable soup, home recipe	28340660
12.5	Chicken vegetable soup with rice, Mexican style (Sopa / Cald	28340670
12.5	Hot and sour soup	28340750
12.5	Chicken soup with vegetables and fruit, Oriental Style	28340800
12.5	Chicken or turkey soup, cream of, canned, reduced sodium, ma	28345030
12.5	Chicken or turkey soup, cream of, canned, reduced sodium, un	28345040
12.5	Chicken or turkey soup, cream of, NS as to prepared with mil	28345110
12.5	Chicken or turkey soup, cream of, prepared with milk	28345120
12.5	TAMALE W/ MEAT &/OR POULTRY (INCL TAMALE, NFS)	58103110
12.5	Tamale casserole with meat	58103310
12.5	Quesadilla with meat and cheese	58104730
12.5	TAQUITOES	58104810
12.5	Meat turnover, Puerto Rican style (Pastelillo de carne; Empa	58116110

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
12.5	Empanada, Mexican turnover, filled with meat and vegetables	58116120
12.5	Dumpling, meat-filled	58121510
12.5	Quiche with meat, poultry or fish	58125110
12.5	Turnover, meat-filled, no gravy	58126110
12.5	Turnover, meat- and cheese-filled, no gravy	58126130
12.5	Turnover, meat- and bean-filled, no gravy	58126140
12.5	Turnover, meat- and cheese-filled, tomato-based sauce	58126150
12.5	Turnover, meat-and vegetable- filled (no potatoes, no gravy)	58126170
12.5	Dressing with chicken or turkey and vegetables	58128220
12.5	Stuffed pepper, with meat	58162090
12.5	Stuffed pepper, with rice and meat	58162110
12.5	Chicken noodle soup	58403010
12.5	Chicken noodle soup, home recipe	58403040
12.5	Chicken rice soup	58404010
12.5	Chicken soup with dumplings	58404520
12.5	Turkey noodle soup, home recipe	58406020
25.0	Turnover, chicken- or turkey-, and cheese-filled, no gravy	58126270
25.0	Turnover, chicken- or turkey-, and vegetable-filled, lower i	58126280
33.0	Chicken or turkey, potatoes, and vegetables (including carro	27341010
33.0	Chicken or turkey, potatoes, and vegetables (excluding carro	27341020
33.0	Chicken or turkey stew with potatoes and vegetables (includi	27341310
33.0	Chicken or turkey stew with potatoes and vegetables (excludi	27341320
33.0	Chicken or turkey stew with potatoes and vegetables (includi	27341510
33.0	Chicken or turkey stew with potatoes and vegetables (excludi	27341520
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343010
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343020
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343470
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343480
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343510
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343520
33.0	Chicken or turkey chow mein or chop suey with noodles	27343910

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
33.0	Chicken or turkey, noodles, and vegetables (including carrot	27343950
33.0	Chicken or turkey, noodles, and vegetables (excluding carrot	27343960
33.0	CHICKEN, NOODLES, VEG (NO CAR/DK GRN), CREAM SAUCE	27343980
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345010
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345020
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345210
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345220
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345310
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345320
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345410
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345420
33.0	Chicken or turkey, rice, and vegetables (including carrots,	27345440
33.0	Chicken or turkey, rice, and vegetables (excluding carrots,	27345520
33.0	Chicken or turkey pot pie	27347100
33.0	Chicken or turkey, dumplings, and vegetables (including carr	27347240
33.0	Chicken or turkey, dumplings, and vegetables (excluding carr	27347250
33.0	Chicken, fried, with potatoes, vegetable (frozen meal)	28140710
33.0	Chicken patty, or nuggets, boneless, breaded, potatoes, vege	28140720
33.0	Chicken patty, breaded, with tomato sauce and cheese, fettuc	28140730
33.0	Chicken patty, or nuggets, boneless, breaded, with pasta and	28140740
33.0	Chicken, fried, with potatoes, vegetable, dessert (frozen me	28140810
33.0	Chicken, fried, with potatoes, vegetable, dessert (frozen me	28141010
33.0	CHICKEN PATTY W/ VEGETABLES (DIET FROZEN MEAL)	28141060
33.0	CHICKEN TERIYAKI W/ RICE, VEGETABLE (FROZEN MEAL)	28141200
33.0	Chicken with rice-vegetable mixture (diet frozen meal)	28141250
33.0	Chicken with rice and vegetable, reduced fat and sodium (die	28141300
33.0	Chicken a la king with rice (frozen meal)	28141600
33.0	Chicken and vegetables in cream or white sauce (diet frozen	28141610
33.0	Chicken and vegetable entree with rice, Oriental (diet froze	28143020
33.0	Chicken and vegetable entree, oriental (diet frozen meal)	28143030
33.0	Chicken chow mein with rice (diet frozen meal)	28143040

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
33.0	Chicken with noodles and cheese sauce (diet frozen meal)	28143080
33.0	Chicken cacciatore with noodles (diet frozen meal)	28143110
33.0	Chicken and vegetable entree with noodles (frozen meal)	28143130
33.0	Chicken and vegetable entree with noodles (diet frozen meal)	28143150
33.0	Chicken in cream sauce with noodles and vegetable (frozen me	28143170
33.0	Chicken in butter sauce with potatoes and vegetable (diet fr	28143180
33.0	Chicken in soy-based sauce, rice and vegetables (frozen meal	28143200
33.0	Chicken in orange sauce with almond rice (diet frozen meal)	28143210
33.0	Chicken in barbecue sauce, with rice, vegetable and dessert,	28143220
33.0	Chicken and vegetable entree with noodles and cream sauce (f	28144100
33.0	Turkey dinner, NFS (frozen meal)	28145000
33.0	TURKEY W/ DRESSING, GRAVY, POTATO (FROZEN MEAL)	28145010
33.0	Turkey with dressing, gravy, vegetable and fruit (diet froze	28145100
33.0	Turkey with vegetable, stuffing (diet frozen meal)	28145110
33.0	Turkey with gravy, dressing, potatoes, vegetable (frozen mea	28145210
33.0	Turkey with gravy, dressing, potatoes, vegetable, dessert (f	28145610
33.0	Burrito with chicken, no beans	58100200
33.0	Burrito with chicken and beans	58100210
33.0	Burrito with chicken, beans, and cheese	58100220
33.0	Burrito with chicken and cheese	58100230
33.0	Burrito with chicken, NFS	58100240
33.0	Enchilada with chicken, tomato-based sauce	58100600
33.0	Enchilada with chicken, beans, and cheese, tomato- based sau	58100620
33.0	Enchilada with chicken and cheese, no beans, tomato- based s	58100630
33.0	Flauta with chicken	58101240
33.0	Soft taco with chicken, cheese, and lettuce	58101450
33.0	Soft taco with chicken, cheese, lettuce, tomato and sour cre	58101460
33.0	Taco or tostada with chicken or turkey, lettuce, tomato and	58101510
33.0	Taco or tostada with chicken, cheese, lettuce, tomato and sa	58101520
33.0	Nachos with chicken or turkey and cheese	58104250
33.0	Chimichanga with chicken and cheese	58104530

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
33.0	Fajita with chicken and vegetables	58105000
33.0	Cornmeal dressing with chicken or turkey and vegetables	58128120
33.0	Rice with chicken, Puerto Rican style (Arroz con Pollo)	58155110
50.0	Chicken or turkey and potatoes with gravy (mixture)	27241010
50.0	Chicken or turkey and noodles, no sauce (mixture)	27242000
50.0	Chicken or turkey and noodles with gravy (mixture)	27242200
50.0	Chicken or turkey and noodles with (mushroom) soup (mixture)	27242250
50.0	Chicken or turkey and noodles with cream or white sauce (mix	27242300
50.0	Chicken or turkey and noodles with cheese sauce (mixture)	27242310
50.0	Chicken or turkey and noodles, tomato-based sauce (mixture)	27242400
50.0	Chicken or turkey and rice, no sauce (mixture)	27243000
50.0	Chicken or turkey and rice with cream sauce (mixture)	27243300
50.0	Chicken or turkey and rice with (mushroom) soup (mixture)	27243400
50.0	Chicken or turkey and rice with tomato-based sauce (mixture)	27243500
50.0	Chicken or turkey and rice with soy-based sauce (mixture)	27243600
50.0	Chicken or turkey with dumplings (mixture)	27246100
50.0	Chicken or turkey with stuffing (mixture)	27246200
50.0	Chicken or turkey and vegetables (including carrots, broccol	27440110
50.0	Chicken or turkey and vegetables (excluding carrots, broccol	27440120
50.0	Chicken or turkey and vegetables (including carrots, broccol	27442110
50.0	Chicken or turkey and vegetables (excluding carrots, broccol	27442120
50.0	Chicken or turkey a la king with vegetables (including carro	27443110
50.0	Chicken or turkey a la king with vegetables (excluding carro	27443120
50.0	Chicken or turkey divan	27443150
50.0	Chicken or turkey and vegetables (including carrots, broccol	27445110
50.0	Chicken or turkey and vegetables (excluding carrots, broccol	27445120
50.0	General Tso (General Gau) chicken	27445150
50.0	Moo Goo Gai Pan	27445180
50.0	Kung pao chicken	27445220
50.0	Almond chicken	27445250

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
50.0	Chicken or turkey chow mein or chop suey, no noodles	27446100
50.0	Chicken or turkey salad	27446200
50.0	Chicken or turkey salad with egg	27446220
50.0	Chicken or turkey garden salad (chicken and/or turkey, tomat	27446300
50.0	Chicken or turkey garden salad (chicken and/or turkey, other	27446310
50.0	Chicken or turkey and vegetables (including carrots, broccol	27446400
75.0	Meat loaf made with chicken or turkey	27246500
75.0	Chicken sandwich, with spread	27540110
75.0	Chicken barbecue sandwich	27540130
75.0	Chicken fillet (breaded, fried) sandwich	27540140
75.0	Chicken fillet (breaded, fried) sandwich with lettuce, tomat	27540150
75.0	Chicken patty sandwich, miniature, with spread	27540170
75.0	Chicken patty sandwich or biscuit	27540180
75.0	Chicken patty sandwich, with lettuce and spread	27540190
75.0	Fajita-style chicken sandwich with cheese, on pita bread, wi	27540200
75.0	Chicken patty sandwich with cheese, on wheat bun, with lettu	27540230
75.0	Chicken fillet, (broiled), sandwich, on whole wheat roll, wi	27540240
75.0	Chicken fillet, broiled, sandwich with cheese, on whole whea	27540250
75.0	Chicken fillet, broiled, sandwich, on oat bran bun, with let	27540260
75.0	Chicken fillet, broiled, sandwich, with lettuce, tomato, and	27540270
75.0	Chicken fillet, broiled, sandwich with cheese, on bun, with	27540280
100.0	Chicken, NS as to part and cooking method, NS as to skin eat	24100000
100.0	Chicken, NS as to part and cooking method, skin eaten	24100010
100.0	Chicken, NS as to part and cooking method, skin not eaten	24100020
100.0	CHICKEN, BONELESS, BROILED, NS PART, NS SKIN	24101000
100.0	CHICKEN, BONELESS, BROILED, NS PART, W/O SKIN	24101020
100.0	Chicken, NS as to part, roasted, broiled, or baked, NS as to	24102000
100.0	Chicken, NS as to part, roasted, broiled, or baked, skin eat	24102010
100.0	Chicken, NS as to part, roasted, broiled, or baked, skin not	24102020
100.0	Chicken, NS as to part, stewed, NS as to skin eaten	24103000
100.0	Chicken, NS as to part, stewed, skin eaten	24103010

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, NS as to part, stewed, skin not eaten	24103020
100.0	Chicken, NS as to part, fried, no coating, NS as to skin eat	24104000
100.0	Chicken, NS as to part, fried, no coating, skin not eaten	24104020
100.0	CHICKEN, BONELESS, FLOURED, BAKED/FRIED, NS SKIN	24105000
100.0	CHICKEN, BONELESS, FLOURED, BAKED/FRIED, W/ SKIN	24105010
100.0	CHICKEN, BONELESS, BREADED, BAKED/FRIED, NS SKIN	24106000
100.0	CHICKEN, BONELESS, BREADED, BAKED/FRIED, W/ SKIN	24106010
100.0	CHICKEN,BONELESS,BREADD,BAKD/FRIED,W/O SKIN,NS COAT	24106040
100.0	CHICKEN,BONELESS,BREADD,BAKED/FRIED,W/O SKIN,W/COAT	24106050
100.0	Chicken, NS as to part, coated, baked or fried, prepared wit	24107000
100.0	Chicken, NS as to part, coated, baked or fried, prepared wit	24107010
100.0	Chicken, NS as to part, coated, baked or fried, prepared wit	24107020
100.0	Chicken, NS as to part, coated, baked or fried, prepared ski	24107050
100.0	CHICKEN, W/ BONE, NFS	24110000
100.0	CHICKEN, W/ BONE, NS AS TO PART, ROASTED, W/ SKIN	24112010
100.0	CHICKEN,W/BONE,NS PART,BREADED,BAKD/FRIED, W/O SKIN	24116020
100.0	Chicken, breast, NS as to cooking method, NS as to skin eate	24120100
100.0	Chicken, breast, NS as to cooking method, skin eaten	24120110
100.0	Chicken, breast, NS as to cooking method, skin not eaten	24120120
100.0	CHICKEN, BREAST, BROILED, NS AS TO SKIN	24121100
100.0	CHICKEN, BREAST, BROILED, W/SKIN	24121110
100.0	CHICKEN, BREAST, BROILED, W/O SKIN	24121120
100.0	Chicken, breast, roasted, broiled, or baked, NS as to skin e	24122100
100.0	Chicken, breast, roasted, broiled, or baked, skin eaten	24122110
100.0	Chicken, breast, roasted, broiled, or baked, skin not eaten	24122120
100.0	Chicken, breast, stewed, NS as to skin eaten	24123100
100.0	Chicken, breast, stewed, skin eaten	24123110
100.0	Chicken, breast, stewed, skin not eaten	24123120
100.0	Chicken, breast, fried, no coating, NS as to skin eaten	24124100
100.0	Chicken, breast, fried, no coating, skin eaten	24124110
100.0	Chicken, breast, fried, no coating, skin not eaten	24124120

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	CHICKEN, BREAST, FLOURED,BAKED/FRIED, NS AS TO SKIN	24125100
100.0	CHICKEN, BREAST, FLOURED, BAKED/FRIED, W/ SKIN	24125110
100.0	CHICKEN, BREAST, FLOURED, BAKED/FRIED, W/O SKIN	24125120
100.0	CHICKEN,BREAST,FLOURED,BAKED/FRIED,W/O SKIN,NS COAT	24125140
100.0	CHICKEN, BREAST, BREADED,BAKED/FRIED, NS AS TO SKIN	24126100
100.0	CHICKEN, BREAST, BREADED, BAKED/FRIED, W/ SKIN	24126110
100.0	CHICKEN, BREAST, BREADED, BAKED/FRIED, W/O SKIN	24126120
100.0	CHICKEN,BREAST,BREADED,BAKED/FRIED, SKINLESS,W/COAT	24126150
100.0	CHICKEN,BREAST,BREADED,BAKED/FRIED,W/O SKIN,NO COAT	24126160
100.0	Chicken, breast, coated, baked or fried, prepared with skin,	24127100
100.0	Chicken, breast, coated, baked or fried, prepared with skin,	24127110
100.0	Chicken, breast, coated, baked or fried, prepared with skin,	24127120
100.0	Chicken, breast, coated, baked or fried, prepared skinless,	24127140
100.0	Chicken, breast, coated, baked or fried, prepared skinless,	24127150
100.0	Chicken, breast, coated, baked or fried, prepared skinless,	24127160
100.0	Chicken, leg (drumstick and thigh), NS as to cooking method,	24130200
100.0	Chicken, leg (drumstick and thigh), NS as to cooking method,	24130220
100.0	CHICKEN, LEG, BROILED, NS AS TO SKIN	24131200
100.0	CHICKEN, LEG, BROILED, W/ SKIN	24131210
100.0	CHICKEN, LEG, BROILED, W/O SKIN	24131220
100.0	Chicken, leg (drumstick and thigh), roasted, broiled, or bak	24132200
100.0	Chicken, leg (drumstick and thigh), roasted, broiled, or bak	24132210
100.0	Chicken, leg (drumstick and thigh), roasted, broiled, or bak	24132220
100.0	Chicken, leg (drumstick and thigh), stewed, NS as to skin ea	24133200
100.0	Chicken, leg (drumstick and thigh), stewed, skin eaten	24133210
100.0	Chicken, leg (drumstick and thigh), stewed, skin not eaten	24133220
100.0	Chicken, leg (drumstick and thigh), fried, no coating, NS as	24134200
100.0	Chicken, leg (drumstick and thigh), fried, no coating, skin	24134210
100.0	Chicken, leg (drumstick and thigh), fried, no coating, skin	24134220
100.0	CHICKEN, LEG, FLOURED, BAKED/FRIED, NS AS TO SKIN	24135200
100.0	CHICKEN, LEG, FLOURED, BAKED/FRIED, W/ SKIN	24135210

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	CHICKEN, LEG, FLOURED, BAKED/FRIED, W/O SKIN	24135220
100.0	CHICKEN, LEG, BREADED, BAKED/FRIED, W/ SKIN	24136210
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137210
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137220
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137240
100.0	Chicken, leg (drumstick and thigh), coated, baked or fried,	24137250
100.0	Chicken, drumstick, NS as to cooking method, NS as to skin e	24140200
100.0	Chicken, drumstick, NS as to cooking method, skin eaten	24140210
100.0	Chicken, drumstick, NS as to cooking method, skin not eaten	24140220
100.0	CHICKEN, DRUMSTICK, BROILED, NS AS TO SKIN	24141200
100.0	CHICKEN, DRUMSTICK, BROILED, W/ SKIN	24141210
100.0	CHICKEN, DRUMSTICK, BROILED, W/O SKIN	24141220
100.0	Chicken, drumstick, roasted, broiled, or baked, NS as to ski	24142200
100.0	Chicken, drumstick, roasted, broiled, or baked, skin eaten	24142210
100.0	Chicken, drumstick, roasted, broiled, or baked, skin not eat	24142220
100.0	Chicken, drumstick, stewed, NS as to skin eaten	24143200
100.0	Chicken, drumstick, stewed, skin eaten	24143210
100.0	Chicken, drumstick, stewed, skin not eaten	24143220
100.0	Chicken, drumstick, fried, no coating, NS as to skin eaten	24144200
100.0	Chicken, drumstick, fried, no coating, skin eaten	24144210
100.0	Chicken, drumstick, fried, no coating, skin not eaten	24144220
100.0	CHICKEN, DRUMSTICK,FLOURED,BAKD/FRIED,NS AS TO SKIN	24145200
100.0	CHICKEN, DRUMSTICK, FLOURED, BAKED/FRIED, W/ SKIN	24145210
100.0	CHICKEN, DRUMSTICK, FLOURED, BAKED/FRIED, W/O SKIN	24145220
100.0	CHICKEN,DRUMSTICK,FLOURD,BAKD/FRID,W/O SKIN,W/ COAT	24145250
100.0	CHICKEN, DRUMSTICK, BREADED, BAKED/FRIED, W/ SKIN	24146210
100.0	CHICKEN, DRUMSTICK, BREADED, BAKED/FRIED, W/O SKIN	24146220
100.0	CHICKEN,DRUMSTICK,BREADED,BAKD/FRID,SKINLESS,W/COAT	24146250
100.0	CHICKEN,DRUMSTICK,BREADD,BAKD/FRID,W/O SKIN,NO COAT	24146260
100.0	Chicken, drumstick, coated, baked or fried, prepared with sk	24147200
100.0	Chicken, drumstick, coated, baked or fried, prepared with sk	24147210

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, drumstick, coated, baked or fried, prepared with sk	24147220
100.0	Chicken, drumstick, coated, baked or fried, prepared skinles	24147240
100.0	Chicken, drumstick, coated, baked or fried, prepared skinles	24147250
100.0	Chicken, drumstick, coated, baked or fried, prepared skinles	24147260
100.0	Chicken, thigh, NS as to cooking method, NS as to skin eaten	24150200
100.0	Chicken, thigh, NS as to cooking method, skin eaten	24150210
100.0	Chicken, thigh, NS as to cooking method, skin not eaten	24150220
100.0	CHICKEN, THIGH, BROILED, NS AS TO SKIN	24151200
100.0	CHICKEN, THIGH, BROILED, W/ SKIN	24151210
100.0	CHICKEN, THIGH, BROILED, W/O SKIN	24151220
100.0	Chicken, thigh, roasted, broiled, or baked, NS as to skin e	24152200
100.0	Chicken, thigh, roasted, broiled, or baked, skin eaten	24152210
100.0	Chicken, thigh, roasted, broiled, or baked, skin not eaten	24152220
100.0	Chicken, thigh, stewed, NS as to skin eaten	24153200
100.0	Chicken, thigh, stewed, skin eaten	24153210
100.0	Chicken, thigh, stewed, skin not eaten	24153220
100.0	Chicken, thigh, fried, no coating, NS as to skin eaten	24154200
100.0	Chicken, thigh, fried, no coating, skin eaten	24154210
100.0	Chicken, thigh, fried, no coating, skin not eaten	24154220
100.0	CHICKEN, THIGH, FLOURED, BAKED/FRIED, NS AS TO SKIN	24155200
100.0	CHICKEN, THIGH, FLOURED, BAKED/FRIED, W/ SKIN	24155210
100.0	CHICKEN, THIGH, FLOURED, BAKED/FRIED, W/O SKIN	24155220
100.0	CHICKEN, THIGH, BREADED, BAKED/FRIED, W/ SKIN	24156210
100.0	CHICKEN, THIGH, BREADED, BAKED/FRIED, W/O SKIN	24156220
100.0	CHICKEN,THIGH,BREADED,BAKD/FRIED,SKINLESS,W/COATING	24156250
100.0	CHICKEN,THIGH,BREADED,BAKED/FRIED,W/O SKIN,NO COAT	24156260
100.0	Chicken, thigh, coated, baked or fried, prepared with skin,	24157200
100.0	Chicken, thigh, coated, baked or fried, prepared with skin,	24157210
100.0	Chicken, thigh, coated, baked or fried, prepared with skin,	24157220
100.0	Chicken, thigh, coated, baked or fried, prepared skinless, N	24157240

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken, thigh, coated, baked or fried, prepared skinless, c	24157250
100.0	Chicken, thigh, coated, baked or fried, prepared skinless, c	24157260
100.0	Chicken, wing, NS as to cooking method, NS as to skin eaten	24160100
100.0	Chicken, wing, NS as to cooking method, skin eaten	24160110
100.0	Chicken, wing, NS as to cooking method, skin not eaten	24160120
100.0	CHICKEN, WING, BROILED, W/ SKIN	24161110
100.0	CHICKEN, WING, BROILED, W/O SKIN	24161120
100.0	Chicken, wing, roasted, broiled, or baked, NS as to skin eat	24162100
100.0	Chicken, wing, roasted, broiled, or baked, skin eaten	24162110
100.0	Chicken, wing, roasted, broiled, or baked, skin not eaten	24162120
100.0	Chicken, wing, stewed, NS as to skin eaten	24163100
100.0	Chicken, wing, stewed, skin eaten	24163110
100.0	Chicken, wing, stewed, skin not eaten	24163120
100.0	Chicken, wing, fried, no coating, NS as to skin eaten	24164100
100.0	Chicken, wing, fried, no coating, skin eaten	24164110
100.0	Chicken, wing, fried, no coating, skin not eaten	24164120
100.0	CHICKEN, WING, FLOURED, BAKED/FRIED, NS AS TO SKIN	24165100
100.0	CHICKEN, WING, FLOURED, BAKED/FRIED, W/ SKIN	24165110
100.0	CHICKEN, WING, FLOURED, BAKED/FRIED, W/O SKIN	24165120
100.0	CHICKEN, WING, BREADED, BAKED/FRIED, W/ SKIN	24166110
100.0	CHICKEN, WING, BREADED, BAKED/FRIED, W/O SKIN	24166120
100.0	Chicken, wing, coated, baked or fried, prepared with skin, N	24167100
100.0	Chicken, wing, coated, baked or fried, prepared with skin, s	24167110
100.0	Chicken, wing, coated, baked or fried, prepared with skin, s	24167120
100.0	Chicken, back	24170200
100.0	CHICKEN, BACK, ROASTED, W/O SKIN	24172220
100.0	CHICKEN, BACK, STEWED, NS AS TO SKIN	24173200
100.0	CHICKEN, BACK, STEWED, W/ SKIN	24173210
100.0	Chicken, neck or ribs	24180200
100.0	Chicken skin	24198440

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Chicken feet	24198500
100.0	CHICKEN, CANNED, MEAT ONLY, LIGHT MEAT	24198550
100.0	Chicken, canned, meat only	24198570
100.0	CHICKEN ROLL, ROASTED, NS AS TO LIGHT OR DARK MEAT	24198640
100.0	Chicken patty, fillet, or tenders, breaded, cooked	24198700
100.0	Chicken, ground	24198720
100.0	Chicken nuggets	24198740
100.0	Chicken crackling, Puerto Rican style (Chicharron de pollo)	24198840
100.0	Turkey, NFS	24201000
100.0	Turkey, light meat, cooked, NS as to skin eaten	24201010
100.0	Turkey, light meat, cooked, skin not eaten	24201020
100.0	Turkey, light meat, cooked, skin eaten	24201030
100.0	Turkey, light meat, breaded, baked or fried, NS as to skin e	24201050
100.0	Turkey, light meat, breaded, baked or fried, skin not eaten	24201060
100.0	Turkey, light meat, roasted, NS as to skin eaten	24201110
100.0	Turkey, light meat, roasted, skin not eaten	24201120
100.0	Turkey, light meat, roasted, skin eaten	24201130
100.0	Turkey, dark meat, roasted, NS as to skin eaten	24201210
100.0	Turkey, dark meat, roasted, skin not eaten	24201220
100.0	Turkey, light and dark meat, roasted, NS as to skin eaten	24201310
100.0	Turkey, light and dark meat, roasted, skin not eaten	24201320
100.0	Turkey, light and dark meat, roasted, skin eaten	24201330
100.0	Turkey, light or dark meat, battered, fried, skin not eaten	24201360
100.0	Turkey, light or dark meat, stewed, NS as to skin eaten	24201400
100.0	Turkey, light or dark meat, stewed, skin not eaten	24201410
100.0	Turkey, light or dark meat, smoked, cooked, NS as to skin ea	24201500
100.0	Turkey, light or dark meat, smoked, cooked, skin not eaten	24201520
100.0	Turkey, drumstick, cooked, skin not eaten	24202010
100.0	Turkey, drumstick, cooked, skin eaten	24202020
100.0	Turkey, drumstick, roasted, NS as to skin eaten	24202050
100.0	Turkey, drumstick, roasted, skin not eaten	24202060

Table D.5 Food Codes for Poultry Items

% Poultry in Food Item	Food Item Description	USDA Food Code
100.0	Turkey, drumstick, roasted, skin eaten	24202070
100.0	Turkey, thigh, cooked, NS as to skin eaten	24202450
100.0	Turkey, thigh, cooked, skin eaten	24202460
100.0	Turkey, thigh, cooked, skin not eaten	24202500
100.0	Turkey, neck, cooked	24202600
100.0	Turkey, wing, cooked, NS as to skin eaten	24203000
100.0	Turkey, wing, cooked, skin not eaten	24203010
100.0	Turkey, wing, cooked, skin eaten	24203020
100.0	Turkey, rolled roast, light or dark meat, cooked	24204000
100.0	Turkey, canned	24206000
100.0	Turkey, ground	24207000
100.0	Turkey, nuggets	24208000
100.0	CHICKEN LIVER, BATTERED, FRIED	25110410
100.0	Chicken liver, braised	25110420
100.0	CHICKEN LIVER, FRIED OR SAUTEED, NO COATING	25110440
100.0	Chicken liver, fried	25110450
100.0	Liver paste or pate, chicken	25112200
100.0	Chicken or turkey cake, patty, or croquette	27246300

Table D.6 Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Beef, NS as to cut, cooked, NS as to fat eaten	21000100
100.0	Beef, NS as to cut, cooked, lean and fat eaten	21000110
100.0	Beef, NS as to cut, cooked, lean only eaten	21000120
100.0	Steak, NS as to type of meat, cooked, NS as to fat eaten	21001000
100.0	Steak, NS as to type of meat, cooked, lean and fat eaten	21001010
100.0	Steak, NS as to type of meat, cooked, lean only eaten	21001020
100.0	Beef, pickled	21002000
100.0	Beef, NS as to cut, fried, NS to fat eaten	21003000
100.0	Beef steak, NS as to cooking method, NS as to fat eaten	21101000
100.0	Beef steak, NS as to cooking method, lean and fat eaten	21101010
100.0	Beef steak, NS as to cooking method, lean only eaten	21101020
100.0	Beef steak, broiled or baked, NS as to fat eaten	21101110
100.0	Beef steak, broiled or baked, lean and fat eaten	21101120
100.0	Beef steak, broiled or baked, lean only eaten	21101130
100.0	Beef steak, fried, NS as to fat eaten	21102110
100.0	Beef steak, fried, lean and fat eaten	21102120
100.0	Beef steak, fried, lean only eaten	21102130
100.0	Beef steak, breaded or floured, baked or fried, NS as to fat	21103110
100.0	Beef steak, breaded or floured, baked or fried, lean and fat	21103120
100.0	Beef steak, breaded or floured, baked or fried, lean only ea	21103130
100.0	Beef steak, battered, fried, NS as to fat eaten	21104110
100.0	Beef steak, battered, fried, lean and fat eaten	21104120
100.0	Beef steak, battered, fried, lean only eaten	21104130
100.0	Beef steak, braised, NS as to fat eaten	21105110
100.0	Beef steak, braised, lean and fat eaten	21105120
100.0	Beef steak, braised, lean only eaten	21105130
100.0	Beef, oxtails, cooked	21301000
100.0	Beef, neck bones, cooked	21302000
100.0	Beef, shortribs, cooked, NS as to fat eaten	21304000
100.0	Beef, shortribs, cooked, lean and fat eaten	21304110

Table D.6 Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Beef, shortribs, cooked, lean only eaten	21304120
100.0	Beef, shortribs, barbecued, with sauce, NS as to fat eaten	21304200
100.0	Beef, shortribs, barbecued, with sauce, lean and fat eaten	21304210
100.0	Beef, shortribs, barbecued, with sauce, lean only eaten	21304220
100.0	Beef, cow head, cooked	21305000
100.0	Beef, roast, roasted, NS as to fat eaten	21401000
100.0	Beef, roast, roasted, lean and fat eaten	21401110
100.0	Beef, roast, roasted, lean only eaten	21401120
100.0	Beef, roast, canned	21401400
100.0	Beef, pot roast, braised or boiled, NS as to fat eaten	21407000
100.0	Beef, pot roast, braised or boiled, lean and fat eaten	21407110
100.0	Beef, pot roast, braised or boiled, lean only eaten	21407120
100.0	Beef, stew meat, cooked, NS as to fat eaten	21410000
100.0	Beef, stew meat, cooked, lean and fat eaten	21410110
100.0	Beef, stew meat, cooked, lean only eaten	21410120
100.0	Beef brisket, cooked, NS as to fat eaten	21417100
100.0	Beef brisket, cooked, lean and fat eaten	21417110
100.0	Beef brisket, cooked, lean only eaten	21417120
100.0	Beef, sandwich steak (flaked, formed, thinly sliced)	21420100
100.0	Ground beef or patty, cooked, NS as to regular, lean, or ext	21500100
100.0	Ground beef, meatballs, meat only, cooked, NS as to regular,	21500110
100.0	Ground beef or patty, breaded, cooked	21500200
100.0	Ground beef, regular, cooked	21501000
100.0	Ground beef, lean, cooked	21501200
100.0	Ground beef, extra lean, cooked	21501300
100.0	Beef, bacon, cooked	21601000
100.0	Beef, bacon, cooked, lean only eaten	21601250
100.0	Beef, dried, chipped, uncooked	21602000
100.0	Beef jerky	21602100
100.0	Beef, pastrami (beef, smoked, spiced)	21603000

Table D.6 Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Beef, baby food, strained	21701010
100.0	Beef liver, braised	25110120
100.0	Beef liver, fried	25110140
100.0	Beef sausage, NFS	25220100
100.0	Beef sausage, fresh, bulk, patty or link, cooked	25220140
66.0	Beef with tomato-based sauce (mixture)	27111000
66.0	Spaghetti sauce with beef or meat other than lamb or mutton,	27111050
66.0	Beef goulash	27111100
66.0	Mexican style beef stew, no potatoes, tomato-based sauce (mi	27111300
66.0	Mexican style beef stew, no potatoes, with chili peppers, to	27111310
66.0	Beef sloppy joe (no bun)	27111500
66.0	Beef with gravy (mixture)	27112000
66.0	Salisbury steak with gravy (mixture)	27112010
66.0	Beef stroganoff	27113100
66.0	Creamed chipped or dried beef	27113200
66.0	Beef with (mushroom) soup (mixture)	27114000
66.0	Beef with soy-based sauce (mixture)	27115000
66.0	Steak teriyaki with sauce (mixture)	27115100
66.0	Beef with barbecue sauce (mixture)	27116200
66.0	Beef with sweet and sour sauce (mixture)	27116300
66.0	Stewed, seasoned, ground beef, Mexican style (Picadillo de c	27116350
66.0	Stewed seasoned ground beef, Puerto Rican style (Picadillo g	27118120
33.0	Beef and potatoes, no sauce (mixture)	27211000
33.0	Beef stew with potatoes, tomato-based sauce (mixture)	27211100
33.0	Mexican style beef stew with potatoes, tomato-based sauce (m	27211110
33.0	Beef goulash with potatoes	27211150
33.0	Beef and potatoes with cream sauce, white sauce or mushroom	27211190
33.0	Beef stew with potatoes, gravy	27211200
33.0	Beef and potatoes with cheese sauce (mixture)	27211500
33.0	Stewed, seasoned, ground beef with potatoes, Mexican style (	27211550

Table D. Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
33.0	Beef and noodles, no sauce (mixture)	27212000
33.0	Beef and macaroni with cheese sauce (mixture)	27212050
33.0	Beef and noodles with tomato-based sauce (mixture)	27212100
33.0	Chili con carne with beans and macaroni	27212120
33.0	Beef goulash with noodles	27212150
33.0	Beef and noodles with gravy (mixture)	27212200
33.0	Beef and noodles with cream or white sauce (mixture)	27212300
33.0	Beef stroganoff with noodles	27212350
33.0	Beef and noodles with (mushroom) soup (mixture)	27212400
33.0	Beef and rice, no sauce (mixture)	27213000
33.0	Beef and rice with tomato-based sauce (mixture)	27213100
33.0	Porcupine balls with tomato-based sauce (mixture)	27213120
33.0	Chili con carne with beans and rice	27213150
33.0	Beef and rice with gravy (mixture)	27213200
33.0	Beef and rice with cream sauce (mixture)	27213300
33.0	Beef and rice with soy-based sauce (mixture)	27213500
66.0	Meat loaf made with beef	27214100
66.0	Meat loaf made with beef, with tomato-based sauce	27214110
12.5	Meat loaf, NS as to type of meat	27260010
12.5	Meatballs, with breading, NS as to type of meat, with gravy	27260050
50.0	Meat loaf made with beef and pork	27260080
33.0	Meat loaf made with beef, veal and pork	27260090
66.0	Beef, potatoes, and vegetables (including carrots, broccoli,	27311110
33.0	Beef stew with potatoes and vegetables (including carrots, b	27311310
33.0	Beef stew with potatoes and vegetables (excluding carrots, b	27311320
33.0	Beef stew with potatoes and vegetables (including carrots, b	27311410
33.0	Beef stew with potatoes and vegetables (excluding carrots, b	27311420
33.0	Shepherd's pie with beef	27311510
33.0	Beef, potatoes, and vegetables (including carrots, broccoli,	27311610
33.0	Beef, potatoes, and vegetables (excluding carrots, broccoli,	27311620

Table D. Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313010
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313020
33.0	Beef chow mein or chop suey with noodles	27313110
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313150
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313160
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313210
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313220
33.0	Beef, noodles, and vegetables (including carrots, broccoli,	27313410
33.0	Beef, noodles, and vegetables (excluding carrots, broccoli,	27313420
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315010
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315020
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315210
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315220
33.0	Stuffed cabbage rolls with beef and rice	27315250
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315310
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315410
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315420
33.0	Beef, rice, and vegetables (including carrots, broccoli, and	27315510
33.0	Beef, rice, and vegetables (excluding carrots, broccoli, and	27315520
33.0	Beef pot pie	27317010
50.0	Beef and vegetables (including carrots, broccoli, and/or dar	27410210
50.0	Beef and vegetables (excluding carrots, broccoli, and dark-g	27410220
50.0	Beef shish kabob with vegetables, excluding potatoes	27410250
50.0	Beef with vegetables (including carrots, broccoli, and/or da	27411100
50.0	Swiss steak	27411120
50.0	Beef rolls, stuffed with vegetables or meat mixture, tomato	27411150
50.0	Beef with vegetables (excluding carrots, broccoli, and dark	27411200
50.0	Beef and vegetables (including carrots, broccoli, and/or dar	27415100
50.0	Beef, tofu, and vegetables (including carrots, broccoli, and	27415120
50.0	Beef chow mein or chop suey, no noodles	27415150

Table D.6 Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
100.0	Pepper steak	27416150
66.0	Beef steak with onions, Puerto Rican style (mixture) (Biftec	27418410
100.0	Liver, beef or calves, and onions	27460750
66.0	Beef barbecue sandwich or Sloppy Joe, on bun	27510110
66.0	Cheeseburger, plain, on bun	27510210
66.0	Cheeseburger, with mayonnaise or salad dressing, on bun	27510220
66.0	Cheeseburger, with mayonnaise or salad dressing and tomatoes	27510230
66.0	Cheeseburger, 1/4 lb meat, plain, on bun	27510240
66.0	Cheeseburger, 1/4 lb meat, with mayonnaise or salad dressing	27510250
66.0	Cheeseburger, 1/4 lb meat, with mushrooms in sauce, on bun	27510260
66.0	Double cheeseburger (2 patties), plain, on bun	27510270
66.0	Double cheeseburger (2 patties), with mayonnaise or salad dr	27510280
66.0	Double cheeseburger (2 patties), plain, on double-decker bun	27510290
66.0	Double cheeseburger (2 patties), with mayonnaise or salad dr	27510300
66.0	Cheeseburger with tomato and/or catsup, on bun	27510310
66.0	Cheeseburger, 1 oz meat, plain, on miniature bun	27510311
66.0	Cheeseburger, 1/4 lb meat, with tomato and/or catsup, on bun	27510320
66.0	Double cheeseburger (2 patties), with tomato and/or catsup,	27510330
66.0	Double cheeseburger (2 patties), with mayonnaise or salad dr	27510340
66.0	Cheeseburger, 1/4 lb meat, with mayonnaise or salad dressing	27510350
66.0	Cheeseburger with mayonnaise or salad dressing, tomato and b	27510360
66.0	Double cheeseburger (2 patties, 1/4 lb meat each), with mayo	27510370
66.0	Triple cheeseburger (3 patties, 1/4 lb meat each), with mayo	27510380
66.0	Double bacon cheeseburger (2 patties, 1/4 lb meat each), on	27510390
66.0	Bacon cheeseburger, 1/4 lb meat, with tomato and/or catsup,	27510400
66.0	Double bacon cheeseburger (2 patties, 1/4 lb meat each), wit	27510430
66.0	Bacon cheeseburger, 1/4 lb meat, with mayonnaise or salad dr	27510440
66.0	Hamburger, plain, on bun	27510500
66.0	Hamburger, with tomato and/or catsup, on bun	27510510
66.0	Hamburger, with mayonnaise or salad dressing and tomatoes, o	27510520

Table D.6 Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
66.0	Hamburger, 1/4 lb meat, plain, on bun	27510530
66.0	Double hamburger (2 patties), with tomato and/or catsup, on	27510540
66.0	Hamburger, 1/4 lb meat, with mayonnaise or salad dressing an	27510560
66.0	Hamburger, with mayonnaise or salad dressing, on bun	27510590
66.0	Hamburger, 1 oz meat, plain, on miniature bun	27510600
66.0	Hamburger, 1/4 lb meat, with tomato and/or catsup, on bun	27510620
66.0	Double hamburger (2 patties), with mayonnaise or salad dress	27510660
66.0	Double hamburger (2 patties), with mayonnaise or salad dress	27510670
66.0	Double hamburger (2 patties, 1/4 lb meat each), with tomato	27510680
66.0	Double hamburger (2 patties, 1/4 lb meat each), with mayonna	27510690
66.0	Meatball and spaghetti sauce submarine sandwich	27510700
66.0	Roast beef sandwich	27513010
66.0	Roast beef submarine sandwich, with lettuce, tomato and spre	27513040
66.0	Roast beef sandwich with cheese	27513050
66.0	Roast beef sandwich with bacon and cheese sauce	27513060
66.0	Steak submarine sandwich with lettuce and tomato	27515000
66.0	Steak sandwich, plain, on roll	27515010
50.0	Beef dinner, NFS (frozen meal)	28110000
50.0	Beef with potatoes (frozen meal, large meat portion)	28110120
50.0	Beef with vegetable (diet frozen meal)	28110150
33.0	Sirloin, chopped, with gravy, mashed potatoes, vegetable (fr	28110220
33.0	Sirloin beef with gravy, potatoes, vegetable (frozen meal)	28110270
33.0	Salisbury steak dinner, NFS (frozen meal)	28110300
33.0	Salisbury steak with gravy, potatoes, vegetable (frozen meal	28110310
33.0	Salisbury steak with gravy, whipped potatoes, vegetable, des	28110330
33.0	Salisbury steak with gravy, potatoes, vegetable, dessert (fr	28110350
33.0	Salisbury steak with gravy, macaroni and cheese, vegetable (	28110370
33.0	Salisbury steak with gravy, macaroni and cheese (frozen meal	28110380
33.0	Salisbury steak, potatoes, vegetable, dessert (diet frozen m	28110390
33.0	Beef, sliced, with gravy, potatoes, vegetable (frozen meal)	28110510

Table D.6 Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
12.5	Meat loaf dinner, NFS (frozen meal)	28160300
12.5	Meat loaf with potatoes, vegetable (frozen meal)	28160310
25.0	Chili beef soup	28310210
12.5	Meatball soup, Mexican style (Sopa de Albondigas)	28310230
25.0	Beef and rice noodle soup, Oriental style (Vietnamese Pho Bo	28310330
25.0	Beef and rice soup, Puerto Rican style	28310420
25.0	Pepperpot (tripe) soup	28311010
25.0	Beef vegetable soup with potato, stew type	28315100
25.0	Beef vegetable soup with noodles, stew type, chunky style	28315120
25.0	Beef vegetable soup with rice, stew type, chunky style	28315130
25.0	Beef vegetable soup, Mexican style (Sopa / caldo de Res)	28315140
33.0	Burrito with beef, no beans	58100100
33.0	Burrito with beef and beans	58100110
33.0	Burrito with beef, beans, and cheese	58100120
33.0	Burrito with beef and cheese, no beans	58100130
33.0	Burrito with beef, beans, cheese, and sour cream	58100140
33.0	Burrito with beef and potato, no beans	58100150
33.0	Enchilada with beef, no beans	58100400
33.0	Enchilada with beef and beans	58100510
33.0	Enchilada with beef, beans, and cheese	58100520
33.0	Enchilada with beef and cheese, no beans	58100530
33.0	Flauta with beef	58101230
33.0	Taco or tostada with beef, cheese and lettuce	58101300
33.0	Taco or tostada with beef, lettuce, tomato and salsa	58101310
33.0	Taco or tostada with beef, cheese, lettuce, tomato and salsa	58101320
33.0	Soft taco with beef, cheese, lettuce, tomato and sour cream	58101350
33.0	Soft taco with beef, cheese, and lettuce	58101400
33.0	Mexican casserole made with ground beef, tomato sauce, chees	58101830
33.0	Taco or tostada salad with beef and cheese, corn chips	58101910
33.0	Taco or tostada salad with beef, beans and cheese, fried flo	58101930

Table D.6 Food Codes for Beef Items

% Beef in Food Item	Food Item Description	USDA food code value
12.5	Tamale casserole with meat	58103310
33.0	Nachos with beef, beans, cheese, and sour cream	58104080
33.0	Nachos with beef, beans, cheese, tomatoes, sour cream and on	58104180
33.0	Chimichanga with beef and tomato	58104450
33.0	Chimichanga, NFS	58104490
33.0	Chimichanga with beef, beans, lettuce and tomato	58104500
33.0	Chimichanga with beef, cheese, lettuce and tomato	58104510
12.5	Quesadilla with meat and cheese	58104730
33.0	Fajita with beef and vegetables	58105050
25.0	Macaroni or noodles with cheese and beef	58145130
12.5	Stuffed pepper, with meat	58162090
12.5	Stuffed pepper, with rice and meat	58162110
12.5	Barley soup	58401010
12.5	Beef noodle soup	58402010
12.5	Beef dumpling soup	58402020
12.5	Beef rice soup	58402030
12.5	Beef noodle soup, home recipe	58402100

Table D.7 Food Codes for Pork Items

% Pork in Food Item	Food Item Description	USDA food code value
12.5	Meat loaf, NS as to type of meat	27260010
12.5	Meatballs, with breading, NS as to type of meat, with gravy	27260050
12.5	Meat loaf dinner, NFS (frozen meal)	28160300
12.5	Meat loaf with potatoes, vegetable (frozen meal)	28160310
12.5	Meatball soup, Mexican style (Sopa de Albondigas)	28310230
12.5	Tamale casserole with meat	58103310
12.5	Quesadilla with meat and cheese	58104730
12.5	TAQUITOES	58104810
12.5	Stuffed pepper, with meat	58162090
12.5	Stuffed pepper, with rice and meat	58162110
25.0	Brunswick stew	27360100
25.0	Gumbo, no rice (New Orleans type with shellfish, pork, and/o	27464000
25.0	Meat and corn hominy soup, Mexican style (Pozole)	28315150
25.0	Pork and rice soup, stew type, chunky style	28320110
25.0	Pork, vegetable soup with potatoes, stew type	28320150
25.0	Pork with vegetable (excluding carrots, broccoli and/or dark	28320300
33.0	Meat loaf made with beef, veal and pork	27260090
33.0	Ham or pork, noodles, and vegetables (including carrots, bro	27320070
33.0	Pork, potatoes, and vegetables (excluding carrots, broccoli,	27320110
33.0	Pork, potatoes, and vegetables (excluding carrots, broccoli,	27320210
33.0	Pork chow mein or chop suey with noodles	27320310
33.0	Pork and vegetables (including carrots, broccoli, and/or dar	27420060
33.0	Greens with ham or pork (mixture)	27420080
33.0	Moo Shu (Mu Shi) Pork, without Chinese pancake	27420160
33.0	Pork and vegetables (excluding carrots, broccoli, and dark-g	27420350
33.0	Pork chow mein or chop suey, no noodles	27420390
33.0	Pork and vegetables (excluding carrots, broccoli, and dark	27420410
33.0	Sausage and vegetables (including carrots, broccoli, and/or	27420450
33.0	Sausage and vegetables (excluding carrots, broccoli, and dar	27420460
33.0	Sausage and peppers, no sauce (mixture)	27420470

Table D.7 Food Codes for Pork Items

% Pork in Food Item	Food Item Description	USDA food code value
33.0	Pork and vegetables (including carrots, broccoli, and/or dar	27420500
33.0	Pork and vegetables (excluding carrots, broccoli, and dark	27420510
33.0	Burrito with pork and beans	58100180
50.0	Meat loaf made with beef and pork	27260080
50.0	Ham or pork salad	27420020
66.0	Pork and rice with tomato-based sauce (mixture)	27220110
66.0	Sausage and rice with tomato-based sauce (mixture)	27220120
66.0	Sausage and rice with (mushroom) soup (mixture)	27220150
66.0	Sausage and noodles with cream or white sauce (mixture)	27220190
66.0	Ham or pork and rice, no sauce (mixture)	27220310
66.0	Ham or pork and potatoes with gravy (mixture)	27220510
66.0	Stewed pig's feet, Puerto Rican style (Patitas de cerdo guis	27221100
66.0	Mexican style pork stew, with potatoes, tomato-based sauce (	27221150
66.0	Pork sandwich, on white roll, with onions, dill pickles and	27520500
100.0	Pork, NS as to cut, cooked, NS as to fat eaten	22000100
100.0	Pork, NS as to cut, cooked, lean and fat eaten	22000110
100.0	Pork, NS as to cut, cooked, lean only eaten	22000120
100.0	Pork, NS as to cut, fried, NS as to fat eaten	22000200
100.0	Pork, NS as to cut, fried, lean and fat eaten	22000210
100.0	Pork, NS as to cut, fried, lean only eaten	22000220
100.0	Pork, NS as to cut, breaded or floured, fried, NS as to fat	22000300
100.0	Pork, NS as to cut, breaded or floured, fried, lean and fat	22000310
100.0	Pork, NS as to cut, breaded or floured, fried, lean only eat	22000320
100.0	Pork, pickled, NS as to cut	22001000
100.0	Pork, ground or patty, cooked	22002000
100.0	Pork, ground or patty, breaded, cooked	22002100
100.0	Pork jerky	22002800
100.0	Pork chop, NS as to cooking method, NS as to fat eaten	22101000
100.0	Pork chop, NS as to cooking method, lean and fat eaten	22101010
100.0	Pork chop, NS as to cooking method, lean only eaten	22101020

Table D.7 Food Codes for Pork Items

% Pork in Food Item	Food Item Description	USDA food code value
100.0	Pork chop, broiled or baked, NS as to fat eaten	22101100
100.0	Pork chop, broiled or baked, lean and fat eaten	22101110
100.0	Pork chop, broiled or baked, lean only eaten	22101120
100.0	Pork chop, breaded or floured, broiled or baked, lean and fa	22101140
100.0	Pork chop, breaded or floured, broiled or baked, lean only e	22101150
100.0	Pork chop, fried, NS as to fat eaten	22101200
100.0	Pork chop, fried, lean and fat eaten	22101210
100.0	Pork chop, fried, lean only eaten	22101220
100.0	Pork chop, breaded or floured, fried, NS as to fat eaten	22101300
100.0	Pork chop, breaded or floured, fried, lean and fat eaten	22101310
100.0	Pork chop, breaded or floured, fried, lean only eaten	22101320
100.0	Pork chop, battered, fried, NS as to fat eaten	22101400
100.0	Pork chop, battered, fried, lean and fat eaten	22101410
100.0	Pork chop, battered, fried, lean only eaten	22101420
100.0	Pork chop, stewed, NS as to fat eaten	22101500
100.0	Pork chop, stewed, lean and fat eaten	22101510
100.0	Pork chop, stewed, lean only eaten	22101520
100.0	Pork chop, smoked or cured, cooked, lean and fat eaten	22107010
100.0	Pork chop, smoked or cured, cooked, lean only eaten	22107020
100.0	Pork steak or cutlet, NS as to cooking method, NS as to fat	22201000
100.0	Pork steak or cutlet, NS as to cooking method, lean and fat	22201010
100.0	Pork steak or cutlet, NS as to cooking method, lean only eat	22201020
100.0	Pork steak or cutlet, battered, fried, NS as to fat eaten	22201050
100.0	Pork steak or cutlet, battered, fried, lean and fat eaten	22201060
100.0	Pork steak or cutlet, battered, fried, lean only eaten	22201070
100.0	Pork steak or cutlet, broiled or baked, NS as to fat eaten	22201100
100.0	Pork steak or cutlet, broiled or baked, lean and fat eaten	22201110
100.0	Pork steak or cutlet, broiled or baked, lean only eaten	22201120
100.0	Pork steak or cutlet, fried, NS as to fat eaten	22201200
100.0	Pork steak or cutlet, fried, lean and fat eaten	22201210

Table D.7 Food Codes for Pork Items

% Pork in Food Item	Food Item Description	USDA food code value
100.0	Pork steak or cutlet, fried, lean only eaten	22201220
100.0	Pork steak or cutlet, breaded or floured, broiled or baked,	22201310
100.0	Pork steak or cutlet, breaded or floured, broiled or baked,	22201320
100.0	Pork steak or cutlet, breaded or floured, fried, NS as to fa	22201400
100.0	Pork steak or cutlet, breaded or floured, fried, lean and fa	22201410
100.0	Pork steak or cutlet, breaded or floured, fried, lean only e	22201420
100.0	Pork, tenderloin, cooked, NS as to cooking method	22210300
100.0	Pork, tenderloin, breaded, fried	22210310
100.0	Pork, tenderloin, braised	22210350
100.0	Pork, tenderloin, baked	22210400
100.0	Pork roast, NS as to cut, cooked, NS as to fat eaten	22400100
100.0	Pork roast, NS as to cut, cooked, lean and fat eaten	22400110
100.0	Pork roast, NS as to cut, cooked, lean only eaten	22400120
100.0	Pork roast, loin, cooked, NS as to fat eaten	22401000
100.0	Pork roast, loin, cooked, lean and fat eaten	22401010
100.0	Pork roast, loin, cooked, lean only eaten	22401020
100.0	Pork roast, shoulder, cooked, lean only eaten	22411020
100.0	Pork roast, smoked or cured, cooked, NS as to fat eaten	22421000
100.0	Pork roast, smoked or cured, cooked, lean and fat eaten	22421010
100.0	Pork roast, smoked or cured, cooked, lean only eaten	22421020
100.0	Canadian bacon, cooked	22501010
100.0	Bacon, NS as to type of meat, cooked	22600100
100.0	Pork bacon, NS as to fresh, smoked or cured, cooked	22600200
100.0	Pork bacon, smoked or cured, cooked	22601000
100.0	Pork bacon, smoked or cured, cooked, lean only eaten	22601020
100.0	Bacon or side pork, fresh, cooked	22601040
100.0	Pork bacon, smoked or cured, lower sodium	22602010
100.0	Pork bacon, formed, lean meat added, cooked	22605010
100.0	Salt pork, cooked	22621000
100.0	Fat back, cooked	22621100

Table D.7 Food Codes for Pork Items

% Pork in Food Item	Food Item Description	USDA food code value
100.0	Pork, spareribs, cooked, NS as to fat eaten	22701000
100.0	Pork, spareribs, cooked, lean and fat eaten	22701010
100.0	Pork, spareribs, cooked, lean only eaten	22701020
100.0	Pork, spareribs, barbecued, with sauce, NS as to fat eaten	22701030
100.0	Pork, spareribs, barbecued, with sauce, lean and fat eaten	22701040
100.0	Pork, spareribs, barbecued, with sauce, lean only eaten	22701050
100.0	Pork, cracklings, cooked	22704010
100.0	Pork ears, tail, head, snout, miscellaneous parts, cooked	22705010
100.0	Pork, neck bones, cooked	22706010
100.0	Pork, pig's feet, cooked	22707010
100.0	Pork, pig's feet, pickled	22707020
100.0	Pork, pig's hocks, cooked	22708010
100.0	Pork skin, rinds, deep-fried	22709010
100.0	Pork skin, boiled	22709110
100.0	PORK LIVER, BREADED, FRIED	25110340
100.0	Pork sausage, fresh, bulk, patty or link, cooked	25221410

Table D.8 Food Codes for Egg Items

% Eggs in Food Item	Food Item Description	USDA Food Code
25	Fried egg sandwich	32201000
25	Egg, cheese, and ham on English muffin	32202010
25	Egg, cheese, and ham on biscuit	32202020
25	Egg, cheese and ham on bagel	32202025
25	Egg, cheese, and sausage on English muffin	32202030
25	Egg, cheese, and beef on English Muffin	32202040
25	Egg, cheese, and steak on bagel	32202045
25	Egg, cheese, and sausage on biscuit	32202050
25	Egg, cheese, and sausage griddle cake sandwich	32202055
25	Egg and sausage on biscuit	32202060
25	Egg, cheese, and bacon on biscuit	32202070
25	Egg, cheese, and bacon griddle cake sandwich	32202075
25	Egg, cheese, and bacon on English muffin	32202080
25	Egg, cheese and bacon on bagel	32202085
25	Egg and bacon on biscuit	32202090
25	Egg and ham on biscuit	32202110
25	Egg, cheese and sausage on bagel	32202120
25	Egg and cheese on biscuit	32202200
25	Egg drop soup	32300100
25	Garlic egg soup, Puerto Rican style (Sopa de ajo)	32301100
25	Burrito with eggs, sausage, cheese and vegetables	58100340
25	Burrito with eggs and cheese, no beans	58100350
25	Croissant sandwich with sausage and egg	58127270
25	Croissant sandwich with ham, egg, and cheese	58127310
25	Croissant sandwich with sausage, egg, and cheese	58127330
25	Croissant sandwich with bacon, egg, and cheese	58127350
33	Egg dessert, custard-like, made with water and sugar, Puerto	32120100
66	Egg foo yung (young), NFS	32105200
66	Chicken egg foo yung (young)	32105210
66	Pork egg foo yung (young)	32105220

Table D.8 Food Codes for Egg Items

% Eggs in Food Item	Food Item Description	USDA Food Code
66	Shrimp egg foo yung (young)	32105230
75	Egg, Benedict	32101500
75	Egg, deviled	32102000
75	Egg salad	32103000
100	Egg, whole, raw	31101010
100	Egg, whole, cooked, NS as to cooking method	31102000
100	Egg, whole, boiled	31103000
100	Egg, whole, poached	31104000
100	Egg, whole, fried	31105000
100	Egg, whole, fried without fat	31105010
100	Egg, whole, baked, fat not added in cooking	31106010
100	Egg, whole, baked, fat added in cooking	31106020
100	Egg, whole, pickled	31107000
100	Egg, white only, cooked	31109010
100	Egg, yolk only, raw	31110010
100	Egg, yolk only, cooked	31111010
100	Egg, creamed	32101000
100	Egg omelet or scrambled egg, NS as to fat added in cooking	32104900
100	Egg omelet or scrambled egg, fat not added in cooking	32104950
100	Egg omelet or scrambled egg, fat added in cooking	32105000
100	Egg omelet or scrambled egg, with cheese	32105010
100	Egg omelet or scrambled egg, with fish	32105020
100	Egg omelet or scrambled egg, with ham or bacon	32105030
100	Egg omelet or scrambled egg, with dark-green vegetables	32105040
100	Egg omelet or scrambled egg, with vegetables other than dark	32105050
100	Egg omelet or scrambled egg, with peppers, onion, and ham	32105060
100	Egg omelet or scrambled egg, with mushrooms	32105070
100	Egg omelet or scrambled egg, with cheese and ham or bacon	32105080
100	Egg omelet or scrambled egg, with cheese, ham or bacon, and	32105085
100	Egg omelet or scrambled egg, with potatoes and/or onions (To	32105100
100	Egg omelet or scrambled egg, with beef	32105110

Table D.8 Food Codes for Egg Items

% Eggs in Food Item	Food Item Description	USDA Food Code
100	Egg omelet or scrambled egg, with sausage and cheese	32105121
100	Egg omelet or scrambled egg, with sausage	32105122
100	Egg omelet or scrambled egg, with hot dogs	32105125
100	Egg omelet or scrambled egg, with onions, peppers, tomatoes,	32105130
100	Egg omelet or scrambled egg, with chorizo	32105160
100	Egg omelet or scrambled egg with chicken	32105170
100	Huevos rancheros	32105180
100	Meringues	32401000

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
50	Cafe con leche prepared with sugar	11561010
50	Ice cream sandwich	13120500
50	Ice cream cookie sandwich	13120550
50	Ice cream cone with nuts, flavors other than chocolate	13120700
50	Ice cream cone, chocolate covered, with nuts, flavors other	13120710
50	Ice cream cone, chocolate covered or dipped, flavors other t	13120720
50	Ice cream cone, no topping, flavors other than chocolate	13120730
50	Ice cream cone, no topping, NS as to flavor	13120740
50	Ice cream cone with nuts, chocolate ice cream	13120750
50	Ice cream cone, chocolate covered or dipped, chocolate ice c	13120760
50	Ice cream cone, no topping, chocolate ice cream	13120770
50	Ice cream cone, chocolate covered, with nuts, chocolate ice	13120780
50	Ice cream sundae cone	13120790
50	Ice cream soda, flavors other than chocolate	13120800
50	Ice cream sundae, fruit topping, with whipped cream	13121100
50	Ice cream sundae, chocolate or fudge topping, with whipped c	13121300
50	Ice cream pie, no crust	13122100
50	Pudding, bread	13210110
50	Pudding, Mexican bread (Capirotada)	13210180
50	Cheese sandwich	14640000
50	Cheese sandwich, grilled	14640100
50	Cheese, nuggets or pieces, breaded, baked, or fried	14660200
75	Pudding, with fruit and vanilla wafers	13241000
100	Milk, NFS	11100000
100	Milk, cow's, fluid, whole	11111000
100	Milk, calcium fortified, cow's, fluid, whole	11111150
100	Milk, cow's, fluid, other than whole, NS as to 2%, 1%, or sk	11112000
100	Milk, cow's, fluid, 2% fat	11112110
100	Milk, cow's, fluid, acidophilus, 1% fat	11112120
100	Milk, cow's, fluid, acidophilus, 2% fat	11112130

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
100	Milk, cow's, fluid, 1% fat	11112210
100	Milk, cow's, fluid, skim or nonfat, 0.5% or less butterfat	11113000
100	Milk, cow's, fluid, lactose reduced, 1% fat	11114300
100	Milk, cow's, fluid, lactose reduced, nonfat	11114320
100	Milk, cow's, fluid, lactose reduced, 2% fat	11114330
100	Milk, cow's, fluid, lactose reduced, whole	11114350
100	Buttermilk, fluid, nonfat	11115000
100	Buttermilk, fluid, 1% fat	11115100
100	Buttermilk, fluid, 2% fat	11115200
100	Milk, goat's, fluid, whole	11116000
100	Yogurt, NS as to type of milk or flavor	11410000
100	Yogurt, plain, NS as to type of milk	11411010
100	Yogurt, plain, whole milk	11411100
100	Yogurt, plain, lowfat milk	11411200
100	Yogurt, plain, nonfat milk	11411300
100	Yogurt, vanilla, lemon, or coffee flavor, NS as to type of m	11420000
100	Yogurt, vanilla, lemon, or coffee flavor, whole milk	11421000
100	Yogurt, vanilla, lemon, maple, or coffee flavor, lowfat milk	11422000
100	Yogurt, vanilla, lemon, maple, or coffee flavor, nonfat milk	11423000
100	Yogurt, vanilla, lemon, maple, or coffee flavor, nonfat milk	11424000
100	Yogurt, chocolate, NS as to type of milk	11425000
100	Yogurt, fruit variety, NS as to type of milk	11430000
100	Yogurt, fruit variety, whole milk	11431000
100	Yogurt, fruit variety, lowfat milk	11432000
100	Yogurt, fruit variety, lowfat milk, sweetened with low-calor	11432500
100	Yogurt, fruit variety, nonfat milk	11433000
100	Yogurt, fruit variety, nonfat milk, sweetened with low-calor	11433500
100	Yogurt, fruit and nuts, lowfat milk	11445000
100	Yogurt, frozen, NS as to flavor, NS as to type of milk	11459990
100	Yogurt, frozen, flavors other than chocolate, NS as to type	11460000
100	Yogurt, frozen, chocolate, NS as to type of milk	11460100

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
100	Yogurt, frozen, NS as to flavor, lowfat milk	11460150
100	Yogurt, frozen, chocolate, lowfat milk	11460160
100	Yogurt, frozen, flavors other than chocolate, lowfat milk	11460170
100	Yogurt, frozen, NS as to flavor, nonfat milk	11460190
100	Yogurt, frozen, chocolate, nonfat milk	11460200
100	Yogurt, frozen, flavors other than chocolate, nonfat milk	11460300
100	Yogurt, frozen, chocolate, nonfat milk, with low-calorie swe	11460400
100	Yogurt, frozen, flavors other than chocolate, nonfat milk, w	11460410
100	Yogurt, frozen, flavors other than chocolate, whole milk	11460440
100	Yogurt, frozen, cone, chocolate	11461250
100	Yogurt, frozen, cone, flavors other than chocolate	11461260
100	Yogurt, frozen, cone, flavors other than chocolate, lowfat m	11461270
100	Yogurt, frozen, cone, chocolate, lowfat milk	11461280
100	Milk, chocolate, NFS	11511000
100	Milk, chocolate, whole milk-based	11511100
100	Milk, chocolate, reduced fat milk-based, 2% (formerly "lowfa	11511200
100	Milk, chocolate, skim milk-based	11511300
100	Milk, chocolate, lowfat milk-based	11511400
100	Cocoa, hot chocolate, not from dry mix, made with whole milk	11512000
100	Cocoa and sugar mixture, milk added, NS as to type of milk	11513000
100	Cocoa and sugar mixture, whole milk added	11513100
100	Cocoa and sugar mixture, reduced fat milk added	11513150
100	Cocoa and sugar mixture, lowfat milk added	11513200
100	Cocoa and sugar mixture, skim milk added	11513300
100	Chocolate syrup, milk added, NS as to type of milk	11513400
100	Chocolate syrup, whole milk added	11513500
100	Chocolate syrup, reduced fat milk added	11513550
100	Chocolate syrup, lowfat milk added	11513600
100	Chocolate syrup, skim milk added	11513700
100	Cocoa, whey, and low-calorie sweetener mixture, lowfat milk	11516000

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
100	Milk beverage, made with whole milk, flavors other than choc	11519000
100	Milk, flavors other than chocolate, whole milk-based	11519050
100	Milk, malted, unfortified, NS as to flavor, made with milk	11520000
100	Milk, malted, unfortified, chocolate, made with milk	11521000
100	Milk, malted, unfortified, natural flavor, made with milk	11522000
100	Milk, malted, fortified, chocolate, made with milk	11526000
100	Milk, malted, fortified, NS as to flavor, made with milk	11527000
100	Eggnog, made with whole milk	11531000
100	Eggnog, made with 2% reduced fat milk (formerly eggnog, made	11531500
100	Milk shake, homemade or fountain-type, NS as to flavor	11541100
100	Milk shake, homemade or fountain-type, chocolate	11541110
100	Milk shake, homemade or fountain-type, flavors other than ch	11541120
100	Milk shake with malt	11541400
100	Milk shake, made with skim milk, chocolate	11541500
100	Milk shake, made with skim milk, flavors other than chocolat	11541510
100	Milk fruit drink	11551050
100	Orange Julius	11552200
100	Fruit smoothie drink, made with fruit or fruit juice and dai	11553000
100	Fruit smoothie drink, NFS	11553100
100	Chocolate-flavored drink, whey- and milk-based	11560000
100	Flavored milk drink, whey- and milk-based, flavors other tha	11560020
100	Instant breakfast, powder, milk added	11612000
100	Instant breakfast, powder, sweetened with low calorie sweete	11613000
100	Cream, NS as to light, heavy, or half and half	12100100
100	Cream, light, fluid	12110100
100	Cream, light, whipped, unsweetened	12110300
100	Cream, half and half	12120100
100	Cream, half and half, fat free	12120110
100	Cream, heavy, fluid	12130100
100	Cream, heavy, whipped, sweetened	12140000
100	Sour cream	12310100

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
100	Sour cream, reduced fat	12310300
100	Sour cream, light	12310350
100	Sour cream, fat free	12310370
100	Dip, sour cream base	12350000
100	Dip, sour cream base, reduced calorie	12350020
100	Spinach dip, sour cream base	12350100
100	Ice cream, NFS	13110000
100	Ice cream, regular, flavors other than chocolate	13110100
100	Ice cream, regular, chocolate	13110110
100	Ice cream, rich, flavors other than chocolate	13110120
100	Ice cream, rich, chocolate	13110130
100	Ice cream, soft serve, flavors other than chocolate	13110200
100	Ice cream, soft serve, chocolate	13110210
100	Ice cream, soft serve, NS as to flavor	13110220
100	ICE CREAM W/ SHERBET	13125100
100	Ice cream, fried	13126000
100	Light ice cream, flavors other than chocolate (formerly ice	13130300
100	Light ice cream, chocolate (formerly ice milk)	13130310
100	Light ice cream, no sugar added, NS as to flavor	13130320
100	Light ice cream, no sugar added, flavors other than chocolat	13130330
100	Light ice cream, no sugar added, chocolate	13130340
100	LIGHT ICE CREAM,PREMIUM, NOT CHOC (FORMERLY ICE MILK)	13130350
100	Light ice cream, soft serve, NS as to flavor (formerly ice m	13130590
100	Light ice cream, soft serve, flavors other than chocolate (f	13130600
100	Light ice cream, soft serve cone, chocolate (formerly ice mi	13130630
100	Light ice cream, soft serve cone, NS as to flavor (formerly	13130640
100	Light ice cream, cone, chocolate (formerly ice milk)	13140550
100	Light ice cream, sundae, soft serve, chocolate or fudge topp	13140660
100	Light ice cream, sundae, soft serve, not fruit or chocolate	13140680
100	LIGHT ICE CREAM,W/ SHERBET OR ICE CREAM (FORMERLY ICE MILK)	13141100

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
100	Sherbet, all flavors	13150000
100	MILK DESSERT, FROZEN, MADE FROM LOWFAT MILK	13160000
100	MILK DESSERT,FZN,LOWFAT,W/LOW CAL SWEET,NOT CHOC	13160100
100	Fat free ice cream, no sugar added, chocolate	13160150
100	Fat free ice cream, no sugar added, flavors other than choco	13160160
100	MILK DESSERT,FROZEN,LOWFAT,NOT CHOCOLATE	13160200
100	MILK DESSERT, FROZEN, LOWFAT, CHOCOLATE	13160210
100	Fat free ice cream, flavors other than chocolate	13160400
100	Fat free ice cream, chocolate	13160410
100	MILK DSRT,FROZ,MILK-FAT FREE,W/SIMPLESSE, NOT CHOC	13160550
100	MILK DESSERT, FROZ, W/ LOW CAL SWEETENER, NOT CHOC	13160600
100	MILK DESSERT, FROZ, W/ LOW CAL SWEETENER, CHOCOLATE	13160650
100	Milk dessert sandwich bar, frozen, made from lowfat milk	13161500
100	Milk dessert bar, frozen, made from lowfat milk and low calo	13161600
100	Light ice cream, bar or stick, with low-calorie sweetener, c	13161630
100	Pudding, NFS	13200110
100	Pudding, chocolate, ready-to-eat, NS as to from dry mix or c	13210220
100	Pudding, chocolate, ready-to-eat, low calorie, containing ar	13210250
100	Pudding, flavors other than chocolate, ready-to-eat, NS as t	13210280
100	Pudding, flavors other than chocolate, ready-to-eat, low cal	13210290
100	Custard	13210300
100	Custard, Puerto Rican style (Flan)	13210350
100	Pudding, rice	13210410
100	Pudding, tapioca, made from home recipe, made with milk	13210500
100	Pudding, tapioca, made from dry mix, made with milk	13210520
100	Pudding, coconut	13210610
100	Puerto Rican pumpkin pudding (Flan de calabaza)	13210810
100	Pudding, flavors other than chocolate, prepared from dry mix	13220110
100	Pudding, chocolate, prepared from dry mix, milk added	13220120
100	Pudding, flavors other than chocolate, prepared from dry mix	13220210

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
100	Pudding, chocolate, prepared from dry mix, low calorie, cont	13220220
100	Mousse, chocolate	13250000
100	Milk dessert or milk candy, Puerto Rican style (Dulce de lec	13252200
100	Barfi or Burfi, Indian dessert, made from milk and/or cream	13252500
100	Tiramisu	13252600
100	Custard pudding, flavor other than chocolate, baby food, NS	13310000
100	Custard pudding, baby food, flavor other than chocolate, str	13311000
100	Custard pudding, baby food, flavor other than chocolate, jun	13312000
100	White sauce, milk sauce	13411000
100	Milk gravy, quick gravy	13412000
100	Cheese, NFS	14010000
100	Cheese, Cheddar or American type, NS as to natural or proces	14010100
100	Cheese, natural, NFS	14100100
100	Cheese, Blue or Roquefort	14101010
100	Cheese, Brick	14102010
100	Cheese, Brie	14103020
100	Cheese, natural, Cheddar or American type	14104010
100	Cheese, Cheddar or American type, dry, grated	14104020
100	Cheese, Colby	14104200
100	Cheese, Colby Jack	14104250
100	Cheese, Feta	14104400
100	Cheese, Fontina	14104600
100	Cheese, goat	14104700
100	Cheese, Gouda or Edam	14105010
100	Cheese, Gruyere	14105200
100	Cheese, Monterey	14106200
100	Cheese, Monterey, lowfat	14106500
100	Cheese, Mozzarella, NFS	14107010
100	Cheese, Mozzarella, whole milk	14107020
100	Cheese, Mozzarella, part skim	14107030
100	Cheese, Mozzarella, nonfat or fat free	14107060

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
100	Cheese, Muenster	14107200
100	Cheese, Muenster, lowfat	14107250
100	Cheese, Parmesan, dry grated	14108010
100	Cheese, Parmesan, hard	14108020
100	Cheese, Parmesan, low sodium	14108050
100	Parmesan cheese topping, fat free	14108060
100	Cheese, Provolone	14108400
100	Cheese, Swiss	14109010
100	Cheese, Swiss, low sodium	14109020
100	Cheese, Swiss, lowfat	14109030
100	Cheese, Cheddar or Colby, low sodium	14110010
100	Cheese, Cheddar or Colby, lowfat	14110030
100	Cheese, Mexican blend	14120010
100	Queso Anejo (aged Mexican cheese)	14131000
100	Queso Asadero	14131500
100	Queso Chihuahua	14132000
100	Queso Fresco	14133000
100	Cheese, cottage, NFS	14200100
100	Cheese, cottage, creamed, large or small curd	14201010
100	Cottage cheese, farmer's	14201200
100	Cheese, Ricotta	14201500
100	Cheese, cottage, with fruit	14202010
100	Cheese, cottage, salted, dry curd	14203020
100	Puerto Rican white cheese (queso del pais, blanco)	14203510
100	Cheese, cottage, lowfat (1-2% fat)	14204010
100	Cheese, cottage, lowfat, with fruit	14204020
100	CHEESE, YOGURT, NFS	14210000
100	Cheese, cream	14301010
100	Cheese, cream, light or lite (formerly called Cream Cheese L	14303010
100	Cheese spread, cream cheese, regular	14420200
100	Cheese, cottage cheese, with gelatin dessert	14610200

Table D.9 Food Codes for Milk Items

% Milk in Food Item	Food Item Description	USDA Food Code
100	Topping from cheese pizza	14620300
100	Topping from vegetable pizza	14620310
100	Topping from meat pizza	14620320
100	Cheese fondue	14630100
100	Cheese sauce	14650100
100	Cheese sauce made with lowfat cheese	14650150
100	Alfredo sauce	14650160

Appendix E Determination of Chemicals for Multipathway Analysis

E.1 Introduction

The AB-2588 program assesses the risk from airborne chemicals that are often emitted by facilities at high temperature and pressure in the presence of particulate matter. Some of these chemicals will be emitted and remain in vapor form. The inhalation cancer risk and noncancer hazard from such volatile chemicals are likely to be much greater than the risk from other possible exposure pathways. Other chemicals, such as semi-volatile organic or metal toxicants, can either be emitted as particles, form particles after emission from the facility, or adhere to existing particles. Some chemicals will partition between the vapor and particulate phases. Some chemicals such as PAHs have been found to have a portion of the particle associated mass in reversible equilibrium with the vapor phase and a portion irreversibly bound (Eiceman and Vandiver, 1983). Chemicals in the particulate phase can be removed from the atmosphere by settling, which can be enhanced by coalescence into larger particles with greater mass.

There are a number of exposure pathways by which humans may be exposed to airborne chemicals in addition to inhalation. Particulate associated chemicals can be deposited directly onto soil, onto the leaves of crops, or onto surface waters. Crops may also be contaminated by root uptake of chemicals. Livestock such as chickens, pigs and cows may be contaminated by inhalation of such chemicals or by consumption of contaminated feed, pasture, or surface waters. Humans may be exposed to these chemicals through inhalation, consumption of crops, soil, surface waters, meat, eggs and dairy products. Infants may be exposed through consumption of human breast milk.

E.2 Criteria for Selection of Chemicals for Multipathway Analysis

Chemicals listed in Appendix A, “Substances for Which Emissions Must be Quantified” that have been previously reported to be emitted by facilities in California under the Air Toxics “Hot Spots” Act were considered as candidates for multipathway analysis. From the chemicals meeting this criteria, chemicals which had been considered in the past to be multipathway chemicals or were thought to be likely candidates were selected for further analysis. We evaluated the extent to which chemicals might be particle bound. Two models were used to determine the fraction of airborne chemical that is in the particle phase, the Junge-Pankow adsorption model and the Koa absorption model.

E.2.1 The Junge-Pankow Adsorption Model as a Means of Determining Gas-Particle Partitioning

Junge (1977) developed a theoretical model for the partitioning of the exchangeable fraction of an airborne chemical between the vapor and particulate phases in the ambient air.

$$\theta = \frac{bS^{(p)}}{P^{s}_{L} + bS^{(p)}} \qquad \textbf{(Eq. E-1)}$$

Where:

θ = fraction of the total mass of chemical on the particle phase (unitless)

b = a constant (mm Hg cm^3/cm^2)

$S^{(p)}$ = total surface area of particle per unit volume of air (cm^2/cm^3)

P^{s}_{L} = saturation pressure of the liquid chemical at ambient temperature (mm Hg)

Junge (1977) did not distinguish between solid and liquid phase vapor pressures. Pankow (1987) recognized the importance of using the liquid phase vapor pressure. When the chemical of interest is a solid at the temperature of interest, the subcooled liquid vapor pressure must be used. The subcooled liquid vapor pressure is an extrapolation of the saturated liquid vapor pressure below the melting point where the compound actually exists as solid (Boethling and McKay, 2000). The subcooled liquid vapor pressure can be estimated using the following equation:

$$P^{s}_{L}/P^{s}_{s} = \exp[\Delta S_f (T_m - T)/RT]/RT \qquad \textbf{(Eq. E-2)}$$

Where:

P^{s}_{L} = sub cooled liquid vapor pressure of the liquid chemical at ambient temperature (Pascal).

P^{s}_{s} = saturated vapor of the solid at room temperature

ΔS_f = entropy of fusion (J/mol K)

T_m = melting point temperature (K)

T = ambient temperature (K)

R = gas constant (8.3143 joules/K mole)

Values for ΔS_f may be obtained in the literature. In cases where a literature value is not available a default value of 56.45 has been suggested by Boethling and McKay (2000).

The percentage of the total mass of chemical (vapor plus particulate fraction) is determined by multiplying θ times 100. The percentage of the total mass of

chemical that is in particulate phase is determined in part by the concentration of particles in the air. For our purposes, we used an average concentration of particles in urban air determined by Whitby (1978). The concentration of particles was 1.04 X 10^{-4} $\mu g/cm^3$. The surface area per μg of particle was assumed to be 0.05 $cm^2/\mu g$. Thus the $S^{(p)}$ is calculated to be 5.2 X 10^{-6} cm^2/cm^3. The value of b used is the default value of 0.1292 mm Hg cm^3/cm^2 recommended by Pankow (1987).

It should be noted that the particle bound associated fraction of some semi-volatile organic toxicants has been found to consist of a non-exchangeable fraction and a fraction which equilibrates with the vapor phase (Bidleman and Foreman, 1987). The equation of Junge (1977) only addresses the exchangeable fraction. This means that the actual fraction of the total mass that is particle bound material may be somewhat higher than the theoretical model which Junge (1977) proposed. The partitioning of semi-volatile organic toxicants between the vapor phase and particles has been experimentally investigated by Bidleman et al. (1986) and Bidleman and Foreman (1987). High volume sampling has been done in several cities in which the particulate and vapor fractions have been collected on filters and adsorbents. This work has supported the validity of the theoretical model of Junge (1977).

The Junge (1977) and Pankow (1987) model appears to be a reasonable model to determine which chemicals emitted by facilities in the AB-2588 program should undergo multipathway analysis. The liquid or subcooled liquid vapor pressure at ambient temperatures determines the fraction of chemical that will be particle associated. The vapor pressure is available for most of the chemicals for which the determination needs to be made.

It should be noted that the Junge (1977) model was designed to look at the partitioning of chemicals between the particle and vapor phases under equilibrium conditions in the atmosphere. The initial conditions under which particle formation may occur as chemicals are emitted into the atmosphere may be different from the conditions assumed by Junge (1977). The chemicals of concern in the AB-2588 program may be emitted at high temperatures and pressures in the presence of a high concentration of particulate matter. Such conditions may favor partitioning of mass toward the particulate fraction. It is also possible that such conditions might favor the formation of a greater fraction of non-exchangeable particle associated chemical which is not taken into account in the Junge (1977) equation. The rapid cooling from high temperature to ambient temperature may also influence the percent of total mass which is particle bound in ways that are not accounted for in the simple equilibrium model of Junge (1977).

E.2.2 The Octanol-Water Partition Coefficient as a Means of Determining Gas-Particle Partitioning

In the past 15 years, there have been advances in the understanding of the partitioning of semi-volatile organic compounds between the gas phase and the organic condensed phase on airborne particles, using the octanol-water partition coefficient as a predictor of gas particle partitioning in the environment. Because the equation for estimating partitioning involves the octanol/air partition coefficient (K_{OA}), this model is referred to as the K_{OA} absorption model, while the Junge-Pankow is known as an adsorption model. Several studies have described the octanol/air partition coefficients for chlorobenzenes, PCBs, DDT, PAHs and polychlorinated naphthalenes (PCNs) (Harner and MacKay, 1995; Komp and McLachlan, 1997; Harner and Bidleman, 1998).

K_{OA} is defined as $K_{OA} = C_o/C_A$, where C_o (mol/L) is the concentration of the compound in 1-octanol and C_A (mol/L) is the gaseous concentration at equilibrium. For the calculation, K_{OA} can be derived as $K_{OA} = K_{OW}/K_{AW} = K_{OW}RT/H$, where K_{OW} is the octanol/water partition coefficient, K_{AW} is the air/water partition coefficient, H is the Henry's Law constant (J/mol), R is the ideal gas constant (J/mol/K), and T is the absolute temperature (degrees K) (Komp and McLachlan, 1997).

The particle/gas partition coefficient (K_P) is defined as $K_P = C_p/C_g$, where C_p is the concentration on particles (ng/µg of particles), and C_g is the gas-phase concentration (ng/m^3 of air) (Harner and Bidleman, 1998). The relation between K_P and K_{OA} is defined as:

$$\log K_P = \log K_{OA} + \log f_{om} - 11.91 \qquad \textbf{(Eq. E-3)}$$

where, f_{om} = organic matter fraction of the particles.

The fraction (ø) of compound in the particle phase is

$$ø = K_P (TSP) / [1 + K_P (TSP)] \qquad \textbf{(Eq. E-4)}$$

where, TSP = total suspended particle concentration.

Using f_{om} = 20% (Harner and Bidleman, 1998) and the afore-mentioned average concentration of particles in urban air determined by Whitby (1978), TSP = 1.04 x 10^{-4} µg/cm^3 = 104 µg/m^3 , we obtained the percentage of compound on particles (ø x 100) for selected chemicals through the K_{OA} absorption model, presented as the last column in Table E.1 below. For many chemicals, the values compare well with those obtained with the Junge-Pankow adsorption model.

A number of studies have been published which evaluated gas-particle partitioning in the urban environment under equilibrium conditions where there were existing particles from a variety of sources (e.g. diesel exhaust, road dust). Existing particles are thought to have a lipid bilayer into which gaseous chemicals can equilibrate. There is some question whether chemicals emitted

from a stack would have time to interact with existing urban particles before reaching nearby receptors. Also, in some cases particulate matter in the air around facilities may not be present in very high concentrations.

E.3 Fraction in particle phase to be considered for multipathway analysis

OEHHA has decided that if either the Koa model or the Junge-Pankow model shows a chemical as $\geq$ 0.5% particle-bound, we will consider it for multipathway assessment. The 0.5% is a relatively small percentage of the total mass. This percentage was chosen in part to compensate for the uncertainties involved in extrapolation of the Junge (1977) model to the conditions under which particles may be formed in the stacks of facilities. Thus chemicals with vapor pressures greater than 1.34×10^{-4} mm Hg at 25° C will not be considered for multipathway analysis. An exception to this rule is the inclusion of hexachlorobenzene (HCB) for multipathway analysis, even though its calculated percentage of total mass in the particulate phase is expected to be below 0.5%. The criteria for including HCB are discussed in Section E.3 below. It should be noted that the chemicals for which noninhalation pathway risks are a significant fraction of the total risk are metals, PAH's, PCB's, polychlorinated dibenzo-p-dioxins and furans. These chemicals have much higher percentages of total mass in the particulate fraction than 0.5%.

There are some toxic compounds without measurable vapor pressure at 25°C such as the metals and their compounds. These metals include lead, mercury compounds, nickel, selenium, fluoride, beryllium, arsenic, chromium VI and cadmium. These toxicants are included on the list of chemicals for multipathway analysis.

In Table E.1 we have calculated the air/particle partition coefficients of the compounds emitted by facilities for which it appeared possible that a significant fraction of the total mass could be in the particulate fraction. In cases where the saturated vapor pressure at a temperature at or near ambient temperature (25°C) is not available; the air/particle coefficient can be calculated using modern tools such as USEPA's SPARC.

For PAHs, consideration for multipathway analysis is largely confined to PAHs with 4 or more fused rings because a significant fraction of their total mass is in the particle phase. Naphthalene contains 2 fused rings and is included in the Hot Spots program as a carcinogen. However, it does not have a significant percentage of its total mass in the particle phase, so is not considered for multipathway analysis. The PAHs with 3 fused rings (e.g., phenanthrene, fluorine, acenaphthene) are also predominantly found in gaseous form and the data are currently too limited or inadequate to list any of them as carcinogens. Laboratory studies of sludge-amended soils containing PAHs have also shown significant loss through volatilization only for PAHs with less than 4 fused rings (Wild and Jones, 1993). Thus, speciated analysis for PAHs that include only the compounds with 4 or more fused rings can be used for multipathway assessment.

Table E1 Calculation of Air/Particle Coefficients and Percent of Particle Associated Total Mass for Selected Chemicals.

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particle Phase	
					Junge-Pankow model	K_{OA} model
4,4-Methylene dianiline	1.0	197	1	NA	NA	31.5
o-Cresol	0.28*	38.2,	2	$2.44x10^{-6}$	$2.44x10^{-4}$	$4.65x10^{-3}$
m-Cresol	0.39**	25	2	$1.71x10^{-6}$	$1.71x10^{-4}$	$6.64x10^{-3}$
p-Cresol	0.37**	25	2	$1.81x10^{-6}$	$1.81x10^{-4}$	$5.45x10^{-3}$
Cellosolve	5.63***	25	3	$1.19x10^{-7}$	$1.19x10^{-5}$	$6.38x10^{-5}$
Cellosolve acetate	2.12***	25	3	$3.17x10^{-7}$	$3.19x10^{-5}$	$3.40x10^{-5}$
Mercury (elemental)	$1.20x10^{-3}$***	25	4	$5.6x10^{-4}$	0.056	NA****
Hexachlorocyclo-hexanes (Lindane)	$1.18x10^{-4}$**	20	5	$5.66x10^{-3}$	0.57	$6.39x10^{-2}$
Phthalates						
Diethylhexylphthalate	$1.97x10^{-7}$***	25	3	$7.73x10^{-1}$	77.3	98.9
Chlorobenzenes						
Chlorobenzene	12.2***	25	6	$5.53x10^{-8}$	$5.53x10^{-6}$	$1.09x10^{-5}$
p-Dichlorobenzene	0.65***	25	6	$1.03x10^{-6}$	$9.93x10^{-5}$	$9.96x10^{-5}$
m-Dichlorobenzene	2.30***	25	6	$1.03x10^{-6}$	$1.03x10^{-4}$	$4.24x10^{-5}$
o-Dichlorobenzene	0.39***	25	6	$1.71x10^{-6}$	$1.71x10^{-4}$	$6.53x10^{-5}$
1,2,3-Trichlorobenzene	0.39*	40	6	$1.71x10^{-6}$	$1.71x10^{-4}$	$3.30x10^{-4}$
1,2,4-Trichlorobenzene	0.45*	38	6	$1.48x10^{-6}$	$1.48x10^{-6}$	$2.88x10^{-4}$
1,2,3,4-Tetrachloro-benzene	$6.58x10^{-2}$*		6	$1.02x10^{-5}$	$1.02x10^{-3}$	$1.39x10^{-3}$
1,2,3,5-Tetrachloro-benzene	0.14*		6	$4.82x10^{-6}$	$4.82x10^{-4}$	$3.41x\ 0^{-4}$
Pentachlorobenzene	$6.67x10^{-3}$*	25	6	$1.01x10^{-4}$	$1.01x10^{-2}$	$7.36x10^{-3}$
Hexachlorobenzene	$2.96x10^{-4}$*	25	6	$2.96x10^{-4}$	$2.96x10^{-2}$	$1.53x10^{-2}$

Table E1 Calculation of Air/Particle Coefficients and Percent of Particle Associated Total Mass for Selected Chemicals.

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particle Phase	
					Junge-Pankow model	K_{OA} model
PAHs						
Naphthalene (2 fused rings)	0.31*	25	7	$2.14x10^{-6}$	$2.14x10^{-4}$	$3.46x10^{-4}$
Acenaphthene (3 fused rings)	$3.02x10^{-3}$*	25	7	$2.23x10^{-5}$	$2.23x10^{-3}$	$4.34x10^{-3}$
Acenaphthylene (3 fused rings)	$6.67x10^{-3}$	25	7	$1.00x10^{-4}$	0.01	$7.55x10^{-3}$
Anthracene (3 fused rings)	$4.2x10^{-6}$*	25	7	$1.57x10^{-2}$	1.57	$6.78x10^{-2}$
Benzo[a]anthracene (4 fused rings)	$4.07x10^{-6}$*	25	7	$1.42x10^{-1}$	14.2	8.15
Chrysene (4 fused rings)	$8.81x10^{-8}$**	25	7	$8.84x10^{-1}$	88.4	$4.82x10^{-5}$
Benzo[a]pyrene (5 fused rings)	$9.23x10^{-8}$	25	7	$8.79x10^{-1}$	87.9	60.2
Benzo[b]fluoranthene (5 fused rings)	$1.59x10^{-7}$	25	7	$8.09x10^{-1}$	80.9	NA****
Benzo[k]fluoranthene (5 fused rings)	$3.7x10^{-8}$*	25	7	$9.48x10^{-1}$	94.8	79.9
Dibenz[a,h]-anthracene (5 fused rings)	$6.07x10^{-11}$**	25	7	$1.00x10^{0}$	100	NA****
Indeno[1,2,3cd]-pyrene (6 fused rings)	$1.19\ x10^{-9}$**	25	8	$9.98x10^{-1}$	99.8	NA****
Chlorophenols						
Pentachlorophenol	$1.73x10^{-3}$*	25	2	$3.88x10^{-4}$	$3.88x10^{-2}$	76.9
2,4,6-Trichlorophenol	$2.8x10^{-02}$*	25	2	$2.34x10^{-5}$	$2.34x10^{-3}$	NA****
2,4,5-Trichlorophenol	$4.59x10^{-02}$*	25	2	$1.46x10^{-5}$	$1.46x10^{-3}$	NA****
Nitrosoamines						
N-Nitrosodiethylamine	$8.60x10^{-1}$***	20	1	$7.81x10^{-7}$	$7.81x10^{-5}$	$2.67x10^{-5}$
N-Nitroso-dimethylamine	8.1***	20	2	$8.29x10^{-8}$	$8.29x10^{-6}$	NA****
N-Nitroso-diphenylamine	$4.12x10^{2}$**	25	2	$1.63x10^{-9}$	$1.63\ x10^{-7}$	NA****
N-Nitrosodi-n-butylamine	$3.0x10^{-2}$***	20	9	$2.24x10^{-5}$	$2.24x10^{-3}$	NA****
N-Nitrosodi-n-propylamine	$4.15x10^{-1}$***	20	2	$1.62x10^{-6}$	$1.62x10^{-4}$	$2.75x10^{-4}$
N-Nintrosopyrrolidine	$7.2x10^{-02}$***	20	9	$9.2x10^{-6}$	$9.2x10^{-4}$	NA****
PCBs						

Table E1 Calculation of Air/Particle Coefficients and Percent of Particle Associated Total Mass for Selected Chemicals.

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particle Phase	
					Junge-Pankow model	K_{OA} model
Aroclor 1016	$1.50x10^{-3}$*	25	6	$4.48x10^{-4}$	$4.48x10^{-2}$	$1.63x10^{-3}$
Aroclor 1221	$1.50x10^{-2}$*	25	6	$4.48x10^{-5}$	$4.48x10^{-3}$	$6.53x10^{-4}$
Aroclor 1232	$4.05x10^{-3}$***	25	6	$1.66x10^{-4}$	0.17	$2.84x10^{-3}$
Aroclor 1242	$4.13x10^{-4}$***	25	6	$1.63x10^{-4}$	0.16	$1.13x10^{-2}$
Aroclor 1248	$3.33x10^{-4}$***	25	6	$1.66x10^{-3}$	0.17	$5.17x10^{-2}$
Aroclor 1254	$7.73x10^{-5}$***	25	6	$8.62x10^{-3}$	0.86	0.142
Aroclor 1260	$4.40x10^{-6}$***	25	6	$1.32x10^{-1}$	13.2	1.23
Dioxins and Furans						
2,3,7,8 Tetrachloro-dibenzo-p-dioxin	$4.5x10^{-7}$*	20	7	$5.97x10^{-1}$	59.7	10.7
2,3,7,8 Tetrachloro-dibenzofuran	$9.21x10^{-7}$*	25	7	$9.97x10^{-1}$	99.7	5.18
1,2,3,4,7 Pentachloro-dibenzodioxin	$5.9x10^{-7}$**	25	7	$5.42x10^{-1}$	54.2	85.7
2,3,4,7,8 Pentachloro-dibenzofuran	$1.63x10^{-7}$*	25	7	$4.22x10^{-1}$	42.2	28.4
1,2,3,4,7,8 Hexachlorodibenzo-p-dioxin	$5.89x10^{-9}$*	25	7	$9.17x10^{-1}$	91.7	78.7
1,2,3,4,7,8 Hexachloro-dibenzofuran	$6.07x10^{-8}$*	25	7	$9.89x10^{-1}$	98.9	30.4
1,2,3,4,6,7,8 Heptachlorodibenzo-p-dioxin	$7.68x10^{-9}$*	25	7	$9.76x10^{-1}$	97.6	83.3
1,2,3,4,6,7,8 Heptachloro-dibenzofuran	$1.68x10^{-8}$*	25	7	$9.76x10^{-1}$	97.6	52.8
1,2,3,4,7,8,9 Heptachloro-dibenzofuran	$9.79x10^{-9}$*	25	7	$9.87x10^{-1}$	98.7	NA****
1,2,3,4,5,6,7,8 Octachloro-dibenzofuran	$1.95x10^{-9}$*	25	7	$9.97x10^{-1}$	99.7	97.1

Table E1 Calculation of Air/Particle Coefficients and Percent of Particle Associated Total Mass for Selected Chemicals.

Chemical	Vapor Pressure (mm Hg)	Temp. (°C)	Ref. (Vapor Press.)	Air/Particle Partition Coefficient (θ)	% Particle Phase	
					Junge-Pankow model	K_{OA} model
1,2,3,4,5,6,7,8 Octachlorodibenzo-p-dioxin	2.08×10^{-9}*	25	7	9.97×10^{-1}	99.7	93.6

1. IARC, 1986;
2. McKay et al. 1992a;
3. McKone et al., 1993;
4. Cohen et al., 1994;
5. ATSDR, 2005;
6. McKay et al., 1992b;
7. McKay et al., 1992c;
8. Montgomery and Welkom, 1990;
9. Klein, 1982

*Indicates subcooled liquid vapor pressure
**Indicates subcooled liquid vapor pressure estimated according to Boethling and McKay, 2000, page 238.
***Indicates Psat liquid (substance is a liquid at 25 °C)
****Not available because Kow and/or Henry's Law constant not found

For the nitrosamines, we were not able to locate saturated vapor pressures for N-nitrosomethylethylamine, N-nitrosomorpholine, and N-nitrosopiperidine. We were able to find saturated vapor pressures for N-nitrosodiethylamine, N-nitrosdimethylamine, N-nitrosodiphenylamine, N-nitrosodi-n-butylamine, N-nitrosodi-n-propylamine and N-nitrosopyrrolidine. None of these compounds had particle associated percentages above 0.5%. N-nitrosopyrrolidine was structurally similar to N-nitrosomorpholine and N-nitrosopiperidine. N-nitrosopyrrolidine has a particle associated percentage of 9.2×10^{-4}. This is well below the 0.5% that we selected as our cutoff. We therefore felt that N-nitrosomorpholine and N-nitrosopiperidine were unlikely to have a particle bound percentage above 0.5% and thus we excluded these compounds from multipathway consideration. N-nitrosomethylethylamine did not appear likely to have a particle bound percentage above N-nitrosodiethylamine, N-nitrosodimethylamine or N-nitrosodi-n-butylamine. All of these nitrosamines are well below the 0.5% cutoff.

Table E2. Chemicals for Which Multipathway Risks Need to be assessed.

4,4 '-methylene dianiline[1]

creosotes

diethylhexylphthalate

hexachlorobenzene

hexachlorocyclohexanes

pentachlorophenol

PAHs (including but not limited to the following:)[2]

- benz[a]anthracene
- benzo[b]fluoranthene
- benzo[j]fluoranthene
- benzo[k]fluoranthene
- benzo[a]pyrene
- dibenz[a,h]acridine
- dibenz[a,j]acridine
- 7H-dibenzo[c,g]carbazole
- 7,12-dimethylbenz[a]anthracene
- 3-methylcholanthrene
- 5-methylchrysene
- dibenz[a,h]anthracene
- dibenzo[a,e]pyrene
- dibenzo[a,h]pyrene
- dibenzo[a,i]pyrene
- dibenzo[a,l]pyrene
- chrysene
- indeno[1,2,3-cd]pyrene

PCBs[3]

Polychlorinated dibenzo-p-dioxins {PCDDs} (including but not limited to the following, but excluding dioxins with less than four chlorines:)[4]

- 2,3,7,8 tetrachlorodibenzo-p-dioxin
- 1,2,3,7,8 pentachloro-p-dioxin
- 1,2,3,4,7,8 hexachlorodibenzo-p-dioxin
- 1,2,3,6,7,8 hexachlorodibenzo-p-dioxin
- 1,2,3,7,8,9 hexachlorodibenzo-p-dioxin
- 1,2,3,4,6,7,8 heptachlorodibenzo-p-dioxin
- 1,2,3,4,5,6,7,8 Octachlorodibenzo-p-dioxin

Table E2. Chemicals for Which Multipathway Risks Need to be Assessed (Cont.).

Polychlorinated dibenzofurans {PCDFs} (including but not limited to the following, but excluding dibenzofurans with less than four chlorines:)[4]

- 2,3,7,8 tetrachlorodibenzofuran
- 1,2,3,7,8 pentachlorodibenzofuran
- 2,3,4,7,8 pentachlorodibenzofuran
- 1,2,3,4,7,8 hexachlorodibenzofuran
- 1,2,3,6,7,8 hexachlorodibenzofuran
- 1,2,3,7,8,9 hexachlorodibenzofuran
- 2,3,4,6,7,8 hexachlorodibenzofuran
- 1,2,3,4,6,7,8 heptachlorodibenzofuran
- 1,2,3,4,7,8,9 heptachlorodibenzofuran
- 1,2,3,4,5,6,7,8 Octachlorodibenzofuran

Metals, semi-metals and inorganic compounds

- arsenic and arsenic compounds
- beryllium and beryllium compounds
- cadmium and cadmium compounds
- soluble compounds of chromium VI
- fluoride and soluble fluoride compounds
- lead and inorganic lead compounds
- inorganic mercury compounds
- nickel and nickel compounds
- selenium and selenium compounds

---

[1] The saturated vapor pressure at 25°C or close to 25°C is not available to our knowledge. The other evidence available, a melting point of 91.5°C and a boiling point of 398-399 °C (Merck, 1989) indicate that it is very likely that a very significant fraction of the chemical emitted into the air would be in the particulate phase. In addition the vapor pressure at 197 °C is only 1 mm (IARC, 1986).

[2] PAHs with three or more fused rings (Table E2) are to be assessed for multipathway analysis. If PAH mixtures are reported instead of speciated PAHs, then the cancer potency of the entire mixture should be treated the same as benzo(a)pyrene.

[3] PCBs is inclusive of all Aroclor mixtures. The information in Table E1 indicates that some of the Aroclor mixtures do not have significant air/particle coefficients. However, it is difficult to determine vapor pressures on mixtures of compounds. OEHHA therefore is proposing to include all of the Aroclors in the list of chemicals for multipathway analysis. The percentage of some individual PCBs in the particulate phase has been measured in air samples (Horstmann and McLachlan, 1998). The particulate phase of tetrachlorinated PCBs (PCB 152) can be expected to be around 1.4%, and increasing to 11.3% for the heptachlorinated PCBs (PCB 180)

[4] From OEHHA analysis (Table E1), it is clear that all polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans should be included in the multipathway analysis.

Table E3 Specific Pathways to be Analyzed for Multipathway Chemicals

Chemical	Soil Ingestion.	Dermal	Meat, Milk & Egg Ingest	Fish Ingestion	Exposed Veg. Ingest.	Leafy Veg. Ingest.	Protected. Veg. Ingest.	Root Veg. Ingest.	Water	Breast Milk Ingestion.
4,4'-methylene dianiline	X	X			X	X			X	
Creosotes	X	X	X	X	X	X			X	
Diethylhexylphthalate	X	X	X	X	X	X			X	
Hexachlorocyclohexanes	X	X	X	X	X	X			X	
Hexachlorobenzene	X	X	X	X	X	X			X	
PAHs	X	X	X	X	X	X			X	X
PCBs	X	X	X	X	X	X			X	X
Pentachlorophenol[a]										
Dioxins & furans	X	X	X	X	X	X			X	X
Inorganic arsenic & cmpds	X	X	X	X	X	X	X	X	X	
Beryllium & compounds	X	X	X	X	X	X	X	X	X	
Cadmium & compounds	X	X	X	X	X	X	X	X	X	
Chromium VI & cmpds	X	X	X[b]	X	X	X	X	X	X	
Lead & compounds	X	X	X	X	X	X	X	X	X	X
Inorganic mercury cmpds	X	X	X	X	X	X	X	X	X	
Nickel & compounds	X	X	X	X	X	X	X	X	X	
Fluoride & compounds	X	X	X		X	X	X	X	X	
Selenium and cmpds	X	X	X	X	X	X	X	X	X	

[a] To be assessed by pathway

[b] Cow's milk only. No multipathway analysis for meat and egg ingestion

OEHHA is recommending that all of the chemicals chosen for multipathway analysis be included in the soil ingestion and dermal pathways. The soil t1/2 values needed to determine concentration in the soil are found in Appendix G. The variates need for the dermal pathway are found in Chapter 6 and Appendix F.

The meat (beef, chicken, pork), cow's milk and egg pathways are listed in one column because the lipid solubility and half-life in the body are common factors which determine if these compounds will be present in these three pathways in appreciable concentrations in the fat of meat, milk and eggs.

E.4 Evidence for Inclusion of Hexachlorobenzene for Multipathway Assessment

In the previous Hot Spots Guidance document, semi-volatile substances with less than 0.5% of their total mass in the particle-associated fraction was not considered for multipathway analysis. Although this is a reasonable cut-off for semi-volatile substances predominantly in the gas phase, an exception is made for hexachlorobenzene (HCB). From Table E1, the Junge model shows HCB with a particle/gas ratio of only 0.0296% at 25 ^{o}C. Normally, this would exclude HCB from multipathway analysis. However, actual field measurements of the air/particle partitioning of HCB in Table E.4 shows that the compound is often found in particle form above 0.5%.

The greater than expected particle fraction for HCB is a likely result of environmental conditions at the locations assessed for HCB. The adsorption of HCB on aerosols and subsequent deposition depends on the vapor pressure, the amount and surface area of aerosol particles, and the relevant environmental temperature (Ballschmiter and Wittlinger, 1991). Colder temperatures and greater airborne particulate levels would increase the particle/gas ratio of HCB. In fact, Ballschmiter and Wittlinger (1991) suggested that the particle fraction found at -8 ^{o}C (3.5%) in a rural region will be similar to the particle fraction in urban areas with higher particulate levels and an air temperature of 15 ^{o}C.

Table E.4. Field study vapor/particle distributions of HCB

Study	Particle fraction Concentration (% particle)	Gas phase Concentration (% gas)
Popp et al., 2000[a] Leipzig area Roitzsch area Greppin area	 0.8 pg/Nm^3 (0.9%) 0.5 pg/Nm^3 (0.3%) 2.6 pg/Nm^3 (0.9%)	 83.1 pg/Nm^3 (99.1%) 145.6 pg/Nm^3 (99.7%) 280.6 pg/Nm^3 (99.1%)
Horstmann and McLachlan, (1998)[b]	0.43 pg/m^3 (0.2%)	210 pg/m^3 (99.8%)
Lane et al., 1992[c] Turkey lake Pt. Petre	 3 pg/m^3 (4.1%) 2 pg/m^3 (2.8%)	 71 pg/m^3 (95.9%) 69 pg/m^3 (97.2%)
Ballschmiter and Wittlinger, 1991[d]	4 pg/m^3 (3.5%)	110 pg/m^3 (96.5%)
Bidleman et al., 1987[e] 20 ^{o}C 0 ^{o}C	 (nd)[f] (0.1%) (nd) (0.7%)	 (nd) (99.9%) (nd) (99.3%)

[a] Air samples collected near chlorobenzene-contaminated sites of Bitterfeld region in Germany over a two-week period during the summer of 1998.
[b] Air samples collected over one year in a forest clearing in Germany from May 1995 to April 1996.
[c] Air samples collected during spring, summer, and fall of 1987 in rural regions of Ontario, Canada.
[d] Air sample taken at a mean ambient temperature of -8 ^{o}C outside a small village near a major road in Germany
[e] Data collected from Stockholm, Denver and Columbia. Vapor phase component possibly overestimated due to volatilization (blowoff) from the particle phase in the sampler.
[f] No concentration data was provided.

In addition, Foreman and Bidleman (1987) have suggested that field measurements of HCB particle fractions may be greater than in laboratory settings because sources in the environment includes combustion-derived HCB particle incorporation. Similar to dioxins, combustion of organic material that includes chlorinated substances has been suggested as a primary source of HCB.

Nevertheless, the minor particle fraction of the HCB results in Table E.4 may still not be sufficient to support a multipathway analysis. However, when the extreme environmental persistence of this compound relative to other predominantly gaseous semi-volatile substances (i.e., nitrosamines and chlorophenols) is taken into account, it appears that even a fraction of the compound depositing in the particle bound phase could result in measurable levels in sediment and soil with possible accumulation over time. Field studies at Lake Superior, a relatively pristine water body in which organics deposit primarily from atmospheric sources, have found that HCB accumulated in water, sediment and fish tissue samples (Eisenreich et al., 1981). In particular, the strong retention of HCB to sediment

particulates in the water allowed much of the historical burden to become immobilized in bottom sediments, with a concomitant reduction in the levels of HCB found in the surface waters.

More evidence for HCB's persistence in soil was observed in a laboratory study. Arial application of HCB in a greenhouse with simulated pasture conditions showed that HCB volatilized fairly rapidly from plant and soil surfaces (Beall, 1976). Only 3.4% of HCB remained in the top 2 cm of soil 19 months after spraying. Residues on the grass grown in the soil volatilized considerably faster, with only 1.5% remaining on the plants after two weeks, and <0.01% at 19 months. However, no significant reduction in HCB was found in the deeper 2-4 cm layer of soil after 19 months, showing HCB to be persistent within the soil, including a resistance to microbial degradation and leaching. The immobilization of HCB within the soil is due to its high Kow, leading to strong adsorption to the soil organic fraction.

E.5 Summary

The theoretical model of Junge (1977) uses the liquid or subcooled liquid vapor pressure to determine the percentage of the total airborne mass of chemical that is particulate. The Koa model uses the octanol-water coefficient as a predictor of gas particle partitioning in the environment. Chemicals with 0.5% of the total mass or more in the particulate fraction at 25°C by either model are considered for multipathway analysis by OEHHA. A list of multipathway chemicals for the AB-2588 program is provided in Table E2. The percentage of the total mass in the particulate phase and the air/particle partition coefficients for these chemicals and a few other selected chemicals are presented in Table E1.

E.6 References

ATSDR, (2005). Toxicological Profile for Hexachlorocylcohexanes. US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry: Atlanta, GA. (as cited by the Intermedia Transport Predictor software developed for the California Air Resources Board by Yorem Cohen, Arthur Winer and Robert Van de Water, UCLA.)

Ballschmiter K, Wittlinger R. (1991). Interhemisphere exchange of hexachlorocyclohexanes, hexachlorobenzene, polychlorobipheneyls, and 1,1-trichloro 2,2-bis(p-chlorolophenyl)ethane in the lower troposphere. Environ Sci Technol 25(6):1103-1111.

Beall ML Jr. (1976). Persistence of aerially applied hexachlorobenzene on grass and soil. J Environ Qual 5:367-369.

Bidleman, T F (1986). Vapor-particle partitioning of semivolatile organic compounds: Estimates from field collections. Environ. Sci. Technol. 20:1038-1043.

Bidleman, T F, Foreman, W T (1987). Vapor-particle partitioning of semivolatile organic compounds. in Sources and Fates of Aquatic Pollutants, Hites, R.A. and Eisenreich, S.J., eds., American Chemical Society: Washington DC, pp 27-56.

Bidleman T F, Idleman, TF, Wideqvist U, Jansson B, Soderlund R. (1987). Organochlorine Pesticides and polychlorinated biphenyls biphenyls in the atomosphere of southern Sweden. Atmos Environ 21 (3):641-654.

Boethling R, McKay D (2000) Handbook of Property Estimation Methods for Chemicals, Environmental Health Sciences, Lewis: Boca Raton

Budavari S, ed. The Merck Index Encyclopedia of Chemicals, Drugs and Biologicals, N.J., Merck and Co. Inc., Rahway, N.J., p469, 1989.

Cohen Y, Winer A M, Creelman L, Stein E, Kwan A, Chu, J (1994). Development of Intermedia Transfer Factors for Toxic Air Pollutants, California Air Resources Board Contract No. A032-170, vol I-VII.

Eiceman G A, Vandiver V J (1983). Adsorption of polycyclic aromatic hydrocarbons on fly ash from a municipal incinerator and a coal-fired plant. Atmos. Environ. 17: 461-465.

Eisenreich S J, Looney B B, Thornton J D. (1981). Airborne organic contaminants in the Great Lakes ecosystem. Environ Sci Tech 15(1):30-38.

Forman WT, Bidleman TF (1987). An experimental system for investigating vapor-particle partitioning of trace organic pollutants. Environ Sci Technol.; 21 (9):869-875.

Harner T, Bidleman TF (1998). Octanol-air partition coefficient for describing particle/gas partitioning of aromatic compounds in urban air. Environ. Sci. Technol.; 32(10):1494-1502

Harner T, MacKay D (1995). Measurement of octanol-air partition coefficients for Chlorobenzenes, PCBs, and DDT. Environ. Sci. Technol.; 29(6):1599-1606

Hortsmann M, McLachlan M S (1998). Atmos deposition of semivolatile organic compounds to two forest canopies. Atmos. Environ.; 32(10):1799-1809.

IARC (1986). Monographs on the Evaluation of the Carcinogenic Risk or Chemicals to Man. World Health Organization, International Agency for Research on Cancer: Geneva. 1972 - present. (multivolume work) 39:348. (as cited by the Intermedia Transport Predictor software developed for the California Air Resources Board by Yorem Cohen, Arthur Winer and Robert Van de Water, UCLA.).

IARC (1986).Monographs on the Evaluation of the Carcinogenic Risk or Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-Present, V. 39, p. 348.

Junge, C. E. (1977). Basic considerations about trace constituents in the atmosphere as related to the fate of global pollutants. in Fate of Pollutants in the Air and Water Environments Part 1, Mechanism of Interaction Between Environments and Mathematical Modeling and The Physical Fate of Pollutants, Volume 8 Advances in Environmental Science and Technology., Suffet, I. H. ed., John Wiley and Sons: New York., pp 1-25.

Klein RG (1982). Toxicol. 23:135-48. (as cited by the Hazardous Substances Data Bank, National Library of Medicine, October, 1996)

Komp P, McLachlan MS (1997). Octanol/air partitioning of Polychlorinated Biphenyls. Environ Toxicol Chem.; 16(12):2433-2437

Lane DA, Johnson ND, Hanely MJ, et al. (1992). Gas-and particle-phase concentrations of alpha-hexachlorocyclohexane, gamma-hexachlorocyclohexane, and hexachlorobenzene in Ontario air. Environ Sci Technol 26(1):126-133.

Mckay, D., Shiu W-Y., and Ma K-C (1992). Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals, Volume IV Oxygen, Nitrogen, and Sulfur Containing Compounds. CRC Lewis: Boca Raton,

Mckay D, Shiu Y-W, Ma K-C (1992) Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals: Monoaromatic Hydrocarbons, Chlorobenzenes and PCBs Vol. 1. Lewis Publishers: Chelsea, MI.

Mckay D, Shiu W-Y and Ma K-C (1992). Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals: Polynuclear Aromatic Hydrocarbons, Polychlorinated Dioxins and Dibenzofurans, Vol. 3. Lewis Publishers: Chelsea, MI.

McKone TE, Daniels JI, Chiao FF, Hsieh DPH (1993) Intermedia Transfer Factors for Fifteen Toxic Pollutants Released in Air Basins in California. California Air Resources Board, Report No. UCRL-CR-115620. (as cited by the Intermedia Transport Predictor software developed for the California Air Resources Board by Yorem Cohen, Arthur Winer and Robert Van de Water, UCLA.)

Montgomery JH, Welkom LM (1990) Groundwater Chemicals Desk Reference. Lewis Publishers: Chelsea, MI. (as cited by the Intermedia Transport Predictor software developed for the California Air Resources Board by Yorem Cohen, Arthur Winer and Robert Van de Water, UCLA.)

Pankow, JF (1987). Review and comparative analysis of the theories on partitioning between the gas and aerosol phases in the atmosphere. Atmos. Environ. 21: 2275-2284.

Popp P, Brüggemann L, Keil P, Thuss U, Weiss H. 2000. Chlorobenzenes and hexachlorocyclohexanes (HCHs) in the atmosphere of Bitterfeld and Leipzig (Germany). Chemosphere. 41(6):849-55.

Whitby, K T (1978). The physical characteristics of sulfur aerosols. Atmos. Environ. 12: 135-159.

Wild, SR, Jones, K.C. 1993. Biological losses of polynuclear aromatic hydrocarbons (PAHs) from soils freshly amended with sewage sludge. Environ Toxicol Chem 12:5-12.

Appendix F

Dermal Exposure to Soil-Bound Hot Spots Multipathway Chemicals: Fractional Absorption (ABS) Values

F.1 Introduction

The absorbed dose resulting from dermal exposure to soil-bound chemicals depends on many factors. An algorithm that describes the uptake of chemicals from soil as a function of exposure duration, exposure frequency, chemical concentration in the soil, soil loading, surface area, body weight, averaging time, and fractional absorption (ABS) is discussed in Chapter 6. The purpose of this appendix is to summarize the derivation of the ABS for the "Hot Spots" multipathway chemicals and present the information used in the development of each chemical ABS. A general discussion of the diverse factors influencing dermal absorption of soil-bound chemicals is presented below preceding the chemical ABS summaries.

A small subset of organic and inorganic compounds evaluated under the Hot Spots program is subject to deposition onto soil, plants and water bodies. Therefore, exposure can occur by pathways other than inhalation. These chemicals are semi-volatile or nonvolatile, and are therefore partially or wholly in the solid or liquid phase after being emitted. Fate and transport of the deposited chemical must then be estimated in order to assess the impact on soil, water and foods that humans come in contact with. The basis for the selection of these compounds as "Hot Spots" multipathway substances can be found in Appendix E. The organic compounds of relevance listed under the "Hot Spots" program include 4,4'-methylene dianiline, hexachlorocyclohexanes, di(2-ethylhexyl)phthalate (DEHP), polychlorinated dibenzodioxins and dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs) and polycyclic aromatic hydrocarbons (PAHs). The inorganic metals and chemicals of relevance include the inorganic salts of arsenic, beryllium, cadmium, fluoride, mercury, lead, nickel, selenium and hexavalent chromium.

F.1.1 Point Estimate Approach for ABS Derivation

An ABS is a chemical-dependent, scenario-dependent value that can vary with the characteristics of the soil matrix and the exposed population. Such characteristics include the relative lipophilicity/hydrophilicity of the compound, soil organic content, soil particle size, soil aging of the chemical, residence time on the skin, and exposed surface area. Some of these issues are discussed in greater detail in Chapter 6. The data necessary to characterize the variability in these variates are often not available. For this reason, the ABS values derived in this document are point estimates. In particular, site specific information on soil organic content and soil particle range are not available. These factors can have

a significant impact on chemical absorption from soil, and the uncertainty in the dose estimate from dermal absorption because of these and other factors can be large.

To derive a point estimate for a chemical, typically the value from the best and sometimes only study available was selected. If multiple studies were available with data collected under similar conditions, the most comprehensive study was selected. Or if the studies were of equal reliability, their absorption values would be averaged for ABS determination. In some cases experimental data are not sufficient for a point estimate ABS and a default ABS is recommended (see below).

F.1.2 Skin Morphology and Dermal Absorption Issues for ABS Determination

The transepidermal uptake of chemicals across skin involves a complex process of transport from the soil matrix to the external protective skin layer called the epidermis, and then through the epidermis to the underlying dermis. The outermost layer of the epidermis is called the stratum corneum, which is thought to provide the major barrier to the absorption of most substances deposited onto the skin surface. The stratum corneum in humans varies in thickness from about 5 µm to over 400 µm on the palms and soles of the feet (Poet and McDougal, 2002; Hostynek, 2003). Below lies the viable epidermis, about 50-100 µm thick, containing keratinocytes that proliferate and differentiate while moving upwards and replacing the stratum corneum cells as they wear away. Below the epidermis lies the hydrous tissue of the dermis perfused by the blood and lymphatic circulation.

Skin appendages, including hair follicles and sweat ducts, transit through all these layers and may provide an alternate pathway for dermal diffusion of some ions such as metal salts (Tregear, 1966; Flynn, 1990). However, skin appendages occupy only a fraction of the surface area of the skin, which may limit their potential as a major diffusion pathway into the systemic circulation.

During the transport through the viable-epidermal and dermal layers, metabolism may also play a role in the absorption process (Kao and Carver, 1990). Metabolism in the dermal layers could also activate a toxicant, resulting in skin as a target organ or producing toxicity elsewhere following systemic absorption. As noted above, specific dermal ABS values for soil-bound chemicals are difficult to obtain due in part to the complex multiphasic nature of the system and lack of published absorption data. Hawley (1985) suggested a default factor of 15 percent to correct for the effect of the soil matrix on the dermal uptake of organic chemicals. Experimental evidence, however, suggests absorption from soil will be chemical dependent. Hence, it is important to determine dermal uptake point estimate values for specific soil-bound chemicals where appropriate data are available, as they will be more accurate than those derived on broad-based assumptions.

To obtain the ABS, a measured amount of chemical in a given amount of soil is administered to the skin surface; this amount (wt chemical/area skin) is referred to as the applied dose. The amount of chemical that crosses the skin barrier is measured and the ABS is calculated by dividing the amount absorbed by the amount applied. When measurements are made in excreta or specific organs, corrections are included for incomplete recovery. In experiments of this type, the administered amount (in soil or solvent) represents a finite level of application. The ABS so calculated is an experimental value that is dependent upon exposure conditions, such as length of exposure and extent of soil loading. The length of exposure used for dermal exposure assessment in this document is 24 hrs. A 24 hr exposure time is commonly used in dermal absorption studies, so it's compatible for ABS calculation. In instances where absorption data did not use 24 hr exposure, an ABS will generally be based on data that are nearest to 24 hr exposure.

In contrast to the studies that utilize the application of finite amounts of chemicals, dermal studies that mimic scenarios such as bathing and swimming, require the applications of infinite volumes, i.e. the volume of the administered dose is much larger than the volume of the exposed skin area and the chemical at the skin surface is continuously replenished. The latter exposure scenario is not applicable to the soil studies described in this chapter, although information obtained from such studies may be useful for discussion purposes. For additional information on dermal uptake of chemicals from water (or vapor), the reader is referred to U.S. EPA (2004). The dermal absorption of chemicals from dermal exposure to contaminated water is not addressed in the "Hot Spots" program because it is likely to be a minor contribution to overall dose if it occurs at all.

F.2 Risk Assessment Issues

Although all dermal absorption studies are useful for understanding the relationship between dermal exposure and absorption, the application of these studies to risk assessment involves specific issues that must be considered to avoid development of a point estimate that may greatly underestimate, or overestimate, the potential for dermal absorption. Included among these issues are biological characteristics, soil properties, and exposure scenarios, and the variability in each can introduce uncertainties into the point estimate determination of ABS. By understanding these issues, the implications of using experimentally derived dermal ABS can be better understood. Specific categories of issues that must be considered when assessing dermal absorption are discussed below.

F.2.1 Definition of Dermal Uptake

Comprehensive dermal absorption studies often include a quantitative analysis of the amount of chemical that has passed through skin into the systemic circulation (for in vivo studies) or appears in the receptor fluid (for in vitro studies), plus the

amount of chemical remaining in the skin at the site of application. Fundamentally, dermal uptake/absorption refers to the amount of dermally applied chemical that is ultimately determined to be systemically available. Because absorbed chemicals may be retained in the skin for long periods of time and act as a reservoir for the slow systemic absorption of chemicals, the chemical remaining in skin at the end of dermal absorption experiments is considered available for systemic absorption unless data are available that shows otherwise.

Some fraction of dermally-absorbed chemicals may be only superficially diffused into skin and deposit in the stratum corneum where they are subject to counter-current forces of skin shedding, or desquamation, and ultimately removed from the body before becoming systemically absorbed. Continuous desquamation with total stratum corneum turnover has been estimated to take 2-3 weeks (Hostynek, 2003). Modeling calculations by Reddy et al. (2000) indicate that epidermal turnover can significantly reduce subsequent chemical absorption into the systemic circulation for highly lipophilic (log K_{ow} > about 4) or high molecular weight chemicals (MW > about 350-400 Da). However, some highly lipophilic chemicals retained in skin at the end of dermal absorption studies have been shown to be predominantly available for eventual absorption into the systemic circulation. Multipathway Hot Spots chemicals that fall into this category include the PAHs and DEHP (Chu et al., 1996).

Loss of absorbed chemical through skin shedding appears to occur more readily with some hydrophilic metal salts in which a portion of the metal becomes irreversibly bound in the epidermis and subject to eventual shedding with skin. Some metal salts have such a slow diffusion (i.e., long lag time) through skin that the stratum corneum turnover rate exceeds the chemical diffusion rate (Hostynek, 2003).

Tape stripping methods to remove thin layers of stratum corneum have been used in several studies discussed below to estimate the fraction of chemical in the stratum corneum that may be lost through desquamation. A more definitive approach used in a few cases is to extend the dermal uptake study for an additional few days (after excess chemical is removed from the skin surface) to determine if more of the chemical retained in the skin becomes available for systemic absorption. Other studies that help determine the fate of chemicals retained in skin include skin localization techniques and skin binding studies (Miselnicky et al., 1988; Yourick et al., 2004). But in many instances the dermal uptake studies for individual chemicals did not provide enough data to determine the fate or location of the chemical retained in skin. Thus, as discussed above, the ABS will then represent that fraction of chemical still retained in skin, plus the fraction that has already passed through the skin.

F.2.2 Dermal Bioavailability of Chemicals in Soil

The term dermal bioavailability as it applies in this section refers to the fraction of chemical in soil that is actually dermally absorbed. Dermal bioaccessibility is another term used in reference to chemical-laden soils and represents that fraction of chemical solubilized from soil, usually into water, sweat, or gastrointestinal fluids that then becomes available for absorption. By definition, bioaccessibility should exceed bioavailability.

Published data for some chemicals considered in this section contain only data for neat application of the chemical to skin in solvent or aqueous vehicle. Generally, there is a lack of absorption data for chemicals bound to soil. To avoid potential overestimation of absorption in these instances, bioaccessibility and soil leaching studies of soil-bound chemicals are considered for adjusting the fractional absorption of the pure chemical applied to skin. These studies can be used to determine the extractable, or bioaccessible, fraction of a soil pollutant that can be deposited on the skin surface. Water added to soil is often used to determine the bioaccessibility of a soil-bound chemical, although human sweat or synthetic sweat has also been used to estimate the amount of a pollutant that can be leached from contaminated soils (Horowitz and Finley, 1993; Filon et al., 2006; Nico et al., 2006).

F.2.3 Soil - Chemical - Tissue Interaction.

Soil is a complex matrix with a highly variable composition and absorptive capacity. Organic content, mineral composition, particle size, and pH are all highly variable. Because the dermal absorption of a compound from soil is often dependent on these characteristics, it follows that transfer of a chemical from soil particles to the skin surface for absorption is likely to vary with soil type.

Transfer of a chemical from soil particles to the skin surface is limited by the chemical's diffusion rate (McKone, 1990). Diffusion through the soil phase, through the air, and through soil moisture is all possible. Fugacity-based interphase transport models were constructed to describe the rate of each of these processes for chemicals in soil particles and to predict the dermal uptake rates. It was shown that predicted dermal uptake of chemicals from soil depends on the Henry's constant (vapor pressure/solubility in water), the octanol/water partition coefficient of a chemical, and the soil thickness on skin. If the Henry's constant is very high, chemicals will be lost from soil particles (or the skin surface) quite rapidly, so net dermal uptake of chemicals added to soil will be low. If the Henry's constant is very low, diffusion through the soil particle layer will be too slow to allow much dermal uptake unless the soil particles are very small. A high octanol/water partition coefficient is associated with tight binding to soil and low water solubility; these properties also limit the ability of a chemical to diffuse through the mixed lipid/water phases of the stratum corneum.

Other mathematical models have been developed by Bunge and Parks (1997) to describe dermal absorption of organic chemicals provided the chemical fits certain assumptions, such as falling within a defined octanol/water partition coefficient range ($1.59 \leq \log_{10}K_{ow} \leq 5.53$), and that the molecular weight of the organic chemical is ≤ 700. Soil constraints for the model include contaminated soils with about 0.2% organic carbon or more, and with a clay fraction less than 60 times the weight fraction of organic carbon. The models were then used to estimate the relative effect of changing exposure conditions (e.g., changes in soil loading, contamination levels, chemical, etc.) compared to published experimental studies. Although the models were generally consistent with the experimental results for some chemicals, such as benzo(a)pyrene (BaP), they were considerably divergent from the experimental results for other chemicals, such as lindane (gamma-hexachlorocyclohexane).

The authors suggested that the fast soil release kinetics on which the models are based may not fit with what was observed experimentally for some chemicals (Bunge and Parks, 1997). Fast soil release kinetics assumes the primary resistance that controls transfer of the chemical from soil to skin resides in the dermal barrier, and that the kinetics of soil desorption are relatively insignificant. Lindane may exhibit slow soil release characteristics in various soils (i.e., soil desorption of the chemical is the controlling influence for dermal absorption), which limits the amount of dermal absorption predicted by the models.

Alternatively, Shatkin et al. (2002) developed a two-stage fugacity-based model specifically for BaP that incorporated both a fast soil desorption phase and a slow desorption phase of BaP from soil. Based on the several parameters investigated that would affect dermal bioavailability, the authors predicted that the fast desorption kinetics of a soil had a greater impact on predicted dermal uptake than any other parameter, including organic carbon content of a soil.

These examples show that the effect of soil on the dermal uptake of organic compounds can be difficult to predict without experimental data. However, dermal absorption by metal salts can be expected to be a more complex process than dermal absorption of organic compounds. Factors affecting absorption of soil-bound metals include pH, metal oxidation state, counter ion, size and solubility (Hostynek, 2003). For example, lead becomes more soluble and available for uptake in soil at low pH. However, a low soil pH tends to convert chromium (VI) to the larger less permeable chromium (III) ion. This reduction in chromium valence can also occur in transit through the skin and considerably slow the absorption of chromium through skin.

F.2.4 Effect of Soil Organic Content on Dermal Absorption

For the soil pollutants discussed in this section, one of the most common soil variables explored for effect on dermal absorption of a chemical is the organic carbon or organic matter content. The chemical adsorbed to the organic carbon phase will generally be less available for transfer to skin than neat chemical

present in a separate liquid phase in the soil, largely due to strong adsorption of the chemical to the organic carbon fraction (Bunge and Parks, 1996). Dermal bioavailability of a chemical in soil also tends to decrease with increasing organic carbon content of the soil (Sheppard and Evenden, 1994; Bunge and Parks, 1997). Consequently, a number of studies compared the effect of varying the soil organic content on the dermal absorption of a chemical. The health protective approach for estimating an ABS would be to base the value on the higher dermal absorption from these studies, often from the soil with lower organic carbon content.

The length of time required for a chemical to partition to the soil organic material may be quite short (a few days) or longer (more than a month), depending on the nature of the deposited chemical, the soil and the weather (Bunge and Parks, 1996). However, early dermal absorption studies of chemicals in soil were usually conducted with freshly spiked soil just prior to exposure. Regardless of the partitioning time to the soil organic carbon, addition of a chemical to soil can often result in a reduction of dermal bioavailability relative to the pure chemical. For a group of selected organic compounds (e.g., DDT, BaP, PCBs, etc.) and arsenic, addition to soil just before loading onto skin reduced the overall dermal uptake by an average of about 60% compared to dermal uptake of the pure chemical (Wester and Maibach, 1999). However, a reduction in absorption from soil relative to a neat solution cannot be predicted for all chemicals. Dermal absorption for some chemicals such as arsenic in soil was found to be essentially unchanged compared to absorption from the neat solution.

F.2.5 Soil Aging Effects

The ABS point estimates presented here are primarily based on soils that were freshly spiked with contaminants and placed on skin for roughly 24 hrs. As such, the ABS point estimates largely represent the initial fast phase of decreased bioavailability when a chemical is freshly added to soil prior to skin exposure (Alexander, 1995; Bunge and Parks, 1997). This phase is generally a reversible process, such that a chemical sorbed to soil may become desorbed and be available for uptake during the skin exposure.

However, over time many chemicals added to soil undergo a slower second phase of decreased bioavailability. The soil-deposited chemicals tend to move from the external surface of soil particles to internal and more remote sites within the soil matrix so that chemicals become increasingly more desorption-resistant, a process known as aging (Alexander, 1995). A number of recent dermal absorption studies discussed below have observed reductions in dermal absorption occurring for up to 3-6 months following addition of the chemical to soil. Reductions of about 50% have been observed for dermal absorption of BaP aged in soil compared to soils freshly spiked prior to skin application (Roy and Singh, 2001). Abdel-Rahman et al. (1999) observed up to a 7.5-fold reduction in dermal absorption for arsenic aged in soil.

The continuous input of chemicals deposited on soils in the vicinity of "Hot Spots" stationary sources will likely result in the less recently deposited chemicals undergoing soil aging. For toxic inorganic metals in soil, the dermal dose equation (Eq. 6.1) does not account for decreased bioaccessibility over time due to soil aging. Leaching and weathering effects are assumed to be very long (i.e., 10^8 days), unless site-specific information shows otherwise. Only a few studies have investigated the decrease in dermal absorption for specific inorganic metals and semi-metals aged in soils, including arsenic, nickel and mercury. The soil aging results from these studies are considered in the development of the ABS, although the volume of literature available is sparse. Therefore, dermal fractional absorption still relies primarily on data for freshly applied metals to soil to avoid underestimation of the ABS.

For organic chemicals, the soil half-life variable in Eq. 6.2 will account to some degree for the effects of soil aging, depending on the rigor of the extraction process used (Abdel-Rahman et al., 2002). Use of a strong acid extraction method may solubilize some of the desorption-resistant chemical from soil and overestimate the dermal bioaccessibility of a soil-aged organic chemical. That is why milder extraction methods have been recommended, such as soil extraction in synthetic sweat, to obtain a more applicable estimate of soil half-life.

F.2.6 Dermal Soil Loading and Adherence Characteristics

The ABS from soil depends on the amount of soil in contact with the skin. Maximal fractional absorption of a soil-bound chemical occurs when a monolayer of soil covers the skin (monolayer threshold). A monolayer can be defined, in this case, as a layer of soil on the skin equal in thickness to the average soil particle diameter. Theoretical calculations and experimental data show that increased soil loading (mg soil/cm^2 skin) beyond monolayer coverage usually leads to decreased fractional absorption as a result of some of the soil not being in direct contact with skin (McKone, 1990; Duff and Kissel, 1996; Bunge and Parks, 1997). Soil loading at which the monolayer exists depends on the soil particle size (Duff and Kissel, 1996). For example, sand with an average particle diameter of 0.044 cm reaches monolayer coverage at 61 mg/cm^2, whereas monolayer coverage with clay at a particle diameter of 0.0092 cm is 13 mg/cm^2 (USEPA, 2004).

Early soil loading experiments were carried out under conditions of high loading, e.g. 20-40 mg/cm^2 (Shu et al., 1988; Wester et al., 1990a; Wester et al., 1992) , without estimating monolayer coverage or providing average soil particle diameter to estimate monolayer coverage. High soil loadings that are greater than monolayer coverage may underestimate the fraction of chemical absorbed from soil. Coarse grain size (180 to 300 µm) used under the high loading conditions of 20-40 mg/cm^2 was at, or only, slightly more than monolayer coverage (Duff and Kissel, 1996). However, using such soil loadings with soils sieved to <150 µm would result in greater than monolayer coverage.

Typical soil loadings under most human exposure scenarios generally ranged from 0.01 to 0.2 mg/cm^2 when averaged over the entire exposed skin surface (USEPA, 2004). Soil loadings on the hands, the skin region with the highest soil loading, averaged about 1 to 5 mg/cm^2 during typical human activities in wet soil with a moisture content of 9 to 18%, and usually less than 0.1 mg/cm^2 with activities in dry soil with a moisture content of 3-4% (Kissel et al., 1998).

During dermal absorption studies, the soil used to measure dermal uptake is applied to the skin as a "dry" formulation, i.e. the solvent used in the preparation of the chemical laden soil is allowed to evaporate prior to dermal application. The uptake of a soil-bound chemical from wet soil is expected to exceed the uptake from dry soil because of the increased humidity and temperature at the skin surface (Wester and Maibach, 1983). Such conditions exist for human exposure scenarios that involve high humidity, high temperature, and skin covering (e.g. gloves and clothing). Some studies are carried out under condition of occluded skin, and these studies could be used to estimate chemical absorption from soil when moisture is present.

In addition, the particle size distribution of soil adhering to skin also needs to be considered in dermal absorption studies. Most recent dermal absorption studies have sieved soil down to <150 µm prior to spiking with chemical and applying to skin. Studies have shown that soil particles in this size range tend to adhere to skin to the greatest extent (Driver et al., 1989; Sheppard and Evenden, 1994; Kissel et al., 1996). In hand press studies by Kissel et al. (1996), small particles $\leq$150 µm were found to adhere preferentially over larger particles $\geq$250 µm in dry soils of <2% moisture. Adherence in wet soils (12-18%) was roughly proportional to the soil particle size distribution of the original soil, although no consistent adherence was seen with soil moisture and particle size with five soils studied. Monolayer coverage with soil sieved to <150 µm will vary depending on the particle characteristics, but was shown in one instance to be about 2 mg/cm^2 with an estimated mean grain size of 12 µm (Duff and Kissel, 1996).

Choate et al. (2006) found that the dermally adhered fractions of two soil samples with wide distributions of particle sizes generally consisted of particles of diameters <63 µm or <125 µm, depending on the soil sampled. Adherence was similar whether the soils were applied dry (1.58-1.85% moisture) or moderately moist (3.35-3.81% moisture). With increasing moisture content of roughly 10% or greater, adherence increases significantly and a greater proportion of larger soil particles >150 µm are represented in the adhered soil (Holmes et al., 1996; Kissel et al., 1996; Choate et al., 2006). Smaller adhering soil particles can be considerably different in composition, especially in organic carbon content, from larger particles that tend to stick to skin in less abundance. However, organic carbon content does not appear to enhance the adherence of any particle sizes (Holmes et al., 1996; Choate et al., 2006).

In a few cases, no dermal absorption data were available for a chemical mixed with soil. Therefore, ABS values were estimated from studies that applied the

chemical directly onto the skin. Kissel (2011) observed that fractional absorption of chemicals applied neat to skin are not generally independent of skin loading conditions. For example, the ABS will decrease as an organic chemical is increasingly loaded onto skin. In other words, absorption of an organic chemical through skin is flux-limited, and loading more chemical onto skin in a defined area will not increase flux, but will decrease the ABS value.

To aid interpretation of dermal absorption-related phenomena, Kissel (2011) proposed a dimensionless variate representing the ratio of mass delivery to plausible absorptive flux under experimental or environmental conditions. High values of this dimensionless dermal variate connote surplus supply (i.e., flux-limited) conditions. This situation is similar to loading skin with chemical-bound soil above monolayer levels. The potential mismeasure of dermal absorption with chemicals applied neat to skin is addressed below for every chemical in which an ABS is derived in this way.

F.2.7 In Vivo Vs. In Vitro Experiments

It is generally recognized that the most reliable method for assessing skin absorption of a chemical is to measure penetration in vivo using the appropriate animal model or human volunteers (Kao, 1990). Thus, in vivo data are preferred over in vitro data for determination of a chemical ABS in this exposure assessment. In vivo data may be lacking for some chemicals of interest in this document due to economic considerations for conducting tests in humans and other mammalian species, or due to ethical concerns for testing in humans.

In vitro studies have the benefit of measuring dermal absorption under more easily controlled environments. Human skin can be tested without the inherent risks of a clinical study, and absorption through skin and retention in skin can be directly measured. Consequently, in vitro dermal absorption studies are frequently performed and provide the basis for an ABS for some chemicals presented in this section, following careful consideration for relevance to in vivo human exposure.

Although good agreement has been found when comparing in vivo and in vitro absorption results for some chemicals, trends towards lower absorption with in vitro exposure have been observed. For example, lipophilic compounds frequently have limited solubility in the buffered aqueous receptor fluids often used for in vitro cell systems, impeding the flow into the receptor fluid and resulting in an underestimation of skin penetration (Wester and Maibach, 1999). In vivo, lipophilic compounds penetrate the stratum corneum and diffuse through skin and, because of the solubilizing and emulsifying abilities of biological fluid, may readily be taken away by the blood in the dermal vasculature.

A reduction in skin viability of excised skin samples may occur due to storage conditions prior to use and may affect dermal absorption measurements. For example, the metabolic properties of human skin are reduced if the skin samples

were previously frozen. Some polycyclic aromatic compounds (PAHs) undergo extensive percutaneous metabolism when absorbed, and reducing the metabolic capabilities of skin samples will reduce dermal penetration of absorbed PAHs (Kao et al., 1985; Ng et al., 1992; Moody et al., 2009a).

For metal salts, it has been postulated that low diffusion values through the stratum corneum in vitro are a result of skin shunts (e.g., hair follicles and sweat ducts) swelling shut upon hydration of skin samples (Tregear, 1966; Hostynek, 2003). Skin shunts that bypass the stratum corneum are thought by some to be a significant absorption route for charged metals. For example, dermal absorption of nickel salts shows there is a surge in diffusion at the earliest stage, which then rapidly decreases towards steady state (Tanojo et al., 2001). The decrease in diffusion rate has been proposed to be a result of the skin tissue becoming hydrated, shutting down the skin shunts.

A further potential limitation under in vitro conditions is that diffusing compounds must traverse the epidermis and the entire dermis in order to reach the receptor fluid. In vivo, the majority of the absorption into the cutaneous microcirculation is thought to occur in the upper dermis and the penetrant compounds may not have to diffuse across the entire thickness of the dermis. However, the bulk of the connective tissue in the dermis is often eliminated from the skin preparation by cutting the skin parallel to the skin surface with a dermatome (Poet and McDougal, 2002).

In vivo studies are not without limitations. Dermally applied chemicals are often radiolabeled to facilitate quantification of the usually low absolute amounts of chemical dermally absorbed. In small mammals, a total accounting of all dermally absorbed radioactivity can be estimated from excreta, carcass, and site of skin absorption. However, in larger mammals measurements of radiotracer are quantified in excreta and measurements from intravenous, intramuscular, or oral dosing are applied as a correction for tissue absorbed chemical. The validity of this method depends on the underlying assumption that metabolism and disposition of the applied compound is route independent, and that the pharmacokinetic behavior of the intravenous and topical doses is similar (Kao, 1990).

F.2.8 Inter- and Intra-Species Specificity

The variability in dermal absorption of chemicals among mammalian species has been investigated in vivo and in vitro. Bartek et al. (1972) suggest that the extent of in vivo uptake among animals follows the rank: rabbit > rat > pig ≈ monkey ≈ humans, based on dermal absorption of benzoic acid, hydrocortisone, testosterone, caffeine, N-acetylcysteine, and butter yellow. However, the species ranking did not strictly hold for all chemicals, indicating not only species-specific differences but also chemical-specific differences.

Comparison of data from other studies does support that in general, the absorption in the rabbit, rat and other rodents can considerably overestimate absorption in humans, while absorption in monkeys and miniature pigs most closely predict human absorption (Wester and Maibach, 1975; Reifenrath et al., 1984; Wester and Maibach, 1985; Bronaugh et al., 1990; Wester et al., 1998a). Alternatively, Kao et al. (1985) found that in vitro permeation of testosterone and BaP through human skin was greater than that for guinea pig, rat, or rabbit, indicating that species-specificity differences likely depend on other factors such as experimental conditions and tissue viability. Variability in dermal absorption depending on the skin area exposed has been investigated (Wester and Maibach, 1983). In humans, absorption across the skin varies by area of the body and may be higher than the commonly used forearm (e.g. scalp, axilla, forehead, jaw angle and scrotum).

F.2.9 Metabolism of Absorbed Chemicals in the Skin

The description of percutaneous absorption is generally based on diffusion models that take into account the physico-chemical characteristics of chemicals and soils. While such descriptions may help to explain the uptake of chemicals across the stratum corneum, the role played by metabolism in the viable epidermal and dermal layers should be included to understand the complete permeation of chemicals through the skin (Wester and Maibach, 1983; Kao and Carver, 1990; Bronaugh et al., 1994).

Viability of the skin refers to the status of active energy turnover, i.e. the utilization of glucose and formation of CO_2 or lactate in skin. Enzymes and metabolic processes in skin may affect the dermal penetration of drugs and other xenobiotics, particularly if absorbed chemicals can be metabolized in the skin. Using production of lactose as the measure of viability, human skin placed in a buffered solution and kept refrigerated remained viable for about 8 days following donor death (Wester et al., 1998b). Skin frozen for storage or heat-treated to separate the epidermis and dermis renders the skin non-viable and may change the dermal penetration dynamics of absorbed chemicals. Some early studies investigating the dermal penetration of chemicals used previously frozen skin samples and may not provide a good basis for ABS determination.

Dermal metabolism of BaP was observed to be considerably reduced in several mammalian species with use of non-viable skin, resulting in reduced penetration of BaP through skin (Kao et al., 1985). In viable human skin, nearly half the BaP that permeated the skin was attributed to BaP metabolites. In non-viable skin, essentially only unchanged BaP was detected in the receptor fluid. In fact, dermal absorption of polycyclic aromatic hydrocarbons (PAH) that include BaP resulted in PAH-DNA adducts in human skin samples, demonstrating that skin is a target organ due to metabolic activation of PAHs in skin (Phillips et al., 1990).

On the other hand, dermal absorption of some chemicals does not appear to be affected by the viability status of the skin samples. Dermal penetration of TCDD through viable and non-viable pig skin was found to be similar (Weber, 1993).

F.2.10 Human Adult and Infant Variability in Skin Permeability

Animal studies are designed to ensure uniformity within the experimental population by using inbred strains and often only one sex. The variability between animals is much less than the genetically diverse human population. Human studies also rarely use children or infants, the elderly, pregnant women and the infirm, partially because of ethical considerations. Dermal uptake may vary due to genetic diversity in the human population and differences in age. This variability will not necessarily be accounted for by experimental data.

A review of the data on human skin permeability to chemicals suggest at least a mean intra-individual coefficient of variation of approximately 40% and a mean inter-individual variation of about 70% (Loth et al., 2000; Hostynek, 2003). A leading cause in the variation is the lipid composition of the stratum corneum, which influences solubility and permeability of drugs. This factor is partly responsible for the high variability in accumulation and permeation measurements (Loth et al., 2000).

There has been increasing awareness in recent years that infants and children are more susceptible than adults to the harmful effects of some pollutants. This can be due to differences in exposure, physiology, absorption, distribution, metabolism, and excretion. Further, organ development and faster cell division influence targets of toxicity. Finally, a large skin surface area to body weight ratio would increase the dose of an absorbed chemical on a mg/kg body weight basis.

Only a few studies have examined age-related differences in the dermal absorption capacity of chemicals in infants and children compared to adults. Preterm infants lack a fully developed dermal barrier function and are particularly prone to accidental poisoning of toxic agents applied to the skin surface (Barrett and Rutter, 1994). In an in vitro system, McCormack et al. (1982) observed increased penetration of some alcohols and fatty acids through skin of premature infants compared to full term infant skin and adult skin. Dermal absorption of sodium salicylate was found to be a hundred- to a thousand-fold greater in infants of 30 weeks gestation or less compared to full term infants (Barker et al., 1987).

In full-term infants, epidermal structure and function matures by 2-3 weeks of age (Holbrook, 1998; Makri et al., 2004). In general, the in vitro system of McCormack et al. (1982) showed full-term baby skin to be a good barrier for some compounds. No difference in penetration of alcohols through full term infant and adult skin was seen. However, penetration of some fatty acids through full term infant skin was greater than that through adult skin. Higher lipid content in the stratum corneum of infants was thought to be the reason for

increased absorption of fatty acids. In addition, a layer of subcutaneous fat develops at approximately 2-3 months of age in infants and continues to exist through the early toddler period (Thompson, 1946; Banks et al., 1990; Cohen Hubal et al., 2000). This layer of fat may act as a sink for lipophilic chemicals absorbed through the skin.

Age-related changes in dermal absorption have also been investigated in experimental animal models. Using TCDD or 2,3,4,7,8-pentachlorodibenzo-p-dioxin (4-PeCDD) in solvent, Banks et al. (1990) observed greater absorption of TCDD or 4-PeCDD in 10-week old rats than 36 - 120-week old rats. 2,4,5,2',4',5'-Hexachlorobiphenyl showed significantly higher fractional penetration in young rats (33 days old) compared to adult rats (82 days old) in vivo, but only at one of three dose levels tested (Shah et al., 1987). Overall, the authors concluded that no clear age-related pattern of dermal absorption was found among a total of 14 pesticides including 2,4,5,2',4',5'-hexachlorobiphenyl.

F.2.11 Use of Default ABS Values

The California South Coast Air Quality Management District's Multi-Pathway Health Risk Assessment Input Parameters Guidance Document (SCAQMD, 1988) recommended using default values of 10% for organic chemicals and 1% for inorganic chemicals when quantitative data are not available to estimate chemical-specific dermal absorption fractions from soil.

Use of these default factors was proposed based on a review of the dermal absorption literature and recommendations by McLaughlin (1984). In his US EPA report, McLaughlin suggests it may be possible to group penetrants into a numerical system using an "order of magnitude" approach (i.e., 100% - 10% - 1% - 0.1% fractional absorption groupings), depending on physical parameters such as partition coefficients and diffusion constants. For example, many of the organic compounds were found to fall into the 10% absorption range. Exceptions included some pesticides, such as the very lipophilic pesticide carbaryl that exhibited a fractional absorption closer to 100%, and the polar pesticide diquat that exhibited a fractional absorption closer to 1%.

More recently, US EPA (2004) also recommended a default dermal absorption fraction for semivolatile organic compounds (SVOCs) of 10% as a screening method for the majority of SVOCs without dermal absorption values. This fraction was suggested because the experimental values for SVOCs determined by US EPA are assumed to be representative of all SVOCs as a class. US EPA (2004) notes that chemicals within classes can vary widely in structure and chemical properties, potentially resulting in a wide range of fractional absorption values. However, OEHHA agrees that a 10% fractional absorption default value is acceptable at this time, based on the range of values (3 to 14%) estimated in Table F.5 for SVOCs. Currently, the OEHHA default ABS value for organic compounds applies only to 4,4'-methylene dianiline.

For inorganic classes of compounds, US EPA (2004) recommended that no default dermal absorption values be used. The premise was that speciation of inorganic compounds is critical to the dermal absorption and there are too little data to extrapolate a reasonable default value. OEHHA notes that the range of ABS point estimate values for the metal and semi-metal salts (see Table F.5) is between 0.2 and 6%. Therefore, it is reasonable to assume that a default ABS of 3% can be used as a screening value, based on the mean ABS value for the metals and semi-metals in which published dermal absorption data exists (i.e., arsenic, cadmium, hexavalent chromium, lead, mercury and nickel). Currently, the default ABS value for inorganic compounds applies only to fluoride, beryllium and selenium.

F.3 Point Estimates for Dermal Absorption (ABS) of Inorganic Compounds

F.3.1 Arsenic and Arsenic Compounds

Recommended point estimate for dermal uptake: 6%

F.3.1.1 Studies Considered

A. Key Studies

Wester et al. (1993a) examined the in vivo percutaneous absorption of radiolabeled soluble arsenic (as $H_3{}^{73}AsO_4$) freshly mixed with soil and applied to skin of female Rhesus monkeys (n = 4 animals per dose group). Dose levels of 0.0004 and 0.6 $\mu g/cm^2$ were used. The soil load on the skin was 40 mg soil/cm^2 skin area. The soil had been sieved to 180-300 µm prior to application, thus, a soil load of 40 mg/cm^2 was likely at or near monolayer coverage. Topical doses were applied to an area of the abdomen for 24 hours. Urine was collected during the dosing period, and through the following 6 days. For comparison, radiolabeled arsenic (as ^{73}As) in water was administered intravenously to four monkeys. Percutaneous absorption was determined by the ratio of urinary arsenic excretion following topical application to that following intravenous administration.

Urinary excretion of the ^{73}As label was complete by day 7, with about half the label excreted in the first 24-48 hrs following topical administration. Results of this study showed that the percutaneous absorption of arsenic from soil was 4.5 $\pm$ 3.2% from the low dose and 3.2 $\pm$ 1.9% from the high dose (nonsignificant difference). An estimate of arsenic retained in the skin was not performed, although 27-28% of the arsenic could not be accounted for following decontamination of the skin.

Lowney et al., (2005) conducted follow-up absorption studies with arsenic aged in soil that paralleled the methodology used in the in vivo Rhesus monkey study. The soil samples collected were adjacent to a pesticide production facility that

had historically produced calcium and lead arsenate compounds. The arsenic was resident in the soil for a minimum of 30 years and was primarily in the sparingly soluble iron oxide and iron silicate mineral phases. Small amounts of more soluble calcium arsenate and arsenic trioxide were also detected in the soil. The particle size fraction was sieved to <150 µm and a skin loading of 4 mg/cm^2 on 100 cm^2 of skin was applied. Total dose was 560 µg arsenic and the duration of dermal exposure was 8 hrs on the abdomens of three monkeys. Following fractional correction of arsenic from i.v. dose, urinary excretion of arsenic ranged from 0.01 to 0.24% of the dermally applied dose, but was not statistically greater than background. Negligible absorption was considered to be due to the presence of soil arsenic primarily in sparingly soluble mineral phases. Direct or indirect estimates of arsenic retained in the skin were not performed.

A sweat extraction technique by Nico et al. (2006) was employed to estimate the soluble arsenic that can be made bioavailable for dermal absorption from the aged arsenic soil used in the in vivo monkey study by Lowney et al. (2005). Sweat extraction of this soil resulted in only 1.8% soluble arsenic. However, a second aged soil sample from a different arsenic-contaminated site resulted in 11% arsenic extracted by sweat. Nico et al. (2006) also used the sweat extraction technique to estimate soluble arsenic from soil samples freshly spiked with arsenic. One sample was sieved to <150 µm while another was sieved to 180-300 µm, similar to that used by Wester et al. (1993a) in the in vivo dermal monkey study. Sweat extraction resulted in 45 and 72% soluble arsenic from the <150 and 180-300 µm soil samples, respectively.

B. Supporting Studies

In addition to the monkey in vivo study, Wester et al., (1993a) conducted an in vitro study using human cadaver skin from three separate donor sources with three replicates from each source. The skin was dermatomed to 500 µm, stored refrigerated in Eagle's medium and used within 5 days to preserve skin viability, although elapsed time from death to harvest of skin was not specified. A dose of 0.0004 µg arsenic per cm^2 skin surface exposed was applied. The soil load on the skin samples was 40 mg soil per cm^2 skin area, and phosphate-buffered saline served as receptor fluid. The in vitro exposure period was 24 hours. As performed in the monkey in vivo study, the soil had been sieved to 180-300 µm prior to application, so monolayer coverage was probably not surpassed. Percutaneous absorption through human cadaver skin was 0.76% (0.43% in receptor fluid; 0.33% in skin) after soap and water wash. While the authors did not speculate as to the reduced in vitro dermal absorption compared to monkey in vivo absorption, Kao (1990) noted that both elapsed time from death to harvest of tissues and treatments and storage of the cadaver could have resulted in a large variability in skin permeability.

Dermal absorption of radiolabeled soluble arsenic (as $H_3{}^{73}AsO_4$) freshly applied or aged in two different soils was determined in vitro through dermatomed pig skin cut 200 µm thick (Abdel-Rahman et al., 1996; Abdel-Rahman et al., 1999).

Soil types included a sandy soil with 4.4% organic matter and a clay soil with 1.6% organic matter, with no apparent sieving before application. Arsenic was applied to skin for 16 hrs either alone in ethanol vehicle, immediately after the addition of 30 mg of the soils to skin, or after aging for 3 months in each soil. Soil loading was calculated to be about 47 mg/cm^2. Applying soil to skin and then applying the arsenic does not allow time for arsenic-soil equilibrium. This method of application allows for direct contact of skin with arsenic or vehicle and not from soil, leading to an overestimation of the fractional absorption (Spalt et al., 2009). In addition, monolayer coverage was probably exceeded with a soil loading of 47 mg/cm^2.

With arsenic freshly added to soil, 0.2% of the arsenic penetrated the skin to receptor fluid from both soil types (Abdel-Rahman et al., 1996; Abdel-Rahman et al., 1999). Total dermal absorption including arsenic retained in skin was 10.0 and 6.0% from the sandy and clay soils, respectively. In comparison, pure arsenic found in receptor fluid and retained in skin was 0.4 and 44.2%, respectively. In aged sandy and clay soil, 0.2 and 0.1% arsenic was found in the receptor fluid, respectively. Total dermal absorption in the aged soils was 1.5 and 0.8% from sandy and clay soils, respectively.

Radiolabeled sodium arsenate was applied in vitro to the skin of mice for 24 hrs as a solid compound, in an aqueous solution, or as an aqueous solution in sandy soil (Rahman et al., 1994). Soil was sieved to $\leq$180 µm and contained 58% sand, 34% silt, 8% clay and 1.4% organic matter. Arsenate was freshly applied to soil prior to skin application, with an average soil loading on the skin of 23 mg/cm^2. Absorption increased linearly with the applied dose from all exposure vehicles, with a constant fraction of the dose being absorbed. Total arsenate absorption was as high as 62% of applied dose from 100 µl water vehicle and about 33% of applied dose as the solid. However, absorption of arsenate from soil was less than 0.3% of applied dose, with about one-third penetrating to the receptor fluid.

A dermal exposure study was conducted to assess the potential for arsenic exposure in children in contact with playground equipment and decks treated with the wood preservative chromated copper-arsenate (CCA) (Wester et al., 2004). Methodology was similar to that used by Wester et al. (1993a) in three monkeys to assess dermal arsenic absorption from CCA-treated wood residues. Following 8-hr dermal application, an increase in urinary excretion of arsenic above background was not detectable, indicating virtually no absorption of arsenic from CCA-treated wood residue. The researchers determined that the absorbed dose would need to be in the range of 0.10 to 0.16% of the applied dose to be detectable above background.

The negligible dermal absorption of arsenic from the CCA residues is a result of arsenic chemically bound with other metals (particularly chromium) and ultimately to the wood structure (Nico et al., 2004). The leaching characteristics of soluble arsenic in CCA residues were also investigated by extraction in human sweat

(Nico et al., 2006). The sweat extraction procedure indicated that up to 12% of total arsenic is available for dermal absorption from CCA-treated wood residue. However, only 1.4% soluble arsenic was extracted with sweat from CCA-residue aged in soil near a CCA-treated utility pole. Gastric leaching conditions resulted in up to 2-3 times greater solubilization of arsenic from CCA-treated wood compared to sweat leaching, indicating soil ingestion of CCA-released arsenic can be a health concern.

F.3.1.2 Discussion and Recommendation for Arsenic and Arsenic Compounds ABS

Dermal exposure of skin to arsenic resulting in passage of arsenic through skin to the bloodstream is the primary concern under the “Hot Spots” program. However, arsenic that becomes bound in skin may also have toxicological consequences. Regardless of route of exposure to arsenic, the skin is a critical target organ for arsenic toxicity due to local absorption and binding of sulfhydryl-group-containing proteins (Hostynek et al., 1993). The affinity for sulfhydryl groups leads to arsenic’s accumulation and tenacious retention in keratin-rich tissues such as hair, nails, and skin. Measurement of in vitro percutaneous absorption of As(III) and As(V) by human epidermal skin cultures for 6 hrs shows strong affinity of arsenic for the keratinocytes, with an estimated 30% of As(V) passing through skin being retained compared to over 90% of the As(III) being retained (Bernstam et al., 2002).

Accumulation of arsenic in the skin is characterized by hyperpigmentation, keratoses of the palms of the hands and soles of the feet, and diffuse macular pigmentation or diffuse darkening of the skin on the limbs and trunk, attributed to the reduction and deposition of the element in the metallic state (Hostynek, 2003). Chronic arsenic accumulation in skin increases the susceptibility of the skin to ultraviolet light and is associated with an increased incidence of tumors of exposed skin, although skin cancer is primarily a result of oral arsenical poisoning and characterized by multifocal lesions over the entire body (Hostynek et al., 1993; OEHHA, 1999).

The key in vivo monkey study by Wester et al. (1993a) provides an average fractional absorption of 3.9% based on two dose levels of arsenic that had been freshly added to soil before application to skin. Some limitations are noted for this study. First, the in vivo study did not estimate arsenic retained in skin. However, the researchers followed excretion of arsenic after exposure and noted that excretion of the labeled arsenic was essentially over by day 7. The remaining arsenic bound to skin proteins will probably remain there and not present a risk of reaching the bloodstream.

Secondly, a sieved soil fraction of 180-300 µm was used, which does not reflect the generally smaller soil particle fraction that sticks to skin following dermal contact. Soil sieved to <150 µm is considered more relevant for dermal studies (Spalt et al., 2009). The sieved soil used by Wester et al. may underestimate

fractional absorption. This assumption is supported by the sweat extraction study by Nico et al. (2006), which found a 63% increase in arsenic bioavailability (45% to 72%) from soil sieved to <150 µm as opposed soil sieved to 180-300 µm.

Finally, there is also some question whether the contaminated soil had continuous contact with the skin of the monkeys (Spalt et al., 2009). From the methodology description, the eye patches used to hold the soil in place on the abdomen of the monkeys were a larger volume than the applied soil. Thus, sloughing of soil off the skin probably occurred when the monkeys sat upright.

Together, these limitations indicate that basing an ABS on the monkey study may underestimate the dermal fractional absorption of arsenic. However, the sweat extraction study by Nico et al. (2006) supports the application of an adjustment to account for use of a soil fraction that likely underestimates fractional absorption. A 63% increase in arsenic bioavailability was observed from soil sieved to <150 µm, compared to soil sieved to 180-300 µm, as used by Wester et al. (1993a). A soil sieved to <150 µm better characterizes the soil particle size that adheres to skin. Thus, a 63% increase was applied to the monkey fraction absorption value of 3.9% resulting in an arsenic ABS of 6% when rounded to the nearest whole number.

The in vitro studies reviewed here gave a range of 0.3 to 10% for total absorption following application of freshly spiked soil to skin samples (Rahman et al., 1994; Abdel-Rahman et al., 1996; Abdel-Rahman et al., 1999; Wester et al., 1993a). However, arsenic aged in two soils gave a total dermal absorption of 0.8-1.5% in pig skin in vitro (Abdel-Rahman et al., 1996). As discussed above, it is difficult to reconcile the difference in dermal absorption in pig skin between arsenic freshly spiked in soil and arsenic aged soil due to differences in methodology. Future in vitro studies using human skin and arsenic freshly applied and aged in soils would help assess the impact of arsenic aged in soil.

F.3.2 Beryllium and Beryllium Compounds

Recommended use of default inorganic compound ABS estimate of 3.0%.

F.3.2.1 Studies Considered

No quantitative data could be found regarding the fractional dermal absorption or skin penetration of beryllium (Be) compounds. Be metal powder can oxidize when suspended in synthetic sweat, whereupon the metallic ions may be absorbed in human skin (Larese et al., 2007). However, Be salts are corrosive to skin, and have a high reactivity with protein substrates that result in strong retention in skin (Hostynek et al., 1993). The reaction of beryllium salts with the proteins in skin acts as a strong sensitizer that cause allergic contact dermatitis. Beryllium compounds typically decompose to form the poorly soluble, amorphous oxide (BeO) or hydroxide ($Be(OH)_2$), resulting in tissue granulomas (i.e., compactly grouped cells that replace normally functioning tissue) and ulcers.

Once lodged in tissue, these amorphous beryllium precipitates are excreted at a very slow rate.

Belman (1969) investigated the interaction of beryllium fluoride and beryllium sulfate with guinea pig epidermal tissue in order to explore a mechanism for the delayed allergic skin reaction observed in humans following beryllium exposure. Using both in vitro and in vivo experiments, he reported that beryllium is taken up into the skin and localized primarily to proteins of the epidermis, with little or no apparent binding to stratum corneum or dermis. Exposure caused a localized immune response and rapid destruction of skin cells. Data are not provided, however, regarding the amount of beryllium taken up by the skin cells, or the fate of beryllium following the immunological response (i.e., whether beryllium is then absorbed into the circulation, or sloughed off with cells.)

Petzow and Zorn (1974) reported on the absorption of beryllium through the tail skin of rats exposed to an aqueous beryllium chloride solution spiked with ^{7}Be. The authors stated that within the first hour of exposure there is an increase in the rate of beryllium uptake. After approximately 90 minutes, the dermal flux of beryllium from the aqueous solution is constant. In addition, Petzow and Zorn reported that the amount of beryllium that diffuses through the skin seems to be dependent upon the concentration of beryllium in contact with the skin.

Worker exposure and likely facility emissions of beryllium compounds are mostly in the form of particulates, primarily BeO (Tinkle et al., 2003; Day et al., 2006). For these poorly soluble beryllium particles, dermal exposure is considered to be of toxicological significance. Chronic beryllium disease (CBD) is an occupational disease that begins as a cell-mediated immune response to inhaled beryllium. Although respiratory and engineering controls have significantly decreased occupational inhalation exposures, reduction in occurrence of beryllium sensitization and CBD has not significantly decreased. The lack of worker skin protection has been postulated as a contributor to the persistence of sensitization and CBD in the workplace.

The concentration of antigen required for elicitation of a cell-mediated immune response is significantly smaller than the concentration required for sensitization, therefore, the failure of respiratory exposure limits to lower the rate of disease is likely related to the continued unchecked skin exposure to beryllium particles (Tinkle et al., 2003; Day et al., 2006; Deubner and Kent, 2007). Thus, in workers with significant beryllium skin exposure, the pulmonary exposure required to elicit a subsequent immune response and granuloma formation would be significantly smaller.

To determine if BeO can penetrate the stratum corneum and reach the immunologically active epidermis, Tinkle et al. (2003) conducted a pilot study in which BeO particles were suspended in petrolatum (1 mg/g), painted on the back of shaved mice, and the area covered with surgical tape. The average amount of beryllium applied to each mouse was 70 µg. Excess BeO was removed from the

surface of the flank skin by gentle washing and tape stripping three times immediately following 24-hr exposure. On day 7 or 14 following the exposure, the amount of beryllium in the flank skin of BeO-treated mice was, on average, 1.2 µg/g tissue, thus confirming that BeO is present in the skin.

Additionally, Tinkle et al. (2003) observed in vitro that polystyrene latex spheres <1 µm in diameter, when applied to skin and coupled with flexing motion, can penetrate intact human skin. The researchers proposed that beryllium particles can similarly penetrate the skin.

F.3.2.2 Discussion and Recommendation for the Beryllium and Beryllium Compound ABS

Due to the lack of quantitative data regarding dermal absorption of beryllium, it is not possible to calculate a chemical-specific fractional absorption value for Be salts. The high reactivity of beryllium with skin suggests penetration to the bloodstream in intact skin is small relative to other inorganic metals discussed in this section. However, it is postulated that a primary concern for dermal exposure to beryllium is related to sensitization, which results in much lower inhaled concentrations of beryllium particles required for elicitation of a cell-mediated immune response leading to progression of CBD (Tinkle et al., 2003; Day et al., 2006). This action only requires penetration to the epidermis where the immune response occurs. Considering that full dermal penetration of beryllium to the bloodstream may not be required to enhance or facilitate a toxicological response, and that particles have been shown to penetrate the skin with flexing, it is recommended that an ABS of 3%, based on the mean ABS for the other Hot Spot metals (Cd, Cr(VI), Pb, Hg, Ni) and semi-metals (As), be used for beryllium for screening purposes to assess dermal exposure.

F.3.3 Cadmium and Cadmium Compounds

Recommended point estimate for dermal uptake: 0.2%

F.3.3.1 Studies Considered

A. Key Studies

Wester et al. (1992) examined the percutaneous absorption of cadmium chloride from soil using human cadaver skin in an in vitro system. Donor skin was used within 5 days of harvest and was kept refrigerated in buffered medium until then. The soil used prior to sieving contained 26% sand, 26% clay, 48% silt and 0.9% organic carbon. The soil was sieved to retain particles in the range of 180 to 300 µm. Radiolabeled cadmium (^{109}Cd) was mixed with soil at a concentration of 13 ppb and applied to the skin samples at a soil loading of 20 mg/cm^2 or 40 mg/cm^2. Two donor skin sources were used with replicates for each of the soil concentrations. Human plasma was used as the receptor fluid. At the end of a 16-hour exposure, soil was removed from the samples by soap and water rinse. Percutaneous absorption, calculated as receptor fluid accumulation plus residual

skin concentration after soap and water wash, ranged from 0.08% to 0.2% of applied dose (Table F.1). No significant differences were observed in absorption between skin samples or soil load concentrations.

Table F.1. In Vitro Human Dermal Fractional Absorption of Cadmium Chloride from Soil[a]

Soil Loading	Skin Source	Percentage Applied Dose		
		Receptor Fluid	Skin	Total
40 mg/cm^2	1	0.02 ± 0.01	0.06 ± 0.02	0.08
	2	0.07 ± 0.03	0.13 ± 0.05	0.20
20 mg/cm^2	3	0.02 ± 0.02	0.08 ± 0.06	0.1
	4	0.02 ± 0.02	0.08 ± 0.06	0.1

[a] Data from Wester et al. (1992); n = 3 replicates per skin source

In another experiment, Wester et al. (1992) applied cadmium in water to human skin samples for 30 min, followed by removal of the cadmium solution from the skin surface and continued perfusion of the skin for an additional 48 hrs. No cadmium appeared in the receptor fluid after 30 min of exposure. However, 0.6 ± 0.8% of the dose had diffused into the receptor fluid after 48 hrs demonstrating the capacity of cadmium to be retained in the skin and be slowly systemically absorbed over time.

B. Supporting Studies

Kimura and Otaki (1972) used liver and kidney accumulation of cadmium in rabbits and hairless mice to estimate dermal absorption. A total dose of 30.5 mg Cd (in an aqueous $CdCl_2$ solution) was administered to rabbit skin (n=1) in 5 doses over 3 weeks. Two weeks after the final application, 0.40% of the applied dose was found in liver and kidney combined. In rabbits (n=2), a total dose of 61 mg Cd was administered in multiple cream-like and milk-like ointment applications, resulting in 0.45 and 0.61%of the applied dose, respectively, in liver and kidney combined. The type of ointment vehicle used did not appear to greatly affect the absorption or accumulation characteristics of Cd. Dermal absorption of cadmium in hairless mice, estimated from kidney and liver accumulation, ranged from 0.07-0.27% after a single application of ointment (0.61 mg Cd). Cadmium absorption after multiple ointment applications on hairless mice ranged from 0.59 - 0.87% of applied dose.

Aqueous 1.0, 0.1 and 0.01% cadmium solutions were painted onto the skin of mice and rats and air dried each day for ten days (Lansdown and Sampson, 1996). Perceptible skin damage occurred at the two highest doses, likely

resulting in increased dermal absorption. At the lowest dose, significantly increased skin content of cadmium was observed in both mice (138 ng Cd/g) and rats (248 ng Cd/g). Adequate data to estimate fractional absorption were not provided.

Although no studies estimated dermal absorption of cadmium aged in soils, Aringhieri et al. (1985) reported that 80% of cadmium added to a soil containing high organic matter (14.2%) and high clay content (60%) was adsorbed to soil particles within 10 min of addition to a soil. Tang et al. (2006) observed that bioaccessibility of cadmium (relating closely to absorption following ingestion of soil) in strongly acidic soils spiked with cadmium reached nearly steady state levels as high as 77% after the first week of aging. In soils highly contaminated with heavy metals by industrial sources, the $MgCl_2$-exchangeable fraction of cadmium was about 37% and was considered the most mobile and biologically available heavy metal in the samples examined (Hickey and Kittrick, 1984).

F.3.3.2 Discussion and Recommendation for a Cadmium and Cadmium Compounds ABS

No in vivo studies investigating fractional absorption of cadmium from soil were located. The human in vitro study by Wester et al. (1992) provided the only quantitative data for dermal absorption of cadmium from soil. The retention and concentrating of cadmium in skin with slow systemic absorption demonstrate the necessity for including the cadmium found in exposed skin for estimating an ABS point estimate.

The lack of quantitative in vivo studies and the use of 16 hr rather than 24 hr exposures support a point estimate based on the highest fractional absorption of 0.2%, rather than a the lower estimate of 0.1% (based on an averaging of different skin sources for each of the two soil loadings). In addition, coarse particle soil loadings of 20 and 40 mg/cm^2 may result in a reduced fractional absorption, although the data suggest monolayer coverage of skin was probably not exceeded (Spalt et al., 2009). The high bioavailability and apparent low capacity for aging of cadmium in some soils indicates that sequestration of cadmium in soil will be small relative to other inorganic metals in soil.

F.3.4 Soluble Compounds of Hexavalent Chromium

Recommended point estimate for dermal uptake: 2%

F.3.4.1 Studies Considered

A. Key Study

Czernielewski et al. (1965) exposed guinea pigs to hexavalent chromium (chromium (VI)) as sodium chromate solution labeled with Cr^{51}. A single dose (15 μg sodium chromate in 0.1 ml solution) was applied to a 4 cm^2 shaved area of skin for 24 hours (n=9 animals). Absorption was estimated by measurement of

the Cr^{51} content of the following: urine, feces, blood (1 ml), heart, liver, spleen, adrenals, kidneys, lungs, lymphatics, and skin. Dermal absorption of chromium (VI) was estimated to be 2.9% of the applied dose from the 24 hour exposure. Based on the average blood volume of adult guinea pigs (27 ml), 1.6% of applied dose was found in blood, 1.1% in excreta, and only 0.2% in organs and tissues including skin.

B. Supporting Studies

Chromium in the hexavalent [Cr(VI)] state does not measurably bind with proteins, whereas the trivalent chromic ion [Cr(III)] shows strong affinity for protein in epithelial and dermal tissues (Samitz et al., 1969; Gammelgaard et al., 1992). Thus, Cr(VI) can permeate through skin relatively easily compared to Cr(III). However, skin has the capacity, though limited, to reduce Cr(VI) to Cr(III) resulting in binding of chromium to skin protein and decreasing the rate of diffusion (Gammelgaard et al., 1992; Hostynek, 2003). Binding of chromium in the skin is characterized as irreversible, leading to protein denaturation with formation of permanent depots in the epidermis (Hostynek, 2003). Some of the bound chromium is likely subject to the counter-current effect of continuous sloughing of the outer skin layers, although no studies have attempted to quantify this removal pathway.

To investigate the level of penetration of Cr(VI) into human skin, Liden and Lundberg (1979) cut 10 µm tangential sections of skin biopsies after application of a 0.5% aqueous potassium chromate solution on a 79 mm^2 patch of skin on the back of volunteers. Dermal exposure durations to the chromate were 5, 24, or 72 hrs. Highest chromium levels were found in stratum corneum. Chromium was also found at the dermal-epidermal junction and the upper mid-dermis. Chromium levels differed considerably between different biopsies, but the content of chromium was the same order of magnitude at all exposure durations indicating that a steady state was reached within 5 hrs of exposure.

Mali et al. (1964) measured the disappearance of a radiolabeled chromate solution absorbed dermally in two human volunteers and determined penetration into stratum corneum by tape stripping. Application of a 0.02 ml 0.25% dichromate solution (containing 50 µg Cr(VI)) on a patch to the arm for 12 hrs resulted in the disappearance, and presumed absorption, of 22 µg Cr into the skin. Tape stripping of stratum corneum removed 0.35 µg of radiolabel in the skin.

Systemic uptake of chromium was studied in four human volunteers following a three hour submersion in a tub of water containing 22 mg/L Cr(VI) as potassium dichromate (Corbett et al., 1997). Urinary chromium excretion showed large inter-individual variability. Five-day total Cr urinary excretion above historical background ranged from 17.5 to 1.4 µg, with an average of 6.1 µg. Urine levels of chromium were normal in three volunteers by day 2, although a fourth volunteer excreted elevated levels of chromium up to the end of the experiment

on day 5. Elevated blood and serum levels of chromium were recorded within 1 hr after end of exposure. Chromium content of red blood cells was generally increased about 2-fold, and serum content was increased about 3- to 5-fold. Chromium levels in red blood cells and serum had returned to control levels 2 days after exposure. The systemic uptake rate through skin ranged from 4.1E-04 to 7.5E-05 μg/cm^2-hr with an average of 1.5E-04 μg/cm^2-hr.

Aqueous solutions of Cr(VI) as potassium dichromate, and Cr(III) as chromium trichloride and chromium nitrate were applied in vitro to full thickness human abdominal skin in diffusion cells at a chromium content of 0.034 M (Gammelgaard et al., 1992). Test solutions of 556 μl/cm^2 were applied over a skin surface area of 1.8 or 0.7 cm^2. After 190 hrs exposure of skin to the dichromate, 134 and 12 μg Cr/cm^2 were found in the epidermis and dermis, respectively. Only 0.037 μg Cr/cm^2 was found in the recipient phase. A total Cr(VI) permeation of 15% was calculated. Significantly less Cr(III) from either the trichloride or nitrate was found in skin. Cr(III) content in skin was no more than 9% of the chromium content applied as Cr(VI), with no chromium found in the recipient phase. The lower permeation of Cr(III) was considered a result of the skin acting as a barrier to absorption of the positive Cr(III) ions.

In other experiments by Gammelgaard et al. (1992), application of the dichromate at concentrations of 0.125, 0.25, and 0.5% to skin for 48 hrs showed increased Cr content in skin with increasing concentration, although no Cr was detected in the recipient phase. Total percent Cr permeation of 0.7, 0.7 and 1.1% was calculated for exposure to the 0.5, 0.25 and 0.125% dichromate solutions, respectively. Increasing dichromate concentration (0.5 to 2.5% Cr solution concentrations) with 168 hr exposure did not result in increased Cr content in skin. Long lag times for appearance of Cr in the recipient phase combined with lack of increased skin concentration with time indicates a high binding capacity for Cr that will interfere with diffusion through the skin, although skin binding sites can eventually be exhausted with time. Gammelgaard et al. (1992) also observed the ratio of Cr(VI) to Cr(III) at pH 10 in the recipient phase to increase over 160 hr of exposure. Appearance of chromium as Cr(VI) in the recipient phase increased from about 60% at 40 hrs, to greater than 90% at 120 hrs. This finding indicated reduced capacity for dermal Cr(VI) reduction, eventually resulting in increased Cr(VI) passing through the skin.

Baranowska-Dutkiewicz (1981) found chromium (VI) from aqueous solutions to be readily absorbed by human skin. Seven volunteers were exposed to sodium chromate solutions (0.01, 0.1, and 0.2 M) on an area of the forearm for 15, 30 or 60 minutes, in a series of experiments. The exposure area was covered with a watch glass throughout the exposure period. Absorption was calculated from the difference between the applied and recovered dose of chromium (VI). The authors reported that percutaneous absorption of chromium is dependent on both concentration and time. Specifically, they found that (1) absorption was highest from the 0.01 molar solution (7.7-23% of applied dose) and lowest from the 0.2 molar solution (3.4-10.6% of applied dose), (2) the rate of absorption decreased

as exposure time increased, and (3) the rate of absorption increased proportionally as exposure concentration increased. Individual data were not provided.

Wahlberg and Skog (1963) used disappearance measurements of radiolabeled chromium to estimate dermal absorption of hexavalent chromium in vivo in guinea pigs. Animals were exposed for 5 hours to various concentrations (0.00048 - 4.870 molar) of sodium chromate labeled with ^{51}Cr. Dermal absorption of chromium was confirmed qualitatively by organ analysis. The maximal disappearance of hexavalent chromium was observed from a 0.261 molar solution. Of the 10 animals exposed to this concentration, the mean disappearance percentage per 5-hour period was 4% of the applied dose.

No studies could be located that examined dermal uptake of Cr(VI) from soils. However, chromium fate in soil and soil bioaccessibility studies (gastrointestinal and sweat leaching) have been conducted.

The relationship between Cr(VI) and Cr(III) in soil is a dynamic one, which is affected by soil type and mineral content, pH, solubility, and other factors (Bartlett, 1991; Fendorf, 1995; Stewart et al., 2003). Cr(VI) exhibits greater mobility and less adsorption in soils compared to Cr(III). Organic matter, Fe(II), and sulfides in soils are capable of reducing Cr(VI) to Cr(III), while manganese oxides in soils are capable of oxidizing Cr(III) to Cr(VI). Usually, part of any Cr(VI) added to soil will be reduced instantly, especially under acid conditions. However, high concentrations of polluting Cr(VI) may quickly exhaust the readily available reducing power of the matrix material and excess Cr(VI) may persist for years in soils without reduction.

Oral bioaccessibility of Cr(VI) from aged soils was determined by Stewart et al. (2003) using a physiologically based extraction test designed to simulate the digestive process of the stomach. It would be expected that bioaccessibility for dermal absorption of soil Cr(VI) would be no greater than oral absorption, and oral absorption has been used to estimate dermal exposure to Cr(VI) in soil in previous health assessments (Sheehan et al., 1991).

In general, Cr(VI) bioaccessibility decreased with the aging of Cr(VI) in soils, with decreased bioaccessibility being most rapid for the first 50 days and then slowing dramatically between 50 and 200 days (Stewart et al., 2003). Chromium bioaccessibility was significantly influenced by reduction processes catalyzed by soil organic carbon. Soils with sufficient organic carbon had lower Cr(VI) bioaccessibility values of about 10 to 20% due to enhanced reduction of Cr(VI) to Cr(III). In soils where organic carbon was limited and reduction processes were minimal, considerably higher Cr(VI) bioaccessibility values of 60-70% were recorded.

Soil samples from two chromium waste sites that varied considerably in Cr(VI) concentration were extracted with a synthetic sweat solution to determine the

potential for dermal bioaccessibility of Cr(VI) from contaminated soils (Wainman et al., 1994). The soils examined were contaminated with slag containing chromium from chromate and bichromate production facilities in New Jersey. One set of soil samples contained 710 µg Cr(VI)/g soil and contained chromate blooms, a thin layer of bright yellow crystals on the soil surface. Approximately 83% Cr(VI) was extracted in sweat from the soil with chromate blooms. Adjusting the pH of the soil from pH 5 to 8 had little effect on Cr(VI) extraction. In the other soil, the Cr(VI) concentration averaged 59 µg/g soil. Sweat extraction of Cr(VI) increased from 15 to 32% with increasing soil pH from pH 5 to 8. No Cr(VI) was extracted from the soil adjusted to pH 4. Extraction with distilled-deionized water was also performed, resulting in 76 and 27% extraction from soil with and without blooms, respectively.

Horowitz and Finley (1993) investigated the leaching of Cr(VI) in human sweat from chromite ore processing residue. The New Jersey ore residue originated from the same or similar processing facility as that investigated by Wainman et al. (1994). The human sweat at a pH of 7.2-8.0 extracted < 0.01% of Cr(VI) from the ore samples. Differences in the parent ore and extraction techniques were suspected to have led to the widely varying extraction of Cr(VI) from samples analyzed by Wainman et al. (1994) and Horowitz and Finley (1993).

Oral bioaccessibility studies have also been conducted on the New Jersey slag material (Hamel et al., 1999). Using two different methods, chromium in the slag material had an average bioaccessibility of 34 or 40%, depending on the method used.

F.3.4.2 Discussion and Recommendation for a Hexavalent Chromium (Soluble Compounds) ABS

In the comprehensive in vitro study by Gammelgaard et al. (1992), a measurable increase in Cr(VI) penetrating full thickness human skin could not be detected with 48 hr exposure and only 1.1% of Cr(VI) had been absorbed into the skin. By 190 hrs of exposure fractional absorption of Cr(VI) increased considerably to 15%. The in vitro data indicate Cr(VI) salts have a long lag phase and are slowly absorbed. In contrast, the in vivo human study by Corbett et al. (1997) suggests a very short lag time for appearance of Cr(VI) systemically, with increased Cr levels in the circulatory system within 3 hrs of immersion in a water tank of dilute aqueous dichromate. The human in vivo study by Baranowska-Dutkiewicz (1981) indirectly supports rapid dermal absorption of Cr(VI) with disappearance of aqueous Cr(VI) salt applied to skin for 15-60 min. Consequently, in vitro human exposure likely underestimates the dermal absorption potential of aqueous Cr(VI) solutions that occurs in vivo.

Alternatively, the indirect estimate of up to 23-44% dermal absorption of the applied dose of Cr(VI) salt by Baranowska-Dutkiewicz (1981) and Mali et al. (1964) likely overestimates the dermal absorption potential due to use of a skin occlusion application and reliance on a disappearance method to estimate

absorption. Mali et al. (1964) found only 0.35 µg of chromium in stratum corneum tape stripping even though a total of 22 µg of Cr(VI) was assumed absorbed by disappearance from the skin surface. This finding does not correspond with data by Liden and Lundberg (1979) in which maximal levels of absorbed Cr(VI) was found in stratum corneum.

The 24 hr guinea pig in vivo study by Czernielewski et al. (1965) was the most comprehensive study available in regard to estimating whole body absorption of a dermally applied radiolabeled Cr(VI) solution. Analysis of excreta, blood, and most tissues yielded a fractional absorption of about 2.9%, of which 2.7% was found in excreta and blood. Dermal absorption in experimental animals often overestimates absorption in humans. The in vitro chromate disappearance constants for dermal exposures up to 24 hrs were 3-5 times greater through guinea pig skin compared to human skin (Wahlberg, 1965). However, recognizing that in vitro studies generate slower absorption rates of Cr(VI) than in vivo, the study by Czernielewski et al. (1965) provides a reasonable health protective absorption estimate (2.9%) when considering a human 48 hr in vitro fractional absorption of 1.1% was estimated by Gammelgaard et al. (1992).

To account for the effect of soil vehicle on dermal absorption of Cr(VI), the maximal Cr(VI) bioaccessibility of 83% in synthetic sweat as determined by Wainman et al. (1994) was taken into account. This bioaccessibility estimate was from a soil sample with about 710 µg Cr(VI) per g soil and contained chromate crystals on the soil surface. The contaminated soil probably represents a matrix described by Bartlett (1991) in which high concentrations of Cr(VI) exhausted the readily available reducing power of the soil and excess Cr(VI) persists on the soil surface without being reduced. Thus, multiplying 2.9% by 0.83 and rounded to the nearest whole number provides an ABS point estimate of 2% for Cr(VI) from soil vehicle.

The Hot Spots risk assessment procedures have previously assumed no reduction of deposited Cr(VI) because typically Cr(VI) deposition is modeled without soil sampling monitoring for the Cr(VI)/Cr(III) ratio and without an evaluation of the redox potential of the soil. This assumption may result in overestimation of Cr(VI) soil concentrations in situations where Cr(VI) is readily reduced to Cr(III). Bioaccessibility is determined in part by the Cr(VI)/Cr(III) ratio. The use of soil with high concentrations of Cr(VI) to determine bioaccessibility is not likely to underestimate bioaccessibility under the conditions typically found in Hot Spots risk assessments, where Cr(VI) is deposited over a long period of time and typically results in lower soil concentrations than the 710 µg/g observed in the study by Wainman et al. (1994).

A Limitation for the ABS not discussed above include lack of a factor for absorbed chromium lost through skin desquamation. Studies show that some Cr(VI) will be reduced to Cr(III) in skin and bind to cellular constituents (Gammelgaard et al., 1992; Hostynek, 2003). If this occurs in the stratum corneum, the chromium will likely be removed through desquamation before

systemic absorption can occur. Another limitation includes reliance on studies in which Cr(VI) is applied directly onto the skin (i.e., neat), rather than combined with soil, for estimation of fractional dermal absorption. Kissel (2011) has noted that fractional absorption is dependent on skin loading conditions for application of organic chemicals directly to skin. However, Baranowska-Dutkiewicz (1981) showed that for Cr(VI) the flux through skin increases proportionally with increasing Cr(VI) load applied to skin, resulting in similar fractional absorption values independent of load onto skin. The constraints in estimating fractional absorption for organic chemicals applied neat, which assumes a constant flux through skin, does not appear to be relevant for the metal salt Cr(VI).

F.3.5 Fluoride and Soluble Fluoride Compounds

Recommended use of default inorganic compound ABS estimate of 3.0%.

F.3.5.1 Studies Considered

Excessive exposure to the negatively charged fluoride ion deposited on soil as an aerosol or as a soluble inorganic fluoride salt is known to have toxic effects in animals through ingestion of contaminated soil (Eagers, 1969). However, no quantitative data could be found regarding the fractional dermal absorption of soil-bound fluoride or fluoride compounds following contact with skin. Two animal studies observed elevated fluoride serum levels or systemic toxicity following dermal exposure to concentrated hydrofluoric acid, but immediate skin corrosion was apparent, which would influenced dermal absorption (Derelanko et al., 1985; Boink et al., 1995).

Much of the fluoride naturally present in soils or deposited from facility emissions will generally be in, or strongly adsorbed to, soil particles and is not in a form accessible for uptake by the body (Davison, 1987). Highest levels of water-soluble, or bioaccessible, fluoride in heavily contaminated soils was about 15-20% of total fluoride (Polomski et al., 1982). Among several studies, the bioaccessible fluoride fraction in uncontaminated soils ranged from 0.06 to 7% of total soil fluoride (Gisiger, 1968; Polomski et al., 1982; Milhaud et al., 1989; Buykx et al., 2004).

F.3.5.2 Discussion and Recommendation for a Fluoride and Soluble Fluoride Compound ABS

Due to the lack of quantitative data regarding dermal absorption of soil-bound fluoride, it is not possible to determine an ABS from the data available. Use of a 3% fractional absorption default value, based on the mean of the derived ABS values for Hot Spots metals and semi-metals (As, Cd, Cr(VI), Pb, Hg, Ni), will likely not underestimate dermal absorption of soil-bound fluoride given the highly ionic nature of fluoride and the strong adsorption of deposited fluoride to soil particles.

F.3.6 Lead and Inorganic Lead Compounds

Recommended point estimate for dermal uptake: 3%

F.3.6.1 Studies Considered

A. Key Study

The in vitro dermal absorption of lead oxide (PbO) powder (<10 µm particle diameter) in human abdominal skin was investigated (Filon et al., 2006). Each diffusion cell had a surface area of about 3.14 cm^2 and was filled with 5 mg PbO/cm^2 and with 2 ml synthetic sweat at pH 5.0. At 24 hrs, a median of 2.9 ng/cm^2 (0.06% fractional absorption) had penetrated the skin to the receiving solution and a median of 321.3 ng/cm^2 (6.4% fractional absorption) was absorbed in the skin following surface decontamination. In another experiment, removal of PbO after 30 min exposure did not cause a reduction of Pb penetration in 24 hrs, but did cause a reduction in skin Pb content. This finding suggested that initial rapid absorption of Pb can occur during the first few min of exposure.

B. Supporting Studies

Bress and Bidanset (1991) studied percutaneous absorption of lead in vitro using human abdominal skin obtained from autopsy, and guinea pig dorsal skin. PbO or lead acetate (10 mg) in saline solution was applied to 1.3 cm^2 skin samples. After 24 hours, the lead content of the saline reservoir fluid was measured. The lead content of the skin samples after exposure was not measured. In this experiment, 0.05% of the applied dose of lead acetate was recovered in the reservoir fluid, and less than 0.01% of the PbO. There was no difference between human and guinea pig skin.

Bress and Bidanset (1991) also examined in vivo percutaneous lead absorption in guinea pigs. Lead acetate or PbO, mixed in aqueous solution, was applied to a shaved area (2 cm^2) of the back (300 mg lead per kg body weight). After exposure for 1 week, the animals were killed and lead was measured in blood, brain, liver and kidney. Percent of applied dose absorbed could not be determined from this study. However, the concentration of lead in the measured tissues following lead oxide exposure was similar to that from control animals. In contrast, the lead concentration in measured tissues following lead acetate exposure was greater than controls, although absorption was considered poor, and statistics were not provided.

Moore et al. (1980) studied percutaneous absorption of lead acetate in humans from two commercial hair dye products. The products (one a lotion and one a cream) were spiked with lead-203 (^{203}Pb) and applied to each subject's forehead (n=8) for 12 hours. The preparations were applied in various forms (wet and dried) with periods of one month between each application. Lead absorption was estimated from blood counts, whole-body counts, and urine activity. Results

were normalized for each subject by administration of an intravenous tracer dose of lead chloride.

The mean uptake of ^{203}Pb activity, measured in whole body at 12 hours, was greatest when the preparation was dried and skin was slightly abraded (0.18% of applied dose). The mean absorption including all methods of application (measured in whole body at 12 hours) was 0.058% with a range of 0-0.3%. It has been noted that the presence of colloidal sulphur in the lead acetate formulations used by Moore et al. (1980) may have led to the formation of insoluble lead sulfide, which would be unlikely to be significantly absorbed through skin (Stauber et al., 1994).

In a series of studies in human volunteers, aqueous solutions of inorganic lead salts including lead chloride and lead nitrate were shown to be rapidly absorbed through skin within 3-6 hrs and enter the extracellular compartment, resulting in increased concentrations of lead in the sweat and saliva but not the blood (Lilly et al., 1988; Stauber et al., 1994). However, application of radiolabeled lead (^{204}Pb) to skin of volunteers resulted in measurable increases of ^{204}Pb in the blood but with a very short residence time (Stauber et al., 1994). Preliminary experiments also showed rapid absorption of lead oxide and elemental lead through the human skin of volunteers and detection in the sweat within a few hours. Only $PbCO_3$ was not absorbed through skin. In mice, skin-absorbed lead concentrated more strongly in skin and muscle, and less in blood and other organs compared to intravenously injected lead (Florence et al., 1998).

The authors proposed that the behavior of skin-absorbed lead in the body is different from lead that is ingested or injected, in that lead which passed through skin is in a physicochemical form with low affinity for erythrocytes and a high affinity for extracellular fluid compartments. The implication is that testing blood for lead exposure may not fully account for absorption of lead through the skin.

Stauber et al. (1994) examined dermal lead absorption by placing lead nitrate and lead nitrate spiked with ^{204}Pb on the arms of volunteers for 24 hrs. Rapid increases of lead were observed in sweat samples from the unexposed arm and in saliva, but only small concentrations of lead in blood and urine. However, high levels of ^{204}Pb in blood and urine were measured 2 and 16 days, respectively, after exposure ended suggesting slow absorption of lead into the blood from lead retained in the skin.

In order to quantify dermal lead absorption, 4.4 mg lead (as 0.5 M $Pb(NO_3)_2$) was dispensed onto filter paper and secured with plastic wrap to the left arm of one subject. After 24 hours, the filter paper was removed and the arm was washed. Of the 4.4 mg lead, 3.1 mg was recovered from the filter paper and wash fluid. Using this disappearance technique, the authors estimated that 29% of the lead was absorbed into or through the skin. In two volunteers, the estimated excretion of skin-absorbed ^{204}Pb in the sweat of two volunteers over 24 hrs was 16 and 46 µg lead/L. Assuming an average sweat production of 500 ml/day, the authors

estimated 0.6% and 1.5% of the total lead that was absorbed was excreted in sweat.

Lead acetate or nitrate was also applied to the skin of mice by the researchers in order to quantitate the amount of lead absorbed and retained in organs and tissues (Florence et al., 1998). Forty µl of aqueous solutions of the lead salts (6.4 mg of lead) were applied to a shaved area of skin and covered with Parafilm. Mice were sacrificed and organs and tissues analyzed for lead content after time periods of 2 hrs to 1 week. A total analysis of the organs, feces, and urine showed that, of the 6.4 mg of lead applied to the skin, 26 µg (0.4%) was absorbed through the skin and entered the circulatory system in 21 hrs. This analysis does not appear to include skin-absorbed lead at the site of application. No differences in absorption of the two lead salts were observed. Increased organ content of lead was noted by 6 hrs of exposure, with maximal organ concentrations generally occurring after 24-48 hrs of exposure.

To investigate the stratum corneum depth profiles of lead in lead battery workers, 10 repeated skin strips were collected from exposed skin (dorsal hand) and nonexposed skin (lower back) of 10 volunteers (Sun et al., 2002). Skin areas to be sampled were washed with soap and water, then ethanol, prior to collection in the morning before work. Total lead in stratum corneum strippings ranged from 20.74 to 86.53 µg (mean = 42.8 µg) from the hand, and 8.94 to 28.32 µg (mean = 17.4 µg) from the back. Approximately 20.8 µg (49%) of the total lead in the stratum corneum were in the first two tape strippings. There was a decreasing amount of lead content from both skin regions going from the outer to the inner layers, suggesting both regions had been contaminated with lead. Total amount of lead in the hand, but not the back, was linearly correlated with the amount of lead in blood. These findings indicate the source of lead in skin was from dermal exposure, rather than absorption of lead from the circulatory system into the skin.

Although the lead compound that workers were exposed to was not specified in the Sun et al. (2002) study, the primary lead compounds emitted during lead-acid battery production are identified as PbO and elemental lead (USEPA, 1998; Ruby et al., 1999). Elemental lead particles that are deposited in soils quickly form coatings of highly bioavailable PbO.

The leaching behavior of lead-contaminated soil can be divided into three stages based on the leachate pH: a high alkalinity leaching stage at pH > 12, where Pb formed soluble hydroxide anion complexes and leached out; a neutral to alkaline immobilization stage in the pH range of 6-12, which was characterized by low Pb leachability by adsorption and precipitation; and an acid leaching stage with pH < 6, where leachability increased exponentially with decreasing pH and was characterized as free Pb-ion (Jing et al., 2004). This study indicates that soluble Pb at the neutral pH found in most soils would only be a fraction of the total Pb content of the soil.

Several leaching studies of Pb-contaminated soils suggest the bioaccessible Pb in soil can vary greatly. Within a pH range of 7-8, soluble Pb ranged from less than 0.01% to 48% of total Pb content of soil (LaPerche et al., 1996; Yang et al., 2001; 2002; Jing et al., 2004). In a major Pb contamination due to a paint spill, the Pb soil content was 34,592 mg/kg, which is roughly an order of magnitude greater than many Pb-contaminated soils (Zhang et al., 1998). Soluble Pb at pH 7 was roughly estimated to be 18% of total soil Pb. At pH 5, fractional soluble Pb increased to about 41% of total soil Pb.

F.3.6.2 Discussion and Recommendation for a Lead and Inorganic Lead Compound ABS

The accumulated in vivo absorption data did not provide enough quantitative information to estimate an ABS point estimate of lead including both systemic absorption and that retained in skin. Additionally, no data could be found that measured dermal absorption of lead from contaminated soil. Thus, the lead ABS point estimate incorporated data from an in vitro human study of lead applied neat and soil leaching tests for lead-contaminated soil.

The most comprehensive human data available were the in vitro study by Filon et al. (2006), which observed 0.06% of applied lead penetrating to the receiving solution and 6.4% of applied lead retained in skin following dermal exposure of PbO in a synthetic sweat solution. The skin depth profile of lead shows 49% of the total lead in the stratum corneum was in the first two tape strippings, and might be removed through desquamation prior to systemic absorption (Sun et al., 2002). However, human in vivo dermal exposure data suggest a relatively short lag time for appearance of lead in blood and continual absorption of lead into the blood from the skin reservoir (Lilly et al., 1988; Stauber et al., 1994). Until further studies are conducted to estimate the fraction of lead removed via desquamation prior to systemic absorption, it is presumed that all the lead absorbed in skin is available for systemic absorption.

Although only 0.06% of the lead reached the receiving solution in the in vitro study by Filon et al. (2006), in vitro dermal absorption studies of metal salts generally do not include a full accounting of absorption due to skin shunts such as hair follicles and sweat ducts. Hostynek (2003) noted that these skin shunts swell shut upon hydration during in vitro dermal absorption studies, and can reduce the movement of some dermally applied metal salts directly into lower skin layers. The human in vivo data support the importance of sweat ducts for lead dermal absorption (Lilly et al., 1988; Stauber et al., 1994). In addition, the rapid reduction of lead dermal absorption early during exposure in the Filon et al. (2006) in vitro study has been considered evidence for skin shunts becoming hydrated and reducing lead absorption by these pathways (Hostynek, 2003). These data further support the reasoning that the lead retained in skin observed by Filon et al. (2006) cannot be discounted for potential systemic absorption.

In soil, aqueous leaching studies suggest soluble Pb can vary greatly depending on the soil characteristics. If sweat is the leachate, the pH can range between 4 and 7, with an average in male Caucasians of 4.85 (Wainman et al., 1994). The acidic nature of sweat will likely enhance Pb bioaccessibility from soil compared to the soil pH ranges of 7-8. Because of the wide range of solubilities of Pb in soil, a health protective point estimate based on the solubility of a heavily Pb contaminated soil at pH 5 (average pH of sweat) is warranted. Zhang et al. (1998) observed an approximate 41% Pb solubility at pH 5 from highly contaminated soil (Pb content = 34,592 mg/kg soil). Adjusting the total fractional dermal absorption of 6.46% observed by Filon et al. (2006) by multiplying by the fraction of soluble Pb in a highly impacted soil (0.41) determined by Zhang et al. (1998) results in an ABS point estimate of 3% after rounding to the nearest whole number.

The ABS of 3% for Pb salts is higher than most other metal salts investigated. However, most of the soil leaching experiments used soils that were environmentally contaminated or incorporated time as a factor to control for soil aging. Absorption of Pb salts has also been shown to be high by the oral route relative to other metals, up to 90% absorption in the acidic environment of the stomach (Ruby et al., 1999). A limitation for this ABS is the reliance on studies in which lead is applied neat to skin, rather than combined with soil, for estimation of fractional dermal absorption. Kissel (2011) has noted that fractional absorption is dependent on skin loading conditions for application of organic chemicals directly to skin. However, Baranowska-Dutkiewicz (1981) showed that for Cr(VI) the flux through skin increases proportionally with increasing Cr(VI) load applied to skin, resulting in similar fractional absorption values independent of load onto skin. Thus, dermal absorption of salts of lead applied neat probably is closer to the dermal absorption kinetics of Cr(VI), rather than to organic compounds.

F.3.7 Inorganic Mercury Compounds

Recommended point estimate for dermal uptake from soil: 3%

F.3.7.1 Studies Considered

Quantitative in vivo dermal absorption studies of Hg-contaminated soils have not been performed. A summary of the in vitro dermal studies exposing human and animal skin to Hg-contaminated soil are shown in Table F-2.

A. Key Studies

The dermal bioavailability of $^{203}HgCl_2$ was tested in vitro on dermatomed male pig skin as pure compound or following addition to sandy soil or clay soil (Skowronski et al., 2000). The Yorkshire pig model was chosen due to histological, physiological, biochemical and pharmacological similarities to human skin. The sandy and clay soil consisted of 4.4% and 1.6% organic matter,

respectively, and a majority of the soil particles were in the range of 50-250 µm. A soil loading of 47 mg/cm^2 was calculated from the data provided and the $HgCl_2$ concentration was 5.3 ng/mg soil. Absorption was estimated up to 16 hrs following application.

In general, dermal absorption of Hg was greater from sandy soil than from clay soil. In both soils, the rate of appearance of Hg in the receptor fluid was rapid during the first hour, then decreased to a steady state for the remaining 15 hrs. In sandy soil freshly spiked with Hg, 0.28% and 37.5% of the applied dose had penetrated the skin to the receptor fluid and was bound to skin, respectively, at 16 hrs. In clay soil freshly spiked with Hg, 0.08% and 39.7% of the applied dose had penetrated the skin to the receptor fluid and was bound to skin, respectively, at 16 hrs. For the pure compound, Skowronski et al. (2000) observed a skin penetration of 0.18%, but the amount bound to skin was 66.3%. For Hg aged 3 months in soil, dermal absorption was reduced to 3.3% in sandy soil and 2.6% in clay soil. Only 0.04% and 0.01% of these totals in the sandy and clay soil, respectively, represented percent of applied dose penetrating to the receptor fluid.

B. Supporting Studies

Radiolabeled mercuric chloride ($^{203}HgCl_2$) was mixed with soil and applied in vitro onto fresh human breast skin (obtained within 24 hrs of harvest) for 24 hrs by means of Bronaugh diffusion cells (Moody et al., 2009b). The same amount of $^{203}HgCl_2$ was also applied without soil to human skin samples. The soil had been sieved to 90-710 µm prior to spiking with the Hg salt. The soil mixture (3.2 mg soil) was added to the diffusion cells resulting in a soil loading of 5 mg/cm^2. At 24 hrs, mean percent dermal absorption including the skin depot was 46.6 and 78.3% with and without soil, respectively. The fraction of total absorbed Hg that entered the diffusion cell in 24 hrs was 1.5 and 1.4% with and without soil, respectively.

A radiolabeled mercury compound ($^{203}HgCl_2$) was applied in soil or water vehicle to human skin in vitro (0.5 µg/cm^2 containing 1 µCi) for 24 hours (Wester et al., 1995; Wester and Maibach, 1998c). The investigators used Yolo County soil (26% sand, 26% clay, 48% silt, 0.9% organic) sieved for 180-300 µm particles. Receptor fluid accumulation from either water vehicle or soil vehicle was 0.07% of applied dose. Previously frozen or fresh skin gave similar results. Skin content of mercury from water vehicle averaged 29% of total dose applied. Using soil loads of 5, 10, and 40 mg, skin content of mercury was 10.4, 6.1, and 7.2% of dose applied, respectively.

In other human in vitro studies by the same research group, 5.5% absorption into skin and 0.01% penetration of pure $HgCl_2$ into receptor fluid was observed with a 30 min exposure (Wester et al., 1995; Wester and Maibach, 1998c). Continued perfusion for 48 hrs following the 30 min exposure increased skin absorption and penetration to receptor fluid to 6.3% and 0.09%, respectively, exhibiting the

ability of Hg to migrate through skin after removal of Hg from the skin surface. When the in vitro exposure was increased from 30 min to 24 hrs, mercury skin absorption and penetration to receptor fluid was increased to 35.4% and 0.06%, respectively. No other results or methodology details were provided.

The dermal bioavailability of liquid and soil-bound $^{203}HgCl_2$ was tested on dermatomed human male skin in vitro (Sartorelli et al., 2003). For the liquid vehicle, $HgCl_2$ was added to buffered water solution (pH = 4.0). For the soil vehicle, $HgCl_2$ was added to loam soil consisting of 60% sand, 30% silt and 10% clay sieved to a particle size of <150 µm. Soil loading on skin was about 40 mg/cm^2, which would be greater than monolayer coverage using a particle size of <150 µm. The concentration of $HgCl_2$ was 0.0069 or 0.1190 nmol/cm^3. After 72 hr exposure, any mercury absorbed from soil and penetrating skin to the receiving fluid was below the detection limit. Mean mercury concentrations in the skin were 10.53% of the applied low dose and 15.04% of the applied high dose. Mercury in the liquid vehicle was also applied at two concentrations of 0.0088 and 0.0607 nmol/cm^3. At the low dose, percent of applied dose penetrating skin to the receptor fluid was 1.64 and 4.80% at 24 and 72 hrs, respectively. At the high dose, percent of applied dose penetrating skin to the receptor fluid was 0.34 and 0.93% at 24 and 72 hrs, respectively. Percent of applied dose retained in skin at 72 hrs was 18.93 and 44.97% for the low and high dose, respectively.

TABLE F.2. In Vitro Dermal Absorption Results of Mercuric Chloride from Soil

Study	Species	Exposure time (hr)	Soil fraction (µm)	% Reaching receptor	% Total absorbed fresh	% Total absorbed aged
Skowronski et al., 2000	pig	16	unsieved	0.28 [a] 0.08 [b]	37.8[a] 39.8[b]	3.3[a] 2.5 [b]
Moody et al., 2009	human	24	90-710	1.5	46.6	ND[c]
Wester et al., 1995	human	24	180-300	0.07	7.9	ND
Sartorelli et al., 2003	human	72	<150	0[d]	13	ND

[a] Sandy soil
[b] Clay soil
[c] Not determined
[d] Below the limit of detection

Hursh et al. (1989) studied dermal absorption of mercury vapor in humans. Each of 5 men exposed the skin of one forearm (a single exposure) to vapors with concentrations ranging from 0.88-2.14 ng $^{203}Hg/cm^3$ for periods of 27 to 43 minutes. The rate of dermal uptake of mercury by the arm was quantified by measuring the difference between accumulated radioactivity on exposed and unexposed forearms following exposure. The mean uptake rate for the 5 subjects was reported as 0.024 ng Hg per cm^2 skin per minute per ng Hg per cm^3

air. At this rate, the authors estimate that dermal absorption of mercury from vapor is approximately 2.6% of the rate of uptake by the lung.

In addition, the study protocol by Hursh et al. (1989) included a procedure in which adhesive strips were applied every 3-4 days post exposure for up to 40 days, which regularly removed cells of the stratum corneum from the same marked skin area following exposure. Larger amounts of Hg were stripped at later time points, suggesting that a substantial fraction of the absorbed Hg was probably associated or bound to keratinocytes rather than stratum corneum. Based on the whole body count of radiolabeled Hg and the amount of Hg absorbed in the skin, the authors note that about half of the Hg eventually reached the bloodstream while the remainder was shed by desquamating cells. The data show estimates of 26, 43, 45, 45 and 46% of the dermally absorbed Hg reaching the bloodstream in the five volunteers. It was theorized that the elemental Hg penetrated the stratum corneum as vapor but that in the epidermis, some, but not all, of the Hg became oxidized to mercuric ions. The ions then became fixed or bound in the skin, some of which then moved upward and was eventually shed.

Baranowska-Dutkiewicz (1982) exposed the forearms of eight male volunteers to aqueous mercuric chloride solutions. Aliquots (0.25 ml) of $HgCl_2$ solutions were applied directly to a 22 cm^2 area of skin and covered with a watch-glass. Percutaneous absorption of mercury was calculated as the difference between the amount applied and the amount recovered after the skin and the watch-glass were washed. In order to examine the effect of concentration on uptake, 3 concentrations (0.01, 0.1, and 0.2 M) were applied for 30 minutes. As concentration increased, rate of uptake increased. In order to examine the influence of exposure time on uptake, 0.1 M $HgCl_2$ was applied for 5, 10, 15, 30 and 60 minutes. The authors reported that the average rate of uptake of mercury decreased from 9.3 $\mu g/cm^2$/min during a 5 minute exposure, to 2.5 $\mu g/cm^2$/min during a 1 hour exposure. The average percutaneous absorption of mercury was calculated for exposures of 5, 10, 15, 30, and 60 minutes resulting in 20%, 29%, 37%, 60% and 64% absorption of the applied dose, respectively.

In vivo application of aqueous $HgCl_2$ (0.1% w/v) to normal human skin followed by biopsy and visualization with electron microscopy found mercury deposits present intracellulary and extracellularly in the stratum corneum within minutes after application (Silberberg, 1972). The presence of mercury in the epidermis was not apparent until 2-4 hrs after application. The finding of immediate absorption of $HgCl_2$ correlates well with the in vivo findings of Baranowska-Dutkiewicz (1982), which observed the disappearance of $HgCl_2$ within 5 min after application to human skin.

An in vivo study in guinea pigs found that dermal absorption of Hg from $HgCl_2$ steadily decreases with increasing dose, suggesting a buildup of a secondary diffusion barrier as a consequence of the electrophilic metal forming irreversible bonds with proteins of the skin (Friberg et al., 1961). Thereby a depot

accumulates in the stratum corneum retarding further penetration in inverse proportion to metal concentration. This secondary barrier build-up retarding absorption was also evident with increasing dermal exposure intervals. $HgCl_2$ applied in vitro on human skin showed greatest percutaneous absorption during the first 5 hrs (Wahlberg, 1965). With later time periods the absorption rate decreased. The average absorption rate over the first 24 hrs was only about one-fourth the rate observed during the first 5 hrs of dermal exposure.

F.3.7.2 Discussion and Recommendation for an Inorganic Mercury Compound ABS

More than 98% of mercury in soils is present as nonalkyl Hg(II) compounds and complexes, with direct deposition a significant component for much of the loading to terrestrial soils (Davis et al., 1997). In the soil, Hg can occur in three different valence states, namely as Hg^0, Hg_2^{2+} and Hg^{2+} (Andersson, 1979). Hg^{2+} forms various complexes with OH^- and Cl^- ions, with the dominating mercuric complexes being $HgCl_2$, $Hg(OH)_2$ and HgOHCl. Only a small fraction of mercuric Hg species occurs free in solution; the major fraction is either bound to or in the soil material. Hg^{2+} and gaseous Hg^0 forms are preferably bound to organic matter in acidic soils, whereas in neutral and slightly alkaline soils, mineral components are active as well. Mercury exhibits a very high affinity for sulfide in reducing environments, forming relatively insoluble HgS (Davis et al., 1997).

Human skin both in vivo and in vitro has been shown to have a large capacity to accumulate metallic mercury vapor or mercury salts (as $HgCl_2$) applied in aqueous solution directly to skin. When freshly mixed with soil, Hg salts appear to have a greater ability for absorption into skin than other metal salts of concern in this section (i.e., Ni, Pb, Cd, etc.). However, similar to other metals, aging of Hg salt in soil significantly reduces the fractional absorption of Hg into skin. Therefore, a fractional absorption of 3% for $HgCl_2$ aged in soil prior to testing was chosen as the basis of the ABS to account for the aging affects in soil.

The Hg ABS is based on the in vitro study in pigs by Skowronski et al. (2000), in which $HgCl_2$ aged in soil for three months resulted in a considerable reduction of fractional absorption compared to $HgCl_2$ freshly mixed with soil. Limitations of this study include use of skin from a non-primate species, less than 24-hr exposure, and likely exceedance of soil monolayer coverage during the exposure. However, the human in vitro studies shown in Table F-2 also have their limitations for estimating fractional absorption, including exceedance of soil monolayer coverage (Sartorelli et al., 2003), or use of soil fractions that do not include soil particles less than 90 to 180 µm, which most commonly adhere to skin (Wester et al., 1995; Moody et al., 2009b).

Given the limitations, it is still unlikely that the ABS will underestimate fractional absorption. While both the human and animal in vitro studies show a large capacity for dermal absorption of Hg salt, very little reaches the diffusion cells (see Table F-2). Other studies reviewed here indicate that some of the Hg^{++} ions

in mercuric salts tend to bind tightly to cellular proteins in all strata of skin, including stratum corneum, which may then impede further diffusion of mercury (Friberg et al., 1961; Silberberg, 1972; Hostynek, 2003). Mercury bound in stratum corneum would likely be removed via desquamation of skin. Hursh et al. (1989) have shown that a considerable portion of absorbed Hg in skin will eventually be lost (up to 50%) due to desquamation.

Nevertheless, the development of a Hg ABS would benefit from human in vitro studies with Hg salts aged in soil, and continued monitoring after 24-hr dermal exposure to better estimate the amount of Hg that reaches the circulation (i.e., reaches the diffusion cells) and how much is likely to be lost due to desquamation. Because the ABS is based on Hg aged in soil, the ABS may underestimate fractional dermal absorption for soils in which a significant fraction of Hg has been very recently deposited on soil, or for soils that are heavily contaminated or saturated with Hg.

F.3.8 Nickel and Nickel Compounds

Recommended point estimate for dermal uptake from soil: 4%

F.3.8.1 Studies Considered

A. Key Studies

Radiolabeled nickel chloride ($^{63}NiCl_2$) was mixed with soil and applied in vitro onto fresh human breast skin (obtained within 24 hrs of harvest) for 24 hrs by means of Bronaugh diffusion cells (Moody et al., 2009b). The same amount of $^{63}NiCl_2$ was also applied without soil to human skin samples. The soil had been sieved to 90-710 µm prior to spiking with nickel salt. The soil mixture (3.2 mg soil) was added to the diffusion cells resulting in a soil loading of 5 mg/cm^2. At 24 hrs, mean percent dermal absorption including the skin depot was 1 and 22.8% with and without soil, respectively. The fraction of total absorbed nickel that entered the diffusion cell in 24 hrs was 0.5 and 1.8% with and without soil, respectively.

In vivo, sequential adhesive tape stripping was implemented to characterize the penetration of nickel salt solutions in methanol and nickel metal powder in human stratum corneum following 24 hr occlusive application to the forearm (Hostynek et al., 2001a; Hostynek et al., 2001b). Hostynek et al. (2001a) investigated stratum corneum depth profiles for chloride, sulfate, nitrate and acetate nickel salts. Penetration of the stratum corneum by nickel salts at levels of 0.001-1% nickel salt was limited and closely related to the counter ion. The total percent dose of each salt recovered in stratum corneum was 26.1, 18.5, 8.8, and 3.3% for the nitrate, acetate, sulfate, and chloride, respectively. Tape stripping of the skin showed that most of the dose remained on the surface or was retained in the superficial layers of the stratum corneum. Depth profiles converged towards non-detectable levels in the lower stratum corneum regardless of concentration

for the acetate, chloride and sulfate. Nickel applied as nitrate is retained at a constant level of approximately 1% of applied dose in the lower layers of the stratum corneum.

The in vitro permeation of 1% aqueous solutions of chloride, sulfate, nitrate, and acetate nickel salts across only the stratum corneum was investigated using human leg skin (Tanojo et al., 2001). An initial surge in permeation rate within the first 24 hrs was observed for the nickel salts, followed by steady-state permeability rate up to 96 hrs that was not significantly different among the four salts. Nickel sulfate penetration of stratum corneum was greatest at 1.09%, whereas nickel nitrate recovery within stratum corneum was greatest at 0.95%. Total absorption (receptor fluid plus bound to stratum corneum) was 1.65, 1.49, 0.92, and 0.12 % for the sulfate, nitrate, chloride, and acetate salts, respectively. Total recovery of absorbed and unabsorbed nickel was virtually complete for all the salts except nickel nitrate, in which 84% recovery was attained.

Permeation of the salts was attributed by Tanojo et al. (2001) solely to the diffusion across the transcellular/intercellular barrier, as hair follicle and gland shunts were shut upon hydration by the aqueous solutions. These pathways swelling shut early during in vitro exposure may explain the decreased rate of absorption of nickel following an initial surge. Lack of ability to account for absorption of nickel via skin shunts may underestimate absorption.

B. Supporting Studies

Nickel reversibly binds to constituents of the epidermis when human epidermis was homogenized and incubated with nickel chloride solutions (Fullerton and Hoelgaard, 1988). Spruit et al. (1965) utilizing human cadaver skin has shown that nickel ions also reversibly bind to the dermis. Nickel powder has also been shown to oxidize when suspended in synthetic sweat, whereupon the metallic ions can be absorbed in vitro through human skin (Larese et al., 2007).

Under the same experimental exposure conditions as used by Hostynek et al., (2001a), nickel metal powder (particle size 3 μm) values were found to decrease from the superficial to the deeper layers of the stratum corneum (Hostynek et al., 2001b). However, nickel was still present at the deepest levels of stratum corneum removed by adhesive stripping, indicating that the metal has likely reached the viable epidermis and has potentially become systemically available. Although the data did not lend itself to estimation of a skin permeation rate, total nickel removed with 20 strips from the skin after 24 hr occlusion with 21.7 mg/cm^2 nickel powder was 38.7 $\mu g/cm^2$ (i.e., approximately 0.18% of the total nickel metal applied was found in the stratum corneum). These data indicated that in intact skin, nickel metal is oxidized to form soluble, stratum corneum-diffusible compounds which penetrate the intact stratum corneum.

Dermal absorption of nickel chloride as $^{63}NiCl_2$ from two different soils was determined in vitro through dermatomed pig skin cut 200 μm thick (Abdel-

Rahman et al., 1997). Soil types included a sandy soil with 4.4% organic matter and a clay soil with 1.6% organic matter. Skin applications included $^{63}NiCl_2$ added immediately after the addition of the two soils (30 mg each) to skin, or after each soil was aged for 6 months with $^{63}NiCl_2$. Nickel chloride was also added alone in ethanol vehicle to separate skin samples. The chemical dose was 113.8 ng/cm^2 and the soil loading was calculated to be 47 mg/cm^2. Monolayer coverage was probably exceeded with a soil loading of 47 mg/cm^2, causing a reduction in the observed fractional absorption.

Following 16 hrs of exposure, 0.3% of freshly applied $^{63}NiCl_2$ in clay soil penetrated the skin to receptor fluid and 12.1% was found bound to skin. No significant difference for dermal absorption from sandy soil was observed. For the nickel solution applied to skin, 0.4 and 57.9% of the dose applied was found in receptor fluid and bound to skin, respectively. In aged sandy and clay soil, 0.03 and 0.05% nickel was found in the receptor fluid, respectively. Only 3.1 and 3.7% of the metal was bound to skin from sandy and clay soil, respectively. Aging nickel in the soils appeared to be complete by 3 months, as further aging in soil for 6 and 12 months did not result in further decreased dermal bioavailability of the metal (Abdel-Rahman et al., 1997; Abdel-Rahman et al., 1999).

Fullerton et al. (1986) examined the permeation of nickel salts, specifically nickel sulfate and nickel chloride, through human full-thickness breast or leg skin in vitro. Skin excised in surgery was exposed to aqueous solutions of 184 μg/cm^2 for each nickel salt for up to 144 hrs. In the first experiment the effect of occlusion on the permeation rate of nickel chloride was examined. Occlusion resulted in a significantly higher permeation rate (approximately 3.6 percent of applied dose) compared with non-occluded exposure (approximately 0.23 percent) after 144 hours.

In the second experiment, nickel ions from a chloride solution were found to pass through the skin about 50 times faster than nickel ions from a sulfate solution. The amount of permeation of nickel chloride was much higher (16%) at 144 hours than nickel sulfate (0.3%). However, dermal penetration of the skin was slow, having a lag-time of about 50 hours. The occluded-skin permeation of nickel chloride was considerably higher in experiment 2 than experiment 1 (9-16% vs 3.6%) and was attributed by the authors to the use of breast skin from different donors.

In another study by the researchers, the stripping method was used in vitro on human full thickness skin following exposure to 5% nickel chloride in a 5% methyl cellulose gel for 96 hrs under occlusion (Fullerton et al., 1988). Nickel penetration from the gel solution gave similar results to nickel penetration of the pure nickel salt. Skin depth profiles found 50.9% was present on and in the stratum corneum (skin was not washed before stripping) with most of the nickel in the upper part of the stratum coeneum, 10.6% in the epidermis, 1.6% in the dermis, and only 0.4% reached the receptor solution.

Although the time frame and doses were different, similar dermal absorption results were obtained by Turkall et al. (2003) with in vitro dermal exposure of pig skin to 64 ng of radiolabeled nickel chloride. Penetration of ^{63}Ni in ethanol through pig skin was 0.4% of initial dose and a total of 58% of the nickel remained in the skin at the end of 16 hrs.

F.3.8.2 Discussion and Recommendation for a Nickel and Nickel Compound ABS

The only study that exposed human skin to soil contaminated with a nickel salt was the in vitro study by Moody et al. (Moody et al., 2009b). However, there is evidence to suggest in vitro tests for dermal absorption of nickel may underestimate absorption in vivo.

Hostynek et al. (2001a) observed a range of 26.1% to 3.3% absorption of applied dose over 24 hrs among four nickel salts tested in vivo on human stratum corneum. However, Tanojo et al. (2001) observed only a range of 1.65% to 0.12% absorption of applied dose over 96 hrs among the same four nickel salts tested in vitro on human stratum corneum. Comparison of these data indicates that reliance on in vitro absorption data probably underestimates the in vivo dermal absorption of nickel salts.

Specifically regarding the nickel chloride salt applied directly to skin, Hostynek et al. (2001a) observed a 24-hr total absorption of 3.3% for human skin in vivo, while Tanojo et al. (2001) observed a 96-hr total absorption of 0.92% for human skin in vitro. These data together suggests a 3.6-fold greater absorption in vivo compared to in vitro absorption.

Although the dermal absorption time used by Tanojo et al. (2001) was 96 hrs, most of the $NiCl_2$ had penetrated the skin in the first 24 hrs (probably greater than 95%) and appearance of nickel into the diffusion cells had attained steady state. Assuming steady state levels of $NiCl_2$ had also been reached in stratum corneum by 24 hrs, it can be estimated that the total absorption of $NiCl_2$ recorded by Tanojo et al. at 96 hrs was similar to that found at 24 hrs.

Applying a 3.6-fold in vivo/in vitro ratio adjustment to the fractional dermal absorption value of 1% for $NiCl_2$ determined by Moody et al. (2009b) results in an ABS value of 3.6% (or 4% when rounded to the nearest whole number). The ABS is similar to the fractional dermal absorption of 2-4% resulting from exposure of pig skin to $NiCl_2$ aged in different soils (Abdel-Rahman et al., 1997; Abdel-Rahman et al., 1999).

F.3.9 Selenium and Selenium Compounds

Recommended use of default inorganic compound ABS estimate of 3.0%.

F.3.9.1 Studies Considered

No quantitative data could be found regarding the fractional dermal absorption of soil-bound selenium (Se) or Se compounds applied to skin.

In dermal absorption studies of Se solutions, Farley et al. (1986) applied a 2.5% selenium sulfide lotion topically overnight on human volunteers. Skin region exposed and surface area covered were not described. Se levels in urine following exposure were significantly increased over control levels, but absorption was considered too slight to result in toxic effects. Repeated overnight treatments in a few volunteers over two days did not result in Se concentrations in the urine which were significantly higher than normal. In another study, increased serum levels of Se could not be measured in human volunteers that applied 2.5% selenium sulfide lotion to their torso overnight (Kalivas, 1993). Used in shampoo as a 1% selenium sulfide concentration, weekly use for a year did not change the normal urinary Se level (Cummins and Kimura, 1971).

Selenium sulfide is insoluble in water and is considerably less toxic via the oral route compared to elemental selenium or ionic forms of water-soluble selenite and selenate salts, such as sodium selenite (Cummins and Kimura, 1971). Lower gastrointestinal absorption of the sulfide salt was thought to be the cause of the lower oral toxicity.

The fraction of applied dose of ^{75}Se internally absorbed following application of selenous acid, a highly water soluble Se compound, onto the pelts of rats was calculated to be 1% per day over a 9-day exposure period (Medinsky et al., 1981).

F.3.9.2 Discussion and Recommendation for a Selenium and Selenium Compounds ABS

Due to the lack of quantitative data regarding dermal absorption of soil-bound Se compounds, it is not possible to determine a chemical-specific point estimate ABS. However, use of a 3% fractional absorption default value for Se and Se salts for screening purposes, based on the mean of the derived ABS values for the Hot Spots metals and semi-metals (As, Cd, Cr(VI), Pb, Hg, Ni), will likely not underestimate dermal absorption of soil-bound Se, given that fractional absorption of highly soluble selenous acid applied neat to the pelts of rats was about 1% of applied dose.

F.4 Point Estimates for Dermal Absorption (ABS) of Organic Compounds

F.4.1 Polychlorinated Biphenyls (PCBs)

Recommended point estimate for dermal uptake from soil: 14%

F.4.1.1 Studies Considered

A. Key Study

The dermal uptake of each of the two commercial PCB formulations Aroclor 1242 and Aroclor 1254 was studied in vivo in female rhesus monkeys (Wester et al., 1993b). Aroclor 1242 is dominated by the tri- and tetra congeners (68 percent) and Aroclor 1254 is dominated by the penta- and hexa congeners (83 percent). Each PCB preparation was adsorbed onto soil particles that before sieving contained 26% sand, 26% clay, 48% silt, and 0.9% organic carbon. The soil was fractionated by particle size to 180 - 300 μm. The soil levels of the PCB preparations were 44 ppm Aroclor 1242 and 23 ppm Aroclor 1254.

The PCB laden soil was applied for 24 hours to a 12 cm^2 area of lightly shaved abdominal skin which was protected by a non-occluded patch. The applied doses were 1.75 μg/cm^2 Aroclor 1242 and 0.91 μg/cm^2 Aroclor 1254. The soil loadings were 40 mg soil/cm^2 skin for both preparations. Following the first 24 hour exposure during which systemic absorption was measured as the content recovered in urine and feces, the patch was removed, the visible soil was removed from the site of application, the treated skin was washed with soap/water, and urine/feces were collected for an additional 34 days. One group of monkeys was exposed to the PCBs intravenously to adjust the cumulative urine/feces recovery of the dermally applied PCBs. The corrected fractional dermal absorption was 13.9% for Aroclor 1242 and 14.1% for Aroclor 1254.

B. Supporting Studies

PCBs are frequently found as complex mixtures of isomers in soil. To determine the effect of chlorine substitution on dermal absorption, Garner and Matthews (1998) applied dermal doses of ^{14}C-labeled mono-, di-, tetra-, and hexachlorobiphenyls to 1 cm^2 areas on the backs of rats for 48 hrs. Dermal penetration varied inversely with the degree of chlorination and ranged from essentially 100% for monochlorobiphenyl to about 30% for the hexachlorobiphenyl. However, the highly chlorinated PCBs tend to have slower metabolism and elimination and remain in the site of exposure longer, resulting in slow diffusion to the systemic circulation.

Mayes et al. (2002) dermally exposed female rhesus monkeys to radiolabeled Aroclor 1260 in soil in a manner similar to that used by Wester et al. (1993b). The soil was classified as sandy silt made up of 20% sand, 54% silt and 20% clay with a total organic carbon content of 5-6%. Sieving to <150 μm prior to application adjusted the total organic carbon content up to 8.7%. Five-hundred

mg of soil either freshly spiked or aged for 88 days with PCBs (about 70 µg PCBs/g soil) was applied to a 12 cm^2 area of the chest/abdominal area and protected by a non-occluded patch. The calculated dermal load was 42 mg/cm^2. One group was exposed to radiolabeled PCBs intravenously to adjust the cumulative urine/feces recovery of dermally applied PCBs. Groups exposed for 12 or 24 hrs to PCBs aged in soil exhibited percutaneous absorption values of 3.43 and 4.26%, respectively, while a group exposed for 24 hrs to soil freshly spiked with PCBs exhibited a dermal absorption value of 4.07%.

Mayes et al. (2002) stated that the reduction in fractional absorption compared to the Wester et al. (1993b) study was due to greater soil content of organic matter, which absorbs highly lipophilic compounds such as PCBs. However, the dermal load of 42 mg/cm^2 used by Mayes et al. likely exceeded monolayer coverage and caused a reduction in fractional absorption. No statistically significant difference was observed between the 12- and 24-hr exposure groups, suggesting PCBs partition quickly into lipid components of the stratum corneum. Likewise, aging of PCBs in soil had no effect on dermal absorption, suggesting rapid binding to the organic fraction of soil. The authors noted that Aroclor 1260 has a slightly higher octanol/water partition coefficient (log K_{ow}) than Aroclors 1242 and 1254 used by Wester et al. (1993b). A higher log K_{ow} would favor greater dermal absorption. However, the higher percentage of congeners with seven or more chlorines in Aroclor 1260 compared to Aroclors 1242 and 1254 tends to reduce dermal absorption, as shown by Garner and Matthews (1998).

The dermal absorption of radiolabeled 3,3',4,4'-tetrachlorobiphenyl (TCB) from liquid and soil mixtures was studied in an ex-vivo Yorkshire-Landrace pig-skin-flap model (Qiao and Riviere, 2000). The soil was described as a dust containing 31.2% sand, 16.8% silt, 53.0% clay (90% kaolinite) and 0.3% organic matter. No particle size fractionation was given. Sixty-five to 70 mg soil containing 200 µg of ^{14}C-TCB (40 $\mu g/cm^2$) was applied onto 5 cm^2 skin surface for 8 hrs, and the area was either left open (non-occlusive) or closed with Parafilm (occlusive). Greatest dermal absorption of TCB occurred from non-occluded soil. Fractional penetration of skin into the perfusate was 0.66%, absorption into dermis and other local tissues excluding stratum corneum was 2.48%, and stratum corneum absorption was 0.90%. Occlusion of the soil mixture significantly decreased dermal absorption 2-3-fold. In addition, dermal absorption from the liquid formulations (acetone, water-acetone mixture, or methylene chloride) was also significantly lower, suggesting TCB dermal absorption data from liquid formulations may considerably underestimate the risk of exposure to TCB in a soil matrix.

Qiao and Riviere (2001) performed a full mass balance in vivo study in Yorkshire-Landrace pigs after iv and dermal exposure to identical doses of 300 µg ^{14}C-TCB. For dermal exposure, TCB in acetone vehicle was applied to a 7.5 cm^2 abdominal area of three pigs and protected by a glass chamber with holes, followed by covering with a nylon sieve screening. Urine and feces were collected for 11 days, with quantitative tissue analysis and tape stripping of the

TCB-exposed dermal region conducted at the end of the 11 day exposure. On average, about 70-71% of the applied dermal and iv doses were recovered. After iv dosing, a total of 60% of the dose was excreted via urinary and fecal routes with 8% of the initial dose remaining in body tissues. However, when TCB was given topically, the total excretion was only 5% but with a much larger tissue residue of 16%. The fraction of applied dermal dose reaching the systemic circulation was estimated at 22%, with 0.85% of the applied dose in stratum corneum following tape stripping of the TCB-exposed skin.

Because of the higher tissue residue levels following dermal absorption of TCB, the researchers noted that dermal absorption of chemicals similar to TCB may be underestimated without a full mass balance analysis (Qiao and Riviere, 2001). In other words, estimating dermal absorption by comparing urinary excretion or blood AUC data with data obtained by the iv route (which represents 100% absorption) would underestimate actual TCB dermal absorption. Use of these indirect methods of absorption would provide a calculated dermal absorption of 6.3-10%.

In addition to their in vivo monkey study described above, Wester et al. (1993b) also estimated in vitro dermal absorption of PCBs through human skin from soil. The percent dose penetrating to the receptor fluid after 24 hr exposure was 0.04% for both Aroclor 1242 and Aroclor 1254. The percent dose absorbed in skin was 2.6% for Aroclor 1242 and 1.6% for Aroclor 1254. The low in vitro dermal absorption compared to their in vivo monkey study results was thought to result from tissue viability issues or solubility limits with receptor fluid. However, in vitro dermal absorption and penetration using water as the vehicle resulted in a fractional absorption of 44-46% for both PCB formulations.

The dermal absorption of purified TCB from soil was studied in rat and human skin in vitro (USEPA, 1992). The soil was comprised mostly of silt with an organic carbon content of 0.45% and a particle size range within 0.05-2 mm. The TCB concentration in the soil was 1000 ppm and soil loading was 10 mg/cm^2 for the rat skin and 6 mg/cm^2 for the human skin. After 96 hours, 7.10% of the applied dose had penetrated the human skin into the perfusate, with another 0.26% remaining in skin after washing. In comparison, total dermal absorption in rat skin was over 4-fold higher. A similar experiment was conducted with rat skin in vitro using a soil with a high organic carbon content of 11.2%. Total dermal absorption of TCB was reduced over 3-fold compared to total absorption from the low organic carbon soil.

Dermal absorption of PCBs was estimated by the disappearance method in a single volunteer exposed to a mixture of ^{13}C-labeled tetra-, penta-, hexa-, and heptachlorobiphenyls (Schmid et al., 1992). Five mg of the PCB mixture were applied to a 4 cm^2 cotton cloth in methylene chloride vehicle and dried. The cotton cloth was then applied to the tip of the forefinger or inner side of the forearm without occlusion for 8 hrs. After recovery of PCBs from the carrier and skin surface, disappearance of the remaining label suggested dermal absorption

was 7 and 47% of total dose applied to finger and forearm, respectively. However, plasma concentrations of ^{13}C-label were at or below the limit of detection (10-20 pg/ml) and were not considered reliable. Application of PCBs to aluminum foil, then rubbed into the skin of the forearm for 10 min, resulted in a fractional absorption of 8% by the disappearance method and a plasma concentration of 56.3 pg/ml. The authors suggested that the lack of measurable serum levels of PCBs was partly due to evaporative loss during exposure.

Dermal absorption of HCB in vivo and in vitro was investigated in young (33 days of age) and adult (82 days of age) female rats (Fisher et al., 1989). Young rats absorbed 3.37 times as much HCB dermally as adults in the first 6 hrs of exposure. This resulted from a lag time for penetration of about 1 hr in young and 4 hrs in adult rats. At 72 hrs in vivo dermal penetration was 35% in young and 26% in adults compared to 1.5% for young and 1.0% for adult as measured with a continuous flow in vitro system, and 2.9% for young and 1.9% for adults as measured with a static in vitro system. By 120 hrs both young and adult rats have the same cumulative dermal absorption.

F.4.1.2 Discussion and Recommendation for a Polychlorinated Biphenyl ABS

The Wester et al. (1993b) study provided the highest fractional dermal absorption value (14%) for PCBs in soil among the in vivo experimental animal species considered most relevant for human exposures (i.e., monkey and pigs). Similar to the Wester study, Mayes et al. (2002) used Rhesus monkeys to estimate dermal absorption of PCBs, but obtained fractional absorption values of only 3-4%. Suggested reasons for the lower value include a greater proportion of highly chlorinated congeners, which reduce absorption. However, this may not be an issue because Wester observed similar fractional absorption values using an Arochlor (1242) dominated by tri- and tetra-congeners, and an Arochlor (1254) dominated by penta- and hexa-congeners. Use of a soil with higher organic carbon content may have also resulted in a lower fraction absorption. Additionally, Spalt et al. (2009) notes that Mayes et al. probably exceeded monolayer coverage during the experiment, whereas Wester et al. did not.

The Wester et al. and Mayes et al. studies also used an indirect mass balance adjustment for dermal absorption by comparing excretion of dermally-applied PCBs to excretion of iv administered PCBs. Qiao and Riviere (2001) showed that this may underestimate dermal absorption up to 2- to 3-fold due to greater organ and tissue content of PCBs following dermal absorption compared to PCBs that were injected by the iv route. Thus, the highest absorption fraction estimate (14%) by Wester et al. (1993b) is recommended as the best health protective value.

Wester et al. (1993b) did not age the PCBs in soil prior to dermal application on the monkeys. However, Mayes et al. (2002) observed that aging of PCBs in soil did not reduce dermal absorption compared to freshly spiked soil.

In vitro dermal absorption studies were not considered for estimating the ABS. Comparison studies applying PCBs both in vivo and in vitro suggest that estimating dermal fractional absorption with an in vitro system would underestimate dermal absorption obtained by in vivo methods (USEPA, 1992; Wester et al., 1993b). A reason for this underestimation may be the limited lipophilicity of the receptor fluid used with the in vitro systems.

F.4.2 Polychlorinated Dibenzo-p-dioxins and Dibenzofurans

"Dioxin" emissions are reported as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) equivalents. Therefore, for purposes of the Hot Spots program, all polychlorinated dibenzo-p-dioxins and dibenzofurans are considered to have the same dermal absorption characteristics as TCDD.

Recommended point estimate for dermal uptake from soil: 3%

F.4.2.1 Studies Considered

A. Key Studies

The dermal absorption of TCDD from high organic (HOS) and low organic (LOS) soils in rats in vitro, and in human skin in vitro and rats in vivo from LOS only, was investigated during exposure intervals up to 96 hours (U.S. EPA, 1992; Roy et al., 2008). The LOS was comprised mostly of silt with an organic carbon content of 0.45% and a particle size range within 0.05-2 mm. For the in vitro studies, the TCDD concentration in the LOS was 1 ppm with soil loading of 10 mg/cm^2 on the rat skin and 6 mg/cm^2 on the human skin. After 24 hrs, 0.28% and 1.17% of the applied dose had penetrated human and rat skin, respectively, to the receptor fluid (Table F-3). Although the dose of TCDD remaining in skin was not determined at 24 hrs, the 96 hr exposure estimate in human and rat skin following skin surface wiping was 0.17 and 1.41%, respectively. The percent of applied dose reaching the receptor fluid at 96 hrs was 2.25% in human skin and 6.32% in rat skin.

The percent of dose absorbed from LOS by rats in vivo was 7.9% at 24 hrs and 16.3% at 96 hrs (Table F-3). TCDD absorbed was estimated indirectly by dividing the percent of applied dose found in the excreta by the fraction of applied dose in the excreta at the same time after i.v. administration. However, TCDD systemically absorbed at 96 hrs was also quantified in all urine, feces and tissues, resulting in 16.3% of dose absorbed. To derive an ABS for human *in vivo* uptake of TCDD from LOS (0.45% organic carbon content) and HOS (11.2% organic carbon content), USEPA (1992) applied corrections by direct ratios to account for rat in vivo, rat in vitro, and human in vitro data. For human TCDD absorption from LOS, the in vivo absorption in rat at 24 hrs was multiplied by the ratio of human to rat total absorption in vitro measured at 96 hrs. The 96 hrs data were used because this was the only measurement in which TCDD in skin was quantified. The final ABS was 2.5% (8.0% x 2.42% / 7.74%).

Table F.3. Percent Dermal Absorption of TCDD over Time from Low Organic Soil[a]

Time (hr)	Rat – in vivo	Rat – in vitro	Human in vitro
24	7.9	1.17	0.28
96	16.3	6.32	2.25
96 (Dose in skin sample after wiping)	NA[b]	1.4	0.2
96 (Total)	16.3	7.7	2.4

[a] Data from US EPA (1992) and Roy et al., 2008
[b] Not applicable

Roy et al. (2008) note that steady state conditions for the TCDD concentration in skin from LOS are reached by 24 hours for the in vitro experiments. Thus it should be reasonable to assume that the amount in the skin after 96 hours is about the same as after 24 hours. The researchers also observed that the rat in vivo percent absorbed results were about twice as high as the rat in vitro results after 96 hours. Assuming the human in vitro results would operate in a similar fashion; Roy et al. obtained a human 24-hr fractional TCDD absorption rate of 0.96% (0.48% x 16.3% / 7.7%). Additionally, a fractional absorption value of 0.1% was derived for TCDD absorbed from HOS (soil with an organic content >10%).

Alternately, it may be more relevant to multiply the rat in vivo percent absorbed at 24 hours (7.9%) by the estimated in vitro rat-to-human ratio for total percent TCDD absorbed at 24 hours (0.48% / 2.75%), rather than rely on any of the results from 96 hr exposure. The resulting human 24-hr fractional TCDD absorption rate by this method is 1.4%.

B. Supporting Studies

Shu et al. (1988) applied soil-bound TCDD to the backs of rats, clipped of hair. Laboratory contaminated TCDD soil was prepared from soil obtained from Times Beach MO and determined not to contain TCDD before the experimental addition of the chemical. Environmentally contaminated soil was also obtained from Times Beach, MO and determined to contain 123 ppb TCDD after sieving through a 40-mesh screen. The organic carbon content of the soils was not specified. Soil loading was 20.8 mg soil/cm^2 skin on a total skin area of 12 cm^2. The TCDD content of the laboratory prepared soil was 10 or 100 pg/mg soil. Occlusion of the skin was minimized by the use of a perforated aluminum eye patch to cover the exposed area. Dermal exposure duration to the TCDD-laden soil was 24 hours and recovery was measured 48 hours following initiation of exposure. In some experiments, 0.5 or 2.0 percent (w/w) used crankcase oil was added to the soil before the addition of TCDD.

Following 24 hour dermal exposure + 24 hour post-exposure (total of 48 hours from initiation of exposure), the TCDD content of the liver was determined. The

uptake of TCDD under the experimental protocols ranged from 0.54 ± 0.06 to 1.01 ± 0.22% and averaged 0.76 ± 0.16%. The percent uptake of TCDD in liver was not affected by the applied TCDD dose (12.5 or 125 ng/kg BW), the presence of crankcase oil in the soil, the use of soil that had been environmentally contaminated with TCDD, or by the use of haired or hairless rats.

Peak liver concentrations for TCDD administered orally and dermally were used to correct for incomplete absorption in the calculation of relative dermal absorption. The calculation is based on the assumption that the source of fecal TCDD following oral exposure is unabsorbed TCDD. The estimated relative dermal bioavailability is 1.5% from laboratory-contaminated soil and 1.6% from environmentally contaminated soil.

Diliberto et al. (1996) note that during the first 48 hours following oral exposure, TCDD in rat feces included both unabsorbed TCDD and absorbed TCDD that was excreted in bile. However the data suggest that at 48 hours, absorbed TCDD contributes only about 10% of the fecal TCDD.

Poiger and Schlatter (1980) applied radiolabeled TCDD in a soil/water paste formulation (26, 350, or 1300 ng in 14.3 mg soil/cm^2 skin) to the backs of hairless rats and measured the appearance of label in the livers. The soil (organic carbon content unspecified) was taken from the Seveso region and was TCDD-free. Measurements were taken 48 hours after the initiation of a 24 hour exposure period.

The average percentage of dose in the liver after dermal application was 0.05, 1.7, and 2.2% for the 26, 350, and 1300 ng dose groups, respectively. The authors noted that other researchers observed that 70% of total body burden of administered TCDD is found in the liver of rats. Using this estimate, the corrected dermal absorption of total applied dose is 0.07, 2.4, and 3.1% for the 26, 350, and 1300 ng dose groups, respectively. The authors also compared the liver uptake of dermally applied TCDD from a soil/water paste to the uptake from methanol, and found the soil/water paste caused a reduction in the fractional uptake (compared to methanol) of 12 percent (1.6 ng TCDD/kg BW) or 15 percent (5.8 ng/kg BW).

TCDD in acetone vehicle was applied to human skin in vitro to estimate the capacity of skin to store TCDD (Weber et al., 1991). Although TCDD did not readily penetrate the skin into the saline receptor fluid (0.03% of dose) after 16.7 hrs exposure, a major portion of the dose was found in skin. The percent of dose absorbed in skin at 16.7 hrs was 56% at a skin loading of 65 ng/cm^2, and 40% at a skin loading of 6.5 ng/cm^2.

Age may be a factor in the absorption of TCDD-like compounds. Anderson et al. (1993) applied radiolabeled TCDD in acetone (111 pmol/cm^2 applied over 1.8 cm^2) to the interscapular region of 3-, 5-, 8-, 10-, and 36-week-old rats and measured dermal absorption 72 hrs later. Dermal absorption was greatest in

3-week-old rats at 64%, decreasing to about 40% in 5-, 8-, and 10-week-old rats, and to about 22% in 36-week-old rats. Although the reason for the age-related changes in dermal absorption was not explored, the authors suggested increased lipids in skin of the young may be a factor.

F.4.2.2 Discussion and Recommendation for a Polychlorinated Dibenzo-p-dioxin and Dibenzofuran ABS

Human skin has the capacity to store TCDD in vitro (Weber et al., 1991; Roy et al., 2008). Once absorbed in skin, lipophilic compounds such as TCDD are anticipated to be eventually absorbed into the systemic circulation. Data for another lipophilic pollutant, lindane, indicates that the chemical retained in skin will be eventually systemically absorbed (Dick et al., 1997a).

Several methods for assessing the dermal exposure data by US EPA (1992) and Roy et al. (2008) were employed above to obtain a total fractional absorption (i.e., amount that reached the bloodstream + amount retained in skin) for TCDD ABS. Since the fractional dermal absorption values presented in this document are based on 24-hr exposure, the most relevant means for estimating an ABS is to rely only on the 24-hr absorption results. The resulting human 24-hr fractional TCDD absorption rate by this method is 1.4%. Roy et al. (2008) employ a monolayer adjustment factor in their assessment, noting that the human in vitro skin test used a soil load of 6 mg/cm^2, which was greater than monolayer load by a factor of 2. Multiplying by this factor, the 24-hr TCDD fractional absorption for human skin is estimated at 2.8% for LOS, which is then rounded up to 3%.

Although both Shu et al. (1988) and Poiger and Schlatter (1980) estimated dermal absorption fractions in rats near 2%, neither study specified the organic carbon content of the TCDD-contaminated soil. The organic carbon content of soil is a major determinant for TCDD dermal absorption. At 96 hrs, USEPA (1992) noted that the ratio of TCDD absorption from low organic carbon soil (0.45% organic carbon) in rat skin measured in vitro to absorption from high organic carbon soil (11.2% organic carbon) in the same system was 7.5. Without the organic carbon content of the soil, it is difficult to compare the findings of Shu et al. (1988) and Poiger and Schlatter (1980) with that of the USEPA study.

TCDD aged in soil prior to dermal application had little effect on absorption, which is supported by the long half-life of TCDD in soil. Shu et al. (1988) observed similar dermal absorption estimates when TCDD was freshly added to soil in the lab and soil that had been environmentally contaminated with TCDD and presumably aged in the soil. In addition, soil aging of polychlorinated biphenyls (PCBs), a group of soil contaminants with some structural similarities to TCDD, is not a significant factor for dermal absorption (Mayes et al., 2002). On the other hand, oral studies of soil-laden TCDD do indicate aging to be factor in the reduction of TCDD intestinal absorption (Poiger and Schlatter, 1980).

F.4.3 Polycyclic Aromatic Hydrocarbons as Benzo[a]pyrene (BaP)

Recommended point estimate for dermal uptake from soil: 13%

Field studies of workers have shown that dermal absorption of PAHs may be significant. Dermal absorption of PAHs, based on the urinary excretion of 1-hydroxypyrene (1-HP), has been documented among petrochemical industry workers, including those digging in PAH-contaminated soil (Boogaard and van Sittert, 1995). Although no attempt was made to quantify the extent of absorption through dermal and inhalation routes, the results of the study strongly suggest dermal uptake is substantial and is mitigated by the use of appropriate protective clothing. Elovaara et al. (1995) compared the levels of urinary 1-HP among 6 creosote workers compared to that expected from the inhalation of the known air levels of PAHs containing $\geq$ 4 rings. Higher levels of urinary 1-HP were observed than could be accounted for solely from the inhalation route of exposure.

F.4.3.1 Studies Considered

A. Key Study

In Wester et al. (1990b), the dermal uptake of soil-bound BaP was studied in vivo in four rhesus monkeys. The systemic absorption of soil-bound BaP was based on urinary excretion following exposure of 12 cm^2 abdominal skin to 10 ppm BaP in soil at a soil loading of 40 mg/cm^2 skin. A nonocclusive cover protected the dermal application site. Prior to sieving to approximately 180-320 µm diameter, the soil composition was 26 percent sand, 26 percent clay, and 48 percent silt with 0.9 percent organic carbon content.

Exposure duration to the chemical laden soil was 24 hours, during which time urine was collected. The cover was removed, visible soil was collected, and the skin application site was washed with soap and water. Urine was then collected for 6 additional days for a cumulative recovery period of 7 days. Incomplete excretion of BaP was corrected by the urinary excretion of BaP following intravenous (iv) administration of the PAH in acetone. The authors report a mean 24 hour dermal absorption factor of 13.2 $\pm$ 3.4 percent (Table F.4).

Radiolabeled BaP (^{14}C-BaP) was mixed with commercial gardening soil and applied in vitro onto fresh human female breast skin (obtained within 1 day of harvest) for 24 hrs by means of Bronaugh diffusion cells (Moody et al., 2007). The same amount of ^{14}C-BaP was also applied without soil to human skin samples. The soil had been sieved to <710 µm prior to spiking with BaP. The soil mixture (3.2 mg soil) was added to the diffusion cells resulting in a soil loading of 5 mg/cm^2. At 24 hrs, the mean total percent dermal absorption including the skin depot was 14.8 and 56.4% with and without soil, respectively. The fraction of total absorbed BaP that entered the diffusion cell in 24 hrs was 7.2 and 11% with and without soil, respectively.

B. Supporting Studies

Yang et al. (1989) studied the in vivo systemic absorption in rats of BaP in soil, fortified with petroleum crude oil (1 percent (w/w)) to which ^{3}H-BaP was added. The soil, which consisted of 46 percent sand, 18 percent clay and 36 percent silt, with an organic content of 1.6 percent, was sieved to a particle size <150 µm. The final BaP level in the soil was 1 ppm and the soil loading was 9 mg/cm^2.

After 24 hours, 1.1 percent of the radioactive label was found in the rat urine and feces; no label was found in the tissues. By 96 hours (4 days) the cumulative total of radioactive label in the excreta + tissues was 9.2 percent, of which 5.8 percent was in the feces. The dermal uptake rate was estimated to be 0.2 ng/cm^2/day. Remaining BaP retained in skin at the site of application was not determined. In vitro absorption of BaP in soil was also determined in rats using a similar exposure protocol. Very good correlation was observed between the in vivo and in vitro data.

In conjunction with the in vivo dermal absorption studies in monkeys, Wester et al. (1990b) also conducted BaP dermal absorption experiments with viable human skin in vitro. Under the same soil and loading conditions of the in vivo monkey study, BaP-laden soil was applied to skin samples (dermatomed to 500 µm thickness) for 24 hrs. The percentage of applied dose in skin and in human plasma receptor fluid was 1.4 and 0.01%, respectively. When acetone was used as the vehicle under the same exposure conditions, BaP found in receptor fluid and in skin was 0.09 and 23.7% of applied dose, respectively.

Dermal absorption of ^{3}H-BaP from two different soils was determined in vitro through dermatomed pig skin cut 200 µm thick (Abdel-Rahman et al., 2002). Soil types included a sandy soil with 4.4% organic matter and a clay soil with 1.6% organic matter. Skin applications included: BaP applied as the pure compound; BaP applied immediately after the addition to each soil type (30 mg each); and pre-sterilized soils aged for three months with BaP. The chemical dose was 1.67 mg/kg and the soil loading was calculated to be 47 mg/cm^2.

Following 16 hrs of exposure, 0.2% of freshly applied BaP in sandy soil penetrated the skin to receptor fluid and 8.3% was found bound to skin. In clay soil, 0.1% of freshly applied BaP was found in the receptor fluid and 3.3% was bound to skin. In comparison, pure BaP applied to skin resulted in 0.2 and 75.8% of the dose found in receptor fluid and bound to skin, respectively. For BaP aged in either sandy or clay soil, 0.1% was found in the receptor fluid. Only 3.7 and 1.7% were bound to skin from sandy and clay soil, respectively. Aging BaP in the soils for three months decreased total dermal adsorption by about 2-fold compared to BaP freshly applied to the soils.

Table F.4. In Vivo and In Vitro Dermal Absorption Results of Pure BaP Freshly Applied or Aged in Soils

Study	Species Treatment	Exposure time (hr)	Soil fraction (µm)	% Total absorbed fresh	% Total absorbed aged
Wester et al. 1990b	monkey in vivo	24	180-320	13.2	ND [a]
Yang et al., 1989	rat in vivo	96	<150	9.2	ND
Moody et al., 2007	human in vitro	24	<710	14.8	ND[c]
Wester et al., 1990b	human in vitro	24	180-320	1.4	ND
Abdel-Rahman et al., 2002	pig in vitro	16	unsieved	8.5[b] 3.4[c]	3.8 [b] 1.8 [c]

[a] Not determined
[b] Sandy soil
[c] Clay soil

Studies were conducted to measure in vitro absorption of BaP through human skin (previously stored frozen) from contaminated soils at manufactured gas plant (MPG) sites. These sites were impacted by PAHs in lampblack, a residue produced from the pyrolysis of oil to produce gas. Roy et al. (1998) collected nine soils from three MPG sites containing targeted PAHs at levels ranging from 10 to 2400 mg/kg. Dermal penetration rates of target PAH from the soils were determined using ^{3}H-BaP as a surrogate. Soils were sieved to <150 µm prior to analytical characterization and loaded onto skin sections at 25 mg/cm^2. Dermal absorption tests ran up to 144 hrs. The recovery of radiolabel in the receptor fluid ranged from 0.19 to 1.0%, while radiolabel absorbed in skin ranged from 0.4 to 1.0%. The highest percent of applied dose (receptor fluid + skin) from a contaminated soil was 1.9%.

Contaminated soils were collected from 7 oil-gas MPG sites in California to assess dermal absorption of BaP in vitro (Stroo et al., 2005a; Stroo et al., 2005b). The soil was sieved to <150 µm and loaded onto human skin at 10 mg/cm^2. The skin samples were dermatomed to a thickness of 350 µm. The percentage of applied dose absorbed across skin over 24 hrs ranged from 0.14 to 1.05%. The lower absorption of BaP in the lampblack samples compared to the Wester et al. (1990b) study was attributed to soil aging effects, but also to tighter binding of BaP to lampblack. Lampblack tends to bind hydrocarbons more tightly then conventional soil organic matter.

To investigate effects of soil loading and aging on PAH dermal absorption, Roy and Singh (2001) loaded PAH-spiked soil onto human skin sections at 1, 2.5, 5 and 10 mg/cm^2 following aging of the PAHs in soil up to 110 days. A field soil was sieved to <150 µm, resulting in a total organic content of 0.43%. The soil

was spiked with coal tar and ^{3}H-BaP to achieve a final soil BaP concentration of 65 ppm. At soil loadings of 1 and 2.5 mg/cm^2, approximately 1% of the applied dose was in the receptor fluid at 24 hrs. The percent of applied dose absorbed decreased with increasing soil loadings of 5 and 10 mg/cm^2, respectively, indicating skin loading above monolayer coverage. In the aging experiment, the dermal bioavailability of coal-tar-derived BaP was reduced by about half by day 110 compared to the soil freshly spiked with ^{3}H-BaP.

The in vitro dermal absorption of BaP applied in acetone to full-thickness skin was compared among six mammalian species (Kao et al., 1985). The percent of applied dose permeating fresh, viable skin in 24 hrs was approximately 10% in mice, 3% in marmosets and humans, 2% in rats and rabbits, and <1% in guinea pigs. However, permeation through skin rendered non-viable by previous freezing was <1% of applied dose in all species. Permeation was accompanied by extensive first-pass metabolism of BaP in viable skin of all species. Nearly half the BaP that permeated viable human skin was attributed to BaP metabolites. In non-viable skin, essentially only unchanged BaP was detected in the receptor fluid.

PAHs have been shown to be poorly absorbed through skin from solids. No percutaneous penetration of PAHs from coal dust occurred across human skin in vitro (Sartorelli et al., 2001).

F.4.3.2 Discussion and Recommendation for a Polycyclic Aromatic Hydrocarbon ABS

A fractional dermal absorption of 13% determined in a primate species in vivo represents a health-protective estimate of human systemic absorption of pure BaP freshly applied to an agricultural soil (Wester et al., 1990b). In support, a similar in vitro fractional absorption (14.8%) was attained by Moody et al. (2007) for 24-hr exposure of human skin to BaP-contaminated soil. The work by Wester et al. and Moody et al. were also one of the few BaP exposure studies that did not exceed monolayer soil coverage of the skin, although the coarse particle soil loadings used in the monkey study may have resulted in a lower fractional absorption.

The only other in vivo study of BaP dermal absorption from soil was in rats, in which a lower fractional absorption of 9.2% was estimated after 4-day exposure (Yang et al., 1989). Although higher organic content of the soil used could be a factor in the lower ABS in rats, the presence of petroleum crude oil (1 percent (w/w)) as a co-contaminant was also likely a factor in the lower absorption in rats compared to monkeys. Stroo et al. (2005a) note that tar in contaminated soils tends to bind hydrocarbons more tightly than conventional soil organic matter and reduces bioavailability for dermal absorption. In addition, a soil loading of 9 mg/cm^2 exceeds monolayer coverage with soil sieved to <150 µm causing a further reduction in the percent fractional absorption.

Wester et al. (1990b) observed a roughly 10-fold lower fractional absorption of BaP in human skin in vitro compared to the human in vitro study by Moody et al. (2007). Use of a course soil fraction (180-320 µm) by Wester et al. may have reduced dermal absorption. The reduction in absorption may also be due, in part, to loss of skin viability. The Wester study used cadaver skin up to 5 days after harvest. The studies of Moody et al. obtained human skin in as little as 2-24 hrs after live donor skin harvest.

The metabolic viability of the skin samples used for in vitro studies is a factor that can affect skin permeation of BaP. Kao et al. (1985) have shown that the rate of cutaneous metabolism of BaP has a positive correlation with the permeation rate of BaP through viable skin. For example, using previously frozen human skin, as was done in some studies discussed above, renders the samples less viable and possibly much less permeable to BaP. When BaP was applied in vitro to fresh skin samples and previously frozen skin from the same individuals, a significant reduction in dermal absorption into the receiver solution was observed for the previously frozen skin (Moody et al., 2009a). However, when the skin depot was included, the difference in dermal absorption between fresh and previously frozen skin was not as pronounced.

The dermal exposure algorithm presented in Chapter 6 includes a half-life variable for BaP in soil, although it is generally assumed the half-life reflects primarily the loss of chemical due to microbial degradation. However, Adbel-Rahman et al. (2002) showed that aging of BaP in sterile soil also resulted in decreased fractional absorption in pig skin. This finding suggests BaP also shows reduced bioaccessibility over time due to partitioning into more remote sites within the soil matrix. Vigorous soil extraction procedures often used to assess soil half-life may overestimate the bioavailability of BaP because it may not be a true representation of BaP's bioaccessibility in soil for dermal absorption. Extraction techniques using human sweat or synthetic sweat would provide a more accurate estimate of the BaP half-life in soil for fractional dermal absorption studies.

F.4.4 Hexachlorobenzene

Recommended use of default organic compound ABS estimate of 4%

F.4.4.1 Studies Considered

No experimental data are available investigating the dermal absorption of HCB from contaminated soil. In a rat in vivo study, ^{14}C-HCB dissolved in tetrachloroethylene was applied neat to the skin and covered with an occlusive patch after the vehicle had evaporated (Koizumi, 1991). The cumulative mean absorbed body burden, not including dosed skin directly contaminated, was 2.67% after 24 hours. Approximately 5% of the total dose remained in or on the dosed area of skin prior to washing. Washing the dosed area of skin resulted in

removal of 4% of the total dose, indicating that 1% of the total dose was absorbed in the skin on which ^{14}C-HCB was directly applied.

A Monte Carlo simulation was developed to produce a probability density function for the dermal uptake fraction of HCB in soil deposited on human skin (McKone, 1991). A two-layer model was used that accounted for chemical properties, skin properties, soil properties, and exposure conditions. The resulting modeled daily dermal uptake fraction had an arithmetic mean value of 0.15 per day (24 hrs), and an arithmetic standard deviation of 0.18 per day.

F.4.4.2 Discussion and Recommendation for a Hexachlorobenzene Compound ABS

A single dermal absorption study in rats observed a 24-hr fractional absorption of 4% (rounded to nearest whole number) for the neat compound. This estimate includes HCB retained in skin at the site of application. Absorption of HCB may have increased as a result of occlusion of the exposed skin area to prevent evaporation of HCB.

A default ABS of 4% is recommended based on the rat dermal exposure study, although the chemical was applied neat to the skin. An HCB modeling study suggests that the fractional absorption of HCB in soil may be 15%, so no adjustment was made to the ABS to account for reduced absorption due to partitioning to soil organic matter (McKone, 1991). In support, HCB is structurally similar to hexachlorocyclohexane (HCH), which has an ABS of 3%. However, the K_{ow} for HCB (log K_{ow} 5.73) is about 100 times greater than that of the HCHs, which would suggest a greater ability for absorption into skin. On the other hand, the high K_{ow} also indicates that HCB will have stronger sorption to soil organic material compared to the HCHs, which usually decreases the dermal absorption potential. Until more relevant dermal absorption studies are conducted, an ABS of 4% is recommended for HCB.

F.4.5 Hexachlorocyclohexanes

Hexachlorocyclohexanes (HCHs) occur as eight isomers. The most common isomer is the gamma, which when purified to 99%, was sold under the trade name of lindane. Lindane was a widely used pesticide but almost all uses of lindane have been banned in the United States due to carcinogenicity concerns, high biopersistence and bioaccumulation. Dermal absorption data exist only for lindane, thus all HCH isomers are considered to have the same dermal absorption characteristics as lindane.

Recommended point estimate for dermal uptake from soil: 3%

F.4.5.1 Studies Considered

A Key Study

The only study located regarding dermal absorption of HCHs from soil was that of Duff and Kissel (1996) who conducted in vitro dermal absorption studies using human full-thickness skin and two lindane-contaminated soils. The organic content of the sieved sub-150 µm soils were 3.87% (sandy loam) and 0.73% (silt loam). The lindane-spiked soils were stored for up to 19 days prior to testing. No effect of aging was observed within this time frame. The studies were carried out for 24 hours with soil loading at 1, 5 or 10 mg/cm^2. The relative percent absorption decreased significantly with soil loads of 5 and 10 mg/cm^2. This was attributed to monolayer coverage of skin occurring at about 2 mg/cm^2, resulting in reduced fractional absorption at the higher soil loadings.

Results of this study showed that most of the mass of absorbed lindane was found in the skin. The average fraction of total dermal uptake found in the receptor fluid for both soils was only about 4%. Mean 24-hour total dermal absorption values (found in receptor fluid + skin) at a soil load of 1 mg/cm^2 was 1.96 and 2.35%, for low and high organic content soil, respectively. Approximately 40% of the lindane was lost to volatilization with a soil load of 1 mg/cm^2, while significantly lesser amounts were lost in the higher loading trials (less than 10% for the sandy loam soil at 10 mg/cm^2; less than 20% for the silt loam soil at 10 mg/cm^2).

B Supporting Studies

Feldman and Maibach (1974) examined the percutaneous absorption of lindane dissolved in acetone and applied to the skin of human subjects (n = 6). Radiolabeled lindane (4 µg/cm^2) was applied to ventral forearm skin and the urinary excretion of ^{14}C was measured for 5 days after the single topical application. The skin sites were not protected and subjects were asked not to wash the area for 24 hours. Data obtained after i.v. dosing were used to correct the skin penetration data for incomplete urinary recovery. Results indicate that 9.3% (SD 3.7) of the dose was absorbed. However, when skin was occluded, the percent of absorbed dose increased dramatically to 82.1%.

In another human study, lindane was dissolved in acetone and applied to the ventral forearm of volunteers and covered with a nonocclusive patch (Dick et al., 1997a). Six hours after application approximately 80% of the applied lindane dose (120 mg lindane per ml acetone) had not been absorbed and 14% of the dose was found in the stratum corneum (measured by tape-stripping). The authors conclude that 5% of the applied dose was absorbed to the systemic circulation by 6 hours. Although the disappearance method was used to estimate systemic absorption, measurable levels of lindane were found in the bloodstream and lindane metabolites were found in the urine. By 24 hours, tape stripping of the remaining volunteers showed the stratum corneum contained

very little of the applied lindane and only about 0.01% of the dose had been lost through desquamation, suggesting that nearly all the lindane detected in the stratum corneum at 6 hours had been systemically absorbed or absorbed into deeper skin layers by 24 hrs.

F.4.5.2 Discussion and Recommendation for a Hexachlorocyclohexane ABS

Although only one study for dermal absorption of lindane from soil is available, the findings provided consistent results for a human in vitro fractional absorption range of 0.45 to 2.35% under different soil loadings and soil types (Duff and Kissel, 1996). The highest fractional absorption of 2.35% was chosen as the basis for the HCH ABS, given that the soil loading (1 mg/cm^2) used was the only one that was at or below monolayer skin coverage. An average of only 4% of the absorbed dose (approximately 0.09% of the applied dose) was found in the receptor fluid after 24 hrs. However, in vivo studies show extensive absorption of lindane into all skin layers, with continued absorption of lindane beyond the stratum corneum 6 hrs after removal of lindane from the skin surface (Dick et al., 1997a). Thus, lindane retained in skin depots should be presumed to be available for eventual systemic absorption.

Duff and Kissel (1996) noted the unexpected result that the soil with the higher organic carbon content generated a higher fractional absorption (2.35%) than the soil with low organic carbon content (1.96%) at equivalent soil loadings of 1 mg/cm^2. Increasing organic carbon content of soil generally reduces transport, and dermal absorption, of organic compounds in soil. The authors theorized that this inconsistent finding at 1 mg/cm^2 was due to inter-individual differences in skin absorption, which would not have occurred had the same skin donors been used for both soils.

To account for known effects of organic content of soil the ABS of 2.35% is rounded up, rather than down, to one significant figure for a final ABS of 3%. In support of this ABS adjustment, soil loadings of 5 and 10 mg/cm^2 from high organic content soil did reduce fractional absorption of lindane compared to lindane in soil with low organic content (Duff and Kissel, 1996). However, monolayer coverage of skin was exceeded at these higher soil loads, resulting in lower fractional absorption compared to fractional absorption at 1 mg/cm^2.

Other data available on percutaneous absorption of lindane or other HCH isomers, which are obtained from studies that use acetone or topical creams and lotions as the vehicle, are not relevant for estimating fractional absorption of lindane from soil (Franz et al., 1996). Use of topical creams and lotions as a vehicle for lindane in dermal absorption studies is related to lindane's use as a medicine to treat scabies.

Theoretical calculations in which release from soil is not the primary limiting factor in the dermal absorption of lindane predict the percent absorbed at 55.6 to 98.5% (Bunge and Parks, 1997). The upper end of this range brackets the

82.1% absorption of applied dose observed by Feldman and Maibach (1974) when the vehicle is acetone and evaporation of lindane is limited by occlusion. However, the lower dermal absorption of lindane from soil observed by Duff and Kissel (1996) is consistent with the theory of slow soil release kinetics, in which partitioning from soil to skin is the limiting factor in dermal absorption for a number of organic compounds (Bunge and Parks, 1997). Oral bioavailability data for absorption of lindane from soil support the dermal data for absorption of lindane from soil. Soil (organic matter content of 9.8%) spiked with lindane and aged was found to have an oral bioavailability of only 7.2% in an in vitro gastrointestinal extraction test (Scott and Dean, 2005).

The dermal exposure scenario used in this document assumes that deposition of contaminated soil occurs on non-occluded skin exposed to the environment. These conditions would promote evaporation of lindane from soil on the skin, resulting in less absorption into skin than might be expected (Wester and Maibach, 1985; Duff and Kissel, 1996). A potential limitation of this ABS is if significant dermal deposition of lindane-contaminated soil occurs on skin under clothing. The situation may then become one of a reservoir for lindane in which enhanced dermal absorption occurs because of limited evaporation. However, the volatilization potential for lindane from soil also suggests that the absorption potential for lindane may be more significant when exposure is from excavated soils or from surface soils soon after the contamination event (Bunge and Parks, 1997). These various countervailing influences on dermal absorption of lindane under the exposure scenario support the assumption that the ABS will not underestimate actual dermal absorption.

F.4.6 Diethylhexylphthalate (DEHP)

Recommend point estimate for dermal uptake from soil: 9%

F.4.6.1 Studies Considered

A Key Studies

No studies were located on dermal absorption of di(2-ethylhexyl)phthalate (DEHP) from soil.

Deisinger et al. (1998) estimated the migration and subsequent absorption of radiolabeled DEHP from polyvinyl chloride film into rat skin in vivo. Based on the amount of DEHP that migrated from film (505.6 mg) with 24 hr dermal exposure, systemic absorption was estimated at 3.4% of the migrated dose. After skin washing, the residual fraction in skin at the site of dermal application was 13.8% of the migrated dose. Assuming the fraction of DEHP in skin will be eventually absorbed systemically, a maximum absorption rate of 0.24 μg/cm^2/hr was calculated.

Barber et al. (1992) carried out an in vitro DEHP dermal exposure study to compare rates of absorption through full thickness rat skin and human stratum

corneum. DEHP was applied to skin samples in saline solution, and absorption expressed in terms of absorption rate after 32 hrs of exposure. Absorption through rat skin and human stratum corneum was 0.42 and 0.10 μg/cm^2/hr, respectively, indicating that DEHP more rapidly penetrated rat skin than human stratum corneum by a factor of 4.2.

Damage to the rat skin observed following exposure was implied as a possible reason for greater permeability of DEHP through rat skin. Scott et al. (1987) compared absorption rates of DEHP through rat and human epidermal membranes (dermal layer removed), obtaining rates of 2.24 and 1.06 μg/cm^2/hr for rat and human skin, respectively. DEHP was applied to the skin sample in 50% v/v aqueous ethanol with exposure up to 53 hrs for rat skin and 72 hrs for human skin. Damage to rat skin, but not human skin, was also observed by Scott et al. (1987) after exposure.

B Supporting Studies

The National Toxicology Program investigated the dermal absorption of ^{14}C-labeled DEHP in male F344 rats (Melnick et al., 1987; Elsisi et al., 1989). The labeled compound was dissolved in ethanol and applied directly to the skin (30 mg DEHP/kg body weight; n = 3 per time point) at a dose of 5-8 mg/cm^2. The ethanol was then evaporated and the site of application was covered with a perforated plastic cap. DEHP showed a very slow rate of excretion over five days, likely reflecting a slow dermal uptake process. After five days, approximately 86% of the applied dose was recovered from the skin at the site of application. However, it was not determined how much of the applied dose remained on the surface of the skin and how much was absorbed into the skin. Approximately 5% of the applied dose was recovered in urine and feces, while the amount of the label remaining in the body five days after dosing was less than 2% of the applied dose of DEHP.

Ng et al. (1992) examined dermal absorption of DEHP both in vivo and vitro in hairless guinea pigs. In an in vivo study, radiolabeled DEHP dissolved in acetone (53 μg DEHP; 34 nmols/cm^2) was applied topically on a dorsal area of the animals which was then covered with a nonocclusive patch. After 24 hours, the patch was removed and the dosing site cleaned to remove any unabsorbed compound. Absorption (estimated from urine and feces) was monitored up to 7 days post treatment. To account for incomplete excretion after the compound was absorbed, a dose of ^{14}C-DEHP was given intramuscularly to a group of animals (n=5) and radioactivity was measured in urine and feces for up to seven days.

After 24 hours, 3% (7% after correction) of the dermally applied dose was eliminated in urine and feces. After seven days, approximately 21% (53% after correction) of the dose had been absorbed by the skin and eliminated, while another 11.3% of the dose had been skin stripped from the dose area. An additional group (n=6) of animals was given DEHP (53 μg) dermally to estimate

the dose remaining in the tissues. After 7 days, ^{14}C content (% of applied dose) was as follows: urine, 18 ± 4; feces, 4 ± 1; skin wash after 24 hrs, 32 ± 10; skin patch, 13 ± 5; skin (dosed area), 5 ± 3; other tissues (liver, fat, muscle, skin), 4 ± 3%. An additional 10% was estimated to be lost to volatilization.

In the in vitro study, Ng et al. (1992) examined absorption of DEHP through viable and non-viable dermatomed guinea pig skin (200 µm sections) with 24-hr exposure. Radiolabeled DEHP was applied in 10 µl acetone at concentrations of 35.6, 153, or 313 nmol/cm^2. The percentage of dose that permeated the viable skin into the receptor fluid was 6, 2.4, and 2.5% for the low-, medium-, and high-dose groups, respectively. The percentage of dose that remained in the skin disc was 41.0, 37.5, and 36.2% for the low-, medium-, and high-dose groups, respectively. Use of nonviable skin resulted in a slightly decreased penetration of 5.0% at the applied dose of 35.6 nmol/cm^2, likely due to decreased metabolism of DEHP. There was a dose-related increase in metabolism but the total metabolites were between 0.5 and 1% of the applied dose for each dose group.

Chu et al. (1996) examined the skin reservoir effects of ^{14}C-labelled DEHP (119-529 µg/cm^2) applied on hairless guinea pigs for 24 hrs, followed by washing of the skin to remove DEHP and analysis of DEHP distribution up to 14 days post-treatment. As DEHP in the dosed skin decreased from 11.1% to 0.66% from 24-hrs to 7 days post-treatment, excreted DEHP gradually increased from 0.74 to 17.3%.

This finding provided evidence that DEHP stored in skin enters the systemic circulation, although the considerable intraspecies variation for percent of absorbed dose precluded a specific estimate of DEHP absorbed systemically after 24 hrs post-treatment. DEHP in the carcass was 1.01 and 0.92% of applied dose at 24 hrs and 7 days, respectively. By 14 days post-treatment, essentially no DEHP remained in dosed skin. Autoradiographic analysis of the dosed skin at 24 hrs revealed dense radiolabel accumulation in the epidermis and along the hair follicles, which indicated hair follicles may be a penetration pathway for DEHP.

The authors also reported that the percent absorbed at 24 hours by Ng et al. (1992) was higher than that found in this study, with nearly identical experimental protocols. They attributed this difference to the higher doses used in the present study (10 times higher when expressed in µg/cm^2) stating that saturation might have occurred at higher doses, resulting in a lower fractional absorption.

F.4.6.2 Discussion and Recommendation for a Diethylhexylphthalate ABS

Although two in vitro dermal absorption studies have been carried out with pure DEHP on human skin, data were not provided to determine ABS values. However, absorption rates were determined for both rat and human skin under similar exposure conditions and compared. The DEHP absorption rate for

humans was 2-4 times less than that for rats (Scott et al., 1987; Barber et al., 1992).

In vivo studies in rats and guinea pigs that determined absorption of DEHP by total mass balance provide the best estimates for fractional dermal absorption in these species. Deisinger et al. (1998) used PVC film as the vehicle for transfer of DEHP to the skin of rats. Using PVC film as the vehicle will slow absorption, as DEHP requires transfer from the film before partitioning into skin can occur. This type of chemical transfer may give a closer estimate of a DEHP ABS from soil, compared to skin application of the pure compound as performed by the other studies. Including both systemic absorption and compound in skin at the site of application, a fractional dermal absorption value of 17.2% is attained from the Deisinger study. The rat-to-human absorption rate ratio of 2.1 determined by Scott et al. (1987) is then applied to give a final ABS of 9% (rounded up from 8.6%).

DEHP in the skin is included in this estimate, as Ng et al. (1992) and Chu et al. (1996) found there is significant systemic absorption of DEHP in skin up to 7 or more days after removal of DEHP from the skin surface. For this reason, the rat study by Melnick et al. (1987) was not considered in this assessment. The Melnick study did not wash DEHP off the site of skin application prior to analysis, so it is unknown how much DEHP was on or retained in the skin at the end of the 5 day exposure.

Similar to rats, Chu et al. (1996) also noted that guinea pig skin is considered generally more permeable to chemicals than human skin. Thus, it is not unexpected that the rat ABS of 17.2% is within the range of 9.5 to 18.9% (DEHP systemically absorbed + DEHP in skin) determined by the authors in guinea pigs. A limitation for this ABS is that both Ng et al. (1992) and Chu et al. (1996) reported that the percent absorbed in guinea pigs appeared to be higher at low application concentrations, although nearly identical experimental protocols were used. They attributed this difference to possible skin saturation occurring at higher doses (about 119-529 $\mu g/cm^2$), resulting in a lower fractional absorption. If saturation of DEHP in rat skin has occurred in the Deisinger et al. (1998) study, this may result in an underestimation of the fractional absorption value at soil concentrations associated with airborne releases.

Another limitation includes reliance on studies in which DEHP is applied directly onto the skin (i.e., neat), rather than combined with soil, for estimation of fractional dermal absorption. Kissel (2011) has reported that fractional absorption is dependent on skin loading conditions for application of organic chemicals directly to skin. Increased skin loading of an organic chemical will result in lower fractional absorption provided complete coverage of the skin at the site of application occurs. Using PVC film as a surrogate for soil for transfer of DEHP from the film to skin is used in the estimation of the ABS, and thus reduces potential mismeasure of dermal absorption of organic compounds applied neat.

Other limitations include lack of data for dermal absorption of the compound bound to soil was located in the literature. In addition, no oral bioavailability studies for DEHP bound to soil could be found. Thus, no further adjustment of the ABS for absorption from a soil was applied.

F.4.7 Dermal Absorption Fraction for 4,4' –Methylenedianiline

Recommended use of default organic compound ABS estimate of 10%.

F.4.7.1 Studies Considered

Brunmark et al. (1995) utilized a patch-test method to evaluate dermal exposure and pharmacokinetics of 4,4'-methylene dianiline (MDA) dissolved in isopropanol. Measurements of MDA were made in plasma and urine of the five human volunteers. The extent of absorption was evaluated by measuring the amount remaining in the patch after 1 hour. Determination of MDA remaining in the patch showed 25 to 29% was absorbed. The authors also describe elimination half-lives from plasma and urine.

Workers were monitored for two consecutive weeks in a fiber glass pipe factory for dermal exposure to MDA (diluted with triethyleneamine) using both cotton glove and hand wash monitoring (Brouwer et al., 1998). Urinary excretion of methylene dianiline was also evaluated. Urinary MDA levels correlated well with exposure measurements. Geometric means of daily exposure ranged from 81 to 1783 μg MDA, while 24 hour urine samples ranged from 8 to 249 μg MDA. Given that the Brunmark study identified a urinary half-life of MDA of 7 hours and that the measurements on the hands and forearms of the workers correlated strongly (0.94) with the urinary excretion of MDA, one can roughly estimate that between 10 and 14% of the MDA on the hands and forearms was absorbed by the workers.

MDA was applied in vitro to unoccluded human and rat skin for 72 hrs at a loading of 17.7-40.6 μg/cm^2 in ethanol (Hotchkiss et al., 1993). Absorption into the receptor fluid at 72 hrs was 6.1 and 13.0% of the applied dose for rat and human skin, respectively. When the skin was occluded, the absorption at 72 hrs was significantly enhanced, reaching 13.3 and 32.9% for rat and human skin, respectively. MDA that remained in human skin at 72 hrs was 23.8 and 37.4% of the applied dose for unoccluded and occluded skin, respectively. For the rat, MDA content of the skin at 72 hrs was 57.6 and 53.1% of the applied dose for unoccluded and occluded skin, respectively. Although the data were only graphically presented, absorption through human skin into the receptor fluid at 24 hrs can be estimated at 8% of the applied dose for unoccluded skin and 20% of the applied dose for occluded skin.

The permeability of rat and human skin in vitro to MDA was assessed by Kenyon et al. (2004) over a large dose range, and the potential for skin to act as a reservoir for MDA was investigated. Dose levels of 0.01, 0.1 and 1 mg per 0.32

cm^2 skin were applied in ethanol:water (50:50) onto occluded skin for 24 hrs. No statistical difference in skin permeability was observed between rat and human skin. After 24 hrs, 27 to 52% of applied MDA had penetrated human skin to the receptor fluid. The percentage of applied MDA retained in human skin was 20%.

In another in vitro experiment, Kenyon et al. (2004) applied 0.1 mg MDA to human skin for 4 hrs, then removed excess MDA on the skin surface and the experiment continued for another 4 hrs. The cumulative absorption rate of MDA into the receptor fluid remained the same for the last 4 hrs, with only a slight decrease noted between 7 and 8 hrs. Of the total 11% of the MDA found in the skin, 5% was removed by tape stripping the stratum corneum. The remaining 6% of MDA was found in the digested skin, suggesting this amount would have been absorbed had the experiment continued longer. Considering that the lag time for appearance of MDA in receptor fluid was about 4 hrs, the authors presumed that the MDA remaining in the stratum corneum at 8 hrs would not be absorbed systemically.

No literature could be located regarding dermal absorption of MDA from soil. However, the fate of MDA added to soil has been investigated. MDA rapidly and strongly absorbs to loam soil which contained a total organic content of 1.3% (Cowen et al., 1998). However, MDA does not appear to form complexes with humic materials or form other irreversible soil binding processes. In one year, the aerobic biodegradation of MDA in silt loam soil was 40%.

F.4.7.2 Discussion and Recommendation for a 4,4'–Methylenedianiline ABS

Dermal absorption of MDA in workers is considered a more significant route of exposure than inhalation (Brouwer et al., 1998). The in vivo worker data support the in vitro human data in that dermal absorption is considerable. However, the exposure/application of MDA involved other organic solvents. The effect of solvent vehicle on absorption was not investigated.

No data could be located regarding dermal or oral absorption of MDA bound to soil. In addition, no oral bioavailability studies for MDA bound to soil could be located. Soil fate studies indicate that MDA binds strongly to soil, which would likely reduce dermal absorption considerably, and biodegrades slowly over a year's time. Thus, the default absorption value of 10% for organic compounds is recommended until soil-bound dermal studies are available.

F.5 Comparison with Other Published Dermal Absorption Factors

Two other agencies have published fractional dermal absorption estimates for some of the Hot Spots chemicals presented in this document. These values are shown in Table F.5 and are compared with the fractional dermal absorption values developed by OEHHA.

Table F.5. Published Point Estimates and Default Dermal Absorption Factors (ABS) as Percent of Selected Chemicals from Soil

CHEMICAL	ABS (percent)		
	OEHHA[a]	US EPA[b]	DTSC[c]
Inorganic chemicals			
Arsenic	6	3	3
Beryllium	3	[d]	[e]
Cadmium	0.2	0.1	0.1
Chromium (VI)	2	[d]	[f]
Fluoride	3	[d]	[e]
Lead	3	[d]	[e]
Mercury	4	[d]	[e]
Nickel	2	[d]	[e]
Selenium	3	[d]	[e]
Organic chemicals			
Di(2-ethylhexyl)phthalate (DEHP)	9	[g]	[g]
Hexachlorobenzene	4	[g]	[g]
Hexachlorocyclohexanes (as lindane)	3	[g]	[g]
4,4'methylene dianiline (MDA)	10	[g]	[g]
Pentachlorophenol	[h]	[g]	[g]
Polychlorinated biphenyls (PCBs)	14	14	15
Polychlorinated dibenzo-p-dioxins and dibenzofurans (as TCDD)	[h] 3	[g] 3, 0.1[i]	[g] 3
Polycyclic aromatic hydrocarbons	13	13	15

[a] ABS values, as presented in this document by OEHHA. In most cases, the OEHHA ABS represent dermal absorption values based on the soil vehicle freshly spiked with the chemical contaminant and placed on skin for up to 24 hrs.
[b] (U.S. EPA, 2004) [c] (DTSC, 1994)
[d] An ABS point estimate is not specifically listed for this chemical. For inorganics with insufficient data, USEPA (2004) states that the speciation of the compound is critical to the dermal absorption and there are too little data to extrapolate a reasonable default value.
[e] California's Department of Toxic Substances Control (DTSC, 1994) recommends using 1% as the default dermal absorption value for metals, based on Clement Associates (1988).
[f] California's Department of Toxic Substances Control (DTSC, 1994) in their Preliminary Endangerment Assessment Guidance Manual does not recommend a fractional absorption value for Cr(VI) due to lack of systemic carcinogenicity via non-inhalation routes of exposure.
[g] No specific default ABS value is listed [h] To be assessed for dermal absorption
[i] USEPA (2004) recommends a dermal absorption fraction from soil of 3%, or a dermal absorption fraction of 0.1% if the soil organic content is > 10%.

F.6. References

Abdel-Rahman MS, Skowronski GA, Kadry AM and Turkall RM (1996). Soil decreases the dermal bioavailability of arsenic in a chemical mixture in pig. In: Contaminated Soils, Vol 1. Calabrese EJ, Kostecki PT, and Bonazountas M, eds., Amherst, MA: Amherst Scientific Publishers, pp. 461-72.

Abdel-Rahman MS, Skowronski GA and Turkall RA (1999). Decreased dermal bioavailability of chemicals aged in soil: Arsenic, nickel, and phenanthrene as models. In: Contaminated Soils, Vol 4. Kostecki PT, Calabrese EJ, and Bonzountas M, eds., Amherst, MA: Amherst Scientific, pp. 173-83.

Abdel-Rahman MS, Skowronski GA and Turkall RA (2002). Assessment of the dermal bioavailability of soil-aged benzo(a)pyrene. Hum Ecol Risk Assess 8(2): 429-41.

Abdel-Rahman MS, Skowronski GA and Turkall RM (1997). Dermal bioavailability of soil-aged nickel in male pig skin in vitro. In: Contaminated Soils, Vol 2. Kostecki PT, Calabrese EJ, and Bonazountas M, eds., Amherst, MA: Amherst Scientific Publishers, pp. 117-26.

Alexander M (1995). How toxic are toxic chemicals in soil? Environ Sci Technol 29: 2713-2717.

Anderson YB, Jackson JA and Birnbaum LS (1993). Maturational changes in dermal absorption of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in Fischer 344 rats. Toxicol Appl Pharmacol 119(2): 214-20.

Andersson A (1979). Mercury in soils. In: The Biogeochemistry of Mercury in the Environment, Nriagu JO, ed., Amsterdam: Elsevier/North-Holland, pp. 79-112.

Banks YB, Brewster DW and Birnbaum LS (1990). Age-related changes in dermal absorption of 2,3,7, 8-tetrachlorodibenzo-p-dioxin and 2,3,4,7,8-pentachlorodibenzofuran. Fundam Appl Toxicol 15(1): 163-73.

Baranowska-Dutkiewicz B (1981). Absorption of hexavalent chromium by skin in man. Arch Toxicol 47(1): 47-50.

Baranowska-Dutkiewicz B (1982). Evaluation of the skin uptake of mercuric chloride in man. J Appl Toxicol 2(5): 223-5.

Barber ED, Teetsel NM, Kolberg KF and Guest D (1992). A comparative study of the rates of in vitro percutaneous absorption of eight chemicals using rat and human skin. Fundam Appl Toxicol 19(4): 493-7.

Barker N, Hadgraft J and Rutter N (1987). Skin permeability in the newborn. J Invest Dermatol 88(4): 409-11.

Barrett DA and Rutter N (1994). Transdermal delivery and the premature neonate. Crit Rev Ther Drug Carrier Syst 11(1): 1-30.

Bartek MJ, LaBudde JA and Maibach HI (1972). Skin permeability in vivo: comparison in rat, rabbit, pig and man. J Invest Dermatol 58(3): 114-23.

Bartlett RJ (1991). Chromium cycling in soils and water: links, gaps, and methods. Environ Health Perspect 92: 17-24.

Belman S (1969). Beryllium binding of epidermal constituents. J Occup Med 11(4): 175-83.

Bernstam L, Lan CH, Lee J and Nriagu JO (2002). Effects of arsenic on human keratinocytes: morphological, physiological, and precursor incorporation studies. Environ Res 89(3): 220-35.

Boink ABTJ, Meulenbelt J, Wemer J, Vaessen HAMG, Dortant P and de Wildt DJ (1995). Systemic fluoride poisoning following dermal hydrofluoric acid exposure: Development of an intravenous sodium fluoride infusion model in rats. J. Toxicol.-Cut. & Ocular Toxicol. 14(2): 75-87.

Boogaard PJ and van Sittert NJ (1995). Urinary 1-hydroxypyrene as biomarker of exposure to polycyclic aromatic hydrocarbons in workers in petrochemical industries: baseline values and dermal uptake. Sci Total Environ 163(1-3): 203-9.

Bress WC and Bidanset JH (1991). Percutaneous in vivo and in vitro absorption of lead. Vet Hum Toxicol 33(3): 212-4.

Bronaugh RL, Collier SW, Macpherson SE and Kraeling ME (1994). Influence of metabolism in skin on dosimetry after topical exposure. Environ Health Perspect 102 Suppl 11: 71-4.

Bronaugh RL, Wester RC, Bucks D, Maibach HI and Sarason R (1990). In vivo percutaneous absorption of fragrance ingredients in rhesus monkeys and humans. Food Chem Toxicol 28(5): 369-73.

Brouwer DH, Hoogendoorn L, Bos PM, Boogaard PJ and van Hemmen JJ (1998). Proposal for the assessment to quantitative dermal exposure limits in occupational environments: Part 2. Feasibility study for application in an exposure scenario for MDA by two different dermal exposure sampling methods. Occup Environ Med 55(12): 805-11.

Brunmark P, Bruze M, Skerfving S and Skarping G (1995). Biomonitoring of 4,4'-methylene dianiline by measurement in hydrolysed urine and plasma after epicutaneous exposure in humans. Int Arch Occup Environ Health 67(2): 95-100.

Bunge AL and Parks JM (1996). Soil contamination: Theoretical descriptions. In: Dermal Absorption and Toxicity Assessment. MS Roberts and KS Walters, eds., Marcell Dekker, New York, NY, pp. 669-695.

Bunge AL and Parks JM (1997). Predicting dermal absorption from contact with chemically contaminated soils. Dwyer FJ, Doane TR and Hinman ML, eds., ASTM STP, 1317. Environmental Toxicology and Risk Assessment: Modeling and Risk Assessment (Sixth Volume); Sixth Symposium on Environmental Toxicology and Risk Assessment, Orlando, FL, USA, April 15-18, 1996. American Society for Testing and Materials: Philadelphia, PA, pp. 227-244.

Buykx SE, van den Hoop MA and de Joode P (2004). Simultaneous extraction of bromide, chloride, fluoride and sulfate from soils, waste- and building materials. J Environ Monit 6(6): 552-8.

Choate LM, Ranville JF, Bunge AL and Macalady DL (2006). Dermally adhered soil: 1. Amount and particle-size distribution. Integr Environ Assess Manag 2(4): 375-84.

Chu I, Dick D, Bronaugh R and Tryphonas L (1996). Skin reservoir formation and bioavailability of dermally administered chemicals in hairless guinea pigs. Food Chem Toxicol 34(3): 267-76.

Cohen Hubal EA, Sheldon LS, Burke JM, McCurdy TR, Berry MR, Rigas ML, Zartarian VG and Freeman NC (2000). Children's exposure assessment: a review of factors influencing children's exposure, and the data available to characterize and assess that exposure. Environ Health Perspect 108(6): 475-86.

Corbett GE, Finley BL, Paustenbach DJ and Kerger BD (1997). Systemic uptake of chromium in human volunteers following dermal contact with hexavalent chromium (22 mg/L). J Expo Anal Environ Epidemiol 7(2): 179-89.

Cowen WF, Gastinger AM, Spanier CE and Buckel JR (1998). Sorption and microbial degradation of toluenediamines and methylenedianiline in soil under aerobic and anaerobic conditions. Environ Sci Technol 32(5): 598-603.

Cummins LM and Kimura ET (1971). Safety evaluation of selenium sulfide antidandruff shampoos. Toxicol Appl Pharmacol 20(1): 89-96.

Czernielewski A, Brykalski D and Depczyk D (1965). Experimental investigations on penetration of radioactive chromium (Cr51) through the skin. Dermatologica 131(5): 384-96.

Davis A, Bloom NS and Que Hee SS (1997). The environmental geochemistry and bioaccessibility of mercury in soils and sediments: a review. Risk Anal 17(5): 557-69.

Davison AW (1987). Pathways of fluoride transfer in terrestrial ecosystems. In: Pollutant Transport and Fate in Ecosystems. Special Publication Number 6 of the British Ecological Sociaty. Coughtrey, P.J., Martin, M.H., and Unsworth, M.H. eds., Blackwell Scientific Publications, Oxford, England, pp. 193-210.

Day GA, Stefaniak AB, Weston A and Tinkle SS (2006). Beryllium exposure: dermal and immunological considerations. Int Arch Occup Environ Health 79(2): 161-4.

Deisinger PJ, Perry LG and Guest D (1998). In vivo percutaneous absorption of [14C]DEHP from [14C]DEHP-plasticized polyvinyl chloride film in male Fischer 344 rats. Food Chem Toxicol 36(6): 521-7.

Derelanko MJ, Gad SC, Gavigan F and Dunn BJ (1985). Acute dermal toxicity of dilute hydrofluoric acid. J. Toxicol.-Cut. & Ocular Toxicol. 4(2): 73-85.

Deubner D and Kent M (2007). Keeping beryllium workers safe: an enhanced preventive model. J Occup Environ Hyg 4(3): D23-30.

Dick IP, Blain PG and Williams FM (1997a). The percutaneous absorption and skin distribution of lindane in man. I. In vivo studies. Hum Exp Toxicol 16(11): 645-51.

Diliberto JJ, Jackson JA and Birnbaum LS (1996). Comparison of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) disposition following pulmonary, oral, dermal, and parenteral exposures to rats. Toxicol Appl Pharmacol 138(1): 158-68.

Driver JH, Konz JJ and Whitmyre GK (1989). Soil adherence to human skin. Bull Environ Contam Toxicol 43(6): 814-20.

DTSC (1994). Preliminary Endangerment Assessment Guidance Manual (A guidance manual for evaluating hazardous substances release sites). Chapter 3, Preparation of the PEA report; Appendix A, Tables for use with screening evaluations; Appendix B, Derivations for equations and complete equation for VOC emission model. Department of Toxic Substances Control, California Environmental Protection Agency, Sacramento, CA. Online at: www.dtsc.ca.gov/PublicationsForms/pubs_index.cfm.

Duff RM and Kissel JC (1996). Effect of soil loading on dermal absorption efficiency from contaminated soils. J Toxicol Environ Health 48(1): 93-106.

Eagers RY (1969). Toxic Properties of Inorganic Fluorine Compounds. Elsevier Publishing Company LTD., New York, NY. .

Elovaara E, Heikkila P, Pyy L, Mutanen P and Riihimaki V (1995). Significance of dermal and respiratory uptake in creosote workers: exposure to polycyclic

aromatic hydrocarbons and urinary excretion of 1-hydroxypyrene. Occup Environ Med 52(3): 196-203.

Elsisi AE, Carter DE and Sipes IG (1989). Dermal absorption of phthalate diesters in rats. Fundam Appl Toxicol 12(1): 70-7.

Farley J, Skelly EM and Weber CB (1986). Percutaneous absorption of selenium sulfide. J Environ Sci Health Part A Environ Sci Eng 21(6): 571-82.

Feldmann RJ and Maibach HI (1974). Percutaneous penetration of some pesticides and herbicides in man. Toxicol Appl Pharmacol 28(1): 126-32.

Fendorf SE (1995). Surface reactions of chromium in soils and waters. Geoderma 67: 55-71.

Filon FL, Boeniger M, Maina G, Adami G, Spinelli P and Damian A (2006). Skin absorption of inorganic lead (PbO) and the effect of skin cleansers. J Occup Environ Med 48(7): 692-9.

Fisher HL, Shah PV, Sumler MR and Hall LL (1989). In vivo and in vitro dermal penetration of 2,4,5,2',4',5'-hexachlorobiphenyl in young and adult rats. Environ Res 50(1): 120-39.

Florence TM, Stauber JL, Dale LS, Henderson D, Izard BE and Belbin K (1998). The absorption of ionic lead compounds through the skin of mice. J Nutr Environ Med (Abingdon) 8(1): 19-23.

Flynn GL (1990). Physicochemical determinants of skin absorption. In: Gerrity TR and Henry CJ, eds., Principles of Route-to-Route Extrapolation for Risk Assessment. Elsevier, New York, pp. 93-127.

Franz TJ, Lehman PA, Franz SF and Guin JD (1996). Comparative percutaneous absorption of lindane and permethrin. Arch Dermatol 132(8): 901-5.

Friberg L, Skog E and Wahlberg JE (1961). Resorption of mercuric chloride and methyl mercury dicyandiamide in guinea-pigs through normal skin and through skin pretreated with acetone, alkylaryl-sulphonate and soap. Acta Derm Venereol Suppl (Stockh) 41: 40-52.

Fullerton A, Andersen JR and Hoelgaard A (1988). Permeation of nickel through human skin in vitro--effect of vehicles. Br J Dermatol 118(4): 509-16.

Fullerton A, Andersen JR, Hoelgaard A and Menne T (1986). Permeation of nickel salts through human skin in vitro. Contact Dermatitis 15(3): 173-7.

Fullerton A and Hoelgaard A (1988). Binding of nickel to human epidermis in vitro. Br J Dermatol 119(5): 675-82.

Gammelgaard B, Fullerton A, Avnstorp C and Menne T (1992). Permeation of chromium salts through human skin in vitro. Contact Dermatitis 27(5): 302-10.

Garner CE and Matthews HB (1998). The effect of chlorine substitution on the dermal absorption of polychlorinated biphenyls. Toxicol Appl Pharmacol 149(2): 150-8.

Gisiger L (1968). The solubility of various fluorine compounds in soil. Fluoride 1: 21-6.

Hamel SC, Ellickson KM and Lioy PJ (1999). The estimation of the bioaccessibility of heavy metals in soils using artificial biofluids by two novel methods: mass-balance and soil recapture. Sci Total Environ 243-244: 273-83.

Hawley JK (1985). Assessment of health risk from exposure to contaminated soil. Risk Anal 5(4): 289-302.

Hickey MG and Kittrick JA (1984). Chemical partitioning of cadmium, copper, nickel and zinc in soils and sediments containing high levels of heavy metals J Environ Qual 13(3): 372-376.

Holbrook KA (1998). Structure and biochemical organogenesis of skin and cutaneous appendages in the fetus and newborn. Chapter 71. In: Fetal and Neonatal Physiology. Volume 1, second edition, Polin RA and Fox WW, eds., W.B. Saunders Co., Philadelphia, PA, pp. 729-52.

Holmes KK, Kissel JC and Richter KY (1996). Investigation of the influence of oil on soil adherence to skin. J Soil Contam 5(4): 301-308.

Horowitz SB and Finley BL (1993). Using human sweat to extract chromium from chromite ore processing residue: applications to setting health-based cleanup levels. J Toxicol Environ Health 40(4): 585-99.

Hostynek JJ (2003). Factors determining percutaneous metal absorption. Food Chem Toxicol 41(3): 327-45.

Hostynek JJ, Dreher F, Nakada T, Schwindt D, Anigbogu A and Maibach HI (2001a). Human stratum corneum adsorption of nickel salts. Investigation of depth profiles by tape stripping in vivo. Acta Derm Venereol Suppl (Stockh)(212): 11-8.

Hostynek JJ, Dreher F, Pelosi A, Anigbogu A and Maibach HI (2001b). Human stratum corneum penetration by nickel. In vivo study of depth distribution after occlusive application of the metal as powder. Acta Derm Venereol Suppl (Stockh)(212): 5-10.

Hostynek JJ, Hinz RS, Lorence CR, Price M and Guy RH (1993). Metals and the skin. Crit Rev Toxicol 23(2): 171-235.

Hotchkiss SAM, Hewitt P and Caldwell J (1993). Percutaneous absorption of 4,4'-methylene-bis-(2-chloroaniline) and 4,4'-methylenedianiline through rat and human skin in vitro. Toxicol In Vitro 7(2): 141-48.

Hursh JB, Clarkson TW, Miles EF and Goldsmith LA (1989). Percutaneous absorption of mercury vapor by man. Arch Environ Health 44(2): 120-7.

Jing C, Meng X and Korfiatis GP (2004). Lead leachability in stabilized/solidified soil samples evaluated with different leaching tests. J Hazard Mater 114(1-3): 101-10.

Kalivas J (1993). Lack of serum selenium rise after overnight application of selenium sulfide. Arch Dermatol 129(5): 646-8.

Kao J (1990). Validity of skin absorption and metabolite studies. In: Methods for Skin Absorption. Kemppainen, BW and Reifenrath, WG, eds. Boca Raton, FL, CRC Press, pp. 191-212.

Kao J and Carver MP (1990). Cutaneous metabolism of xenobiotics. Drug Metab Rev 22(4): 363-410.

Kao J, Patterson FK and Hall J (1985). Skin penetration and metabolism of topically applied chemicals in six mammalian species, including man: an in vitro study with benzo[a]pyrene and testosterone. Toxicol Appl Pharmacol 81(3 Pt 1): 502-16.

Kenyon SH, Bhattacharyya J, Benson CJ and Carmichael PL (2004). Percutaneous penetration and genotoxicity of 4,4'-methylenedianiline through rat and human skin in vitro. Toxicology 196(1-2): 65-75.

Kimura M and Otaki N (1972). Percutaneous absorption of cadmium in rabbit and hairless mouse. Industrial Health 10: 7-10.

Kissel JC (2011). The mismeasure of dermal absorption. Journal of exposure science & environmental epidemiology 21(3): 302-9.

Kissel JC, Richter KY and Fenske RA (1996). Factors affecting soil adherence to skin in hand-press trials. Bull Environ Contam Toxicol 56(5): 722-8.

Kissel JC, Shirai JH, Richter KY and Fenske RA (1998). Investigation of dermal contact with soil in controlled trials. J Soil Contam 7(6): 737-52.

Koizumi A (1991). Experimental evidence for the possible exposure of workers to hexachlorobenzene by skin contamination. Br J Ind Med 48: 622-8.

Lansdown AB and Sampson B (1996). Dermal toxicity and percutaneous absorption of cadmium in rats and mice. Lab Anim Sci 46(5): 549-54.

LaPerche V, Traina SJ, Gaddam P and Logan TJ (1996). Chemical and mineralogical characterizations of Pb in a contaminated soil: Reactions with synthetic apatite. Environ Sci Technol 30(11): 3321-3326.

Larese F, Gianpietro A, Venier M, Maina G and Renzi N (2007). In vitro percutaneous absorption of metal compounds. Toxicol Lett 170(1): 49-56.

Liden S and Lundberg E (1979). Penetration of chromium in intact human skin in vivo. J Invest Dermatol 72(1): 42-5.

Lilly SG, Florence TM and Stauber JL (1988). The use of sweat to monitor lead absorption through the skin. Sci Total Environ 76: 267-78.

Loth H, Hauck G, Borchert D and Theobald F (2000). Statistical testing of drug accumulation in skin tissues by linear regression versus contents of stratum corneum lipids. Int J Pharm 209(1-2): 95-108.

Lowney YW, Ruby MV, Wester RC, Schoof RA, Holm SE, Hui XY, Barbadillo S and Maibach HI (2005). Percutaneous absorption of arsenic from environmental media. Toxicol Ind Health 21(1-2): 1-14.

Makri A, Goveia M, Balbus J and Parkin R (2004). Children's susceptibility to chemicals: a review by developmental stage. J Toxicol Environ Health B Crit Rev 7(6): 417-35.

Mali JWH, van Kooten WJ, van Neer FCJ and Spruit D (1964). Quantitative aspects of chromium sensitization. Acta Derm. Venereol. 44: 44-8.

Mayes BA, Brown GL, Mondello FJ, Holtzclaw KW, Hamilton SB and Ramsey AA (2002). Dermal absorption in rhesus monkeys of polychlorinated biphenyls from soil contaminated with Aroclor 1260. Regul Toxicol Pharmacol 35(3): 289-95.

McCormack JJ (1982). Neonatal Skin: Structure and Function. Marcel Dekker, New York, pp. 149-164.

McKone TE (1990). Dermal uptake of organic chemicals from a soil matrix. Risk Anal 10(3): 407-19.

McKone TE (1991). The Precision of a Fugacity-Based Model for Estimating Dermal Uptake of Chemicals from Soil. In: Hydrocarbon Contaminated Soils, Chapter 38, Chelsea, MI, Lewis Publishers, pp. 555-74.

McLaughlin T (1984). Review of dermal absorption. Office of Health and Environmental Assessment, US EPA. Wasjington, D.C. EPA/600/8-84/033.

Medinsky MA, Cuddihy RG and McClellan RO (1981). Systemic absorption of selenious acid and elemental selenium aerosols in rats. J Toxicol Environ Health 8(5-6): 917-28.

Melnick RL, Morrissey RE and Tomaszewski KE (1987). Studies by the National Toxicology Program on di(2-ethylhexyl)phthalate. Toxicol Ind Health 3(2): 99-118.

Milhaud G, Clauw M and Joseph-Enriquez B (1989). Bioavailability in soil fluoride in sheep. Fluoride 22(4): 188-94.

Miselnicky SR, Lichtin JL, Sakr A and Bronaugh RL (1988). Influence of solubility, protein binding, and percutaneous absorption on reservoir formation in skin. J Soc Cosmet Chem 39: 169-177.

Moody RP, Joncas J, Richardson M and Chu I (2007). Contaminated soils (I): In vitro dermal absorption of benzo[a]pyrene in human skin. J Toxicol Environ Health A 70(21): 1858-65.

Moody RP, Joncas J, Richardson M, Petrovic S and Chu I (2009b). Contaminated soils (II): in vitro dermal absorption of nickel (Ni-63) and mercury (Hg-203) in human skin. J Toxicol Environ Health A 72(8): 551-9.

Moody RP, Yip A and Chu I (2009a). Effect of cold storage on in vitro human skin absorption of six 14C-radiolabeled environmental contaminants: benzo[a]pyrene, ethylene glycol, methyl parathion, naphthalene, nonyl phenol, and toluene. J Toxicol Environ Health A 72(8): 505-17.

Moore MR, Meredith PA, Watson WS, Sumner DJ, Taylor MK and Goldberg A (1980). The percutaneous absorption of lead-203 in humans from cosmetic preparations containing lead acetate, as assessed by whole-body counting and other techniques. Food Cosmet Toxicol 18(4): 399-405.

Ng KM, Chu I, Bronaugh RL, Franklin CA and Somers DA (1992). Percutaneous absorption and metabolism of pyrene, benzo[a]pyrene, and di(2-ethylhexyl) phthalate: comparison of in vitro and in vivo results in the hairless guinea pig. Toxicol Appl Pharmacol 115(2): 216-23.

Nico PS, Fendorf SE, Lowney YW, Holm SE and Ruby MV (2004). Chemical structure of arsenic and chromium in CCA-treated wood: implications of environmental weathering. Environ Sci Technol 38(19): 5253-60.

Nico PS, Ruby MV, Lowney YW and Holm SE (2006). Chemical speciation and bioaccessibility of arsenic and chromium in chromated copper arsenate-treated wood and soils. Environ Sci Technol 40(1): 402-8.

OEHHA (1999). Air Toxics Hot Spots Program Risk Assessment Guidelines, Part I: The Determination of Acute Reference Exposure Levels for Airborne Toxicants. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Sacramento CA. Availble online at: http://www.oehha.ca.gov.

Petzow VG and Zorn H (1974). Zur Toxikologie berylliumhaltiger stoffe. Chemiker-Zeitung 98(5): 236-241.

Phillips DH, Schoket B, Hewer A and Grover PL (1990). DNA adduct formation in human and mouse skin by mixtures of polycyclic aromatic hydrocarbons. IARC Sci Publ(104): 223-9.

Poet TS and McDougal JN (2002). Skin absorption and human risk assessment. Chem Biol Interact 140(1): 19-34.

Poiger H and Schlatter C (1980). Influence of solvents and adsorbents on dermal and intestinal absorption of TCDD. Food Cosmet Toxicol 18(5): 477-81.

Polomski J, Fluhler H and Blaser P (1982). Accumulation of airborne fluoride in soils. J. Environ. Qual. 11(3): 457-61.

Qiao G and Riviere JE (2000). Dermal absorption and tissue disposition of 3,3',4, 4'-tetrachlorobiphenyl (TCB) in an ex-vivo pig model: assessing the impact of dermal exposure variables. Int J Occup Environ Health 6(2): 127-37.

Qiao GL and Riviere JE (2001). Enhanced systemic tissue distribution after dermal versus intravenous 3,3',4,4'-tetrachlorobiphenyl exposure: limited utility of radiolabel blood area under the curve and excretion data in dermal absorption calculations and tissue exposure assessment. Toxicol Appl Pharmacol 177(1): 26-37.

Rahman MS, Hall LL and Hughes MF (1994). In vitro percutaneous absorption of sodium arsenate in B6C3F1 mice. Toxicol In Vitro 8(3): 441-8.

Reddy MB, Guy RH and Bunge AL (2000). Does epidermal turnover reduce percutaneous penetration? Pharm Res 17(11): 1414-9.

Reifenrath WG, Chellquist EM, Shipwash EA and Jederberg WW (1984). Evaluation of animal models for predicting skin penetration in man. Fundam Appl Toxicol 4(2 Pt 2): S224-30.

Roy TA, Hammerstrom K and Schaum J (2008). Percutaneous absorption of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) from soil. J Toxicol Environ Health A 71(23): 1509-15.

Roy TA, Krueger AJ, Taylor BB, Mauro DM and Goldstein LS (1998). Studies estimating the dermal bioavailability of polynuclear aromatic hydrocarbons from manufactured gas plant tar-contaminated soils. Environ Sci Technol 32: 3113-17.

Roy TA and Singh R (2001). Effect of soil loading and soil sequestration on dermal bioavailability of polynuclear aromatic hydrocarbons. Bull Environ Contam Toxicol 67(3): 324-31.

Ruby MV, Schoof R, Brattin W, Goldade M, Post G, Harnois M, Mosby DE, Casteel SW, Berti W, Carpenter M, Edwards D, Cragin D and Chappell W (1999). Advances in evaluating the oral bioavailability of inorganics in soil for use in human health risk assessment. Environ Sci Technol 33(21): 3697-3705.

Samitz MH, Katz SA, Scheiner DM and Gross PR (1969). Chromium-protein interactions. Acta Derm Venereol 49(2): 142-6.

Sartorelli P, Montomoli L, Sisinni AG, Barabesi L, Bussani R and Cherubini Di Simplicio F (2003). Percutaneous penetration of inorganic mercury from soil: an in vitro study. Bull Environ Contam Toxicol 71(6): 1091-9.

Sartorelli P, Montomoli L, Sisinni AG, Bussani R, Cavallo D and Foa V (2001). Dermal exposure assessment of polycyclic aromatic hydrocarbons: in vitro percutaneous penetration from coal dust. Toxicol Ind Health 17(1): 17-21.

SCAQMD (1988). Multi-pathway health risk assessment input parameters guidance document. South Coast Air Quality Management District, El Monte CA. Prepared by: Clement Associates, Inc., Fairfax, VA.

Schmid P, Buhler F and C. S (1992). Dermal absorption of PCB in man. Chemosphere 24: 1283-1292.

Scott RC, Dugard PH, Ramsey JD and Rhodes C (1987). In vitro absorption of some o-phthalate diesters through human and rat skin. Environ Health Perspect 74: 223-7.

Scott WC and Dean JR (2005). An assessment of the bioavailability of persistent organic pollutants from contaminated soil. J Environ Monit 7(7): 710-5.

Shah PV, Fisher HL, Sumler MR, Monroe RJ, Chernoff N and Hall LL (1987). Comparison of the penetration of 14 pesticides through the skin of young and adult rats. J Toxicol Environ Health 21(3): 353-66.

Shatkin JA, Wagle M, Kent S and Menzie CA (2002). Development of a biokinetic model to evaluate dermal absorption of polycyclic aromatic hydrocarbons from soil. Hum Ecol Risk Assess 8: 713-734.

Sheehan PJ, Meyer DM, Sauer MM and Paustenbach DJ (1991). Assessment of the human health risks posed by exposure to chromium-contaminated soils. J Toxicol Environ Health 32(2): 161-201.

Sheppard SC and Evenden WG (1994). Contaminant enrichment and properties of soil adhering to skin. J Environ Qual 23: 604-13.

Shu H, Teitelbaum P, Webb AS, Marple L, Brunck B, Dei Rossi D, Murray FJ and Paustenbach D (1988). Bioavailability of soil-bound TCDD: dermal bioavailability in the rat. Fundam Appl Toxicol 10(2): 335-43.

Silberberg I (1972). Ultrastructural identification of mercury in epidermis. A method for visualization of gold-mercury amalgams in skin from normal and allergic persons and those with primary irritant reactions to mercury. Arch Environ Health 24(2): 129-44.

Skowronski GA, Turkall RM and Abdel-Rahman MS (2000). In vitro penetration of soil-aged mercury through pig skin. J Toxicol Environ Health A 61(3): 189-200.

Spalt EW, Kissel JC, Shirai JH and Bunge AL (2009). Dermal absorption of environmental contaminants from soil and sediment: a critical review. J Expo Sci Environ Epidemiol 19(2): 119-48.

Spruit D, Mali JW and De Groot N (1965). The interaction of nickel ions with human cadaverous dermis. Electric potential, absorption, swelling. J Invest Dermatol 44: 103-6.

Stauber JL, Florence TM, Gulson BL and Dale LS (1994). Percutaneous absorption of inorganic lead compounds. Sci Total Environ 145(1-2): 55-70.

Stewart MA, Jardine PM, Brandt CC, Barnett MO, Fendorf S, McKay LD, Mehlhorn TL and Paul K (2003). Effects of contaminant concentration, aging, and soil properties on the bioaccessibility of Cr(III) and Cr(VI) in soil. Soil Sediment Contam 12: 1-21.

Stroo HF, Nakles DV, Kreitinger JP, Loehr RC, Hawthorne SB, Luthy RG, Holman HY and LaPierre A (2005a). Improving risk assessments for manufactured gas plant soils by measuring PAH availability. Integr Environ Assess Manag 1(3): 259-66.

Stroo HF, Roy TA, Liban CB and Kreitinger JP (2005b). Dermal bioavailability of benzo[a]pyrene on lampblack: implications for risk assessment. Environ Toxicol Chem 24(6): 1568-72.

Sun CC, Wong TT, Hwang YH, Chao KY, Jee SH and Wang JD (2002). Percutaneous absorption of inorganic lead compounds. AIHA J (Fairfax, Va) 63(5): 641-6.

Tang XY, Zhu YG, Cui YS, Duan J and Tang L (2006). The effect of ageing on the bioaccessibility and fractionation of cadmium in some typical soils of China. Environ Int 32(5): 682-9.

Tanojo H, Hostynek JJ, Mountford HS and Maibach HI (2001). In vitro permeation of nickel salts through human stratum corneum. Acta Derm Venereol Suppl (Stockh)(212): 19-23.

Thompson H (1946). Physical Growth. Chapter 5. In: Manual of Child Psychology. New York, John Wiley and Sons, pp. 255-94.

Tinkle SS, Antonini JM, Rich BA, Roberts JR, Salmen R, DePree K and Adkins EJ (2003). Skin as a route of exposure and sensitization in chronic beryllium disease. Environ Health Perspect 111(9): 1202-8.

Tregear RT (1966). The permeability of mammalian skin to ions. J Invest Dermatol 46(1): 16-23.

Turkall RM, Skowronski GA, Suh DH and Abdel-Rahman MS (2003). Effect of a chemical mixture on dermal penetration of arsenic and nickel in male pig in vitro. J Toxicol Environ Health A 66(7): 647-55.

U.S. EPA (1992). Dermal Exposure Assessment: Principles and Applications. Interim Report. U.S. Environmental Protection Agency, Washington D.C. January 1992. EPA/600/8-91/011B, pp. 6-1 to 6-43. Available online at: http://www.epa.gov/nceawww1/pdfs/derexp.pdf.

U.S. EPA (2004). Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual (Part E, supplemental guidance for dermal risk assessment). Final. Office of Superfund Remediation and Technology Innovation, U.S. Environmental Protection Agency, Washington DC.

USEPA (1992). Dermal Exposure Assessment: Principals and Applications. Interim Report. U.S. Environmental Protection Agency, Washington D.C. January 1992. EPA/600/8-91/011B, pp. 6-1 to 6-43. Available online at: http://www.epa.gov/nceawww1/pdfs/derexp.pdf.

USEPA (1998). Locating and estimating air emissions from sources of lead and lead compounds. U.S. Environmental Protection Agency, Research Triangle Park, NC. EPA-454/R-98-006, pp. 1-1 to 1-5, 2-1 to 2-4, 3-1 to 3-15, 6-1 to 6-16. Available online at: www.epa.gov/ttnchie1/le/lead.pdf.

USEPA (2004). Risk assessment guidance for superfund, Volume 1: Human Health evaluation manual (Part E, supplemental guidance for dermal risk assessment). Final. Office of Superfund Remediation and Technology Innovation, U.S. Environmental Protection Agency, Washington DC.

Wahlberg JE (1965). Percutaneous absorption of sodium chromate (51Cr), cobaltous (58Co), and mercuric (203Hg) chlorides through excised human and guinea pig skin. Acta Derm Venereol 45(6): 415-26.

Wahlberg JE and Skog E (1963). The percutaneous absorption of sodium chromate (51Cr) in the guinea pig. Acta Derm Venereol Suppl (Stockh) 43: 102-8.

Wainman T, Hazen RE and Lioy PJ (1994). The extractability of Cr(VI) from contaminated soil in synthetic sweat. J Expo Anal Environ Epidemiol 4(2): 171-81.

Weber LW (1993). The penetration of 2,3,7,8-tetrachlorodibenzo-p-dioxin into viable and non-viable porcine skin in vitro. Toxicology 84(1-3): 125-40.

Weber LW, Zesch A and Rozman K (1991). Penetration, distribution and kinetics of 2,3,7,8-tetrachlorodibenzo-p-dioxin in human skin in vitro. Arch Toxicol 65(5): 421-8.

Wester R, Logan F, Maibach H, Wade M and Hoang K (1995). In vitro percutaneous absorption of mercury from water and soil through human skin. Proceedings of the Society of Toxicology 34th Annual Meeting. The Toxicologist 15(1): 135-36.

Wester RC, Christoffel J, Hartway T, Poblete N, Maibach HI and Forsell J (1998b). Human cadaver skin viability for in vitro percutaneous absorption: storage and detrimental effects of heat-separation and freezing. Pharm Res 15(1): 82-4.

Wester RC, Hui X, Barbadillo S, Maibach HI, Lowney YW, Schoof RA, Holm SE and Ruby MV (2004). In vivo percutaneous absorption of arsenic from water and CCA-treated wood residue. Toxicol Sci 79(2): 287-95.

Wester RC and Maibach HI (1975). Percutaneous absorption in the rhesus monkey compared to man. Toxicol Appl Pharmacol 32(2): 394-8.

Wester RC and Maibach HI (1983). Cutaneous pharmacokinetics: 10 steps to percutaneous absorption. Drug Metab Rev 14(2): 169-205.

Wester RC and Maibach HI (1985). In vivo percutaneous absorption and decontamination of pesticides in humans. J Toxicol Environ Health 16(1): 25-37.

Wester RC and Maibach HI (1998c). Percutaneous absorption of hazardous substances from soil and water. In: Dermal Absorption and Toxicity Assessment, Roberts MS and Walters KA, eds., New York: Marcel Dekker, pp. 697-707.

Wester RC and Maibach HI (1999). Skin contamination and absorption of chemicals from water and soil. In: Percutaneous Absorption: Drugs, Cosmetics, Mechanisms and Methodology. Bronaugh RL and Maibach HI, eds., Dekker, New York, pp. 133-148.

Wester RC, Maibach HI, Bucks DA, McMaster J, Mobayen M, Sarason R and Moore A (1990a). Percutaneous absorption and skin decontamination of PCBs: in vitro studies with human skin and in vivo studies in the rhesus monkey. J Toxicol Environ Health 31(4): 235-46.

Wester RC, Maibach HI, Bucks DA, Sedik L, Melendres J, Liao C and DiZio S (1990b). Percutaneous absorption of [14C]DDT and [14C]benzo[a]pyrene from soil. Fundam Appl Toxicol 15(3): 510-6.

Wester RC, Maibach HI, Sedik L, Melendres J, DiZio S and Wade M (1992). In vitro percutaneous absorption of cadmium from water and soil into human skin. Fundam Appl Toxicol 19(1): 1-5.

Wester RC, Maibach HI, Sedik L, Melendres J and Wade M (1993a). In vivo and in vitro percutaneous absorption and skin decontamination of arsenic from water and soil. Fundam Appl Toxicol 20(3): 336-40.

Wester RC, Maibach HI, Sedik L, Melendres J and Wade M (1993b). Percutaneous absorption of PCBs from soil: in vivo rhesus monkey, in vitro human skin, and binding to powdered human stratum corneum. J Toxicol Environ Health 39(3): 375-82.

Wester RC, Melendres J, Sedik L, Maibach H and Riviere JE (1998a). Percutaneous absorption of salicylic acid, theophylline, 2, 4-dimethylamine, diethyl hexyl phthalic acid, and p-aminobenzoic acid in the isolated perfused porcine skin flap compared to man in vivo. Toxicol Appl Pharmacol 151(1): 159-65.

Yang J, Mosby DE, Casteel SW and Blanchar RW (2001). Lead immobilization using phosphoric acid in a smelter-contaminated urban soil. Environ Sci Technol 35(17): 3553-9.

Yang J, Mosby DE, Casteel SW and Blanchar RW (2002). In vitro lead bioaccessibility and phosphate leaching as affected by surface application of phosphoric acid in lead-contaminated soil. Arch Environ Contam Toxicol 43(4): 399-405.

Yang JJ, Roy TA, Krueger AJ, Neil W and Mackerer CR (1989). In vitro and in vivo percutaneous absorption of benzo[a]pyrene from petroleum crude-fortified soil in the rat. Bull Environ Contam Toxicol 43(2): 207-14.

Yourick JJ, Koenig ML, Yourick DL and Bronaugh RL (2004). Fate of chemicals in skin after dermal application: does the in vitro skin reservoir affect the estimate of systemic absorption? Toxicol Appl Pharmacol 195(3): 309-20.

Zhang P, Ryan JA and Yang J (1998). In vitro soil Pb solubility in the presence of hydroxyapatite. Environ Sci Technol 32(18): 2763-2768.

Appendix G. Chemical-specific Soil Half-life

G.1 Algorithm for Estimating Chemical-specific Soil Half-life

The average concentration of a substance in soil (Csoil) is a function of several different variables, including deposition rate, accumulation period, mixing depth, soil bulk density, and the chemical-specific half-life, as shown in equation G-1 below:

$$Csoil = [GLC\ (Dep\text{-}rate)\ (86{,}400)\ (X)] / [Ks\ (SD)\ (BD)\ (Tt)] \quad \textbf{(Eq. G-1)}$$

where: Csoil = average soil concentration at a specific receptor location over the evaluation period (µg/kg)

GLC = ground level concentration from the air dispersion modeling (µg/m^3)

Dep-rate = vertical rate of deposition (m/sec) (see Chapter 2 for values)

86,400 = seconds per day conversion factor

X = integral function accounting for soil half-life

Ks = soil elimination time constant = 0.693/T1/2

SD = soil mixing depth = 1 cm for dermal scenario

BD = bulk density of soil = 1333 kg/m^3

Tt = total averaging time = 70 years = 25,550 days

The soil half-life is part of the integral function X determined as below:

$$X = [\{Exp\ (-Ks \times Tf) - Exp\ (-Ks \times To)\} / Ks] + Tt \quad \textbf{(Eq. G-2)}$$

where: EXP = Exponent base e = 2.72

Ks = soil elimination constant = 0.693/ T1/2

T1/2 = chemical-specific soil half-life

Tf = end of exposure duration (days); 25,500 for a 70-year exposure

T0 = beginning of exposure duration (days) = 0 days

Tt = total days of exposure period = Tf - T0 (days)

Estimating toxicant soil concentration is necessary for estimating dose from incidental soil ingestion by home raised meat, home raised produce, and dermal absorption via contact with contaminated soil.

Since the chemicals that the Hot Spots program is concerned with are emitted into the air and then subject to deposition to the soil, there are only two classes of chemicals considered. These classes are the semivolatilve organic chemicals, such as PAHs, PCBs and dioxins, and toxic metals such as hexavalent chromium, cadmium, lead,

arsenic, and beryllium. Other programs that consider hazardous waste sites may be concerned with other classes of chemicals such as volatile organic solvents.

Soil extraction studies were often used to estimate soil half-life by using rigorous extraction techniques with an organic solvent (e.g., dichloromethane) to release as much of the remaining chemical from soil as possible. The amount of chemical extracted from soil is considered the fraction that is bioaccessible for uptake. The bioaccessible fraction of a pollutant in soil, which is reduced over time by various processes, is used to estimate the soil half-life of chemicals.

An extraction procedure that mimics or parallels bioavailability is preferable for assessing exposure and risk than one whose sole virtue is the removal of the largest percentage of the compound from soil (Kelsey, 1997; Reid, 2000; Tang, 1999). These investigations suggest that mild, selective extractants may prove more useful as predictors of exposure than the methods currently used for regulatory purposes in some programs. The solvent needed for predictive purposes may vary with the pollutant and the species of concern.

Another common method to determine soil half-life of organic compounds is through mineralization, or ultimate degradation, studies. Instead of measuring the parent organic compound remaining in soil through extraction methods, mineralization studies add the radiolabeled chemical to soil, and measure the release of $^{14}CO_2$ from soil resulting from “ultimate” breakdown of the compound by microbial degradation.

Mineralization studies may be quite useful for determining the soil half-life of organic chemicals, if abiotic loss processes are minor, and if mineralization of the chemical occurs quickly once primary degradation (and presumably loss of toxicity) of the chemical takes place.

G.2 Metals and Other Inorganic Compounds

Biodegradation as such is not expected to occur with metals and other elements because of their elemental nature. However, once a metal is deposited to soil, leaching or weathering may eventually result in movement of the metal out of the system. The valence and charge of the metal in soil affects its sorption, solubility, and retention in soil. Additionally, soil pH and availability of charged sites on soil surfaces are the primary factors controlling formation of the ionic species, charged metal complexes or precipitates (US EPA, 2003).

Soil with predominately negatively charged sites is more plentiful in the United States; less than 5% of the total available charge on the soil surface is positively charged (US EPA, 2003; Fairbrother et al., 2007). For the metals that largely exist as cations in soil (beryllium, cadmium, lead, inorganic mercury and nickel), there is a greater propensity to be sorbed to soil particles. This makes them less bioavailable, but it also results in greater loading of metals into the soil because of reduced mobility and leaching.

Under most relevant scenarios, arsenic, chromium, fluoride and selenium deposition to soil typically results in an anion or formations of anionic complexes with oxygen (US EPA, 2003; Fairbrother et al., 2007). The most common forms of arsenic are arsenate (As(V)) and arsenite (As(III)), which are present in soil solution in the form of AsO_4^{3-} and AsO_3^{3-}, respectively. Similarly, selenium can be present as selenates (SeO_4^{2-}) and selenites (SeO_3^{2-}). Hexavalent chromium (Cr(VI)) can exist as chromate (CrO_4^{2-}) which is usually considered more soluble, mobile, and bioavailable than the sparingly soluble chromite (Cr(III)), which is normally present in soil as the precipitate $Cr(OH)_3$. Anionic metals generally move into pore water where they can leach out of the system faster, but are also more bioavailable.

As a default estimate, the metal content of soil is assumed to decay with a half-life of 10^8 days unless site-specific information is presented showing that soil conditions will result in the loss of soil metal content, i.e., soil aging or leaching. The 10^8 default means that significant loss or removal is not occurring within the risk assessment time frame of interest.

Some fraction of chromium (VI) will undergo reduction to the less toxic chromite (CrIII) species when deposited to soil (Bartlett, 1991; Fendorf, 2004; Stewart et al., 2003). However, oxidation reactions of chromium (III) to chromium (VI) can also occur at the same time in soil. Characterizing the reduction of chromium (VI) to chromium (III) is complex and "it is not possible to predict how chromium compounds will behave in soil until the soil environment has been adequately characterized" (Cohen et al., 1994a, citing Gochfeld and Whitmer, 1991). Several tests have been suggested for evaluating the reducing capacity of soils and may be considered in the development of site-specific information (Cohen et al., 1994a, citing Bartlett and James, 1988; Walkley and Black, 1934). These tests are described as follows:

> "(1) Total Cr(VI) Reducing Capacity. Use the Walkley-Black (1934) soil organic matter determination in which carbon oxidizable by $K_2Cr_2O_7$ is measured by titrating the Cr(VI) not reduced by a soil sample (in suspension with concentrated H_2SO_4) with $Fe(NH_4)_2(SO_4)_2$.
>
> (2) Available Reducing Capacity. Shake 2.5 cm^3 of moist soil 18 hours with 25 mL of 0.1 to 10mM chromium as $K_2Cr_2O_7$ in 10mM H_3PO_4, filter or centrifuge, and determine Cr(VI) not reduced in the extract by the s-diphenylcarbazide method.
>
> (3) Reducing Intensity. The procedure is the same as that used in (2) above except that 10mM KH_2PO_4 should be used in the matrix solution in place of H_3PO_4."

In the absence of site-specific data, the public health protective assumption is to assume that hexavalent chromium remains in the hexavalent form in the soil. In most instances this will lead to an over prediction of hexavalent chromium concentration from airborne deposition.

G.3 Organics

Organic compounds deposited in soil are subject to degradation or loss by both biotic and abiotic processes. Biotic processes include degradation by soil microorganisms. Abiotic loss of organic compounds in soil includes such processes as photochemical reactions (if on the surface of the soil) or volatilization from the soil.

For some persistent organic chemicals, such as PAHs, soil aging is the abiotic process causing the most loss. Aging is associated with a continuous diffusion and retention of compound molecules into remote and inaccessible regions within the soil matrix over time, often on the order of weeks or months, thereby occluding the compounds from abiotic and biotic processes (Northcott and Jones, 2001).

Many earlier soil half-life studies assumed that decreased soil extractability and bioavailability of chemicals with time was due to biodegradation by soil microorganisms, when, in fact, soil aging is a significant or dominant factor. Soil aging represents an abiotic loss process in which chemicals in soil become inaccessible for microbial degradation. Soil half-life of an organic compound can vary to a large extent depending on pre-treatment of soils before or after addition of the chemical to soil, the methodology used for soil extraction of the compound, and soil organic content. Other variables that can influence a soil half-life include vegetation coverage, weather and climate, and the presence of co-contaminants.

The organic carbon content of soil is often a major factor influencing the half-life of an organic compound. Increasing the organic carbon content of soils will increase sequestration and decrease bioavailability of organic chemicals. The amount of organic material in the soil is expressed as either organic carbon or organic matter. A conversion factor of 1.724 can be used to approximate the OC content of a soil that is expressed as OM (Northcott and Jones, 2001). The OC or organic matter (OM) contents of the soils used are identified in the summaries below if included in the study methodology. The OC content of the contaminated soil at a particular site can be taken into consideration if enough data are present to show that the OC content is a significant factor for the soil half-life of an individual chemical. A default assumption is available for the Hot Spots program, in which the fraction organic carbon in soil is 10%.

Considerable differences between field and laboratory half-life estimates have also been found for some organic chemicals such as PAHs (Doick et al., 2005). Pollutant fate studies are frequently performed under laboratory conditions and over short time periods. Field studies under realistic environmental conditions and protracted time frames probably represent a better estimate of the soil half-life and, therefore, carry more weight for estimating the soil half-life.

G.3.1 Creosotes

Creosotes are of concern primarily because of the polycyclic aromatic hydrocarbon content, which represent 85-90% of creosote constituents (Cerniglia, 1992). Therefore, in terms of soil half-life of this complex mixture, OEHHA recommends using the PAH half-life of 429 days for creosotes (see below).

G.3.2 Diethylhexylphthalate

Phthalates share the same basic structure of an esterified benzenedicarboxylic acid with two alkyl chains, and are chemically stable in the environment (Cartwright et al., 2000; Staples et al., 1997). Thus, the general absence of high concentrations of phthalates in the environment indicates the importance of biodegradative processes, specifically those mediated by microorganisms because higher organisms are unable to cleave phthalate's aromatic ring.

Metabolism of DEHP often results in the formation of the MEHP and phthalic acid. These metabolites retain some toxicological properties but are metabolized at a much faster rate than DEHP. Therefore, mineralization (i.e., ultimate degradation) of DEHP represents a reasonable and health protective indicator of the destruction of the phthalate's toxicological potential (Maag and Lokke, 1990). The very high Koc and Kow values for DEHP relative to other phthalates promote slower degradation in soil because a major fraction of this compound can eventually become strongly sorbed to soil organic material (i.e., soil aging) and therefore becomes much less bioavailable to soil microorganisms (Gejlsbjerg et al., 2001; Madsen et al., 1999).

Numerous microbial DEHP degradation studies are available in the literature, many of which measured degradation in unadulterated agricultural/garden soil. Only two studies were located in which DEHP soil degradation was investigated outdoors. In one study, DEHP-polluted sandy soil was mixed with compost topsoil and fertilizer, and then layered over a grass-covered plot (Maag and Lokke, 1990). White clover and grass were sown into the plot with four soil samples collected for analysis over 192 days. The depletion of extracted parent compound from soil roughly followed first-order kinetics with a half-life of 73 days.

In the other outdoor study, [^{14}C]DEHP was applied to sandy soil (pH 6.8, organic matter 0.3%) and potatoes planted the first year, followed by planting of barley during the second year (Schmitzer et al., 1988). Only 6.9% of the applied radiocarbon, mainly as DEHP, was recovered after 111 days when the potatoes were harvested. Nearly all the remaining activity, at least 92.3%, was lost to the atmosphere as $^{14}CO_2$. After 446 days when the barley was harvested, only 1.7% of the radiocarbon was found in the soil. A half-life was not determined, although assuming first order kinetics, the half-life would roughly be 30 days over the first four months of the study.

In a highly detailed laboratory study, Madsen et al. (1999) revealed that there are actually two phases in the mineralization of [^{14}C]DEHP in a sandy loam soil (pH 5.9, OC 2.5%) over a 130 day exposure - an initial phase during the first 30-60 days described

well by first-order kinetics, and a late phase in which mineralization activity was much lower. This second phase was thought to represent mineralization that was increasingly regulated by strong sorption to organic matter, resulting in decreased bioavailability to soil microbes. The researchers also observed mineralization was strongly regulated by temperature, with the rate of mineralization increasing with increasing temperature. To account for diurnal swings in temperature that would occur in the field, the mean half-life over the temperature range examined (5, 10 and 20 °C) was 99 days during the initial phase and 161 days during the late phase.

A similar two-phase degradation rate for [^{14}C]DEHP was observed by Roslev et al. (1998) in a sludge-amended soil (DEHP is a common contaminant in sludge). The half-life for mineralization in a sandy loam soil (pH 5.9, organic matter 2.5%) was found to increase 2.5-fold in the late phase from 58 to 147 days.

Slow degradation of DEHP has been observed in other laboratory studies. Cartwright et al. (2000) observed that only 10% of DEHP added to a sandy clay loam soil (pH 6.25, OC 3.78%) was removed by indigenous microbes by day 70. Gejlsbjerg et al. (2001) observed an average mineralization of [^{14}C]DEHP in three Danish agricultural soils (pH 6.0-6.6, OC 2.2-3.0) to be only 13% (range = 8.46 to 21.8%) over two months. In both studies, strong sorption to soil organic matter was assumed to be the reason for slow microbial degradation.

On the other hand, rapid soil degradation of DEHP has also been observed. Kirchmann et al. (1991) determined a half-life of 20-80 days for loss of parent DEHP extracted from soil (pH 7.3, OC 1.77%), although the data suggested more of a linear disappearance of DEHP with time, rather than a first order disappearance. Shanker et al. (1985) observed a half-life of 15 days for loss of parent DEHP extracted from garden soil (pH 8.2) under a relatively high incubation temperature (30 °C).

The soil half-life of DEHP can vary greatly depending on the soil conditions, with a significant amount of the parent compound eventually being sorbed to soil organic matter for long periods and becoming recalcitrant to breakdown by soil microbes. The soil half-life of 73 days based on the field study by Maag and Lokke (1990) is used here as the default soil half-life for DEHP. Similar results were obtained in comprehensive soil mineralization studies by Madsen et al. (1999) and Roslev et al. (1998), although first order kinetics were not strictly followed over the full length of the studies.

G.3.3 Hexachlorobenzene

Hexachlorobenzene is a persistent soil contaminant that does not appear to be significantly degraded in soils by either abiotic or biodegradation processes (Isensee et al., 1976; Beall, 1976). In a simulated field experiment conducted in a greenhouse, HCB applied to soil almost completely volatilized from the first two cm of soil after 19 months. However, only about 20% of the HCB was lost at a soil depth of 2-4 cm over 19 months. Only the parent compound was found in soil throughout the experiment suggesting HCB could be quite stable and persistent in a plowed field. It should be

noted that this study used a single addition of HCB to the soil and the distribution of HCB with long-term low level (deposition) is likely to be different.

A soil half-life estimate for HCB was obtained from a controlled laboratory experiment conducted in plastic-covered pots over a period of 600 days (Beck and Hansen, 1974; Bro-Rasmussen et al., 1970). Analysis for parent compound following soil extraction showed a soil half-life for disappearance of HCB to be 969-2089 days with a mean of 4.2 years. In a similar experiment, Isensee et al. (1976) observed no loss of HCB from soil in covered beakers over a one-year period.

The data show loss of HCB from soil to be primarily by volatilization with essentially no loss due to microbial degradation. It is recommended that as a default estimate, the deposition of HCB to soil in particle form be assumed to decay with a half-life of 10^8 days, similar to the metals.

HCB accumulation in the soil from airborne sources has been shown to occur in field studies. There are a couple of mechanisms that could account for this observation. HCB could partition and bind tightly onto airborne particulate matter and then be subject to deposition. Alternatively, tight binding of gaseous HCB to soil could effectively make the soil a sink for gaseous airborne hexachlorobenzene. The studies in which hexachlorobenzene is added directly to soil establish that hexachlorobenzene below a certain depth remains in the soil, presumably bound.

G.3.3 Hexachlorocyclohexanes

The α- and γ-forms of the HCHs are the most common isomers in technical grade HCH, while the β-isomer is generally the most environmentally persistent. Similar to HCB, loss of HCH deposited on soil is expected to be primarily from volatilization, although some microbial degradation has been shown to occur with the HCHs (Spencer et al., 1988; Jury et al., 1987). HCH tilled into soil will adsorb to soil organic matter significantly reducing the potential for volatilization. HCHs can undergo dehydrochlorination by soil microbes in moist, acidic-to-neutral soils (Yule et al., 1967). Anaerobic soil conditions tend to favor faster degradation over aerobic conditions (MacRae et al., 1984).

No recent soil half-life studies for HCHs conducted in the U.S. could be located. Early field studies in the U.S. suggested a soil half-life for Lindane (γ-HCH) to be on the order of months to years (Lichtenstein and Schultz, 1959; Lichtenstein and Polivka, 1959). However, the method of detection used also included detection of relatively non-toxic degradation products of Lindane. It was also unclear if offsite atmospheric deposition of HCHs onto the field plots was occurring, which can dramatically increase the apparent half-life of HCHs if not taken into account (Meijer et al., 2001).

Table G.1 Soil half-lives (days) for HCHs in subtropical environments of India.

	Singh et al., 1991[a]	Kaushik, 1989[a]	Srivastava & Yadav, 1977
α-HCH	55	-	-
γ-HCH	85	-	-
β-HCH	142	-	-
Technical HCH	-	23	44

[a] Half-lives are an average of cropped and uncropped soils

In an Indian field study, Kaushik (1989) monitored the loss of technical grade HCH sown into the top 15 cm of a field that remained fallow, and a field that contained plants and was watered regularly. The climate was characterized as subtropical, and the soil in both fields was sandy loam with a pH of 8.2 and an OC content of 0.8-1.0%. In the fallow field, the HCH half-life in the upper and lower 7.5-cm soil layers was 21 and 41 days, respectively, with a combined total half-life of 26 days. In the planted field, a total half-life of 20 days was recorded, with little difference in HCH loss observed between the upper and lower soil layers field.

In another Indian field study, Singh et al. (1991) determined the soil half-lives for several HCH isomers sown into the top 10 cm of cropped and uncropped sandy loam soil (pH 7.8; OC 0.63%) over a 1051 day period. Half-life values in the subtropical climate showed similar persistence in cropped and uncropped treatments. The longest half-life was observed for β-HCH (100 days cropped; 183 days uncropped) and the shortest half-life was observed for α-HCH (56.1 days cropped; 54.4 days uncropped). Another field study in India observed an average soil half-life of 44 days (range: 35 to 54 days) for a low concentration of technical grade HCH applied under cover of maize crop over three years of planting (Srivastava and Yadav, 1977).

Researchers have noted that the soil half-life for HCHs estimated in tropical climates likely underestimates the half-life for HCHs in cooler, temperate climates of the U.S. due to greater volatility, and probably higher microbial degradation, at warmer temperatures (Singh et al., 1990; Kaushik, 1989). Because temperate climate of California will tend toward lower volatility of HCHs from soil, the longer HCH half-lives determined by Singh et al. (1991) in Table G.1 are recommended for use in the “Hot Spots” program. If the HCH isomer profile in the soil is unknown, an average of the three isomer soil half-lives (94 days) can be used.

G.3.4 4,4'-Methylenedianiline

Cowen et al. (1998) investigated biodegradation of 4,4'-methylenedianiline under aerobic and anaerobic conditions using ^{14}C labeled methylenedianiline. The data showed that, after 365 days of aerobic biodegradation in silt loam soil, 59.9% of 4,4'-methylenedianiline remained intact. Based on the aerobic biodegradation data from this study, using first-order kinetics default for dissipation of chemicals, OEHHA derived a soil half life of 455 days for 4,4'-methylenedianiline.

G.3.5 Polychlorinated Biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are a mixture of chlorinated biphenyl congeners that vary in the degree of chlorination. The degree of chlorination has a major impact on soil half-life. Several different mixtures were marketed and used widely before PCBs were banned because of their toxicity, environmental persistence and bioaccumulative properties. Small amounts are generated as combustion byproducts and these emissions are subject to the Hot Spots program. The toxicity of individual congeners varies widely. For these reasons, meaningful overall soil half-life for PCBs is difficult to ascertain for situations in which PCB emissions are not speciated and the cancer potency factor for the entire mixture is applied. A half-life of 940 days for Aroclor 1254 was derived by Hsieh et al. (1994). This value is used by the Department of Toxic Substances Control in CalTOX. In 2000, OEHHA proposed to use this value for all Aroclor mixtures and airborne emissions of unspeciated PCB mixtures generated from Hot Spots facilities.

Harner et al. (1995) studied four PCB congeners (28, 52, 138, 153) in air, herbage, and soil of the southern U.K. over the period 1942-1992 and observed soil half-lives ranging from 7 to 25 years (mean 18 years) (6570 days). Wania and Daly (2002) estimated soil half-lives of seven PCB congeners (8, 28, 52, 101, 153, 180, 194) ranging from 550 hours (23 days) to 1,700,000 hours (70,833 days).

Sinkkonen and Paasivirta (2000) suggested soil half-lives for eleven PCB congeners, ranging from 26,000 hours (1,083 days) to 330,000 hours (13,750 days), based on the work of Lake et al. (1992), Beurskens et al. (1993) and Brown et al. (1984).

Doick et al. (2005) studied long-term fate of two PCBs in an agricultural soil in Germany. Their observation over 152 months concluded that the soil half-lives were 10.9 years (3979 days) for PCB 28 and 11.2 years (4088 days) for PCB 52. The authors attributed the much longer soil half-lives of PCBs than estimates in other studies to length of study, field study conditions, vegetation (type and coverage), weather and climate, the presence of co contaminants and, particularly, soil type -- a high silt, high clay content, "heavy" soil with reduced water infiltration, compared with higher porosity, sandy soils.

There is great variability in soil half-lives among the PCB congeners in the above studies. The OEHHA adopted Toxicity Equivalency Factors (TEF) for individual PCB congeners (WHO97-TEF) (OEHHA 2003a); thus, it is appropriate to apply the soil half-life data for these individual congeners where speciation of PCBs has been performed

on facility emissions. Based on the studies above, only the data for PCB congeners with a WHO TEF (IUPAC # 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 189) were used for estimating soil half-lives in this document, unless only total PCBs are available (OEHHA 2003b).

Among the above studies, Lake et al. (1992) derived a half-life of 7.5 years for PCB 105 and 6.8 yrs for PCB 118, using the anaerobic dechlorination reaction in sediment of 15-17.5 cm deep from New Bedford Harbor, Connecticut. Beurskens et al. (1993) have estimated a half-life time of nine years for PCB 105, PCB 126, PCB 156 and PCB 169 in the anaerobic sediment. Brown et al. (1984) found the average elimination half-life for PCB 105 and PCB 118 in Hudson River sediments was 10 years. The OEHHA acknowledges that the degree of biodegradation in sediment would be different from that for a dry land scenario. Until studies in dry soil become available, the river sediment data appear to be the best choice.

Table G-2. Soil half-lives (days) for PCBs (IUPAC #) relevant to the "Hot Spots" program

Study	105	118	126	156	169	Total PCBs
Lake et al. 1992	2738	2482				
Beurskens et al., 1993	3285		3285	3285	3285	
Brown et al. 1984	3650	3650				
Arithmetic mean half-lives	3224	3066	3285	3285	3285	3229

The arithmetic mean half-lives for each PCB are shown at the bottom of Table G-2, and a grand mean half-life including all studied PCBs is 3229 days. This overall half-life of 3229 days is recommended as the estimated soil half-life for PCBs.

G.3.6 Polycyclic Aromatic Hydrocarbons (PAHs)

There are a variety of polycyclic aromatic hydrocarbons emitted from combustion sources. The structures vary by number and placement of fused aromatic carbon rings and functional groups on those rings. In general, it has been observed that the soil half-life increases with the increasing number of fused rings on a PAH and is correlated directly with molecular weight and K_{ow} (Northcott and Jones, 2001; Wild and Jones, 1993). The PAHs currently of toxicological concern under the "Hot Spots" program consist almost entirely of four or more rings with the prototype PAH, benzo(a)pyrene, containing five fused benzene rings. Naphthalene is carcinogenic and only has two rings but it is too volatile to be a multipathway chemical subject to deposition. Therefore, OEHHA chose to base the soil PAH half-life on those compounds with greater than three rings to avoid underestimating the accumulation of the carcinogenic PAHs in the soil.

Studies where PAHs have been added to soil have noted that those PAHs with three rings or less show significant volatilization from soil and microbial degradation, whereas PAHs with greater than three rings show little or no volatilization and slower microbial degradation (Wild and Jones, 1993; Cerniglia, 1992). In addition, a broad inverse relationship has been observed between the rate of biodegradation and the organic carbon (OC) content of the soil (Northcott and Jones, 2001; Wild and Jones, 1993). Soil half-life estimates for PAHs that currently have a potency equivalency factor (PEF) were given the greatest weight in determining a default soil half-life. Table G-3 shows the PAH half-life results from the most comprehensive studies found in the literature and a brief summary of the studies is given below.

Doick et al. (2005) conducted a field study and determined the long-term fate of ^{12}C and ^{14}C analogues of benzo[a]pyrene spiked in a cultivated agricultural soil subject to typical agricultural practices. The soil had a pH=7.2 and an organic matter content of 2.2%. Their observation over 152 months found that the soil half-life for benzo[a]pyrene was 2.7 years (982 days). These half-life values are much longer than estimates in other studies and are thought to be a result of the soil type, length of the study, use of field conditions rather than laboratory conditions, and vegetation (type and coverage).

Sewage sludge containing PAHs was applied to two agricultural soils at five dose levels (30 to 600 ton/ha) in field plots, followed by cultivation with annual crops or a perennial (willow) for up to 54 months (Oleszczuk and Baran, 2005). It was unclear from the description of the methodology if this work was an actual field study. Before addition of the sewage sludge, the soil with the annual crops had a pH=4.3 and a total organic carbon (OC) content of 1.12%. The soil with the perennials had a pH=5.8 and a total OC content of 1.21%. Analysis of 16 PAHs showed longer half-lives in the soil with the annual crops. However, the sewage sludge properties were considered as important as the type of crop used. The investigators suggested that longer half-lives of PAHs compared to other studies may have occurred due to the increased soil aging process in a soil-sludge matrix.

In a climate-controlled greenhouse experiment, sewage sludge containing PAHs was applied to four different soils to determine the soil half-life for a number of individual PAHs (Wild and Jones, 1993). The four soils ranged from a sandy clay loam agricultural soil (pH=6.6, organic carbon content, 6.04%) to a coniferous forest soil (pH=2.9, organic carbon content, 58%). Although the half-lives among 12 PAHs measured in the forest soil tended to be longest, the overall average of the sum of the PAH half-lives was not considerably higher in forest soil ($t_{1/2}$=192 d) compared to the overall average of the sum of the half-lives in the agricultural soils ($t_{1/2}$=146 d and 165 d) and a roadside soil (177 d). The authors noted that the controlled environmental conditions in the greenhouse optimize biodegradation compared to field conditions, and likely results in more rapid losses of PAHs from the soil.

Two different sandy loam soils were spiked with 14 PAHs in incubation chambers and their soil half-lives estimated over an exposure period of up to 196 days (Park et al., 1990). One soil (Kidman sandy loam) had a pH=7.9 and an OC content of 0.5%, and the other soil (McLaurin sandy loam) had a pH=4.8 and an OC content of 1.1%. The

half-lives for PAHs with PEF values ranged from 24 days to 391 days. Although the organic content and pH of the two soils differed, the biological degradation rates of the PAH compounds were not statistically different between the two soils.

In another laboratory study, Coover and Sims (1987) spiked a sandy loam agricultural soil (pH=7.9; OC content, 0.5%) with 16 PAHs and estimated the soil half-lives over a 240 day incubation period. Increasing the soil temperature was observed to increase the apparent loss of low molecular weight PAHs but had little effect on loss of five- and six-ring PAHs.

Table G.3 Soil half-lives (days) for PAHs relevant to the "Hot Spots" program

Study	Ch	BaA	BaP	BbF	BkF	DahA	DaiP	Ind	DaA
Coover & Sims, 1987[a]	1000	430	290	610	1400	750		730	
Park et al., 1990[b]	379	212	269	253		391	297	289	24
Wild & Jones, 1993[c]	215	215	211	202	301				
Doick et al., 2005			982						
Arithmetic mean half-lives	531	286	438	355	851	571	297	510	24

Abbreviations: Ch, chrysene; BaA, benz[a]anthracene; BaP, benzo[a]pyrene; BbF, benzo[b]fluoranthene; BkF, benzo[k]fluoranthene; DahA, dibenz[a,h]anthracene; DaiP, dibenzo[a,i]pyrene; Ind, Indeno[1,2,3-c,d]pyrene; DaA, 7,12-Dimethylbenz [a] anthracene

[a] Environmental temperature held at 20C

[b] Average half-life values for two sandy loam soils

[c] Average half-life values for four different soils. Ch and BaA co-eluted; the $t_{1/2}$ is for both PAHs combined

The arithmetic mean half-lives for each PAH are shown at the bottom of Table G.3, and a grand mean half-life including all PAHs is 429 days. Greater differences in PAH half-lives are seen between studies rather than within studies. One possible reason is that longer half-lives are attained from field studies (Doick et al., 2005) compared to laboratory studies (Coover & Sims, 1987; Park et al., 1990; Wild & Jones, 1993).

However, the limited number of field studies makes it difficult to confirm this assumption. The overall PAH half-life of 429 days is recommended until further field studies are conducted.

G.3.7 Polychlorinated Dibenzo-p-dioxins and Dibenzofurans (PCDD/F)

The prototype compound and most potent of the dioxin and furan family of compounds is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). The degree and placement of chlorination affects both the toxicity and soil half life of dioxins and furans. Sampling of 32 sites in Seveso, Italy, produced an initial calculated regression half-life of one year (365 days) (Di Domenico et al., 1980). Experimental application of TCDD to two different soil types (loamy sand and silty clay loam) for 350 days produced calculated half-life values ranging from 394 to 708 days (Kearney et al., 1972; Kearney et al., 1973). Soil half-life estimates ranging from 10 to 12 years (3650-4380 days) were reported based upon experimental measured soil concentrations of TCDD from a contaminated site at an Air Force base in Florida (Young, 1981). Soil half-life estimates of 10 to 100 years (3650-36500 days) were reported, depending on the depth of the contamination, with deeper soil having reduced biodegradation rates (Nauman and Schaum, 1987). An estimated soil half-life of 3609 days has also been reported (calculated from a soil reaction rate constant of 8×10^{-6} hr^{-1}) (Mackay et al., 1985).

Several other half-life estimates have also been identified and summarized (Cohen et al., 1994b). Soil samples showing loss of TCDD content by volatilization produced estimated half-lives of 7-24 days (Nash and Beall, 1980). TCDD measured in soils from the contaminated site in Seveso, Italy, produced a half-life estimate of 9.1 years (3322 days) (Cerlesi et al., 1989). A half-life estimate of 3 days was made based on loss of TCDD content from soil by both photodecomposition and volatilization (Di Domenico et al., 1982).

McLachlan et al. (1996) studied PCDD/F persistence in a sludge-amended soil sample with presence of PCDD/Fs from 1968 to 1990. Half-lives for these PCDD/Fs in the sludge-amended soil after 1972 were of the order of 20 years (7300 days).

The arithmetic mean of the suggested values from ten studies (6,986 days) cited above is recommended as the estimated soil half-life of PCDD/Fs if the facility is reporting emissions for all dioxins and furan congeners as total PCDD/Fs.

There is great variability in soil half-lives among the PCDD/F congeners among the above studies. Soil half-life estimates for PCDD/Fs that currently have a toxicity equivalency factor (TEF) were given the greatest weight in determining a default soil half-life, where speciation of PCDD/Fs has been performed on facility emissions, unless only total PCDD/Fs are available (OEHHA, 2003). Table G-4 shows the PCDD/F half-life results from the study (Kjeller and Rappe, 1995) found in the literature which speciated PCDD/F congeners in sediment.

Table G.4. Half-lives (days) for PCDD/Fs in sediment

Compound	TEF_{WHO-97}	Half-life (days) from Kjeller and Rappe (1995)
PCDDs		
2378-TCDD	1	37,500
12378-PeCDD	1	42,000
123478-HxCDD	0.1	100,000
123789-HxCDD	0.1	29,200
123678-HxCDD	0.1	23,000
1234678-HpCDD	0.01	37,500
12346789-OCDD	0.0001	54,200
PCDFs		
2378-TCDF	0.1	23,000
12378-PeCDF	0.05	18,750
23478-PeCDF	0.5	23,000
123478-HxCDF	0.1	25,000
123789-HxCDF	0.1	20,800
123678-HxCDF	0.1	29,200
234678-HxCDF	0.1	18,750
1234678-HpCDF	0.01	14,600
1234789-HpCDF	0.01	12,500
12346789-OCDF	0.0001	10,400
Arithmetic mean half-lives		30,600

G.3.8 Summary

The chemical-specific soil half-lives for each chemical are summarized as Table G-5 below.

Table G-5. Summary of Soil Half-life Values (Days).

Compound	Soil Half-life (days)
Arsenic	1.0 E+08
Beryllium	1.0 E+08
Cadmium	1.0 E+08
Chromium	1.0 E+08
Diethylhexylphthalate	1.5 E+01
Fluoride	1.0 E+08
Hexachlorobenzene	1.0 E+08
Hexachlorocyclohexanes	9.4 E+01
Lead	1.0 E+08
Mercury	1.0 E+08
4,4'-methylenedianiline	4.6 E+02
Pentachlorophenol	-[a]
PAHs	4.3 E+02
PCBs	3.2 E+03
PCDD/F	7.0 E+03
Selenium	1.0 E+08

[a] To be assessed for soil half-life

For a chemical with individual congeners, such as PCBs, PAHs, PCDD/Fs, only the grand average was presented in Table G-5. When speciation of these chemicals in soil has been performed on facility emissions, soil half-life data for individual congeners are summarized in Table G-2 (PCBs), and Table G-3 (PAHs).

G.4 References

Bartlett, R J (1991). Chromium Cycling in Soils and Water: Links, Gaps, and Methods. Environmental Health Perspectives 92: 17-24.

Bartlett, R J. and James, B.R. (1988). Mobility and bioavailability of chromium in soils. In: Chromium in the Natural and Human Environments. Nriagu, J.O. and Nierboor, E. (eds). John Wiley and Sons, New York, pp.267-383.

Beall, M L, Jr. (1976). Persistence of aerially applied hexachlorobenzene on grass and Soi" J Environ Quality 5(4): 367-369.

Beck, J. and Hansen, K.E. (1974). The degradation of quintozene, pentachlorobenzene, hexachlorobenzene, and pentachloroaniline in soil. Pesticide Science, 5:41-8.

Beurskens, J.E.M., Mol, G.A.J., Barreveld, H.L., van Munster, B., Winkels, H.J. (1993). Geochronology of priority pollutants in a sedimentation area of the Rhine River. Environ. Toxicol. Chem. 12, 1549-1566.

Bro-Rasmussen, F E. Noddegaard, et al. (1970). Comparison of the disappearance of eight organophosphorus insecticides from soil in laboratory and in outdoor experiments. Pesticide Science 1(5): 179-182.

Brown Jr., J F, Wagner, RE, Bedard, D L, Brennan, M J, Carnahan J C, May, R J, 1984. PCB transformation in upper Hudson sediments. Northeastern Environ. Sci. 3, 166-178.

Cartwright, C D, Thompson I P (2000). Degradation and impact of phthalate plasticizers on soil microbial communities. Environ Toxicol and Chem 19(5): 1253-1261.

Cerlesi S., Domenico A., and Ratti S. (1989). 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) persistence in the Seveso (Milan, Italy) soil. Ecotoxicol Environ Safety, 18:149-64.

Cerniglia C E (1992). Biodegradation of polycyclic aromatic hydrocarbons. Biodegradation 3(2): 351-368.

Cohen Y, Winer A M, Creelman L. (1994a). Development of intermedia transfer factors for hexavalent chromium. Final report prepared for the Air Resources Board in fulfillment of contract number A032-170.

Cohen Y, Winer A M, Pesinova V. (1994b). Development of intermedia transfer factors for 2,3,7,8-TCDD. Final report prepared for the Air Resources Board in fulfillment of contract number A032-170.

Coover, MP, Sims R C C. (1987). The effects of temperature on polycyclic aromatic hydrocarbon persistence in an unacclimated agricultural soil. Hazardous Waste and Hazardous Materials, 4:69-82.

Cowen, W. F., A. M. Gastinger, et al. (1998). Sorption and Microbial Degradation of Toluenediamines and Methylenedianiline in Soil under Aerobic and Anaerobic Conditions. Environmental Science & Technology 32(5): 598-603.

Di Domenico A, Silano V, Viviano G, Zapponi G. (1980). Accidental release of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at Seveso, Italy. V. Environmental persistence of TCDD in soil. Di Domenico, A., Viviano, G., and Zapponi, G. (1982). Environmental persistence of 2,3,7,8-TCDD at Seveso. In: Chlorinated Dioxins and Related Compounds: Impact on the Environment. pp.105-14.

Doick, K J, Klingelmann E. (2005). Long-term fate of polychlorinated biphenyls and polycyclic aromatic hydrocarbons in an agricultural soil. Environl Science Technol 39(10): 3663-3670.

Fairbrother, A, Wenstel R, (2007). Framework for metals risk assessment. Ecotoxicol Environ Safety 68(2): 145-227.

Fendorf, S., M. J. La Force, et al. (2004). Temporal changes in soil partitioning and bioaccessibility of arsenic, chromium, and lead. J Environl Quality 33(6): 2049-2055.

Gejlsbjerg, B., C. Klinge, et al. (2001). Mineralization of organic contaminants in sludge-soil mixtures. Environ Toxicol Chem 20(4): 698-705.

Gochfeld, M,. Whitmer C.. (1991). A research agenda for environmental health aspects of chromium. Environ Health Perspect, 92:141-4.

Harner, T, Mackay D.. (2002). Model of the Long-Term Exchange of PCBs between Soil and the Atmosphere in the Southern U.K. Environ Science Technol 29(5): 1200-1209.

Hsieh, D P H., McKone, TE, Chiao F, CurrieR C, Kleinschmidt, L. (1994). Final Draft Report: Intermedia transfer factors for contaminants found at hazardous waste sites. Prepared for the Office of Scientific Affairs, Department of Toxic Substances Control, California Environmental Protection Agency. November, 1994.

Isensee, A R, Holden E R, (2002). Soil persistence and aquatic bioaccumulation potential of hexachlorobenzene (HCB). Journal of Agricultural and Food Chemistry 24(6): 1210-1214.

Jury, W A, Focht D. D. (1987). Evaluation of pesticide groundwater pollution potential from standard indices of soil-chemical adsorption and biodegradation. J Environ Quality 16(4): 422-428.

Kaushik, C P (1989). Loss of HCH from surface soil layers under subtropical conditions. Environ Pollution 59(3): 253-264.

Kearney, PC, Woolson, EA , and Ellington, CP (1972). Persistence and metabolism of chlorodioxins in solids. Environ Sci Technol, 5:273-7.

Kearney, PC, Woolson E A, Isensee AR, Helling C S. (1973). Tetrachlorodibenzodioxin in the environment: sources, fate, and decontamination. Environ Health Perspect, 5:273-7.

Kelsey, J W, Kottler, B D, Alexander, M. (1997). Selective chemical extractants to predict bioavailability of soil-aged organic chemicals. Environ. Sci. Technol. 31:214-217.

Kirchmann H., Astrom H., and Jonsall G. (1991). Organic pollutants in sewage sludge. 1. Effect of toluene, naphthalene, 2-methylnaphthalene, 4-n-nonylphenol and di-2-ethylhexyl phthalate on soil biological processes and their decomposition in soil. Swedish J. Agric. Res. 21:107-113.

Kjeller, L.-O. and C. Rappe (1995). Time yrends in levels, patterns, and profiles for polychlorinated dibenzo-p-dioxins,dibenzofurans, and biphenyls in a sediment core from the baltic proper. Environ Science Technol 29(2): 346-355.

Lake, J L, Pruell R J, Osterman F A., 1992. An examination of dechlorination processes and pathways in New Bedford Harbor sediments. Mar. Environ. Res. 33, 31-47.

Lichtenstein, E P, Polivka J B. (1959). Persistence of Some Chlorinated Hydrocarbon Insecticides in Turf Soils 1. Journal of Economic Entomol 52: 289-293.

Lichtenstein, E P, Schulz, K R. (1959). Persistence of some chlorinated hydrocarbon insecticides as influenced by soil types, rate of application and temperature1,2. J Economic Entomol 52: 124-131.

Maag J, Lokke H. (1990). Land Farming of DEHP Contaminated Soil, in Contaminated Soil '90, Eds. Arendt, F., Hiusenveld, M., Van den Brunk, W. J. Kluwer Academic Pulishers, Netherlands, pp. 975-982.

Mackay D, Paterson S, Cheung B, Neely W B. (1985). Evaluating the environmental behavior of chemicals with a level III fugacity model. Chemosphere, 14:335-74.

McLachlan, M. S., A. P. Sewart, et al. (1996). Persistence of PCDD/Fs in a sludge-amended soil. Environ Science Technol 30(8): 2567-2571.

MacRae IC, Y Yamaya, T Yoshida. (1984) Persistence of hexachlorocyclohexane isomers in soil suspensions. Soil Biol. Biochem. 16:285-6.

Madsen P L, Thyme J B. (1999). Kinetics of di-(2-ethylhexyl)phthalate mineralization in sludge-amended soil. Environ Science Technol 33(15): 2601-2606.

Meijer S N, Halsall C J. (2001). Organochlorine pesticide residues in archived UK. Soil. Environ Science Technol 35(10): 1989-1995.

Nash R G, Beall, M L (1980). Distribution of silver, 2,4-D and TCDD applied to turf in chambers and field pots. J Agric Food Chemistry, 28:614-23.

Nauman, C.H. and Schaum, J.L. (1987). Human exposure estimation for 2,3,7,8-TCDD. Chemosphere, 16:1851-6.

Northcott, G L, Jones K C. (2001). Partitioning, extractability, and formation of nonextractable PAH residues in soil. 1. compound differences in aging and sequestration. Environ ScienceTechnology 35(6): 1103-1110.

OEHHA, 2003. Technical Support Document for Describing Available Cancer Potency Factors, Appendix A (revised August, 2003) : Use of the Revised Toxicity Equivalency Factor (TEFWHO-97) Scheme for Estimating Toxicity of Mixtures of Dioxin-Like Chemicals, September 2003. Available at **www.oehha.ca.gov**.

Park, K S, Sims, R C. (1990). Fate of PAH Compounds in two soil types: influence of volatilization, abiotic loss and biological activity. Environ Toxicol chem 9(2): 187-195.

Reid, B. J., K. C. Jones, and K. T. Semple. 2000. Bioavailability of persistent organic pollutants in soils and sediments—a perspective on mechanisms, consequences and assessment. Environ. Pollut. 108:103-112.

Roslev P, Madsen P L. (1998). Degradation of phthalate and di-(2-ethylhexyl)phthalate by indigenous and inoculated microorganisms in sludge-amended soil. Applied Environ Microbiology 64(12): 4711-4719.

Schmitzer, J.L, Scheunert I, Korte F. (1988). Fate of bis(2-ethylhexyl) [^{14}C] phthalate in laboratory and outdoor soil-plant systems. J. Agric. Food Chem. 36: 210-215

Shanker R C, Ramakrishna, PK . (1985). Degradation of some phthalic acid esters in soil. Environ. Pollut. Ser. A. 39:1-7.

Singh G, Kathpal T S, Spencer W F, et al. (1991). Dissipation of some organochlorine insecticides in cropped and uncropped soil. Environ Pollut 70:219-240.

Sinkkonen S, Paasivirta J. (2000) Degradation half-life times of PCDDs, PCDFs and PCBs for environmental fate modeling. Chemosphere 40(9-11): 943-949.

Spencer W F, Cliath M M. (1988). Volatilization of organic chemicals from soil as related to their Henry's law constants. J EnvironQuality 17(3): 504-509.

Srivastava, BP, Yadav PR. (1977). Dissipation of BHC in clay loam soil under the cover of maize (Zea mays) crop. Ind. J. Plant Protec., 5, 62-69.

Staples C A, Peterson D R. (1997). The environmental fate of phthalate esters: A literature review. Chemosphere 35(4): 667-749.

Stewart, M A, Jardine P. M. (2003). Effects of contaminant concentration, aging, and soil properties on the bioaccessibility of Cr(III) and Cr(VI) in soil. Soil Sediment Contamin: An International J 12(1): 1-21.

Tang J, Alexander M. (1999). Mild extractability and bioavailability of polycyclic aromatic hydrocarbons in soil. Environ. Toxicol. Chem. 18(12):2711–2714.

US EPA (Environmental Protection Agency), 2003. Framework for Cumulative Risk Assessment. Risk Assessment Forum, Washington, DC (EPA/630/P-02/001F). Available online at: http://oaspub.epa.gov/eims/eimscomm.getfile?p_download_id=36941

Walkley, A. and Black, I.A. (1934). An examination of the Degtjareff method for determining soil organic matter and a proposed modification of the chromic acid titration method. Soil Science, 37:29-38.

Wania, F, Daly G L. (2002). Estimating the contribution of degradation in air and deposition to the deep sea to the global loss of PCBs. Atmos Environ 36(36-37): 5581-5593.

Wild S R, Jones K C. (1993). Biological and abiotic losses of polynuclear aromatic hydrocarbons (PAHs) from soils freshly amended with sewage sludge. Environ Toxicol Chem 12(1): 5-12.

Young A L. (1981). Long-term studies on the persistence and movement of TCDD in a natural ecosystem. In: Human and Environmental Risks of Chlorinated Dioxins and Related Compounds. Tucker, R.E., Young, A.L., and Gray, A.P. (eds). Plenum, New York.

Yule, W. N., M. Chiba, et al. (2002). Fate of insecticide residues. Decomposition of lindane in soil. Journal of Agricultural and Food Chemistry 15(6): 1000-1004.

Appendix H. Root Uptake Factors

H.1 Introduction

Root uptake factors for crops have been estimated for toxic metals in the "Hot Spots" program. These toxic metals are subject to soil deposition and subsequent uptake by the roots of home raised produce. A root uptake factor is necessary to estimate a concentration in the plant from the concentration in the soil. An estimate of produce consumption can be applied to estimate dose to the residential receptor (Chapter 7). The soil-to-plant uptake factor (UF) is the ratio of the fresh weight contaminant concentration in the edible plant or plant part over the total concentration of the contaminant in wet weight soil:

$$UF = C_{f.w.plant} / C_{wet.w.\ soil} \qquad \textbf{(Eq. H-1)}$$

where: $C_{f.w.plant}$ = fresh weight concentration in the plant (mg/kg)

$C_{wet.w.\ soil}$ = wet weight concentration in soil (mg/kg)

In the last 25 years, a large number of studies have been published that investigated metal concentrations in edible plants grown in contaminated soils. Although most of these studies did not calculate the UF, data were often presented from which a UF could be calculated. OEHHA assembled the data from these studies into a database from which basic statistical analyses for chemical UFs were determined. The volume of studies that could be included in the database is quite large for some inorganic metals, with new studies frequently published. Our database is not an exhaustive compilation of all plant uptake studies published, however, enough data were found to reasonably estimate default UFs for most of the toxic metals and metalloids of concern.

The UFs calculated by OEHHA are based on the total metal concentration in soil and reflect the fact that most crop uptake studies estimate total metal soil concentration, usually by extraction with strong or moderately strong acids (e.g., 4 N sulfuric acid). A smaller body of uptake studies uses various mild soil extraction processes (e.g., extraction with diethyltriaminopentaacetic acid) to estimate plant bioaccessible metal concentrations in soil. Once more studies become available using an established method for estimating bioaccessible metals in contaminated soil, OEHHA may also consider developing an algorithm that incorporates a bioaccessible metal uptake factor.

The ability for crops to accumulate and translocate toxic inorganic metals and metalloids to edible parts depends to a large extent on soil and climatic factors, plant genotype and agronomic management (McLaughlin et al., 1999). In order to be most applicable to Hot Spots risk analysis, a set of criteria was applied for the selection of data used in developing soil-to-plant uptake factors.

Data used to determine root uptake factors were limited to studies that estimated contaminant concentrations in edible portions of crops raised and harvested at maturity

for human consumption. Crops that are commonly grown in backyard gardens in California were considered most relevant. For example, plant uptake studies in crops grown in tropical climates were not included in the database. Grain crops such as wheat and rice were also not included in the database because these crops are unlikely to be grown in backyard gardens. In most field studies background soil contaminant levels were unknown or not presented. However, field studies were included in the database if the study indicated that the soil was contaminated due to human causes, or that the soil contaminant concentration was considered above background levels.

Another data selection factor was soil pH because soil pH is a major influence on root uptake. Most agricultural soils in California are near neutral, with a geometric mean pH=7.2 (Holmgren et al., 1993). The range of pHs for most agricultural soils in California are roughly estimated at between 5.5 and 7.6. Thus, plant uptake studies that investigated soils with pH values within this range were considered most useful for estimating soil-to-crop uptake factors. Acidic soils tend to increase the bioavailability of divalent cationic metals such as cadmium, lead, and mercury. UFs based on acidic soils may overestimate metal uptake from pH neutral soils.

A distinction is made in the database for contaminant source between freshly added inorganic salts and other forms of the chemicals. In general, fresh addition of metal salts to soil in laboratory experiments will represent the most available form of the metal to plants. UFs developed from these studies likely represent an upper limit for plant accumulation. Where possible, UFs were calculated based on field studies that estimated plant uptake due to human-caused contamination of soils. These sources primarily included mine waste, smelter deposits, vehicle and other urban emissions, other industrial sources, wastewater effluent, compost, fertilizer, dredged material, sewage sludge, fly ash and flue dust. Ideally, UFs would be based on airborne deposition of contaminants due to emissions from nearby industrial facilities. However, uptake data from these sources were often very limited.

Most of the plant uptake studies summarized in the database presented their contaminant concentration results on a dry weight basis for both the plants and the soil. However, the soil-to-plant UF in Eq. 7.6 (Chapter 7) is expressed as a ratio of fresh weight crop concentration per wet weight soil concentration. To adjust the soil-to-plant UFs to a fresh weight crop basis, dry-to-wet weight fractions of edible portions of crops were applied using literature sources containing water content data of raw fruits and vegetables (Watt and Merrill, 1975; Baes et al., 1984; USDA, 2009). A default value of 0.8 was applied to all UFs for the dry-to-wet weight adjustment of soil, unless water content data of soil was presented in the study (Clement Associates, 1988).

As a result, two types of soil-to-plant UFs can be generated for each metal contaminant: one based on the dry weight plant over dry weight soil, and the other based on fresh weight plant over wet weight soil. A UF based on dry weights of plant and soil may be beneficial because the ratio avoids the naturally wide variations in water content of the crops and the soil. On the other hand, estimates of fruit and vegetable consumption are based on fresh weight values for the crops, which were grown in irrigated soils. This

type of UF is most applicable for contaminant exposure via the crop consumption pathway (Eq. 7.6).

Finally, some studies also presented uptake data for reference soils. This information was also entered into the database to estimate crop uptake based on control soils as well as crop uptake specifically due to deposited contaminants (i.e., contaminated soil minus control soil metal concentration). Metals of concern naturally present in soils may be largely present in the mineral fraction of the soil and not available for uptake by plants. However, it may be beneficial to know what the background soil-to-plant UF is for toxic metals to estimate the impact of anthropogenic sources of the same metals is on the soils and plants.

The database of the studies used in the analysis is presented at the end of this appendix. Studies were grouped according to each metal/metalloid for comparison purposes.

H.2 Arsenic

Arsenic can be present in well-drained soil as $H_2AsO_4^{-1}$ if the soil is acidic or as $HAsO_4^{-2}$ if the soil is alkaline (Bhumbla and Keefer, 1994). Arsenite (As(III)), the reduced state of inorganic arsenic, is a toxic pollutant in natural environments. It is much more toxic and more soluble and mobile in soil than the oxidized state of inorganic arsenic, arsenate (As(V)). Under flooded conditions, As(III) would dominate, whereas aerobic conditions would favor the oxidation of As(III) to As(V). Arsenic accumulates in roots of plants grown on soils contaminated by arsenic pesticides. However, arsenic is not readily translocated to above-ground parts.

Although background mean levels of arsenic in U.S. agricultural soils could not be located, a review by Wiersma et al. (1986) showed mean levels of arsenic in European and Canadian agricultural soils to be in the range of 5 to 12 mg/kg dry soil. Kloke et al. (1984) reports that the range of arsenic in arable land to be 0.1 to 20 mg/kg dry soil. The typical dry weight concentration of arsenic in plants has been listed as 0.1 to 5 mg/kg (Vecera et al., 1999). In this document, all crops grown in As-polluted soils had an overall average dry weight arsenic concentration of about 2.5 mg/kg, which is within the range of typical plant concentrations.

Table H.1 Distribution Parameters for Arsenic Fresh Weight Soil-to-plant Uptake Factors

	Leafy	Exposed	Protected	Root
n	27	22	8	17
minimum	0.000275	0.0000538	0.000115	0.000338
maximum	0.055	0.132	0.27	0.045
mean	0.00983	0.0158	0.066	0.00828
median	0.00531	0.00138	0.032	0.00399
90^{th} percentile	0.0257	0.0403	0.19	0.0236
95^{th} percentile	0.0481	0.0674	0.23	0.0361

It was observed that lower UFs were recorded in plants growing in high As-polluted soils compared to plants growing in low-level As-polluted soils. This finding, in part, led to the large range in UF values shown in Table H.1 for some types of crops. For example, in soils with low-level As contamination of < 12 mg/kg, a UF of 0.01 was calculated for both exposed and leafy crops. In exposed and leafy crops grown in soils with >12 to 745 mg/kg As (mean: 343 mg/kg), calculated UFs were 0.0002 and 0.002, respectively. This seems to suggest that many crops have the ability to resist uptake, or have a high excretion rate, of excessive amounts of As in highly polluted soils. The crop UFs in Table H.1 are based on the arithmetic mean value for low- to high-level As polluted soils.

H.3 Beryllium

Very little data could be found regarding plant uptake of beryllium from the soil. Measurable amounts of beryllium in plants are rarely observed and the toxicity of this metal to plants is reported to be high (Shacklette et al., 1978; Baes et al., 1984). Kloke et al. (1984) estimates that a general dry weight plant/soil transfer coefficient for Be is in the range of 0.01 - 0.1, similar to that found for lead and mercury.

Single soil-to-plant data points from Baes et al. (1984) for leafy and protected crops were used in Table 7-6 to represent these particular crop types. These were the only UFs that could be located in the literature. Due to expected similarities in soil-to-plant transfer, the lead UFs for root and exposed crops were used to represent the root and exposed UFs for beryllium.

H.4 Cadmium

Cadmium has the most extensive literature on root uptake of any of the toxic metals Compared to Pb, Cd is readily taken up by plants, but unlike the other heavy metals, Cd is not phytotoxic at low plant concentrations that pose a concern to human health (McLaughlin et al., 1999). Cadmium exists in solution mostly as the divalent cation, Cd^{2+}. Plant uptake of Cd is governed by a number of factors that include soil pH, organic matter, cation exchange capacity, clay type and amount, hydrous metal oxides, carbonates, and other inorganic compounds (Mahler et al., 1987; McLaughlin et al., 1996). Acidic soils, and soils with lower clay and humus content will increase availability of Cd to plants.

The mean concentration of Cd in uncontaminated U.S. agricultural soils is 0.27 mg/kg d.w., with 5th and 95th percentiles of 0.036 and 0.78 mg/kg d.w., respectively (Holmgren et al., 1993). The mean concentration of Cd for field-contaminated soils reviewed in this document was about 8 to 9 mg/kg d.w., with a range of 0.16 to 106.5 mg/kg d.w. Typical dry weight levels of Cd in plants are expected to be between 0.1 and 1 mg/kg (Vecera et al., 1999). In this document, the overall Cd concentration in crops grown in Cd-polluted soil was about 6 mg/kg.

Figure H.1. Cumulative distribution of the leafy crop UFs for cadmium from field studies in the literature (n=73, skewness=3.05, kurtosis=9.09)

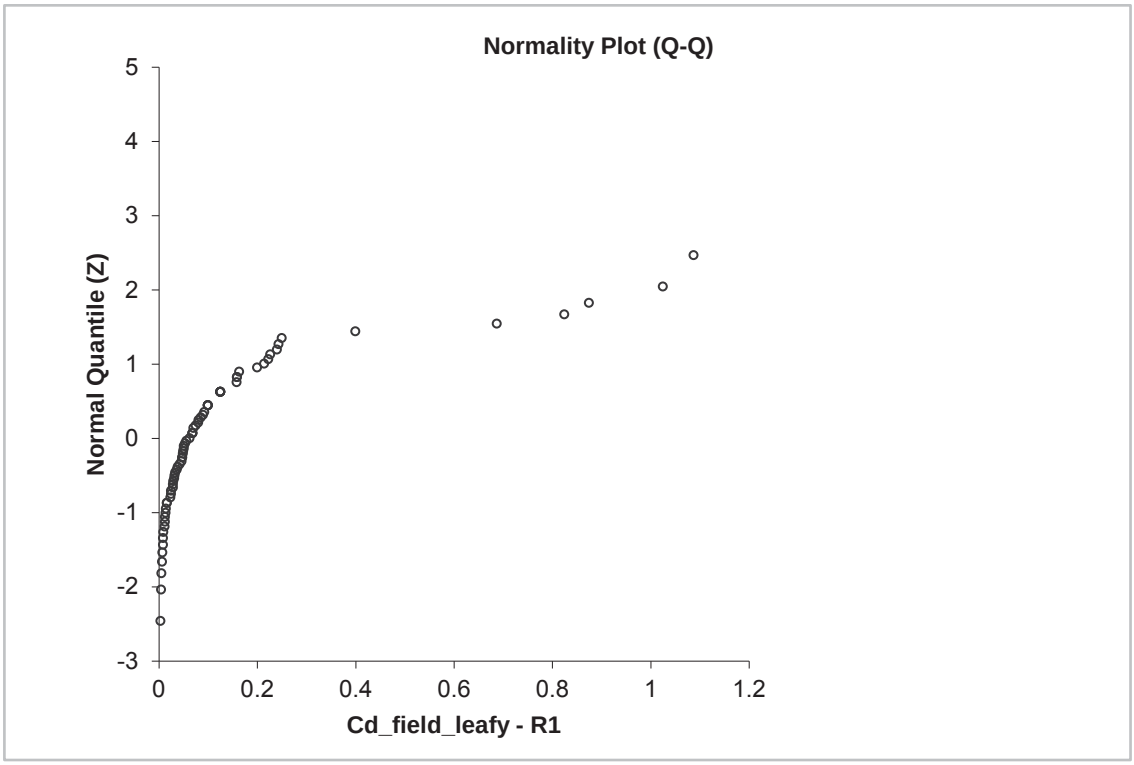


Table H.2 presents the UF distributions from field data only. UFs calculated from laboratory studies in which Cd salts were added to soils were not included in Table H.2, although there are a considerable number of these types of studies. Comparison of UFs calculated from field and Cd salt studies showed significantly greater UFs were obtained in crops grown in Cd salt-contaminated soil. For example, the mean leafy UF from Cd salt studies was 0.5 (n=27), which was significantly greater ($p<0.0001$) than the leafy UF of 0.1 based on field studies (Table H.2). The field studies were chosen to calculate the UFs because they are likely more relevant for "Hot Spots" facility soil contamination.

Table H.2: Percentile Distribution for Cadmium Fresh Weight Soil-to-plant Uptake Factors

	Leafy	Exposed	Protected	Root
n	81	41	27	62
minimum	0.00375	0.0001	0.0002	0.00113
maximum	1.09	0.148	0.0688	0.913
mean	0.139	0.0216	0.0134	0.0683
median	0.0688	0.008	0.0064	0.0244
90^{th} percentile	0.244	0.0541	0.0294	0.124
95^{th} percentile	0.688	0.0863	0.0552	0.172

H.5 Chromium VI

Exposure to hexavalent chromium (Cr(VI)) as a contaminant in soil has been a contentious and complex risk assessment issue that has never been satisfactorily resolved. In both industrial and environmental situations Cr(III) and Cr(VI) can inter-convert, with reduction of Cr(VI) to Cr(III) generally being favored in most soils and sediments. Rapid oxidation of a portion of Cr(III) salts or hydroxides added to almost any soil with a pH above 5 was found to occur readily, provided the soil sample was fresh and kept moist and directly from the field (Bartlett and James, 1988). However, oxidation of Cr(III) to Cr(VI) in field soils is slow compared to well mixed soils in laboratory studies, and given opportunities for its reduction, accumulated Cr(VI) from inorganic sources may rarely be measurable.

Cr(VI) added to soils may be reduced, or absorbed, or may remain in solution depending on the organic matter content, pH, and texture of the soil (Cary, 1982). In neutral to basic soil, chromium will be more available to growing plants than in acidic soil probably due to the increased stability and presence of Cr(VI) in the basic pH range.

For example, when Cr(VI) was added to near-neutral pH soil (6.65) under field conditions, most of the Cr(VI) was extracted from the soil unchanged three weeks later (Bloomfield and Pruden, 1980). Under the same field conditions, most of the added Cr(VI) to an acidic soil (pH 4.20) was reduced three weeks later. These results suggest that in some neutral pH agricultural soils, such as those found in California, constant deposition of Cr(VI) may result in accumulation of Cr(VI) in the soil and ground water.

As a soluble anion, Cr(VI) readily penetrates cell membranes, whereas Cr(III) is soluble at biological pHs only when organically complexed in low molecular weight organic complexes and, therefore, soil forms probably do not penetrate membranes (Bartlett and James, 1988). The difficulty for risk assessors is attempting to estimate what proportion of chromium deposited as Cr(VI) to soil will be available for plant uptake, presumably as Cr(VI). This problem is compounded by the difficulty of estimating the actual speciation of chromium in biological tissues during analysis. As a result, most studies only measure total chromium contents of plant parts.

Cr(III) in soil probably does not penetrate plant cell membranes as such, but is thought to undergo enhanced solubility in soil due to organic acids exuded by roots (James and Bartlett, 1984; Bartlett and James, 1988). This in turn leads to an increased oxidation of Cr(III) to Cr(VI) by soil manganese oxides. The oxidation of Cr(III) to anionic Cr(VI) enables its absorption by the roots. However, once absorbed by root tissues, it appears that most of the Cr(VI) is reduced again to Cr(III) and retained by the roots in a tightly bound or insoluble form or in a soluble complex (e.g., trioxalato chromate(III)) that is not translocated to the above-ground plant parts.

Evidence for the low translocation of chromium from roots has been observed by Lahouti (1979), in which crops that accumulated chromium from nutrient solutions labeled with either ^{51}Cr(III) or ^{51}Cr(VI) retained about 98% of the elements in the roots. Of nine species of crops examined, the roots supplied with ^{51}Cr(III) contained more chromium than those supplied with ^{51}Cr(VI), but chromium added as ^{51}Cr(VI) was slightly better translocated to the shoots. In another study, onion plants were grown in soil after equivalent doses (total dose not provided) of either Cr(III) or Cr(VI) added to the soil (Srivastava et al., 1994). At the lower levels that did not injure the onion plants, the chromium concentration in the plants with Cr(VI) added to soil was only marginally higher than those with Cr(III) added to soil, with most of the chromium retained in the roots and bulb.

This finding seems to suggest that much of the chromium, either added as Cr(VI) or Cr(III), had reached an equilibrium in the soil prior to uptake by the roots.Field studies in which soils were contaminated by anthropogenic sources of Cr(VI) were difficult to come by. Soils contaminated with chromium, generally from sewage sludge, tannery waste, inorganic native chromium in mine waste, are mainly present as Cr(III). Often, the contaminated soils did not exhibit concentrations above the range of typical soil chromium levels of 2 to 50 mg/kg (Kloke et al., 1984), and no chromium control level was provided in the study. Quantitative data for plant uptake of chromium added as Cr(VI) in greenhouse studies are also limited. Cary et al. (1977a, 1977b) added Cr(VI) as K_2CrO_4 to soil over the first 29-40 days after seeding several crop varieties in pots,

and then harvested the crops at maturity 70-110 days after seeding. From these data, leafy, exposed and protected crop UFs for total chromium were estimated (Table H.3). For the root UF, it was observed that roughly 10% of the chromium added as Cr(VI) to soil was incorporated in the above-ground plant parts, with the remainder incorporated into roots and bulbs (Srivastava et al., 1994). The difference between above-ground and root chromium was also reflected by a 10-fold greater concentration of chromium in roots compared to above-ground plant parts. Thus, the root UF is 10-fold greater than the leafy UF. It is currently unknown what proportion of chromium as Cr(VI) will be found in edible crops following absorption and translocation from the roots (Cary, 1982; Kimbrough et al., 1999). Bartlett and James (1988) surmised that if Cr(III) were to be translocated to above-ground plant parts, it is not unreasonable to think that if it enters the chloroplasts it might be oxidized to Cr(VI) in the powerful oxidative environment within the chloroplasts where water is oxidized to O^{2-}. Skeffington (1976) showed that 0.5% of the Cr(III) mixed with ground fresh barley roots was oxidized to Cr(VI). These data would suggest that a fraction of the chromium in roots is present as Cr(VI). Until further characterization of the form of chromium found in edible crops is determined, the health protective assumption is that the chromium found in crops due to root uptake is in the form of Cr(VI).

Table H.3: Crop uptake factors for total chromium, added originally as chromium(VI) to the soil[a]

	Leafy	Exposed	Protected	Root
N	3	1	3	-[b]
Minimum	0.18	-	0.0034	-
Maximum	0.42	-	0.19	-
Mean	0.3	0.02	0.07	3

[a] Data were too limited to determine percentiles.
[b] No quantitative data could be found for a root UF. The general finding that root levels of chromium are 10-fold greater than above-ground plant parts was to devise a root UF.

H.6 Fluoride

Fluoride (F) is strongly sorbed to soil when added as a salt, much stronger than the other halide salts of iodine, bromine and chlorine (Sheppard et al., 1993). The generally low soluble F in most soils coupled with the fact that the root endodermis acts as a barrier means that transport from root to shoot will be limited (Davison, 1982). The lack of soil-to-plant field data for fluoride resulted in a reliance on laboratory studies which added fluoride salts to the soils. The resulting UFs are shown in Table H.4.

The most important F exposure route for plants is uptake via airborne deposition of soluble fluorides of HF and particulate fluoride salts on leaf surfaces. Fluoride that deposits on leaf surfaces can be taken up through stomata of leaves once it deposits on

the surface. Uptake of F into plant leaves occurs by passive permeation of the undissociated HF molecule across the plasmalemma (Kronberger, 1987). Thus, HF behaves like a weak acid (pKa = 3.4) when dissolved in water, where the ionic species becomes trapped within membrane-surrounded compartments after nonionic diffusion. Little fluoride moves downward in plants to roots, from leaf to leaf or from leaves to fruits. Assessing fluoride UFs for leafy crops near airborne industrial emissions of fluoride compounds may eventually require a different algorithm to estimate airborne fluoride accumulation in leafy crops.

Tea plants (*Camellia sinensis*) are known to accumulate high concentrations of F in their leaves from soil containing elevated levels of F, resulting in considerable amounts of F in tea beverages (Davison, 1983). However, it is not known if significant cultivation of tea plants occurs in California. There is also some evidence spinach can accumulate F from soil to a greater degree than other leafy crops (Kumpulainen and Koivistoinen, 1977). The maximum fluoride UF for leafy crops shown in Table H.4 is for spinach.

Table H.4: Fresh weight soil-to-plant uptake factors for fluoride[a]

	Leafy	Exposed	Protected	Root
N	5	-[b]	1	2
Minimum	0.0006	-	-	0.003
Maximum	0.16	-	-	0.014
Mean	0.036	0.004	0.004	0.009

[a] Data were too limited to determine percentiles.
[b] No quantitative data could be found for an exposed crop UF, so the protected crop UF was used

H.7 Lead

Deposited lead (Pb) is strongly retained by most soils, resulting in lower plant concentrations (and lower UFs) relative to more bioaccessible metals such as cadmium and nickel (McLaughlin et al., 1999). Because of the usually low soil-to-root uptake, the above-ground plant parts are likely predominantly contaminated by airborne deposition of lead-containing dust or aerosols onto the plant surface (McBride, 1998). This finding emphasizes the importance of selecting studies in which the leafy plant samples are thoroughly washed prior to assessing root uptake and translocation of lead. Because inorganic lead most often exists as a divalent cation, maintaining alkaline soil conditions will reduce lead mobility in soil, while acidic soil conditions has been shown in some cases to increase soil mobility and uptake of lead through plant roots.

The mean concentration of Pb in uncontaminated U.S. agricultural soils is 12.3 mg/kg, with 5th and 95th percentiles of 4.0 and 23.0 mg/kg, respectively (Holmgren et al., 1993). The range of Pb concentrations in field-contaminated soils reviewed in this document

was large, ranging from 11 mg/kg dry soil to nearly 5500 mg/kg dry soil. Typical dry weight concentrations of Pb in plants are reported to be 0.1 to 5 mg/kg (Vecera et al., 1999), whereas the overall average Pb concentration in crops grown in Pb-polluted soil reviewed in this document was about 9.5 mg/kg.

Table H.5: Percentile distribution for lead fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	77	38	24	57
minimum	0.0000375	0.00002	0.000075	0.0000425
maximum	0.0413	0.0475	0.0278	0.0375
mean	0.00770	0.00693	0.00282	0.00403
median	0.00298	0.00228	0.000912	0.00125
90^{th} percentile	0.0248	0.0214	0.00465	0.00962
95^{th} percentile	0.0308	0.0406	0.00711	0.015

H.8 Mercury

Determining the crop uptake of inorganic mercury (Hg) from soil can be problematic. (Caille et al., 2005) found that following application of radiolabeled $^{203}HgCl_2$ to sediment in a pot experiment, 33-73% of the leaf content in cabbage, rapeseed and pasture grass was due to volatilized Hg absorbed into the leaves. Presumably, the applied inorganic Hg^{2+} was emitted from the soil after reduction to Hg^0 in the soil whereupon it was absorbed by the leaves. Lindberg et al. (1979) observed the same phenomena in alfalfa grown in a chamber, in that above-ground plant parts primarily absorbed Hg vapor released from the soil originally contaminated with mercury mine waste including cinnabar (mercury(II) sulfide). However, the root levels of mercury were determined by direct uptake from contaminated soil and reflected the total Hg concentrations in the soil. Significantly, any Hg vapor emitted by a facility could also be absorbed directly onto leafy crops.

Nearly all studies examined by OEHHA for crop Hg uptake from soil measured total Hg content and did not account for potential volatilization of elemental Hg from soil. Therefore, the soil-to-plant UF for mercury in above-ground plant parts (primarily leafy) includes both root uptake from soil and leaf uptake through volatilization from soil. It is unclear what portion of Hg oxidizes to inorganic Hg once absorbed by leaves, although mercury in food stuffs are mainly in the inorganic form (WHO, 1991). Therefore, a health protective assumption is that the Hg in crops is all in the inorganic form.

Another possible factor to consider is the uptake of methyl mercury (MeHg) by plants. Although it is not expected that Hot Spots facilities would emit MeHg, a fraction of total Hg emitted and deposited to soil could be converted to MeHg in soil. Generally, this may not be a concern in cropland soils, as the content of MeHg would be very low. Nevertheless, results by Gnamus et al. (2001) observed MeHg to be approximately 10 times more phytoavailable then total Hg in an ecotoxicology field study of an Hg-polluted region. Phytoavailability of both total Hg and MeHg increases with decreasing soil pH below 7 and decreased soil content of organic matter.

In rice paddies exposed to Hg smelting and mining facilities, it was found that the percent of total Hg in soil that was MeHg ranged from 0.092 to 0.003 percent (Horvat et al., 2003). However, the percent of total Hg that was MeHg in brown rice grown in the contaminated region ranged from 5 to 84 percent, indicating preferential uptake of MeHg from soil. The resulting UFs for rice ranged from 550 to 6000, suggesting rice may be a high accumulator of MeHg. However, the risk assessment conducted by Horvat et al. (2003) could not establish a clear correlation between total Hg and MeHg in soil and in rice, indicating that uptake and retention of Hg in rice is influenced by a number of factors other than total Hg in soil. Although background mean levels of Hg in U.S. agricultural soils could not be located, a review by Wiersma et al. (1986) showed mean levels of Hg in European and Canadian agricultural soils to be in the range of 0.06 to 0.2 mg/kg dry soil. On average, the concentration of Hg in polluted soils reported in studies reviewed for this document was about 3.6 mg/kg. Typical dry weight plant concentrations of Hg are listed as 0.001 to 0.3 mg/kg (Vecera et al., 1999). In this document, the overall Hg concentration in crops grown in Hg-polluted soils was about 0.2 mg/kg.

Table H.6: Percentile distribution for mercury fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	33	23	15	18
minimum	0.00021	0.000248	0.000106	0.00111
maximum	0.0813	0.0938	0.0363	0.0588
mean	0.0163	0.00855	0.00804	0.0119
median	0.00875	0.00225	0.00514	0.00553
90th percentile	0.0478	0.0175	0.016	0.0274
95th percentile	0.06	0.0198	0.0223	0.0545

H.9 Nickel

Nickel (Ni) is considered to be one of the more mobile heavy metals in soils (Sauerbeck and Hein, 1991). However, in contrast to Cd, the toxicity of Ni in mammals is lower and phytotoxicity occurs at lower concentrations. Similar to other divalent, cationic metals, acidification of soil increases bioavailability, and liming of soil decreases bioavailability, of Ni to plants. The UF data presented in Table H.7 are based on field-contaminated studies. One study that added Ni salts to soil can be found in the database, but appeared to result in increased plant uptake compared to the field data and was, thus, not included for the UF calculations.

The mean concentration of Ni in uncontaminated U.S. agricultural soils is 23.9 mg/kg, with 5th and 95th percentiles of 4.1 and 56.8 mg/kg, respectively (Holmgren et al., 1993). The mean concentration of Ni for field-contaminated soils reviewed in this document was about 70 mg/kg d.w., with a range of 13 to 122 mg/kg d.w. Typical Ni levels in plants are expected to be in the range of 0.1 to 5 mg/kg dry weight (Vecera et al., 1999). In this report, the overall mean dry weight concentration of Ni in crops was about 9 mg/kg.

Table H.7 Percentile distribution for nickel fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	11	13	9	11
minimum	0.00135	0.00025	0.00875	0.00163
maximum	0.0375	0.00625	0.075	0.0175
mean	0.0145	0.00293	0.0305	0.00638
median	0.00888	0.00224	0.025	0.00463
90th percentile	0.0250	0.00610	0.055	0.0125
95th percentile	0.0313	0.00618	0.065	0.0150

H.10 Selenium

The major inorganic species of selenium (Se) in plant sources is selenate, which is translocated directly from the soil and is less readily bound to soil components than selenite (McLaughlin et al., 1999; Rayman, 2008) .The more reduced forms, selenide and elemental Se, are virtually insoluble and do not contribute directly to plant uptake. Other major Se species in plants are biosynthesized, including selenomethionine, smaller amounts of selenocysteine, and Se-containing proteins. At pH values around 7.0 or greater, oxidation to the more soluble selenate ion is favored. Thus, endemic vegetation in alkaline, seleniferous soil of the western U.S. has evolved that is highly tolerant and can hyperaccumulate Se (McLaughlin et al., 1999).

However, potential Se-accumulators that are food sources for humans are largely limited to Brazil nuts, a tree crop that is not grown in California (Rayman et al., 2008). Crops of the Brassica (e.g., broccoli, cabbage) and Allium (e.g., onions, garlic, leeks, chives) families appear to more readily accumulate Se than other crops, and form the Se detoxification products Se-methyl-selenocysteine and gamma-glutamyl-Se-methyl-selenocysteine. Se-enriched plants have been shown in animals to have potent anti-tumor effects that are attributed to these Se detoxification products (Rayman et al., 2008).

Though there is no direct evidence in humans, it is generally accepted on the basis of animal studies that inorganic forms of Se are more acutely toxic than organic species, selenite being slightly more toxic than selenate (Rayman et al., 2008). In chronic studies of humans, lower toxicity is seen with organically bound Se, although there are limited data on the toxicity of individual compounds.

Selenomethionine is known to be the main Se species present in the diet of Chinese who developed chronic selenosis from consumption of high-Se-containing maize and rice. Based on these Chinese studies, 1540 and 819 µg/day were established as the LOAEL and NOAEL, respectively, for total daily Se intake (Rayman, 2008). However, the levels found in crops rarely accumulate greater than 25-30 µg/g even in seleniferous areas suggesting other sources of Se are also contributors to chronic Se toxicity.

Although the UF data for Se were limited, an overall mean dry weight crop Se concentration of about 4 mg/kg was calculated from the reviewed studies, with a maximum crop concentration of 19 mg/kg. Kloke et al. (1984) observed a general dry weight UF for Se in plants would be 0.1 to 10. Based on the studies examined in this document, an overall dry weight uptake factor of 0.9 was calculated for crops grown in Se-polluted soils, which was within the range predicted. Field contamination studies were the primary source of the UF distribution data in Table H.8. The Se pollution sources included mainly fly ash, smelters and compost.

Table H.8: Percentile distribution for selenium fresh weight soil-to-plant uptake factors

	Leafy	Exposed	Protected	Root
n	12	10	7	10
minimum	0.006	0.00132	0.00625	0.005
maximum	0.25	0.25	1.25	0.375
mean	0.0587	0.0415	0.256	0.0689
median	0.0328	0.0106	0.07	0.0195
90th percentile	0.12	0.104	0.678	0.15
95th percentile	0.179	0.177	0.964	0.263

H.11 Summary and Recommendations

OEHHA recommends the root uptake factors in Table H.16 for metals and metalloids.

Table H.16 Recommended Soil-to-plant uptake factors for inorganic metals and metalloids in edible crops[a]

Element	Leafy	Exposed	Protected	Root
Arsenic	1×10^{-2}	2×10^{-2}	7×10^{-2}	8×10^{-3}
Beryllium	2×10^{-4}	8×10^{-3}	3×10^{-4}	5×10^{-3}
Cadmium	1×10^{-1}	2×10^{-2}	1×10^{-2}	8×10^{-2}
Chromium (VI)	3×10^{-1}	2×10^{-2}	7×10^{-2}	3×10^{0}
Fluoride	4×10^{-2}	4×10^{-3}	4×10^{-3}	9×10^{-3}
Lead	8×10^{-3}	7×10^{-3}	3×10^{-3}	4×10^{-3}
Mercury	2×10^{-2}	9×10^{-3}	1×10^{-2}	2×10^{-2}
Nickel	1×10^{-2}	3×10^{-3}	3×10^{-2}	6×10^{-3}
Selenium	6×10^{-2}	4×10^{-2}	3×10^{-1}	7×10^{-2}

[a] Soil-to-plant UFs represent the fresh weight concentration of a contaminant in the plant part over the wet weight concentration of contaminant in the soil.

H.12 Database

The database that lists all of the studies, values, with references is presented as Table H.9-1 through Table H.15-4 in the following pages.

Abbreviations in these tables:

soil conc bckd: the concentration of the chemical in the control soil samples

soil conc contam: the concentration of the chemical in the soil treated with the chemical

tissue conc bckg: the concentration of the chemical in the control tissue samples of the crop

tissue conc contam: the concentration of the chemical in the tissue of the crop grown in the soil treated with the chemical

contam: the related sample treated with the chemical

wt: weight

dw: dry weight

wet w: wet weight

ww: wet weight

Calculation:

$$\text{Uptake factor (contam) dry wt} = \frac{\text{tissue conc contam dry wt} - \text{tissue conc bckg dry wt}}{\text{soil conc contam} - \text{soil conc bckd}}$$

$$\text{Uptake factor (contam) wet wt plant/dw soil} = \text{Uptake factor (contam) dry wt} \times \text{dry-to-wet wt conversion factor}$$

$$\text{Uptake factor (contam) ww plant/wet w soil} = \frac{\text{Uptake factor (contam) wet wt plant/dw soil}}{\text{dry-to-wet weight fraction for soil (0.8)}}$$

Table H.9-1 Arsenic field studies on leafy crops.

Study Type	soil conc bckd mg/kg	soil conc contam mg/kg	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Field		377	leaf mustard		20	0.05305	0.08	0.004244	0.005305	Clemente et al. (2005)
25% mine waste - greenhouse	23.3	187	lettuce	5.47	21.5	0.11497	0.045	0.005	0.00625	Cobb et al., (2000)
field-fly ash - pot	8.8	9.5	cabbage	0.2	0.3	0.03	0.08	0.003	0.00375	Furr et al. (1978a)
Field		6.04	Chinese cabbage			0.025	0.08	0.002	0.0025	Huang et al. (2006)
Field		6.04	leaf mustard			0.07125	0.08	0.0057	0.007125	Huang et al. 2006
Field		6.04	lettuce			0.046	0.05	0.0023	0.002875	Huang et al. 2006
Field		6.04	pakchoi			0.04625	0.08	0.0037	0.004625	Huang et al. 2006
Field		6.04	water spinach			0.07375	0.08	0.0059	0.007375	Huang et al. 2006
Field			amaranthus			0.55	0.08	0.044	0.055	Huq and Naidu (2005)
Field			cabbage			0.44	0.08	0.0352	0.044	Huq and Naidu 2005
wood preserve. Factory-field	3.4	17.9	kale	0.078	0.1	0.0056	0.08	0.00045	0.000563	Larsen et al., (1992)
wood preserve. Factory-field	3.4	17.9	lettuce	0.048	0.086	0.0048	0.05	0.00024	0.0003	Larsen et al., 1992
mining, smelting-field		446.64	cabbage		1.48	0.0033	0.08	0.00027	0.000338	Li et al., (2006)
mining, smelting-field		446.64	cabbage		1.21	0.0027	0.08	0.00022	0.000275	Li et al., 2006
mining, smelting-field		446.64	Chinese cabbage		1.85	0.0041	0.08	0.00034	0.000425	Li et al., 2006
mining, smelting-field		446.64	spinach		1.37	0.0031	0.08	0.00025	0.000313	Li et al., 2006
Field		6.01	amaranth		0.67	0.11148	0.08	0.008918	0.011148	Liu et al. (2006)
Field		6.01	cabbage		0.81	0.13478	0.08	0.010782	0.013478	Liu et al. 2006
Field		6.01	celery		0.49	0.08153	0.08	0.006522	0.008153	Liu et al. 2006
Field		6.01	Chinese cabbage		0.45	0.07488	0.08	0.00599	0.007488	Liu et al. 2006
Field		6.01	Chinese chive		0.57	0.09484	0.08	0.007587	0.009484	Liu et al. 2006
Field		5.54	leek		0.62	0.11191	0.08	0.008953	0.011191	Liu et al. 2006
field		6.01	pakchoi		3	0.49917	0.08	0.039933	0.049917	Liu et al. 2006

Table H.9-1 Arsenic field studies on leafy crops.

Study Type	soil conc bckd mg/kg	soil conc contam mg/kg	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
pot	9.83	745	Radish	0.28	14.4	0.01933	0.08	0.001546	0.001933	Mathe-Gaspar and Anton (2002)
pot	9.83	745	Radish	0	48.7	0.06537	0.08	0.00523	0.006537	Mathe-Gaspar and Anton 2002
Env polluted soil - field		118	lettuce		7.2	0.06102	0.049	0.003	0.00375	Mattina et al., (2003)
Env polluted soil - field		125.9	spinach		1.55	0.012	0.093	0.0011	0.001375	Mattina et al., 2003

Average Arsenic uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00666±0.00982

Table H.9-2 Arsenic field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-fly ash - pot	8.8	9.5	tomato	0.03	0.1	0.01	0.059	0.0006	0.00075	Furr et al. 1978
field		6.04	bottle gourd			0.00397	0.126	0.0005	0.000625	Huang et al. 2006
field		6.04	cauliflower			0.00873	0.126	0.0011	0.001375	Huang et al. 2006
field		6.04	celery			0.05873	0.126	0.0074	0.00925	Huang et al. 2006
field		6.04	cowpea			0.00272	0.257	0.0007	0.000875	Huang et al. 2006
field		6.04	eggplant			0.00822	0.073	0.0006	0.00075	Huang et al. 2006
field		6.04	onion			0.0088	0.125	0.0011	0.001375	Huang et al. 2006
field		6.04	towel gourd			0.00397	0.126	0.0005	0.000625	Huang et al. 2006
field			bean			0.27	0.111	0.02997	0.037463	Huq and Naidu 2005
field			cauliflower			0.84	0.126	0.10584	0.1323	Huq and Naidu 2005
field			tomato			0.55	0.059	0.03245	0.040563	Huq and Naidu 2005
mining, smelting-field		446.64	capsicum		0.75	0.0017	0.074	0.00013	0.000163	Li et al., 2006
mining, smelting-field		446.64	cucumber		0.49	0.0011	0.039	0.000043	5.38E-05	Li et al., 2006
mining, smelting-field		446.64	eggplant		0.45	0.001	0.073	0.000074	9.25E-05	Li et al., 2006
field		5.54	broccoli		0.59	0.1065	0.126	0.013419	0.016773	Liu et al. 2006
field		6.48	cucumber		0.53	0.08179	0.039	0.00319	0.003987	Liu et al. 2006
field		6.01	Eggplant		0.98	0.16306	0.073	0.011903	0.014879	Liu et al. 2006
field		6.01	kidney bean		2.98	0.49584	0.111	0.055038	0.068798	Liu et al. 2006
field		6.01	pepper		0.39	0.06489	0.126	0.008176	0.01022	Liu et al. 2006
field		6.01	tomato		0.46	0.07654	0.059	0.004516	0.005645	Liu et al. 2006
air dep, mine waste, poll. Water		459.02	capsicum		1.3		0.074	0.00021	0.000263	Liu et al., (2005)
air dep, mine waste, poll. Water	96.92	459.02	string bean	0.54	1.33	0.0029	0.111	0.00032	0.0004	Liu et al., 2005

Average Arsenic uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0158±0.0313

Table H.9-3 Arsenic field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
25% mine waste - greenhouse	23.3	187	bush bean	0.184	0.304	0.099	0.00016	0.0002	Cobb et al., 2000
field-fly ash - pot	8.8	9.5	corn	0.1	0.2	0.895	0.02	0.025	Furr et al. 1978
field			cowpea			0.257	0.03341	0.041763	Huq and Naidu 2005
field			garlic			0.222	0.12654	0.158175	Huq and Naidu 2005
field			pea			0.257	0.21331	0.266638	Huq and Naidu 2005
field			pumpkin			0.222	0.03108	0.03885	Huq and Naidu 2005
mining, smelting-field		446.64	pumpkin		0.5	0.082	0.000092	0.000115	Li et al., 2006
air dep, mine waste, poll. Water		459.02	corn		0.21	0.261	0.00012	0.00015	Liu et al., 2005

Average Arsenic uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0664±0.0962

Table H.9-4 Arsenic field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-ground water		13.3 (4-14)	potato		0.8	0.0602	0.222	0.013364	0.016706	Alam et al. (2003)
25% mine waste - greenhouse	23.3	187	radish	0.593	2.94	0.01572	0.047	0.00075	0.000938	Cobb et al., 2000
field-fly ash - pot	8.8	9.5	carrot (peeled)	0.05	0.2	0.02	0.118	0.002	0.0025	Furr et al. 1978
field-fly ash - pot	8.8	9.5	Onion (peeled)	0.1	0.3	0.03	0.125	0.004	0.005	Furr et al. 1978
field-fly ash - pot	8.8	9.5	Potato (peeled)	0.1	0.1	0.01	0.222	0.002	0.0025	Furr et al. 1978
field		6.04	garlic			0.0245	0.2	0.0049	0.006125	Huang et al. 2006
field		6.04	radish			0.0285	0.2	0.0057	0.007125	Huang et al. 2006
field		6.04	taro			0.0165	0.2	0.0033	0.004125	Huang et al. 2006
field			carrot			0.23	0.118	0.02714	0.033925	Huq and Naidu 2005
field			radish			0.18	0.2	0.036	0.045	Huq and Naidu 2005
wood preserve. Factory-field	3.4	17.9	carrot (unpeeled)	0.032	0.042	0.0023	0.118	0.00027	0.000338	Larsen et al., 1992
wood preserve. Factory-field	3.4	17.9	potato (unpeeled)	0.037	0.077	0.0043	0.222	0.00095	0.001188	Larsen et al., 1992
field		5.54	carrot		0.15	0.02708	0.118	0.003195	0.003994	Liu et al. 2006
field		6.01	radish		0.22	0.03661	0.2	0.007321	0.009151	Liu et al. 2006
landfill-field		27	carrot (unpeeled)		0.17	0.0063	0.106	0.00067	0.000838	Samsoe-Petersen et al., (2002)
landfill-field		27	potato (unpeeled)		0.127	0.0047	0.094	0.00044	0.00055	Samsoe-Petersen et al., 2002
landfill-field		27	radish		0.27	0.01	0.059	0.00059	0.000738	Samsoe-Petersen et al., 2002

Average Arsenic uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00828±0.0129

Table H.10-1 Cadmium field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field	0.69	1.6	amaranth	0.81	3.85	2.406	0.08	0.1925	0.2406	Hu and Ding (2009)
field		0.16	amaranth		0.16	1.000	0.08	0.0800	0.1000	Liu et al. 2006
indust. Poll. Depo. - field		12	amaranthus		5.66	0.470	0.08	0.0380	0.0475	Pandey and Pandey, (2009)
Indust. sewage wastes - field	0.5	22	amaranthus	0.14	1.1	0.050	0.08	0.0040	0.0050	Srikanth et al., (1991)
field-wastewater	0.12	0.87	basil	0.16	0.6	0.690	0.08	0.0550	0.0688	Shariatpanahi and Anderson (1986)
field		4.4	cabbage		0.3	0.068	0.08	0.0055	0.0068	Chumbley and Unwin (1982)
sewage sludge - pots		23.22	cabbage		1.77	0.076	0.08	0.0061	0.0076	Jackson & Alloway, (1991)
mining, smelting-field		7.43	cabbage		0.71	0.096	0.08	0.0077	0.0096	Li et al., 2006
mining, smelting-field		7.43	cabbage		1.29	0.170	0.08	0.0130	0.0163	Li et al., 2006
field		0.16	cabbage		0.076	0.475	0.08	0.0380	0.0475	Liu et al. 2006
sewage sludge - field		10.5	cabbage		2.1	0.200	0.08	0.0200	0.0250	Muntau et al., (1987)
Indust. sewage wastes - field	0.5	22	cabbage	0.02	2.88	0.130	0.078	0.0100	0.0125	Srikanth et al., 1991
field - smelter	0.108	4.99	cabbage				0.052	0.1740	0.2175	Zheng et al. (2007a)a
field		1.6	celery		3.57	2.231	0.08	0.1785	0.2231	Hu and Ding 2009
field		0.16	celery		0.1	0.625	0.08	0.0500	0.0625	Liu et al. 2006
field - smelter	0.108	12.5	celery				0.058	0.1310	0.16375	Zheng et al. 2007a
mining, smelting-field		7.43	Chinese cabbage		1.31	0.180	0.08	0.0130	0.0163	Li et al., 2006
field		0.16	Chinese cabbage		0.2	1.250	0.08	0.1000	0.1250	Liu et al. 2006
field		0.515	Chinese cabbage		0.2625	0.510	0.08	0.0408	0.0510	Wang et al. (2006)
field - smelter	0.108	22.8	Chinese cabbage				0.055	0.1280	0.16	Zheng et al. 2007a
field		0.16	Chinese chive		0.12	0.750	0.08	0.0600	0.0750	Liu et al. 2006
sewage sludge-field-grnhs		2.55	chinese leek		0.9	0.350	0.089	0.0310	0.0388	Yang et al., (2009)
field-wastewater	0.12	0.87	garden cress	0.1	0.6	0.690	0.08	0.0550	0.0688	Shariatpanahi and Anderson 1986

Table H.10-1 Cadmium field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field - smelter	0.108	43.4	green onion				0.085	0.0440	0.055	Zheng et al. 2007a
field		0.17	leek		0.055	0.324	0.08	0.0259	0.0324	Liu et al. 2006
field - smelter	0.108	39.2	leek			2.250	0.08	0.1800	0.2250	Zheng et al. 2007a
field		7.8	lettuce		4.2	0.538	0.05	0.0269	0.0337	Chumbley and Unwin 1982
25% mine waste - greenhouse	1.38	6.06	lettuce	1.61	5.37	0.890	0.045	0.0400	0.0500	Cobb et al., 2000
Env. contam. Soil 1a - potted		1.8	lettuce		2.5	1.400	0.049	0.0686	0.0858	Crews & Davies, (1985)
Env. contam. Soil 1b - potted		2.2	lettuce		7.8	3.500	0.049	0.1715	0.2144	Crews & Davies, 1985
Env. contam. Soil 2 - potted		4.5	lettuce		11.8	2.600	0.049	0.1274	0.1593	Crews & Davies, 1985
Env. contam. Soil 3 - potted		5.5	lettuce		20.5	3.700	0.049	0.1813	0.2266	Crews & Davies, 1985
field	0.69	1.6	lettuce	1.49	4.19	2.619	0.05	0.1309	0.1637	Hu and Ding 2009
fertilizer	0.53	0.6-0.86	lettuce				0.05	0.1950	0.2438	Huang et al. (2003)
fertilizer in field			lettuce				0.05	0.3199	0.3998	Huang et al. (2004)
sewage sludge - pots		23.22	lettuce		10.57	0.460	0.05	0.0230	0.0288	Jackson & Alloway, 1991
Env polluted soil - field		1	lettuce		2.6	2.600	0.049	0.1274	0.1593	Mattina et al., 2003
sewage sludge-field		2.2	lettuce		2.8	1.300	0.05	0.0650	0.0813	Preer et al., (1995)
smelter area - urban gardens	0.8	12.6	lettuce	0.41	7.55	0.600	0.049	0.0294	0.0368	Pruvot et al., (2006)
landfill-field		2.4	lettuce		0.552	0.230	0.05	0.0115	0.0144	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	lettuce		0.21	0.400	0.05	0.0200	0.0250	Samsoe-Petersen et al., 2002
fertilizer-field	ND	0.311	lettuce	ND	0.06	0.200	0.05	0.0100	0.0125	(Schroeder and Balassa, 1963)
fertilizer-field	ND	0.311	lettuce	ND	0.5	1.600	0.045	0.0720	0.0900	Schroeder & Balassa, 1963
urban gardens-field-to-grnhs	0.08	3.28	lettuce	0.65	1.73	0.760	0.045	0.0342	0.0428	Sterrett et al., (1996)
field - smelter	0.108	4.99	lettuce				0.042	0.2030	0.25375	Zheng et al. 2007
field-wastewater	0.12	0.87	mint	0.11	0.7	0.800	0.08	0.0640	0.0800	Shariatpanahi and Anderson 1986
field - smelter	0.108	20.1	mustard				0.071	0.0870	0.10875	Zheng et al. 2007
field		1.6	pakchoi		2.53	1.581	0.08	0.1265	0.1581	Hu and Ding 2009
field		0.16	pakchoi		0.11	0.688	0.08	0.0550	0.0688	Liu et al. 2006

Table H.10-1 Cadmium field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		0.515	Pakchoi		0.275	0.534	0.08	0.0427	0.0534	Wang et al. 2006
field		15.8	Pakchoi		0.21	0.090	0.08	0.0072	0.0090	Yan et al. (2007)
sewage sludge-field-greenhouse		2.55	pakchoi		1.25	0.490	0.076	0.0370	0.0463	Yang et al., 2009
field (industrial sewage irrigation)		2.69	palak (spinach)		1.5	0.560	0.08	0.0450	0.0563	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		2.26	palak (spinach)		2.1	0.930	0.08	0.0740	0.0925	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		2.8	palak (spinach)		2.85	1.000	0.08	0.0800	0.1000	Kumar Sharma et al., 2007
pot	0.167	30.5	Radish	0.388	8.78	0.288	0.08	0.0230	0.0288	Mathe-Gaspar and Anton 2002
pot	0.167	30.5	Radish	0.448	9.05	0.297	0.08	0.0237	0.0297	Mathe-Gaspar and Anton 2002
flooded gardens		1.31	sorrel		0.115	0.088	0.08	0.0070	0.0088	Sipter et al. (2008)
non-flooded gardens		0.43	sorrel		0.101	0.235	0.08	0.0188	0.0235	Sipter et al. 2008
field		4.6	spinach		4.6	1.000	0.08	0.0800	0.1000	Chumbley and Unwin 1982
high-Cd fertilizer - greenhouse	0.25	0.2625	spinach	1.48	2.18	8.300	0.08	0.6600	0.8250	He and Singh (1994)
high-Cd fertilizer - greenhouse	0.25	0.2625	spinach	2.32	2.85	10.860	0.08	0.8700	1.0875	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	spinach	1.48	1.74	6.890	0.08	0.5500	0.6875	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	spinach	2.32	2.58	10.210	0.08	0.8200	1.0250	He and Singh 1994
sewage sludge-field	0.48	5.32	spinach	0.94	12.76	1.991	0.08	0.1600	0.2000	Hooda et al., 1997
sewage sludge-field	1.6	4.3	spinach	0.01	0.14	0.030	0.08	0.0030	0.0038	Jamali et al., 2007
mining, smelting-field		7.43	spinach		1.06	0.140	0.08	0.0110	0.0138	Li et al., 2006
field (sewage-fed lake irrigation)			Spinach			2.500	0.08	0.2000	0.2500	Lokeshwari and Chandrappa 2006
Env polluted soil - field		0.7	spinach		5.3	7.600	0.093	0.7000	0.8750	Mattina et al., 2003
indust. Poll. Depo. - field		12	spinach		5.84	0.490	0.08	0.0390	0.0488	Pandey and Pandey, 2009

Table H.10-1 Cadmium field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Indust. sewage wastes - field	0.5	22	spinach	0.13	6.4	0.290	0.086	0.0250	0.0313	Srikanth et al., 1991
field - smelter	0.108	43.4	spinach				0.088	0.0980	0.1225	Zheng et al. 2007
field		9.3	spring greens		1.1	0.118	0.08	0.0095	0.0118	Chumbley and Unwin 1982
sewage sludge - chamber	0.9	8.4	Swiss chard	2.2	11.2	1.300	0.08	0.1000	0.1250	Mahler et al., 1987
sewage sludge + limed - chamber	0.9	8.4	Swiss chard	1.7	8.4	1.000	0.08	0.0800	0.1000	Mahler et al., 1987
fertilizer-field greenhouse	0.07	1.13	Swiss chard	0.26	1.61	1.400	0.08	0.1000	0.1250	Mulla et al., (1980)
drilling fluid-greenhouse	0.6	19.4	swiss chard	1.5	26.9	1.400	0.08	0.1000	0.1250	Nelson et al., (1984)
sewage sludge-field		2.2	Swiss chard		3.15	1.400	0.08	0.1000	0.1250	Preer et al., 1995
field-wastewater	0.12	0.87	tarragon	0.14	0.05	0.060	0.08	0.0046	0.0058	Shariatpanahi and Anderson 1986
field		0.515	Water spinach		0.3625	0.704	0.08	0.0563	0.0704	Wang et al. 2006
field survey						0.507	0.08	0.0406	0.0507	Cambra et al. 1999

Average cadmium uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.139±0.214

Table H.10-2 Cadmium field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field - smelter	0.108	39.2	aubergine			0.513	0.081	0.0416	0.0519	Zheng et al. 2007a
indust. sewage-field-Egypt	ND	28	bell pepper		0.05	0.002	0.074	0.0001	0.0001	Gorbunov et al., 2003
field - smelter	0.108	20.1	bitter melon				0.066	0.0050	0.00625	Zheng et al. 2007a
landfill-field		2	blackberry					0.0025	0.0031	Samsoe-Petersen et al., 2002
field		0.17	broccoli		0.048	0.282	0.126	0.0356	0.0445	Liu et al. 2006
mining, smelting-field		7.43	capsicum		0.41	0.055	0.074	0.0040	0.0050	Li et al., 2006
air dep, mine waste, poll. Water		6.77	capsicum		1.37	0.200	0.074	0.0150	0.0188	Liu et al., 2005
field - smelter	0.108	39.2	capsicum			0.258	0.066	0.0170	0.0213	Zheng et al. 2007a
field		3.5	cauliflower		0.7	0.200	0.126	0.0252	0.0315	Chumbley and Unwin 1982
indust. sewage-field-Egypt	ND	28	cucumber		0.06	0.002	0.039	0.0001	0.0001	Gorbunov et al., 2003
mining, smelting-field		7.43	cucumber		0.66	0.089	0.039	0.0035	0.0044	Li et al., 2006
field		0.16	cucumber		0.059	0.369	0.039	0.0144	0.0180	Liu et al. 2006
sewage sludge-field-grnhs		2.55	cucumber		0.2	0.080	0.04	0.0031	0.0039	Yang et al., 2009
mining, smelting-field		7.43	eggplant		0.4	0.054	0.073	0.0039	0.0049	Li et al., 2006
field		0.16	Eggplant		0.16	1.000	0.073	0.0730	0.0913	Liu et al. 2006
indust. Poll. Depo. - field		12	eggplant		4.18	0.350	0.073	0.0260	0.0325	Pandey and Pandey, 2009
field		0.515	Eggplant		0.3	0.638	0.073	0.0466	0.0583	Wang et al. 2006
indust. sewage-field-Egypt	ND	28	fig		0.015	0.001	0.126	0.0001	0.0001	Gorbunov et al., 2003
sewage sludge-field	1.6	4.3	Indian squash	0.08	0.24	0.060	0.082	0.0050	0.0063	Jamali et al., (2007)
field		0.16	kidney bean		0.036	0.225	0.111	0.0250	0.0312	Liu et al. 2006
field-wastewater	0.12	0.87	leek	0.14	0.5	0.570	0.12	0.0690	0.0863	Shariatpanahi and Anderson 1986
indust. sewage-field-Egypt	ND	28	olive		0.03	0.001	0.126	0.0001	0.0001	Gorbunov et al., 2003
landfill-field		2	pear					0.0034	0.0043	Samsoe-Petersen et al., 2002
sewage sludge-field			pepper				0.0408	0.0290	0.0362	Giordano et al., (1979)
field		0.16	pepper		0.15	0.938	0.126	0.1181	0.1477	Liu et al. 2006
field survey			peppers			0.053	0.126	0.0066	0.0083	Cambra et al. (1999)
landfill-field		2	plum					0.0006	0.0008	Samsoe-Petersen et al., 2002

Table H.10-2 Cadmium field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
sewage sludge-field			squash				0.082	0.0098	0.0123	Giordano et al., 1979
flooded gardens		1.31	squash		0.033	0.025	0.082	0.0021	0.0026	Sipter et al. 2008
non-flooded gardens		0.43	squash		0.005	0.012	0.082	0.0010	0.0012	Sipter et al. 2008
air dep, mine waste, poll. Water	2.08	6.77	string bean	0.21	0.67	0.099	0.111	0.0110	0.0138	Liu et al., 2005
25% mine waste - greenhouse	1.38	6.06	tomato	0.523	0.704	0.120	0.065	0.0078	0.0098	Cobb et al., 2000
field		0.15	tomato		0.11	0.733	0.059	0.0433	0.0541	Liu et al. 2006
indust. Poll. Depo. - field		12	tomato		4.96	0.410	0.059	0.0240	0.0300	Pandey and Pandey, 2009
smelter area - urban gardens	0.8	12.6	tomato	0.15	1.23	0.098	0.065	0.0063	0.0079	Pruvot et al., 2006
flooded gardens		1.31	tomato		0.06	0.046	0.059	0.0027	0.0034	Sipter et al. 2008
non-flooded gardens		0.43	tomato		0.008	0.019	0.059	0.0011	0.0014	Sipter et al. 2008
smelter contam - field	0.08	4.4	tomato		0.43	0.098	0.065	0.0064	0.0080	Tomov & Alandjiyski, (2006)
sewage sludge-field-grnhs		2.55	tomato		0.2	0.080	0.033	0.0026	0.0033	Yang et al., 2009
field - smelter	0.11	43.4	tomato				0.056	0.0030	0.00375	Zheng et al. 2007a
field		0.515	Towel gourd		0.0976	0.189	0.082	0.0155	0.0194	Wang et al. 2006

Average cadmium uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0216±0.0304

Table H.10-3 Cadmium field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant /wet w soil	References
flooded gardens		1.31	bean		0.02	0.01527	0.111	0.001695	0.0021	Sipter et al. 2008
non-flooded gardens		0.43	bean		0.01	0.02326	0.111	0.002581	0.0032	Sipter et al. 2008
indust. sewage-field-Egypt	ND	28	bean (spot)		0.28	0.01	0.111	0.001	0.0013	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	28	bean (white)		0.26	0.009	0.111	0.001	0.0013	Gorbunov et al., 2003
sewage sludge-pot-field		4.6	beans		0.27	0.06	0.222	0.013	0.0163	Sauerbeck, 1991
field survey			broad beans			0.0108	0.126	0.001361	0.0017	Cambra et al. 1999
25% mine waste - grhs	1.38	6.06	bush bean	0.145	0.01	0.0017	0.099	0.00017	0.0002	Cobb et al., 2000
sewage sludge-field			cantelope				0.06	0.0192	0.0240	Giordano et al., 1979
sewage sludge-field	1.6	4.3	cluster beans	0.04	0.2	0.05	0.111	0.005	0.0063	Jamali et al., 2007
field	0.26	25.3889	corn		0.2	0.00788	0.261	0.002056	0.0026	Bi et al. (2006)
air dep, mine waste, poll. Water		6.77	corn		0.47	0.069	0.261	0.018	0.0225	Liu et al., 2005
indust. sewage-field	0.072	3.72	corn	0.002	0.23	0.062	0.895	0.055	0.0688	Nan et al., (2002)
smelter area - ag field	0.4	8.1	corn	0.07	0.18	0.022	0.273	0.0062	0.0078	Pruvot et al., 2006
field		0.515	Cowpea		0.02724	0.05289	0.257	0.013592	0.0170	Wang et al. 2006
field - smelter	0.108	43.4	cowpea				0.097	0.004	0.005	Zheng et al. 2007a
landfill-field		2	green bean		0.098	0.041	0.027	0.0011	0.0014	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	green bean		0.009	0.02	0.111	0.002	0.0025	Samsoe-Petersen et al., 2002
landfill-field		2	hazelnut					0.004	0.0050	Samsoe-Petersen et al., 2002
field - smelter	0.108	39.2	kidney bean			0.119	0.103	0.012257	0.0153	Zheng et al. 2007a
fertilizer-field	ND	0.311	onion	ND	0.024	0.08	0.125	0.01	0.0125	Schroeder & Balassa, 1963
fertilizer-field	ND	0.311	pea	ND	0.04	0.1	0.257	0.03	0.0375	Schroeder & Balassa, 1963
sewage sludge-field	1.6	4.3	peas	0.075	0.2	0.05	0.257	0.01	0.0125	Jamali et al., 2007
sewage sludge-pot-field		4.6	peas		0.2	0.04	0.257	0.01	0.0125	Sauerbeck, 1991
mining, smelting-field		7.43	pumpkin		0.46	0.062	0.082	0.0051	0.0064	Li et al., 2006
field - smelter	0.108	43.4	pumpkin				0.065	0.001	0.001	Zheng et al. 2007a
fertilizer-field	ND	0.311	string bean	ND	0.015	0.05	0.111	0.01	0.0125	Schroeder & Balassa, 1963
field		7.8	sweet corn		1.5	0.19231	0.261	0.050192	0.0627	Chumbley and Unwin 1982

Average cadmium uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0134±0.0175

Table H.10-4 Cadmium field studies on root crops.

Study Type	soil conc bcgd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bcgd(T) dry wt (mg/kg)	tissue conc contam(C) dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
fertilizer-field	ND	0.311	beet	ND	0.045	0.100	0.2	0.0300	0.0375	Schroeder & Balassa, 1963
field		6.5	beetroot		2	0.308	0.222	0.0683	0.0854	Chumbley and Unwin 1982
smelter - field - home gardens		40.6	carrot		4.4	0.110	0.118	0.0130	0.0163	Chaney et al., (1988)
sewage sludge-field	0.48	5.32	carrot	0.63	1.71	0.350	0.118	0.0410	0.0513	Hooda et al., 1997
field		0.17	carrot		0.085	0.500	0.118	0.0590	0.0738	Liu et al. 2006
indust. Poll. Depo. - field		12	carrot		2.06	0.170	0.118	0.0200	0.0250	Pandey and Pandey, 2009
smelter area - urban gardens	0.8	12.6	carrot	0.085	1.53	0.120	0.118	0.0140	0.0175	Pruvot et al., 2006
fertilizer-field	ND	0.311	carrot	ND	0.068	0.200	0.118	0.0300	0.0375	Schroeder & Balassa, 1963
flooded gardens		1.31	carrot		0.13	0.099	0.118	0.0117	0.0146	Sipter et al. 2008
non-flooded gardens		0.43	carrot		0.068	0.158	0.118	0.0187	0.0233	Sipter et al. 2008
contam-irrig. water - greenhouse		3.6	carrot		1.22	0.340	0.135	0.0460	0.0575	Zheng et al., (2008)
sewage sludge-field-greenhouse		2.55	carrot		0.7	0.270	0.11	0.0300	0.0375	Yang et al., 2009
field - smelter	0.108	39.2	carrot			0.752	0.088	0.0662	0.0827	Zheng et al. 2007a
high-Cd fertilizer - greenhouse	0.25	0.2625	carrot	0.115	0.145	0.550	0.118	0.0650	0.0813	He and Singh 1994
high-Cd fertilizer - greenhouse	0.25	0.2625	carrot	0.125	0.165	0.630	0.118	0.0740	0.0925	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	carrot	0.115	0.135	0.530	0.118	0.0630	0.0788	He and Singh 1994
low-Cd fertilizer - greenhouse	0.25	0.2527	carrot	0.125	0.15	0.590	0.118	0.0700	0.0875	He and Singh 1994
fertilizers w/ Cd		0.3	carrot (unpeeled)		0.25	0.800	0.11	0.0900	0.1125	Jansson and Oborn, (2000)
landfill-field		2.4	carrot (unpeeled)		0.26	0.110	0.127	0.0140	0.0175	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	carrot (unpeeled)		0.12	0.200	0.118	0.0300	0.0375	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		4.6	carrots		0.9	0.200	0.118	0.0200	0.0250	Sauerbeck, 1991
field survey			chard			0.519	0.2	0.1038	0.1298	Cambra et al. 1999
indust. sewage-field-Egypt	ND	28	garlic		0.21	0.008	0.125	0.0009	0.0011	Gorbunov et al., 2003
smelter area - urban gardens	0.8	12.6	leek	0.14	1.58	0.130	0.146	0.0180	0.0225	Pruvot et al., 2006
field		3.1	leeks		0.8	0.258	0.2	0.0516	0.0645	Chumbley and Unwin 1982
indust. sewage-field-Egypt	ND	28	onion		0.27	0.010	0.125	0.0010	0.0013	Gorbunov et al., 2003
field-wastewater	0.12	0.87	onion	0.12	0.3	0.340	0.125	0.0400	0.0500	Shariatpanahi and Anderson 1986
flooded gardens		1.31	onion		0.07	0.053	0.125	0.0067	0.0083	Sipter et al. 2008
non-flooded gardens		0.43	onion		0.056	0.130	0.125	0.0163	0.0203	Sipter et al. 2008

Table H.10-4 Cadmium field studies on root crops.

Study Type	soil conc bcgd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bcgd(T) dry wt (mg/kg)	tissue conc contam(C) dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field survey			onions			0.105	0.125	0.0132	0.0164	Cambra et al. 1999
fertilizer-field	ND	0.311	parsnip	0.15	0.7	2.200	0.2	0.5000	0.6250	Schroeder & Balassa, 1963
smelter - field - home gardens		13.2	potato		3.6	0.270	0.202	0.7300	0.9125	Chaney et al., 1988
field		10.8	potato		0.6	0.056	0.222	0.0123	0.0154	Chumbley and Unwin 1982
smelter flue-dust	0.3	106.5	potato	0.16	1.67	0.016	0.222	0.0035	0.0044	Dudka et al. 1996
smelter flue-dust	0.3	54.4	potato	0.16	2.12	0.039	0.222	0.0087	0.0108	Dudka et al. 1996
smelter flue-dust	0.3	7.1	potato	0.16	0.53	0.075	0.222	0.0166	0.0207	Dudka et al. 1996
smelter flue-dust	0.3	3.2	potato	0.16	0.42	0.131	0.222	0.0291	0.0364	Dudka et al. 1996
smelter area - ag field	0.4	8.1	potato	0.3	0.45	0.056	0.202	0.0110	0.0138	Pruvot et al., 2006
smelter area - urban gardens	0.8	12.6	potato	0.05	0.54	0.043	0.202	0.0087	0.0109	Pruvot et al., 2006
fertilizer-field	ND	0.311	potato	ND	0.015	0.050	0.222	0.0100	0.0125	Schroeder & Balassa, 1963
smelter contam - field	0.08	4.4	potato		0.097	0.022	0.202	0.0044	0.0055	Tomov & Alandjiyski, 2006
sewage sludge - pots		23.22	potato (peeled)		0.3	0.013	0.222	0.0029	0.0036	Jackson & Alloway, 1991
sewage sludge-field		2.77	potato (peeled)		0.07	0.030	0.218	0.0055	0.0069	Smith (1994)
landfill-field		2.4	potato (unpeeled)		0.089	0.037	0.135	0.0050	0.0063	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	potato(unpeeled)		0.05	0.090	0.222	0.0200	0.0250	Samsoe-Petersen et al., 2002
field		2.7	radish		1.7	0.630	0.222	0.1398	0.1747	Chumbley and Unwin 1982
25% mine waste - greenhouse	1.38	6.06	radish	0.01	2.31	0.380	0.047	0.0180	0.0225	Cobb et al., 2000
indust. sewage-field-Egypt	ND	28	radish		0.28	0.010	0.085	0.0009	0.0011	Gorbunov et al., 2003
field		0.16	radish		0.083	0.519	0.2	0.1038	0.1297	Liu et al. 2006
field (sewage-fed lake irrigation)			Radish			1.600	0.2	0.3200	0.4000	Lokeshwari and Chandrappa 2006
indust. Poll. Depo. - field		12	radish		2.61	0.220	0.085	0.0190	0.0238	Pandey and Pandey, 2009
smelter area - urban gardens	0.8	12.6	radish	0	2.12	0.170	0.047	0.0079	0.0099	Pruvot et al., 2006
landfill-field		2.4	radish		0.19	0.080	0.041	0.0033	0.0041	Samsoe-Petersen et al., 2002
moderate urban poll -field		0.56	radish		0.071	0.100	0.085	0.0100	0.0125	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		4.6	radish		1.1	0.200	0.05	0.0100	0.0125	Sauerbeck, 1991
fertilizer-field	ND	0.311	radish	ND	0.1	0.300	0.2	0.0600	0.0750	Schroeder & Balassa, 1963
field-wastewater	0.12	0.87	radish	0.18	0.45	0.520	0.085	0.0400	0.0500	Shariatpanahi and Anderson 1986

Table H.10-4 Cadmium field studies on root crops.

Study Type	soil conc bcgd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bcgd(T) dry wt (mg/kg)	tissue conc contam(C) dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
contam-irrig. water - greenhouse		3.6	radish		1.09	0.300	0.083	0.0250	0.0313	Zheng et al., 2008
sewage sludge-field-greenhouse		2.55	radish		0.5	0.200	0.05	0.0098	0.0123	Yang et al., 2009
field		4.8	salad onions		1	0.208	0.125	0.0260	0.0326	Chumbley and Unwin 1982
fertilizer-field	ND	0.311	turnip	ND	0.15	0.500	0.2	0.1000	0.1250	Schroeder & Balassa, 1963
field - smelter	0.108	39.2	turnip			0.027	0.108	0.0029	0.0036	Zheng et al. 2007a

Average cadmium uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0683±0.144

Table H.11-1 Lead field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt mg/kg	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
pots -env. chamber	30	300	cabbage		2.4	0.0080	0.08	0.0006	0.00075	Caille et al., 2005
pots -env. chamber	30	300	rape		2.3	0.0080	0.08	0.0006	0.00075	Caille et al., 2005
field		117	cabbage		0.3	0.0026	0.08	0.000205	0.0002564	Chumbley and Unwin 1982
field		155	lettuce		2.3	0.0148	0.05	0.000742	0.0009274	Chumbley and Unwin 1982
field		124	spinach		3.7	0.0298	0.08	0.002387	0.0029839	Chumbley and Unwin 1982
field		214	spring greens		2.3	0.0107	0.08	0.00086	0.0010748	Chumbley and Unwin 1982
field		532	leaf mustard		21	0.0395	0.08	0.003158	0.0039474	Clemente et al. 2005
25% mine waste - grnhs	60.9	3600	lettuce	29.8	227	0.0631	0.045	0.002838	0.0035469	Cobb et al., 2000
Env. contam. Soil 1a - potted - outside		301	lettuce		2	0.0066	0.049	0.000326	0.000407	Crews & Davies, 1985
Env. contam. Soil 1b - potted - outside		169	lettuce		7.7	0.0456	0.049	0.002233	0.0027907	Crews & Davies, 1985
Env. contam. Soil 2 - potted - outside		754	lettuce		5.7	0.0076	0.049	0.00037	0.000463	Crews & Davies, 1985
Env. contam. Soil 3 - potted - outside		850	lettuce		14.3	0.0168	0.049	0.000824	0.0010304	Crews & Davies, 1985
urban gardens-field			cilantro				0.08	0.002	0.0025	Finster et al., 2004
urban gardens-field			collard greens				0.147	0.0004	0.0005	Finster et al., 2004
urban gardens-field			coriander				0.08	0.003	0.00375	Finster et al., 2004
urban gardens-field			ipasote				0.08	0.002	0.0025	Finster et al., 2004
urban gardens-field			lemon balm				0.08	0.001	0.00125	Finster et al., 2004
urban gardens-field			mint				0.08	0.0009	0.001125	Finster et al., 2004
urban gardens-field			rhubarb				0.052	0.00047	0.0005875	Finster et al., 2004
urban gardens-field			Swiss chard				0.089	0.0027	0.003375	Finster et al., 2004
sewage sludge-field	70	259	spinach	0.82	0.95	0.0080	0.08	0.0006	0.00075	Hooda et al., 1997
field	65.9	361	amaranth	2.66	45.7	0.1266	0.08	0.010127	0.0126593	Hu and Ding 2009
field		361	celery		22.1	0.0612	0.08	0.004898	0.0061219	Hu and Ding 2009
field	65.9	361	lettuce	1.14	37.5	0.1039	0.05	0.005194	0.0064924	Hu and Ding 2009
field		361	pakchoi		36.2	0.1003	0.08	0.008022	0.0100277	Hu and Ding 2009

Table H.11-1 Lead field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt mg/kg	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Pb arsenate - grnhs	60.9	342.3	lettuce	10.2	12.5	0.0400	0.05	0.002	0.0025	Hutchinson et al. 1974
sewage sludge-field	21.1	67.4	spinach	0.33	1.2	0.0200	0.08	0.001	0.00125	Jamali et al., 2007
mining, smelting-field		223.22	cabbage			0.0500	0.08	0.004	0.005	Li et al., 2006
mining, smelting-field		223.22	cabbage			0.0490	0.08	0.0039	0.004875	Li et al., 2006
mining, smelting-field		223.22	Chinese cabbage			0.0780	0.08	0.0062	0.00775	Li et al., 2006
mining, smelting-field		223.22	spinach			0.0700	0.08	0.0056	0.007	Li et al., 2006
field		14.48	amaranth		1.91	0.1319	0.08	0.010552	0.0131906	Liu et al. 2006
field		14.48	cabbage		1	0.0691	0.08	0.005525	0.0069061	Liu et al. 2006
field		14.48	celery		1.76	0.1215	0.08	0.009724	0.0121547	Liu et al. 2006
field		14.48	Chinese cabbage		2.05	0.1416	0.08	0.011326	0.0141575	Liu et al. 2006
field		14.48	Chinese chive		2.53	0.1747	0.08	0.013978	0.0174724	Liu et al. 2006
field		14.48	pakchoi		2.02	0.1395	0.08	0.01116	0.0139503	Liu et al. 2006
pot	18.5	2897	Radish	2.9	94.3	0.0326	0.047	0.00153	0.0019124	Mathe-Gaspar and Anton 2002
pot	18.5	2897	Radish	2.4	272.4	0.0940	0.047	0.004419	0.0055242	Mathe-Gaspar and Anton 2002
sewage sludge - field		775	cabbage		0.31	0.0004	0.08	0.00003	0.0000375	Muntau et al., 1987
drilling fluid-grnhs	17	1131	swiss chard	1.7	9.2	0.0080	0.08	0.0007	0.000875	Nelson et al., 1984
Env. contam. Soil (paint?) - potted - grnhs		2000	collard		8	0.0040	0.147	0.0006	0.00075	Nicklow et al., (1983)
Env. contam. Soil (paint?) - potted - grnhs		2000	kale		7	0.0035	0.173	0.0006	0.00075	Nicklow et al., 1983
Env. contam. Soil (paint?) - potted - grnhs		2000	lettuce		25	0.0125	0.049	0.000613	0.0007656	Nicklow et al., 1983
indust. Poll. Depo. - field		165.85	amaranthus		18.44	0.1100	0.08	0.0088	0.011	Pandey and Pandey, 2009
indust. Poll. Depo. - field		165.85	spinach		19.58	0.1200	0.08	0.0096	0.012	Pandey and Pandey, 2009
sewage sludge-field		98	lettuce			0.0200	0.05	0.001	0.00125	Preer et al., 1995

Table H.11-1 Lead field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt mg/kg	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
sewage sludge-field		98	Swiss chard			0.0300	0.08	0.003	0.00375	Preer et al., 1995
smelter area - urban gardens - field	84	872	lettuce	2.24	6.93	0.0079	0.049	0.000387	0.0004839	Pruvot et al., 2006
landfill-field		1000	lettuce		1.3	0.0013	0.05	0.000065	8.125E-05	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	lettuce		0.25	0.0020	0.05	0.0001	0.000125	Samsoe-Petersen et al., 2002
field-wastewater	0.32	2.04	basil	0.18	0.84	0.4100	0.08	0.033	0.04125	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	garden cress	0.16	0.8	0.3900	0.08	0.031	0.03875	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	mint	0.29	0.78	0.3800	0.08	0.031	0.03875	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	tarragon	0.15	0.68	0.3300	0.08	0.027	0.03375	Shariatpanahi and Anderson 1986
flooded gardens		85.2	sorrel		0.99	0.0116	0.08	0.00093	0.001162	Sipter et al. 2008
non-flooded gardens		27.8	sorrel		0.295	0.0106	0.08	0.000849	0.0010612	Sipter et al. 2008
sewage sludge-field			spinach				0.08	0.00048	0.0006	Sridhara Chary et al., 2008
Indust. sewage wastes - field	3.4	183.5	amaranthus	0.12	12.2	0.0660	0.08	0.0054	0.00675	Srikanth et al., 1991
Indust. sewage wastes - field	3.4	183.5	cabbage	0.64	7.52	0.0410	0.078	0.0032	0.004	Srikanth et al., 1991
Indust. sewage wastes - field	3.4	183.5	spinach	0.05	14.94	0.0810	0.086	0.007	0.00875	Srikanth et al., 1991
urban gardens-field-to-grnhs	12	1601	lettuce	2.22	8.67	0.0080	0.045	0.00036	0.00045	Sterrett et al., 1996
field		71.31	Chinese cabbage		0.65	0.0091	0.08	0.000729	0.0009115	Wang et al. 2006
field		71.31	Pakchoi		0.7625	0.0107	0.08	0.000855	0.0010693	Wang et al. 2006
field		71.31	Water spinach		1.2125	0.0170	0.08	0.00136	0.0017003	Wang et al. 2006
field		400.3	Pakchoi		3.28	0.0680	0.08	0.00544	0.0068	Yan et al. 2007
field - smelter	21.6	319.6	leek			0.2760	0.08	0.02208	0.0276	Zheng et al. 2007a
field - smelter		158	Chinese cabbage				0.055	0.018	0.023	Zheng et al. 2007b
field - smelter		297	green onion				0.085	0.006	0.008	Zheng et al. 2007b
field - smelter		297	spinach				0.088	0.025	0.03	Zheng et al. 2007b

Table H.11-1 Lead field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt mg/kg	tissue conc contam dry wt mg/kg	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field - smelter		139	celery				0.058	0.016	0.02	Zheng et al. 2007b
field - smelter		111	cabbage				0.052	0.019	0.024	Zheng et al. 2007b
field - smelter		111	lettuce				0.042	0.024	0.03	Zheng et al. 2007b
field - smelter		167	mustard				0.071	0.021	0.026	Zheng et al. 2007b

Average lead uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0077±0.0104

Table H.11-2 Lead field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		12	peach		1.4	0.1167	0.131	0.015283	0.0191042	Basar and Aydmalp (2005)
field		12	peach		2.9	0.2417	0.131	0.031658	0.0395729	Basar and Aydmalp 2005
field		11	peach		0.8	0.0727	0.131	0.009527	0.0119091	Basar and Aydmalp 2005
field		137	cauliflower		2	0.0146	0.126	0.001839	0.0022993	Chumbley and Unwin 1982
indust. sewage-field-Egypt	ND	334	bell pepper		0.4	0.0010	0.074	0.00007	0.0000875	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	cucumber		0.3	0.0009	0.039	0.00004	0.00005	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	fig		0.6	0.0020	0.225	0.00045	0.0005625	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	olive		0.3	0.0009	0.2	0.0002	0.00025	Gorbunov et al., 2003
sewage sludge-field	21.1	67.4	Indian squash	0.33	1.4	0.0200	0.082	0.002	0.0025	Jamali et al., 2007
mining, smelting-field		223.22	capsicum			0.0370	0.074	0.0027	0.003375	Li et al., 2006
mining, smelting-field		223.22	cucumber			0.0460	0.039	0.0018	0.00225	Li et al., 2006
mining, smelting-field		223.22	eggplant			0.0220	0.073	0.0016	0.002	Li et al., 2006
field		14.49	broccoli		0.34	0.0235	0.126	0.002957	0.0036957	Liu et al. 2006
field		14.48	cucumber		1.39	0.0960	0.039	0.003744	0.0046797	Liu et al. 2006
field		14.48	Eggplant		1.3	0.0898	0.073	0.006554	0.0081923	Liu et al. 2006
field		14.48	kidney bean		0.91	0.0628	0.111	0.006976	0.0087198	Liu et al. 2006
field		14.48	pepper		4.25	0.2935	0.126	0.036982	0.0462276	Liu et al. 2006
field		14.47	tomato		5.23	0.3614	0.059	0.021325	0.026656	Liu et al. 2006
air dep, mine waste, poll. Water		751.98	capsicum		4.58	0.0061	0.074	0.00045	0.0005625	Liu et al., 2005
air dep, mine waste, poll. Water	60.49	751.98	string bean	0.84	5.82	0.0077	0.111	0.00086	0.001075	Liu et al., 2005
indust. Poll. Depo. - field		165.85	eggplant		13.15	0.0790	0.073	0.0058	0.00725	Pandey and Pandey, 2009
indust. Poll. Depo. - field		165.85	tomato		15.2	0.0920	0.059	0.0054	0.00675	Pandey and Pandey, 2009
smelter area - urban gardens - field	84	872	tomato	0	1.38	0.0016	0.065	0.0001	0.000125	Pruvot et al., 2006
Kalvebod area		613	blackberry					0.000026	0.0000325	Samsoe-Petersen et al., 2002
Kalvebod area		613	pear					0.000016	0.00002	Samsoe-Petersen et al., 2002
Kalvebod area		613	plum					0.000016	0.00002	Samsoe-Petersen et al., 2002
field-wastewater	0.32	2.04	leek	0.2	0.65	0.3200	0.12	0.038	0.0475	Shariatpanahi and Anderson

Table H.11-2 Lead field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
										1986
flooded gardens		85.2	squash		0.673	0.0079	0.082	0.000648	0.0008097	Sipter et al. 2008
flooded gardens		85.2	tomato		0.48	0.0056	0.059	0.000332	0.0004155	Sipter et al. 2008
non-flooded gardens		27.8	squash		0.079	0.0028	0.082	0.000233	0.0002913	Sipter et al. 2008
non-flooded gardens		27.8	tomato		0.083	0.0030	0.059	0.000176	0.0002202	Sipter et al. 2008
smelter contam - field	22	163	tomato		7.15	0.0440	0.065	0.0029	0.003625	Tomov & Alandjiyski, 2006
field		71.31	Eggplant		0.3973	0.0056	0.073	0.000407	0.0005083	Wang et al. 2006
field		71.31	Towel gourd		0.3415	0.0048	0.082	0.000393	0.0004908	Wang et al. 2006
field - smelter	21.6	319.6	aubergine			0.0240	0.066	0.001584	0.00198	Zheng et al. 2007a
field - smelter	21.6	319.6	capsicum			0.0240	0.081	0.001944	0.00243	Zheng et al. 2007a
field - smelter		297	tomato				0.056	0.002	0.003	Zheng et al. 2007b
field - smelter		167	bitter melon				0.066	0.003	0.004	Zheng et al. 2007b

Average lead uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00693±0.0124

Table H.11-3 Lead field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field	50	318.056	corn		1.1	0.0035	0.261	0.000903	0.0011283	Bi et al. 2006
field		156	sweet corn		0.1	0.0006	0.261	0.000167	0.0002091	Chumbley and Unwin 1982
25% mine waste - grnhs	60.9	3600	bush bean	5.53	0	-	0.099	0.00017	0.0002125	Cobb et al., 2000
indust. sewage-field-Egypt	ND	334	bean (spot)		2.2	0.0070	0.894	0.006	0.0075	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	bean (white)		0.9	0.0030	0.894	0.003	0.00375	Gorbunov et al., 2003
sewage sludge-field	21.1	67.4	cluster beans	0.104	0.6	0.0090	0.111	0.001	0.00125	Jamali et al., 2007
sewage sludge-field	21.1	67.4	peas	0.22	0.74	0.0100	0.257	0.003	0.00375	Jamali et al., 2007
mining, smelting-field		223.22	pumpkin			0.0470	0.082	0.0039	0.004875	Li et al., 2006
air dep, mine waste, poll. Water		751.98	corn		1.91	0.0025	0.261	0.00066	0.000825	Liu et al., 2005
field (sewage-fed lake irrigation)			Beans			0.2000	0.111	0.0222	0.02775	Lokeshwari and Chandrappa 2006
smelter area - ag field	30	440	corn	0	0.92	0.0021	0.273	0.00057	0.0007125	Pruvot et al., 2006
Kalvebod area		613	hazelnut					0.00073	0.0009125	Samsoe-Petersen et al., 2002
landfill-field		1000	green bean		1.4	0.0014	0.042	0.00006	0.000075	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	green bean		0.18	0.0010	0.111	0.0002	0.00025	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		154	beans			0.0080	0.222	0.002	0.0025	Sauerbeck, 1991
sewage sludge-pot-field		154	peas			0.0010	0.257	0.0003	0.000375	Sauerbeck, 1991
flooded gardens		85.2	bean		0.26	0.0031	0.111	0.000339	0.0004234	Sipter et al. 2008
non-flooded gardens		27.8	bean		0.141	0.0051	0.111	0.000563	0.0007037	Sipter et al. 2008
field		71.31	Cowpea		0.2023	0.0028	0.257	0.000729	0.0009115	Wang et al. 2006
field - smelter	21.6	319.6	kidney bean			0.0320	0.103	0.003296	0.00412	Zheng et al. 2007a
field - smelter		297	cowpea				0.097	0.003	0.004	Zheng et al. 2007b
field - smelter		297	pumpkin				0.065	0.001	0.001	Zheng et al. 2007b

Average lead uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00282±0.00565

Table H.11-4 Lead field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-ground water		28	potato		0.5	0.0179	0.222	0.003974	0.0049673	Alam et al. 2003
salt	40.5	744.5	carrot	0.312	5.754	0.0077	0.118	0.000912	0.00114	Alexander et al. (2006)
salt	40.5	744.5	Onion	1.418	7.458	0.0100	0.125	0.001252	0.0015652	Alexander et al. 2006
smelter - field - home gardens		130	carrot		2.2	0.0169	0.118	0.002	0.0025	Chaney et al., 1988
smelter - field - home gardens		48	potato		2.6	0.0542	0.202	0.01	0.0125	Chaney et al., 1988
field		103	beetroot		0.4	0.0039	0.222	0.000862	0.0010777	Chumbley and Unwin 1982
field		97	leeks		0.8	0.0082	0.2	0.001649	0.0020619	Chumbley and Unwin 1982
field		176	potato		0.2	0.0011	0.222	0.000252	0.0003153	Chumbley and Unwin 1982
field		110	radish		2.9	0.0264	0.222	0.005853	0.0073159	Chumbley and Unwin 1982
field		107	onions		0.6	0.0056	0.125	0.000701	0.0008762	Chumbley and Unwin 1982
25% mine waste - grnhs	60.9	3600	radish	0	92.4	0.0257	0.047	0.0012	0.0015	Cobb et al., 2000
smelter flue-dust	6.8	146.3	potato	0.2	0.2	0.0014	0.222	0.000303	0.0003794	Dudka et al. (1996)
smelter flue-dust	6.8	340	potato	0.2	0.4	0.0012	0.222	0.000261	0.0003265	Dudka et al. 1996
smelter flue-dust	6.8	2202.5	potato	0.2	0.7	0.0003	0.222	7.06E-05	8.82E-05	Dudka et al. 1996
smelter flue-dust	6.8	5452.5	potato	0.2	0.9	0.0002	0.222	3.66E-05	4.58E-05	Dudka et al. 1996
urban gardens-field			carrot				0.118	0.0006	0.00075	Finster et al., (2004)
urban gardens-field			onion				0.125	0.004	0.005	Finster et al., 2004
urban gardens-field			radish				0.047	0.00094	0.001175	Finster et al., 2004
indust. sewage-field-Egypt	ND	334	garlic		1	0.0030	0.387	0.001	0.00125	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	onion		1.1	0.0030	0.125	0.0004	0.0005	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	334	radish		2.3	0.0070	0.047	0.0003	0.000375	Gorbunov et al., 2003
sewage sludge-field	70	259	carrot	0.33	0.48	0.0040	0.118	0.0005	0.000625	Hooda et al., 1997
Pb arsenate - grnhs	60.9	342.3	carrot	3.9	13.3	0.0400	0.118	0.005	0.00625	Hutchinson et al. (1974)
Pb arsenate - grnhs	60.9	342.3	onion	10	75.4	0.2000	0.125	0.03	0.0375	Hutchinson et al. 1974
Pb arsenate - grnhs	60.9	342.3	parsnip	7.8	14.8	0.0400	0.209	0.008	0.01	Hutchinson et al. 1974
Pb arsenate - grnhs	60.9	342.3	radish	7.9	27.5	0.0800	0.047	0.004	0.005	Hutchinson et al. 1974
field		14.49	carrot		0.92	0.0635	0.118	0.007492	0.0093651	Liu et al. 2006
field		14.49	leek		0.92	0.0635	0.146	0.00927	0.0115873	Liu et al. 2006
field		14.48	radish		0.47	0.0325	0.047	0.001526	0.0019069	Liu et al. 2006
Env. contam. Soil (paint?) - potted - grnhs		2000	beet		19	0.0095	0.127	0.001	0.00125	Nicklow et al., 1983

Table H.11-4 Lead field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Common Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Env. contam. Soil (paint?) - potted - grnhs		2000	carrot		34	0.0170	0.118	0.002	0.0025	Nicklow et al., 1983
Env. contam. Soil (paint?) - potted - grnhs		2000	turnip		22	0.0110	0.085	0.0009	0.001125	Nicklow et al., 1983
indust. Poll. Depo. - field		165.85	carrot		8.16	0.0490	0.118	0.0058	0.00725	Pandey and Pandey, 2009
indust. Poll. Depo. - field		165.85	radish		11.7	0.0710	0.047	0.0033	0.004125	Pandey and Pandey, 2009
smelter area - ag field	30	440	potato	0.099	0.099	0.0002	0.202	0.000045	5.625E-05	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	carrot	0.25	1.17	0.0013	0.118	0.00024	0.0003	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	leek	0.34	2.67	0.0031	0.146	0.00045	0.0005625	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	potato	0	0.15	0.0002	0.202	0.000034	0.0000425	Pruvot et al., 2006
smelter area - urban gardens - field	84	872	radish	0	3.83	0.0044	0.047	0.00021	0.0002625	Pruvot et al., 2006
landfill-field		1000	carrot unp		5.1	0.0051	0.104	0.00053	0.0006625	Samsoe-Petersen et al., 2002
landfill-field		1000	potato unp		2	0.0020	0.113	0.00023	0.0002875	Samsoe-Petersen et al., 2002
landfill-field		1000	radish		7.4	0.0074	0.036	0.00027	0.0003375	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	carrot unp		0.93	0.0070	0.118	0.0009	0.001125	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	potato unp		0.18	0.0010	0.222	0.0003	0.000375	Samsoe-Petersen et al., 2002
moderate urban poll -field		130	radish		1.65	0.0100	0.085	0.001	0.00125	Samsoe-Petersen et al., 2002
sewage sludge-pot-field		154	carrots			0.0030	0.118	0.0004	0.0005	Sauerbeck, 1991
sewage sludge-pot-field		154	radish			0.0200	0.05	0.0009	0.001125	Sauerbeck, 1991
field-wastewater	0.32	2.04	onion	0.22	0.46	0.2300	0.125	0.028	0.035	Shariatpanahi and Anderson 1986
field-wastewater	0.32	2.04	radish	0.28	0.73	0.3600	0.047	0.02	0.025	Shariatpanahi and Anderson 1986
flooded gardens		85.2	carrot		0.81	0.0095	0.118	0.001122	0.0014023	Sipter et al. 2008
flooded gardens		85.2	onion		1.06	0.0124	0.125	0.001555	0.001944	Sipter et al. 2008
non-flooded gardens		27.8	carrot		0.278	0.0100	0.118	0.00118	0.001475	Sipter et al. 2008
non-flooded gardens		27.8	onion		0.13	0.0047	0.125	0.000585	0.0007307	Sipter et al. 2008
smelter contam - field	22	163	potato		2.95	0.0180	0.202	0.0037	0.004625	Tomov & Alandjiyski, 2006
field - smelter	21.6	319.6	carrot			0.0320	0.108	0.003456	0.00432	Zheng et al. 2007a
field - smelter	21.6	319.6	turnip			0.0270	0.088	0.002376	0.00297	Zheng et al. 2007a
field - smelter		167	potato				0.11	0.001	0.001	Zheng et al. 2007b

Average lead uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00403±0.0075

Table H.12-1 Mercury field studies on leafy crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
Hgt pots -env. chamber		17.6	cabbage		1.5	0.09	0.08	0.007	0.00875	Caille (2005)
Hgt pots -env. chamber		17.6	rape		1.7	0.09	0.08	0.008	0.01	Caille et al., 2005
field-compost			lettuce				0.05	0.0122355	0.0152944	Cappon 1987
field-compost			spinach				0.08	0.0137064	0.017133	Cappon 1987
field-compost			Swiss chard				0.08	0.01201	0.0150125	Cappon 1987
field		4.77	amaranth		0.27	0.0566038	0.08	0.0045283	0.0056604	Liu et al. 2006
field		4.77	cabbage		0.21	0.0440252	0.08	0.003522	0.0044025	Liu et al. 2006
field		4.77	celery		0.31	0.0649895	0.08	0.0051992	0.006499	Liu et al. 2006
field		4.77	Ch cabbage		0.15	0.0314465	0.08	0.0025157	0.0031447	Liu et al. 2006
field		4.77	Ch chive		0.32	0.067086	0.08	0.0053669	0.0067086	Liu et al. 2006
field		5.5	leek		0.19	0.0345455	0.08	0.0027636	0.0034545	Liu et al. 2006
field		4.77	pakchoi		0.41	0.0859539	0.08	0.0068763	0.0085954	Liu et al. 2006
field-contam fungicide -greenhouse grown	ND	1.64	lettuce		0.173	0.10549	0.05	0.0052745	0.0065931	(MacLean, 1974)
field-contam fungicide -greenhouse grown	ND	7.13	lettuce		0.103	0.01445	0.05	0.0007225	0.0009031	MacLean 1974
sewage sludge - field		2.5	cabbage		0.01	0.004	0.08	0.0003	0.000375	Muntau et al., 1987
field-wastewater	0.06	0.16	basil	0.05	0.08	0.5	0.08	0.04	0.05	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	gard cress	0.04	0.12	0.75	0.08	0.06	0.075	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	mint	0.06	0.08	0.5	0.08	0.04	0.05	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	tarragon	0.04	0.13	0.81	0.08	0.065	0.08125	Shariatpanahi and Anderson 1986
flooded gardens		0.81	sorrel		0.06	0.0740741	0.08	0.0059259	0.0074074	Sipter et al. 2008
field - smelter	0.037	1.28	leek			0.139	0.08	0.01112	0.0139	Zheng et al. 2007a
field - smelter	0.037	0.76	Ch cabbage				0.055	0.016	0.02	Zheng et al. 2007a
field - smelter	0.037	1.5	Grn onion				0.085	0.01	0.0125	Zheng et al. 2007a
field - smelter	0.037	1.5	spinach				0.088	0.005	0.00625	Zheng et al. 2007a
field - smelter	0.037	0.4	celery				0.058	0.01	0.0125	Zheng et al. 2007a
field - smelter	0.037	0.5	cabbage				0.052	0.031	0.03875	Zheng et al. 2007a
field - smelter	0.037	0.5	lettuce				0.042	0.015	0.01875	Zheng et al. 2007a

Average mercury uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0163±0.0202

Table H.12-2 Mercury field studies on exposed crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field survey			peppers			0.00222	0.126	0.0002797	0.0003497	Cambra et al. 1999
field-compost			broccoli				0.126	0.0145385	0.0181731	Cappon 1987
field-compost			cabbage				0.08	0.0120093	0.0150117	Cappon 1987
field-compost			cucmber				0.039	0.0002636	0.0003295	Cappon 1987
field-compost			pepper				0.074	0.0014145	0.0017681	Cappon 1987
field-compost			squash				0.082	0.0016629	0.0020787	Cappon 1987
field-compost			tomato				0.059	0.0036445	0.0045557	Cappon 1987
field		5.5	broccoli		0.12	0.0218182	0.126	0.0027491	0.0034364	Liu et al. 2006
field		4.03	cucumber		0.15	0.0372208	0.039	0.0014516	0.0018145	Liu et al. 2006
field		4.77	Eggplant		0.26	0.0545073	0.073	0.003979	0.0049738	Liu et al. 2006
field		4.77	kidney bean		0.27	0.0566038	0.111	0.006283	0.0078538	Liu et al. 2006
field		4.77	pepper		0.14	0.0293501	0.126	0.0036981	0.0046226	Liu et al. 2006
field		4.77	tomato		0.13	0.0272537	0.059	0.001608	0.00201	Liu et al. 2006
pots - phenyl mercuric acetate	0.08	5.24	tomato	0.034	0.037	0.0071	0.059	0.00042	0.000525	MacLean 1974
field-wastewater	0.06	0.16	leek	0.04	0.1	0.63	0.12	0.075	0.09375	Shariatpanahi and Anderson 1986
flooded gardens		0.81	squash		0.037	0.045679	0.082	0.0037457	0.0046821	Sipter et al. 2008
flooded gardens		0.81	tomato		0.01	0.0123457	0.059	0.0007284	0.0009105	Sipter et al. 2008
field - smelter	0.037	1.28	aubergine			0.003	0.066	0.000198	0.0002475	Zheng et al. 2007a
field - smelter	0.037	1.28	capsicum			0.007	0.081	0.000567	0.0007088	Zheng et al. 2007a
field - smelter	0.037	1.5	tomato				0.056	0.004	0.005	Zheng et al. 2007a
field - smelter	0.037	0.3	bitter melon				0.066	0.016	0.02	Zheng et al. 2007a

Average mercury uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00855±0.0194

Table H.12-3 Mercury field studies on protected crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field survey			broad beans			0.003506	0.126	0.0004418	0.0005522	Cambra et al. 1999
field-compost			bean				0.111	0.0011126	0.0013907	Cappon 1987
field	0.15	0.38	corn		0.011	0.0289474	0.261	0.0075553	0.0094441	Feng et al. (2006)
Hgt field-smelter-9 sites			brown rice			0.002	0.888	0.002	0.0025	Horvet et al., 2003
Hgt field-smelter-2 sites			brown rice			0.0001	0.888	0.00009	0.0001125	Horvet et al., 2003
Hgt field-clean area-2 sites			brown rice			0.009	0.888	0.008	0.01	Horvet et al., 2003
field		0.21	wheat		0.003	0.0142857	0.875	0.0125	0.015625	Huang et al. (2008)
HgCl2 - pots - chamber	ND		oats	0.009	0.013	0.002	0.917	0.0018	0.00225	John 1972
HgCl2 - pots - chamber	ND		peas	0.001	0.002	0.00033	0.257	0.000085	0.0001063	John 1972
Hgt field-smelter-23 sites		0.1782	corn		0.0061	0.03	0.261	0.0089	0.011125	Li et al., (2008)
pots - phenyl mercuric acetate	0.08	5.24	oats	0.113	0.163	0.031	0.917	0.029	0.03625	MacLean 1974
pots - phenyl mercuric acetate	0.08	5.24	soybeans	0.074	0.076	0.015	0.925	0.013	0.01625	MacLean 1974
flooded gardens		0.81	bean		0.03	0.037037	0.111	0.0041111	0.0051389	Sipter et al. 2008
field - smelter	0.037	1.28	kidney bean			0.067	0.103	0.006901	0.0086263	Zheng et al. 2007a
field - smelter	0.037	1.5	cowpea				0.097	0.001	0.00125	Zheng et al. 2007a

Average mercury uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00804±0.0096

Table H.12-4 Mercury field studies on root crops.

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-compost			Beet				0.164	0.0104746	0.0130932	Cappon 1987
field-compost			carrot				0.118	0.0036308	0.0045385	Cappon 1987
field-compost			onion				0.125	0.0105478	0.0131847	Cappon 1987
field-compost			radish				0.222	0.0129371	0.0161713	Cappon 1987
field-compost			turnip				0.222	0.0056406	0.0070507	Cappon 1987
HgCl2 - pots - chamber	ND		carrot	0.044	0.053	0.0075	0.118	0.00089	0.0011125	John (1972)
HgCl2 - pots - chamber	ND		radish	0.013	0.026	0.02	0.085	0.0017	0.002125	John 1972
field		5.5	carrot		0.24	0.0436364	0.118	0.0051491	0.0064364	Liu et al. 2006
field		4.77	radish		0.21	0.0440252	0.2	0.008805	0.0110063	Liu et al. 2006
pots - phenyl mercuric acetate	0.08	5.24	carrot	0.086	0.18	0.034	0.118	0.0041	0.005125	MacLean 1974
pots - phenyl mercuric acetate	0.08	5.24	potato	0.047	0.055	0.01	0.222	0.0023	0.002875	MacLean 1974
field-wastewater	0.06	0.16	onion	0.06	0.06	0.38	0.125	0.047	0.05875	Shariatpanahi and Anderson 1986
field-wastewater	0.06	0.16	radish	0.04	0.08	0.5	0.085	0.043	0.05375	Shariatpanahi and Anderson 1986
flooded gardens		0.81	carrot		0.02	0.0246914	0.118	0.0029136	0.003642	Sipter et al. 2008
flooded gardens		0.81	onion		0.02	0.0246914	0.125	0.0030864	0.003858	Sipter et al. 2008
field - smelter	0.037	1.28	carrot			0.044	0.108	0.004752	0.00594	Zheng et al. 2007a
field - smelter	0.037	1.28	turnip			0.034	0.088	0.002992	0.00374	Zheng et al. 2007a
field - smelter	0.037	0.3	potato				0.11	0.002	0.0025	Zheng et al. (2007b)

Average mercury uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0119±0.0167

Table H.13-1 Nickel field studies on leafy crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field (industrial sewage irrigation)		13.37	palak (spinach)		4.2	0.31	0.08	0.02	0.025	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		15.61	palak (spinach)		5.9	0.38	0.08	0.03	0.0375	Kumar Sharma et al., 2007
field (industrial sewage irrigation)		14.52	palak (spinach)		2.6	0.18	0.08	0.02	0.025	Kumar Sharma et al., 2007
indust. Poll. Depo. - field		119.32	amaranthus		9.5	0.08	0.08	0.0064	0.008	Pandey and Pandey, 2009
indust. Poll. Depo. - field		119.32	spinach		10.62	0.089	0.08	0.0071	0.008875	Pandey and Pandey, 2009
landfill-field		49	lettuce		1.23	0.025	0.05	0.00125	0.0015625	Samsoe-Petersen et al., 2002
sewage sludge - field		120	cabbage		24	0.2	0.08	0.02	0.025	Muntau et al., 1987
sewage sludge-field	22.5	51.8	spinach	4.76	9.46	0.178	0.08	0.014	0.0175	Hooda et al., 1997
sewage sludge-field	28.1	34.6	spinach	0.88	1.2	0.03	0.08	0.003	0.00375	Jamali et al., 2007
sewage sludge-field			spinach				0.08	0.0048	0.006	Sridhara Chary et al., (2008)
urban gardens-field-to-greenhouse	10	50.7	lettuce	0.73	1.25	0.024	0.045	0.00108	0.00135	Sterrett et al., 1996

Average nickel uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0145±0.0121

Table H.13-2 Nickel field studies on exposed crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field		112	peach		1.5	0.0133929	0.131	0.0017545	0.0021931	Basar and Aydmalp 2005
field		117	peach		1.6	0.0136752	0.131	0.0017915	0.0022393	Basar and Aydmalp 2005
field		122	peach		2	0.0163934	0.131	0.0021475	0.0026844	Basar and Aydmalp 2005
highly contam area		53	blackberry					0.0021	0.002625	Samsoe-Petersen et al., 2002
highly contam area		53	pear					0.0013	0.001625	Samsoe-Petersen et al., 2002
highly contam area		53	plum					0.0007	0.000875	Samsoe-Petersen et al., 2002
indust. Poll. Depo. - field		119.32	eggplant		7.92	0.066	0.073	0.0048	0.006	Pandey and Pandey, 2009
indust. Poll. Depo. - field		119.32	tomato		9.85	0.083	0.059	0.0049	0.006125	Pandey and Pandey, 2009
indust. sewage-field-Egypt	ND	106	bell pepper		0.7	0.007	0.074	0.0005	0.000625	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	cucumber		0.43	0.004	0.039	0.0002	0.00025	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	fig		1.6	0.02	0.225	0.0045	0.005625	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	olive		0.41	0.004	0.2	0.0008	0.001	Gorbunov et al., 2003
sewage sludge-field	28.1	34.6	Indian squash	1.3	2.1	0.06	0.082	0.005	0.00625	Jamali et al., 2007

Average nickel uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00293±0.00226

Table H.13-3 Nickel field studies on protected crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field (sewage-fed lake irrigation)			Beans			0.1	0.111	0.0111	0.013875	Lokeshwari and Chandrappa (2006)
highly contam area		53	hazelnut					0.033	0.04125	Samsoe-Petersen et al., 2002
indust. sewage-field-Egypt	ND	106	bean (spot)		6.9	0.07	0.894	0.06	0.075	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	bean (white)		1.9	0.02	0.894	0.02	0.025	Gorbunov et al., 2003
landfill-field		49	green bean		6.37	0.13	0.076	0.0099	0.012375	Samsoe-Petersen et al., 2002
sewage sludge-field	28.1	34.6	cluster beans	1.21	2.1	0.06	0.111	0.007	0.00875	Jamali et al., 2007
sewage sludge-field	28.1	34.6	peas	1.12	1.18	0.03	0.257	0.009	0.01125	Jamali et al., 2007
sewage sludge-pot-field		25	beans			0.3	0.099	0.03	0.0375	Sauerbeck, 1991
sewage sludge-pot-field		25	peas			0.2	0.257	0.04	0.05	Sauerbeck, 1991

Average nickel uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0306±0.0224

Table H.13-4 Nickel field studies on root crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
indust. Poll. Depo. - field		119.32	carrot		3.65	0.031	0.118	0.0037	0.004625	Pandey and Pandey, 2009
indust. Poll. Depo. - field		119.32	radish		3.98	0.033	0.047	0.0016	0.002	Pandey and Pandey, 2009
indust. sewage-field-Egypt	ND	106	garlic		2.6	0.02	0.125	0.003	0.00375	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	onion		3.1	0.03	0.125	0.004	0.005	Gorbunov et al., 2003
indust. sewage-field-Egypt	ND	106	radish		3.8	0.04	0.085	0.003	0.00375	Gorbunov et al., 2003
landfill-field		49	carrot (unpeeled)		1.86	0.038	0.132	0.005	0.00625	Samsoe-Petersen et al., 2002
landfill-field		49	potato (unpeeled)		0.34	0.007	0.185	0.0013	0.001625	Samsoe-Petersen et al., 2002
landfill-field		49	radish		1.57	0.032	0.048	0.0015	0.001875	Samsoe-Petersen et al., 2002
sewage sludge-field	22.5	51.8	carrot	2.17	5.28	0.118	0.118	0.014	0.0175	Hooda et al., (1997)
sewage sludge-pot-field		25	carrots			0.08	0.118	0.009	0.01125	Sauerbeck, 1991
sewage sludge-pot-field		25	radish			0.2	0.05	0.01	0.0125	Sauerbeck, 1991

Average nickel uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.00638±0.00516

Table H.15-1 Selenium field studies on leafy crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-fly ash	1.5	1.7	cabbage	0.07	0.2	0.1	0.08	0.009	0.01125	Furr et al. 1978
sewage sludge - field		0.4	cabbage		1.1	2.8	0.08	0.2	0.25	Muntau et al., 1987
field-compost			lettuce				0.05	0.008482	0.0106025	Cappon 1987
field-compost			lettuce				0.05	0.010372	0.012965	Cappon 1987
field		9.84	lettuce		19.16	1.94715	0.05	0.0973575	0.1216969	van Mantgem et al. (1996)
field		6.18	lettuce		5.61	0.90777	0.05	0.0453885	0.0567356	van Mantgem et al. 1996
field		15.9	lettuce		13.63	0.85723	0.05	0.0428615	0.0535769	van Mantgem et al. 1996
field		16.83	lettuce		27.9	1.65775	0.05	0.0828875	0.1036094	van Mantgem et al. 1996
field		17.37	lettuce		12.37	0.71215	0.05	0.0356075	0.0445094	van Mantgem et al. 1996
field-compost			spinach				0.08	0.016888	0.02111	Cappon 1987
field-compost			Swiss chard				0.08	0.00957	0.0119625	Cappon 1987

Average selenium uptake factor in leafy crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0587±0.0713

Table H.15-2 Selenium field studies on exposed crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-fly ash-potted soil	0.3	1.2	apple (w/o seeds)	0.01	0.03	0.03	0.159	0.004	0.005	Furr et al. (1979)
field-compost			broccoli				0.126	0.0130125	0.0162656	Cappon 1987
field-fly ash-potted soil	0.3	1.2	cabbage	0.04	2.4	2	0.08	0.2	0.25	Furr et al. 1979
field-compost			cabbage				0.08	0.0216667	0.0270833	Cappon 1987
field-compost			cucmber				0.039	0.0010563	0.0013203	Cappon 1987
field-compost			pepper				0.074	0.0025107	0.0031384	Cappon (1987)
field-compost			squash				0.082	0.0027089	0.0033862	Cappon 1987
field-fly ash-potted soil	0.3	1.2	tomato	0.015	1.5	1.2	0.059	0.07	0.0875	Furr et al. 1979
field-compost			tomato				0.059	0.0099387	0.0124234	Cappon 1987
field-fly ash - pot	1.5	1.7	tomato	0.01	0.02	0.01	0.059	0.007	0.00875	Furr et al. 1978

Average selenium uptake factor in exposed crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0415±0.0776

Table H.15-3 Selenium field studies on protected crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-compost			bean				0.111	0.0070366	0.0087958	Cappon 1987
field-smelter		16.9	brown rice		1.06	0.06	0.888	0.056	0.07	Horvet et al., (2003)
field-fly ash - pot	1.5	1.7	bush bean	0.02	0.07	0.04	0.111	0.005	0.00625	Furr et al. 1978
field-fly ash-potted soil	0.3	1.2	bush bean	0.025	1.3	1.1	0.111	0.1	0.125	Furr et al. 1979
field-fly ash - pot	1.5	1.7	corn	0.02	0.05	0.03	0.895	0.03	0.0375	Furr et al. 1978
field-fly ash-potted soil	0.3	1.2	Japanese millet grain	0.025	1.4	1.1	0.888	1	1.25	Furr et al. 1979
field-fly ash-potted soil			onion		2.3	1.9	0.125	0.2375	0.296875	Furr et al. 1979

Average selenium uptake factor in protected crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.256±0.450

Table H.15-4 Selenium field studies on root crops

Study Type	soil conc bckd (mg/kg)	soil conc contam (mg/kg)	Crop Name	tissue conc bckg dry wt (mg/kg)	tissue conc contam dry wt (mg/kg)	Uptake factor (contam) dry wt	dry-to-wet wt conver-sion factor	Uptake factor (contam) wet wt plant/dw soil	Uptake factor (contam) ww plant/wet w soil	Reference
field-compost			Beet				0.164	0.0098107	0.0122634	Cappon 1987
field-fly ash-potted soil	0.3	1.2	carrot	0.015	1.5	1.3	0.118	0.1	0.125	Furr et al. 1979
field-compost			carrot				0.118	0.0082179	0.0102723	Cappon 1987
field-fly ash - pot	1.5	1.7	carrot (peeled)	0.02	0.06	0.04	0.118	0.004	0.005	Furr et al. 1978
field-compost			onion				0.125	0.0550223	0.0687779	Cappon 1987
field-fly ash - pot	1.5	1.7	Onion (peeled)	0.02	0.21	0.1	0.125	0.02	0.025	Furr et al. 1978
field-fly ash-potted soil	0.3	1.2	potato	0.025	1.8	1.5	0.222	0.3	0.375	Furr et al. 1979
field-fly ash - pot	1.5	1.7	Potato (peeled)	0.02	0.03	0.02	0.222	0.004	0.005	Furr et al. (1978b)
field-compost			radish				0.222	0.0391143	0.0488929	Cappon 1987
field-compost			turnip				0.222	0.0112321	0.0140402	Cappon 1987

Average selenium uptake factor in root crops (fresh weight conc. in plant / wet weight conc. in soil) = 0.0689±0.114

H.13 References

Alam MGM, Snow ET and Tanaka A (2003). Arsenic and heavy metal contamination of vegetables grown in Samta village, Bangladesh. Sci Total Environ 308(1-3): 83-96.

Alexander PD, Alloway BJ and Dourado AM (2006). Genotypic variations in the accumulation of Cd, Cu, Pb and Zn exhibited by six commonly grown vegetables. Environ Pollut 144(3): 736-745.

BaÅŸar H and Aydinalp C (2005). Heavy metal contamination in peach trees irrigated with water from a heavily polluted creek. J Plant Nutr 28(11): 2049 - 2063.

Baes CFI, Sharp RD, Sjoreen AL and Shor RW. (1984). A review and analysis of parameters for assessing transport of environmentally released radionuclides through agriculture. ORNL-5786. U. S. Dept. of Energy. Oak Ridge, TN.

Bartlett RJ and James BR (1988). Mobility and bioavailability of chromium in soils. In: Chromium in the Natural and Human Environments. Nraigu J. O. and Nieborer E., eds Wiley. New York: 267-303.

Bhumbla D and Keefer R (1994). Arsenic mobilization and bioavailability in soils. In: Advances in Environmental Science and Technology. Nriagu J. O. John Wiley and Sons, Inc. New York, New York, USA; Chichester, England, UK. : 26: 51-82.

Bi X, Feng X, Yang Y, Qiu G, Li G, Li F, Liu T, Fu Z and Jin Z (2006). Environmental contamination of heavy metals from zinc smelting areas in Hezhang County, western Guizhou, China. Environ Int 32(7): 883-890.

Bloomfield C and Pruden G (1980). The behaviour of Cr(VI) in soil under aerobic and anaerobic conditions. Environmental Pollution Series A, Ecological and Biological 23(2): 103-114.

Caille N, Vauleon C, Leyval C and Morel J-L (2005). Metal transfer to plants grown on a dredged sediment: use of radioactive isotope 203Hg and titanium. Sci Total Environ 341(1-3): 227-239.

Cambra K, MartÃ-nez T, Urzelai A and Alonso E (1999). Risk Analysis of a Farm Area Near a Lead- and Cadmium-Contaminated Industrial Site. J Soil Contamin 8(5): 527 - 540.

Cappon CJ (1987). Uptake and speciation of mercury and selenium in vegetable crops grown on compost-treated soil. Water Air Soil Pollut 34(4): 353-361.

Cary E (1982). Chromium in air, soil and natural waters. In: Biological and Environmental Aspects of Chromium, Langard S. ed., Elsevier. Amsterdam: 49-64.

Cary EE (1977a). Control of chromium concentrations in food plants. 1. Absorption and translocation of chromium by plants. J Agric Food Chem 25(2): 300-304.

Cary EE (1977b). Control of chromium concentrations in food plants. 2. Chemistry of chromium in soils and its availability to plants. Journal of agricultural and food chemistry 25(2): 305-309.

Chaney RL, Beyer WN, Gifford CH and Sileo L (1988). Effects of zinc smelter emissions on farms and gardens at Palmerton, PA. in: Trace Substances in Environmental Health - 22. Hemphill DD (ed), University of Missouri, Columbia, pp 263-280

Chumbley CG and Unwin RJ (1982). Cadmium and lead content of vegetable crops grown on land with a history of sewage sludge application. Environmental Pollution Series B, Chemical and Physical 4(3): 231-237.

Clement Associates I. (1988). Multi-pathway Health Risk Assessment Input Parameters Guidance Document. Prepared for the South Coast Air Quality Management District by Clement Associates, Inc. Fairfax, Virginia

Clemente R, Walker DJ and Bernal MP (2005). Uptake of heavy metals and As by Brassica juncea grown in a contaminated soil in Aznalcóllar (Spain): The effect of soil amendments. Environ Pollut 138(1): 46-58.

Cobb GP, Sands K, Waters M, Wixson BG and Dorward-King E (2000). Accumulation of heavy metals by vegetables grown in mine wastes. Environ Toxicol Chem 19(3): 600-607.

Crews HM and Davies BE (1985). Heavy metal uptake from contaminated soils by six varieties of lettuce (Lactuca sativa L.). J Agric Sci 105(03): 591-595.

Davison A (1982). The effects of fluorides on plant growth and forage quality. In: Effects of Gaseous Pollutants in Agriculture and Horticulture. Unsworth M. and Ormrod D. University of Nottingham School of Agriculture Butterworth, London: 267-292.

Davison A (1983). Uptake, transport and accumulation of.soil and airborne fluorides by vegetation. In: Fluorides : Effects on Veftetation, Animals atid Humans. Shupe J., Peterson H. and Leone N.(eds) Paragon Press. Salt Lake City, Utah 61-82.

Dudka S, Piotrowska M and Terelak H (1996). Transfer of cadmium, lead, and zinc from industrially contaminated soil to crop plants: A field study. Environ Pollut 94(2): 181-188.

Feng X, Li G and Qiu G (2006). A preliminary study on mercury contamination to the environment from artisanal zinc smelting using indigenous methods in Hezhang County, Guizhou, China: Part 2. Mercury contaminations to soil and crop. Sci Total Environ 368(1): 47-55.

Finster ME, Gray KA and Binns HJ (2004). Lead levels of edibles grown in contaminated residential soils: a field survey. Sci Total Environ 320(2-3): 245-257.

Furr AK (1978a). Elemental content of tissues and excreta of lambs, goats, and kids fed white sweet clover growing on fly ash. J Agric Food Chem 26(4): 847-851.

Furr AK (1978b). Elemental content of vegetables, grains, and forages field-grown on fly ash amended soil. J Agric Food Chem 26(2): 357-359.

Furr AK, Parkinson TF, Elfving DC, Gutenmann WH, Pakkala IS and Lisk DJ (1979). Elemental content of apple, millet, and vegetables grown in pots of neutral soil amended with fly ash. J Agric Food Chem 27(1): 135-138.

Giordano PM, Mays DA and Behel AD (1979). Soil temperature effects on uptake of cadmium and zinc by vegetables grown on sludge-amended soil. J Environ Qual 8(2): 233-236.

Gnamus A, Zupan M and Sajn R (2001). Mercury and methylmercury in soil and vegetation of various polluted areas in Slovenia. RMZ - Materials and Geoenvironment 48(1): 94-108.

Gorbunov AV, Frontasyeva MV, Kistanov AA, Lyapunov SM, Okina OI and Ramadan AB (2003). Heavy and Toxic Metals in Staple Foodstuffs and Agriproduct from Contaminated Soils. J Environ Sci Health B 38(2): 181 - 192.

He QB and Singh BR (1994). Crop uptake of cadmium from phosphorus fertilizers: I. Yield and cadmium content. Water Air Soil Pollut 74(3): 251-265.

Holmgren GGS, Meyer MW, Chaney RL and Daniels RB (1993). Cadmium, lead, zinc, copper, and nickel in agricultural soils of the United States of America. J Environ Qual 22(2): 335-348.

Hooda PS, McNulty D, Alloway BJ and Aitken MN (1997). Plant availability of heavy metals in soils previously amended with heavy applications of sewage sludge. J Sci Food Agric 73(4): 446-454.

Horvat M, Nolde N, Fajon V, Jereb V, Logar M, Lojen S, Jacimovic R, Falnoga I, Liya Q, Faganeli J and Drobne D (2003). Total mercury, methylmercury and selenium in mercury polluted areas in the province Guizhou, China. Sci Total Environ 304(1-3): 231-256.

Hu X and Ding Z (2009). Lead/Cadmium Contamination and Lead Isotopic Ratios in Vegetables Grown in Peri-Urban and Mining/Smelting Contaminated Sites in Nanjing, China. Bull Environ Contam Toxicol 82(1): 80-84.

Huang B, Kuo S and Bembenek R (2003). Cadmium uptake by lettuce from soil amended with phosphorus and trace element fertilizers. Water Air Soil Pollut 147(1): 109-127.

Huang B, Kuo S and Bembenek R (2004). Availability of cadmium in some phosphorus fertilizers to field-grown lettuce. Water Air Soil Pollut 158(1): 37-51.

Huang M, Zhou S, Sun B and Zhao Q (2008). Heavy metals in wheat grain: Assessment of potential health risk for inhabitants in Kunshan, China. Sci Total Environ 405(1-3): 54-61.

Huang R-Q, Gao S-F, Wang W-L, Staunton S and Wang G (2006). Soil arsenic availability and the transfer of soil arsenic to crops in suburban areas in Fujian Province, southeast China. Sci Total Environ 368(2-3): 531-541.

Huq SMI and Naidu R (2005). Arsenic in groundwater and contamination of the food chain: Bangladesh scenario. In: Natural Arsenic in Groundwater: Occurrence, Remediation and Management. Bundschuh J. (ed). Taylor & Francis Group. London: 95-101.

Hutchinson TC, Czuba M and Cunningham L (1974). Lead, cadmium, zinc, copper and nickel distributions in vegetables and soils of an intensely cultivated area and levels of copper, lead and zinc in the growers. in: Trace Substances in Environmental Health - 8, University of Missouri, Columbia, pp 81-93

Jackson AP and Alloway BJ (1991). The transfer of cadmium from sewage-sludge amended soils into the edible components of food crops. Water Air Soil Pollut 57-58(1): 873-881.

Jamali MK, Kazi TG, Arain MB, Afridi HI, Jalbani N and Memon AR (2007). Heavy metal contents of vegetables grown in soil, irrigated with mixtures of wastewater and sewage sludge in Pakistan, using ultrasonic-assisted pseudo-digestion. J Agron Crop Sci 193(3): 218-228.

James BR and Bartlett RJ (1984). Plant-soil interactions of chromium. J Environ Qual 13(1): 67.

Jansson G and Ã–born I (2000). Cadmium Content of Swedish Carrots and the Influence of Soil Factors. Acta Agric Scand B 50(2): 49 - 56.

John MK (1972). Mercury uptake from soil by various plant species. Bull Environ Contam Toxicol 8(2): 77-80.

Kimbrough DE, Cohen Y, Winer A, Creelman L and Mabuni C (1999). A critical assessment of chromium in the environment. Crit Rev Environ Sci Tech 29: 1.

Kloke A, Sauerbeck D and Vetter H (1984). The contamination of plants and soils with heavy metals and the transport of metals in terrestrial food chains. In: Changing Metal Cycles and Human Health: Report of the Dahlem Workshop on Changing Metal Cycles and Human Health. Nriagu J. Springer-Verlag, Berlin. Berlin, Germany: 113-141.

Kronberger W (1987). Kinetics of nonionic diffusion of hydrogen fluoride in plants I. Experimental and theoretical treatment of weak acid permeation. Phyton (Horn) 27(2): 241-265.

Kumar Sharma R, Agrawal M and Marshall F (2007). Heavy metal contamination of soil and vegetables in suburban areas of Varanasi, India. Ecotoxicol Environ Safety 66(2): 258-266.

Kumpulainen J and Koivistoinen P (1977). Fluorine in foods. Residue Rev 68: 37-57.

Lahouti M (1979). Chromium accumulation and distribution in crop plants. J Sci Food Agric 30(2): 136-142.

Larsen EH, Moseholm L and Nielsen MM (1992). Atmospheric deposition of trace elements around point sources and human health risk assessment. II: Uptake of arsenic and chromium by vegetables grown near a wood preservation factory. Sci Total Environ 126(3): 263-275.

Li G, Feng X, Qiu G, Bi X, Li Z, Zhang C, Wang D, Shang L and Guo Y (2008). Environmental mercury contamination of an artisanal zinc smelting area in Weining County, Guizhou, China. Environ Pollut 154(1): 21-31.

Li Y, Wang Y, Gou X, Su Y and Wang G (2006). Risk assessment of heavy metals in soils and vegetables around non-ferrous metals mining and smelting sties, Baiyin, China. J Environ Sci 18(1124-1134).

Lindberg SE, Jackson DR, Huckabee JW, Janzen SA, Levin MJ and Lund JR (1979). Atmospheric emission and plant uptake of mercury from agricultural soils near the Almaden mercury mine. J Environ Qual 8(4): 572-578.

Liu H, Probst A and Liao B (2005). Metal contamination of soils and crops affected by the Chenzhou lead/zinc mine spill (Hunan, China). Sci Total Environ 339(1-3): 153-166.

Liu WX, Li HH, Li SR and Wang YW (2006). Heavy Metal Accumulation of Edible Vegetables Cultivated in Agricultural Soil in the Suburb of Zhengzhou City, People's Republic of China. Bull Environ Contam Toxicol 76(1): 163-170.

Lokeshwari H (2006). Impact of heavy metal contamination of Bellandur Lake on soil and cultivated vegetation. Curr Science (Bangalore) 91(5): 622.

MacLean AJ (1974). Mercury in plants and retention of mercury by soils in relation to properties and added sulfur. Can J Soil Sci 54: 287.

Mahler RJ, Ryan JA and Reed T (1987). Cadmium sulfate application to sludge-amended soils I. Effect on yield and cadmium availability to plants. Sci Total Environ 67(2-3): 117-131.

Mathe-Gaspar G and Anton A (2002). Heavy metal uptake by two radish varieties. Acta Biol Szegediensis 46: 113-114.

Mattina MI, Lannucci-Berger W, Musante C and White JC (2003). Concurrent plant uptake of heavy metals and persistent organic pollutants from soil. Environ Pollut 124(3): 375-378.

McBride MB (1998). Growing food crops on sludge-amended soils: problems with the U.S. Environmental Protection Agency method of estimating toxic metal transfer. Environ Toxicol Chem 17(11): 2274-2281.

McLaughlin MJ, Parker DR and Clarke JM (1999). Metals and micronutrients - food safety issues. Field Crops Res 60(1-2): 143-163.

McLaughlin MJ, Tiller KG, Naidu R and Stevens DP (1996). The behavior and environmental impact of contaminants in fertilizers. Austr J Soil Res 34(1): 1-54.

Mulla DJ, Page AL and Ganje TJ (1980). Cadmium accumulations and bioavailability in soils from long-term phosphorus fertilization. J Environ Qual 9(3): 408-412.

Muntau H, Crössmann G, Schramel P, Gallorini M and Orvini E (1987). Trace and nutrient element transfer from sewage sludge-amended soil to crop. Fresenius' J Anal Chem 326(7): 634-635.

Nan Z, Li J, Zhang J and Cheng G (2002). Cadmium and zinc interactions and their transfer in soil-crop system under actual field conditions. Sci Total Environ 285(1-3): 187-195.

Nelson DW, Liu SL and Sommers LE (1984). Extractability and plant uptake of trace elements from drilling fluids. J Environ Qual 13(4): 562-566.

Nicklow CW (1983). Influence of varying soil lead levels on lead uptake of leafy and root vegetables. J Am Soc Hortic Sci 108(2): 193.

Pandey J and Pandey U (2009). Accumulation of heavy metals in dietary vegetables and cultivated soil horizon in organic farming system in relation to atmospheric deposition in a seasonally dry tropical region of India. Environ Monit Assess 148(1): 61-74.

Preer JR, Abdi AN, Sekhon HS and Murchison GB (1995). Metals in urban gardens - effect of lime and sludge. J Environ Sci Health A 30(9): 2041 - 2056.

Pruvot C, Douay F, Hervé F and Waterlot C (2006). Heavy metals in soil, crops and grass as a source of human exposure in the former mining areas. J Soil Sed 6(4): 215-220.

Rayman MP (2008). Food-chain selenium and human health: emphasis on intake. Br J Nutr 100(2): 254-268.

Rayman MP, Infante HG and Sargent M (2008). Food-chain selenium and human health: spotlight on speciation. Br J Nutr 100(2): 238-253.

Samsæ-Petersen L, Larsen EH, Larsen PB and Bruun P (2002). Uptake of trace elements and PAHs by fruit and vegetables from contaminated soils. Environ Sci Technol 36(14): 3057-3063.

Sauerbeck DR and Hein A (1991). The nickel uptake from different soils and its prediction by chemical extractions. Water Air Soil Pollut 57-58(1): 861-871.

Schroeder HA and Balassa JJ (1963). Cadmium: Uptake by vegetables from superphosphate in soil. Science 140(3568): 819-820.

Shacklette H, Erdman J and Harms T (1978). Trace elements in plant foodstuffs. In: Toxicity of Heavy Metals in the Environment, Part 1. Oehme F.(ed) New York: Marcel Dekker**:** 25-43.

Shariatpanahi M and Anderson AC (1986). Accumulation of cadmium, mercury and lead by vegetables following long-term land application of wastewater. Sci Total Environ 52(1-2): 41-47.

Sheppard SC, Evenden WG and Amiro BD (1993). Investigation of the soil-to-plant pathway for I, Br, Cl and F. J Environ Radioact 21(1): 9-32.

Sipter E, Rózsa E, Gruiz K, Tátrai E and Morvai V (2008). Site-specific risk assessment in contaminated vegetable gardens. Chemosphere 71(7): 1301-1307.

Skeffington RA (1976). Chromium uptake and transport in barley seedlings (Hordeum vulgare L.). Planta 132(3): 209-214.

Smith SR (1994). Effect of soil pH on availability to crops of metals in sewage sludge-treated soils. II. Cadmium uptake by crops and implications for human dietary intake. Environ Pollut 86(1): 5-13.

Sridhara Chary N, Kamala CT and Samuel Suman Raj D (2008). Assessing risk of heavy metals from consuming food grown on sewage irrigated soils and food chain transfer. Ecotoxicol Environ Saf 69(3): 513-524.

Srikanth R and Reddy SRP (1991). Lead, cadmium and chromium levels in vegetables grown in urban sewage sludge--Hyderabad, India. Food Chem 40(2): 229-234.

Srivastava M, Juneja A, Dass S, Srivastava R, Srivastava S, Srivastava S, Mishra S, Singh V and Prakash S (1994). Studies on uptake of trivalent and hexavalent chromium in onion (Allium cepa). Chem Speciat Bioavail 6: 27-30.

Sterrett SB, Chaney RL, Gifford CH and Mielke HW (1996). Influence of fertilizer and sewage sludge compost on yield and heavy metal accumulation by lettuce grown in urban soils. Environ Geochem Health 18(4): 135-142.

Tomov A and Alandjiyski D (2006). Lead and cadmium in the system soil-plant in industrially polluted area. Field experiment. J Environ Protect Ecol 7(2): 313-318.

USDA. (2009). Website for water content of fruits and vegetables. from http://www.nal.usda.gov/fnic/foodcomp/Data/SR21/nutrlist/sr21a255.pdf.

Van Mantgem PJ, Wu L and Banuelos GS (1996). Bioextraction of selenium by forage and selected field legume species in selenium-laden soils under minimal field management conditions. Ecotoxicol Environ Saf 34: 228-238.

Vecera Z, Mikuska P, Zdráhal Z, Docekal B, Buckova M, Tynova Z, Parizek P, Mosna J and Marek J. (1999). Additional comments about trace elements in crop plants. Analysis of plant, soil, water and chemical treatment samples, from http://www.dsa.unipr.it/phytonet/fertilia/partners/vecera3.htm.

Wang G, Su M-Y, Chen Y-H, Lin F-F, Luo D and Gao S-F (2006). Transfer characteristics of cadmium and lead from soil to the edible parts of six vegetable species in southeastern China. Environ Pollut 144(1): 127-135.

Watt B and Merrill A (1975). Composition of Foods, Raw, Processed, Prepared. Agricultural Handbook No. 8. Washington D.C.: Consumer and Food Economics Institute, Agricultural Research Service, U.S. Dept. of Agriculture.

WHO. (1991). Inorganic Mercury. Environmental Health Criteria, Vol 118. World Health Organization. Geneva, Switzerland

Wiersma D (1986). Cadmium, lead, mercury and arsenic concentrations in crops and corresponding soils in the Netherlands. J Agric Food Chem 34(6): 1067-1074.

Yan S, Ling Q and Bao Z (2007). Metals contamination in soils and vegetables in metal smelter contaminated sites in Huangshi, China. Bull Environ Contam Toxicol 79(4): 361-366.

Yang Y, Zhang F-S, Li H-F and Jiang R-F (2009). Accumulation of cadmium in the edible parts of six vegetable species grown in Cd-contaminated soils. J Environ Manage 90(2): 1117-1122.

Zheng N, Wang Q and Zheng D (2007a). Health risk of Hg, Pb, Cd, Zn, and Cu to the inhabitants around Huludao Zinc Plant in China via consumption of vegetables. Sci Total Environ 383(1-3): 81-89.

Zheng N, Wang QC, Zhang XW, Zheng DM, Zhang ZS and Zhang SQ (2007b). Population health risk due to dietary intake of heavy metals in the industrial area of Huludao city, China. Sci Total Environ 387(1-3): 96-104.

Zheng R-L, Li H-F, Jiang R-F and Zhang F-S (2008). Cadmium accumulation in the edible parts of different cultivars of radish, *Raphanus sativus* L., and carrot, *Daucus carota* var. sativa, grown in a Cd-contaminated soil. Bull Environ Contam Toxicol 81(1): 75-79.

Appendix I. Fish Bioaccumulation Factors

I.1 Introduction

The algorithm used in the AB-2588 risk assessment to estimate exposure to contaminants via intake of angler-caught fish contains a chemical-specific variable known as a bioaccumulation factor (BAF). Fish are exposed to chemicals that are deposited into their aqueous environment from airborne sources. Only a small subset of Hot Spots chemicals are wholly or partially in the particulate phase and thus subject to deposition. These chemicals include semivolatile organic chemicals and toxic metals and semi-metals. Table I-1 presents the chemical-specific BAF values derived by OEHHA for the Hot Spots program. This appendix outlines the methods used for estimating BAFs and summarizes the available literature used for deriving the chemical-specific BAFs recommended in Table I-1.

Table I-1. Recommended Default Fish BAFs for Edible (Muscle) Tissue[a]

Organic Chemicals[b]	
Diethylhexylphthalate (DEHP)	40
Hexachlorobenzene (HCB)	80,000
Hexachlorocylcohexanes (HCH)	3000
Pentachlorophenol	[c]
Polycyclic aromatic hydrocarbons (PAH)	800
Polychlorinated biphenyls (PCB)	2,000,000
Polychlorinated dibenzo-p-dioxins and furans (PCDD/F)	300,000
Inorganic Metals and Semi-Metals[d]	
Arsenic	20
Beryllium	40
Cadmium	40
Chromium	20
Lead	20
Inorganic mercury	80
Nickel	20
Selenium	1000

[a] All BAFs were rounded to the nearest whole number.
[b] Lipid-normalized to adult rainbow trout with 4% lipid content in muscle tissue, and based on the freely dissolved fraction of organic chemical in water under conditions of average POC and DOC in U.S. lakes and other water bodies.
[c] To be assessed for bioaccumulation in fish
[d] Based on wet weight muscle tissue concentration, and on the total water concentration of the metal or semi-metal in water.

Accumulation of a chemical in fish is a physical-chemical process by which chemicals tend to apportion themselves between the fish and the fish's contact with its environment. The environment in this case is defined broadly to include the water, food that the fish eats, and contact with materials other than water. Accumulation of

chemicals in fish may result in human exposure from fish consumption, which may be significant relative to other exposure pathways considered in the Hot Spots Program.

The Hot Spots program previously only considered the physical-chemical transfer of chemicals from the water column to the fish. This approach does not address other potentially important sources of toxic contaminant contributions to fish and can thus underestimate human exposure for some chemicals. This issue is discussed in more detail below.

The BAF reflects the uptake and retention of a chemical by fish from all surrounding media (e.g., water, food, sediment) when a steady-state concentration has been reached between the fish and the media. The BAF will vary depending on the organ or tissue of interest, but is also often expressed as the chemical accumulation in the whole fish. The BAF is defined under the Hot Spots program as representing the ratio of a concentration of a chemical in edible tissue, specifically the whole muscle tissue or muscle lipid fraction, to its concentration in the surrounding water in situations where the organism and its food are exposed and the ratio does not change substantially over time. The BAF is calculated as:

$$BAF = C_t / C_w \qquad \textbf{(Eq. I.1)}$$

where:

C_t = concentration of the chemical in wet tissue
C_w = concentration of chemical in water

Lipophilic, organic chemicals tend to concentrate in the lipid fraction of fish and the resulting BAF is often lipid normalized to express the concentration of chemical in lipid (see below). The concentration of a chemical in water is often expressed in milligrams or micrograms of chemical per liter of water (i.e., mg/L or µg/L) and the concentration in tissue is often expressed in µg of chemical per kg tissue (µg/kg, or ppb). The BAF can be represented as a unitless factor through conversion of a volume of water to a mass (1 L water ≈ 1 kg), or simply represented in L/kg.

In some instances, the BAF may be based on a bioconcentration factor (BCF). The BCF is defined as representing the ratio of a concentration of a chemical in tissue to its concentration in the surrounding water only when a steady-state concentration has been reached between the two media. Potential fish exposure via food sources is not included. Laboratory accumulation studies often determine BCFs due to the simplicity of the test and easier comparison with other BCF studies. Currently, U.S. EPA (2003a) recommends use of BCFs only for exposure to inorganic metals, presumably because intake of inorganic metals by fish via food sources is minor compared to uptake from water. However, a review of the literature by OEHHA suggests contaminated food sources can also be an important source of metal accumulation in fish tissues. Thus, reliance on BCFs to estimate fish exposure may also underestimate the actual accumulation of a metal in fish.

For semi- or non-volatile organic chemicals that are highly persistent and hydrophobic (generally with a log K_{ow}>4), the magnitude of bioaccumulation by fish via food sources can be substantially greater than the magnitude of bioaccumulation via exposure to water. For such chemicals, only true BAFs adequately assess accumulation of the chemical in fish tissues. For many of these persistent organic chemicals, biomagnification can occur. Biomagnification is the process through which chemical concentrations in fish increase as the chemical moves up the food chain, essentially through food sources. This process occurs because there are fewer organisms feeding off of more organisms at each level in the food chain, thus concentrating the chemical contaminants.

Numerous variables can affect uptake of persistent organic chemicals and inorganic metals in fish, therefore literature sources that reflected potential chemical accumulation as might occur under the “Hot Spots” program were our primary focus. That is, BCF/BAFs were primarily based on the edible portion (i.e., muscle tissue) of freshwater sport fish common to California lentic environments. Lentic environments consist mainly of standing water bodies including lakes, reservoirs and ponds. Sport fish that are caught and consumed in California are predominantly in trophic levels 3 and 4. These fish are typically of highest economic value and include predatory and carnivorous fish that feed on lower trophic level animals. BAF values for trophic level 2 organisms (e.g., zooplankton and larval fish stages) and non-sport fish, such as mosquito fish and the fathead minnow, were not considered unless there was a lack of accumulation data for higher trophic level sport fish.

The muscle tissue is defined here as the edible tissue of fish, although some ethnic groups may also eat various organs of fish. OEHHA’s California fish advisories recommend against eating the liver and other organs of fish, because they may have higher concentrations of organic contaminants than the muscle tissue (OEHHA, 2003). In addition, most inorganic metals will also concentrate in the organs, particularly the kidney and liver. Thus, the BAFs derived in this document cannot be used for estimating accumulation of chemicals in organs other than muscle tissue, as doing so could seriously underestimate the dose received by consuming fish organs and tissues other than muscle.

In California, common freshwater sport fish caught for consumption include various species of trout, catfish, bass, perch, sunfish and carp (CDFG, 2007). Mean muscle lipid content and trophic level data for some sport-fish are shown in Table I-2. In general, the size of the sport fish should be representative of the size being consumed by the target human population. Thus, the mean values are based on fish sizes that are caught and consumed by anglers. As Table I-2 shows, both muscle lipid content and trophic level can increase with increasing length (and age) of the fish. In some instances, lipid content or trophic level based on fish length, in cm, is provided.

Table I-2. Percent Muscle Lipid Content and/or Mean Trophic Level for some Freshwater Sport-Fish Found in California

Common Name	Mean % Muscle Lipid	Mean Trophic Level
Carp (*Cypinus carpio*)	4.45	3 (10-23 cm)[a] 2.4 (>23 cm)
Catfish		
Black bullhead	1.12	3
Brown bullhead	2.79	3
Channel catfish	5.00	3.1 (5-30 cm) 2.8-4 (36-54 cm)
White catfish	2.15	
Yellow catfish	0.75	
Blue catfish		3
Flathead catfish		3.8
Perch		
Yellow perch	0.66	3.4
Trout		
Rainbow trout	4.00	3 (<30 cm) 3.6 (30-50 cm) 4 (>50 cm)
Brook trout	1.51	3.2
Brown trout	3.81	
Cutthroat trout	1.23	3 (<40 cm) 3.2 (>40 cm)
Lake trout	10.90	3.7 (20-30 cm) 3.9 (30-40 cm) 4.2 (>40 cm)
Bass		
Smallmouth	1.1	
Largemouth	1.03 (35-48 cm) 3.1 (54 cm)	
Black crappie	0.57 (14-23 cm)	

Sources: U.S. EPA (1998); OEHHA (1999); SFBRWQCB (2005); Morrison et al. (1997)
[a] Length of fish shown in parentheses

I.1.1 Uptake and Accumulation of Semi- or Non-Volatile Organic Chemicals in Fish Tissues

Much of the field data for BAFs of organic chemicals comes from studies in the Great Lakes region (Eisenreich et al., 1981). The large surface area of the lakes, long hydraulic residence times, and major pollution sources near and upwind of the lakes have a significant impact on airborne deposited trace organic inputs.

For lipophilic, bioaccumulative organic chemicals, U.S. EPA (1998) recommends calculating a BAF based on the concentration of freely dissolved chemical in the

ambient water and the lipid-normalized concentration in tissue. Regarding lipid normalization, the BAF of lipophilic organic chemicals is usually directly proportional to the percent lipid content in the tissue of interest (U.S. EPA, 1998). For example, a fish with four percent lipid content would accumulate twice the amount of a chemical as a fish with two percent lipid content, all else being equal. Normalizing BAFs or BCFs to lipid content allows comparison between different fish species on the basis of factors other than percent lipid content. The lipid-normalized concentration is expressed as:

$$C = C_t / f \qquad \textbf{(Eq. I.2)}$$

where:

C_t = Concentration of chemical in wet tissue (either whole fish or specified tissue)
f = Fraction lipid content in the organism

The lipid fraction of the edible muscle tissue is generally estimated because this is where the lipophilic chemicals will reside. However, the lipid content of muscle tissue can vary considerably among freshwater sport fish species (see Table I-1) as well as among the same species of different sizes and in different habitats. For this document, the rainbow trout lipid muscle content (4%) is used as the basis for point estimate BAFs for lipophilic organic chemicals. The rainbow trout is a common freshwater sport fish species caught and consumed in California and represents a reasonable “average’ lipid content value among California sport fish. However, muscle lipid content can increase well above 10% in some fish species (carp, lake trout, and certain catfish) as they reach maximum size and age. The BAFs determined in this document may underestimate chemical intake if proportionally high consumption rates of such fish occur.

The tendency of an organic compound to bioconcentrate has been shown to be related to its lipophilicity and inversely related to the chemical’s water solubility. However, correlations between bioconcentration and physical properties are poor for very large molecules of high molecular weight and for chemicals metabolized by fish (Oliver and Niimi, 1985). Large molecules (about 300 to 500 MW) appear to be less efficiently transferred from water and food to fish tissues, but can have very long half lives in lentic/lotic environments (U.S. EPA, 2003a). Comparison of laboratory and field bioaccumulation studies in fish show that use of laboratory BCFs (kinetic and steady state studies), in which water was the only media for bioconcentration, would severely underestimate the field residue levels of large organic molecules in fish, particularly if they are poor substrates for metabolic enzymes. This is a clear indication that water is not the primary route of fish exposure for these chemicals; consumption of contaminated food is likely the major chemical source.

U.S. EPA (1998) derived some BAFs from field measured biota-sediment accumulation factors (BSAFs) for very hydrophobic, organic compounds such as PCDD/Fs. The BSAF is the ratio of the lipid-normalized concentration of a chemical in tissue to its organic carbon-normalized concentration in surface sediment. Water concentrations of highly hydrophobic compounds can be difficult to measure accurately for field-measured BAFs, so U.S. EPA (2003a) recommends the BSAF as the only field-based method that can be used to estimate the concentration of certain organic compounds in ambient

water. The California "Hot Spots" PCDD/F BAF point estimates discussed below in Section I.3.1.6 were derived from field-measured BSAF data by U.S. EPA (1998).

U.S. EPA (1998) recommends that for organic chemicals with a log K_{ow} greater than four, the concentrations of particulate organic carbon (POC) and dissolved organic carbon (DOC) in the ambient water should be either measured or reliably estimated. For these chemicals, the concentration of the chemical that is dissolved in ambient water excludes the portion sorbed onto particulate or dissolved organic carbon. The freely dissolved concentration is considered to represent the most bioavailable form of an organic chemical in water and, thus, is the form that best predicts bioaccumulation. The freely dissolved concentration is calculated as:

$$C^{fdw} = (f_{fd}) \times (C^{tw}) \qquad \textbf{(Eq. I.3)}$$

Where:

C^{fdw} = freely dissolved concentration of the organic chemical in ambient water

f_{fd} = faction of the total chemical in ambient water that is freely dissolved

C^{tw} = total concentration of the organic chemical in ambient water

If F_{fd} is not known, it may be calculated using the equation:

$$F_{fd} = \frac{1}{1 + POC \times K_{ow} + DOC \times 0.08 \times K_{ow}} \qquad \textbf{(Eq. I.4)}$$

For the California BAFs, DOC and POC were sometimes based on U.S. EPA (2003a) national default estimates of 2.9 mg/L for DOC and 0.5 mg/L for POC. These values reflect the central tendency estimated for DOC and POC for lakes and reservoirs distributed throughout the United States.

Field-based estimates of the freely dissolved concentration of an organic chemical in water (C^{fdw}) are preferred in order to predict BAF point estimates. However, Eq. I.4 was used to estimate f_{fd} in a number of instances when sufficient data were lacking in studies used to estimate a BAF.

I.1.2 Uptake and Accumulation of Inorganic Metals in Fish Tissues

In aquatic systems the availability of a metal to fish depends on many physico-chemical as well as biological factors. As summarized by Dallinger et al. (1987), availability is influenced by the chemical speciation of the ionic forms. The chemistry of the water including factors such as pH, hardness, and the presence of organic compounds and suspended particles may change the activity of free metal ions and influence the speciation of heavy metals. Binding to, and release from the sediment also affects the availability of metals to fish. Among the biological factors affecting metal availability, species-specific differences like feeding behavior and habitat preferences play a dominant role. These basic features are modified by physiological factors, such as accumulation rates and the binding capacity in various fish species. The three ways by

which inorganic metals may enter fish include body surface, the gills, and the alimentary tract. However, fish seem to be able to homeostatically regulate some heavy metals that they are exposed to. Thus, BCFs and BAFs for metals will generally be smaller compared to BCFs and BAFs for persistent bioaccumulative organic chemicals.

In general, soluble metal fractions may accumulate preferentially via the gills, and particulate metal fractions via the alimentary tract (Dallinger et al., 1987). Unlike persistent, hydrophobic organic chemicals, bioconcentration and biotransferance factors of metals tend to decrease with increasing trophic level up to fish, although the organometal methylmercury is an exception. However, even if biomagnification is not observed, or bioconcentration factors are small, the amount of metal transferred via food or water can be high enough to reach levels that are harmful to humans. This is because under chronic exposure of a water system, very high metal levels may occur in sediments, macrophytes and benthic animals in relation to the water levels. Thus, ingestion of sediment and sediment-dwelling invertebrates by bottom-dwelling fish species may be an important route of metal uptake by these fishes.

The wet weight muscle tissue concentrations of metals are used for determination of the BAF values. If the reference data are expressed only as a dry weight muscle tissue concentration, the tissue concentration was adjusted to a wet weight concentration using a factor of 0.24 (i.e., water content of fish muscle is roughly 75-76% by weight) if specific conversion data are not presented in the reference to calculate the adjustment.

An inverse relationship between metal accumulation and weight/size of the fish has been observed; metal in tissues decreases with increasing size or weight of fish (Liao et al., 2003). This effect has been attributed to growth dilution, increased metabolic rate in juvenile fish and increased ability to depurinate the metals as the fish matures. As a result, metal uptake studies in fingerlings or juvenile fish may overestimate bioaccumulation of mature sport fish caught and consumed by anglers and were usually not used in this document to derive accumulation factors.

Another factor to take into account is exposure duration. Numerous accumulation studies summarized below have observed long exposure times, on the order of months, before steady-state levels of a metal are reached in fish tissues. Thus, short-term exposure studies may underestimate bioaccumulation of a metal in fish.

Based on the bioaccumulation literature for metals of interest in the “Hot Spots” program, some general statements can be made. Waterborne exposure to an inorganic metal will result in greatest metal accumulation in gill, kidney and liver. Metals in the diet will increase levels in the gut as well. Muscle tissue will have the lowest accumulation of the metals. Basing BAFs on whole body concentrations of a metal may overestimate metal intake, as the concentration of an inorganic metal can be quite high in the viscera (e.g., kidney and liver), with organ-specific BAFs of 1000 or greater. Where sufficient data were present, laboratory-measured BCFs were lower for a metal than those derived using data from field studies. BCF studies often did not account for intake via contaminated food, which in some studies summarized below was shown to be an important route of exposure for inorganic metals. Also, many of the laboratory

BCF studies likely did not attain steady-state concentrations because exposures were too short.

In almost all instances, acidic water bodies (generally with a pH of 6.5 or lower) will increase accumulation of the cationic metals and oxy-anionic chromium in fish organs and tissues compared to pH neutral (7.0 to 7.5) water bodies. The default BAFs in this document are primarily based on pH neutral lentic water bodies, as these are the most common in California. Consequently, the default BAFs may underestimate the actual accumulation of a metal in fish if the water body is acidic.

I.2 Derivation of Fish BAFs

I.2.1 Semi- or Non-Volatile Organic Chemicals

I.2.1.1 Diethylhexylphthalate (DEHP)

DEHP has been detected in marine and lake sediments, as well as in marine and freshwater sport fish (Stalling et al., 1973; McFall et al., 1985; Camanzo et al., 1987; Mackintosh et al., 2004). However, the source of the DEHP found in these marine and lake sediments is not likely to be solely from air emissions. The very high K_{ow} of 7.73 and model calculations suggest that DEHP could readily bioaccumulate in fish and that dietary uptake would be an important route of exposure (Staples et al., 1997; Gobas et al., 2003). However, bioaccumulation and biomagnification studies of DEHP in fish show roughly three orders of magnitude lower BCFs/BAFs than predicted based on the K_{ow} of DEHP. This finding is a result of trophic dilution and lack of biomagnification through the aquatic food web, primarily due to the metabolic transformation of DEHP in fish (Staples et al., 1997; Mackintosh et al., 2004). The term trophic dilution means that the BAF tends to decrease as the trophic level increases.

The only freshwater study from which a field-measured BAF was developed was based on a Dutch study investigating the occurrence of DEHP in the freshwater and fish throughout the Netherlands (Peijnenburg and Struijs, 2006). Twenty-five samples of bream and roach fish and 66 freshwater samples from 23 sites were collected throughout the country. Based on the geometric mean DEHP concentration of 1.8 µg/kg wet fish and the dissolved freshwater DEHP concentration of 0.33 µg/L, a BAF of 5.5 is calculated (Table I.3). We corrected for the lipid fraction in the whole fish samples (median: 0.5% lipid), generating a lipid-normalized DEHP BAF of 1.1×10^3. Finally, we also corrected for the muscle lipid content of rainbow trout (4%), which is approximately eight times greater than that of the bream and roach fish, generating a BAF of 44.

An assumption used for this BAF is that the influence of collecting fish and water samples at different times and from different locations on this BCF is not large. Another factor to consider is that the fish in the Dutch study were collected from both lentic and lotic water bodies. Lentic environments are characterized by still (not flowing) water, as in lakes and reservoirs. But the lotic environments are characterized by flowing water, as in streams and rivers.

Gobas et al. (2003) and Mackintosh et al. (2004) conducted a saltwater field study to assess the food-web bioaccumulation of a range of phthalate esters including DEHP. The calculated lipid-normalized BAF for the staghorn sculpin, a forage fish, and the dogfish, a predatory species, were 16,000 and 580, respectively (Table I.3). The larger dogfish (3 kg BW) has a smaller BAF than the sculpin (0.1 kg BW) due to gill elimination and fecal egestion rates dropping with increasing organism size and becoming negligible compared to growth rates.

Table I.3. BAF Values for DEHP in Fish

Fish Species	Total BAF[a]	BAF(fd)[b]	BAF(rt)[c]
Staghorn Sculpin	ND[d]	16,000	640
Spiny Dogfish	ND	580	23
Bream & Roach	5.5	1091	44

[a] Total concentration in whole fish divided by the total concentration of chemical in water
[b] Freely dissolved, lipid-normalized concentration
[c] BAF(rt) for sport-sized rainbow trout (rt) based on muscle lipid content of 4%
[d] No data

Supporting studies from other laboratories report BCFs in small sport and non-sport fish. Whole-fish BCFs of 17 and 30 were estimated in separate studies in small rainbow trout (Mehrle and Mayer, 1976; Tarr et al., 1990). Mayer (1976) estimated a BCF of 594 in fathead minnows, and Karara and Hayton (1984) estimated a BCF of 637 in sheepshead minnows. The estimated BCF values are based on the parent compound (i.e., they did not estimate a total BCF including DEHP and its metabolites) and did not include data that appeared to suffer from water solubility problems or lack of steady state attainment.

Basing the bioaccumulation of DEHP on BCF values does not take into account accumulation of DEHP from food or sediment sources, which may result in an underestimation of the BAF. In addition, basing a BAF on fingerlings or small fish may overestimate BAFs for sport-sized fish. Until field-based bioaccumulation studies for specific lentic water bodies are published for DEHP, we recommend that the BAF of 44, based on the Dutch freshwater field study, be used in the “Hot Spots” program as the default point estimate for DEHP accumulation in sport fish.

I.2.1.2 Hexachlorobenzene

HCB in the atmosphere is predicted to be predominantly in the vapor phase (see Appendix E). HCB concentrations in the vapor phase averaged 96.6% (range: 92-100%) of the total HCB concentration in air samples over Ontario, Canada (Lane et al., 1992). This finding would suggest that airborne deposition of HCB into water bodies would be small enough to disregard. However, due to the extreme persistence of HCB in air, water and soil, accumulation of HCB into water bodies by both dry and wet deposition can be significant (Eisenreich et al., 1981; Kelly et al., 1991). Field studies at Lake Superior, a relatively pristine water body in which organics deposit primarily from

atmospheric sources, report HCB in water, sediment and fish tissue samples (Eisenreich et al., 1981).

Niimi and Oliver (1989) determined the percent lipid content and HCB concentration in muscle tissue of four salmonid species (brown, lake, and rainbow trout and coho salmon) collected from Lake Ontario. Based on the published water concentration of HCB in Lake Ontario, the researchers calculate a total BAF of 101,333. The total BAF was lipid-normalized based on 4% muscle lipid content in the fish, and adjusted for the concentration of freely dissolved HCB in water, assuming a DOC content of 0.25 mg/L in Lake Ontario from Gobas (1993). The resulting BAF(fd) is 2.6×10^6.

We did not adjust the BAF(fd) to the muscle lipid fraction of rainbow trout (0.04) used in the California "Hot Spots" program because it is the same as the fish investigated by Niimi and Oliver (1989). We calculated the freely dissolved HCB fraction in water (0.78) from Eq. H.4 using the national default DOC and POC content of lakes and reservoirs (U.S. EPA, 2003a). A final BAF point estimate of 81,120 ($2.6 \times 10^6 \times 0.04 \times 0.78$) is recommended for California fish.

U.S. EPA (1998) calculates a similar BAF(fd) of log 6.40 (2.5×10^6) using Lake Ontario whole fish HCB data from Oliver and Niimi (1988). This BAF(fd) is similar to that estimated by Niimi and Oliver (1989) using only the muscle HCB concentration (BAF(fd) = 2.6×10^6) of the fish presented. U.S. EPA (1998) also calculated a mean log BAF(fd) of 5.70 (5.0×10^5) derived from BSAF data for HCB. Pereria et al. (1988) and Burkhard et al. (1997) determined a similar log BAF(fd) in the range of 6.03 to 6.68 for bioaccumulation of HCB in small, mostly non-sport fish in estuarine environments.

I.2.1.3 Hexachlorocylcohexanes

Technical grade hexachlorocyclohexane (HCH) generally consists of five isomers, including α-, β-, γ-, δ-, and ε-HCH. α-HCH is the most common isomer in technical grade HCH, and γ-HCH, also known as lindane, is most often isolated and used for its insecticidal action. Consequently, most environmental fate and bioaccumulation studies have investigated the α- and γ-isomers.

Lindane is a relatively small MW compound with a short half-life in fish, so rapid equilibrium occurs between the chemical concentration in fish and the water (Oliver and Niimi, 1985). The short half-life is probably a result of its log $K_{ow} < 4$. The high chlorine content of HCHs prevents metabolism of the isomers by rainbow trout (Konwick et al., 2006). The half-life of lindane in sport-sized fish (11-13 days) is longer than in juvenile fish (about 4 days). However, Geyer et al. (1997) report that α-HCH has a longer half-life of 14.8 days in juvenile rainbow trout. In addition, they observed a positive correlation for fish lipid content and the BCF for lindane.

The major factor governing residue levels for HCHs appears to be the chemical concentration in the water (Oliver and Niimi, 1985). Thus, good agreement between field BAFs and laboratory BCFs in rainbow trout is achieved. For lindane, the whole-fish laboratory BCF was 1200 and the whole-fish field BAF in Lake Ontario fish was 1000.

For α-HCH, the whole-fish laboratory BCF was 1600 and the whole-fish BAF in Lake Ontario fish was 700.

In a subsequent comprehensive investigation at Lake Ontario, Oliver and Niimi (1988) report total BAFs for α-HCH and lindane of 5357 and 9333, respectively. The lipid-normalized whole fish BAFs shown in Table I.4 were based on a weighted average lipid content of 11% for the four fish species examined (i.e., brown, lake, and rainbow trout, coho salmon).

Normalizing the BAFs to represent the freely dissolved fraction in water based on the national default DOC and POC values for lakes and reservoirs had little effect on the freely dissolved fraction of the HCHs, as chemicals with log Kow < 4 (the lindane and α-HCH log K_{ow}s are 3.67 and 3.78, respectively) will not partition significantly to OC. Normalizing the muscle concentration of the HCHs based on the muscle lipid content of rainbow trout (4%) results in point estimate BAFs of 3394 for lindane, and 1948 for α-HCH.

Table I.4. BAF Values Based on Lake Ontario Salmonids

HCH Isomer	Total BAF[a]	BAF(fd)[b]	BAF(rt)[c]
Lindane (γ-HCH)	9333	84,845	3394
α-HCH	5357	48,700	1948

[a] Total concentration in whole fish divided by the total concentration of chemical in water
[b] Freely dissolved, lipid-normalized concentration based on 11% lipid content in whole fish
[c] BAF point estimates based on muscle lipid content of 4% for sport-sized rainbow trout

Niimi and Oliver (1989) determined the percent lipid content and HCH concentrations in muscle tissue, rather than only whole fish (apparently from the same fish examined in their previous study). The HCH concentrations in muscle adjusted for an average muscle lipid content of 4% for rainbow trout are 5.7 and 1.4 µg/kg for α-HCH and lindane, respectively. Using the water concentrations of 2.8 and 0.3 ng/L for α-HCH and lindane, respectively, from Oliver and Niimi (1988) provides BAFs of 2036 (α-HCH) and 4667 (lindane).

Because the muscle HCH concentration data in Niimi and Oliver (1989) was at or below the limit of detection for some fish, particularly for lindane, the California BAF point estimate is based on the Oliver and Niimi (1988) data presented in Table I.4. We recommend a BAF(rt) point estimate of 2671 for the “Hot Spots” program, which is the arithmetic average of the muscle tissue BAF(rt)s for the two major HCH isomers in Table I.4.

I.2.1.4 Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are compounds with two or more fused benzene rings and often contain alkyl side groups. In water and sediment, low molecular weight PAHs (i.e., containing two or three aromatic rings) are more easily degraded by microbes, whereas the high molecular weight PAHs (i.e., containing four or more aromatic rings), including benzo[a]pyrene (BaP), tend to persist (Meador et al., 1995).

Bioaccumulation of PAHs in fish has not been rigorously studied, in part because PAHs undergo liver metabolism in fish resulting in low to non-detectable concentrations of the parent PAHs in fish tissues (Meador et al., 1995). Bioaccumulation of PAHs tends to decline with increasing K_{ow}, probably due to low gut assimilation efficiency and increased metabolism. However, low molecular weight PAHs tend to be less persistent in fish than the high molecular weight PAHs, probably due to more ready diffusion in and out of lipid pools.

BaP has been shown to be extensively metabolized in fish. In small bluegill sunfish (4 to 12 g wet weight) exposed to ^{14}C-labelled BaP in water, only 5% of the radiolabel in whole fish samples at the end of 24 hr exposure was found to be the parent compound (McCarthy and Jimenez, 1985). In their risk assessment, Boyce and Garry (2003) estimated a whole fish BCF of 14 for BaP based on the average value reported from relevant laboratory bioaccumulation studies in the literature.

Using the assumption that a typical lipid fraction of whole fish is 0.05 (Staples et al., 1997), and a muscle/whole body lipid ratio of 0.20 for adult rainbow trout (Niimi and Oliver, 1983), we calculated the lipid-normalized muscle tissue BCF as 56 for BaP. Adequate data for the DOC and POC water concentrations were not supplied by the studies used to derive the BCF, so the influence of this factor on the BAF could not be accounted for in the final estimate.

Burkhard and Lukasewycz (2000) determined field-measured BAFs for several PAHs found in water, sediment and lake trout muscle lipid of Lake Superior. The total BAF and BAF(fd) in Table I.5 were calculated by the researchers for lake trout in Lake Superior. The BAF(rt) was calculated by OEHHA for PAHs in rainbow trout (4% muscle lipid content) using default DOC + POC content for U.S. lakes and reservoirs. The relative order of metabolism was obtained by dividing the BAF of the chemical by its corresponding K_{ow}. By increasing rate of metabolism in the fish, the relative order was pyrene, benz[*a*]anthracene, chrysene/triphenylene, fluoranthrene, and phenanthrene. Thus, metabolism of the parent PAH compound appears to primarily control accumulation in the muscle tissue.

Table I.5. BAF Values for Polycyclic Aromatic Hydrocarbons

PAH congener (# of rings)[a]	PEF[b]	Total BAF[c]	BAF(fd)[d]	BAF(rt)[e]
Phenanthrene (3)	ND[f]	18	89	4
Fluoranthrene (4)	ND	331	1660	62
Pyrene (4)	ND	10,471	52,481	2067
Benz[*a*]anthracene (4)	0.1	9550	53,703	1573
Chrysene/triphenylene (4)	0.01 (chrysene only)	759	4074	124[g]

[a] Number of benzene rings per PAH compound shown in parentheses
[b] Potency Equivalency Factor for carcinogenicity, using benzo[a]pyrene as the index PAH compound with a PEF=1.
[c] Total concentration in fillet of lake trout divided by the total concentration of chemical in water
[d] Freely dissolved, lipid-normalized concentration based on 20.5% lipid content in fish fillet samples
[e] BAF point estimates based on muscle lipid content of 4% for rainbow trout and default DOC + POC content for U.S. lakes and reservoirs from U.S. EPA (2003a).
[f] Not determined, as a result of inadequate or no evidence for carcinogenicity in animals.
[g] Assumed to represent BAF(rt) for both chrysene and triphenylene

The data in Table I.5 suggest that PAHs with four rings are more likely to accumulate in fish than PAHs with three rings. A study by Zabik et al. (1996) found some five- and six-ring PAHs in muscle fat of lake trout from Lake Superior. This study did not detect BaP in the fish tissue, but did find dibenzo[ah]pyrene which has a potency equivalency factor (PEF) value of 10. BAFs could not be calculated for any PAHs with five or more rings, either because dissolved levels of these congeners could not be detected in the water, or because the congener could not be detected in the fish (Baker and Eisenreich, 1989; 1990; Zabik et al., 1996). Another reason is that the individual PAHs quantified in water and fish were not all the same between various studies.

We calculated an average BAF(rt) of 849 from the congener groups in Table I.5 that have PEFs (i.e., benz[*a*]anthracene and chrysene), and is recommended as the default point estimate of BAF(rt) for PAHs. Considering that measurable levels of high molecular weight carcinogenic PAHs have been detected in fish muscle (although not enough data are present to estimate BAFs), but that a BAF for BaP is likely below the BAF(rt) of 849, a point estimate based on the most bioaccumulative carcinogenic PAHs should be sufficiently health protective to avoid underestimation of a BAF for the carcinogenic PAHs.

I.2.1.5 Polychlorinated biphenyls (PCBs)

PCBs are a group (209 congeners) of organic chemicals, based on various substitutions of chlorine atoms on a basic biphenyl molecule. However, probably less than 100 congeners are found at concentrations of significance in commercial PCB mixtures and environmental samples, and fewer represent a toxicological concern (Niimi, 1996). Solubilities and octanol-water partition coefficients (K_{ow}) for PCB congeners range over several orders of magnitude. The K_{ow}s, which are often used as estimators of the potential for bioconcentration, are highest for the most chlorinated PCB congeners.

Since log K_{ow} values of most PCB congeners are higher than 5, biomagnifications through trophic transfer is the primary mechanism governing the accumulation of these compounds in fish (Oliver and Niimi, 1985; van der Oost et al., 2003). Thomann and Connolly (1984) demonstrated that more than 99% of PCBs in Lake Michigan lake trout came from food. A food web bioaccumulation PCB study by Morrison et al. (1997) noted that over 99% of PCB 153 accumulated in fish through consumption of contaminated food and 79.9% of PCB 42 accumulation was through food (PCB 42 has a lower K_{ow}).

Food-web relationships and biomagnification may be more related to the PCBs in sediment rather than water. Therefore, biota sediment accumulation factors (BSAF) have been developed for PCBs as an indicator of bioavailability to fish because sediment is an important source for hydrophobic chemicals such as PCBs (Niimi, 1996). However, the PCBs found in the highest concentrations in fish generally reflected their high concentrations in water and sediment (Oliver and Niimi, 1988).

In the comprehensive field study by Oliver and Niimi (1988), the most common classes of PCB isomers in various salmon and trout species from Lake Ontario were the penta- and hexachlorobiphenyls, making up about 65% of the total isomeric composition. The tetra- and heptachlorobiphenyls made up another 30% of the isomeric composition. Eleven single and co-eluting PCB congeners (153, 101, 84, 138, 110, 118, 180, 87 + 97, 149, 187 + 182, and 105) constituted over half the PCBs in fish. The single most common congener was 153 (2,2', 4,4',5,5'-hexachlorobiphenyl). The tri, tetra, and penta congeners comprised a much higher fraction in water than in the fish. Thus, the PCB accumulation pattern in fish is not an accurate reflection of the aqueous composition of the mixture found in the lake.

Because the calculated total BAFs for the most common PCBs accumulating in fish gave a roughly 10-fold range for the values, a weighted average total BAF was calculated for the four most common chlorinated classes of PCB congeners in fish from the study by Oliver and Niimi (1988). These were the tetra-, penta-, hexa-, and hepta-CBs, which constituted about 95% of the overall PCBs accumulated in whole fish. The resulting weighted-average total BAF was 6.12×10^6.

We calculated a lipid-normalized BAF of 5.56×10^7 based on the whole fish lipid content of 11% determined in the study by Oliver and Niimi (1988). The mean percent contribution of PCB congeners was similar for whole fish and muscle among the

species even though total concentrations vary widely (Niimi and Oliver, 1989). Consistency among congener contribution in whole fish and muscle was also demonstrated by cumulative percent of the more common PCB congeners. The freely dissolved PCB portion in water is based on data by Gobas (1993) who found about half of total PCBs in Lake Ontario water was in the freely dissolved form. The resulting calculated lipid-normalized, freely dissolved BAF, or BAF(fd), is 1.11×10^8.

Next, we adjusted the BAF(fd) to generate a BAF point estimate to be used in the California "Hot Spots" program. Correcting the BAF(fd) for the muscle lipid fraction of 0.04 in rainbow trout, and correcting for the freely dissolved PCB fraction in water (0.25, or 50% of that calculated for Lake Ontario) gives a final BAF point estimate of 2.22×10^6 ($1.11 \times 10^8 \times 0.04 \times 0.50$).

I.2.1.6 Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans (PCDDs and PCDFs)

PCDDs and PCDFs are two groups of toxic compounds composed of 135 and 75 individual isomers, respectively. Most studies have focused on the 17 congeners with lateral Cl substitutions at the 2,3,7,8 positions (Niimi, 1996). These congeners appear to be primarily responsible for the accumulation and toxicity of PCDD/Fs. The 2,3,7,8-TCDD, 1,2,3,7,8-PCDD, 2,3,7,8-PCDF and 2,3,4,7,8-PCDF congeners were common in four fish species (brown trout, lake trout, rainbow trout, coho salmon) examined from Lake Ontario. Dietary uptake of PCDD/Fs appears to be of more importance than waterborne uptake, although dietary absorption efficiencies in fish are consistently lower and more variable compared to PCBs.

The two main lateral substituted PCDDs, 2,3,7,8-TCDD and 1,2,3,7,8-PCDD, constituted about 89% of the sum of all PCDDs in the fish (Niimi, 1996). The two main PCDFs, 2,3,7,8-PCDF and 2,3,4,7,8-PCDF, constituted 51% of the sum of all PCDFs in the fish. Since these congeners are the most bioaccumulative and have the greatest toxicity concern, the PCDD/F BAFs will be representative of these four congeners.

U.S. EPA (1998) derived lipid-normalized, freely dissolved BAFs (i.e., BAF(fd)) from field measured BSAFs. The high hydrophobic nature of PCDD/Fs makes it difficult to accurately determine field-measured BAFs (i.e., based on water concentrations) for this group of chemicals. U.S. EPA (2003a) recommends the BSAF as the only field-based method that can be used to estimate the concentration of these compounds in ambient water. Using a weighted-average approach for the main congeners found in fish, the BAF(fd)s were 1.00×10^7 and 5.50×10^6 for PCDDs and PCDFs, respectively.

We then adjusted the BAF(fd)s to generate BAF point estimates to reflect the muscle lipid fraction of rainbow trout (0.04) for the "Hot Spots" program. The final BAF point estimates of 400,000 and 220,000 were calculated for PCDDs and PCDFs, respectively, for California fish. The average BAF of these two values, 310,000, is the recommended BAF point estimate for the "Hot Spots" program.

I.2.2 Derivation of Fish BCFs – Inorganic Metal and Semi-Metal Chemicals

I.2.2.1 Arsenic

Inorganic arsenic (As), either as As(III) or As(V), are the predominant forms in aquatic ecosystems such as sediment and water, but organoarsenic compounds may be present at significant levels in freshwater fish. Average concentrations of As in ambient freshwater are generally <1 to 10 µg/L (U.S. EPA, 2003b). U.S. EPA (2003b) states that recent research shows each of the major inorganic and organic As species, including As(III), As (V), arsenobetaine (AsB), dimethylarsenic acid (DMA), and monomethylarsonic acid (MMA), may exhibit different toxicities, and it may be important to take into account the fraction of total As present in the inorganic and organic forms when estimating the potential risk posed through consumption of As-contaminated fish. Ideally, the most appropriate BAFs would incorporate the most bioavailable and toxic form(s). This is currently not possible, so the point estimate BAF in this document will be based on total As in sport fish muscle tissue.

Direct accumulation of As in tilapia was proportional to the concentration of arsenicals in water (Suhendrayatna et al., 2002). Approximately 25% of absorbed arsenic from water in whole fish as either As(III) or As(V) was transformed to methylated arsenic, primarily methyl-, dimethyl-, and trimethyl- forms. Whether absorbed as As(III) or As(V) from water, metabolism in fish resulted in roughly equivalent concentrations of both inorganic arsenic species in whole fish, although As(III) was absorbed more easily than As(V).

Accumulation and transformation of As in the food chain has been investigated. In a three-step freshwater food chain (algae-shrimp-tilapia), exposure to As(III) in water resulted in total As concentrations decreasing in the organisms with each step up the food chain (Suhendrayatna et al., 2002). Inorganic As species were the predominant forms in each organism (As(III), 9-41%; As(V), 50-90%), with only a limited degree of As methylation at each step in the food chain. However, when As(V) was the dominant As species in water, mouthbreeder fish raised long-term in aquaculture ponds contained predominantly organoarsenic species in muscle tissue, with inorganic As equaling only 7.4% of total As (Huang et al., 2003).

Predicted and measured As concentrations in major organs of tilapia from culture ponds high in As observed highest As concentrations in the alimentary canal, blood and liver, and lowest concentrations in muscle tissue (Liao et al., 2005). Steady-state concentration of As in muscle tissue took up to 300 days to be achieved.

Arsenic bioaccumulation studies in fish have been conducted in laboratory, aquaculture pond, and field investigations, although exposure durations to achieve steady-state concentrations in fish tissues were only observed for the aquaculture and field studies. The BAFs findings are presented in Table I.6.

In aquaculture studies, an average BCF of 8.2 (range: 5.4 to 11) was determined for bioconcentration of As in muscle of mouthbreeder fish raised long-term in ponds from three different regions in Taiwan (Huang et al., 2003). The fish were collected from

ponds containing 14.4 to 75.8 µg/L As in water. A BCF of 3.5 was recorded for As in muscle tissue of large-scale mullet raised in a Taiwanese aquaculture pond (Lin et al., 2001). In farmed tilapia fish exposed to As in water for 300 days, a muscle BCF = 4 was calculated (Liao et al., 2005). In a similar study, BCFs of 15 and 53 were obtained for As from tilapia muscle raised in two aquaculture ponds containing 49.0 and 17.8 µg/L As in water, respectively (Liao et al., 2003). Because the fish in these aquaculture studies were fed with artificial bait that did not contain As, the accumulation factors may better represent BCF values rather than BAF values.

Only two field studies were located that presented data to determine a muscle tissue BAF for fish in As-contaminated lentic water bodies. A BAF of 28 was determined from muscle tissue of the common carp exposed to As in four wastewater treatment basins in Pennsylvania (Skinner, 1985). Channel catfish and large-mouth bass from a reservoir impacted by mining and agricultural runoff had muscle BAF values of 12.5 for As (Baker and King, 1994).

Table I.6. BAFs for Arsenic in Muscle Tissue of Fish from Lentic Water Bodies

Location	Species	Arsenic Water Concentration	Arsenic Muscle Concentration	BAF	Reference
Taiwanese Aquaculture Studies					
Putai Pond	mouthbreeder	75.8 µg/L	0.41 µg/g	5.4	Huang et. al., 2003
Yichu Pond	mouthbreeder	15.1	0.12	7.9	Huang et. al., 2003
Hsuehchia Pond	mouthbreeder	14.4	0.16	11.1	Huang et. al., 2003
Putai Pond	large-scale mullet	169.7	2.41	14.2	Lin et. al., 2001
Hsuehchia Pond	tilapia	17.8	0.95	53.4	Liao et. al., 2003
Yichu Pond	tilapia	49.0	0.75	15.3	Liao et. al., 2003
Tilapia farms	tilapia	94	1.5	16	Liao et al., 2005
Field Studies					
San Carlos Reservoir, AZ	large-mouth bass	8	0.1	12.5	Baker & King, 1994
San Carlos Reservoir, AZ	channel catfish	8	0.1	12.5	Baker & King, 1994
Wastewater treatment basins, PA	common carp	3.0 – 16.0	0.22 - <0.05	28	Skinner, 1985

Among the studies presented in Table I.6, average BCF/BAFs were calculated for six fish species: 8.1 for mouthbreeder, 14.2 for large-scale mullet, 28 for tilapia, 12.5 for

large-mouth bass and channel catfish, and 28 for common carp. The arithmetic average BAF combined for all species is 17, which we recommend as the BAF point estimate for As.

I.2.2.2 Beryllium

Little information could be found for bioaccumulation of beryllium in fish. U.S. EPA (1980) estimated a BCF of 19 in whole bluegill after 28 days of exposure in water. It is unknown if steady state levels were attained in the fish, although the whole-body elimination half-life was observed to be one day. Limited data by Eisler (1974) suggest that whole-fish accumulation of inorganic beryllium in mummichogs from seawater is similar to some other cationic metals such as cadmium, in that whole fish uptake of beryllium appears to be a passive process.

No information could be found regarding the accumulation of beryllium in muscle tissue of fish. Based on BCF and BAF studies of other cationic metals discussed in this appendix, steady state levels were probably not reached in bluegills during the 28-day exposure (U.S. EPA, 1980). The muscle BAFs for other cationic metals (i.e., cadmium, inorganic mercury, lead, nickel) presented in Table H.2 range from 20 to 80. We recommend that a mean cationic metal BAF of 40 be used for beryllium in sport fish until more comprehensive bioaccumulation studies are conducted.

I.2.2.3 Cadmium

A considerable number of cadmium (Cd) bioaccumulation studies have been carried out in fish. Freshwater sport fish accumulate Cd mainly in gills, kidney, liver, and gastrointestinal tract (Sangalang and Freeman, 1979; Harrison and Klaverkamp, 1989; Spry and Wiener, 1991; Szebedinszky et al., 2001). However, Cd does not accumulate as appreciably in muscle tissue of exposed sport fish and the concentration is generally low relative to other tissues and organs.

The Cd concentration in fish varies with the proportion of free divalent Cd in water, typically increasing with increasing water concentration (Camusso et al., 1995). Direct uptake across the gills has been generally considered the primary influx of the metal for fish in dilute waters (Spry and Wiener, 1991). However, absorption of Cd from contaminated food sources can be a significant route of exposure, and may be the dominant source of Cd in bodies of water with high pH and calcium levels (Ferard et al., 1983; Harrison and Klaverkamp, 1989; Farag et al., 1994; Kraal et al., 1995; Thomann et al., 1997).

The main characteristics of lakes that enhance bioaccumulation of Cd in fish include low pH (pH ≤6), low aqueous calcium (often <2 mg/L), and low DOC (usually <3 mg/L) (Spry and Wiener, 1991). In the eastern U.S., whole-body Cd levels in bluegill fish from low pH lakes were as much as 10-fold higher compared to cadmium in bluegills from circumneutral-pH lakes. In addition, accumulation of Cd in fish is more sensitive to changes in water hardness, usually expressed in mg/L $CaCO_3$, rather than changes in DOC (Wiener and Giesy, 1979).

Steady-state equilibrium of Cd in muscle and other tissues was obtained in brook trout at about 20 weeks exposure in a three-generation exposure study by Benoit et al. (1976). Benoit et al. (1976) also recorded a muscle BCF = 3.5 in brook trout exposed to aqueous Cd in Lake Superoir water for 70 weeks. Equilibrium of Cd in tissues was also reached at 20 weeks of exposure.

Perhaps significantly, the numerous laboratory studies that measured muscle Cd content show an inverse relationship with water hardness. In several laboratory studies, BCFs varied between 1.6 to 4.8 for Cd in muscle of rainbow trout, carp and brook trout with a water hardness between 33 and 93 mg /L $CaCO_3$ (Benoit et al., 1976; Giles, 1988; Harrison and Klaverkamp, 1989; de Conto Cinier et al., 1997). Exposure durations for these studies ranged from 3 to 17 months, and tissue and organ Cd concentrations increased with increasing exposure duration. Two other laboratory studies that recorded somewhat higher BCFs of 17-19 in muscle of rainbow and brook trout also had the lowest water hardness (19-22 mg /L $CaCO_3$) (Sangalang and Freeman, 1979; Kumada et al., 1980). The exposure duration of fish to Cd-contaminated water for both of these studies was about 3 months. Alternatively, laboratory studies exposing rainbow trout to Cd in water with considerably higher hardness (140-320 mg/L $CaCO_3$) at circumneutral-to-high pH (7.4-8.2) for up to 80 weeks recorded BCFs from 0 to 2 in muscle tissue (Roberts et al., 1979; Calamari et al., 1982; Brown et al., 1994; Szebedinszky et al., 2001).

The level of DOC in the water of the laboratory BCF studies above were not discussed, but were likely low. Low DOC levels would allow water hardness to be the main factor affecting bioaccumulation of Cd.

Although comparatively few field studies have been published that investigated Cd accumulation in muscle tissue of sport fish, the field study by Wiener and Giesy (1979) supports the assumption that water hardness (and perhaps pH) is a more important factor in controlling tissue accumulation then the DOC content. In this study, a Cd muscle BAF = 12 was determined in bluegill stocked in an acidic (pH = 4.6), highly organic pond for 511 days. Measured total organic carbon of the pond was anywhere from 15 to >30 mg/L, but the $CaCO_3$ content of the pond was very low, averaging 2.1 mg/L.

Two field studies examined the effect of acidified water in New York lakes on fish tissue levels of various heavy metals as a result of acid deposition (i.e., acid rain) (Heit et al., 1989; Stripp et al., 1990). In general, higher BAFs were recorded for Cd in muscle tissue of yellow perch and white sucker from the most acidic lentic water body, Darts Lake, compared to two other lakes, Rondaxe and Moss lakes, with higher pH values (Table I.7). All three lakes were clear-water lakes with comparable concentrations of DOC.

Table I.7. BAFs for Cadmium in Muscle Tissue of Fish from U.S. Lakes

Location	Species	Lake pH	Cd Water Concentration (µg/L)	Cd Muscle Concentration (µg/g)	BAF
Darts Lake (1)	White sucker	4.9-5.4	0.7	0.062	89
Darts Lake (1)	Yellow perch	4.9-5.4	0.7	0.048	69
Darts Lake (2)	White sucker	5.1-5.4	0.26	0.038	146
Darts Lake (2)	Yellow perch	5.1-5.4	0.26	0.028	108
Rondaxe Lake (1)	White sucker	5.8-6.7	1.1	0.024	22
Rondaxe Lake (1)	Yellow perch	5.8-6.7	1.1	0.024	22
Rondaxe Lake (2)	White sucker	5.8-6.7	0.61	0.025	41
Rondaxe Lake (2)	Yellow perch	5.8-6.7	0.61	0.038	62
Moss Lake (1)	White sucker	6.5-6.8	0.6	0.022	36
Moss Lake (1)	Yellow perch	6.5-6.8	0.6	0.034	56
Skinface Pond, SC (3)	Bluegill	4.6	0.17	0.0021	12

Sources: (1) Stripp et al., (1990); (2) Heit et al., (1989); (3) Wiener and Giesy (1979).

The few field studies examining muscle tissue levels of Cd in contaminated lakes indicate that basing a BAF on laboratory BCF studies would underestimate the accumulation potential of Cd in fish. However, it is probably not appropriate basing a BAF on data from highly acidified lakes (i.e., Darts Lake and Skinface Pond), as California generally does not have the lake acidification problem that exists in the northeastern U.S. Thus, we recommend default BAF point estimate for Cd of 40 based on fish from the variable pH (Rondaxe Lake) and circumneutral lakes (Moss Lake), which is the arithmetic average BAF combining both fish species (white sucker and yellow perch, which represent trophic level 3 and 4 fish, respectively) from these lakes.

I.2.2.4 Chromium

Hexavalent chromium (Cr(VI)) in water readily penetrates the gill membrane of fish and is the main route of uptake (Holdway, 1988). Organs and tissues that accumulate Cr(VI) include gills, spleen, kidney, gall bladder, gastrointestinal tract, opercular bone, and brain. Accumulation in muscle tissue is minor compared to these other tissues. No biomagnifications occur at higher trophic levels. Cr(VI) uptake is a passive process with resulting tissue concentrations directly proportional to exposure concentrations. Chromium bioavailability to fish increases with decreasing pH (7.8 to 6.5), resulting in increased bioaccumulation in tissues and organs (Van der Putte et al., 1981).

In a laboratory study, six-month exposure of rainbow trout to Cr(VI) as potassium dichromate ($K_2Cr_2O_7$) in water resulted in a muscle tissue BCF of 3 (Calamari et al., 1982).

A small freshwater aquatic ecosystem containing adult catfish was created in a small tank, and a single dose of potassium dichromate was added to the system (Ramoliya et al., 2007). After 21 days of exposure, a muscle tissue BCF <1 was calculated for the

catfish based on the average water concentration of Cr(VI) over the 21 days. However, the Cr(VI) content in the catfish had not reached equilibrium at the end of exposure, and was still increasing with increasing exposure duration. High levels of Cr(VI) in the intestine of the catfish suggest Cr(VI) may be absorbed via food sources.

Rainbow trout that were reared for two years in either a hatchery or river water that was contaminated with low levels of sodium dichromate had muscle tissue BCFs of 40 and 12, respectively (Buhler et al., 1977). Exposing the same fish to high concentrations of Cr(VI) (2.5 mg/L) for 22 days increased muscle levels of Cr(VI), but the resulting BCF was only 0.1-0.2.

Two field studies from South Africa determined the bioaccumulation of chromium in muscle tissue of fish. In adult African sharptooth catfish, muscle tissue BAFs of 10 and 16 were calculated for fish kept in a treated sewage maturation pond and in a reservoir, respectively, for 12 months (Van den Heever and Frey, 1996). Nussey et al. (2000) calculated an average muscle tissue BAF of 23.6 in the moggel, a cyprinid fish, collected from a different reservoir over a period of 15 months.

Based on the long-term field exposure studies, an average muscle BAF of 26 was calculated for rainbow trout in the Buhler et al. study, and an average muscle BAF of 13 was calculated for the African sharptooth catfish in the van den Heever and Frey study. Combined with the muscle tissue BAF of 23.6 in the moggel from Nussey et al. (2000), we calculate an arithmetic mean BAF of 21 and recommend this value as the BAF point estimate for Cr.

I.2.2.5 Lead

Similar to Cd, factors that may increase accumulation of cationic metals such as lead in fish include low pH (6.0-6.5 or less) in the water body, low concentrations of aqueous calcium that compete with lead for absorption through the gills, and low DOC (Varanasi and Gmur, 1978; Spry and Wiener, 1991; Lithner et al., 1995). Pb appears to have a greater tendency than Cd to associate with DOC and particulate matter in lake water, with accumulation in fish varying inversely with the concentration of dissolved organics in water (Wiener and Giesy, 1979). When Merlini and Pozzi (1977a) added a Pb salt to lake water, only 8% remained in the ionic form with the remainder presumably associating with dissolved organics.

Accumulation of Pb by fish typically increases with increasing exposure concentration in water, although Pb does not biomagnify in aquatic food chains (Spry and Wiener, 1991). Pb chiefly accumulates in the bone, scales, gill, kidney, and liver. Pb does not accumulate as appreciably in skeletal muscle tissue of fish. Primary mode of absorption has been suggested to be direct uptake of Pb in the ionic state across the gills, with lead from food sources being minor or insignificant (Merlini and Pozzi, 1977a; Spry and Wiener, 1991; Farag et al., 1994). On the other hand, another laboratory study found that lead uptake in fish via food was significant, if not more important than uptake via water (Vighi, 1981).

In a three-generation laboratory study, a BCF of 2 to 3 was estimated for Pb in muscle tissue of first and second generation brook trout (Holcombe et al., 1976). Exposure to Pb in water was for 38 and 70 weeks in first and second generation fish, respectively. The concentration of Pb in muscle had reached equilibrium at about 20 weeks of exposure.

Whole bluegill Pb concentrations have been shown to be as much as 10 times higher in bluegills from low-pH lakes (pH≤6.0) compared to bluegills from circumneutral-pH lakes (pH 6.7-7.5) (Spry and Wiener, 1991). In another study, whole-fish Pb levels in sunfish increased almost three-fold when lake water pH was decreased from 7.5 to 6.0 (Merlini and Pozzi, 1977b).

In other field studies, Pb accumulated to greater extent in muscle of white suckers and yellow perch from an acidic lake compared to more neutral lakes (Heit et al., 1989; Stripp et al., 1990) (Table I.8). With increasing lake acidity, muscle bioaccumulation of Pb became increasingly higher in bottom-dwelling, omnivorous white suckers compared to carnivorous yellow perch. Thus, contact with sediments by bottom-dwelling fish increases Pb bioaccumulation.

A considerably greater concentration of Pb was found in surface sediments (880-1005 µg/g) of the lakes compared to the water (2.0-3.0 ng/g) (Stripp et al., 1990). It was postulated that higher levels in fish tissues from acidic lakes result from increased mobilization of the cationic Pb species from sediments coupled with an increase in the cationic Pb species in the acidic water.

The field data indicate higher muscle BAFs in fish from highly acidified lakes (Table I.8). California generally does not have the acidification problem that exists in the northeastern U.S. Thus, a BAF point estimate for Pb was based on fish from the variable pH and circumneutral lakes. The BAF data from Nussey et al. (2000) was also included, although water pH data were not provided in the report. We calculate an arithmetic average BAF of 19 combining all fish species (white sucker, yellow perch and moggel) from these lakes and recommend this value as the Pb BAF point estimate.

Table I.8. BAFs for Lead in Muscle Tissue of Fish from Lentic Ecosystems

Location	Species	Lake pH	Pb Water Concentration	Pb Muscle Concentration	BAF
Acidic water bodies					
Darts Lake (1)	White sucker	4.9-5.4	3.0 µg/L	0.13 µg/g	43
Darts Lake (1)	Yellow perch	4.9-5.4	3.0 µg/L	0.058	19
Darts Lake (2)	White sucker	4.9-5.4	1.5	0.13	87
Darts Lake (2)	Yellow perch	4.9-5.4	1.5	0.055	37
Acidic lakes & ponds, NJ (3)	Yellow perch	3.7-4.6	0.8 – 3.6	0.067 – 0.11	40
Variable and circumneutral water bodies					
Rondaxe Lake (1)	White sucker	5.8-6.7	2.0	0.048	24
Rondaxe Lake (1)	Yellow perch	5.8-6.7	2.0	0.058	29
Rondaxe Lake (2)	White sucker	5.8-6.7	2.3	0.050	22
Rondaxe Lake (2)	Yellow perch	5.8-6.7	2.3	0.050	22
Moss Lake (1)	White sucker	6.5-6.8	2.5	0.031	12
Moss Lake (1)	Yellow perch	6.5-6.8	2.5	0.024	10
Witbank Dam, South Africa (4)	Moggel	ND*	140	2.00	14

Sources: (1) Stripp et al. (1990), (2) Heit et al. (1989), (3) Sprenger et. al. (1988), (4) Nussey et al. (2000)
* No data

I.2.2.6 Mercury (inorganic) and Methylmercury

Mercury, like other metals deposited into water, can occur in a number of physical and chemical forms. Physically, mercury can be freely dissolved or bound to organic matter or particles suspended in water. Mercury can be found as elemental mercury (Hg^0), inorganic ionic mercury (primarily Hg^{++}), or organic mercury (e.g., methylmercury (MeHg) or dimethylmercury).

Mercury (Hg) enters aquatic ecosystems primarily as inorganic Hg, but MeHg is the dominant form of Hg found in muscle tissue of freshwater fish (Spry and Wiener, 1991). MeHg has been shown to constitute virtually all, about 99% or greater, of the total Hg in muscle of trophic level 3-4 freshwater sport fish even though much of the Hg analyzed in the water was in inorganic Hg (Bloom, 1992; Kuwabara et al., 2007). In whole fish, the proportion of inorganic Hg is greater (5% or more of total Hg) because whole body samples include visceral tissue, such as kidney and liver, which is the principal site of inorganic Hg accumulation in fish (Hill et al., 1996; Watras et al., 1998). BAFs discussed for MeHg in this document are for informational purposes only and are not specific to the Hot Spots program. Mercury compounds emitted by facilities are almost exclusively in the elemental or inorganic form, so MeHg is not directly applicable to the Hot Spots program.

As summarized by Southworth et al. (2004), MeHg is produced in aquatic environments by the action of microorganisms on inorganic Hg. It can also be removed from the

aquatic systems by microorganisms that demethylate MeHg. Once formed, MeHg is taken up by microorganisms, primary producers, aquatic invertebrates, and fish. MeHg in the organisms shows the classical biomagnification process, with MeHg concentration increasing with trophic level. The concentrations of MeHg that are accumulated in fish are greatly affected by the nature of the aquatic food chain, and are sensitive to factors such as aquatic community composition and productivity. In many waters, minute concentrations (<10 ng/L) of waterborne inorganic Hg are capable of sustaining MeHg production at rates high enough to support bioaccumulation of MeHg in fish to levels warranting fish consumption advisories. The concentrations of MeHg and inorganic Hg are positively related in natural waters, which would appear to support expressing a BAF for MeHg in fish as a ratio based on total or dissolved inorganic Hg in water. Calculating MeHg bioaccumulation in fish using such a ratio (i.e., estimate the concentration of dissolved MeHg in water based in the total Hg concentration deposited in water), introduces another level of uncertainty compared to development of BAFs directly from published reports.

Using the dissolved MeHg fraction in water to derive BAFs is recommended, as this is the primary form of MeHg that is bioaccumulated in fish. MeHg is also more toxic than other forms of mercury. However, dissolved MeHg was not always the form measured in the studies U.S. EPA (2001) identified for inclusion in their database. Thus, translators were necessary to convert between other forms of Hg measured in water and dissolved MeHg for BAF calculations. For lentic systems (i.e., lakes, reservoirs and ponds), the translators that may be used in the Hot Spots program include dissolved MeHg ($MeHg_d$) over the total Hg (Hg_t) and the $MeHg_d$ over the total MeHg ($MeHg_t$). The lentic U.S. EPA translators are $MeHg_d$/ Hg_t = 0.032 and $MeHg_d$/ $MeHg_t$ = 0.61.

U.S. EPA (2001) derived the mean dissolved MeHg/total Hg translator of 3.2% for lentic ecosystems, and used it to convert between other forms of Hg measured in water and dissolved MeHg for BAF calculations. Thus it can be interpreted that 3.2% of inorganic Hg that has deposited into a lake will be converted by microorganisms and found in the form of dissolved MeHg.

Table I.9 presents various BAFs for methylmercury from U.S. EPA (2001) and California data (OEHHA, 2006). Although U.S. EPA presents the geometric means of BAFs, OEHHA recommends the use of arithmetic means of the BAFs to provide a more health protective estimate. In developing their BAFs, U.S. EPA assumed that 100 percent of the mercury measured as total mercury in both trophic levels 3 and 4 was MeHg. This assumption provides a more health protective estimate.

Table I.9. Methylmercury BAFs for Lentic/Lotic[a] Ecosystems from U.S. EPA and California Data

Agency	Environment/Comments	Mean	Trophic Level	
			3	4
U.S. EPA	Lentic Only	Geometric	1.1×10^6	5.7×10^6
U.S. EPA	Lentic Only	Arithmetic	1.5×10^6	6.2×10^6
California	Lentic Alternative	Geometric	NP	NP
California	Lentic Alternative	Arithmetic	NP	NP
U.S. EPA	Lotic Only	Geometric	5.7×10^5	1.2×10^6
U.S. EPA	Lotic Only	Arithmetic	1.3×10^6	3.9×10^6
California	Lotic Alternative	Geometric	6.8×10^5	1.1×10^6
California	Lotic Alternative	Arithmetic	1.4×10^6	3.5×10^6
U.S. EPA	Lentic/Lotic Combined	Arithmetic	1.4×10^6	5.0×10^6

[a] Lentic environments are characterized by still (not flowing) water, as in lakes and reservoirs. Lotic environments are characterized by flowing water, as in streams and rivers.

In California, using a MeHg BAF developed by U.S. EPA is complicated by the large number of Hg point sources originating from legacy mining activities, a situation somewhat unique to California. Atmospheric deposition of Hg into water bodies may be overshadowed by the existing Hg already present due to legacy mining. In addition, very little published data exist for California lentic ecosystems in order to determine if total Hg concentrations are good predictors of MeHg concentration. The BAFs and translators developed by U.S. EPA were based primarily on atmospheric deposition of Hg into water bodies. Hg speciation in water and fish may be quite different depending on whether the Hg originated from mining or atmospheric deposition.

Nevertheless, OEHHA (2006) found that the national values predicted California fish MeHg concentrations very well except for some water bodies where Hg concentrations in water were statistically higher. Hg concentrations (≥0.2 ng/L) in these water bodies were found to be more than one standard derivation from the mean for other data used in these tests. We concluded that the national default values for BAFs and translators may not work well for all water bodies in California. However, based on the limited comparisons possible, BAFs and translators based on the California data and international studies (U.S. EPA database) were found to be similar. Thus, a MeHg BAF = 6,200,000 (log 6.79) from Table I.9 for sport fish caught and consumed from lentic ecosystems, and a translator of 3.2% to convert total Hg deposited in water to dissolved MeHg in water may be relevant MeHg variates to use in California.

In partial support, Kelly et al. (1995) observed that total Hg concentration was not a good predictor of MeHg concentration in stream water or in lakes in general, but it appeared to be a good predictor for lakes within individual geographic areas. In lotic ecosystems, Southworth et al. (2004) concluded that it is not valid to assume that the fraction of total waterborne Hg comprised by MeHg would remain constant while total Hg varies at high total Hg concentrations (roughly >50 ng/L) typical of systems affected by point-source or legacy contamination. However, at total Hg concentrations less than 10 ng/L, the %MeHg varies little. They postulated that such a relationship results from

saturation of the ecosystems capacity to methylate inorganic Hg at high total Hg concentrations.

Inorganic Hg is absorbed by fish less efficiently then MeHg from both food and water, but if absorbed, is eliminated more rapidly. For example, rainbow trout fed inorganic Hg-contaminated prey resulted in Hg predominantly accumulating in the intestines, and the Hg was not significantly absorbed into the body (Boudou and Ribeyre, 1985). During the decontamination phase, Hg that had accumulated in the intestines was rapidly excreted.

In water, the most important route for uptake of inorganic Hg in fish is likely the gills, with accumulation of Hg mainly in the gills, kidney and liver (Allen et al., 1988; Gottofrey and Tjalve, 1991). Whole-body accumulation of inorganic Hg in rainbow trout and carp increases with decreasing water pH from 9 to 5, but did not reach equilibrium during a 17-day exposure in water (Wakabayashi et al., 1987).

MeHg is the primary concern for estimating Hg bioaccumulation. Since relatively little of the Hg in fish muscle is in the inorganic form, there are very little field data to estimate a BAF for inorganic Hg.

In a laboratory tank study investigating the relationship between inorganic Hg body burden levels and toxicity, a mean muscle BCF of 84 was calculated in rainbow trout exposed to HgCl in water for 60 to 130 days (Niimi and Kissoon, 1994). Steady-state levels in muscle tissue were reached by 60 days of exposure to high levels of HgCl (64 µg/L); these levels were eventually lethal to the fish. Since most lakes of concern contain inorganic Hg levels in the ng/L to low µg/L range, such high exposure conditions may not reflect an ideal situation for estimating an inorganic Hg BAF. In addition, it has been found that food sources containing inorganic Hg are also important for fish Hg bioaccumulation (Hill et al., 1996).

U.S. EPA (2001) has used a national criteria of 51 ng/L of total Hg in water as a measure that may result in the MeHg concentration of concern of 0.3 µg/g in fish. Using the assumption that, at most, 1% of the MeHg concentration in fish muscle is actually inorganic Hg, a BAF of 59 for inorganic Hg is calculated (0.3 µg/g (0.01) ÷ 51 ng/L). Although this BAF derivation is a rather crude estimate of the inorganic Hg BAF, the value is near that calculated from the BCF study (BCF = 84) by Niimi and Kissoon (1994). OEHHA recommends using the inorganic Hg BAF point estimate = 84 (rounded to 8×10^1) derived from the Niimi and Kissoon study.

I.2.2.7 Nickel

In aquarium tank studies, brown trout exposed to water containing radioactive nickel (^{63}Ni) showed the greatest accumulation of the metal in the gills, kidneys and liver, with relatively low accumulation in muscle tissue (Tjalve et al., 1988). The Ni concentration in muscle was related to the water concentration of Ni (Van Hoof and Nauwelaers, 1984). Similar to other cationic metals, increasing the acidity of water increases accumulation of Ni in fish.

A muscle BCF of 1.5 was recorded in the brown trout following 3 week exposure to Ni in a water tank. However, equilibrium of Ni between water and fish tissues had not been attained. Rainbow trout exposed to Ni in hard water (hardness = 320 mg $CaCO_3$/L) for six months accumulated little or no Ni in muscle tissue (BCF = 0.8-1.1) (Calamari et al., 1982).

In a field study, Nussey et al. (2000) calculated an average muscle tissue BAF of 19 in the moggel, a cyprinid fish, collected from a reservoir containing various heavy metals, including Ni, over a period of 15 months. Average muscle BAFs of 4 and 39 were calculated in common carp collected from two different wastewater treatment basins in Pennsylvania (Skinner, 1985). The acidity of the treatment basin water was not discussed, so it is unknown if water acidity played a role for the variation in BAF values.

In laboratory studies, accumulation of Ni in fish muscle tissues is relatively low compared to other inorganic metals discussed in this document. There are also relatively few published reports investigating fish bioaccumulation of Ni. Based on the BAFs from the two field studies by Nussey et al. (2000) and Skinner (1985), we calculated an arithmetic mean average BAF of 21 and recommend this value as a point estimate BAF for Ni.

I.2.2.8 Selenium

Selenium (Se) occurs in the environment in several oxidation states with different physicochemical and biological properties (Besser et al., 1993). Se from both natural and anthropogenic sources enters surface waters primarily as the highly soluble Se(IV) and Se(VI) oxidation states, which form selenite, SeO_3^{2-}, and selenate, SeO_4^{2-}, respectively. Organic selenides, Se(-II), including Se-amino acids and Se-proteins, methyl selenides, and other Se-substituted analogs of organosulfur compounds, are produced by biological reduction of selenite and usually occur at lower concentrations in water than inorganic Se species. Little information is available for organic selenides, so the BAF is based on total Se.

Se is an essential micronutrient for most aquatic organisms but is also toxic at relatively low environmental concentrations. It is reported that Se concentrations in fish muscle rarely exceed 1 ppm (wet weight) in the absence of exposure to Se from geologic sources or from industrial wastes (Cumbie and Van Horn, 1979).

Four-month exposure of juvenile bluegill and largemouth bass to selenite (Na_2SeO_3) in water resulted in BCF values of 288 and 153, respectively, and was independent of

water temperature and hardness (Lemly, 1982). Accumulation of Se in muscle was relatively slow, reaching a steady-state concentration after 90 days of exposure in both fish species. Accumulation of Se in fish skeletal muscle was presumed to be a result of the high affinity of Se for sulfhydryl groups found on many organic molecules in muscle tissue. However, bioconcentration in muscle was quite low compared to BCF values for other organs and tissues. Lemly (1982) observed higher bioconcentration of Se in the spleen, heart, liver, kidney, gill, and erythrocytes.

In a food-chain study (algae-daphnids-bluegill), whole bluegill fry accumulated greater Se concentrations from food than from water in selenite-based exposures, and aqueous and food-chain Se bioaccumulation were approximately additive (Besser et al., 1993). However, in both aqueous and food-chain exposures based on selenite and selenate, Se bioaccumulation was greatest in algae and least in bluegills. Se concentrations in whole bluegill fry did not differ significantly between selenite and selenate treatments in either aqueous or food-chain exposures. Inorganic Se BCF values ranged from 13 to 106 in whole blue gill fry with 30- to 40-day exposures, although a steady-state concentration was not attained.

In a field study, Cumbie and van Horn (1979) analyzed muscle Se levels in various species of fish, primarily bluegill, other sunfish, carp and bullhead, during spring and summer from a reservoir with a high Se concentration. The range of muscle BAFs among all fish was 632 to 5450 with an arithmetic average of about 1780. Further research at the same reservoir observed muscle BAFs in warmwater sportfish (primarily various species of perch, catfish, sunfish and crappie) ranging from 739 to 2019 with an arithmetic average of 1351 (Lemly, 1985). There was evidence of biomagnification of Se through the food-chain, although when considering only muscle tissue of fish, levels of Se appeared to be similar to that of mulluscs, insects, annelids and crustaceans found at the reservoir.

Lower Se BAFs of 124 and 216 were calculated in muscle of white suckers and yellow perch, respectively, from an acidic lake in New York (Stripp et al., 1990). Based upon geochemistry, Se would be expected to be less soluble in acidic lakes. BAFs of 454 and 490 were determined for Se in muscle tissue of crappie and carp, respectively, collected from a wastewater treatment basin in Pennsylvania (Skinner, 1985).

The accumulation data indicate Se uptake from both food and water results in accumulation of Se in muscle tissue, and that BAF/BCF values can be quite variable even between different fish species within the same water body. The two related field studies investigating Se accumulation in fish from a North Carolina reservoir (Cumbie and Van Horn, 1979; Lemly, 1985) gave an average BAF of 1566 (1351 + 1780 / 2) combining all trophic level 3 and 4 fish. Not including the data from the acidic lake, we calculate an arithmetic mean BAF of 1019 when the average BAF from the North Carolina reservoir is combined with the average fish BAF from the Pennsylvania wastewater treatment basin from Skinner (1985). In support, the BAF is within the predicted intervals (at water Se concentrations above 0.5 µg/L) of the Se whole fish bioaccumulation model for lentic systems developed by Brix et al. (2005). We recommend a default point estimate BAF of 1000 for selenium for use in the Hot Spots program.

I.3 Non-Bioaccumulated Chemicals

Some organic “Hot Spot” chemicals in which a significant airborne fraction can be found in the particle phase do not appear to be bioaccumulated in fish. For example, although data show that methylenedianiline (MDA) exists partly in the particle phase and is persistent in soils, the low log Kow of 1.59 (HSDB, 2008) and rapid metabolism in higher trophic level animals (ATSDR, 1998) indicate this chemical will likely not bioaccumulate in fish tissues. In addition, unpublished evidence summarized in ATSDR (1998) suggests that MDA does not bioaccumulate in carp. Until published evidence shows otherwise, a fish BAF for MDA will not be included in the fish pathway in the “Hot Spots” program.

In addition, OEHHA is proposing that fluoride should not be included in the fish pathway because fresh weight fluoride concentrations in muscle or the fillet portion of fish were found to be less than the water concentration, regardless of the weight of the fish (Gikunju, 1992; Mwaniki and Gikunju, 1995).

I.4 References

Allen P, Min SY and Keong WM (1988). Acute effects of mercuric chloride on intracellular GSH levels and mercury distribution in the fish Oreochromis aureus. Bull Environ Contam Toxicol 40(2): 178-84.

ATSDR. (1998). *Toxicological Profile for Methylenedianiline*. Agency for Toxic Substances and Disease Registry. Available online at: www.atsdr.cdc.gov/toxprofiles/tp122.html.

Baker DL and King KA. (1994). *Environmental contaminant investigation of water quality, sediment and biota of the Upper Gila River Basin, Arizona. Project No. 22410-1130-90-2-053*. U.S. Fish and Wildlife Service.

Baker JE and Eisenreich SJ (1989). PCBs and PAHs as tracers of particulate dynamics in large lakes. J Great Lakes Res 15(1): 84-103.

Baker JE and Eisenreich SJ (1990). Concentrations and fluxes of polycyclic aromatic hydrocarbons and polychlorinated biphenyls across the air-water interface of Lake Superior (USA and Canada). Environ Sci Technol 24(3): 342-52.

Benoit DA, Leonard EN, Christensen GM and Fiandt JT (1976). Toxic effects of cadmium on three generations of brook trout (Salvelinus fontinalis). Trans Am Fish Soc 105: 550-60.

Besser JM, Canfield TJ and La Point TW (1993). Bioaccumulation of organic and inorganic selenium in a laboratory food chain. Environ Toxicol Chem 12(1): 57-72.

Bloom NS (1992). On the chemical form of mercury in edible fish and marine invertebrate tissue. Can J Fish Aquat Sci 49(5): 1010-7.

Boudou A and Ribeyre F (1985). Experimental study of trophic contamination of Salmo-Gairdneri by 2 mercury compounds: Mercuric chloride and methylmercuric chloride anaylsis at the organism anf organ levels. Water Air Soil Pollut 26(2): 137-48.

Boyce CP and Gary MR (2003). Developing risk-based target concentrations for carcinogenic polycyclic aromatic hydrocarbon compounds assuming human consumption of aquatic biota. J Toxicol Environ Health B 6: 497-520.

Brix KV, Toll JE, Tear LM, DeForest DK and Adams WJ (2005). Setting site-specific water-quality standards by using tissue residue thresholds and bioaccumulation data. Part 2. Calculating site-specific selenium water-quality standards for protecting fish and birds. Environ Toxicol Chem 24(1): 231-7.

Brown V, Shurben D, Miller W and Crane M (1994). Cadmium toxicity to rainbow trout Oncorhynchus mykiss Walbaum and brown trout Salmo trutta L. over extended exposure periods. Ecotoxicol Environ Saf 29(1): 38-46.

Buhler DR, Stokes RM and Caldwell RS (1977). Tissue Accumulation and Enzymatic Effects of Hexavalent Chromium in Rainbow Trout (Salmo gairdneri). J Fish Res Board Can 34(1): 9-18.

Burkhard LP and Lukasewycz MT (2000). Some bioaccumulation factors and biota-sediment accumulation factors for polycyclic aromatic hydrocarbons in lake trout. Environ Toxicol Chem 19: 1427-9.

Burkhard LP, Sheedy BR, McCauley DJ and DeGraeve GM (1997). Bioaccumulation factors for chlorinated benzenes, chlorinated butadienes and hexachloroethane. Environ Toxicol Chem 16(8): 1677-86.

Calamari D, Gaggino GF and Pacchetti G (1982). Toxicokinetics of Low Levels of Cd, Cr, Ni and Their Mixture in Long-Term Treatment on Salmo gairdneri Rich. Chemosphere 11(1): 59-70.

Camanzo J, Rice CP, Jude DJ and Rossmann R (1987). Organic priority pollutants in nearshore fish from 14 Lake Michigan tributaries and embayments, 1983. J Great Lakes Res 13(3): 296-309.

Camusso M, Vigano L and Balestrini R (1995). Bioconcentration of trace metals in rainbow trout: a field study. Ecotoxicol Environ Saf 31(2): 133-41.

CDFG (2007). California Fishing Passport. California Department of Fish and Game. Available online at: www.dfg.ca.gov/fishingpassport/book.asp.

Cumbie PM and Van Horn SL (1979). Selenium accumulation associated with fish reproductive failure. Proc Ann Conf SE Assoc Fish and Wildl Agencies 32: 612-24.

Dallinger R, Prosi F, Segner H and Back H (1987). Contaminated food and uptake of heavy metals by fish: A review and a proposal for further research. Oecologia (Berl) 73(1): 91-8.

de Conto Cinier C, Petit-Ramel M, Faure R and Garin D (1997). Cadmium bioaccumulation in carp (Cyprinus carpio) tissues during long-term high exposure: analysis by inductively coupled plasma-mass spectrometry. Ecotoxicol Environ Saf 38(2): 137-43.

Eisenreich SJ, Looney BB and Thornton JD (1981). Airborne organic contaminants in the Great Lakes ecosystem. Environ Sci Technol 15: 30-38.

Eisler R (1974). Radiocadmium Exchange with Seawater by Fundulus heteroclitus (L.) (Pisces: Cyprinodontidae). J Fish Biol 6: 601-12.

Farag AM, Boese CJ, Woodward DF and Bergman Hl (1994). Physiological changes and tissue metal accumulation in rainbow trout exposed to foodborne and waterborne metals. Environ Toxicol Chem 13(12): 2021-9.

Ferard JF, Jouany JM, Truhaut R and Vasseur P (1983). Accumulation of cadmium in a freshwater food chain experimental model. Ecotoxicol Environ Saf 7(1): 43-52.

Geyer HJ, Scheunert I, Brueggmann R, Langer D, Korte F, Kettrup A, Mansour M, Steinberg CEW, Nyholm N and Muir DCG (1997). Half-lives and bioconcentration of lindane (gamma-HCH) in different fish species and relationship with their lipid content. Chemosphere 35(1-2): 343-51.

Gikunju JK (1992). Fluoride concentration in Tilapia fish (Oreochromis Leucostictus) from Lake Naivasha, Kenya. Fluoride 25(1): 37-43.

Giles MA (1988). Accumulation of cadmium by rainbow trout, Salmo gairdneri, during extended exposure. Can J Fish Aquat Sci 45(1045-53).

Gobas FAPC (1993). A model for predicting the bioaccumulation of hydrophobic organic chemicals in aquatic food-webs: Application to Lake Ontario. Ecol Modell 69(1-2): 1-17.

Gobas FAPC, Mackintosh CE, Webster G, Ikonomou M, Parkerton TF and Robillard K (2003). Bioaccumulation of PEs in aquatic food-webs. In: The Handbook of Environmental Chemistry. CA Staples ed. Springer. Berlin, Germany: 3, part Q: 201-25.

Gottofrey J and Tjalve H (1991). Effect of lipophilic complex formation on the uptake and distribution of mercury and methylmercury in brown trouts (Salmo trutta): Studies with some compounds containing sulphur ligands. Water Air Soil Pollut 56(0): 521-32.

Harrison SE and Klaverkamp JF (1989). Uptake, elimination and tissue distribution of dietary and aqueous cadmium by rainbow trout (Salmo gairdneri Richardson) and lake whitefish. Environ Toxicol Chem 8(1): 87-98.

Heit M, Schofield CL and Driscoll CT (1989). Trace element concentrations in fish from three Adirondack lakes with different pH values. Water Air Soil Pollut 44: 9-30.

Hill WR, Stewart AJ and Napolitano GE (1996). Mercury speciation and bioaccumulation in lotic primary producers and primary consumers. Can J Fish Aquat Sci 53(4): 812-9.

Holcombe GW, Benoit DA, Leonard EN and McKim JM (1976). Long-term effects of lead exposure on three generations of brook trout (Salvelinus fontinalis). J Fish Res Board Can 33: 1731-41.

Holdway DA (1988). The toxicity of chromium to fish. In: Chromium in the Natural and Human Environments. Nriagu J., Niebor E. and eds. Wiley. New York: 369-97.

HSDB (2008). 4,4'-Diaminodiphenylmethane. U.S. National Library of Medicine. Hazardous Substances Data Bank. Available online at: www.toxnet.nlm.nih.gov

Huang YK, Lin KH, Chen HW, Chang CC, Liu CW, Yang MH and Hsueh YM (2003). Arsenic species contents at aquaculture farm and in farmed mouthbreeder

(Oreochromis mossambicus) in blackfoot disease hyperendemic areas. Food Chem Toxicol 41(11): 1491-500.

Karara AH and Hayton WL (1984). Pharmacokinetic model for the uptake and disposition of di-2-ethylhexyl phthalate in sheepshead minnow Cyprinodon variegatus. Aquat Toxicol (Amst) 5(3): 181-96.

Kelly CA, Rudd WM, St. Louis VL and Heyes A (1995). Is total mercury concentration a good predictor of methyl mercury concentration in aquatic systems? Water Air Soil Pollut 80((1-4)): 715-24.

Kelly TJ, Czuczwa JM, Sticksel PR, Sticksel PR, Sverdrup GM, Koval PJ and Hodanbosi RF (1991). Atmospheric and tributary inputs of toxic substances to Lake Erie. J Great Lakes Res 17(4): 504-16.

Konwick BJ, Garrison AW, Black MC, Avants JK and Fisk AT (2006). Bioaccumulation, biotransformation, and metabolite formation of fipronil and chiral legacy pesticides in rainbow trout. Environ Sci Technol 40(9): 2930-6.

Kraal MH, Kraak MH, de Groot CJ and Davids C (1995). Uptake and tissue distribution of dietary and aqueous cadmium by carp (Cyprinus carpio). Ecotoxicol Environ Saf 31(2): 179-83.

Kumada H, Kimura S and Yokote M (1980). Accumulation and biological effects of cadmium in rainbow trout. Bull Jpn Soc Sci Fish 46: 97-103.

Kuwabara JS, Arai Y, Topping BR, Pickering IJ and George GN (2007). Mercury speciation in piscivorous fish from mining-impacted reservoirs. Environ Sci Technol 41(8): 2745-9.

Lane DA, Johnson ND, Hanely MJ, Schroeder WH and Ord DT (1992). Gas-and particle-phase concentrations of alpha-hexachlorocyclohexane, gamma-hexachlorocyclohexane, and hexachlorobenzene in Ontario air. Environ Sci Technol 26(1): 126-33.

Lemly AD (1982). Response of juvenile centrachids to sublethal concentrations of waterborne selenium. I. Uptake, tissue distribution, and retention. Aquat Toxicol 2: 235-52.

Lemly AD (1985). Toxicology of selenium in a freshwater reservoir: implications for environmental hazard evaluation and safety. Ecotoxicol Environ Saf 10(3): 314-38.

Liao CM, Chen BC, Singh S, Lin MC, Liu CW and Han BC (2003). Acute toxicity and bioaccumulation of arsenic in tilapia (Oreochromis mossambicus) from a blackfoot disease area in Taiwan. Environ Toxicol 18(4): 252-9.

Liao CM, Liang HM, Chen BC, Singh S, Tsai JW, Chou YH and Lin WT (2005). Dynamical coupling of PBPK/PD and AUC-based toxicity models for arsenic in tilapia

Oreochromis mossambicus from blackfoot disease area in Taiwan. Environ Pollut 135(2): 221-33.

Lin MC, Liao CM, Liu CW and Singh S (2001). Bioaccumulation of arsenic in aquacultural large-scale mullet Liza macrolepis from blackfoot disease area in taiwan. Bull Environ Contam Toxicol 67(1): 91-7.

Lithner G, Holm K and Borg H (1995). Bioconcentration factors for metals in humic waters at different pH in the Ronnskar Area (N. Sweden). Water Air Soil Pollut 85(2): 785-90.

Mackintosh CE, Maldonado J, Hongwu J, Hoover N, Chong A, Ikonomou MG and Gobas FA (2004). Distribution of phthalate esters in a marine aquatic food web: comparison to polychlorinated biphenyls. Environ Sci Technol 38(7): 2011-20.

Mayer FL (1976). Residue dynamics of di-2-ethylhexyl phthalate in fathead minnows (Pimephales promelas). J Fish Res Board Can 33: 2610-3.

McCarthy JF and Jimenez BD (1985). Reduction in bioavailability to bluegills of polycyclic aromatic hydrocarbons bound to dissolved humic material. Environ Toxicol Pharmacol 4: 511-21.

McFall JA, Antoine SR and DeLeon IR (1985). Organics in the water column of Lake Pontchartrain. Chemosphere 14: 1253-65.

Meador JP, Stein JE, Reichert WL and Varanasi U (1995). Bioaccumulation of polycyclic aromatic hydrocarbons by marine organisms. Rev Environ Contam Toxicol 143: 79-165.

Mehrle PM and Mayer FL (1976). Di-2-ethylhexyl phthalate: Residue dynamics and biological effect in rainbow trout and fathead minnows. Trace Subst Environ Health 10: 519-24.

Merlini M and Pozzi G (1977a). Lead and Freshwater Fishes: Part 2-Ionic Lead Accumulation. Environ Pollut 13(1): 119-26.

Merlini M and Pozzi G (1977b). Lead and Freshwater Fishes: Part I-Lead Accumulation and Water pH. Environ Pollut 12(3): 167-72.

Morrison HA, Gobas FAPC, Lazar R, Whittle DM and Haffner GD (1997). Development and verification of a benthic/pelagic food web bioaccumulation model for PCB congeners in Western Lake Erie. Environ Sci Technol 31(11): 3267-3273.

Mwaniki DL and Gikunju JK (1995). Fluoride concentration in tissues of fish from low fluoride fresh water lakes in Kenya. Discov Innov 7(2): 173-6.

Niimi AJ (1996). Evaluation of PCBs and PCDDs retention by aquatic organisms. Sci Total Environ 192(2): 123-50.

Niimi AJ and Kissoon GP (1994). Evaluation of the critical body burden concept based on inorganic and organic mercury toxicity to rainbow trout (Oncorhynchus mykiss). Arch Environ Contam Toxicol 26(2): 169-178.

Niimi AJ and Oliver BG (1983). Biological half-lives of polychlorinated biphenyl congeners in whole fish and muscle of rainbow trout (Salmo gairdneri). Can J Fish Aquat Sci 40(9): 1388-94.

Niimi AJ and Oliver BG (1989). Distribution of polychlorinated biphenyl congeners and other halocarbons in whole fish and muscle among Lake Ontario salmonids. Environ Sci Technol 23: 83-8.

Nussey G, van Vuren JHJ and du Preez HH (2000). Bioaccumulation of chromium, manganese, nickel, and lead in the tissues of the moggel, Labeo umbratus (Cyrinidae), from Witbank Dam, Mpumanalnga. Water SA 26: 269-84.

OEHHA. (1999). *Prevalence of Selected Target Chemical Contaminants in Sport Fish from Two California Lakes: Public Health Designed Screening Study. Final Project Report*. Pesticide and Environmental Toxicology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.

OEHHA. (2003). *Methylmercury in Sport Fish: Information for Fish Consumers* Pesticide and Environmental Toxicology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Available online at: www.oehha.ca.gov/fish/pdf/HGfacts.pdf.

OEHHA. (2006). *Evaluation of Bioaccumulation Factors and Translators for Methylmercury*. Pesticide and Environmental Toxicology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.

Oliver BG and Niimi AJ (1985). Bioconcentration factors of some halogenated organics for rainbow trout (*Salmo gairdneri*) limitations in their use for prediction of environmental residues Environ Sci Technol 19(9): 842-9.

Oliver BG and Niimi AJ (1988). Trophodynamic analysis of polychlorinated biphenyl congeners and other chlorinated hydrocarbons in the Lake Ontario ecosystem. Environ Sci Technol 22: 388-97.

Peijnenburg WJ and Struijs J (2006). Occurrence of phthalate esters in the environment of The Netherlands. Ecotoxicol Environ Saf 63(2): 204-15.

Pereria WE, Rostad CE, Chiou CT, Brinton TI, Barber LB, Demcheck DK and Demas CR (1988). Contamination of estuarine water, biota and sediment by halogenated organic compounds: A field study. Environ Sci Technol 22: 772-8.

Ramoliya J, Kamdar A and Kundu R (2007). Movement and bioaccumulation of chromium in an artificial freshwater ecosystem. Indian J Exper Biol 45(5): 475-9.

Roberts KS, Cryer A, Kay J, Solbe JF, Wharfe JR and Simpson WR (1979). The effects of exposure to sub-lethal concentrations of cadmium on enzyme activities and accumulation of the metal in tissues and organs of rainbow and brown trout (Salmo gairdneri, Richardson and Salmo trutta Fario L.). Comp Biochem Physiol C 62C(2): 135-40.

Sangalang GB and Freeman HC (1979). Tissue uptake of cadmium in brook trout during chronic sublethal exposure. Arch Environ Contam Toxicol 8(1): 77-84.

SFBRWQCB. (2005). *Edible Fish Tissue Trace Organic Chemistry Data for Reservoirs. Appendix IV*. San Francisco Bay Regional Water Quality Control Board. Available online at: www.waterboards.ca.gov/sanfranciscobay/water_issues/available_documents/swamp.

Skinner WF (1985). Trace element concentrations in wastewater treatment basin-reared fishes: Results of a pilot study. 61st Annual Meeting of the Pennsylvania Academy of Science.Proc PA Acad Sci. 59(2): 155-61. Lancaster, PA, Apr. 21-23, 1985.

Southworth GR, Peterson MJ and Bogle MA (2004). Bioaccumulation factors for mercury in stream fish. Environ Prac 6: 135-43.

Spry DJ and Wiener JG (1991). Metal bioavailability and toxicity to fish in low-alkalinity lakes: A critical review. Environ Pollut 71(2-4): 243-304.

Stalling DL, Hogan JW and Johnson JL (1973). Phthalate ester residues--their metabolism and analysis in fish. Environ Health Perspect 3: 159-73.

Staples CA, Peterson DR, Parkerton TF and Adams WJ (1997). The environmental fate of phthalate esters: A literature review. Chemosphere 35(4): 667-749.

Stripp RA, Heit M, Bogen DC, Bidanset J and Trombetta L (1990). Trace element accumulation in the tissues of fish from lakes with different pH values. Water Air Soil Pollut 51(75-87).

Suhendrayatna OA, Nakajima T and Maeda S (2002). Studies on the accumulation and transformation of arsenic in freshwater organisms II. Accumulation and transformation of arsenic compounds by Tilapia mossambica. Chemosphere 46(2): 325-31.

Szebedinszky C, McGeer JC, McDonald DG and Wood CM (2001). Effects of chronic Cd exposure via the diet or water on internal organ-specific distribution and subsequent gill Cd uptake kinetics in juvenile rainbow trout (Oncorhynchus mykiss). Environ Toxicol Chem 20(3): 597-607.

Tarr BD, Barron MG and Hayton WL (1990). Effect of body size on the uptake and bioconcentration of di-2-ethylhexyl phthalate in rainbow trout. Environ Toxicol Chem 9(8): 989-96.

Thomann RV and Connolly JP (1984). Model of PCB in the Lake Michigan lake trout food chain. Environ Sci Technol 18: 65-71.

Thomann RV, Shkreli F and Harrison S (1997). A pharmacokinetic model of cadmium in rainbow trout. Environ Toxicol Chem 16(11): 2268-74.

Tjalve H, Gottofrey J and Borg K (1988). Bioaccumulation, Distribution and Retention of $^{63}Ni^{2+}$ in the Brown Trout (*Salmo trutta*). Water Res 22(9): 1129-36.

U.S. EPA. (1980). *Ambient Water Quality Criteria for Beryllium. EPA 440 5-80-024.* . United States Environmental Protection Agency. Available online at: www.epa.gov/waterscience/criteria/library/ambientwqc/beryllium80.pdf.

U.S. EPA. (1998). *Ambient Water Quality Criteria Derivation Methodology for the Protection of Human Health - Technical Support Document. Final Draft. EPA/822/B-98/005*. United States Environmental Protection Agency.

U.S. EPA. (2001). *Water Quality Criteria: Notice of Availability of Water Quality Criterion for the Protection of Human Health: Methylmercury*. 66. Federal Register Environmental Documents, United States Environmental Protection Agency.

U.S. EPA. (2003a). *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000). Technical Support Document Volume 2: Development of National Bioaccumulation Factors*. United States Environmental Protection Agency. EPA-822-R-03-030.

U.S. EPA. (2003b). *Techinical Summary of Information Available on the Bioaccumulation of Arsenic in Aquatic Organisms. EPA-822-R-03-032*. United States Environmental Protection Agency.

Van den Heever DJ and Frey BJ (1996). Human health aspects of certain metals in tissue of the African sharptooth catfish, Clarias gariepinus, kept in treated sewage effluent and the Krugersdrift Dam: Chromium and mercury. Water SA 22(1): 73-8.

van der Oost R, Beyer J and Vermeulen NPE (2003). Fish bioaccumulation and biomarkers in environmental risk assessment: a review. Environ Toxicol Pharmacol 13(2): 57-149.

Van der Putte I, Lubbers J and Kolar Z (1981). Effect of pH on uptake, tissue distribution and retention of hexavalent chromium in rainbow trout (Salmo gairdneri). Aquatic Toxicol 1: 3-18.

Van Hoof F and Nauwelaers JP (1984). Distribution of nickel in the roach (Rutilus rutilus L.) after exposure to lethal and sublethal concentrations. Chemosphere 13(9): 1053-8.

Varanasi U and Gmur DJ (1978). Influence of water-borne and dietary calcium on uptake and retention of lead by coho salmon (Oncorhynchus kisutch). Toxicol Appl Pharmacol 46(1): 65-75.

Vighi M (1981). Lead uptake and release in an experimental trophic chain. Ecotoxicol Environ Saf 5(2): 177-93.

Wakabayashi M, Kikuchi M, Oh Y-K, Yoshida T, Kojima H and Saito H (1987). Bioconcentration of $^{203}HgCl_2$ in rainbow trout and carp at low concentrations. Bull Jpn Soc Sci Fish (Nippon Suisan Gakkaishi) 53(5): 841-5.

Watras CJ, Back RC, Halvorsen S, Hudson RJM, Morrison KA and Wente SP (1998). Bioaccumulation of mercury in pelagic freshwater food webs. Sci Total Environ 219(2): 183-208.

Wiener JG and Giesy JP (1979). Concentrations of Cd, Cu, Mn, Pb, and Zn in fishes in a highly organic softwater pond. J Fish.Res Board Can 36: 270-9.

Zabik ME, Booren A, Zabik MJ, Welch R and Humphrey H (1996). Pesticide residues, PCBs and PAHs in baked, charbroiled, salt boiled and smoked Great Lakes lake trout. Food Chem 55(3): 231-9.

Appendix J. Lactational Transfer

J.1 Introduction

Some toxic chemicals in the environment can accumulate in a woman's body and transfer to her milk during lactation. Chronic exposure to pollutants that accumulate in the mother's body can transfer a daily dose to the infant much greater than the mother's daily intake from the environment. For example, the mother's milk pathway can be responsible for about 25% of total lifetime exposure to dioxins and furans (USEPA, 2000).

Several reviews have listed numerous toxic chemical contaminants in human breast milk (Abadin et al., 1997; Liem et al., 2000; van Leeuwen and Malisch, 2002; LaKind et al., 2005; Li et al., 2009). Many of these chemical contaminants are carcinogens and/or have non-cancer health impacts on people who inhale or ingest them. Data suggest that infants during the first two years of life have greater sensitivity to many toxic chemicals compared to older children and adults (OEHHA, 2009).

Multiple chemical contaminants have been measured in breast milk or have properties that increase their likelihood of partitioning to milk during lactation. OEHHA grouped these chemicals into the following four major categories:

1) Persistent highly-lipophilic, poorly metabolized organic contaminants, such as polychlorinated biphenyls (PCBs), polychlorinated dibenzofurans (PCDFs), and polychlorinated dibenzo-p-dioxins (PCDDs), are by far the most documented group. These, by virtue of their lipophilicity, are found almost entirely in the milk fat. PCBs, methyl sulfones, and hexachlorobenzene (HCB) methyl sulfones have also been measured in the lipid phase of breast milk.

2) Lipophilic but more effectively metabolized organic contaminants such as polycyclic aromatic hydrocarbons (PAHs) occur in breast milk. The PAHs are a family of over 100 different chemicals formed during incomplete combustion of biomass (e.g. coal, oil and gas, garbage, tobacco or charbroiled meat). Some of the more common parent compounds have been measured in breast milk and research suggests that chronic exposure to PAHs produces stores in maternal fat that can transfer (carryover) to breast milk (Fürst et al., 1993; Costera et al., 2009).

3) Inorganic compounds, metals, and some organo-metallics, including the heavy metals arsenic, lead, cadmium, and mercury, have been found in breast milk. These inorganics are generally found in the aqueous phase and most are bound to proteins, small polypeptides, and free amino acids. The lipid phase may also contain some organometallics (e.g. methyl mercury) and metalloids (such as arsenic and selenium).

4) Chemicals with relatively low octanol:water partition coefficients such as phenol, benzene, halobenzenes, halophenols, some aldehydes and the more polar metabolites of PCBs, PAHs, and pesticides may occur in both the aqueous and lipid phases of breast milk.

Since this document supports risk assessments conducted under the Air Toxics Hot Spots program, we are primarily discussing Hot Spots chemicals emitted from stationary sources.

Many of these persistent chemicals are ubiquitous in the environment and are global pollutants found in low concentrations in air, water and soil. Because some of these chemicals bio-concentrate in animal fat, the primary pathway of exposure to breastfeeding mothers would be consumption of animal products such as meat, milk, and eggs. Nearby polluting facilities can be a local source of exposure and can add to the mother's body burden of contaminants from global pollution through multiple pathways.

This appendix develops lactational transfer coefficients for use in estimating the concentration of a multipathway chemical in mother's milk from an estimate of chronic incremental daily dose to the mother from local stationary sources. OEHHA derived human lactation transfer coefficients from studies that measured contaminants in human milk and daily intake from inhalation or oral routes of exposure from global pathways (e.g. air, cigarette smoke or diet) in the same or a similar human population.

Briefly, human milk transfer coefficients (Tco_{hm}) represent the transfer relationship between the chemical concentration found in milk and the mother's chronic daily dose (i.e. concentration (µg/kg-milk)/dose (µg/day) under steady state conditions. In its simplest form, the biotransfer factor is:

$$Tco_{hm} = C_m / D_t \qquad \textbf{(Eq. J-1)}$$

where:

Tco_{hm} = transfer coefficient from ingested and inhaled media (day/kg)
C_m = concentration of chemical in mother's milk (µg/kg-milk)
D_t = total maternal dose through all exposure routes (µg/day)

Equation J-2 estimates the concentration of contaminants in mother's milk by incorporating the Tco in the following way:

$$Cm = [DOSEair + DOSEwater + DOSEfood + DOSEsoil + DOSEdermal\ ] \times Tco_{hm} \times BW \qquad \textbf{(Eq. J-2)}$$

where:

BW = the body weight of the mother at age 25 (default = 70.7 kg)
DOSEair = dose to the mother through inhalation (µg/kg-BW-day)
DOSEwater = dose to the mother though drinking water ingestion (µg/kg-BW-day)

DOSEfood = dose to the mother through ingestion of food sources (µg/kg-BW-day)
DOSEsoil = dose to the mother through incidental ingestion of soil (µg/kg-BW-day)
DOSEdermal = dose to the mother through dermal exposure to contaminated soil (µg/kg-BW-day)

However, if separate biotransfer information is available for the oral and inhalation route, equation J-3 incorporates route-specific Tcos in the following way:

$$Cm = [(D\_inh \times Tco_{m\_inh}) + (D\_ing \times Tco_{m\_ing})] \times BW \quad \textbf{(Eq. J-3)}$$

where:

D_ing = the sum of DOSEfood + DOSEsoil + DOSEwater through ingestion (mg/kg-BW-day)
D_inh = the sum of DOSEair + DOSEdermal through inhalation and dermal absorption (mg/kg-BW-day)
Tcom_inh = biotransfer coefficient from inhalation to mother's milk (d/kg-milk)
Tcom_ing = biotransfer coefficient from ingestion to mother's milk (d/kg-milk)

These coefficients, applied to the mother's chronic daily dose estimated by the Hot Spots exposure model, estimate a chemical concentration in her milk (see Table J.1-1).

Table J.1-1: Default Tcos (d/kg) for Mother's Milk

Chemical/chem. group	Tco	LCL	UCL
PCDDs - oral	3.7	2.68	5.23
PCDFs - oral	1.8	1.27	2.43
Dioxin-like PCBs - oral	1.7	0.69	4.40
PAHs – inhalation	1.55	0.731	3.281
PAHs – oral	0.401	0.132	1.218
Lead - inhalation	0.064	0.056	0.074

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

Table J.1-1 lists the transfer coefficients for dioxins, furans, dioxin-like PCBs, PAHs and lead that OEHHA has estimated from data found in the peer-reviewed literature and reviewed in this appendix. One key factor that plays a role in the difference between oral and inhalation transfer coefficient (e.g., for PAHs) is first pass metabolism which is lacking in dermal and inhalation exposures. Thus, for simplicity, OEHHA recommends applying the transfer coefficients from inhalation to the dermal absorption pathway for lead and PAHs. For lead, we recommend using the inhalation Tco for all the other pathways of exposure to the mother. Likewise, for PCDD/Fs and dioxin-like PCBs, we recommend using the oral Tco for the other pathways of exposure to the mother in Eq. J-2.

Estimates of toxicant biotransfer to breast milk are ideally chemical-specific. Data necessary to develop a transfer model are available in the open literature for a limited number of chemicals. Therefore, for some toxicants OEHHA has modeled the transfer of a class of chemicals with similar physical-chemical properties using a single Tco when data in the open literature are lacking.

The Hot Spots exposure model can estimate long-term total dose from an individual facility or group of facilities through many pathways of contamination and routes of exposure to the mother and ultimately to her infant. In this appendix, "multipathway toxicants" refers to airborne-released chemicals that can cause exposure through pathways in addition to inhalation. The indirect exposure pathways evaluated under the Hot Spots program include incidental ingestion of contaminated soil, ingestion of contaminated home-raised meat and milk, surface drinking water, homegrown produce, angler-caught fish and skin contact with contaminated soil.

Relative to the lifetime average daily dose to the infant from other exposure pathways in the Hot Spots exposure model, the dose of some chemicals from mother's milk will be negligible. However, the mother's milk pathway may be a substantial contributor to the estimated total lifetime cancer risk for some chemicals emitted from a Hot Spots facility. Exposure from global sources is expected to make up most (almost all) of a mother's toxicant body burden for chemcials like PCDDs. Therefore, the contribution to a mother's toxicant body burden from a single Hot Spot facility is expected to be very small. Regardless of the mother's toxicant body burden from both local and global sources, the benefits of breastfeeding outweigh the risks to the infant exposed to these toxicants during breastfeeding. Breast-feeding has a number of universally accepted benefits for the infant as well as for the mother (Mukerjee, 1998).

We established transfer coefficients (Tcos) for individual congeners of PCDDs/Fs and dioxin-like PCBs, individual and summary carcinogenic PAHs and lead through equations J.1-1 through J.1-3. We used data on exposure and breast milk contamination from background (global), accidental and occupational sources, and a set of simplifying assumptions. We assume that a mother's intake and elimination rates remain constant before lactation. We also assume that changes in a woman's body due to the onset of lactation occur as a single shift in elimination rate and do not change over the lactation period. Unless a study reported the geometric mean or median, we converted arithmetic mean and standard deviation to geometric mean and GSD.

In the following sections, we describe the methods for deriving specific Tcos from measurements of human milk intake and transfer estimates from studies of populations published in the open literature. In some cases, OEHHA adjusted some measurements of human milk and contaminant intake to account for confounding factors. In such cases, OEHHA describes the method of adjustment in the text and table containing adjusted values.

J.2 Mothers' Milk Transfer Coefficients for PCDD/Fs and PCBs

Polychlorinated dibenzo-p-dioxins and furans (PCDD/Fs) are two series of almost planar tricyclic aromatic compounds with over 200 congeners, which form as impurities in the manufacture of other chemicals such as pentachlorophenol and PCBs. PCDD/Fs also form during combustion (e.g. waste incineration) and the breakdown of biomass (e.g. in sewage sludge and garden compost) (Liem et al., 2000). IARC has classified many dioxins and dioxin-like compounds as known or possible carcinogens (WHO, 1997; OEHHA, 2009). Their carcinogenic potency is related to the potency of 2,3,7,8-TCDD in a toxic equivalent (TEQ) weighting scheme (OEHHA, 2009).

The main exposure to PCDD/Fs in the general population from global sources is through the intake of food of animal origin. PCB exposure has been linked to fish consumption. For example, Jensen (1987) observed that congener distribution patterns in contaminated fish and human milk were very similar suggesting that one of the primary sources of human exposure to PCBs in the study population was ingestion of contaminated fish (Jensen, 1987).

Estimates of PCDD/F and PCB TEQ-intake from dietary sources contaminated by global sources can vary by 3 to 4-fold within some populations and by as much as 29-fold between populations (Liem et al., 2000; Focant et al., 2002). Exposure from diet can be at least an order of magnitude higher than intake from ambient air or cigarette smoking (i.e., 0.1 to 4 pg/day) (Liem et al., 2000).

J.2.1 Biotransfer of PCDD/Fs and PCBs to Human Milk

The potential health impacts from exposure to PCBs, PCDDs and PCDFs include carcinogenicity, developmental, endocrine disruption, reproductive toxicity, and neurotoxicity. These persistent, lipophilic compounds can accumulate in the fat of women, transfer to breast milk, and thus result in infant exposure. Some countries implemented measures to reduce dioxin emissions in the late 1980s (Liem et al., 2000). PCBs were banned in the late 1970's and are no longer used in commercial products. Nevertheless, following the PCB ban and efforts to reduce PCDDs, PCDFs emissions, these toxicants are still detected worldwide in human milk, although at declining levels.

The World Health Organization (WHO) has carried out a series of international studies on levels of approximately 29 dioxins and dioxin-like contaminants in breast milk. The first WHO-coordinated study took place in 1987-1988, the second round in 1992-1993 and the third round was initiated in 2000-2003. In the second round, in which concentrations of PCBs, PCDDs and PCDFs were determined in milk samples collected in 47 areas from 19 different countries, mean levels in industrialized countries ranged from 10-35 pg I-TEQ/g-milk (Liem et al., 2000).

Much lower levels (40% lower than 1993) were detected in the 3rd round (Liem et al., 1995; Liem et al., 2000; van Leeuwen and Malisch, 2002) WHO exposure study. Nevertheless, several recent investigators have continued to measure levels of dioxin-like compounds in breast milk (LaKind et al., 2004; Barr et al., 2005; Wang and

Needham, 2007; Li et al., 2009). PCBs still appear in human milk and are still much higher than the total concentrations of PCDDs and PCDFs. Several studies report pg/g-fat levels of PCDD/Fs compared to ng/g-fat levels of PCBs (100 to 1000 times higher) measured in human milk (Chao et al., 2003; Chao et al., 2004; Hedley et al., 2006; Sasamoto et al., 2006; Harden et al., 2007; Wittsiepe et al., 2007; Raab et al., 2008; Todaka et al., 2008).

Thus, nursing infants have the potential to ingest substantial doses during the breastfeeding period, relative to typical total lifetime dose of these compounds from global sources. Consequently, this pathway of exposure may supply a substantial fraction of PCDDs and PCDFs (about 25%) of the infant's total lifetime dose of these compounds (USEPA, 2000). Several studies have detected higher levels of PCBs in the sera (Schantz et al., 1994), adipose tissues (Niessen et al., 1984; Teufel et al., 1990) and bone marrow (Scheele et al., 1995) of mostly breast-fed children relative to partially breast fed infants. These studies were conducted many years after PCBs were banned and no longer used in commercial products. Some investigators have reported a 4-fold greater level of PCBs in the blood of fully breast-fed compared to partially breast-fed infants (Niessen et al., 1984).

In another study, Abraham et al (1994, 1996, 1998) measured elevated PCB concentrations in nursing infants after approximately one year of feeding (Abraham et al., 1994; Abraham et al., 1996; Abraham et al., 1998). These authors reported levels of 34 to 45 ppt (pg TEQ/g blood lipid) among breastfed infants versus 3 to 3.3 ppt blood lipid PCDD/F TEQ concentrations among formula fed infants.

Numerous studies have measured dioxins, furans and dioxin-like PCBs in mother's milk (Liem et al., 2000) The twenty nine dioxin-like PCBs listed in Table J.2-1 are recognized by OEHHA as carcinogens and have potency factors associated with them (OEHHA, 2008). Concentrations of TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin), the most toxic PCDD, are low relative to other PCDDs and more than 50% of the total PCDD content consists of Octa-CDD. Early studies found around 70% of the total Hexa-CDDs (HxCDDs) is 1,2,3,6,7,8-HxCDD, and the remainder is mainly 1,2,3,4,7,8-HxCDD and 1,2,3,7,8,9-HxCDD (USEPA, 1998). These proportions have not shifted in recent studies (Sasamoto et al., 2006; Zhao et al., 2007; Raab et al., 2008).

PeCDD (1,2,3,7,8 Penta-CDD) is always found in the emissions from waste incinerators (USEPA, 1998). Early studies indicated that the presence of 1,2,3,7,8-PeCDD with other PCDDs/PCDFs in human milk suggested that the major source of exposure came from waste incinerator emissions (Buser and Rappe, 1984; Rappe et al., 1985; Mukerjee and Cleverly, 1987). Note that these congeners are measurable in human milk currently (Sasamoto et al., 2006; Zhao et al., 2007; Raab et al., 2008).

Levels of PCDFs in human milk tend to be lower than PCDDs. However, PCDFs dominate in particulates emitted by combustion sources, including hazardous waste incinerators, and are present in higher concentrations in the atmosphere than PCDDs (USEPA, 1998). HxCDDs/HxCDFs and HpCDDs/HpCDFs are prevalent in pentachlorophenol. Incineration of wood and other products impregnated with

pentachlorophenol results in the formation of these congeners and emissions of hexa- and hepta-CDDs/CDFs. Both 1,2,3,7,8 and 2,3,4,7,8-PeCDFs have been detected in human milk, but 90% of the PeCDFs is generally 2,3,4,7,8-PeCDF. 1,2,3,4,7,8-, and 1,2,3,6,7,8- HxCDFs, 2,3,4,6,7,8-HxCDFs, and 1,2,3,4,6,7,8-HpCDF are also prevalent.

Several investigators have observed that dose, degree of chlorination, degree of lipophilicity, and molecular weight influence how much PCDD/F congener is absorbed through the lungs or gut, metabolized and transferred from blood to milk (Yakushiji, 1988; Abraham et al., 1998; Schecter et al., 1998; Kostyniak et al., 1999; Oberg et al., 2002; Wittsiepe et al., 2007).

Numerous studies have attempted to correlate exposure to individual dioxins, furans and dioxin-like PCBs from ingestion of contaminated food with levels in human biological samples such as blood and milk. Transfer from intake sources to human milk has often been estimated in the context of accidental or occupational exposures or after a substantial decline in environmental concentrations (Liem et al., 1995; Pinsky and Lorber, 1998; Liem et al., 2000; Focant et al., 2002; Furst, 2006; Milbrath et al., 2009). Steady state conditions are not reached in these studies because the half-lives of these compounds are in years and exposure changed considerably over the period evaluated in each study.

Others have attempted to model the relationship between maternal intake and concentration in mother's milk using an indicator compound such as TCDD (Smith, 1987; Lorber and Phillips, 2002). Less understood is the relationship between modeled and measured transfer estimates of individual dioxins, furans and dioxin-like PCBs. The following sections describe the sources of data and methods for deriving estimates of transfer for an array of dioxins, furans and dioxin-like PCBs that have accounted to some extent for the non-steady state condition and other confounders.

J.2.2 Oral Biotransfer

OEHHA located a series of studies conducted on the Dutch population that allows for an oral biotransfer estimate of dioxins and furans, and accounts for changing exposure conditions. In 1988, Albers et al. collected and analyzed three hundred nineteen breast milk samples from women enrolled through 28 maternity centers located throughout the Netherlands. Maternity centers were selected based on geographic distribution and degree of urbanization. Human milk samples were analyzed for 17 PCDD/F congeners and 8 PCB congeners (Albers et al., 1996).

Liem et al. (1995) took a similar approach to collect about 100 samples from first-time mothers enrolled in 1993 through maternity centers dispersed throughout The Netherlands. Based on information obtained from a questionnaire about characteristics of the study subject, investigators determined that the 1993 cohort appeared to be comparable to the cohort studied in 1988. With one exception, (1,2,3,4,7,8- HxCDD), a consistent downward trend can be seen among congeners of PCDD/Fs and PCB-118 that were analyzed during both sampling periods, (Table J.2-1).

Table J.2-1: Summary Estimates of Dioxin-like Compounds Dietary Intake during Three Periods Over 15 years, and Human Milk Levels over Five Years in the Dutch Population

Chemical/ group	TEF	1978 (diet)[a]	1984/5 (diet)[a]	1994 (diet)[a]	1988 (milk)[b]	1993 (milk)[a]
		Mean, SD	Mean, SD	Mean, SD	Mean, SD	Mean, SD
		pg/d*	pg/d*	pg/d*	pg/kg-milk	pg/kg-milk
2,3,7,8-TCDD	1	13.2, 1.32	6, 2.94	3.6, 1.26	264,14	124, 56
1,2,3,7,8-PeCDD	1	39.6, 6.73	15, 4.65	4.8, 2.26	435,185	324, 116
1,2,3,4,7,8-HxCDD	0.1	85.8, 23.17	23.4, 17.55	7.2, 5.98	328,51	344, 192
1,2,3,6,7,8-HxCDD	0.1	325.8, 45.61	89.4, 42.02	19.8, 22.77	2445,349	1484, 668
1,2,3,7,8,9-HxCDD	0.1	105, 21.0	32.4, 21.38	10.8, 9.61	395,32	276, 132
1,2,3,4,6,7,8-HpCDD	0.01	2016, 463.68	1908, 2671.2	150, 120	3242,114	1796, 984
OctaCDD	0.0001	12420, 4595	9180, 10281	1170, 749	28844,2896	11788, 6708
2,3,7,8-TCDF	0.1	106.8, 9.61	84, 31.08	21, 14.7	100,8	16, 16
1,2,3,7,8-PeCDF	0.05	24.6, 4.67	6.6, 2.71	3.6, 1.51	30,10	8, 8
2,3,4,7,8-PeCDF	0.5	178.8, 25.03	65.4, 13.73	23.4, 12.87	807,108	720, 300
1,2,3,4,7,8-HxCDF	0.1	178.8, 30.40	43.8, 9.20	27.6, 11.04	293,20	208, 92
1,2,3,6,7,8-HxCDF	0.1	54, 3.78	27, 6.21	13.8, 5.52	261,17	176, 84
1,2,3,7,8,9-HxCDF	0.1	<0.05	<0.04	<0.04	NA	NA
2,3,4,6,7,8-HxCDF	0.1	55.8, 6.70	25.2, 6.80	9, 5.76	133,19	96, 52
1,2,3,4,6,7,8-HpCDF	0.01	471, 117.75	176.4, 65.27	51.6, 22.19	523,55	240, 124
1,2,3,4,7,8,9-HpCDF	0.01	39, 4.68	7.8, 5.07	3, 1.62	NA	4, 4
OctaCDF	0.0001	466.8, 107.36	195, 78.0	69.6, 37.58	49,10	12, 12
PCB-77	0.0001	NA	NA	NA	NA	452, 872
PCB-81	0.0001	NA	NA	NA	NA	NA
PCB-126	0.1	1350, 202.5	924, 221.76	378.6, 87.08	NA	3284, 1448
PCB-169	0.01	270, 54.0	181.2, 86.98	174, 214.02	NA	2320, 988

Table J.2-1: Summary Estimates of Dioxin-like Compounds Dietary Intake during Three Periods Over 15 years, and Human Milk Levels over Five Years in the Dutch Population

Chemical/ group	TEF	1978 (diet)[a]	1984/5 (diet)[a]	1994 (diet)[a]	1988 (milk)[b]	1993 (milk)[a]
		Mean, SD	Mean, SD	Mean, SD	Mean, SD	Mean, SD
		ng/d*	ng/d*	ng/d*	ng/kg-milk	ng/kg-milk
PCB-105	0.0001	71.4, 13.57	70.2, 33.7	13.2, 5.54	NA	160, 80
PCB-114	0.0005	6.6, 0.92	11.4, 8.66	1.8, 1.35	NA	NA
PCB-118	0.0001	289.2, 43.38	247.2, 111.24	49.2, 15.25	1009,565	971.2, 456
PCB-123	0.0001	18.6, 3.91	15, 7.65	2.4, 0.89	NA	NA
PCB-156	0.0005	191.4, 63.16	27.6, 8.28	9, 2.79	NA	564, 236
PCB-157	0.0005	22.2, 6.44	4.8, 1.73	1.8, 0.72	NA	108, 48
PCB-167	0.00001	79.2, 22.18	11.4, 2.51	3.6, 1.01	NA	152, 64
PCB-189	0.0001	43.8, 13.14	2.4, 0.53	1.2, 0.31	NA	48.4, 48

[a] (Liem et al., 2000); [b] (Albers et al., 1996), NA, not available

* Conversion from g-fat to kg-milk = 0.04 g-fat/g-milk*1000g/kg; Liem et al. reported dietary intake estimates in units of mass/body weight/day. Therefore, we converted their estimates to units of mass/day by multiplying by the default 60 kg body weight used by Liem et al (Liem et al., 2000).

Liem et al. (2000) reported dietary intake for three time-periods (see Table J.2-1)(Liem et al., 2000). Dietary intake estimates were based on concentrations of PCDD/Fs and PCBs measured in composite samples of 24-hr duplicate diets in the Dutch adult population in 1978, 1984-85, and 1994 and combined with individual consumption data collected in 1987-1988 (Albers et al., 1996) (briefly summarized previously) for approximately 6000 individuals from 2200 families over a 2-day period . In a separate study, these same investigators estimated dioxin and dioxin-like compounds in human milk fat collected in the period 1992-1993 from more than 80 women (Liem et al., 1995; Liem et al., 2000).

Liem et al. (2000) observed a downward trend in estimated dietary intake of individual congeners of PCDDs PCDFs and PCBs in the Dutch population during three intervals from 1978 to 1994 (see Table J.2-1)(Liem et al., 2000). A downward trend was also seen in a study of these toxicant levels in the diet and human milk of the German population from 1983 - 2003 (Furst, 2006; Wilhelm et al., 2007). However, about half of the mono-ortho PCBs did not show a similar linear decline. This pattern is consistent with observations made by Alcock et al., (1996) who reported some evidence that the environmental load of PCDD/Fs increased in the 1960s, peaked around 1975 and then began to decline (Alcock et al., 1996).

OEHHA has derived lactational transfer coefficients for PCDD/Fs and dioxin-like PCBs from studies of exposure from global sources and by multiple pathways. The proportional contribution from various exposure pathways to total exposure from a single Hot Spots facility is likely to be quite different from that found from global sources.

However, we assume that the estimate of transfer to milk from global sources, such as that derived from the Dutch studies, reasonably represents the transfer in persons from communities near Hot Spot facilities in California.

The Hot Spots program allows for reporting emissions of individual congeners of dioxins, furans and PCBs, when emissions are speciated. It also permits reporting of emissions as total dioxins and furans or PCBs. Speciation of emissions produces a more accurate (and lower) risk estimate. This is because unspeciated emissions are assumed to be 2,3,7,8-TCDD, which has the highest potency factor among the dioxins and furans. Therefore, OEHHA has derived congener Tcos for individual PCBs and dioxins that can be used when emissions are speciated.

J.2.3 Mothers' Milk Transfer Coefficients (Tco) for PCDD/Fs and PCBs

To calculate oral Tcos, OEHHA used adjusted reference half-lives for the chemicals in adults estimated from dietary and occupational exposures. OEHHA estimated oral Tcos for these chemicals using estimates of body weight reported in Chapter 10 of this document, reference half-lives reported in Milbrath et al. (2009) and the steady-state equation developed by Smith (1987) (Smith, 1987; Milbrath et al., 2009).

Milbrath et al., (2009), in a systematic review of studies reporting half-lives in the human body, developed average human biological reference half-lives for 28 out of 29 dioxins and dioxin-like PCBs with OEHHA-recognized potency factors (see Table J-2-2) (Milbrath et al., 2009).

Each reference half-life was derived from data on occupational exposures (Flesch-Janys et al., 1996; van der Molen et al., 1996) or dietary intake of the general population (Ogura, 2004). Note that mean half-lives vary by more than 2-fold among dioxin, 5-fold among furans and more than 100-fold among PCB congeners.

Table J.2-2: Half-lives of PCDD/Fs and Dioxin-like PCB Congeners in Humans as Measured in Blood (Milbrath et al., 2009)

Chemical	N studies	Half-life range (yrs)	Mean half-life in adult (yrs)	Median half-life in adult (yrs)	Study
TCDD	10	1.5 – 15.4	7.2	6.3	a
1,2,3,7,8-PeCDD	4	3.6 – 23.1	11.2	8.5	a
1,2,3,4,7,8-HxCDD	3	1.4 – 19.8	9.8	10.9	a
1,2,3,6,7,8-HxCDD	4	2.9 – 70	13.1	12	a
1,2,3,7,8,9-HxCDD	3	2.0 – 9.2	5.1	6.8	a
1,2,3,4,6,7,8-HpCDD	4	1.6 – 16.1	4.9	3.7	a
OctaCDD	4	1.8 - 26	6.7	5.7	a
2,3,7,8-TCDF	1	0.4	2.1	0.9	b
1,2,3,7,8-PeCDF	4	0.9-7.5	3.5	1.9	b
2,3,4,7,8- PeCDF	16	1.5-36	7	4.9	b
1,2,3,4,7,8-HxCDF	14	1.5-54	6.4	4.8	a
1,2,3,6,7,8-HxCDF	6	2.1-26	7.2	6	a
2,3,4,6,7,8-HxCDF	6	1.5-19.8	2.8	3.4	b
1,2,3,4,6,7,8-HpCDF	11	2.0-7.2	3.1	3	a
1,2,3,4,7,8,9-HpCDF	1	2.1-3.2	4.6	5.2	b
OctaCDF	1	0.2	1.4	1.6	b
PCB-77	2	0.1-5.02	0.1	0.1	c
PCB-81	-	-	0.7	0.73	c
PCB-126	3	1.2-11	1.6	2.7	c
PCB-169	3	5.2-10.4	7.3	10.4	c
PCB-105	4	0.56-7.0	2.4	2.4	c
PCB-114	2	7.4-31.7	10	25	c
PCB-118	10	0.82-33.7	3.8	1.6	c
PCB-123	2	5.3-15.3	7.4	12	c
PCB-156	7	1.62-100	16	5.35	c
PCB-157	2	13-26	18	20	c
PCB-167	2	8.7-35	12	12	c
PCB-189	2	16-166.7	22	41	c

[a] (Flesch-Janys et al., 1996); [b] (van der Molen et al., 1996); [c] (Ogura, 2004)

In an initial review of the literature, Milbrath et al (2009) reviewed evidence about factors that can affect elimination rates. Personal factors such as body fat, smoking status and past lactation practices can affect body burden and elimination rates. For example, smoking has been associated with a 30% to 100% increase in elimination rates of some dioxin congeners (Flesch-Janys et al., 1996; Milbrath et al., 2009). As well, the onset of lactation sets a new elimination pathway into effect and can substantially reduce the maternal body burden of PCBs during 6 months of lactation (Niessen et al., 1984; Landrigan et al., 2002).

Half-lives derived from children would be less than that from older adults due, in part, to the effects of the growing body on estimates of blood concentrations. Models based on rat data demonstrate a linear relationship between increasing fat mass and half-life length at low body burdens, with the impact of adipose tissue on half-life becoming less important at high body burdens (Emond et al 2006). At high body burdens, dioxins are known to up-regulate the enzymes responsible for their own elimination. Human data suggest that the serum concentration of TCDD where this transition occurs is 700 pg/g and 1,000 – 3,000 pg/g for PCDFs (Kerger et al 2006, Leung et al 2005). Therefore, investigators selected a subset of data based on the following criteria:

- blood serum concentrations of PCDD/Fs were less than 700 pg /g blood lipid total toxic equivalents (TEQs) at the time of sampling
- subjects were adults
- measurements were not reported as inaccurate in later studies

Milbrath et al selected the reference values to represent a 40- to 50-year-old adult with blood dioxin concentrations in the range where fat drives the rate of elimination (i.e. at lower body burdens). In addition, Milbrath rejected half-lives longer than 25 years if the original study calculated half-lives assuming steady-state conditions.

For the retained subset, the investigators calculated the mean and range of half-lives to establish a representative set of half-lives for each congener in a moderately exposed adult (Milbrath et al., 2009). They also adjusted reference half-lives for age, body fat, smoking habits and breast-feeding status as these factors were all strong determinants of half-life in humans (Milbrath et al., 2009).

A generally accepted approach to estimating the concentration of a lipophilic chemical in milk is outlined by Smith (1987). This approach is based on average maternal daily intake, an estimate of the half-life ($t_{1/2}$) of PCDDs/PCDFs and PCBs and body weight-normalized (BW) proportionality factors. The chemical concentration in breast milk can be calculated by equation J-4:

$$Cm = (Emi)(t_{1/2})(f1)(f3)/(f2)(0.693) \quad \textbf{(Eq. J-4)}$$

Where:

Cm = chemical concentration in milk (mg/kg milk)
Emi = average daily maternal intake of contaminant (mg/kg-BW/day)
$t_{½}$ = biological half-life (days)
f1 = proportion of chemical in mother that partitions into fat (e.g. 0.8)
f2 = proportion of mother's body weight that is fat (e.g. 0.33 = kg-fat/kg-BW)
f3 = proportion of breast milk that is fat (e.g., 0.04 = kg-fat/kg-milk)

Smith's approach requires an estimate of the biological half-life of PCBs and PCDDs/PCDFs in the adult human and is restricted to poorly metabolized, lipophilic chemicals that act predominantly by partitioning into the fat component and quickly reaching equilibrium in each body tissue (including breast milk).

Because of Milbrath's approach, Tco-estimates for dioxins, furans and dioxin-like PCBs apply the following conservative assumptions regarding factors that affect elimination rates:

- lower enzyme induction based on nonsmokers with a body burden below 700 ppt in the blood
- adult age
- no recent history of breast-feeding
- body fat estimates based on older adults

Transfer coefficients (Ng, 1982) are ideally calculated from the concentration of contaminant in milk following relatively constant long-term exposure that approximates steady state conditions. Because Smith's equation is linear, it can be rearranged to solve ratio of the chemical concentration in milk to the chemical taken into the body per day, which is the transfer coefficient (Equation J-5).

$$Tco = Cm/(Cf)(I) \quad \textbf{(Eq J-5)}$$

Where:

Tco is the transfer coefficient (day/kg or day/liter)
Cm = measured chemical concentration in milk (µg/kg or mg/liter milk)
Cf = measured chemical concentration in exposure media (e.g. food) (µg/kg food)
I = reported daily intake of exposure media (kg/day of food)

The following equation (Eq-J-6) is equation Eq J-5 substituted into equation Eq J-4 and rearranged to solve for Tco.

$$Tco = (t_{1/2})(f1)(f3)/(BW)(f2)(0.693) \quad \textbf{(Eq J-6)}$$

Note that Emi in equation J-4 = (Cf)(I)/BW with units of mg/kg-BW/day. BW is the average adult body weight of the mother (kg).

Transfer coefficients (Tcos) in Table J.2-3 (column-2) combine milk data (milk concentration of PCDD/Fs and PCBs) with dietary intake estimates listed in Table J.2-1. OEHHA derived individual Tcos from data presented in (Liem et al., 1995; Albers et al., 1996; Liem et al., 2000). Because the median is a reasonable estimate of the geometric mean in skewed distributions, Tcos were derived from median half-lives listed in column-5 of Table J.2-2. Tcos range from less than one to more than ten d/kg-milk among dioxins and furan and less than two to more than 20 d/kg-milk among dioxin-like compounds.

Table J.2-3: Arithmetic Mean Transfer Coefficients (Tcos) for Individual PCDD/F and PCB Congeners Measured in Human Milk and Dietary Intake from a Dutch Population (d/kg-milk) Compared to the Median and Geometric Mean Tcos Derived from Reference Half-lives ($t_{1/2}$) and Equation J-6

Chemical/group	Tcos (GM) based on slope factors	Tco based on median reference half life (Milbrath et al 2007)	Tco based on t1/2 GM*	Tco based on t1/2 GSD	Tco based on t1/2 LCL	Tco based on t1/2 UCL
2,3,7,8-TCDD	49.62	5.36	4.02	2.76	2.14	7.53
1,2,3,7,8-PeCDD	8.76	7.24	6.53	2.16	3.07	13.90
1,2,3,4,7,8-HxCDD	0.98	9.28	5.60	3.41	1.40	22.48
1,2,3,6,7,8-HxCDD	11.02	10.21	3.27	4.20	0.80	13.32
1,2,3,7,8,9-HxCDD	4.89	5.79	3.32	1.91	1.60	6.88
1,2,3,4,6,7,8-HpCDD	2.88	3.15	1.96	2.74	0.73	5.26
OctaCDD	5.54	4.85	2.29	3.25	0.72	7.28
2,3,7,8-TCDF	3.18	0.77	1.76	1.36	0.96	3.23
1,2,3,7,8-PeCDF	3.43	1.62	1.91	2.49	0.78	4.68
2,3,4,7,8- PeCDF	2.77	4.17	1.78	4.24	0.88	3.62
1,2,3,4,7,8-HxCDF	2.16	4.09	0.99	5.29	0.41	2.38
1,2,3,6,7,8-HxCDF	7.89	5.11	2.64	3.01	1.09	6.39
1,2,3,7,8,9-HxCDF	NA	NA	NA	NA	NA	NA
2,3,4,6,7,8-HxCDF	3.18	2.89	0.55	3.18	0.22	1.39
1,2,3,4,6,7,8-HpCDF	2.40	2.55	1.82	1.63	1.36	2.44
1,2,3,4,7,8,9-HpCDF	NA	4.43	3.63	1.34	2.06	6.42
OctaCDF	0.32	1.36	0.99	2.83	0.13	7.55
PCB-77	NA	NA	0.06	6.38	0.004	0.72
PCB-81	NA	NA	0.38	1.35	0.248	0.57
PCB-126	NA	2.30	0.34	2.61	0.11	1.01
PCB-169	NA	8.85	5.60	1.27	4.28	7.32
PCB-105	NA	2.04	1.07	3.02	0.36	3.16
PCB-114	NA	2.04	2.74	3.11	0.57	13.20
PCB-118	0.01	1.36	0.55	6.17	0.18	1.70
PCB-123	NA	1.36	2.93	2.63	0.77	11.18
PCB-156	NA	4.55	3.23	7.10	0.76	13.81
PCB-157	NA	17.02	14.10	1.21	10.84	18.34
PCB-167	NA	10.21	5.93	1.76	2.70	13.00
PCB-189	NA	34.90	4.23	2.77	1.03	17.33

slope factors obtained from the longest interval between measures of diet (1978-1994) and milk (1988-1993) in the Dutch population; * GM, geometric mean, GSD, geometric standard deviation derived from natural log of three half-life values, low, high and median reported in Milbrath et al. (Milbrath et al., 2009) LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

OEHHA evaluated the relationship between Tcos predicted by Equation J-6 (column 3) using median reference half-lives and those derived from slope factors (column 2). Briefly, slope factors were calculated by taking the difference between cross-sectional dietary intake estimates taken in 1978 and 1994 and the difference between cross-sectional human milk concentrations taken in 1988 and 1993 from the Dutch population. Most Tcos derived from reference half-lives compare reasonably well with those derived from slope factors.

In columns 4-7 of Table J.2-3 the GM, GSD and 95%CLs of transfer coefficients (Tcos) for individual dioxins and dioxin-like congeners are derived from equation J-6 and geometric distribution estimates and 95% confidence intervals of half-lives provided in (Milbrath et al., 2009).

A Random-effects model derived summary estimates shown in Table J.2-4 from individual summary estimates shown in columns 4-7 of Table J.2-3.

Table J.2-4: Tco Estimates Stratified by Dioxin, Furan and Dioxin-like PCB Congeners (mean, 95%CI from Random-effects Model)

Chemical group	N congeners	Tco	LCL	UCL
PCDDs - oral	7	3.7	2.68	5.23
PCDFs - oral	9	1.8	1.27	2.43
Dioxin-like PCBs - oral	12	1.7	0.69	4.40

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

OEHHA believes that a Random-effects model is appropriate because OEHHA assumes that the compounds found in exposure studies are a subgroup from a population of congeners in each subgroup (i.e., dioxins and dioxin-like compounds). Random-effects models assume there are multiple central estimates and incorporate a between-compound estimate of error as well as a within-compound estimate of error in the model. In contrast, a Fixed-effects model assumes that observations scatter about one central estimate (Kleinbaum, 1988).

J.2.4 Carryover Rate

Looking at mother's milk Tcos in terms of carryover rate suggests that accumulation of dioxins and dioxin-like compounds in the mother's body occurs but varies by more than 100-fold among individual compounds (based on Tcos derived from equation J-6).

Carryover rate, a term commonly used in the dairy literature (McLachlan et al., 1990) is defined as the daily output of dioxins and dioxin-like compounds in mother's milk (µg/day) over the daily intake of dioxins and dioxin-like compounds (µg/day). This rate is estimated by multiplying a dioxin's and dioxin-like Tco by the daily output of mother's milk. Since milk production in human mothers are about 1.0 kg/day, a dioxins and dioxin-like Tco is the carryover rate for a typical 60 kg woman.

A carryover rate > 1 would suggest that dioxins and dioxin-like compounds could accumulate in body fat and transfer to the fat in mother's milk. With an average dioxin Tco of 3.7 d/kg, 370% of the mother's average daily intake from ingested sources, transfers to mother's milk. This high transfer-value suggests that accumulation or concentrating of carcinogenic dioxins and dioxin-like compounds occur in the mother's body. Oral Tcos less than one d/kg (e.g., 1,2,3,4,7,8-HxCDF and 2,3,4,6,7,8-HxCDF) suggest that net metabolism or excretion occurs in the mother's body.

J.3 Mothers' Milk Transfer Coefficients for PAHs

The polycyclic aromatic hydrocarbons (PAHs), a family of hundreds of different chemicals, are characterized by fused multiple ring structures. These compounds are formed during incomplete combustion of organic substances (e.g. coal, oil and gas, garbage, tobacco or charbroiled meat). Thus, PAHs are ubiquitous in the environment and humans are likely to be exposed to these compounds on a daily basis. PAHs are a common pollutant emitted from Hot Spots facilities and are evaluated under the program.

Only a small number of the PAHs have undergone toxicological testing for cancer and/or noncancer health effects. PAHs with cancer potency factors are the only ones that can be evaluated for cancer risk using risk assessment. However, PAHs that lack cancer potency factors have been measured in various studies and can serve as a useful surrogate for PAHs with cancer potency factors because of their physical-chemical similarity to PAHs with cancer potency factors.

Less than 30 specific PAHs are measured consistently in biological samples or in exposure studies. For example, Table J.3-1 lists commonly detectable PAHs in food and the environment (Phillips, 1999). In one analysis, pyrene and fluoranthene together accounted for half of the measured PAH levels in the diet (Phillips, 1999). Table J.3-1 includes nine PAHs that have cancer potency factors and are recognized by OEHHA as presenting a carcinogenic risk to humans (OEHHA, 2009).

Table J.3-1: PAHs with and without Cancer Potency Factors Commonly Measured in Food (Phillips, 1999)

PAHs without Cancer Potency Factors	PAHs with Cancer Potency Factors
Benzo[ghi]perylene	Dibenz[a,h]anthracene
Fluoranthene	Indeno[1,2,3-cd]pyrene
Pyrene	Benzo[a]pyrene
Phenanthrene	Benzo[k]fluoranthene
Anthracene	Chrysene
Fluorene	Benzo[b]fluoranthene
Acenaphthylene	Benz[a]anthracene
Acenaphthene	Naphthalene
Benzo[b]naphtho[2,1-d]thiophene	Benzo[j]fluoranthene
Benzo[ghi]fluoranthene	
Cyclopenta[cd]pyrene	
Triphenylene	
Perylene	
Benzo[e]pyrene	
Dibenz[a,j]anthracene	
Anthanthrene	
Coronene	

Few investigators have attempted to correlate PAH exposure from contaminated food and ambient air with PAH concentrations in human biological samples such as the blood or mother's milk. This is likely due to insensitive limits of detection for PAHs yielding few positive measurements, possibly due to the rapid and extensive metabolism of PAHs in mammals (West and Horton, 1976; Hecht et al., 1979; Bowes and Renwick, 1986).

This extensive metabolism often results in low or immeasurable concentrations of PAHs in mother's milk and blood (e.g. (Kim et al., 2008)). Nevertheless, emissions of PAHs from stationary sources are common and the increased sensitivity of infants to carcinogens necessitates looking into development of mother's milk transfer factors (Tco) for carcinogenic PAHs.

Four studies have measured PAHs in mother's milk of smokers and non-smokers (see Table J.3-2). The 16 PAHs reported in these studies are among the most common PAHs released into the environment and found in biological samples (Phillips, 1999; Ramesh et al., 2004).

TABLE J.3-2: Measured Concentrations (µg/kg-milk) of PAHs in Human Milk

Chemical / chemical group	Urban smokers (Italy) n=11[a] (Zanieri et al., 2007)	Urban non-smokers (Italy) n=10 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=11 (Zanieri et al., 2007)	Rural Non-smokers (Italy) n=10 (Del Bubba et al., 2005)	Non-smokers (USA) n=12 (Kim et al., 2008)	Unknown (Japan) n=51 (Kishikawa et al., 2003)
PAHs with Cancer Potency Factors AM, SD						
Naphthalene	10.54, 6.08	6.83, 2.18	4.42, 1.17	4.70, 2.44	NA[d]	NA
Chrysene	0.90, 2.09	0.59, 0.94	<0.018	<0.018	--[c]	0.06, 0.08
Benzo[a] anthracene	0.98, 1.47	0.61, 0.94	0.07, 0.16	0.974, 1.82	--	0.004, 0.01
Benzo[b] fluoranthene	0.53, 1.24	0.55, 0.80	<0.019	0.560, 1.39	--	0.41, 0.26
Benzo[k] fluoranthene	0.13, 0.30	<0.018	<0.018	0.114, 0.343	--	0.01, 0.01
Benzo[a]pyrene	0.52, 0.65	<0.018	<0.018	<0.018	--	0.002, 0.003
Dibenzo[a,h] anthracene	1.33, 3.33	<0.014	<0.014	<0.014	--	0.01, 0.01
Indeno[1,2,3-c,d] pyrene	0.42, 0.94	<0.011	<0.011	<0.011	--	0.003, 0.01
Sum	15.35	8.58	4.5	6.4	--	0.5
PAHs without Cancer Potency Factors AM, SD						
Anthracene	0.16, 0.45	0.71, 1.57	0.21, 0.56	0.616, 1.58	--[c]	0.01, 0.01
Acenaphthylene	7.73, 11.95	9.09, 3.08	4.11, 3.62	6.95, 4.18	NA[d]	NA
Phenanthrene	3.67, 2.39	0.97, 0.51	0.64, 0.58	0.553, 0.493	0.49, 0.44	0.25, 0.16
Fluorene	5.13, 9.45	1.50, 1.60	0.06, 0.21	1.06, 1.70	0.13, 0.13	NA
Acenaphthene	10.55, 17.73	3.12, 1.79	1.37, 1.31	2.72, 1.69	NA	NA
Pyrene	1.03, 1.25	1.40, 3.01	0.21, 0.30	0.620, 1.64	0.05, 0.04	0.02, 0.05
Fluoranthene	2.86, 2.60	0.54, 0.76	0.53, 1.03	0.250, 0.441	0.06, 0.05	0.02, 0.03
Benzo[g,h,i] perylene	1.51, 2.24	<0.018	<0.018	<0.018	--	--
Sum	32.64	17.33	7.13	12.8	0.73	0.3

[a] group includes one rural smoker; [b]values below detection limits were treated as zero in estimates of the mean; [c] – indicates all measurements were below the detection limits; [d] not assessed; (Kishikawa et al., 2003; Del Bubba et al., 2005; Zanieri et al., 2007; Kim et al., 2008) µg, microgram; kg, kilogram; n, number of samples; AM, Arithmetic Mean; SD, Standard Deviation

In this section, we estimated Tcos for PAHs with and without cancer potency factors. Additionally, none of the PAHs has a chronic Reference Exposure Level (REL) value. PAHs without cancer potency factors (other) are included because they:

- have structures similar to carcinogenic PAHs and are thus suitable as surrogate compounds
- are frequently measured in exposure studies
- produce measurements at detectable levels

In Table J.3-2, the sum of carcinogenic PAHs in human milk of Italian women is about 2-fold lower than the sum of other PAHs.

Because of their similarities in structure, the Tcos developed from other abundant PAHs are expected to compare reasonably well with the Tcos developed for less abundant carcinogenic PAHs.

J.3.1 Inhalation Biotransfer of PAHs to Mother's Milk

Biotransfer of PAHs to breast milk via the mother's inhalation pathway must be considered separately from biotransfer of PAHs to breast milk from the mother's oral route. PAHs will show a different pattern of metabolism depending on the route of exposure because of first pass metabolism in the liver from oral exposure, different rates and patterns of metabolism in the lung, and other factors. Smoking cigarettes represents a significant source of PAHs resulting in measurable levels of PAHs in mother's milk. Therefore, OEHHA chose a study that measured PAH concentrations in breast milk in smoking women and nonsmoking women to estimate inhalation Tcos for PAHs.

Of the four studies listed in Table J.3-2, the Italian study by Zanieri et al. (2007) allowed correlation of PAH intake via chronic smoking with PAH levels found in human milk (Zanieri et al., 2007). These investigators reported individual PAH concentrations in the milk of urban smoking and nonsmoking mothers, and in rural smoking and nonsmoking mothers.

Zanieri et al (2007) had obtained self-reported smoking habits (an arithmetic average of 5.4 cigarettes smoked per day) but not the daily dose of PAHs due to smoking (Zanieri et al., 2007). Therefore, OEHHA estimated daily PAH doses using published estimates of the amounts of PAHs a smoker voluntarily consumes during smoking per cigarette from simulated cigarette smoking studies. Ding et al. (2005) measured the amount of 14 individual PAHs that would be inhaled because of smoking major U.S. cigarette brands (Table J.3-3). Two other simulated smoking studies were included that estimated the inhaled amounts of two additional PAHs not covered in the Ding study (Gmeiner et al., 1997; Forehand et al., 2000).

Table J.3-3: Summary Estimates of Polycyclic Aromatic Hydrocarbons (PAHs) Intake from Cigarettes (µg/cigarette)

PAH	Ding et al (n=5)	Ding et al (n=50)	Ding et al (n=5)	Gmeiner et al (n=3)	Forehand et al (n=4)	Pooled
With Cancer Potency Factors	1[#] AM, SD[1]	2 AM, SD	3 AM, SD	1 AM, SD	1 AM, SD	AM, SD
Naphthalene	0.3503, 0.021	0.192, 0.044	0.407, 0.187	0.236, 0.019	0.362, 0.011	0.292, 0.087
Chrysene	0.0157, 0.0003	0.0197, 0.0024	0.0314, 0.0028	0.0218, 0.0009	0.0112, 0.0003	0.015, 0.0017
Benzo[a] anthracene	0.0134, 0.0007	0.0165, 0.0015	0.0226, 0.0025	0.0132, 0.0005	0.014, 0.0004	0.015, 0.0014
Benzo[b] fluoranthene	0.0094, 0.003	0.0106, 0.0013	0.0183, 0.0024	0.0086, 0.0003	0.0112, 0.0003	0.010, 0.0012
Benzo[k] fluoranthene	0.0015, 0.00014	0.0019, 0.00029	0.0039, 0.00070	0.0015, 0.00008	NA	0.0020, 0.0004
Benzo[a]pyrene	0.0103, 0.00041	0.011, 0.00077	0.0147, 0.00118	0.0079, 0.00024	0.0076, 0.00023	0.0092, 0.00067
Dibenzo[a,h] anthracene	NA	NA	NA	0.0006, 0.00013	0.0023, 0.00021	0.0023, 0.00017
Indeno[1,2,3-c,d] pyrene	NA	NA	NA	0.0035, 0.00039	NA	0.0035, 0.00039
Without Cancer Potency Factors	1 AM, SD	2 AM, SD	3 AM, SD	1 AM, SD	1 AM, SD	AM, SD
Anthracene	0.0749, 0.0052	0.0698, 0.0084	0.074, 0.0089	0.0381, 0.0023	0.0358, 0.0011	0.043, 0.0060
Acenaphthylene	0.1169, 0.0082	0.0883, 0.0097	0.153, 0.0306	0.0504, 0.0040	NA	0.083, 0.0167
Phenanthrene	0.1348, 0.0054	0.1452, 0.0131	0.144, 0.0144	0.11, 0.0033	0.1477, 0.0044	0.134, 0.0094
Fluorene	0.2175, 0.0087	0.1563, 0.0188	0.257, 0.0257	0.119, 0.0048	0.239, 0.0048	0.184, 0.0151
Acenaphthene	0.0848, 0.0025	0.0513, 0.0072	0.088, 0.0167	0.0253, 0.0013	NA	0.062, 0.0092
Pyrene	0.0486, 0.0029	0.0495, 0.0069	0.077, 0.0231	0.0332, 0.0017	0.0321, 0.0010	0.036, 0.0109
Fluoranthene	0.0744, 0.0037	0.063, 0.0107	0.101, 0.0121	0.0462, 0.0018	0.0516, 0.0026	0.056, 0.0076
Benzo[g,h,i] perylene	NA	NA	NA	0.0025, 0.00030	0.0023, 0.00018	0.0023, 0.00025

[1]AM arithmetic mean,, SD standard deviation ; #, Experiment number listed in the study reference by the first author in row one of columns two through six in the table (Gmeiner et al., 1997; Forehand et al., 2000; Ding et al., 2005)

Based on the estimated intake of 16 measured PAHs in simulated smoking studies and the PAHs found in breast milk from long-time smoking mothers by Zanieri et al. (2007), OEHHA was able to estimate transfer coefficients (Tco) with a modified version of Equation J-1:

$$Tco_{hmi} = Cm_i/(C_{cig\_i} \times I_{cig/day} \times f_{smoke}) \quad \textbf{(Eq. J-7)}$$

where:

Cm_i = adjusted geometric average ith PAH concentration due to smoking (µg per kg milk as wet weight)

C_{cig_i} = geometric average dose of the ith PAH per cigarette (µg/cigarette averaged across experiments)

$I_{cig/day}$ = geometric average number of cigarettes smoked (4.75 cigarettes/day)

f_{smoke} = adjustment for under-reporting of smoking frequency (2)

Cm_i is the adjusted geometric average of the ith PAH in whole milk due to smoking. OEHHA obtained these estimates by converting arithmetic estimates to geometric estimates of the mean and standard deviation and subtracting the GM concentration in the milk of primarily urban nonsmokers from the GM concentration in the milk of urban smokers. This adjustment accounts for oral intake of PAHs from dietary sources and inhalation of PAHs in urban air from combustion sources other than cigarettes. Implicit in this adjustment is the assumption by OEHHA that oral intake and exposure to other airborne PAHs is similar between smokers and nonsmokers who participated in the Zanieri study.

OEHHA also included a 2-fold smoking habit adjustment-factor (f_{smoke}) in Eq. J-7 based on published data to account for the recognized tendency of smokers to under-report their smoking habits. The studies examined the accuracy of self-reported smoking habits among pregnant women and parents with small children (Marbury et al., 1993; Graham and Owen, 2003). They measured airborne nicotine in the smoker's breathing zone and obtained the number of cigarettes smoked per day by each smoker. The data presented in Figure (1) of Marbury et al suggest that mothers under-reported their smoking rate by 50% (Marbury et al., 1993).

Table J.3-4 presents the Tcos for cancer and noncancer PAHs calculated using Eq. J-7. However, Zanieri and Del Bubba did not find measurable levels of some PAHs, particularly PAHs with 5 or 6 carbon rings, in milk from nonsmokers. In these cases, the concentration representing half the limit of detection (between 0.006-0.014 µg/kg) was used as the background concentration of the PAH in mother's milk.

There are two main limitations in the data provided in Table J.3-4. For some PAHs, no individual Tco was calculated because the concentration of the individual PAH was higher in mother's milk of nonsmokers than in smokers. For example, in column two of Table J.3-4, mother's milk benzo[b]fluoranthene, pyrene and anthracene have negative concentration values.

These discrepancies could be due to the natural variation in the ability of individuals to transfer inhaled PAHs to milk, or as Zaneiri et al. suggested, a result of greater exposure to certain PAHs in some foods compared to cigarette smoke. The small sample sets (n=11 for each group of smokers and nonsmokers) in the Zanieri study are less likely to represent the true mean in the study population and magnify the large variation in this biological response.

Additional uncertainties in the use of smokers to estimate PAH transfer coefficients include that fact that lung metabolism may be different in smokers because of the much higher doses of PAHs that smokers receive relative to those only exposed in ambient pollution. Cytochrome P-450 enzymes are known to be induced when exposure is greater and therefore metabolism could be proportionately greater in smokers. In addition, at higher dose levels some enzyme systems may become saturated which could alter the pattern of metabolism.

However, smokers are the best population for estimating PAH Tcos because the inhalation dose can be separated from background inhalation and dietary exposure, and the inhalation dose from the cigarettes can be estimated. OEHHA requested raw data from the investigators for individual women in the study, but unfortunately, only the summary statistics from the published paper were available to us.

Table J.3-4: Inhalation Transfer Coefficients (Tcos) for Individual PAHs with and without Potency factors from Geometric Mean and Standard Deviation Estimates (GM, GSD) of Human Milk (Cm) and Intake from Cigarettes (Ccig) (d/kg-milk)

PAH (no. of rings)[a]	Adjusted Cm (µg/kg wet wt.)	C_{cig} (µg/cig)	Inhalation Tco[b] (d/kg)
With Cancer Potency Factors	**GM, GSD**	**GM, GSD**	**GM, GSD**
Naphthalene (2)	2.78, 1.63	0.2798, 1.34	1, 2.66
Chrysene (4)	0.04, 5.34	0.0149, 1.12	0.28, 8.11
Benzo[a]anthracene (4)	0.20, 4.31	0.0149, 1.1	1.4, 6.52
Benzo[b]fluoranthene (5)	*-0.09, 5.01*	0.0099, 1.13	NA[c]
Benzo[k]fluoranthene (5)	0.05, 2.95	0.002, 1.22	0.26, 4.6
Benzo[a]pyrene (5)	0.26, 2.29	0.0092, 1.08	2.97, 3.45
Dibenzo[a,h]anthracene (5)	0.46, 3.85	0.0023, 1.08	2.11, 5.81
Indeno[1,2,3-c,d]pyrene (6)	0.16, 3.65	0.0035, 1.12	4.81, 5.54
Without Cancer Potency Factors	**GM, GSD**	**GM, GSD**	**GM, GSD**
Anthracene (3)	*-0.22, 6.29*	0.0426, 1.15	NA
Acenaphthylene (3)	*-4.56, 2.9*	0.0814, 1.22	NA
Phenanthrene (3)	2.00, 1.94	0.0035, 1.07	1.57, 2.92
Fluorene (3)	1.31, 4.1	0.1336, 1.09	0.75, 6.19
Acenaphthene (3)	2.48, 3.26	0.0613, 1.16	4.21, 5
Pyrene (4)	0.04, 4.57	0.0345, 1.34	0.12, 7.48
Fluoranthene (4)	1.63, 3.29	0.0555, 1.14	3.06, 5.02
Benzo[g,h,i]perylene (6)	0.77, 2.72	0.0023, 1.11	35.24, 4.13

[a] no. of rings, number of rings are an indicator of lipophilicity (greater # of rings, more likely to partition to body fat); [b] Sum of each PAH found in mother's milk microgram per kilogram (µg/kg) over the sum of the daily intake (µg/day) of the same PAH x 4.75 cigarettes/day x an adjustment factor of 2; [c] NA, not available because the concentration of PAH in mother's milk of smokers was lower than the concentration in nonsmokers, so an individual Tco could be calculated

Tco values for carcinogenic PAHs in Table J.3-4 are determined for all available PAHs and included in a summary estimate (see Table J.3-7 near the end of this section).

Unlike the other PAHs with cancer potency factors, naphthalene is not considered a multipathway chemical under the Hot Spots program because it is regarded as a gas, and therefore not subject to appreciable deposition onto soil, etc. Naphthalene was included in this analysis because this PAH constitutes a large proportion of the total mass of PAHs inhaled. Among the carcinogenic PAHs in Table J.3-4, naphthalene predominates in both mainstream smoke (63% of total carcinogenic PAHs) and in mother's milk (56% of total carcinogenic PAHs). Naphthalene is also the only PAH that

is considered a gas, and therefore, its physical properties are different from other larger PAHs that are semi-volatile or exist primarily as a solid. In spite of these differences, the summary estimate did not change when naphthalene was excluded in the analysis (summary Tco = 1.55 versus 1.60).

Due to few measurable levels of carcinogenic PAHs in milk samples, there is more uncertainty in the carcinogenic PAH Tco compared to the PAH Tco for PAHs without cancer potency values. Nevertheless, summary estimates for PAH Tcos from inhaled sources differ by less than a factor of two (Tco for carcinogens, 1.2 versus Tco without cancer potency values, 2.06) suggesting that there may be no systematic difference between these two groups of chemicals. Therefore, OEHHA combined individual Tcos for PAHs from both groups into an overall inhalation Tco (see Table J.3-7 and Figure J.3-1 at the end of this section of the Appendix). In Figure J.3-1, the top seven estimates of inhalation Tcos are carcinogenic PAHs and the bottom six estimates are PAHs without cancer potency values.

The combined estimate is the summary of all 13 PAH estimates combined using a Random-effects model. OEHHA assumes that the PAHs found in exposure studies are a subgroup from a population of PAHs. Random-effects models assume there are multiple central estimates and incorporate a between-PAH estimate of error as well as a within-PAH estimate of error. In contrast, a Fixed-effects model assumes observations scatter about one central estimate (Kleinbaum, 1988).

OEHHA recommends using the inhalation Tco based on the summary estimates provided in Table J.3-7 rather than using the individual PAH Tcos values provided in Table J.3-4, to assess transfer of individual inhaled PAHs to mother's milk. There are a high number of non-detects and small sample sizes in these data. The estimation of PAH Tco values with this method might be improved with more sensitive methods for measurement of breast milk PAH content and larger study populations to better estimate biological variation and estimates of PAH transfer from air to mother's milk. Such improved data could allow for a robust determination of the Tco values for individual compounds.

The key assumption underlying the development of these Tcos is that the variability in individual PAHs Tcos is sufficiently small to justify the use of an average value for individual PAH congeners. This approach appears to be the best available given the available studies.

J.3.2 Oral Biotransfer of PAHs to Mother's Milk

Diet is the largest contributor by pathway to total PAH intake from ubiquitous background sources for the general public and other situations where airborne levels are not remarkably high (Lioy et al., 1988). In a risk assessment of a reference nonsmoking male, a mean total PAH intake of 3.12 µg/d was estimated of which dietary intake was 96.2%, air 1.6%, water 0.2% and soil 0.4% (Menzie et al., 1992; Ramesh et al., 2004). Inhalation, soil ingestion and homegrown produce pathways can be important when considering total dose from a single stationary source. PAHs

contaminate homegrown produce and soil through direct deposition. Milk and meat from home-raised animals or commercial sources would be less of a contributor because many PAHs are highly metabolized by these animals following intake from contaminated pastures and soil.

There are no studies available that relate PAH dietary intake directly to mother's milk concentrations for these compounds, although studies of PAH dietary intake have been performed in several countries. Therefore, the PAH biotransfer efficiency to mother's milk from food was calculated using PAH dietary intake data and mother's milk PAH data from separate studies. OEHHA recognizes the uncertainty in this approach but it appears to be the best currently available. Table J.3-5 shows the daily dietary intake of carcinogenic PAHs from published studies of European residents.

Regional preferences, ethnicity, and individual dietary preferences will influence the amount of PAHs ingested with food. In addition, there were differences among the intake studies in the number and type of PAHs investigated in foods. Even though dietary habits and PAH analysis methods can result in different levels of PAH intake, the total dietary intakes of PAHs in each of five studies in Table J.3-5 were generally within an order of magnitude of each other.

Table J.3-5: Summary Estimates of PAHs with and without Cancer Potency Factors Dietary Intake (µg/day)

PAH (no. of rings[a])	**Italian** Lodovici et al (1995) Adults	**Dutch** De Vos et al. (1990)[c] Adult males	**Spanish** Marti-Cid et al. (2008) Adults	**Spanish** Falco et al. (2003) Adults	**U.K.** Dennis et al. (1983) Adults
With Cancer Potency Factors	**AM[b], SD**	**AM***	**AM***	**AM, SD**	**AM***
Naphthalene (2)	NA[d]	NA	1.846	0.823, 0.056	NA
Chrysene (4)	0.84, 0.0131	0.86 – 1.53	0.204	0.564, 0.037	0.5
Benzo[a]anthracene (4)	0.47, 0.0093	0.2 – 0.36	0.139	0.310, 0.021	0.22
Benzo[b]fluoranthene (5)	0.17, 0.0101	0.31 – 0.36	0.137	0.188, 0.014	0.18
Benzo[k]fluoranthene (5)	0.06, 0.0043	0.1 – 0.14	0.086	0.094, 0.006	0.06
Benzo[a]pyrene (5)	0.13, 0.0003	0.12 – 0.29	0.083	0.113, 0.008	0.25
Dibenzo[a,h]anthracene (5)	0.01, 0.0026	NDd[e]	0.084	0.048, 0.003	0.03
Indeno[1,2,3-c,d]pyrene (6)	ND	0.08 – 0.46	0.102	0.045, 0.003	ND
Without Cancer Potency Factors	**AM, SD**	**AM***	**AM***	**AM, SD**	**AM***
Anthracene (3)	NA	0.03 – 0.64	0.428	0.088, 0.006	NA
Acenaphthylene (3)	NA	NA	0.354	0.402, 0.026	NA
Phenanthrene (3)	NA	NA	3.568	2.062, 0.150	NA
Fluorene (3)	NA	NA	0.934	0.206, 0.017	NA
Acenaphthene (3)	NA	NA	0.368	0.071, 0.005	NA
Pyrene (4)	0.19, 0.0043	NA	1.084	1.273, 0.092	1.09
Fluoranthene (4)	1.03, 0.0106	0.99 – 1.66	1.446	0.848, 0.062	0.99
Benzo[g,h,i]perylene (6)	0.20, 0.0009	0.2 – 0.36	0.112	0.214, 0.017	0.21

[a] no. of rings, number of rings are an indicator of lipophilicity (greater # of rings, more likely to partition to body fat);
[b] Arithmetic mean (AM), Standard Deviation (SD);
[c] The Dutch dietary intakes were presented as the range of lower bound values (calculated by taking values below the detection limit to be zero) to upper bound values (calculated by taking values below the detection limit to be equal to the limit)
[d] *NA,* Not available; [e] ND, Not determined;
* no measure of variance was reported (Dennis et al., 1983a; Dennis et al., 1983b; De Vos et al., 1990; Lodovici et al., 1995; Falcó et al., 2003; Martí-Cid et al., 2008)

Based on the estimated intake of the same measured PAHs in dietary studies and the PAHs found in breast milk from nonsmoking mothers (Del Bubba et al., 2005; Zanieri et al., 2007), OEHHA was able to estimate transfer coefficients (Tco) by Equation J-8, a version of Equation J-1:

$$Tco_{hmoi} = Cm_{oi} / (D_{oi}) \quad \textbf{(Eq. J-8)}$$

where:

Cm_{oi} = geometric average ith PAH concentration in mother's milk (µg per kg milk as wet weight)
D_{oi} = geometric average dose of the ith PAH per day from dietary sources (µg/day)

Cm_{oi} is the geometric average of the ith PAH in whole milk from nonsmoking, rural dwelling women. OEHHA obtained estimates of GM and GSD by pooling and converting arithmetic estimates to geometric estimates of the mean and standard deviation from two studies of nonsmoking rural-dwelling women (Del Bubba et al., 2005; Zanieri et al., 2007). D_{oi} is the geometric average of the ith PAH taken in through dietary sources. Oral PAH Tcos for both carcinogenic and noncancer PAHs are shown in Table J.3-6.

The Italian dietary study by Lodovici et al. (1995) supplied data in which OEHHA could calculate estimates of dietary intake of nine PAHs among a population living mostly in urban settings. OEHHA obtained GM and GSD estimates by converting arithmetic estimates of dietary intake reported in Lodovici et al (1995) and estimates of intake variability from Buiatti et al (1989).

These investigators estimated that the entire study population consumes about 1.9 µg of carcinogenic PAHs per day from dietary sources. Approximately 46% of the total carcinogenic PAH intake comes from cereal products, non-barbecued meat, oils and fats. Even though meat barbecued on wood charcoal has the highest PAH levels, the contribution of these barbecued foods is only about 13% of the carcinogenic PAH intake.

A limitation of the Italian dietary intake study is that the population examined was 58% men, and the study did not report any body weight adjustments. Thus, the sample population may not represent the female population sampled by Zanieri et al (2007). Other studies that have compared dietary PAH intake levels between men and women indicate that men consume slightly higher levels of PAHs than women do (5% to 15% on a µg/kg-body weight-day basis) (Falco et al 2003, Marti-Cid et al 2008), so the bias introduced by this assumption may not be significant.

Table J.3-6 presents the dietary intake and mother's milk concentrations for individual PAHs from the Italian studies. OEHHA calculated Tcos for individual PAHs common to both the studies of dietary intake and mother's milk concentration. The mother's milk concentrations for individual PAHs represents the pooled average reported in the Zanieri et al. and Del Bubba et al. studies.

Table J.3-6: Oral Transfer Coefficients (Tcos) for Individual PAHs Based on Italian Data from a Daily PAH Dietary Intake Study (Lodovici et al., 1995; Del Bubba et al., 2005; Zanieri et al., 2007) and Mother's Milk PAH Concentration Studies (Del Bubba et al., 2005; Zanieri et al., 2007).

PAH	Mother's milk PAH concentration (µg/kg-milk)	Daily PAH intake (µg/d)	Oral PAH Tco (d/kg)
With Cancer Potency Factors	GM[a], GSD[b]	GM, GSD	GM, GSD
Naphthalene	4.12, 1.41	NA[c]	NA
Chrysene	0.01, 3.36	0.49, 2.82	0.02, 4.93
Benzo[a]anthracene	0.12, 5.41	0.27, 2.82	0.44, 7.25
Benzo[b]fluoranthene	0.21, 3.61	0.1, 2.82	2.1, 5.21
Benzo[k]fluoranthene	0.055, 3.01	0.034, 2.82	1.62, 4.54
Benzo[a]pyrene	0.01, 3.36	0.076, 2.82	0.13, 4.93
Dibenzo[a,h]anthracene	0.007, 3.36	0.003, 2.82	2.33, 4.93
Indeno[1,2,3-c,d]pyrene	0.011, 3.36	NA	NA
Without Cancer Potency Factors	GM, GSD	GM, GSD	GM, GSD
Anthracene	0.13, 4.26	NA	NA
Acenaphthylene	4, 1.99	NA	NA
Phenanthrene	0.41, 2.03	NA	NA
Fluorene	0.12, 6.32	NA	NA
Acenaphthene	1.39, 2.16	NA	NA
Pyrene	0.15, 3.47	0.11, 2.82	1.35, 5.05
Fluoranthene	0.16, 3.34	0.6, 2.82	0.27, 4.91
Benzo[g,h,i]perylene	0.01, 3.37	0.116, 2.82	0.08, 4.94

[a] GM, geometric mean; [b]GSD, geometric standard deviation; [c] NA, Not available;

Oral Tcos were calculated for each individual PAH by equation J-8. The average Tco for carcinogenic and PAHs without cancer potency factors was calculated as the sum of the Tco values over the total number of PAHs evaluated. Similar Tco values are obtained for both groups of PAHs (0.46 d/kg) and 0.31 d/kg, respectively). This finding suggests that, on average, the PAHs with cancer potency factors as a whole transfer to mother's milk with about the same efficiency as some of the most common PAHs without cancer potency factors that are taken in through the diet.

Summary Tcos were calculated using a Random-effects model to pool across individual PAH-Tcos. OEHHA found no systematic difference between summary estimates stratified by PAHs with or without cancer potency factors (data not shown). Therefore, we pooled Tcos for both groups by route of intake (see Table J.3-7).

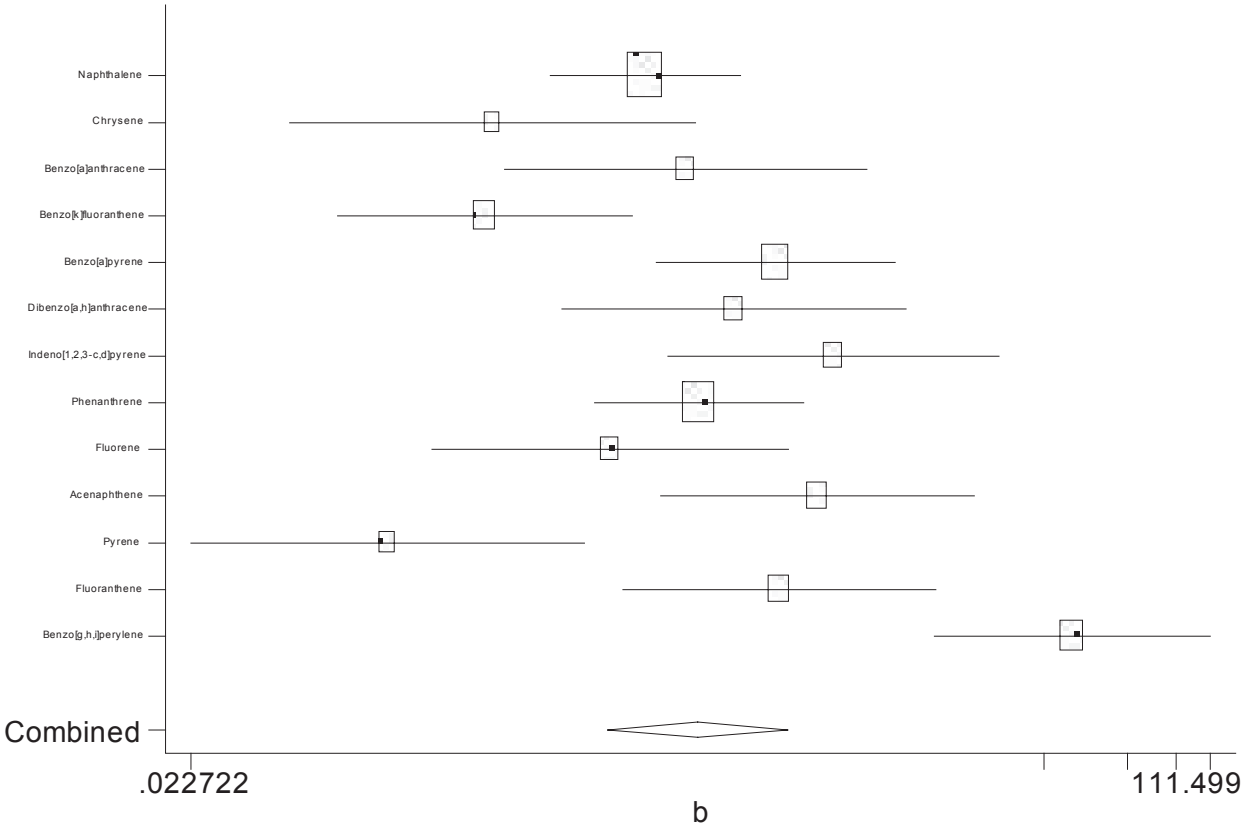
Table J.3-7: Random Effects Estimate and 95% Confidence Intervals of Tcos Stratified by Intake Route and Data Source

Tco (data source)	No. PAHs	summary estimate (random effects model)	LCL	UCL
Inhalation	13	1.55	0.731	3.281
Oral (Italian)	9	0.401	0.132	1.218

LCL, lower 95% confidence limit of the mean Tco;
UCL, upper 95% confidence limit of the mean Tco.

Similar to the inhalation Tco derivation, limitations of the oral Tco derivations include the small number of women examined for PAHs in mother's milk (n=21) and the large number of "below detection limit" results for milk concentrations, particularly for the larger PAHs with more than four rings. OEHHA assumed that the arithmetic estimates, minimum and maximum values reported by investigators represented a lognormal distribution and converted estimates from arithmetic to geometric. Nevertheless, the use of sparse data to derive an inhalation Tco and data from potentially two different study populations to generate an oral Tco – one for dietary PAH intake and another for mother's milk PAH concentrations - introduces considerable uncertainty.

Figure J.3-1: Inhalation Tcos (b, 95% CL) Based on Italian Data, (Random-effects Model)



The top seven estimates are PAHs with potency factors and bottom six estimates are PAHs without potency factors; summary of all 13 PAHs is labeled “combined” = 1.55 d/kg; b, the Tco in units of day/kg-milk

J.3.3 Comparison and Use of Inhalation and Oral PAH Tcos

Comparison of the oral and inhalation Tcos also presents a number of interesting findings. For example, comparing the averaged inhalation and oral mother's milk Tcos generated from the Italian studies for carcinogenic PAHs, the mean inhalation Tco is about four times greater than the oral Tcos based on Italian study data.

Although studies in humans are lacking, (Grova et al., 2002) showed that BaP is poorly absorbed through the gut in goats when administered orally in vegetable oil. Radiolabeled BaP fed to these animals led to 88% recovery of the radioactivity in feces, indicating little BaP reached the bloodstream where it could be taken up in mother's milk. In contrast, respiratory absorption of PAHs in particulate form through smoking is about 75% efficient (Van Rooij et al., 1994).

The following factors may have influenced the difference between oral Tco values and inhalation Tco values:

- First-pass metabolism in the liver following oral intake before reaching the blood supply of the breast versus entering systemic blood circulation prior to passage through the liver with the inhalation route (however, some PAH metabolism occurs in the lung)
- Gut assimilation of PAHs is likely to occur at a different rate than the rate of passage across the lung

Looking at mother's milk Tcos in terms of carryover rate suggests that accumulation of PAHs in the mother's body occurs more readily when inhaled versus ingested. Carryover rate, defined here as the daily output of PAHs in mother's milk (µg/day) over the daily intake of PAHs (µg/day), can be estimated by multiplying a PAH Tco by the daily output of mother's milk. Since milk production in human mothers are about 1.0 kg/day, the calculated carryover rate turns out to be the same as the PAH Tco value. A carryover rate greater than one in PAH transfer suggests that accumulation occurs in the mother's body prior to lactation.

The average inhalation Tco of 1.6 d/kg daily inhalation of a PAH mixture, indicates that 160% of the daily intake from inhaled sources transfers to mother's milk. This high transfer-value suggests that some accumulation of PAHs with cancer potency factors may occur in the mother's body before lactation begins. An average oral Tco of 0.40 d/kg for PAHs with cancer potency factors indicates 40% of the daily intake from diet transfers to mother's milk following oral intake of PAHs.

This suggests that metabolism occurs in the mother's body. The uncertainties in our Tco estimation methods could account for both of these results. If the Tco estimation is correct, the mother may be metabolizing a considerable fraction of her intake prior to partitioning into the fat stores. There could also be inefficient transfer to mother's milk for unknown reasons or metabolism following transfer of PAHs to mother's milk.

J.4 Mothers' Milk Transfer Coefficients for Inorganic Lead

Inorganic lead is naturally present on the earth's crust and may enter terrestrial and aquatic ecosystems due to the weathering of rocks. Traces of lead can not only be found in the immediate vicinity of emission sources but also are present, albeit at very low levels, in every part of the world (Castellino and Castellino, 1995).

Lead particulate matter is the primary form of lead present in the air (OEHHA 1997). Atmospheric movements may transport lead aerosol in the form of very fine particles, a long way from its place of emission. Refineries, mineral extraction industries, and smelting plants for lead and other metals are largely responsible for emitting lead-containing aerosols into the atmosphere (Castellino and Castellino, 1995) in the U.S.

Human intake of lead can occur by inhalation of airborne particles and ingestion of lead-contaminated food and water. Furthermore, people can be exposed using lead-glazed or painted cooking and eating utensils. Lead may also be ingested in foods or drinks contaminated with the metal during the industrial processes of food production or preservation (Castellino and Castellino, 1995). The potential pathways of concern with Hot Spots facilities would be inhalation, soil ingestion, and dermal absorption, home raised meat, homegrown produce, surface drinking water consumption, and breast milk consumption.

Background levels of lead in the blood of the U.S. population have declined in recent years mainly resulting from the removal of lead from gasoline and paint. Results from an NHANES study (1991 – 1994) show that the geometric mean blood lead level in the U.S. adult population (20 – 69 years of age) was about 4 µg/dL (Pirkle et al., 1994), which is over a 70% decline in blood lead from blood lead levels obtained from 1976 to 1980. The NHANES IV survey (1999- 2000) found an additional 50% reduction (1.75 µg/dL) in the U.S. adult population (CDC, 2005).

As of the date of this report, measured levels of lead at ambient air quality monitoring sites in California are very low. Lead exposure in the California population is likely to occur from sources other than Hot Spots facility emissions, such as old lead-based paint. However, no threshold has been identified for lead-induced neurotoxicity in children and therefore an evaluation of all potential routes of exposure for Hot Spots facilities is prudent. Further, there are significant lead emissions from some Hot Spots facilities.

In an effort to derive lactation transfer coefficients for inorganic lead, OEHHA drew from studies conducted on subjects exposed to lead through multiple pathways at higher levels from other areas of the world. OEHHA assumes that the transfer of lead derived from these studies serves as a reasonable surrogate for the transfer of lead from contaminated media near a Hot Spots facility in California.

J.4.1 Inorganic Lead in Human Milk

Breast milk levels of lead correlate with levels of lead in whole blood but are generally much lower (Sternowsky and Wessolowski, 1985; Castellino and Castellino, 1995; Li et al., 2000; Ettinger et al., 2004). Castellino et al (1995) reviewed 11 studies conducted between 1933 to 1989 and observed that in the vast majority of cases, the mean values of lead in breast milk vary from 0.17 to 5.6 µg/L (Castellino and Castellino, 1995).

Ursinyova and Masamova (2005) published a table of 32 human milk summary estimates from studies published between 1983 and 2001. Mean human milk levels of lead generally ranged from 0.5 to 50 µg/L (Ursinyova and Masanova, 2005). Average blood lead levels during that timeframe ranged from 24 to 460 (µg/L) (Gulson et al., 1998a).

Because lead levels in milk correlate well with whole blood, OEHHA searched for studies that reported both lead levels in milk and blood before and/or during lactation for derivation of a lactational Tco for lead. However, several investigators have questioned high results from early studies of lead in breast milk. For example, Ettinger et al (2004), Gulson (1998b) and others cautioned that high levels of lead in breast milk might be due to contamination from some past sample collection techniques (Hu et al., 1996; Newman, 1997; Gulson et al., 1998a; Smith et al., 1998; Ettinger et al., 2004). These sources of lead include the use of the following products to prepare nipples or express breast milk:

- lead acetate ointment
- lead in nipple shields
- lead in alcohol wipes from foil wrap

Gulson et al (1998a) also suggested that analytical problems, indicated by an unusually wide range in lead concentrations for the quality control standard in Parr et al (1991), warrant verification by follow-up studies (Parr et al., 1991; Gulson et al., 1998a). Gulson et al (1998a) assessed lead concentrations in maternal blood versus the concentration of lead in breast milk per concentration in maternal whole blood from studies conducted over 15 years prior to 1998. From this assessment, they suggested that milk lead levels less than about 15% of maternal blood lead levels best represent the relationship between lead in maternal blood and milk. In other words, milk lead levels that were greater than 15% of blood lead levels were suspected of being contaminated with lead during sample collection and/or assessment. Therefore, OEHHA has included only summary estimates from studies published after 1990 that did not report or show evidence of breast milk contamination.

OEHHA located eight studies that met our inclusion criteria. Table J.4-1 summarizes key attributes of the study populations.

Table J.4-1: Studies with Summary Estimates of Concurrent Maternal Blood and Milk Levels of Lead)

Study	Country	Group	Study period	Measurement	# Study subjects
(Nashashibi et al., 1999)	Greece	Residents of Athens and surrounding areas	~1999	At delivery, at onset of lactation	47
(Li et al., 2000)	China, Shanghai	Not occupationally exposed	prior to 2000	At delivery, at onset of lactation	32
(Counter et al., 2004)	Equador, Pujili	Pottery glazers	2003	Post partum	13
(Ettinger et al., 2004)	Mexico, Mexico City	Exclusive breast feeders	1994-1995	One month postpartum	88
(Ettinger et al., 2004)	Mexico, Mexico City	Partial breast feeders	1994-1995	One month postpartum	165
(Namihira et al., 1993)	Mexico, Mexico City	Reside near New Smelter	1986	postpartum	35
(Hallen et al., 1995)	Sweden	Reside in Rural areas	1990-1992	6 weeks postpartum	39
(Hallen et al., 1995)	Sweden	Reside near Smelter area	1990-1992	6 weeks postpartum	35
(Baum and Shannon, 1996)	U.S.A Camden, New Jersey	Mothers of lead poisoned infants	1996	Postpartum	2
(Gulson et al., 1998b)	Australia	Immigrants from eastern Europe	Early 1990s	At delivery and average during lactation	9

Regression analyses suggest a linear relationship between lead in maternal blood and milk among women with substantially elevated levels of lead in blood. For example, Namihira et al (1993) reported a significant linear relationship (r = 0.88) between levels of lead in blood and milk for blood lead levels in the range of 35 µg/dL -100 µg/dL from a study of 35 lactating women living in Mexico City (Namihira et al., 1993). At these levels of lead in blood, authors reported a univariate regression of 4.3% representing the average level of lead in breast milk relative to the average level of lead in blood.

A similar study of 47 lactating women conducted by Nashashibi et al also reported a significant linear relationship (r=0.77) between lead in milk and blood for blood lead levels in the range of 5 µg/dL - 25 µg/dL (Nashashibi et al., 1999). Based on a univariate regression, the average level of lead in breast milk was about 7% the average level of lead in blood. OEHHA calculated similar estimates of the milk/blood lead ratio from Li et al (2000), Counter et al (2002) and Ettinger et al (2004) (see Table J.4-2).

Table J.4-2 Concurrent Measurements of the Lead Concentration (µg/L) in Mother's Milk and Blood

Study	N	Blood AM,SD	Milk AM,SD	Blood GM,GSD	Milk GM,GSD
(Nashashibi et al., 1999)	47	149, 41.1	20,5	143.64, 1.31	19.4, 1.28
(Li et al., 2000)	119	142.5, 69.14	5.63,4.39	128.21, 1.58	4.44, 1.99
(Counter et al., 2004)	13	171, 91	4.6,5.3	150.96, 1.65	3.02, 2.51
(Ettinger et al., 2004)	88[a]	94, 48	1.4,1.1	83.72, 1.62	1.1, 2
(Ettinger et al., 2004)	165[b]	95, 43	1.5,1.2	86.55, 1.54	1.17, 2.02
(Namihira et al., 1993)	35	459, 198.8	29.94,25.75	421.19, 1.51	24.7, 1.86
(Hallen et al., 1995)	39[c]	31.4, 6.7	0.5,0.3**	30.71, 1.23	0.43, 1.74
(Hallen et al., 1995)	35[d]	31.7, 10.2	0.9,0.4***	30.18, 1.37	0.82, 1.53
(Baum and Shannon, 1996)	2	315, 35.4	5.02,0.50	313.03, 1.12	5, 1.1
(Gulson et al., 1998b)	9	29, 8	0.73,0.7	27.96, 1.31	0.53, 2.24

[a]exclusively breast fed; [b] partially breast fed; [c] rural setting; [d] near smelter; * < LOD taken as 1/2 LOD as GM and 9.9 = max, **based on LOD of 0.5 µg/L and 2 out of 39 samples above LOD; *** based on 16/35 above LOD

Li et al. (2000) stratified milk lead levels by low, medium and high blood lead levels. Their findings suggest that slightly higher transfer rates occur at low levels relative to high levels of lead in blood (Li et al., 2000). This may be due to more efficient transfer rates at lower body burdens of lead or it could result from very slight breast milk contamination during collection and/or assessment.

J.4.2 Biotransfer from Bone to Blood during Pregnancy and Lactation

Lead transferred from blood to human milk reflects both the mother's current and ongoing intake of lead exposure as well as lead mobilized due to physiological changes of pregnancy and lactation from bone stores due to past exposures. Several studies provided indications of internal transfer of lead from bone stores. Internal transfer was evident by comparing the rise in blood lead levels during lactation to blood lead levels measured prior to lactation (see Table J.4-3).

Table J.4-3: Change in Blood Lead Levels from Pregnancy (bloodpreg) to Lactation (bloodlac) (µg/L)

Study	N	Bloodpreg	Bloodlac	Bloodpreg	Bloodlac
		AM,SD	AM,SD	GM,GSD	GM,GSD
(Gulson et al., 1997)**	8	22.4, 6	32, 8.4	21.64, 1.30	30.95, 1.29
(Ettinger et al., 2004)	~86-88 excl	81, 38	94, 48	73.33, 1.56	83.72, 1.62
(Ettinger et al., 2004)	164-165 part	90, 44	95, 43	80.85, 1.59	86.55, 1.54
(Tellez-Rojo et al., 2002)	425	84, 40	93.7, 43.04	75.84, 1.57	85.15, 1.55
(Sowers et al., 2002)*	15	13.7, 7.75	17, 5.29	11.93, 1.69	16.23, 1.36
(Rothenberg et al., 2000)	311	27.59, 26.49	32.03, 21.78	22, 1.96	28, 1.68

* SD for blood lead level during lactation estimated for blood lead at 6-months from figure 2;
** bloodlact is max blood lead level during pregnancy and lactation;
excl, exclusively breastfed part, partially breastfed.

These investigators conducted longitudinal monitoring of blood samples to determine stable lead isotope profiles by mass spectrometry and chemical analyses of blood samples for total lead content over a 300-day period. Gulson et al followed Australian women (15 immigrants and 7 non-immigrants) to study the mobilization of lead from the maternal skeleton during pregnancy and lactation (Gulson et al., 1995; Gulson et al., 1997; Gulson et al., 1998a; Gulson et al., 1998b; Gulson et al., 1999; Gulson et al., 2001). Investigators measured maternal and infant blood, urine, diet, and breast milk from 21 mothers and 24 infants. The arithmetic mean and standard deviation lead concentration in breast milk were AM (SD) 0.73 (0.70) µg/kg and the geometric mean and standard deviation were GM (GSD) 0.55 (2.24) respectively. Levels ranged from 0.09 to 3.1 µg/kg.

Gulson et al (1997) provided evidence that lead in female immigrants to Australia was mobilized from skeletal stores during pregnancy, with increases in blood lead concentration of about 20% and a mean increase in skeletal lead contribution to blood lead of 31%. Authors concluded that between 45% and 70% of lead in blood comes from mobilized long-term tissue lead stores (Gulson et al., 1997).

Investigators obtained environmental samples of house dust, drinking water, urban air, gasoline, and a 6-day duplicate diet quarterly. The GM (GSD) blood lead concentration for the immigrant females on arrival in Australia (either prior to or during early pregnancy) was 3.0 µg/dL (SD 1.56) (range: 1.9 to 20 µg/dL) and for the Australian controls was 3.1 µg/dL (range: 1.9 to 4.3 µg/dL). Skeletal lead contribution to blood lead was significantly greater ($p< 0.001$) during the post pregnancy period than during the 2nd and 3rd trimesters.

The contribution of skeletal lead to blood lead during the post-pregnancy period remained constant at the increased level even though the duration of breast-feeding varied from 1 week to 6 months. The authors concluded that the increased contribution of skeletal lead both during pregnancy and in the post pregnancy period is consistent with increased bone resorption and may be associated with inadequate calcium intake.

Sowers et al (2000) followed lactating women enrolled in prenatal program located in Camden, New Jersey between 1997 and 2000 (Sowers et al., 2002). These women were part of a larger cohort of 962 women enrolled in study of calcium metabolism in pregnancy and lactation. A nested cohort of 15 women with a mean (standard deviation) age of 23.7 (5.42) years, who provided breast milk samples through 6 months postpartum or longer and were unaware of their blood lead levels, was included in the study. Blood and milk lead levels along with measures of bone loss and osteocalcin concentrations were evaluated. Authors reported the precautions taken to avoid contamination of milk samples by environmental lead.

The arithmetic mean (standard deviation) (µg/dL) of blood lead levels at delivery for 15 breast-feeding and 30 randomly selected bottle-feeding women were 1.37 (0.78) and 1.31 (1.10) respectively. Mean maternal blood lead levels rose to 1.6, (1.7) µg/dL at three and six months during lactation, respectively. Compared to bottle-feeding women, blood lead levels from breast-feeding women were consistently higher by 15 – 35% during the first six months postpartum. Authors found that breast-feeding women had greater bone loss as reflected in the bone change data and higher serum osteocalcin concentrations than bottle-feeding women.

The arithmetic mean of lead in breast milk samples (standard deviation) were 5.6 (4.2) and 5.9 (3.87) µg/L at three and six months post partum. Breast milk lead was also measured 1.5 and 12 months post partum. However, authors did not measure blood lead at 1.5 months, did not indicate how many women were still breast-feeding and did not attempt to estimate how many liters/day study subjects produced. The relative increase in blood lead levels from delivery to an active lactating period (e.g. one to 6 months) is consistent with the relative increases in blood lead found in other studies (see Table J.4-3).

Tellez-Rojo et al (2002) concluded that maternal bone lead levels are an important predictor of maternal blood lead levels over the course of lactation. In fact, bone lead from past exposures can contribute an additional 40% of the lead measured in blood during lactation (see Table J.4-3) (Tellez-Rojo et al., 2002).

Ettinger et al (2004) measured relatively high maternal blood lead levels in women exposed to lead in the air while living in Mexico City. Between January 1994 and June 1995, investigators selected 1398 women from three maternity hospitals in Mexico City for participation in a randomized control trial (Tellez-Rojo et al., 2002; Hernandez-Avila et al., 2003; Ettinger et al., 2004). From this study population, 629 women agreed to participate. Ettinger et al. (2004) examined a nested cohort of 255 women with a mean (standard deviation) age of 24 (5) years with both breast milk, maternal and infant blood lead levels at delivery and one-month post partum. The authors reported the precautions taken to avoid contamination of milk samples by environmental lead.

For breast-feeding women, the arithmetic mean (standard deviation) of blood lead level at delivery was 8.7 (4.2) and at one-month post partum was 9.4 (4.5) µg/dL. At one-month post partum, the average (standard deviation) lead level in breast milk was 1.5 (1.2) µg/L. After adjusting for parity, calcium intake, infant weight change and breastfeeding status, an increase in blood lead was associated with a 33% increase in breast milk lead.

Rothenberg et al (2000) recruited immigrant women, almost exclusively from Latin America, from outpatient clinics in South Central Los Angeles to examine bone lead contribution to blood lead. Investigators contacted subjects from June 1995 through July 1998. Three hundred eleven subjects were followed from late pregnancy to one or two months after delivery. The investigators evaluated bone lead levels after delivery and blood lead levels both pre- and post-delivery. Ages ranged from 15 to 44 years. Prenatal blood lead was lower on average GM = 2.2 µg/dL (0.4 to 38.7) than postnatal blood lead GM = 2.8 µg/dL (0.4 to 25.4). In fact, postnatal blood lead level increased by 27% relative to the prenatal blood lead level.

A questionnaire was administered including questions about present breast feeding practice (presently nursing yes/no) and past history of breast feeding (ever nursed and total months nursed). Breast milk samples were not obtained from this cohort. Tibia and calcaneus bone lead levels were associated with prenatal blood lead levels and calcaneus but not tibia lead was associated with postnatal blood lead levels (Rothenberg et al., 2000).

J.4.3 Inhalation Biotransfer of Lead to Mother's Milk

Ideally, lead transfer to human milk would include estimates of lead in ambient air and major sources of oral exposure over time along with human milk estimates from the exposed lactating population. However, few studies have attempted to correlate lead exposure from multiple pathways (e.g. oral sources such as contaminated food, water, dust and soil and inhalation sources such as ambient air) with lead concentrations in human mother's milk. This is likely due to the multiple effects of daily intake from environmental sources (Sannolo et al., 1995) and internal transfer from lead released from bone stores during pregnancy and lactation (Gulson et al., 1997).

Although exposure to lead can come from many sources, ambient air contaminated from combustion sources has been a significant source of exposure in the U.S.

population and European countries (U.S. EPA 1998). The relationship between air lead and blood lead has been studied extensively in both field studies and experimental chamber studies. OEHHA evaluated studies conducted prior to 1997 in their health risk assessment of inorganic lead under the toxic air contaminant program (OEHHA, 1997).

Briefly, in the OEHHA report, the contribution of airborne lead to blood lead levels was examined using several different methods – disaggregate, aggregate, uptake biokinetic, and physiologically based pharmaco-kinetic models (OEHHA, 1997). Findings were evaluated for linearity over a wide range of air and blood lead levels and are expected to apply to some exposure scenarios under the Hot Spots program. Most of these studies were conducted prior to 1985 when both air and blood lead levels were much higher than they are now. For example, the level of lead in the air used in chamber studies was 3.2 $\mu g/m^3$ representing low exposure and 10.9 $\mu g/m^3$ representing high exposure, while background air was typically between 7 $\mu g/m^3$ and 8 $\mu g/m^3$ in the city of Los Angeles during similar time-periods – late 1960s / early 1970s. Lead in Los Angeles air is 100-fold lower today (Ospital et al., 2008).

The relationship between air lead concentration and blood lead is not linear. Higher slopes are observed at lower air lead concentrations. However, the aggregate model was chosen because it implicitly incorporates all air-related pathways (i.e. soil, dust, water, contaminated food, etc.) and has averaged slopes estimated from a wide range of air concentrations. Using this model OEHHA estimated that an average change of 1.8 µg/dL in adult blood lead levels ($\mu g/m^3$) per $\mu g/m^3$ air lead concentration with current ambient air levels in California.

As part of our effort to estimate a lactational transfer factor for lead (Tco), we searched for studies that examined slope factors in other populations or were conducted subsequent to our 1997 report (OEHHA, 1997).

In addition to the kinetics of lead in the general adult population, recent studies have observed that - under similar exposure conditions - plasma lead rises by about 20% – 80% during lactation (Gulson et al., 1997; Gulson et al., 1998b; Gulson et al., 1999; Rothenberg et al., 2000; Tellez-Rojo et al., 2002). Findings from these and other investigations suggest that, in addition to daily environmental sources of exposure, breast milk levels of lead also reflect lead released from lead accumulated in the lactating woman's bones.

We were not able to locate studies that measured both long-term exposure to ambient air lead and lead levels in breast milk. Therefore, we calculated estimates of transfer from blood to human milk from separate study populations to combine with estimates of lead transfer from air to blood.

J.4.4 Population Transfer Coefficient (Tco) for Lead

OEHHA has derived transfer coefficients for lead using Equation J-9

$$Tco_{hma}=(Cma/C_{blood}^{+})x(C_{blood}^{+}/C_{blood})x(C_{blood}/(C_{air} \times BR))xF_{c1} \times F_{c2} \quad \textbf{Eq. J-9}$$

where:

Cma = geometric mean human milk lead level (µg/L-milk as wet weight)
C_{blood}^{+} = geometric mean blood lead level during lactation (µg/dL)
C_{blood} = geometric mean blood lead level during non-lactating state (µg/dL)
C_{air} = geometric mean concentration of lead in ambient air (µg/m^3)
BR = geometric mean breathing rate for adult women (14 m^3/day)
F_{c1} = conversion factor (L-milk)/(kg-milk) ~ **(**0.97**)**
F_{c2} = conversion factor (dL)/(L) = 10

Cm_a is the geometric mean human milk lead level that incorporates all (aggregated) air-related pathways of lead. C_{blood}^{+} is the geometric mean blood lead level among lactating women measured during lactation (µg/L). C_{blood} is the geometric mean blood lead level taken from the general population during a non-lactating state (µg/L). C_{air} is the geometric mean concentration of lead in the ambient air (µg/m^3) inhaled by the same population where blood lead levels were measured. BR is the geometric mean breathing rate for adult women (14 m^3/day) (see Chapter 2). F_{c1} is the inverse of the specific gravity of breast milk (1.03 g/ml)(Sergen, 2006). F_{c2} is the conversion from deciliters to liters.

J.4.4.1 Biotransfer from Blood to Milk

Three groups measured maternal blood lead before and during lactation along with lead in mother's milk (Gulson et al., 1997; Gulson et al., 1998a; Gulson et al., 1998b; Sowers et al., 2002; Ettinger et al., 2004). However, Sowers et al. reported unusually high levels of lead in breast milk relative to blood, which suggest contamination problems. It is possible that breast milk samples were contaminated by the sampling collection technique (e.g. lead in the nipple shields). However, it is also possible that a more efficient active transport mechanism at lower blood lead levels could explain higher levels of lead in breast milk relative to blood. More studies of mothers with low blood lead levels are needed to further verify the results reported by Sowers et al.

For our purposes, Gulson et al (1995, 1997, 1998a, 1998b) and Ettinger et al (2004) provide the best estimates of the change in blood lead levels before the onset of lactation, during lactation and relative to the levels of lead in breast milk (Gulson et al., 1997; Gulson et al., 1998a; Gulson et al., 1998b; Ettinger et al., 2004).

J.4.4.2 Transfer from Air to Blood

Equation J-10 describes estimation of aggregate transfer from airborne and associated sources that appears in the OEHHA 1997 report on the health effects of airborne inorganic lead (OEHHA, 1997):

Slope factor = $(C_{bloode} - C_{bloodr})/(C_{aire} - C_{airr})$ **Eq.-J-10**

$(C_{bloode} - C_{bloodr})$ is the difference between lead concentration in the blood of exposed compared to reference group and $(C_{aire} - C_{airr})$ is the difference in air lead between exposed and reference group. This simplified model assumes that the exposed and reference communities are similar in confounders such as age and smoking habits and reasonably comparable in their exposure to other sources of lead (e.g. paint).

Subsequent to OEHHA's 1997 report, Ranft et al (2008) published results from studies conducted on exposure to air pollutants among residents living near industrial sources along the rivers Rhine, Ruhr and Wupper in North Rhine-Westphalia Germany during five time-periods from 1983 to 2000. Authors reported the distribution of ambient air lead levels for each of the five time-periods (Ranft et al., 2008).

During the early years (1983 – 1991), ambient air lead levels ranged from 0.100 – 0.510 $\mu g/m^3$. Whereas, during the later years (1997 – 2000), air lead levels were much more variable - ranging from 0.025 to 0.729 $\mu g/m^3$. The 50^{th} percentile (P 50) declined by almost a factor of 20 from years 1983 to 2000. During the earliest years (1983 – 1991), P 50 declined by a factor of four from 0.465 to 0.100 $\mu g/m^3$. Based on data collected from 1991 to 2000, these investigators reported that childhood blood lead would decrease by a factor of 6.4: 95%CI (6.02 – 6.80) from the decrease in lead concentration in polluted ambient air (m^3/dL).

OEHHA calculated a similar slope factor from the study of 500, 55-yr-old women living in industrial areas of the North Rhine – Westphalia, Germany from 1985 to 1990 by Wilhelm and associates (Wilhelm et al., 2007). The investigators reported that mean blood lead levels among these women declined from 7.2 to 5.0 µg/dL. Based on ambient air levels of lead reported in Ranft et al (2008), OEHHA estimated that blood lead levels in 55-year old women would change by 6-fold per unit of change in ambient air levels of lead (µg/dL) over a similar period (GM, 6.2; 95% CI 6.1 – 6.4) (Ranft et al., 2008). This estimate is within the range of slope factors reported previously by OEHHA for the general adult population (OEHHA, 1997).

J.4.4.3 Transfer from Air and Body Stores to Milk

Tables J.4-4 and J.4-5 show the Tcos derived by combining air to blood and blood to milk transfer of inorganic lead from the available data. Table J.4-4 shows the transfer factors derived from the study of eight women who provided samples of blood before and during lactation as well as samples of milk during lactation (Gulson et al., 1998a; Gulson et al., 1998b). The geometric mean and standard deviation blood lead levels prior to lactation were low (GM 2.2 µg/dL, GSD1.3).

Table J.4-4: Transfer Coefficients (Tcos) for Inorganic Lead Measured in Human Blood and Milk (d/kg-milk) from Data Reported in (Gulson et al., 1998a; Gulson et al., 1998b) and the Change in Blood Lead with the Change in Lead Concentration Measured in Ambient Air (slope factor)

Source	Slope factor m^3/dL	Tco (d/kg milk) GM	GSD	LCL	UCL
OEHHA	1.8	0.024	3.19	0.009	0.061
Willhelm/Ranft	6.2	0.08	3.19	0.031	0.203

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

Table J.4-5 shows the transfer factors derived from the study of 253 women who provided samples of blood prior-to and during lactation as well as samples of milk during lactation (Ettinger et al., 2004).

Table J.4-5: Biotransfer Coefficients (Tcos) for Inorganic Lead Measured in Human Blood and Milk (d/kg-milk) from Data Reported in (Ettinger et al., 2004) and the Change in Blood Lead with the Change in Lead Concentration Measured in Ambient Air (slope factor)

Source	Slope factor m^3/dL	Tco (d/kg milk) GM	GSD	LCL	UCL
OEHHA	1.8	0.019	3.00	0.017	0.022
Willhelm/Ranft	6.2	0.064	3.00	0.056	0.074

LCL, lower 95% confidence limit of the mean Tco; UCL, upper 95% confidence limit of the mean Tco

Compared to Gulson et al (1998), the geometric mean, blood lead levels prior to lactation observed by Ettinger et al (2004) were about 4-fold higher (7.3 and 8.0 for exclusive and partial lactators, respectively)(Gulson et al., 1998b; Ettinger et al., 2004). However, the transfer factors derived from residents of Mexico and immigrants to Australia differ by less than a factor of two.

J.4.5 Study Limitations, Influencing Factors and Uncertainty (inorganic compounds)

Our Tco estimate for lead has not considered the influence of maternal age, parity, length of lactation, and body weight on concentration of lead in milk.

J.5 Summary and Recommendations

This appendix develops lactational transfer coefficients for use in estimating the concentration of a multipathway chemical in mother's milk from an estimate of chronic incremental daily dose to the mother from local stationary sources. OEHHA derived human lactational transfer coefficients from studies that measured contaminants in human milk and daily intake from inhalation or oral exposure (e.g. air, cigarette smoke or diet) in the same or a similar human population. These coefficients can be applied to the mother's chronic daily dose estimated by the Hot Spots exposure model to estimate a chemical concentration in her milk.

We established transfer coefficients (Tcos) for individual congeners and WHO-TEQ summary PCDDs/Fs and dioxin-like-PCBs, individual and summary carcinogenic PAHs, and lead through equations J-1-3, data on exposure and breast milk contamination from background (global), accidental and occupational sources, and a set of simplifying assumptions. We assume that a mother's intake and elimination is constant before lactation. We also assume that changes in a woman's body due to the onset of lactation occur as a single shift in elimination rate over the lactation period. In some cases, OEHHA adjusted some measurements of human milk and contaminant intake to account for confounding factors. In such cases, OEHHA describes the method of adjustment in the text and table containing adjusted values.

We described the methods for deriving specific Tcos from measurements of human milk, intake and transfer estimates from studies of populations exposed to general global sources of pollutants. Although the proportional contribution from various exposure pathways to total exposure from a single Hot Spots facility is likely to be quite different from exposure found with global sources, we believe Tcos in this appendix have been derived from data that serve as reasonable surrogates of transfer from Hot Spot facility exposures.

J.5.1 Dioxins and Furans

Personal factors such as body fat, smoking status and past lactation practices can affect body burden and elimination rates. For example, smoking has been associated with a 30% to 100% increase in elimination rates of some dioxin congeners (Milbrath et al. 2009, Flesch-Janys et al. 1996). As well, the onset of lactation sets a new elimination pathway into effect and can substantially reduce the maternal body burden of PCBs during 6 months of lactation (Niessen et al.1984, Landrigan et al. 2002).

Therefore, OEHHA incorporated conservative assumptions regarding these factors into our model (i.e. reference half-lives based on body burden below 700 ppt in the blood, adult age, nonsmoker, no recent prior breast-feeding period and percent body fat of older adults) in addition to accounting for the substantial variability between individual congeners of PCDDs, PCDFs and dioxin-like PCBs.

To calculate oral Tcos, OEHHA used adjusted reference half-lives for the chemicals in the adult human body derived from dietary and occupational exposures. OEHHA

estimated oral Tcos for these chemicals from estimates of body weight reported in Chapter 10 of this document, the steady-state equation developed by Smith (1987) and reference half-lives reported in Milbrath et al (2009). Milbrath et al (2009) adjusted reference half-lives for age, body fat, smoking habits and breast-feeding status as these factors were all strong determinants of half-life in humans.

A carryover rate > 1 would suggest that dioxins and dioxin-like compounds could accumulate in body fat and transfer to the fat in mother's milk. An average dioxin Tco of 3.7 d/kg indicates that 370% of the daily intake from ingested sources transfers to mother's milk. This high transfer-value suggests that some accumulation of carcinogenic dioxins and dioxin-like compounds occurs in the mother's body. For individual congeners, an oral Tco less than one (e.g. 1,2,3,4,7,8-HxCDF and 2,3,4,6,7,8-HxCDF) suggests that some metabolism occurs in the mother's body.

J.5.2 PAHs

Based on the estimated intake of 16 measured PAHs in simulated smoking studies and the PAHs found in breast milk from long-time smoking mothers (Zanieri et al. 2007), OEHHA was able to estimate transfer coefficients (Tco) with a modified version of Equation J-1.

The key assumption underlying the development of these Tcos is that the variability in an individual PAHs Tcos is sufficiently small to justify the use of an average value for individual PAH congeners. This approach appears to be the best available given the available studies.

OEHHA calculated oral Tcos for each individual PAH by Equation J-8. The average Tco for carcinogenic and PAHs without cancer potency factors was calculated as the sum of the Tco values over the total number of PAHs evaluated. Similar Tco values are obtained for both groups of PAHs (0.46 d/kg) and 0.31 d/kg, respectively). This finding suggests that, on average, the PAHs with cancer potency factors as a whole transfer to mother's milk with about the same efficiency as some of the most common PAHs without cancer potency factors that are taken in through the diet. Therefore, summary Tcos were calculated by pooling across individual PAH-Tcos from both groups (see Table J.3-7).

J.5.3 Inorganic Lead

In an effort to derive lactational transfer coefficients for inorganic lead, OEHHA has drawn from studies conducted on subjects exposed to lead through multiple pathways at higher levels from other areas of the world. OEHHA assumes that the transfer of lead derived from these studies serves as a reasonable surrogate for the transfer of lead from contaminated media near a Hot Spots facility in California.

We were not able to locate studies that measured both long-term exposure to ambient air lead and lead levels in breast milk. Therefore, we calculated estimates of transfer from blood to human milk from separate study populations to combine with estimates of lead transfer from air to blood.

For our purposes, Gulson et al (1995, 1997, 1998a, 1998b) and Ettinger et al (2004) provide the best estimates of the change in blood lead levels due to the onset of lactation as well as during lactation relative to the levels of lead in breast milk.

Based on ambient air levels of lead reported in Ranft et al (2008), OEHHA estimated that blood lead levels in 55-year old women would change by 6-fold per unit of change in ambient air levels of lead (µg/dL) over a similar period (GM, 6.2; 95% CL 6.1 – 6.4).

Compared to Gulson et al (1998), the geometric mean blood lead levels prior to lactation observed by Ettinger et al (2004) were about 4-fold higher (7.3 and 8.0 for exclusive and partial lactators, respectively) (Gulson et al., 1998b; Ettinger et al., 2004).

The transfer factors derived from residents of Mexico and immigrants to Australia differ by less than a factor of two. However, our Tco estimate for lead has not considered the influence of maternal age, parity, length of lactation, and body weight on concentration of lead in milk.

J.5.4 Recommendations

OEHHA recommends using the Tcos based on the summary estimates provided in Table J.1-1 rather than the individual compound Tcos provided in Tables J.2-3, J.3-4, and J.3-6 to assess transfer of compounds to mother's milk. Tcos of individual compound are less robust that summary Tcos listed in Table J.1-1 because in some cases they have derived from data containing a high number of non-detects and small sample sizes. Additional studies might improve the estimation of individual Tco values, especially studies that incorporate more sensitive methods for analyzing breast milk PAH content and larger study populations to better estimate biological variation and estimates of PAH transfer from air to mother's milk. Such improved data could allow for a robust determination of the Tco values for individual compounds (see Table J.1-1).

Table J.1-1: Default Tcos (d/kg) for Mother's Milk

Chemical/chem. group	Tco	LCL	UCL
PCDDs - oral	3.7	2.68	5.23
PCDFs - oral	1.8	1.27	2.43
Dioxin-like PCBs - oral	1.7	0.69	4.40
PAHs – inhalation	1.55	0.731	3.281
PAHs – oral	0.401	0.132	1.218
Lead - inhalation	0.064	0.056	0.074

LCL, lower 95% confidence interval of the mean Tco; UCL, upper 95% confidence interval of the mean Tco

When calculating cancer risk from speciated PCDD/Fs, dioxin-like PCBs and PAHs, assume that the ratios of congeners measured in the emissions are preserved when transferred from the mother's body to breast milk. OEHHA recommends a single Tco for each chemical group (e.g. PCDDs oral). Risk assessors can apply TEQs to the

infant dose after applying the Tco for a chemical group to each congener in the group to calculate infant cancer risk for the mother's milk pathway.

The mother's exposure from multiple pathways should be included in estimating the concentration of contaminant in mother's milk. One key factor that plays a role in the difference between oral and inhalation transfer coefficient (e.g., for PAHs) is first pass metabolism which is lacking in dermal and inhalation exposures. Thus, for simplicity, OEHHA recommends applying the transfer coefficients from inhalation to the dermal absorption pathway for lead and PAHs. For lead, we recommend using the inhalation Tco for all the other pathways of exposure to the mother. Likewise for PCDD/Fs and dioxin-like PCBs, we recommend using the oral Tco for the other pathways of exposure to the mother in Eq. J-2.

J.6 References

Abadin HG, Hibbs BF, Pohl HR (1997). Breast-feeding exposure of infants to cadmium, lead, and mercury: a public health viewpoint. Toxicol Ind Health 13(4): 495-517.

Abraham K., Hille A., Ende M. and Helge H. (1994). Intake and fecal excretion of PCDDs, PCDFs, HCB and PCBs (138, 153, 180) in a breast-fed and a formula-fed infant. Chemosphere 29(9-11): 2279-86.

Abraham K., Knoll A., Ende M., Papke O. and Helge H. (1996). Intake, fecal excretion, and body burden of polychlorinated dibenzo-p-dioxins and dibenzofurans in breast-fed and formula-fed infants. Pediatr Res 40(5): 671-9.

Abraham K., Papke O., Gross A., Kordonouri O., Wiegand S., Wahn U. and Helge H. (1998). Time course of PCDD/PCDF/PCB concentrations in breast-feeding mothers and their infants. Chemosphere 37(9-12): 1731-41.

Albers J.M.C., Kreis I.A., Liem A.K. and van Zoonen P. (1996). Factors that influence the leel of contamination of human milk with poly-chlorinated organic compounds. Arch Environ Contam Toxicol 30: 285-291.

Alcock R.E., Bacon J., Bardget R.D., Beck A.J., Haygarth P.M., Lee R.G., Parker C.A. and Jones K.C. (1996). Persistence and fate of polychlorinated biphenyls (PCBs) in sewage sludge-amended agricultural soils. Environ Pollut 93(1): 83-92.

Barr D.B., Wang R.Y. and Needham L.L. (2005). Biologic monitoring of exposure to environmental chemicals throughout the life stages: requirements and issues for consideration for the National Children's Study. Environ Health Perspect 113(8): 1083-91.

Baum C.R. and Shannon M.W. (1996). Lead in breast milk. Pediatrics 97(6 Pt 1): 932.

Bowes S.G. and Renwick A.G. (1986). The hepatic metabolism and biliary excretion of benzo[a]pyrene in guinea-pigs fed normal, high-fat or high-cholesterol diets. Xenobiotica 16(6): 531-42.

Buser H.R. and Rappe C. (1984). Isomer-specific separation of 2378-substituted polychlorinated dibenzo-p -dioxins by high-resolution gas chromatography mass spectrometry. Anal Chem 56(3): 442-448.

Castellino N. and Castellino P. (1995). Lead metabolism. In: Inorganic lead exposure metabolism and intoxication. Castellino N. CRC Press, Inc. Bota Raton, Fl.

CDC (2005). Blood lead levels--United States, 1999-2002. MMWR Morb Mortal Wkly Rep 54(20): 513-6.

Chao H.R., Wang S.L., Lee C.C., Yu H.Y., Lu Y.K. and Päpke O. (2004). Level of polychlorinated dibenzo-p-dioxins, dibenzofurans and biphenyls (PCDD/Fs, PCBs) in human milk and the input to infant body burden. Food Chem Toxicol 42(8): 1299-1308.

Chao H.R., Wang S.L., Lin L.Y., Yu H.Y., Lu Y.K., Chou W.L., Guo Y.L. and Chang L.W. (2003). Polychlorinated biphenyls in taiwanese primipara human milk and associated factors. Bull Environ Contam Toxicol 70(6): 1097-103.

Costera A., Feidt C., Dziurla M.A., Monteau F., Le Bizec B. and Rychen G. (2009). Bioavailability of polycyclic aromatic hydrocarbons (PAHs) from soil and hay matrices in lactating goats. J Agric Food Chem 57(12): 5352-7.

Counter S.A., Buchanan L.H. and Ortega F. (2004). Current pediatric and maternal lead levels in blood and breast milk in Andean inhabitants of a lead-glazing enclave. J Occup Environ Med 46(9): 967-73.

De Vos R.H., Van Dokkum W., Schouten A. and De Jong-Berkhout P. (1990). Polycyclic aromatic hydrocarbons in Dutch total diet samples (1984-1986). Food Chem Toxicol 28(4): 263-8.

Del Bubba M., Zanieri L., Galvan P., Donzelli G.P., Checchini L. and Lepri L. (2005). Determination of polycyclic aromatic hydrocarbons (PAHs) and total fats in human milk. Ann Chim 95(9-10): 629-41.

Dennis J.M., Massey R.C., McWeeny D.J. and Watson D.H. (1983a). Polycyclic aromatic hydrocarbons in the U.K. diet. Polynucl. Aromat. Hydrocarbons, Int. Symp., 7th: 405-12.

Dennis M.J., Massey R.C., McWeeny D.J., Knowles M.E. and Watson D. (1983b). Analysis of poly cyclic aromatic hydro carbons in UK total diets. Food Chem Toxicol 21(5): 569-74.

Ding Y.S., Trommel J.S., Yan X.J., Ashley D. and Watson C.H. (2005). Determination of 14 polycyclic aromatic hydrocarbons in mainstream smoke from domestic cigarettes. Environ Sci Technol 39(2): 471-8.

Ettinger A.S., Tellez-Rojo M.M., Amarasiriwardena C., Gonzalez-Cossio T., Peterson K.E., Aro A., Hu H. and Hernandez-Avila M. (2004). Levels of lead in breast milk and their relation to maternal blood and bone lead levels at one month postpartum. Environ Health Perspect 112(8): 926-31.

Falcó G., Domingo J.L., Llobet J.M., Teixidó A., Casas C. and Müller L. (2003). Polycyclic aromatic hydrocarbons in foods: human exposure through the diet in Catalonia, Spain. J Food Prot 66(12): 2325-31.

Flesch-Janys D., Becher H., Gurn P., Jung D., Konietzko J., Manz A. and Papke O. (1996). Elimination of polychlorinated dibenzo-p-dioxins and dibenzofurans in occupationally exposed persons. J Toxicol Environ Health 47(4): 363-78.

Focant J.F., Eppe G., Pirard C., Massart A.C., André J.E. and De Pauw E. (2002). Levels and congener distributions of PCDDs, PCDFs and non-ortho PCBs in Belgian foodstuffs: Assessment of dietary intake. Chemosphere 48(2): 167-179.

Forehand J.B., Dooly G.L. and Moldoveanu S.C. (2000). Analysis of polycyclic aromatic hydrocarbons, phenols and aromatic amines in particulate phase cigarette smoke using simultaneous distillation and extraction as a sole sample clean-up step. J Chromatogr A 898(1): 111-24.

Furst P. (2006). Dioxins, polychlorinated biphenyls and other organohalogen compounds in human milk. Levels, correlations, trends and exposure through breastfeeding. Mol Nutr Food Res 50(10): 922-33.

Fürst P., Krause G.H.M., Hein D., Delschen T. and Wilmers K. (1993). PCDD/PCDF in cow's milk in relation to their levels in grass and soil. Chemosphere 27(8): 1349-1357.

Gmeiner G., Stehlik G. and Tausch H.J. (1997). Determination of seventeen polycyclic aromatic hydrocarbons in tobacco smoke condensate. J Chromatogr A 767: 163-69.

Graham H. and Owen L. (2003). Are there socioeconomic differentials in under-reporting of smoking in pregnancy? Tob Control 12(4): 434.

Grova N., Feidt C., Crepineau C., Laurent C., Lafargue P.E., Hachimi A. and Rychen G. (2002). Detection of polycyclic aromatic hydrocarbon levels in milk collected near potential contamination sources. J Agric Food Chem 50(16): 4640-2.

Gulson B.L., Jameson C.W., Mahaffey K.R., Mizon K.J., Korsch M.J. and Vimpani G. (1997). Pregnancy increases mobilization of lead from maternal skeleton. J Lab Clin Med 130(1): 51-62.

Gulson B.L., Jameson C.W., Mahaffey K.R., Mizon K.J., Patison N., Law A.J., Korsch M.J. and Salter M.A. (1998a). Relationships of lead in breast milk to lead in blood, urine, and diet of the infant and mother. Environ Health Perspect 106(10): 667-74.

Gulson B.L., Mahaffey K.R., Jameson C.W., Mizon K.J., Korsch M.J., Cameron M.A. and Eisman J.A. (1998b). Mobilization of lead from the skeleton during the postnatal period is larger than during pregnancy. J Lab Clin Med 131(4): 324-9.

Gulson B.L., Mahaffey K.R., Mizon K.J., Korsch M.J., Cameron M.A. and Vimpani G. (1995). Contribution of tissue lead to blood lead in adult female subjects based on stable lead isotope methods. J Lab Clin Med 125(6): 703-12.

Gulson B.L., Mizon K.J., Palmer J.M., Patison N., Law A.J., Korsch M.J., Mahaffey K.R. and Donnelly J.B. (2001). Longitudinal study of daily intake and excretion of lead in newly born infants. Environ Res 85(3): 232-45.

Gulson B.L., Pounds J.G., Mushak P., Thomas B.J., Gray B. and Korsch M.J. (1999). Estimation of cumulative lead releases (lead flux) from the maternal skeleton during pregnancy and lactation. J Lab Clin Med 134(6): 631-40.

Hallen I.P., Jorhem L., Lagerkvist B.J. and Oskarsson A. (1995). Lead and cadmium levels in human milk and blood. Sci Total Environ 166: 149-55.

Harden F.A., Toms L.M., Symons R., Furst P., Berry Y. and Muller J.F. (2007). Evaluation of dioxin-like chemicals in pooled human milk samples collected in Australia. Chemosphere 67(9): S325-33.

Hecht S.S., Grabowski W. and Groth K. (1979). Analysis of faeces for benzo[a]pyrene after consumption of charcoal-broiled beef by rats and humans. Food Cosmet Toxicol 17(3): 223-7.

Hedley A.J., Wong T.W., Hui L.L., Malisch R. and Nelson E.A. (2006). Breast milk dioxins in Hong Kong and Pearl River Delta. Environ Health Perspect 114(2): 202-8.

Hernandez-Avila M., Gonzalez-Cossio T., Hernandez-Avila J.E., Romieu I., Peterson K.E., Aro A., Palazuelos E. and Hu H. (2003). Dietary calcium supplements to lower blood lead levels in lactating women: a randomized placebo-controlled trial. Epidemiology 14(2): 206-12.

Hu H., Hashimoto D. and Besser M. (1996). Levels of lead in blood and bone of women giving birth in a Boston hospital. Arch Environ Health 51(1): 52-8.

Jensen A.A. (1987). Polychlorobiphenyls (PCBs), polychlorodibenzo-p-dioxins (PCDDs) and polychlorodibenzofurans (PCDFs) in human milk, blood and adipose tissue. Sci Total Environ 64(3): 259-93.

Kim S.R., Halden R.U. and Buckley T.J. (2008). Polycyclic aromatic hydrocarbons in human milk of nonsmoking U.S. women. Environ Sci Technol 42(7): 2663-7.

Kishikawa N., Wada M., Kuroda N., Akiyama S. and Nakashima K. (2003). Determination of polycyclic aromatic hydrocarbons in milk samples by high-performance liquid chromatography with fluorescence detection. J Chromatogr B Analyt Technol Biomed Life Sci 789(2): 257-64.

Kleinbaum D.G. (1988). Applied regression analysis and other multivariable methods. Duxbury series in statistics and decision sciences. Boston: PWS-Kent Pub. Co., c1988.

Kostyniak P.J., Stinson C., Greizerstein H.B., Vena J., Buck G. and Mendola P. (1999). Relation of Lake Ontario fish consumption, lifetime lactation, and parity to breast milk polychlorobiphenyl and pesticide concentrations. Environ Res 80(2 Pt 2): S166-S174.

LaKind J.S., Amina- Wilkins A. and Berlin C.M., Jr. (2004). Environmental chemicals in human milk: a review of levels, infant exposures and health, and guidance for future research. Toxicol Appl Pharmacol 198(2): 184-208.

LaKind J.S., Brent R.L., Dourson M.L., Kacew S., Koren G., Sonawane B., Tarzian A.J. and Uhl K. (2005). Human milk biomonitoring data: interpretation and risk assessment issues. J Toxicol Environ Health A 68(20): 1713-69.

Landrigan P.J., Sonawane B., Mattison D., McCally M. and Garg A. (2002). Chemical contaminants in breast milk and their impacts on children's health: an overview. Environ Health Perspect 110(6): A313-5.

Li J., Zhang L., Wu Y., Liu Y., Zhou P., Wen S., Liu J., Zhao Y. and Li X. (2009). A national survey of polychlorinated dioxins, furans (PCDD/Fs) and dioxin-like polychlorinated biphenyls (dl-PCBs) in human milk in China. Chemosphere 75(9): 1236-1242.

Li P.-J., Sheng Y.-Z., Wang Q.-Y., Gu L.-Y. and Wang Y.-L. (2000). Transfer of lead via placenta and breast milk in human. Biomed Environ Sci 13(2): 85-89.

Liem A.K., Furst P. and Rappe C. (2000). Exposure of populations to dioxins and related compounds. Food Addit Contam 17(4): 241-59.

Liem A.K.D., Albers J.M.C., Baumann R.A., van Beuzekom A.C., den Hartog R.S., Hoogerbrugge R., de Jong A.P.J.M. and Marsman J.A. (1995). RGBs, PCDDs, PCDFs and organochlorine pesticides in human milk in the Netherlands, levels and trends. Organohal Compounds 26: 69-74.

Lioy P.L., Waldman J.M., Greenberg A., Harkov R. and Pietarinen C. (1988). The Total Human Environmental Exposure Study (THEES) to benzo(a)pyrene: comparison of the inhalation and food pathways. Arch Environ Health 43(4): 304-12.

Lodovici M., Dolara P., Casalini C., Ciappellano S. and Testolin G. (1995). Polycyclic aromatic hydrocarbon contamination in the Italian diet. Food Addit Contam 12(5): 703-713.

Lorber M. and Phillips L. (2002). Infant exposure to dioxin-like compounds in breast milk. Environ Health Perspect 110(6): A325-32.

Marbury M.C., Hammond S.K. and Haley N.J. (1993). Measuring exposure to environmental tobacco smoke in studies of acute health effects. Am J Epidemiol 137(10): 1089-97.

Martí-Cid R., Bocio A. and Domingo J.L. (2008). Dietary exposure to PCDD/PCDFs by individuals living near a hazardous waste incinerator in Catalonia, Spain: Temporal trend. Chemosphere 70(9): 1588-1595.

McLachlan M., Thoma H., Reissinger M. and Hutzinger O. (1990). PCDD/F in an agricultural food chain. Part 1: PCDD/F mass balance of a lactating cow. Chemosphere 20(7-9): 1013-20.

Menzie C.A., Potocki B.B. and Santodonato J. (1992). Exposure to carcinogenic PAHs in the environment. Environ Sci Technol 26: 1278-84.

Milbrath M.O., Wenger Y., Chang C.W., Emond C., Garabrant D., Gillespie B.W. and Jolliet O. (2009). Apparent half-lives of dioxins, furans, and polychlorinated biphenyls as a function of age, body fat, smoking status, and breast-feeding. Environ Health Perspect 117(3): 417-25.

Mukerjee D. (1998). Health risk of endocrine-disrupting ortho-substituted PCBs emitted from incinerators. Environ Eng Sci 15(2): 157-169.

Mukerjee D. and Cleverly D.H. (1987). Risk from exposure to polychlorinated dibenzo p dioxins and dibenzofurans emitted from municipal incinerators. Waste Manage Res 5: 269-283.

Namihira D., Saldivar L., Pustilnik N., Carreon G.J. and Salinas M.E. (1993). Lead in human blood and milk from nursing women living near a smelter in Mexico City. J Toxicol Environ Health 38(3): 225-32.

Nashashibi N., Cardamakis E., Bolbos G. and Tzingounis V. (1999). Investigation of kinetic of lead during pregnancy and lactation. Gynecol Obstet Invest 48(3): 158-62.

Newman J. (1997). Caution regarding nipple shields. J Hum Lact 13(1): 12-3.

Ng Y.C. (1982). A review of transfer factors for assessing the dose from radionuclides in agricultural products. Nucl Safety 23(1): 57-71.

Niessen K.H., Ramolla J., Binder M., Brugmann G. and Hofmann U. (1984). Chlorinated hydrocarbons in adipose tissue of infants and toddlers: inventory and studies on their association with intake of mothers' milk. Eur J Pediatr 142(4): 238-44.

Oberg M., Sjodin A., Casabona H., Nordgren I., Klasson-Wehler E. and Hakansson H. (2002). Tissue distribution and half-lives of individual polychlorinated biphenyls and serum levels of 4-hydroxy-2,3,3',4',5-pentachlorobiphenyl in the rat. Toxicol Sci 70(2): 171-82.

OEHHA (1997). *Proposed Identification of Inorganic Lead as a Toxic Air Contaminant.* Part B: Health Effects Air Toxicology and Epidemiology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. http://www.arb.ca.gov/toxics/lead/tsdb.pdf.

OEHHA (2008). *The Air Toxics Hot Spots Program Risk Assessment Guidelines Part III: Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels*. Air Toxicology and Epidemiology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.

OEHHA (2009). *Air Toxics Hot Spots Program Risk Assessment Guidelines. Part II: Technical Support Document for Cancer Potency Factors: Methodologies for derivation,*

listing of available values and adjustments to allow for early life stage exposures. California Environmental Protection Agency.

Ogura I. (2004). Half-life of each dioxin and PCB congener in the human body. Organohalogen Compounds 66: 3329-3337.

Ospital J., Cassmassi J. and Chico T. (2008). *Multiple Air Toxics Exposure Study in the South Coast Air Basin MATES III Final Report*. South Coast Air Quality Management District. http://www.aqmd.gov/prdas/matesIII/Final/Appendices/f-MATESIIIAppendixVIFinal92008.pdf.

Parr R.M., DeMaeyer E., Iyengar V., Byrne A., Kirkbright G., Schoch G., Ninisto L., Pineda O., Vis H. and Hofvander Y. (1991). Minor and trace elements in human milk from Guatemala, Hunpary, Nigeria, Philippines, Sweden, and Zaire. Biol Trace Elem Res 29: 51-75.

Phillips D.H. (1999). Polycyclic aromatic hydrocarbons in the diet. Mutat Res 443(1-2): 139-47.

Pinsky P.F. and Lorber M.N. (1998). A model to evaluate past exposure to 2,3,7,8-TCDD. J Expo Anal Environ Epidemiol 8(2): 187-206.

Pirkle J.L., Brody D.J., Gunter E.W., Kramer R.A., Paschal D.C., Flegal K.M. and Matte T.D. (1994). The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys (NHANES). JAMA 272(4): 284-91.

Raab U., Preiss U., Albrecht M., Shahin N., Parlar H. and Fromme H. (2008). Concentrations of polybrominated diphenyl ethers, organochlorine compounds and nitro musks in mother's milk from Germany (Bavaria). Chemosphere 72(1): 87-94.

Ramesh A., Walker S.A., Hood D.B., Guillen M.D., Schneider K. and Weyand E.H. (2004). Bioavailability and risk assessment of orally ingested polycyclic aromatic hydrocarbons. Int J Toxicol 23(5): 301-33.

Ranft U., Delschen T., Machtolf M., Sugiri D. and Wilhelm M. (2008). Lead concentration in the blood of children and its association with lead in soil and ambient air" trends between 1983 and 2000 in Duisburg. J Toxicol Environ Health, Part A: Current Issues 71(11): 710 - 715.

Rappe C., Nygren M., Marklund S., Keller L.O., Bergqvist P.A. and Hansson M. (1985). Assessment of human exposure to polychlorinated dibenzofurans and dioxins. Environ Health Perspect 60: 303-4.

Rothenberg S.J., Khan F., Manalo M., Jiang J., Cuellar R., Reyes S., Acosta S., Jauregui M., Diaz M., Sanchez M., Todd A.C. and Johnson C. (2000). Maternal bone lead contribution to blood lead during and after pregnancy. Environ Res 82(1): 81-90.

Sannolo N., Carelli G., De Lorenzo G. and Castellino N. (1995). Environmental Exposure. Chapter 5. In: Inorganic Lead Exposure Metabolism and Intoxication. Castellino N., Castellino P. and Sannolo N. CRC Press, Inc. Boca Raton, Fl.

Sasamoto T., Horii S., Ibe A., Takada N. and Shirota K. (2006). Concentration changes of PCDDs, PCDFs, and dioxin-like PCBs in human breast milk samples as shown by a follow-up survey. Chemosphere 64(4): 642-649.

Schantz S.L., Jacobson J.L., Humphrey H.E., Jacobson S.W., Welch R. and Gasior D. (1994). Determinants of polychlorinated biphenyls (PCBs) in the sera of mothers and children from Michigan farms with PCB-contaminated silos. Arch Environ Health 49(6): 452-8.

Schecter A., Kassis I. and Päpke O. (1998). Partitioning of dioxins, dibenzofurans, and coplanar PCBS in blood, milk, adipose tissue, placenta and cord blood from five American women. Chemosphere 37(9-12): 1817-1823.

Scheele J., Teufel M. and Niessen K.H. (1995). A comparison of the concentrations of certain chlorinated hydrocarbons and polychlorinated biphenyls in bone marrow and fat tissue of children and their concentrations in breast milk. J Environ Pathol Toxicol Oncol 14(1): 11-4.

Sergen J., ed. (2006). Concise Dictionary of Modern Medicine. Breast milk. McGraw-Hill New York.

Smith A.H. (1987). Infant exposure assessment for breast milk dioxins and furans derived from waste incineration emissions. Risk Anal 7(3): 347-53.

Smith D.R., Ilustre R.P. and Osterloh J.D. (1998). Methodological considerations for the accurate determination of lead in human plasma and serum. Am J Ind Med 33(5): 430-8.

Sowers M.R., Scholl T.O., Hall G., Jannausch M.L., Kemp F.W., Li X. and Bogden J.D. (2002). Lead in breast milk and maternal bone turnover. Am J Obstet Gynecol 187(3): 770-6.

Sternowsky H.J. and Wessolowski R. (1985). Lead and cadmium in breast milk higher levels in urban vs. rural mothers during the 1st 3 months of actation. Arch Toxicol 57(1): 41-45.

Tellez-Rojo M.M., Hernandez-Avila M., Gonzalez-Cossio T., Romieu I., Aro A., Palazuelos E., Schwartz J. and Hu H. (2002). Impact of breastfeeding on the mobilization of lead from bone. Am J Epidemiol 155(5): 420-8.

Teufel M., Niessen K.H., Sartoris J., Brands W., Lochbuhler H., Waag K., Schweizer P. and von Oelsnitz G. (1990). Chlorinated hydrocarbons in fat tissue: analyses of residues in healthy children, tumor patients, and malformed children. Arch Environ Contam Toxicol 19(5): 646-52.

Todaka T., Hirakawa H., Kajiwara J., Hori T., Tobiishi K., Onozuka D., Kato S., Sasaki S., Nakajima S., Saijo Y., Sata F., Kishi R., Iida T. and Furue M. (2008). Concentrations of polychlorinated dibenzo-p-dioxins, polychlorinated dibenzofurans, and dioxin-like polychlorinated biphenyls in blood and breast milk collected from 60 mothers in Sapporo City, Japan. Chemosphere 72(8): 1152-1158.

Ursinyova M. and Masanova V. (2005). Cadmium, lead and mercury in human milk from Slovakia. Food Addit Contam 22(6): 579-89.

USEPA (1998). *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emisssions EPA 600/R-98/137 National Center for Environmental Assessment*

USEPA (2000). *Draft Dioxin Reassessment. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. National Center for Environmental Assessment*.

van der Molen G.W., Kooijman S.A. and Slob W. (1996). A generic toxicokinetic model for persistent lipophilic compounds in humans: an application to TCDD. Fundam Appl Toxicol 31(1): 83-94.

van Leeuwen F. and Malisch R. (2002). Results of the third round of the WHO coordinated exposure study on the levels of PCBs, PCDDs and PCDFs in human milk. Organohalogen Compounds 56: 311-316.

Van Rooij J.G.M., Veeger M.M.S., Bodelier-Bade M.M., Scheepers P.T.J. and Jongeneelen F.J. (1994). Smoking and dietary intake of polycyclic aromatic hydrocarbons as sources of interindividual variability in the baseline excretion of 1-hydroxypyrene in urine. Int Arch Occup Environ Health 66: 55-65.

Wang R.Y. and Needham L.L. (2007). Environmental Chemicals: From the Environment to Food, to Breast Milk, to the Infant. 8. Taylor; Francis.

West C.E. and Horton B.J. (1976). Transfer of polycyclic hydrocarbons from diet to milk in rats, rabbits and sheep. Life Sci 19(10): 1543-51.

WHO (1997). *Monographs on the Evaluation of Carcinogenic Risks To Humans. Polychlorinated Dibenzo- para-dioxins and Polychlorinated Dibenzofurans*. Volume 69. International Agency for Research on Cancer (IARC)

Wilhelm M., Ewers U., Wittsiepe J., Fürst P., Hölzer J., Eberwein G., Angerer J., Marczynski B. and Ranft U. (2007). Human biomonitoring studies in North Rhine-Westphalia, Germany. Int J Hyg Environ Health 210(3-4): 307-318.

Wittsiepe J., Fürst P., Schrey P., Lemm F., Kraft M., Eberwein G., Winneke G. and Wilhelm M. (2007). PCDD/F and dioxin-like PCB in human blood and milk from German mothers. Chemosphere 67(9): S286-S294.

Yakushiji T. (1988). Contamination, clearance, and transfer of PCB from human milk. Rev Environ Contam Toxicol 101: 139-64.

Zanieri L., Galvan P., Checchini L., Cincinelli A., Lepri L., Donzelli G.P. and Del Bubba M. (2007). Polycyclic aromatic hydrocarbons (PAHs) in human milk from Italian women: influence of cigarette smoking and residential area. Chemosphere 67(7): 1265-74.

Zhao G., Xu Y., Li W., Han G. and Ling B. (2007). PCBs and OCPs in human milk and selected foods from Luqiao and Pingqiao in Zhejiang, China. Sci Total Environ 378(3): 281-292.

Appendix K

Meat, Milk, and Egg Transfer Coefficients

K.1 Chemical Transfer Coefficient (Tco) Derivation Methodology

Meat, cow's milk and eggs can become contaminated when food-producing animals inhale or ingest contaminated materials that then transfer into these food products. The transfer coefficients (Tco) presented in Tables K.1 and K.2 were derived from published studies investigating chemical concentrations in food products resulting from animal intake of the chemical. In most studies, the chemicals were mixed into the animal's feed, although some studies investigated the bioaccumulation of chemicals from contaminated soil in poultry feed. The Tcos, expressed in day/kilogram (d/kg), represent the ratio of contaminant concentration in fresh weight animal product (in mg/kg, for example) to the daily intake of contaminant by the animal (in mg/day). Tcos were determined only for the main food-producing animal sources, including cow's milk, eggs, and meat from cattle, pigs and chickens.

The studies selected to estimate Tcos were usually of long enough duration to allow steady-state concentrations to be reached in milk-, meat- and egg-producing animals. Steady-state concentrations in the tissues are a function of the tissue elimination half-lives (MacLachlan and Bhula, 2008). Assuming a first-order process, an exposure duration that is five times greater than the tissue elimination half-life has been used to represent time to steady-state conditions (i.e., the ratio of the measured concentration at five half-lives to steady-state concentration is 0.968).

Realistically, fast-growing animals used for food may never attain a true tissue steady-state for persistent organic chemicals due to the competing factors of growth, fattening and lactation (Fries, 1996; Hoogenboom, 2005). A steady-state concentration in food-producing animals will likely be reached more quickly than in humans due to these factors and may even show declining levels in fat during the fattening phase of the animals' prior to slaughter (Fries, 1996). The most practical approach is to base the Tco on exposure studies that expose the animal for a majority of the animals' life span up to or near marketable weight. The studies that followed tissue and milk contaminant levels during exposures over most of the animals' productive lifespan have shown that a sufficient semblance of steady-state is reached during the productive life of lactating dairy cattle and laying hens, and in meat animals prior to slaughter.

Default consumption rates of contaminated feed were used for estimating Tcos if no consumption data were provided in the primary studies. Usually, the food-producing animals in biotransfer studies were caged or treated similar to commercial farming practices. However, this exposure assessment document is primarily concerned with small farm or family farm situations in which the food-producing animals may be allowed to roam more freely than in commercial operations. This is particularly relevant for pigs and chickens. Free-range and organic farming will result in greater feed intake, slower

growth, and potentially greater contaminant exposure from range forage and soil ingestion (MacLachlan, 2010).

Specifically regarding poultry food products, the term “poultry” refers to a number of avian species that are food sources for humans. Due to the substantial human consumption of eggs and meat from chickens, the Tcos described here were exclusively based on data from chickens, laying hens (usually Leghorns) for the egg Tcos and usually meat chickens (broilers) for the meat Tcos. However, these values could also be reasonably applied to other home-raised avian species, such as turkeys and quail.

Compared to chickens and dairy cattle, fewer swine and beef cattle exposure studies could be found to estimate the biotransfer of ingested contaminants to muscle tissue. Rather than simply adopting the same cattle Tco values for swine when biotransfer data are lacking, contaminant transfer models are employed by OEHHA to estimate differences in chemical accumulation among livestock. For transfer of organic lipophilic chemicals, MacLachlan (2009) developed Physiologically Based Pharmacokinetic (PBPK) models to derive scaling factors that are used to assist the extrapolation of transfer studies, carried out most often on lactating dairy cows, to beef cattle and pigs. Given the estimated half-life (or extraction ratio for liver) of the chemical in the animal and the ratio of the chemical concentration in milk fat to body fat of dairy cows, the appropriate scaling factor can be selected and combined with the Tco derived from lactating dairy cattle to improve estimates of residues in beef cattle and pigs.

For metal Tcos, a metabolic weight adjustment can be made that accounts for differences in tissue transfer of chemicals in animals of different weight (i.e., a lower metabolic rate is expected in larger animals such as cattle compared to smaller animals such as swine, resulting in slower rates of transfer into tissues). A similar metabolic weight approach has been used to estimate the transfer of metals to dairy cattle from data in sheep (Crout et al., 2004). This adjustment is reasonable considering most of the metal compounds of interest have passive uptake and elimination processes and are subject to little or no metabolism.

The effect of metabolic weight is apparent when comparing the meat Tco values between chicken and cattle in Tables K-1 and K-2. Where published data were used to directly estimate individual chemical Tco values, the chicken Tcos were greater than cattle Tcos. For chemicals in which biotransfer could not be estimated from published reports in pigs, a default meat Tco was estimated with the following formula:

$$\text{Pig } Tco_i = (W^{0.75}_{cow}) / (W^{0.75}_{pig}) \times \text{cow } Tco_i \qquad \text{Eq. K-1}$$

Where: $W^{0.75}_{cow}$ = live-weight in kg of a cow to the 0.75 power

$W^{0.75}_{pig}$ = live weight in kg of a pig to the 0.75 power

Pig Tco_i = pig meat Tco for chemical i

Cattle Tco_i = cow meat Tco for chemical i

Using average live weights of 500 kg for cattle and 60 kg for swine, the metabolic weight ratio adjustment is 4.8.

Table K.1 Meat, Milk and Egg Transfer Coefficients for Persistent Organic Chemicals

Organic Chemical	Tcos (d/kg)[a]				
	Cow's Milk	**Chicken Egg**	**Chicken Meat**	**Cattle Meat**	**Pig Meat**
Diethylhexylphthalate	9×10^{-5}	0.04	0.002	6×10^{-4}	5×10^{-4}
Hexachlorobenzene	0.02	20	10	0.2	0.08
Hexachlorocylcohexanes	0.01	7	5	0.2	0.09
PAH's	0.01	0.003	0.003	0.07	0.06
Pentachloropenol	[b]	[b]	[b]	[b]	[b]
PCB Congeners					
77	0.001	6	4	0.07	0.4
81	0.004	10	7	0.2	0.4
105	0.01	10	7	0.6	0.7
114	0.02	10	7	0.9	0.7
118	0.03	10	7	1	0.7
123	0.004	10	7	0.2	0.7
126	0.04	10	7	2	0.7
156	0.02	10	8	0.9	2
157	0.01	10	8	0.5	2
167	0.02	10	8	1	2
169	0.04	10	8	2	2
189	0.005	10	8	0.2	1
Unspeciated	0.01	10	7	0.2	0.5
PCDD/F's Congeners					
2378-TCDD	0.02	10	9	0.7	0.1
12378-PeCDD	0.01	10	9	0.3	0.09
123478-HxCDD	0.009	10	6	0.3	0.2
123678-HxCDD	0.01	10	6	0.4	0.1
123789-HxCDD	0.007	7	3	0.06	0.02
1234678-HpCDD	0.001	5	2	0.05	0.2
OCDD	0.0006	3	1	0.02	0.1
2378-TCDF	0.004	10	6	0.1	0.02
12378-PeCDF	0.004	30	10	0.1	0.01
23478-PeCDF	0.02	10	8	0.7	0.09
123478-HxCDF	0.009	10	5	0.3	0.1
123678-HxCDF	0.009	10	6	0.3	0.09
234678-HxCDF	0.008	5	3	0.3	0.06
123789-HxCDF	0.009	3	3	0.3	0.03
1234678-HpCDF	0.002	3	1	0.07	0.06
1234789-HpCDF	0.003	3	1	0.1	0.02
OCDF	0.002	1	0.6	0.02	0.03
Unspeciated	0.001	6	5	0.03	0.09

[a] All Tco values were rounded to the nearest whole number
[b] To be assessed for transfer to meat, milk and eggs

Table K.2 Meat, Milk and Egg Transfer Coefficients for Inorganic Metals and Chemicals

Inorganic Metals	Tcos (d/kg)[a]				
	Cow's Milk	Chicken Egg	Chicken Meat	Cattle Meat	Pig Meat
Arsenic	5×10^{-5}	0.07	0.03	2×10^{-3}	0.01[b]
Beryllium	9×10^{-7}	0.09	0.2	3×10^{-4}	0.001
Cadmium	5×10^{-6}	0.01	0.5	2×10^{-4}	0.005
Chromium (VI)	9×10^{-6}	NA[c]	NA	NA	NA
Fluoride	3×10^{-4}	0.008	0.03	8×10^{-4}	0.004[b]
Lead	6×10^{-5}	0.04	0.4	3×10^{-4}	0.001[b]
Mercury	7×10^{-5}	0.8	0.1	4×10^{-4}	0.002[b]
Nickel	3×10^{-5}	0.02	0.02	3×10^{-4}	0.001
Selenium	0.009	3	0.9	0.04	0.5

[a] All Tco values were rounded to the nearest whole number.
[b] The meat Tco was estimated using the metabolic weight adjustment ratio of 4.8 from cattle to pig
[c] NA – no data available or were not applicable

Speciated data existed that allowed the derivation of individual Tcos for polychlorinated biphenyls (PCBs) and polychlorinated dibenzo-p-dioxins and furans (PCDD/F), shown in Table K.1, that are of toxicological concern under the "Hot Spots" program. Tcos for unspeciated mixtures of PCBs and PCDD/Fs have also been calculated by OEHHA from literature sources and are shown in Table K.1. In risk assessments in which only the unspeciated mixture is determined, OEHHA recommends using the Tcos for PCB126 to represent the PCBs, and the Tcos for 2,3,7,8-TCDD to represent the PCDD/F's. These compounds are one of the most persistent and toxic congeners within their respective classes. The unspeciated Tco values in K.1 are for only comparison to the other Tco values. Different emissions sources of these chemicals may result in different mixtures of PCBs and PCDD/Fs, and thus influence the unspeciated Tco value.

K.2 Tco Derivations for Milk, Meat and Eggs

K.2.1 Semi- and Non-Volatile Organic Chemicals

The exposure studies used to derive organic compound Tcos often normalized the muscle tissue, egg and cow's milk contaminant concentrations to their respective fat content. The Tcos presented here are based on fresh, whole meat, egg and milk concentrations of the contaminants. If necessary, the fat concentration of a chemical was adjusted to the average fresh weight concentration using fat content default factors derived from reference sources: 0.11 for egg, 0.07 for chicken meat, 0.19 for beef cattle meat, 0.23 for pig meat, and 0.04 for cow's milk (Malisch et al., 1996; Pirard and De Pauw, 2005; U.S. EPA, 2005). If only the fat concentration of the organic chemical in egg yolk was provided in the key study, the fresh weight whole egg concentration was derived based on a fat content default value of 0.30 for yolk, and a yolk volume of 0.32

for the whole egg. If the study determined the fat content in food products, these were used for adjustment to fresh weight concentration in lieu of the default values.

For chicken meat, organic chemical content in skin was usually not included by the studies, although skin has a higher fat content and is often consumed with the meat. This would suggest that the skin could have a higher contaminant content than the muscle tissue. Due to lack of skin chemical concentration data and potential loss or destruction of organic chemicals in skin when the meat is cooked, the concentration of chemical in skin was considered similar to the concentration of a chemical in muscle for Tco derivation.

In general, extensive bioaccumulation of persistent, organic chemicals is not as great in either beef or dairy cattle as might be expected, even though beef cattle have no major fat excretion pathway as dairy cattle do with milk production (McLachlan, 1996). This finding is a result of the short life spans and rapid growth dilution that is characteristic of modern animal husbandry. A beef cow develops 100-150 kg of fat in which to deposit the chemical that it absorbs over its 1.5-year life. While a milk cow might excrete its absorbed contaminant in 300 kg of milk fat over the same period, it consumes more feed (and contaminant) in this time. Hence, the chemical concentrations in milk fat were not always much lower compared to beef fat (McLachlan, 1996; RTI, 2005).

Interestingly, the lower-than-expected bioaccumulation of persistent, hydrophobic chemicals in cow's milk does not translate to human milk (McLachlan, 1996). Persistent, organic chemicals tend to bioaccumulate in human milk by an order of magnitude greater than in cow's milk, presuming similar chemical concentrations in the diet on a mg/kg basis. This pronounced difference in bioaccumulation is due to a more limited capability of humans to excrete these chemicals. In addition, the extent of contaminant absorption from food in the human digestive tract may be greater. For example, nursing human infants absorb over 95% of PCBs and most PCDD/Fs while absorption in cows for these same compounds averages closer to 80%.

K.2.1.1 Diethylhexylphthalate (DEHP)

At high concentrations (1% DEHP in feed), Tcos for chicken eggs and breast muscle were estimated by OEHHA to be 0.04 and 0.002 d/kg (Ishida et al., 1981; Ishida, 1993). The low transfer values for DEHP relative to other organic chemicals are likely due to rapid metabolism and excretion of DEHP in the chicken.

In dairy cattle, DEHP was observed to be extensively metabolized prior to secretion into the milk (Bluthgen and Ruoff, 1998). OEHHA surmised that much of the metabolism begins in the rumen, where DEHP ester-bond cleavage would occur. Consequently, steady-state is reached in about 7 days and a low milk Tco of 9×10^{-5} d/kg was calculated by OEHHA. Cessation of DEHP administration resulted in nearly undetectable milk levels within 3 days post-exposure. No data could be found regarding residue levels of DEHP in cattle muscle, so a Tco of 4×10^{-4} d/kg was estimated after adjusting for the average fat content difference between cow's milk and cattle muscle. PBPK modeling by MacLachlan (2009) observed a ratio of about 1.5 for residues of

highly metabolized lipophilic compounds, such as DEHP, in body fat of non-lactating cows and steers to the same compound in body fat of lactating dairy cows. Thus, the Tco of 4×10^{-4} d/kg was increased by a factor of 1.5 to arrive at a Tco of 6×10^{-4} d/kg for DEHP in meat of beef cattle.

Bioaccumulation data are lacking for DEHP in pigs. Thus, a scaling factor by MacLachlan (2009) was applied for the transfer of lipophilic xenobiotics from lactating cattle to other livestock species. For chemicals such as DEHP that are extensively metabolized in the animal and have a short half-life ($t_{1/2}$ <5.8 d in lactating cows), the ratio of simulated residues in the body fat of pigs to the body fat of lactating dairy cows was essentially equal to 1. Therefore, the dairy cattle muscle Tco determined above (4×10^{-4} d/kg) was only adjusted for the difference in muscle fat content in pig to beef cattle (ratio = 1.2) to arrive at a default Tco of 5×10^{-4} d/kg for pig meat.

K.2.1.2 Hexachlorobenzene (HCB)

HCB in the atmosphere is predicted to be predominantly in the vapor phase (Lane et al., 1992). However, due to the extreme persistence of HCB and other chlorinated organic compounds in the environment, deposition and accumulation of non-volatile forms of these organics onto crops, soil and sediment are significant pathways of exposure (Eisenreich et al., 1981; Kelly et al., 1991; Douben et al., 1997; Horstmann and McLachlan, 1998).

In dairy cattle, two studies recorded nearly identical cow's milk HCB Tcos of 0.015-0.016 d/kg with 60-70 days of exposure (Fries and Marrow, 1976; Firestone et al., 1979). The data suggested near steady-state levels in milk were attained with this duration of exposure. A higher Tco of 0.030 d/kg was recorded in pregnant dairy cattle after about 8 months of exposure (Vreman et al., 1980). Steady-state was reached in milk of the pregnant dairy cattle after about 5 months. The average HCB Tco from these three studies is 0.02 d/kg.

In his review, Kan (1978) provided bioaccumulation data from which to calculate Tcos for HCB. The Tco for egg and chicken muscle were estimated at 16 and 13 d/kg, respectively.

In beef cattle, steady-state levels of HCB were at or near attainment in subcutaneous fat following ten weeks of exposure in the feed (Dingle and Palmer, 1977; RTI, 2005). A muscle Tco estimated from this study was 0.090 d/kg. Exposure to HCB in dairy cattle provided similar Tco values. A muscle Tco of 0.070 d/kg was calculated from HCB concentrations in body fat of lactating dairy cattle following 60 day exposure in the feed (Fries and Marrow, 1976). An eight-month HCB exposure in dairy cattle resulted in a muscle Tco of 0.16 d/kg (Vreman et al., 1980). Because the Vreman study provided a considerably longer exposure overall for cattle, the Tco was based on this study. The PBPK-based scaling factor data by MacLachlan (2009) was applied to estimate the transfer of HCB from lactating cattle to body fat of steers. Using data supplied by Fries and Marrow (1976), a slow elimination half-life of HCB in lactating dairy cattle (average: 50 days) and a small ratio for milk fat concentration over body fat concentration at

steady state (0.04) suggests that the PBPK-generated ratio of simulated HCB level in body fat of steers to body fat of lactating dairy cows would be about 1.5. The final default beef Tco is 0.24 d/kg (0.16 d/kg x 1.5)

No data for HCB accumulation in pig muscle tissue could be found. Therefore, a PBPK-based scaling factor was also applied to estimate the transfer of HCB from lactating cattle to pigs (MacLachlan, 2009). The PBPK model results generated a ratio of 0.5 for the simulated HCB level in body fat of pigs to body fat of lactating dairy cows. The final default pig Tco is 0.08 d/kg (0.16 d/kg x 0.5)

K.2.1.3 Hexachlorocyclohexanes (HCH)

HCH Tcos of 7.3 d/kg for egg and 5.1 d/kg for chicken meat were calculated from contaminated feed data provided by Kan (1978) and Szokolay et al. (1977). The beta-isomer tended to have roughly 10-fold greater bioaccumulation in poultry egg and muscle than the other major isomers (i.e., alpha and gamma isomers), but is generally found to a lesser extent in the environment. Hence, the Tcos represent a mean of the three major HCH isomers. MacLachlan (2008) developed a model that adequately reproduced the pattern of lindane (gamma-HCH) residue levels in fat and eggs of hens consuming contaminated feed. Utilizing the authors' data, the egg and muscle Tcos at steady-state were estimated to be 1.3 and 1.5 d/kg, respectively. These lindane Tcos were similar to those calculated from data by Kan (1978) and Szokolay et al. (1977) for eggs, 1.7 and 4.2 d/kg, respectively, and in muscle, 1.8 and 1.2 d/kg, respectively.

As in eggs and meat, the major isomers of HCH (alpha-, beta-, and gamma-HCH) had different patterns of accumulation in cow's milk. The beta isomer has the largest transfer factor, 0.025 d/kg, but generally is in the smallest proportion relative to the other 2 major isomers found in the environment (van den Hoek et al., 1975; Vreman et al., 1976; Vreman et al., 1980). Average Tco values for the alpha- and gamma- (Lindane) isomers were 0.0054 and 0.0014 d/kg, respectively (Williams and Mills, 1964; van den Hoek et al., 1975; Vreman et al., 1980; Surendra Nath et al., 2000). An average Tco for these three HCH isomers is 0.011 d/kg. Surendra Nath et al. (2000) provided data for the industrial grade HCH isomer mixture resulting in a Tco of 0.003 d/kg. The HCH mixture contained 21% gamma-HCH, but further speciation data were not included.

Vreman et al. (1980) fed dairy cows diets containing alpha- and beta-HCH for up to eight months. The calculated muscle Tcos were 0.045 and 0.19 d/kg for alpha- and beta-HCH, respectively. For lindane (gamma-HCH), a Tco of 0.027 d/kg was calculated from a different study following 12-week exposure in non-lactating dairy cattle (Claborn et al., 1960).

We applied a scaling factor by MacLachlan (2009) to estimate the transfer of HCHs from lactating cattle to beef cattle. Using data supplied by Vreman *et al.* (1980) that showed a cow's milk elimination half-life of 9-19 days for alpha- and beta-HCH, and the data by van den Hoek et al. (1975) that showed similar levels of HCH isomers in milk fat and body fat, the PBPK-generated ratio of simulated HCH levels in body fat of steers to body fat of lactating dairy cows is approximately 2. We multiplied the alpha- and beta-

HCH Tcos of 0.045 and 0.19 d/kg, respectively, which were determined in dairy cattle by the scaling factor of 2. The gamma-HCH Tco remained unchanged since non-lactating cows and steers have similar steady state HCH levels in body fat. The average Tco for these three isomers is 0.17 d/kg and is the recommended Tco for beef cattle.

No data for HCH accumulation in pig muscle tissue could be found, so we used a scaling factor by MacLachlan (2009) to estimate the transfer of HCHs from lactating cattle to pigs. Based on the HCH half-lives and milk fat to body fat ratios in dairy cattle discussed above, the PBPK-generated ratio of simulated HCH levels in body fat of pigs to body fat of lactating dairy cows is very close, or slightly greater, than 1. Thus, Tcos of the three isomers in lactating and non-lactating dairy cows were averaged by us and used as the default for pig meat (0.045 + 0.19 + 0.027 d/kg / 3 = 0.087 d/kg).

K.2.1.4 Polycyclic Aromatic Hydrocarbons (PAH)

Although there are a considerable number of studies investigating PAH exposure in the environment, there are surprisingly few studies that provide reliable data for estimating Tcos in food-producing animals. Exposure of fish, poultry and dairy cattle to a mixture of PAHs results in the presence of mainly low molecular weight PAHs (i.e., three or four cyclic rings) in the fat of meat and milk (Meador et al., 1995; Grova et al., 2000; Grova et al., 2002; Schaum et al., 2003; Lutz et al., 2006). Many of the high molecular weight PAHs with five or more cyclic rings, such as benzo[a]pyrene (BaP), are known carcinogens or possible carcinogens. Bioaccumulation of PAHs declines with increasing number of aromatic rings and the associated increase in K_{ow}, likely due to both lower gut assimilation efficiency and increased metabolism rate. Another factor appears to be that lower levels of the larger carcinogenic PAHs contaminate pastures and feed compared to the smaller PAHs, often resulting in animal milk and tissue concentrations below the detection limits of analysis equipment (EC, 2002). For example, Muhlemann et al. (2006) found that the larger carcinogenic PAHs in contaminated feed comprised only 8.3% of total PAHs, while the smaller PAHs of four rings or less contributed most of the remaining fraction.

Broiler chickens fed a diet containing low levels of PAHs found in de-inking paper sludge did not exhibit increased PAH levels in abdominal fat for nearly all carcinogenic PAHs examined (Beauchamp et al., 2002). However, the low molecular weight PAHs fluoranthene and pyrene showed increasing levels in abdominal fat with increasing levels of PAHs from paper sludge in the diet of broilers. The carcinogenic potential of these PAHs are undetermined, due to inadequate evidence of carcinogenicity in animals. The calculated broiler muscle Tco for total PAHs was 0.003 d/kg (due mainly to accumulation of pyrene and fluoranthene), and the individual PAH Tcos for pyrene and fluoranthene were 0.1 and 0.04 d/kg, respectively. The total PAH Tco of 0.003 d/kg was chosen as a poultry muscle default value for PAHs, as Tcos for the larger carcinogenic PAHs would likely not surpass this value. No data could be found for PAH accumulation in eggs. Thus, the poultry muscle Tco was also applied to the egg Tco.

The presence of PAHs in milk and milk products suggests that these foods can represent a significant part of human intake of PAHs (Schaum et al., 2003). Among PAHs, the lightest and least lipophilic ones, such as naphthalene, phenanthrene, fluoranthene and pyrene, are detected in the greatest amounts in milk from farms exposed to airborne PAHs (Grova et al., 2000; Grova et al., 2002; Cavret et al., 2005; Lutz et al., 2006). Higher molecular weight PAHs with more than four rings, including possible carcinogens or known carcinogens such as BaP, chrysene and benz[a]anthracene, have been largely undetectable in cow's milk. Of the larger carcinogenic and possibly carcinogenic PAHs, only benz[a]anthracene was detected in tank milk (pooled milk from many cows) sampled near several potential contamination sources (Grova et al., 2002). Levels of this PAH in milk fat ranged from 1.9-2.2 ng/g in milk fat (approximately 0.08-0.09 ng/g in whole milk).

Based on the pasture grass concentrations and corresponding cow's milk concentrations of the three most abundant PAHs (phenanthrene, anthracene, and pyrene) from 10 rural and urban farms investigated by Grova et al. (2000), the range of PAH Tco values in milk were 0.02 to 0.002 d/kg. However, some assumptions were made to arrive at this estimate, including pasture grass as the only source of ingested PAHs, and intake of pasture grass ranged between 10 to 100% of the cow's diet.

A cow's milk Tco range of 0.002 to 2 x 10^{-5} d/kg for total PAHs was calculated by OEHHA from the risk assessment by Muhlemann et al. (2006), based on measurement of total PAHs (roughly 19 PAHs measured) in contaminated feed. Although BaP consisted of only 1.5% of total PAHs, the calculated Tco was within an expected range of 0.013-0.00013 d/kg for BaP. We chose a cow's milk Tco of 0.01 d/kg for total PAHs based primarily on the high-end accumulation of BaP in cow's milk from Muhlemann et al. The recommended Tco is also within the range of 0.02 to 0.002 d/kg estimated for PAHs from data published by Grova et al. (2000).

No data could be found regarding residue levels of PAHs in cattle muscle. The ratio of simulated PAH residues in body fat of steers to body fat of lactating dairy cows for extensively metabolized lipophilic compounds is about 1.4, based on PBPK modeling (MacLachlan, 2009). Assuming equal PAH concentrations in milk fat and body fat of dairy cattle, and application of a scaling factor of 1.4 for dairy cattle to steers, we calculated a default beef Tco for PAHs of 0.067 d/kg (0.01 d/kg x 0.19/0.04 x 1.4).

Accumulation data are also lacking for PAHs in pigs. Using the assumptions from MacLachlan (2009) for transfer of extensively metabolized lipophilic compounds to body fat in livestock, the ratio of PAHs in body fat of pigs to dairy cattle is close to 1. Based on a milk Tco of 0.01 d/kg, adjusting for fat content in pig meat and a scaling factor of 1, we calculate a default pig meat Tco of 0.058 d/kg (0.01 d/kg x 0.23/0.04 x 1).

K.2.1.5 Polychlorinated Biphenyls (PCB)

Specific congener Tcos are recommended due to variation in absorption and metabolism of PCBs in dairy cattle, and also due to the degree of chlorination and the position of the chlorine atoms. Some PCBs are transferred effectively unchanged from grass to milk and dairy products (e.g. PCBs 118, 138, 153, 180), with the cow acting as an efficient conduit to humans, while others (e.g. PCBs 52, 101, 149) are largely removed from the environment and the human food chain if ingested by the dairy cow because they are readily metabolized by the cow (Thomas et al., 1999b). Tcos for individual PCB congeners were estimated from published data and are presented in Table K-1 (Slob et al., 1995; Thomas et al., 1998; Thomas et al., 1999a; Kerst et al., 2004; Huwe and Smith, 2005). Kerst et al. (2004) provided TEQ-adjusted data from which a Tco (WHO-TEQ) of 0.014 d/kg was estimated for unspeciated PCBs.

In dairy cattle, Willett et al. (1990) reviewed early studies that examined the transfer of Aroclor 1254 applied to feed to cow's milk. Tcos of 0.008 to 0.009 d/kg were obtained with doses ranging from 3.5-200 mg/d and exposures ranging from 60-107 days. A cow's milk Tco of 0.01 d/kg for unspeciated PCBs from data by Thomas et al. (1999a) was calculated for the sum of 28 PCB congeners found both in feed and the milk.

Only one study could be found that allowed development of poultry meat Tcos for a limited number of individual PCB congeners. Pirard and De Pauw (2005) determined bioconcentration factors for coplanar-PCBs (PCBs 77, 81, 126, 169) in chicken breast muscle. Traag et al. (2006) provided bioconcentration data in abdominal chicken fat for all PCBs but exposure lasted only seven days. Because steady-state was not attained, Tcos could not be reliably determined. However, the data do indicate that based on the number of chlorines, the coplanar-PCBs are similarly, or more, bioaccumulative in fat compared to the other PCB congeners with the same number of chlorines. Thus, Tcos for the non-coplanar PCB congeners in Table K-1 were based on the co-planar PCBs with the same number of chlorines.

No reliable data could be found for developing individual congener Tcos for chicken eggs. Thus, the muscle Tcos for individual PCB congeners were also used for eggs, following adjustment for the higher fat content of eggs (11%) compared to muscle (7%).

A general PCB egg Tco of 6.7 d/kg was calculated from a laboratory study in which seven reference congeners (only one of which (#118) is listed in Table K-1) were spiked in the diet of hens (De Vos et al., 2005). Because none of the more bioaccumulative co-planar PCBs were investigated in this study, the co-planar PCB Tco of 10 d/kg was used for unspeciated PCBs. Numerous unspeciated PCB feed-to-muscle tissue studies have been published in chickens, resulting in a range of Tco values of 2.5 to 7.7 d/kg (Hansen et al., 1983; De Vos et al., 2003; Maervoet et al., 2004; De Vos et al., 2005; Pirard and De Pauw, 2005). A Tco of 7 d/kg for unspeciated PCBs was selected as the default value to reflect the median Tco of the individual congeners listed in Table K-1, and because this value is within the range of Tcos for unspeciated PCBs.

No reliable data could be found that estimated transfer of PCBs consumed in food to body fat of beef cattle. In dairy cattle, Willett et al. (1990) reviewed early experiments that examined the transfer of Aroclor 1254 from feed to adipose tissue. Fresh weight dairy beef Tcos of 0.013 to 0.027 d/kg were obtained for doses ranging from 10-200 mg/d with 60 day exposures. In another study, a beef Tco of 0.024 d/kg was calculated for dairy cattle following 14-week consumption of PCBs that naturally contaminated pastures (Thomas et al., 1999a).

On a fat weight basis, Thomas et al. (1999b) observed that not only are the PCB concentrations in body fat and milk fat similar, but that the congener patterns were similar as well. Thus, even though comprehensive congener-specific data are lacking for PCBs in muscle, congener-specific beef Tcos can be estimated from the cow's milk Tco data by adjusting for the greater fat content in muscle tissue (19%) compared to the milk fat content (4%).

We applied a PBPK-generated scaling factor developed by MacLachlan (2009) to estimate the transfer of PCBs from body fat of lactating cattle to body fat of beef cattle. Using data by Huwe and Smith (2005) that found a cow's milk half-life of 39-196 days for some co-planar PCBs, and the data by Thomas et al. (1999b) that showed similar levels of PCBs in milk fat and body fat, the ratio of simulated co-planar PCB levels in body fat of steers to body fat of lactating dairy cows is approximately 10. We multiplied the scaling factor of 10 by the PCB milk Tcos in Table K-1 following adjustment for differences in fat content between milk and beef to generate Tcos for beef.

In swine, Arochlor 1254 was added to feed for 6 months resulting in an unspeciated PCB Tco of 0.52 d/kg (Hansen et al., 1983). Speciated Tcos for 16 PCBs could be determined from the data, although only one PCB (#118) is currently listed in Table K-1. Thus, Tcos for individual PCBs in Table K-1 were based on the highest calculated PCB Tco with the same number of chlorines from the Hansen et al. study.

K.2.1.6 Polychlorinated Dibenzo-p-Dioxins and Furans (PCDD/F)

Numerous studies have investigated the feed-to-cow's milk transfer of PCDD/Fs. Several of these studies were conducted in the field near municipal solid waste incinerators, or estimated the mass balance of PCDD/F intake resulting from exposure to background or elevated levels of PCDD/Fs in pasture and soil (McLachlan et al., 1990; Slob et al., 1995; Schuler et al., 1997b; McLachlan and Richter, 1998; Lorber et al., 2000). These types of studies likely represent the best data for developing individual congener and overall unspeciated transfer factors of PCDD/Fs from "Hot Spots" facilities. Averaged congener Tco values were estimated from these data and are presented in Table K-1.

The milk Tco decreases by an order of magnitude or more for some of the higher chlorinated PCDD/Fs. This trend agrees with models showing that the percent transfer of chemical from feed to milk decreases for compounds with log Kow larger than about 6.5 (McLachlan, 1996). This reduced absorption is attributed to the presence of an aqueous resistance that limits diffusion of very hydrophobic compounds through the

intestinal wall. Thus, a Tco for total PCDD/Fs (unspeciated PCDD/Fs) has not been pursued by researchers in their exposure studies. Nevertheless, a Tco for unspeciated dioxin-like PCDD/Fs of 0.001 d/kg can be calculated from the data by McLachlan et al. (1990).

Several studies provided data from which Tcos could be estimated for individual PCDD/F congeners found in eggs and chicken meat. For eggs, transfer factor data were derived from three studies in which feed was mixed with soil environmentally contaminated with PCDD/Fs (Petreas et al., 1991; Stephens et al., 1995; Schuler et al., 1997a), and one study of feed contaminated with fly ash (Pirard and De Pauw, 2006). Individual congener Tcos among the studies were similar, often within a factor of five between values. An average Tco was calculated for each congener from the four studies and is shown in Table K-1.

Many of the same studies in chickens also estimated accumulation values for the sum of all PCDD/F congeners, or unspeciated PCDD/Fs, in eggs and meat. In egg, four studies in free-range and laboratory chickens exposed to contaminated soil provided an average Tco of 5.5 d/kg (range: 1.9 to 13.1 d/kg) for unspeciated PCDD/Fs (Petreas et al., 1991; Stephens et al., 1995; Malisch et al., 1996; Schuler et al., 1997a). In chicken muscle, three contaminated feed or soil studies provided accumulation data from which an average Tco of 4.6 d/kg (range: 1.0 to 7.6 d/kg) was calculated (Stephens et al., 1995; Iben et al., 2003; Pirard and De Pauw, 2005).

For the controlled laboratory feed-to-egg studies in which PCDD/Fs in fly ash or oil were added to feed (i.e., no contaminated soil was added to the diet), egg Tcos ranged from 8.5 to 17 d/kg with a mean of 12 d/kg (Pirard and De Pauw, 2005; 2006; Van Eijkeren et al., 2006).

For field studies, calculated egg Tcos of free-foraging chickens in various regions with PCDD/F-contaminated soil showed greater variation and was higher (Schuler et al., 1997a; Harnly et al., 2000; Hoogenboom et al., 2006). The Tcos ranged from 12 to 37 d/kg with an average of 23 d/kg. An assumption was made that the PDCC/F source for the free-foraging hens was contaminated soil, and that the soil ingestion rate was 10 g soil/day. There is general support among researchers for this soil ingestion rate by free-foraging chickens (De Vries et al., 2006). The larger egg Tco in field studies compared to controlled laboratory studies may be a result of free-foraging chickens consuming soil organisms and herbs and grass which may also be contaminated. However, greater bioavailability of soil PCDD/Fs in the field, or a higher soil ingestion rate than predicted may also play a role in a larger egg Tco under field conditions.

Overall, the range of mean values for these three types of studies is not large (within a factor of 10), considering the different sources of PCDD/Fs that the poultry were exposed to. A grand mean from the three types of exposure studies (contaminated soil field study, controlled contaminated soil study and contaminated feed study) is 13 d/kg (3.6 + 23 + 12 d/kg / 3), which we recommend as the default egg Tco for PCDD/Fs.

For edible muscle tissue (usually thigh or breast tissue), TEQ-adjusted Tcos could be calculated from several studies that investigated PCDD/F concentrations in chickens given contaminated feed. In a controlled laboratory study in which 10% of the diet was PCDD/F-contaminated soil, a Tco of 7.4 d/kg was calculated (Stephens et al., 1995). In three contaminated feed studies where PCDD/Fs in oil or fly ash were added to diet, similar Tcos of 8.6, 9.0 and 4.1 d/kg were calculated (Iben et al., 2003; Pirard and De Pauw, 2005; 2006).

Congener-specific data for development of beef Tcos were not as comprehensive as that for development of cow's milk Tcos. Two long-term pentachlorophenol (PCP) feeding studies in dairy cattle determined body fat concentrations for several PCDD/F congeners (1, 2, 3, 6, 7, 8- and 1, 2, 3, 7, 8, 9-HxCDD, 1, 2, 3, 4, 6, 7, 8-HpCDD, OCDD, 1, 2, 3, 4, 6, 7, 8-HpCDF, and OCDF) that were contaminants in the PCP formulation (Firestone et al., 1979; Parker et al., 1980). Beef Tcos based on dairy cattle for the other congeners and unspeciated PCDD/Fs were estimated with the assumption that the fat concentration is similar in milk and beef, and were adjusted upward to account for the greater fat content in muscle tissue (19%) compared to the fat content in milk (4%). As noted above, the concentration of PCBs in milk fat and body fat have been shown to be similar in exposure studies (Thomas et al., 1999b). We then applied scaling factors by MacLachlan (2009) to estimate the transfer of PCDD/Fs from body fat of lactating cattle to body fat of beef cattle. Data by Huwe and Smith (2005) found that half-lives were mostly 30-50 days for the PCDD/Fs; the major exceptions were OCDF ($t_{1/2}$ = 14 days) and OCDD ($t_{1/2}$ = 72.6 days). A ratio of 7 is estimated for the simulated PCDD/F levels in body fat of steers to body fat of lactating dairy cows for most PCDD/Fs. A ratio of 4 was estimated for OCDF and a ratio of 10 was estimated for OCDD.

Pig Tcos for individual and unspeciated PCDD/Fs in Table K-1 were estimated from a comprehensive study in which PCDD/Fs were added to the diet in feed of pigs during the 12-week fattening period (Spitaler et al., 2005). This exposure period represents the last 12-weeks prior to slaughter in the typical 6-month life of a pig. Notably, the researchers did not observe a reduction of residues due to roasting of the meat.

K.2.2 Tcos for Inorganic Metals and Chemicals

The studies used to derive inorganic metal Tcos listed in Table K-2 usually presented data as fresh weight concentrations in muscle, milk and eggs. Occasionally, dry weight concentrations were reported. Unless the study noted the water content of the food source, default factors of 0.87 for cow's milk, 0.35 for chicken egg, 0.25 for chicken meat, and 0.30 for beef and pork were used for adjusting to fresh weight concentration (USDA, 1975).

Biotransfer studies for pig muscle could not be found for most of the metals. As noted in the beginning of this appendix, biotransfer data in cattle were more abundant. Where specific metal biotransfer data were missing in pigs but present in cattle, the pig meat Tco was estimated using a simple metabolic weight adjustment from cattle to pig as shown in Eq. K-1.

In general, low concentrations of inorganic metals are transferred from contaminated feed to muscle tissue, cow's milk and eggs and are not as great a concern relative to other potential sources of heavy metals in multipathway exposures. However, many of the inorganic metals such as cadmium, lead and mercury tend to accumulate over time in organs, particularly kidney and liver. Thus, frequent consumption of organs from exposed food animals may present a much greater toxic hazard to humans than consumption of the meat. Cadmium is of particular concern due to its relatively high toxicity and high potential for accumulation in the kidney and liver. Kidney and liver-specific Tcos for cadmium and a few other metals are presented in the text below for some of these food-producing animals only for comparison purposes. Tcos for accumulation in bone for some of the metals (i.e., lead) are also noted or calculated for some of the food products.

Another toxicological concern is that chickens can convert some of the ingested inorganic mercury in controlled feeding studies to methyl mercury, which is then found primarily in the poultry meat and egg white (Kiwimae et al., 1969). The inorganic mercury Tcos for poultry meat and eggs in Table K-2 represents total mercury, although some will be present as organic methyl mercury. Because methyl mercury is not emitted from facilities (i.e., only inorganic or elemental mercury is emitted), it is not accounted for in health risk assessments. However, Tcos for methyl mercury were calculated by OEHHA and presented in Section K.2.2.7 only for comparison to the inorganic mercury Tcos.

K.2.2.1 Arsenic

Only one study could be located that recorded a measurable increase of arsenic in cow's milk following dairy cattle consumption of contaminated feed. We calculated a Tco of 5×10^{-5} d/kg from data in dairy cattle exposed to As(III) as arsenic trioxide for 15-28 months (Vreman et al., 1986).

In poultry, organic arsenic compounds are an approved dietary supplement that can result in increased levels of total arsenic in meat and eggs (Lasky et al., 2004). Both organic and inorganic forms of arsenic are found in poultry, with inorganic forms more toxic than organic forms. Analysis of poultry and meat samples indicates that about 65% of total arsenic is in the inorganic form.

We calculated a Tco of 0.07 d/kg for total arsenic in eggs from hens fed a diet containing arsenic trioxide (Holcman and Stibilj, 1997). In muscle, total arsenic Tcos of 0.06 and 0.02 d/kg were determined in chickens from two studies following addition of arsenic trioxide to feed (Overby and Frost, 1962; Vadnjal et al., 1997). The proportion of arsenic in the inorganic form was not determined. In drinking water, soluble As(V) was added to the water resulting in a total arsenic Tco of 0.2 d/kg in muscle of broiler chickens (Pizarro et al., 2004). However, only 10% of arsenic in muscle was in the inorganic form. Over 50% was present as dimethylarsinic acid, which is considered a methylation detoxification pathway for arsenic. Thus, the inorganic arsenic Tco was 0.02 d/kg. We calculated an average muscle Tco of 0.03 d/kg from the three studies for transfer of arsenic from diet to chicken meat.

In beef cattle, Vreman et al. (1988) administered arsenic trioxide in the feed for 143 days to 16 bulls at about 12.5 mg/d resulting in a muscle Tco of 2.4×10^{-3} d/kg. The same Tco was calculated from data by Ham et al. (1949) that dosed adult steers daily with 270 mg arsenic trioxide for 201 days. In another study in steers, Bruce et al. (2003) estimated the daily intake of arsenic from grazing pasture grass, ingesting dust adhering to pasture, and direct ingestion of soil in an area contaminated with arsenic-laced mine tailings. Based on the daily intake and muscle concentration of arsenic at sacrifice after 237 days of exposure, a Tco of 2.8×10^{-4} d/kg was derived. We calculated an average muscle Tco of 1.7×10^{-3} d/kg from these three studies, which we recommend as the default value for beef cattle. Long-term arsenic feeding studies have also been conducted in lactating dairy cows. A slightly lower muscle Tco of 7.1×10^{-4} d/kg was calculated from these studies (Peoples, 1964; Vreman et al., 1986).

Arsenic exposure in beef and dairy cattle has not shown tissue-specific sequestering in liver or kidney, unlike some of the inorganic metals (e.g., cadmium, lead, and mercury). Similar Tcos were estimated for muscle, liver and kidney (Ham et al., 1949; Peoples, 1964; Vreman et al., 1988).

K.2.2.2 Beryllium

No inorganic beryllium accumulation studies could be found in the literature for poultry. Thus, we calculated poultry egg and meat Tcos for beryllium based on the average Tco value of the other “Hot Spots” divalent, cationic metals in Table K-2 (i.e., cadmium, lead, inorganic mercury, and nickel) providing beryllium Tcos for egg and muscle of 0.09 and 0.2 d/kg, respectively.

No multiple day inorganic beryllium exposure studies have been conducted in cattle or swine. In a single bolus study, Ng (1982) estimated a cow’s milk Tco of 9.1×10^{-7} d/kg based on recovery of radiolabeled beryllium chloride given to dairy cattle. For beef, we determined a beryllium Tco of 3×10^{-4} d/kg based on the average Tco value of the divalent, cationic metals cadmium, lead, and inorganic mercury. Beef Tcos for these three metals were determined directly from published studies. A default pork Tco was determined by us by the same method as that used for beef, resulting in a pig meat Tco of 1×10^{-3} d/kg.

K.2.2.3 Cadmium

Very low accumulation of cadmium occurs in cow’s milk, and concentrations of cadmium in cow’s milk are often below the detection limit. In his review, Stevens (1991) estimated an average Tco of 1.3×10^{-6} d/kg in cow’s milk from two long-term cadmium exposure studies by Vreman et al. (1986). More recently, we estimated a milk Tco of 1.3×10^{-5} d/kg from exposure data in a single cow exposed to cadmium for 77 days (Mehennaoui et al., 1999). The average Tco from the three exposure studies is 5×10^{-6} d/kg, which we recommend as a default Tco.

Numerous cadmium accumulation studies have been conducted in poultry. Similar to cow’s milk, very low accumulation of cadmium occurred in hen’s eggs with exposure in

feed; the levels of cadmium in eggs are sometimes below the detection limit. We calculated an average egg Tco of 0.01 d/kg from the best available data (Leach et al., 1979; Sharma et al., 1979; Hinesly et al., 1985). In muscle, we determined cadmium Tcos in exposed chickens ranging from 0.2 to 1 d/kg (Leach et al., 1979; Sharma et al., 1979; Hinesly et al., 1985; Pribilincova et al., 1995; Bokori et al., 1996). The average value from these studies was 0.5 d/kg, which we recommend as the Tco.

Similar cadmium Tcos in muscle of dairy and beef cattle have been observed in long-term feeding studies lasting 3.5 to 28 months. We calculated an average Tco of 2.0 x 10^{-4} d/kg with a range of 1.2 – 3.2 x 10^{-4} d/kg (Johnson et al., 1981; Vreman et al., 1986; 1988). A muscle Tco of 6.5 x 10^{-5} d/kg was obtained from a feeding study by Lamphere et al. (1984) describing cadmium body burden in calves exposed for 60 days. However, the short exposure duration only during growth of the animal may result in an underestimation of the Tco compared to exposure to adulthood.

Cadmium accumulates to a much greater extent in some organs compared to muscle tissue. In poultry, exposure studies suggest that cadmium accumulation in the kidney and liver increases with increasing exposure duration and may not attain a steady-state concentration. Eighty-week exposure to cadmium in chickens resulted in a Tco of 800 d/kg in the kidney and 70 d/kg in the liver (Hinesly et al., 1985). In dairy and beef cattle, cadmium Tcos for liver and kidney did not vary greatly even though exposure durations varied. Average calculated Tcos were about 0.03 d/kg (range: 0.01 to 0.048 d/kg) for liver, and 0.1 d/kg (range: 0.09 to 0.19 d/kg) for kidney (Sharma et al., 1979; Sharma et al., 1982; Vreman et al., 1986; 1988).

Only one study could be found that measured cadmium muscle levels in pigs following exposure to cadmium in feed. Cousins et al. (1973) only found measurable cadmium levels in skeletal muscle at the highest of four doses tested (1350 ppm) following a six-week exposure, but this level caused severe toxicity. More accurate estimates of muscle uptake were found in heart tissue, which exhibited increased tissue concentration with increasing dose and may represent the upper end of the cadmium concentration found in skeletal muscle. The average Tco we calculated in heart muscle was 0.0051 d/kg. In the liver and kidneys of pigs, cadmium Tcos as high as 0.48 and 2.53 d/kg, respectively, were calculated from a study by Sharma et al. (1979).

K.2.2.4 Chromium (Hexavalent)

Only a portion of ingested hexavalent chromium (Cr(VI)), perhaps 1-2%, is expected to be systemically absorbed in the hexavalent form due to rapid reduction to the less soluble and less toxic trivalent chromium in the acidic environment of the stomach (Costa, 1997; NTP, 2008). Trivalent chromium (Cr(III)) is an essential micronutrient, but no cancer potency or noncancer reference exposure level is currently available for this form of chromium. Cr(VI) that is absorbed can then be actively transported into all cells and tissues of the body in place of anions, such as phosphates. Once inside the cell, the Cr(VI) is reduced to various unstable reactive intermediates and, finally, stable Cr(III) is ultimately formed inside the cell.

Current analytical procedures cannot differentiate between the oxidation states of chromium in biological tissues (NTP, 2008). However, it has been advocated that any Cr(VI) transported into meat and eggs would be converted to the more stable Cr(III) form and would presumably not pose a risk for human consumption (Chundawat and Sood, 2005). Based on these findings no Cr(VI) Tco is currently recommended by OEHHA for meat and eggs.

However, a similar situation may not be the case for cow's milk. Lameiras et al. (1998) found Cr(VI) in cow's milk, which was 25-50% of total chromium. In whole milk, the average total chromium concentration was 2.70 ug/L (range: 1.42-5.70 ug/L) and the average Cr(VI) concentration was 0.68 ug/L (range: 0.20-1.20 ug/L). No multiple day Cr(VI) exposure studies in dairy cattle could be found in the literature. Following a single oral dose of radiolabeled sodium chromate (Na_2CrO_4), Van Bruwaene et al. (1984) calculated a steady-state cow's milk Tco of 1.0×10^{-5} d/kg for total chromium. Stevens (1991) estimated a similar Tco of 1.4×10^{-5} d/kg from the same data based on a half-life of 26 days for total chromium in cow's milk. These studies did not attempt to estimate the proportion of total chromium that was secreted as Cr(VI) into milk.

Multiplying the Stevens total chromium Tco by the fraction of total chromium that is Cr(VI) in normal milk (1.4×10^{-5} d/kg x 0.68/2.70 ug/L) provided a modified Tco of 3.5×10^{-6} d/kg. Until valence-speciated cow's milk data are available from Cr(VI) exposure studies, we chose a midpoint Tco value between the Stevens Tco and this modified Tco adjusted for Cr(VI) content in normal milk (8.75×10^{-6} d/kg) as a health-protective cow's milk default value for Cr(VI).

K.2.2.5 Fluoride

In a series of long-term exposure studies on fluorides' effect on milk production, the fluoride concentration in the milk of dairy cows given fluoride in feed resulted in an estimated cow's milk Tco of 0.0003 d/kg (Stoddard et al., 1963; Harris et al., 1964).

Fluoride in the diet of hens resulted in very low accumulation of fluoride in muscle, and yolk and albumin of eggs (Hahn and Guenter, 1986). We calculated a Tco in whole eggs of 0.008 d/kg from the exposure data. Considerably greater accumulation occurs in egg shell. Muscle accumulation in the fluoride-exposed hens resulted in a Tco of 0.03 d/kg.

Specific data concerning accumulation of fluoride in the skeletal muscle tissue of exposed cattle could not be found. However, in cases of high fluoride intake, fluoride levels in the soft tissue (i.e., brain, liver, kidney, pancreas, intestines, etc.) are reported to increase only two or three times the normal value in meat producing animals. Fluoride does not accumulate in the edible portions of the animal (Suttie et al., 1958; Shupe et al., 1964). However, considerably greater accumulation of fluoride occurred in bone. In heart tissue, we calculated a fluoride Tco of 8.4×10^{-4} d/kg for Holstein cows fed fluoride-contaminated rations for 5.5 years, which we recommend as the default muscle Tco for range cattle (Suttie et al., 1958). It is assumed that similar

pharmacokinetic properties, and similar Tcos, occur for fluoride in both skeletal and heart muscle tissue.

K.2.2.6 Lead

Only three contaminated feed studies observed measurable levels of lead in milk from both control and exposed dairy cows. Based on data from a 15-28 month lead exposure study of dairy cows kept indoors, a cow's milk Tco of 2.6 x 10^{-5} d/kg was calculated (Vreman et al., 1986). A three-month outdoor lead exposure study by the same researchers produced a Tco of 5.4 x 10^{-5} d/kg. Stating that the half-life of lead in dairy cows is about 45 days, Stevens (1991) adjusted the Tco of the three-month outdoor study to 7.1 x 10^{-5} d/kg. However, Willett et al. (1994) observed that steady-state was attained in cow's milk after only 14 days of a 49-day lead exposure study, generating a Tco of 7.9 x 10^{-5} d/kg. Using the steady-state-corrected Tco by Stevens (1991) for the outdoor Vreman study, we recommend an average Tco of 5.9 x 10^{-5} d/kg from these three studies.

An average Tco of 0.4 d/kg in muscle was calculated by OEHHA for lead in broiler chicks fed contaminated feed for 20 days (Stoddard et al., 1963; Harris et al., 1964; Latta and Donaldson, 1986a; 1986b). For comparison, a roughly 10-fold higher Tco was calculated for lead in kidney. However, lead tends to accumulate most in bone, generating a Tco of 70 d/kg. Lead in bone is not expected to be a problem, unless contaminated bone is ground into bone meal and fed to animals. Accumulation of lead in eggs was very low, generating a Tco of 0.04 d/kg (Meluzzi et al., 1996).

Vreman et al. (1988) administered lead acetate in feed to young bulls for 143 days during the fattening period. The resulting muscle Tco was 2.7 x 10^{-4} d/kg. A slightly lower muscle Tco of 6.7 x 10^{-5} d/kg in lactating dairy cows fed lead mixed with their feed (Vreman et al., 1986).

Roughly 10- to 100-fold greater accumulation of lead occurs in the kidney and liver of cattle compared to their muscle tissue. We calculated Tcos of 4.8 x10^{-3} and 1.4 x 10^{-2} d/kg for liver and kidney, respectively, in the bulls from the Vreman et al. (1988) study. In addition to liver and kidney, lead was also found to accumulate in bone. In a three-month feeding study in dairy cattle, a bone Tco of 0.02 d/kg was calculated from the data by Sharma et al. (1982). In one of the few biotransfer studies conducted in pigs, a liver Tco of 1.4 x 10^{-2} d/kg was recorded in pigs fed diets containing either 5 or 25 ppm lead acetate for 90 days (Sharma and Street, 1980).

K.2.2.7 Inorganic Mercury

Addition of inorganic mercury (Hg(II)) to the feed of hens for 140 days resulted in a muscle tissue Tco of 0.1 d/kg (Kiwimae et al., 1969). However, some Hg(II) was converted to methyl mercury (MeHg) in the chickens, resulting in a muscle Tco of 0.09 d/kg for MeHg. When only MeHg is added to the diet in prolonged feeding studies, an average Tco of 10 d/kg was calculated with virtually all the mercury in the muscle as MeHg (Kiwimae et al., 1969; Soares et al., 1973; Hilmy et al., 1978). Some Hg(II) added

to feed is also endogenously methylated in the hens and transported to the eggs. Addition of Hg(II) to the feed of hens for 140 days resulted in a calculated egg Tco of 0.8 d/kg for total mercury, and 0.5 d/kg for MeHg (Kiwimae et al., 1969). An average egg Tco of 11 d/kg was calculated when only MeHg was added to feed (Scott et al., 1975; Hilmy et al., 1978).

Vreman et al. (1986) observed a small, but statistically insignificant increase in mercury in cow's milk with exposure of dairy cattle to inorganic mercury in feed for 15-28 months. The Tco range was 7 to 40 x 10^{-5} d/kg with an average of 2 x 10^{-4} d/kg. Stevens (1991) calculated Tcos of 9.2 x 10^{-6} and 1.3 x 10^{-5} d/kg from oral single bolus studies of radiolabeled inorganic mercury by Mullen et al. (1975) and Potter et al. (1972). The steady-state Tcos were calculated by use of study-specific half-lives of 1.2 (Potter et al., 1972) or 5.5 days (Mullen et al., 1975) for mercury. We calculated an average Tco of 7 x 10^{-5} d/kg from the three studies, which we recommend for transfer of inorganic mercury to cow's milk.

Similar to cow's milk, only a small, but statistically insignificant increase in inorganic mercury could be measured in muscle tissue following long-term exposure of dairy and beef cattle to soluble mercury (Vreman et al., 1986; 1988). Calculated maximum muscle Tco values from these two studies were 6.7-18 x 10^{-4} d/kg, but we lack confidence in this value due to the detection limit of these studies. To calculate the biotransfer of ingested mercury to muscle, Stevens (1992) relied on three oral bolus dose studies that determined the half-life of inorganic mercury in blood of dairy cattle (Potter et al., 1972; Ansari et al., 1973; Mullen et al., 1975). Operating on a reasonable assumption that muscle is a well-perfused tissue and shares the same kinetic compartment as blood, Stevens calculated an average muscle Tco of 3.5 x 10^{-4} d/kg (range: 1.8-4.4 x 10^{-4} d/kg). This value is comparable with the Tcos estimated from the Vreman studies, which we recommend as the point estimate Tco for inorganic mercury in beef.

Although it is not anticipated that human exposure to methyl mercury via cow's milk and beef would be a significant pathway (e.g., as compared to fish), biotransfer information is included here for completeness. There are few published data that investigated ruminant methylmercury uptake and accumulation. However, background exposure and accumulation of inorganic and methylmercury in meat products are reported to be very low (U.S. EPA, 1997). In their risk assessment guidelines, U.S. EPA (2005) suggests that only 13% of total mercury in ruminants is present as methylmercury, an indication that ruminants have little exposure to methylmercury.

In vitro, cow rumen microflora does not methylate added inorganic mercury (as $HgCl_2$) to methylmercury (Kozak and Forsberg, 1979). On the other hand, rumen microflora was found to demethylate up to 40% of added methylmercury to elemental (Hg^0), or metallic, mercury, which would then be presumably excreted with little or no absorption. This finding suggests that ruminants can detoxify some of the ingested methylmercury.

Stevens (1991) estimated that the Tco for methylmercury in cow's milk is roughly one order of magnitude greater than that for inorganic mercury (i.e., 7 x 10^{-4} d/kg). His

finding was based on a study by Neathery et al. (1974), in which two dairy cows were given a bolus dose of radiolabeled methylmercuric chloride and followed for the appearance of label in milk for 14 days. A milk excretion half-life of 6 days was calculated from the data. It was suspected that the lipophilic nature of methyl mercury resulted in its accumulation in milk fat. Of the labeled methylmercury that was absorbed, 72% of the total body burden was found in muscle tissue 15 days after the single bolus dose. However, there are insufficient data to estimate the biotransfer of ingested methylmercury in cattle and pigs with chronic exposure.

K.2.2.8 Nickel

Only two studies were found in the literature that attempted to estimate the nickel concentration in cow's milk following 1.5 to 2 month exposure of the dairy cattle to inorganic nickel-contaminated feed (Archibald, 1949; O'Dell et al., 1970). Neither study used analysis methods that were sensitive enough to record measurable increases of nickel in the cow's milk. Stevens (1991) used the maximum value approach by dividing the detection limit (0.1 ppm) of the studies by two, arriving at an average cow's milk Tco of 2.7×10^{-5} d/kg. Until more sensitive studies are conducted, we recommend this Tco as the default value for inorganic nickel.

Limited data for nickel indicate low accumulation of this metal occurs in eggs and tissues of chickens (Ling and Leach, 1979; Meluzzi et al., 1996). We calculated Tcos of 0.02 d/kg for both eggs and muscle tissue of hens fed inorganic nickel mixed in their diet. As with other inorganic metals, greatest nickel accumulation occurred in the kidney (Tco = 0.68 d/kg), resulting in a Tco over 30-fold higher than that found in muscle or eggs.

No adequate studies investigating biotransfer of ingested inorganic nickel to beef or pork could be located. As with the approach used for beryllium, we determined a beef Tco based on an average of the three divalent cationic metal Tcos (i.e., cadmium, lead and inorganic mercury) that had sufficient biotransfer data available in the literature. The resulting beef Tco was 3×10^{-4} d/kg. We then developed a pig meat Tco of 0.001 d/kg based on the cow-to-pig metabolic weight ratio adjustment (Eq. K-1). OEHHA recognizes that these Tcos developed for beef and pork are more uncertain than would be desirable. However, the data available in other food-producing animals and similar Tcos developed for other cationic metal contaminants indicates the nickel muscle Tco is likely not underestimated in cattle and pigs.

K.2.2.9 Selenium

The selenium concentration in milk tends to increase as intake of selenium increases from about 2 to 6 mg/day (Fisher et al., 1980; Maus et al., 1980; Beale et al., 1990). Secretion of selenium into milk then appears to reach a temporary limit when selenium intake is about 6 to 12 mg/day. The mammary gland is either limited in the limited amount of selenium it can secrete into milk, or, more likely, the net absorption of selenium from the gut is controlled in the face of increased selenium intake. Only when selenium intake increases above 50-100 mg/day does the ability of the protection

mechanism become exceeded, resulting in selenium toxicity and increased selenium concentration in milk. We calculated a Tco of 0.009 d/kg based on the average value for studies that supplemented feed with 6 mg/d selenium or less.

Optimum levels of selenium in the diet of poultry are about 0.1 to 0.2 ppm (Arnold et al., 1973; Moksnes and Norheim, 1982). Concentrations of selenium above 3 ppm may result in toxicity. At concentrations of 1 to 9 ppm selenite in the feed, we calculated an average egg Tco of 3 d/kg (Arnold et al., 1973; Ort and Latshaw, 1978; Moksnes and Norheim, 1982; Davis and Fear, 1996). In broiler chicks, an average Tco of 0.9 d/kg for muscle was calculated (Moksnes and Norheim, 1982; Echevarria et al., 1988a; 1988b). Laying hens had a lower Tco of 0.4 d/kg for muscle tissue, possibly due to eggs acting as an elimination pathway for selenium (Arnold et al., 1973; Ort and Latshaw, 1978; Moksnes and Norheim, 1982). Thus, the muscle Tco for selenium is based on the findings in meat (broiler) chickens.

In beef cattle, groups of calves were fed sodium selenite in a milk replacer at concentrations of 0.2 to 5 ppm for six weeks (Jenkins and Hidiroglou, 1986). We calculated an average muscle Tco of 6.6×10^{-2} d/kg from the exposure data. In another study, inorganic selenium was intraruminally administered in beef cows through two soluble-glass boluses to slowly release Se over approximately 11 months (Hidiroglou et al., 1987). We calculated a Tco of 7.1×10^{-3} d/kg in the muscle tissue. The average muscle Tco from the two studies is 0.037 d/kg, which we recommend as the default selenium transfer factor. Jenkins and Hidiroglou (1986) also observed greater accumulation of selenium in the liver and kidney cattle compared to muscle, resulting in calculated Tcos of 2.7 and 0.25 d/kg, respectively.

In pigs, selenium muscle concentrations have been measured following unsupplemented intake or supplementation of selenium in diets. No studies could be located that estimated tissue levels of selenium following prolonged intake of toxic or near-toxic levels of selenium. Using a study by Ku et al. (1972), we calculated an average muscle Tco of 0.61 d/kg in groups of adult pigs that had been fed diets containing selenium at levels ranging from 0.027 to 0.493 ppm. A positive correlation between selenium level in the diet and muscle concentration was observed. Using another study, which exposed pigs to diets containing 0.78-0.88 ppm selenium during the growth phase, we calculated a muscle Tco of 0.35 d/kg in pigs at market weight (Jenkins and Winter, 1973).

Similar to the phenomena observed in dairy cattle, supplementation of pig diets with selenium (0.1 to 1.0 ppm) did not always result in an increase in tissue selenium levels. Tcos based on these studies are as much as 10-fold lower compared to Tcos calculated from baseline levels of selenium found in feed (Groce et al., 1971). However, it is not known if this protective mechanism also operates at higher selenium levels in feed that may produce toxic effects in pigs. Thus, we recommend a default pig Tco based on the average Tco (0.48 d/kg) determined using Ku et al. (1972) and Jenkins and Winter (1973), which covered a range of baseline selenium intakes in feed from 0.027 to 0.88 ppm.

K.3 References

Ansari MS, Miller WJ, Gentry RP, Neathery MW and Stake PE (1973). Tissue 203 Hg distribution in young Holstein calves after single tracer oral doses in organic and inorganic forms. J Anim Sci 36(2): 415-9.

Archibald JG (1949). Nickel in cow's milk. J Dairy Sci 32: 877-80.

Arnold RL, Olson OE and Carlson CW (1973). Dietary selenium and arsenic additions and their effects on tissue and egg selenium. Poult Sci 52: 847-54.

Beale AM, Fasulo DA and Craigmill AL (1990). Effects of oral and parenteral selenium supplements on residues in meat, milk and eggs. Rev Environ Contam Toxicol 115: 125-50.

Beauchamp CJ, Boulanger R, Matte J and Saint-Laurent G (2002). Examination of the contaminants and performance of animals fed and bedded using de-inking paper sludge. Arch Environ Contam Toxicol 42(4): 523-8.

Bluthgen A and Ruoff U (1998). Carry-over of diethylhexylphthalate and aromatic nitro compounds into milk of lactating cows. Third Karlsruhe Nutrition Symposium European Research towards Safer and Better Food.Review and Transfer Congress, Congress Centre, Karlsruhe, Germany, October 18-20, 1998. pp. 25-32.

Bokori J, Fekete S, Glavits R, Kadar I, Koncz J and Kovari L (1996). Complex study of the physiological role of cadmium. IV. Effects of prolonged dietary exposure of broiler chickens to cadmium. Acta Vet Hung 44(1): 57-74.

Bruce SL, Noller BN, Grigg AH, Mullen BF, Mulligan DR, Ritchie PJ, Currey N and Ng JC (2003). A field study conducted at Kidston Gold Mine, to evaluate the impact of arsenic and zinc from mine tailing to grazing cattle. Toxicol Lett 137(1-2): 23-34.

Cavret S, Feidt C, Le Roux Y and Laurent F (2005). Short communication: Study of mammery epithelial role in polycyclic aromatic hydrocarbons transfer to milk. J Dairy Sci 88(1): 67-70.

Chundawat RS and Sood PP (2005). Vitamins deficiency in developing chick during chromium intoxication and protection thereof. Toxicology 211(1-2): 124-31.

Claborn HV, Radeleff RD and Bushland RC. (1960). *Pesticide Residues in Meat and Milk. A Research Report*. ARS-33-63. Prepared by U.S. Department of Agriculture, Agriculture Research Service. pp. 1-46.

Costa M (1997). Toxicity and carcinogenicity of Cr(VI) in animal models and humans. Crit Rev Toxicol 27(5): 431-42.

Cousins RJ, Barber AK and Trout JR (1973). Cadmium toxicity in growing swine. J Nutr 103(7): 964-72.

Crout NMJ, Beresford NA, Dawson JM, Soar J and Mayes RW (2004). The transfer of ^{73}As, ^{109}Cd and ^{203}Hg to the milk and tissues of dairy cattle. J Agric Sci 142: 203-12.

Davis RH and Fear J (1996). Incorporation of selenium into egg proteins from dietary selenite. Br Poult Sci 37(1): 197-211.

De Vos S, Maervoet J, Schepens P and De Schrijver R (2003). Polychlorinated biphenyls in broiler diets: their digestibility and incorporation in body tissues. Chemosphere 51(1): 7-11.

De Vos S, Verschueren D and De Schrijver R (2005). Digestibility, retention and incorporation of low-level dietary PCB contents in laying hens. Chemosphere 58(11): 1553-62.

De Vries M, Kwakkel RP and Kijistra A (2006). Dioxins in organic eggs: a review. Njas-Wageningen J Life Sci 54(2): 207-21.

Dingle JHP and Palmer WA (1977). Residues of hexachlorobenzene in subcutaneous and butter fat of cattle. Aust J Exp Agric Animal Husb 17: 712-17.

Douben PE, Alcock RE and Jones KC (1997). Congener specific transfer of PCDD/Fs from air to cows' milk: an evaluation of current modelling approaches. Environ Pollut 95(3): 333-44.

EC. (2002). *Opinion of the Scientific Committee on Food on the Risk to Human Health of Polycyclic Aromatic Hydrocarbons in Food*. European Commission, Health and Consumer Protection Directorate-General. SCF/CS/CNTM/PAH/29 Final. Available online at: http://europa.eu.int/comm/food/fs/sc/scf/out153_en.pdf.

Echevarria MG, Henry PR, Ammerman CB, Rao PV and Miles RD (1988a). Estimation of the relative bioavailability of inorganic selenium sources for poultry. 1. Effect of time and high dietary selenium on tissue selenium uptake. Poult Sci 67(9): 1295-301.

Echevarria MG, Henry PR, Ammerman CB, Rao PV and Miles RD (1988b). Estimation of the relative bioavailability of inorganic selenium sources for poultry. 2. Tissue uptake of selenium from high dietary selenium concentrations. Poult Sci 67(11): 1585-92.

Eisenreich SJ, Looney BB and Thornton JD (1981). Airborne organic contaminants in the Great Lakes ecosystem. Environ Sci Technol 15: 30-38.

Firestone D, Clower M, Jr., Borsetti AP, Tseke RH and Long PE (1979). Polychlorodibenzo-p-dioxin and pentachlorophenol residues in milk and blood of cows fed technical pentachlorophenol. J Agric Food Chem 27(6): 1171-7.

Fisher LJ, Hoogendoorn C and Montemurro J (1980). The effect of added dietary selenium on the selenium content of milk, urine and feces. Can J Anim Sci 60: 79-86.

Fries GF (1996). A model to predict concentrations of lipophilic chemicals in growing pigs. Chemosphere 32(3): 443-51.

Fries GF and Marrow GS (1976). Hexachlorobenzene retention and excretion by dairy cows. J Dairy Sci 59(3): 475-80.

Groce AW, Miller ER, Keahey KK, Ullrey DE and Ellis DJ (1971). Selenium supplementation of practical diets for growing-finishing swine. J Anim Sci 32(5): 905-11.

Grova N, Feidt C, Crepineau C, Laurent C, Lafargue PE, Hachimi A and Rychen G (2002). Detection of polycyclic aromatic hydrocarbon levels in milk collected near potential contamination sources. J Agric Food Chem 50(16): 4640-2.

Grova N, Laurent C, Feidt C, Rychen G, Laurent F and Lichtfouse E (2000). Gas chromatography-mass spectrometry study of polycyclic aromatic hydrocarbons in grass and milk from urban and rural farms. Eur J Mass Spectrometry 6(5): 457-460.

Hahn PH and Guenter W (1986). Effect of dietary fluoride and aluminum on laying hen performance and fluoride concentration in blood, soft tissue, bone, and egg. Poult Sci 65(7): 1343-9.

Ham WE, Kline EA and Ensminger ME (1949). Residual arsenic and strychnine in the tissues of drug-treated cattle. Am J Vet Res 10(35): 150-3.

Hansen LG, Tuinstra LG, Kan CA, Strik JJ and Koeman JH (1983). Accumulation of chlorobiphenyls in chicken fat and liver after feeding Aroclor 1254 directly or fat from swine fed Aroclor 1254. J Agric Food Chem 31(2): 254-60.

Harnly ME, Petreas MX, Flattery J and Goldman LR (2000). Polychlorinated dibenzo-p-dioxin and polychlorinated dibenzofuran contamination in soil and home-produced chicken eggs near pentachlorophenol sources. Environ Sci Technol 34(7): 1143-9.

Harris LE, Raleigh RJ, Stoddard GE, Greenwood DA, Shupe JL and Nielsen HM (1964). Effects of fluorine on dairy cattle. III. Digestion and metabolism trials. J Anim Sci 23: 537-46.

Hidiroglou M, Proulx J and Jolette J (1987). Effect of intraruminally administered, selenium soluble-glass boluses on selenium status in cows and their calves. J Anim Sci 65(3): 815-20.

Hilmy MI, Rahim SA, Abbas AH and Taka RY (1978). Toxicity of organic mercury in sheep and hens. Clin Toxicol 12(4): 445-56.

Hinesly TD, Hansen LG, Bray DJ and Redborg KE (1985). Transfer of sludge-borne cadmium through plants to chickens. J Agric Food Chem 33(2): 173-80.

Holcman A and Stibilj V (1997). Arsenic residues in eggs from laying hens fed with a diet containing arsenic (III) oxide. Arch Environ Contam Toxicol 32(4): 407-10.

Hoogenboom LA, Kan CA, Zeilmaker MJ, Van Eijkeren J and Traag WA (2006). Carry-over of dioxins and PCBs from feed and soil to eggs at low contamination levels--influence of mycotoxin binders on the carry-over from feed to eggs. Food Addit Contam 23(5): 518-27.

Hoogenboom LAP (2005). Behavior of polyhalogenated and polycyclic aromatic hydrocarbons in food-producing animals. Rev Food Nutr Toxicity 2: 269-99.

Horstmann M and McLachlan MS (1998). Atmospheric deposition of semivolatile organic compounds to two forest canopies. Atmos Environ 32(10): 1799-1809.

Huwe JK and Smith DJ (2005). Laboratory and on-farm studies on the bioaccumulation and elimination of dioxins from a contaminated mineral supplement fed to dairy cows. J Agric Food Chem 53(6): 2362-70.

Iben C, Bohm J, Tausch H, Leibetseder J and Luf W (2003). Dioxin residues in the edible tissue of broiler chicken. J Anim Physiol Anim Nutr (Berl) 87(3-4): 142-8.

Ishida M (1993). Reduction of phthalate in chicken eggs, liver and meat by several cooking methods. J Food Hyg Soc Japan 34(6): 529-31.

Ishida M, Suyama K and Adachi S (1981). Occurrence of dibutyl and di(2-ethylhexyl) phthalate in chicken eggs. J Agric Food Chem 29(1): 72-4.

Jenkins KJ and Hidiroglou M (1986). Tolerance of the preruminant calf for selenium in milk replacer. J Dairy Sci 69(7): 1865-70.

Jenkins KJ and Winter KA (1973). Effects of selenium supplementation of naturally high selenium swine rations on tissue levels of the element. Can J Anim Sci 53: 561-67.

Johnson DE, Kienholz EW, Baxter JC, Spangler E and Ward GM (1981). Heavy metal retention in tissues of cattle fed high cadmium sewage sludge. J Anim Sci 52(1): 108-14.

Kan CA (1978). Accumulation of organochlorine pesticides in poultry: a review. J Agric Food Chem 26(5): 1051-5.

Kelly TJ, Czuczwa JM, Sticksel PR, Sticksel PR, Sverdrup GM, Koval PJ and Hodanbosi RF (1991). Atmospheric and tributary inputs of toxic substances to Lake Erie. J Great Lakes Res 17(4): 504-16.

Kerst M, Waller U, Reifenhauser W and Korner W (2004). Carry-over rates of dioxin-like PCB from grass to cow’s milk. Organohalogen Compd 66: 2440-4.

Kiwimae A, Swensson A, Ulfvarson U and Westoo G (1969). Methylmercury compounds in eggs from hens after oral administration of mercury compounds. J Agric Food Chem 17(5): 1014-6.

Kozak S and Forsberg CW (1979). Transformation of mercuric chloride and methylmercury by the rumen microflora. Appl Environ Microbiol 38(4): 626-36.

Ku PK, Ely WT, Groce AW and Ullrey DE (1972). Natural dietary selenium, -tocopherol and effect on tissue selenium. J Anim Sci 34(2): 208-11.

Lameiras J, Soares ME, Bastos ML and Ferreira M (1998). Quantification of total chromium and hexavalent chromium in UHT milk by ETAAS. Analyst 123(10): 2091-5.

Lamphere DN, Dorn CR, Reddy CS and Meyer AW (1984). Reduced cadmium body burden in cadmium-exposed calves fed supplemental zinc. Environ Res 33(1): 119-29.

Lane DA, Johnson ND, Hanely MJ, Schroeder WH and Ord DT (1992). Gas-and particle-phase concentrations of alpha-hexachlorocyclohexane, gamma-hexachlorocyclohexane, and hexachlorobenzene in Ontario air. Environ Sci Technol 26(1): 126-33.

Lasky T, Sun W, Kadry A and Hoffman MK (2004). Mean total arsenic concentrations in chicken 1989-2000 and estimated exposures for consumers of chicken. Environ Health Perspect 112(1): 18-21.

Latta DM and Donaldson WE (1986a). Lead toxicity in chicks: interactions with dietary methionine and choline. J Nutr 116(8): 1561-8.

Latta DM and Donaldson WE (1986b). Modification of lead toxicity and organ distribution by dietary sulfur amino acids in chicks (Gallus domesticus). Comp Biochem Physiol C 84(1): 101-4.

Leach RM, Jr., Wang KW and Baker DE (1979). Cadmium and the food chain: the effect of dietary cadmium on tissue composition in chicks and laying hens. J Nutr 109(3): 437-43.

Ling JR and Leach RM, Jr. (1979). Studies on nickel metabolism: interaction with other mineral elements. Poult Sci 58(3): 591-6.

Lorber M, Fries G, Winters D, Ferrario J and Byrne C (2000). A study of the mass balance of dioxins and furans in lactating cows in background conditions. Part 2: Mass balance and bioconcentration factors. Organohalogen Compd 46: 326-9.

Lutz S, Feidt C, Monteau F, Rychen G, Le Bizec B and Jurjanz S (2006). Effect of exposure to soil-bound polycyclic aromatic hydrocarbons on milk contaminations of parent compounds and their monohydroxylated metabolites. J Agric Food Chem 54(1): 263-8.

MacLachlan DJ (2008). Transfer of fat-soluble pesticides from contaminated feed to poultry tissues and eggs. Br Poult Sci 49(3): 290-8.

MacLachlan DJ (2009). Influence of physiological status on residues of lipophilic xenobiotics in livestock. Food Addit Contam Part A Chem Anal Control Expo Risk Assess 26(5): 692-712.

MacLachlan DJ (2010). Physiologically based pharmacokinetic (PBPK) model for residues of lipophilic pesticides in poultry. Food Addit Contam Part A Chem Anal Control Expo Risk Assess 27(3): 302-14.

MacLachlan DJ and Bhula R (2008). Estimating the residue transfer of pesticides in animal feedstuffs to livestock tissues, milk and eggs: a review. Aust J Experimental Agric 48(5): 589-98.

Maervoet J, Chu SG, De Vos S, Covaci A, Voorspoels S, De Schrijver R and Schepens P (2004). Accumulation and tissue distribution of selected polychlorinated biphenyl congeners in chickens. Chemosphere 57(1): 61-6.

Malisch R, Schmid P, Frommberger R and Furst P (1996). Results of a quality control study of different analytical methods for determination of PCDD in egg samples. Chemosphere 32(1): 31-44.

Maus RW, Martz FA, Belyea RL and Weiss MF (1980). Relationship of dietary selenium to selenium in plasma and milk from dairy cows. J Dairy Sci 63(4): 532-7.

McLachlan M and Richter W (1998). Uptake and transfer of PCDDs by cattle fed naturally contaminated feedstuffs and feed contaminated as a result of sewage sludge application: 1. Lactating cows. J Agric Food Chem 46(3): 1166-72.

McLachlan M, Thoma H, Reissinger M and Hutzinger O (1990). PCDD/F in an agricultural food chain. Part 1: PCDD/F mass balance of a lactating cow. Chemosphere 20(7-9): 1013-20.

McLachlan MS (1996). Bioaccumulation of hydrophobic chemicals in agricultural food chains. Environ Sci Technol 30(1): 252-9.

Meador JP, Stein JE, Reichert WL and Varanasi U (1995). Bioaccumulation of polycyclic aromatic hydrocarbons by marine organisms. Rev Environ Contam Toxicol 143: 79-165.

Mehennaoui S, Delacroix-Buchet A, Duche A, Enriquez B, Kolf-Clauw M and Milhaud G (1999). Comparative study of cadmium transfer in ewe and cow milks during rennet and lactic curds preparation. Arch Environ Contam Toxicol 37(3): 389-95.

Meluzzi A, Simoncini F, Sirri F, Vandi L and Giordani G (1996). Feeding hens diets supplemented with heavy metals (chromium, nickel and lead). Archiv fuer Gefluegelkunde 60(3): 119-25.

Moksnes K and Norheim G (1982). Selenium concentrations in tissues and eggs of growing and laying chickens fed sodium selenite at different levels. Acta Vet Scand 23(3): 368-79.

Muhlemann M, Sieber R, Schallibaum M and Zoller O (2006). Polycyclic aromatic hydrocarbons in Swiss dry feed for dairy cattle and contamination resulting in milk and meat - a risk assessment. Mitt Lebensm Hyg 97: 121-39.

Mullen AL, Stanley RE, Lloyd SR and Moghissi AA (1975). Absorption, distribution and milk secretion of radionuclides by the dairy cow IV. Inorganic radiomercury. Health Phys 28: 685-91.

Neathery MW, Miller WJ, Gentry RP, Stake PE and Blackmon DM (1974). Cadmium-109 and methyl mercury-203 metabolism, tissue distribution, and secretion into milk of cows. J Dairy Sci 57(10): 1177-83.

Ng YC (1982). A review of transfer factors for assessing the dose from radionuclides in agricultural products. Nucl Safety 23(1): 57-71.

NTP (2008). NTP Technical Report on the Toxicology and Carcinogenesis Studies of Sodium Dichromate Dihydrate (CAS NO. 7789-12-0) in F344/N Rats and B6C3F1 Mice (Drinking Water Studies). NTP TR 546, NIH Publication No. 08-5887, National Toxicology Program, Research Triangle Park, NC. Online at: http://ntp.niehs.nih.gov.

O'Dell GD, Miller WJ, King WA, Ellers JC and Jurecek H (1970). Effect of nickel supplementation on production and composition of milk. J Dairy Sci 53(11): 1545-8.

Ort JF and Latshaw JD (1978). The toxic level of sodium selenite in the diet of laying chickens. J Nutr 108(7): 1114-20.

Overby LR and Frost DV (1962). Nonretention by the chicken of the arsenic in tissues of swine fed arsanilic acid. Toxicol Appl Pharmacol 4: 745-51.

Parker CE, Jones WA, Matthews HB, McConnell EE and Hass JR (1980). The chronic toxicity of technical and analytical pentachlorophenol in cattle. II. Chemical analyses of tissues. Toxicol Appl Pharmacol 55(2): 359-69.

Peoples SA (1964). Arsenic toxicity in cattle. Ann N Y Acad Sci 111: 644-9.

Petreas MX, Goldman LR, Hayward DG, Chang RR, Flattery JJ, Wiesmuller T and Stephens RD (1991). Biotransfer and bioaccumulation of PCDD/PCDFs from soil: Controlled exposure studies of chickens. Chemosphere 23(11-12): 1731-41.

Pirard C and De Pauw E (2005). Uptake of polychlorodibenzo-p-dioxins, polychlorodibenzofurans and coplanar polychlorobiphenyls in chickens. Environ Int 31(4): 585-91.

Pirard C and De Pauw E (2006). Toxicokinetic study of dioxins and furans in laying chickens. Environ Int 32(4): 466-9.

Pizarro I, Gomez MM, Fodor P, Palacios MA and Camara C (2004). Distribution and biotransformation of arsenic species in chicken cardiac and muscle tissues. Biol Trace Elem Res 99(1-3): 129-43.

Potter GD, McIntyre DR and Vattuone GM (1972). Metabolism of 203 Hg administered as HgCl 2 in the dairy cow and calf. Health Phys 22(1): 103-6.

Pribilincova J, Maretta M, Janotikova I and Marettova E (1995). The effect of cadmium treatment on breeding hens and cocks and early viability of their chickens. Vet Med (Praha) 40(11): 353-7.

RTI. (2005). *Research Triangle Institute. Methodology for predicting cattle biotransfer factors*. RTI Project Number 08860.002.015, Research Triangle Institute, Research Triangle Park, NC, USA.

Schaum J, Schuda L, Wu C, Sears R, Ferrario J and Andrews K (2003). A national survey of persistent, bioaccumulative, and toxic (PBT) pollutants in the United States milk supply. J Expo Anal Environ Epidemiol 13(3): 177-86.

Schuler F, Schmid P and Schlatter C (1997a). The transfer of polychlorinated dibenzo-p-dioxins and dibenzofurans from soil into eggs of foraging chicken. Chemosphere 34(4): 711-8.

Schuler F, Schmid P and Schlatter C (1997b). Transfer of airborne polychlorinated dibenzo-p-dioxins and dibenzofurans into dairy milk. J Agric Food Chem 45(10): 4162-7.

Scott ML, Zimmermann JR, Marinsky S, Mullenhoff PA, Rumsey GL and Rice RW (1975). Effects of PCBs, DDT, and mercury compounds upon egg production, hatchability and shell quality in chickens and Japanese quail. Poult Sci 54(2): 350-68.

Sharma RP and Street JC (1980). Public health aspects of toxic heavy metals in animal feeds. J Am Vet Med Assoc 177(2): 149-53.

Sharma RP, Street JC, Shupe JL and Bourcier DR (1982). Accumulation and depletion of cadmium and lead in tissues and milk of lactating cows fed small amounts of these metals. J Dairy Sci 65(6): 972-9.

Sharma RP, Street JC, Verma MP and Shupe JL (1979). Cadmium uptake from feed and its distribution to food products of livestock. Environ Health Perspect 28: 59-66.

Shupe JL, Miner ML and Greenwood DA (1964). Clinical and pathological aspects of fluorine toxicosis in cattle. Ann N Y Acad Sci 111: 618-37.

Slob W, Olling M, Derks HJ and de Jong AP (1995). Congener-specific bioavailability of PCDD/Fs and coplanar PCBs in cows: laboratory and field measurements. Chemosphere 31(8): 3827-38.

Soares JH, Miller D, Lagally H, Stillings BR, Bauersfeld P and Cuppett S (1973). The comparative effect of oral ingestion of methyl mercury on chicks and rats. Poult Sci 52(452-8).

Spitaler M, Iben C and Tausch H (2005). Dioxin residues in the edible tissue of finishing pigs after dioxin feeding. J Anim Physiol Anim Nutr (Berl) 89(3-6): 65-71.

Stephens RD, Petreas MX and Hayward DG (1995). Biotransfer and bioaccumulation of dioxins and furans from soil: chickens as a model for foraging animals. Sci Total Environ 175(3): 253-73.

Stevens JB (1991). Disposition of toxic metals in the agricultural food chain: 1. Steady-state bovine milk biotransfer factors. Environ Sci Technol 25(7): 1289-94.

Stevens JB (1992). Disposition of toxic metals in the agricultural food chain. 2. Steady-state bovine tissue biotransfer factors. Environ Sci Technol 26(10): 1915-21.

Stoddard GE, Bateman GQ, Harris LE and Shupe JLGDA (1963). Effects of fluorine on dairy cattle. IV. Milk production. J Dairy Sci 46(7): 720-6.

Surendra Nath B, Unnikrishnan V, Preeja CN and Rama Murthy MK (2000). A study on the transfer of organochlorine pesticide residues from the feed of the cattle into their milk. Pesticide Res J 12(1): 68-73.

Suttie JW, Phillips PH and Miller RF (1958). Studies of the effects of dietary sodium fluoride on dairy cows. III. Skeletal and soft tissue fluorine deposition and fluorine toxicosis. J Nutr 65(2): 293-304.

Szokolay A, Madaric A and Uhnak J (1977). Relative cumulation of beta-BHC in ecological and biological system. J Environ Sci Health B 12(3): 193-212.

Thomas GO, Sweetman AJ and Jones KC (1999a). Input-output balance of polychlorinated biphenyls in a long-term study of lactating dairy cows. Environ Sci Technol 33(1): 104-12.

Thomas GO, Sweetman AJ and Jones KC (1999b). Metabolism and body-burden of PCBs in lactating dairy cows. Chemosphere 39(9): 1533-44.

Thomas GO, Sweetman AJ, Lohmann R and Jones KC (1998). Derivation and field testing of air-milk and feed-milk transfer factors for PCBs. Environ Sci Technol 32(22): 3522-8.

Traag WA, Kan CA, van der Weg G, Onstenk C and Hoogenboom LA (2006). Residues of dioxins (PCDD/Fs) and PCBs in eggs, fat and livers of laying hens following consumption of contaminated feed. Chemosphere 65(9): 1518-25.

U.S. EPA. (1997). *Mercury Study Report to Congress Volume III: Fate and Transport of Mercury in the Environment, Chapter 3, Measured Concentrations.* . U.S. Environmental Protection Agency, EPA-452/R-97-005.

U.S. EPA. (2005). *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities*. U.S. Environmental Protection Agency, Office of Solid Waste, EPA 530-R-05-006. Online at: www.epa.gov/osw.

USDA. (1975). *Composition of Foods: Raw, Processed, Prepared*. Agriculture Handbook No. 8, U.S. Department of Agriculture.

Vadnjal R, Stibilj V, Holcman A and Dermelj M (1997). Distribution of selenium and iodine in the tissues of laying hens fed with As2O3 added to the diet. Zootehnika 70: 195-200.

Van Bruwaene R, Gerber GB, Kirchmann R, Colard J and Van Kerkom J (1984). Metabolism of 51Cr, 54Mn, 59Fe and 60Co in lactating dairy cows. Health Phys 46(5): 1069-82.

van den Hoek J, Salverda MH and Tuinstra LGMT (1975). The excretion of six organochlorine pesticides into the milk of the dairy cow after oral administration. Neth Milk Dairy J 29: 66-78.

Van Eijkeren JC, Zeilmaker MJ, Kan CA, Traag WA and Hoogenboom LA (2006). A toxicokinetic model for the carry-over of dioxins and PCBs from feed and soil to eggs. Food Addit Contam 23(5): 509-17.

Vreman K, Poortvliet LJ and van den Hoek J (1980). Transfer of organochlorine pesticides from feed into the milk and body fat of cows. Long-term experiment with intake at low levels. Neth Milk Dairy J 34: 87-105.

Vreman K, Tuinstra LGMT, Van den Hoek J, Bakker J, Roos AH, de Visser H and Westerhuis JH (1976). Aldrin, heptachlor and beta-hexachlorocyclohexane to dairy cows at three oral dosages. 1. Residues in milk and body fat of cows early and late in lactation. Neth J Agric Sci 24: 197-207.

Vreman K, van der Veen NG, van Der Molen EJ and de Ruig WG (1986). Transfer of cadmium, lead, mercury and arsenic from feed into milk and various tissues of dairy cows: chemical and pathological data. Neth J Agric Sci 34: 129-44.

Vreman K, Van der Veen NG, Van Der Molen EJ and De Ruig WG (1988). Transfer of cadmium, lead, mercury and arsenic from feed into tissues of fattening bulls: chemical and pathological data. Neth J Agric Sci 36: 327-38.

Willett LB, Blanford JJ, Becker CJ and Bromund RH. (1994). *Distribution of Lead in Lactating Cows*. 145. Special circular-Ohio Agricultural Research and Development Center, OARDC Dairy Science, pp. 9-11.

Willett LB, Liu TT and Fries GF (1990). Reevaluation of polychlorinated biphenyl concentrations in milk and body fat of lactating cows. J Dairy Sci 73(8): 2136-42.

Williams S and Mills PA (1964). Residues in milk of cows fed rations containing low concentrations of five chlorinated hydrocarbon pesticides. J A O A C 47(6): 1124-8.

Appendix L Activity Data Analysis Report

L.1 Introduction

The Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) staff have updated the exposure assessment methodologies and the data used for conducting Health Risk Assessments (HRA) as prescribed under the Air Toxics “Hot Spots” Information and Assessment Act (Assembly Bill 2588; Health and Safety Code Section 44300 et seq.). The mandates of the Air Toxics "Hot Spots" Act are to collect emission data, to identify facilities having localized impacts, to ascertain health risks, to notify nearby residents of significant risks, and to reduce those significant risks to acceptable levels. This report focuses on two of the exposure variables (i.e. exposure duration and exposure frequency) used in estimating a person’s lifetime average daily dose by considering the time a person lives in his or her primary residence and the time a person spends daily at home.

Staff looked into various data sources to determine the residency duration at the household level and the daily activity pattern at the individual level. The data sources the staff examined include the National Human Activity Pattern Survey (NHAPS), the National Household Travel Surveys (NHTS), the National Longitudinal Surveys, the American Time Use Survey Data Extract Builder, the Integrated Public Use Microdata Series (IPUMS-USA) census data, the Southern California Association of Governments (SCAG) 2000 regional travel survey data, and the California Department of Transportation (Caltrans) 2000-2001 California Statewide Household Travel Survey (CHTS) data. The staff determined that IPUMS-USA, SCAG 2000 regional travel survey, and Caltrans 2000-2001 CHTS represent the most current and California-specific residence and activity data and therefore were used as the basis for the conclusions stated in this report.

Results show that, from 2006 to 2009, over 91% of California householders had lived at their current home address for less than 30 years, and over 63% of householders had lived at their current residence for 9 years or less. No data were available for householders who lived in their homes over a 70 year period.

The 2000-2001 CHTS data show that, on average, Californians spend approximately 73% of their time at home per day. When looking at the data by age group, the time increases to 85% for children under 2 years old. Individuals that are 2 years or older, but less than 16 years old, spend 72% of their time at home whereas Californians that are 16 years or older spend 73% of their time at home.

L.2 Data Sources Analyzed

L.2.1 IPUMS-USA data

IPUMS-USA consists of more than fifty samples of the American population drawn from fifteen federal censuses and from the American Community Surveys (ACS). ACS is a nationwide survey that collects and produces population and housing information every year from about three million selected housing unit addresses across every county in the nation (ACS). IPUMS-USA samples, which draw on every surviving census from 1850-2000 and the 2000-2009 ACS samples, collectively constitute the quantitative information on long-term changes in the American population. These records for the period since 1940 only identify geographic areas with equal or larger than 100,000 residents (250,000 in 1960 and 1970) (IPUMS-USA).

IPUMS-USA census data contain residency duration, travel to work data, residence and work location, age, household and personal income, and ethnicity data.

L.2.2 SCAG Year 2000 Post-Census Regional Household Travel Survey Data

The second set of data the staff evaluated was the Post-Census Regional Household Travel Survey sponsored by the Southern California Association of Governments (SCAG). SCAG is the federally designated metropolitan planning organization (MPO) for the Los Angeles region of California. The survey targeted households in the six counties of the SCAG region: Imperial, Los Angeles, Orange, San Bernardino, Riverside, and Ventura (SCAG, 2003).

SCAG survey has data of time spent at home, trip data, geo code for locations, home address, age, income, ethnicity, and limited residency duration (months lived at home location).

L.2.3 Caltrans 2000-2001 California Statewide Household Travel Survey Data

Caltrans maintains statewide household travel data to estimate, model, and forecast travel throughout the State. The information is used to help in transportation planning, project development, air quality analysis, and other programs. The CHTS obtained sample household socioeconomic and travel data at the regional and statewide levels.

In the raw survey database obtained from Caltrans, there are data about trip duration, activity duration, location type, geo code for destination, address, age, income, and ethnicity. There are no data about residency duration.

Caltrans is currently developing a new 2011-2012 CHTS, which is a joint effort among Caltrans, SCAG and other MPOs. ARB is part of the Steering Committee.

L.2.4 Data Sources Summary

Table L.1 summarizes the activity data sources the staff analyzed, which include IPUMS census data, SCAG 2000 regional travel survey data, and Caltrans 2000-2001 CHTS data. It shows the data availability based on the HRA related categories.

Table L.1 Activity Data Sources

Sources			
HRA related Categories	**IPUMS-USA Census Data 2000-2009**	**SCAG 2000 Travel Survey**	**Caltrans 2000-2001 CHTS**
Residency duration	Year moved in	Months lived at home location	N/A*
Time at home per day	N/A	At home activity duration	At home activity duration
Time away from home	Hours worked, Travel time to work	Trip duration, activity duration	Trip duration, activity duration
Trip distance	N/A	Geo code for origin and destination	Geo code for destination
Residence location	City. No zip code	Address	Address
Age	Yes	Yes	Yes
Income level	Income Variables	Household income	Household income
Seasonal trend	N/A	N/A	N/A
Ethnicity	Yes	Yes	Yes
Data Set	Federal censuses (1850-2000), American Community Surveys (2000-2009)	2000-2002 Six-county Los Angeles region of CA	2000-2001 CA Statewide weekday travel survey

* N/A: Data are not available.

L.3 Methodologies and Findings:

In this section, we outline the methodologies we used in each of the data sources to estimate a person's time period lived in his or her residence and the time spent in different activities each day. We also examined how different environmental factors such as socioeconomic status, age, and ethnicity affect residency duration and daily activity patterns. We conclude with a discussion of the findings of each of the data sources.

L.3.1 IPUMS-USA data

L.3.1.1 Methodology

The staff used IPUMS online analysis tool (IPUMS Tool) to analyze the residency duration data based on ACS 2006-2009 data. The results are compiled and discussed below.

There are IPMUS_USA ACS data from 2000 to 2005 as well. However, the IPMUS_USA ACS data from 2006 to 2009 are more recent and have the same sample size percentage (i.e. 1%) for each year. In addition, these data include persons in group quarters and the smallest identifiable geographic unit is the Public Use Microdata Area (PUMA) containing at least 100,000 persons (IPUMS Samples). Group quarters consist of both institutions and units housing either a primary family or a primary individual plus a given number of persons unrelated to the head (IPUMA GQ).

L.3.1.2 Findings and Discussions

L.3.1.2.1 California Statewide Residency Duration Distributions

Table L.2 presents California statewide time moved into residence distributions compiled from the analysis results of ACS 2006, 2007, 2008, 2009 single year samples and ACS 2006-2008 3-year sample using IPUMS-USA online data analyzing tool. The time moved into residence variable has 7 values in ACS data as listed in "Time Moved into Residence" column in Table L.2, including "5 to 9 years ago" and "30 years ago". The statistical data provided have the samples' household weight applied. Household weight indicates how many households in the U.S. population are represented by a given household in an IPUMS sample (IPUMS Weights). Each cell besides the row and column headers in Table L.2 contains a household percent and the number of householders presented by that percent.

In summary, IPUMS-USA ACS 2006 to 2009 data show that the percentage of the California householders with a residency period of 30 years or greater is less than 9%. In other words, over 91% of California householders had lived in their current residence location for less than 30 years. These data also show that over 63% California householders had lived at their current residence for 9 years or less.

Table L.2* California Statewide Time Moved into Residence Distribution by Year

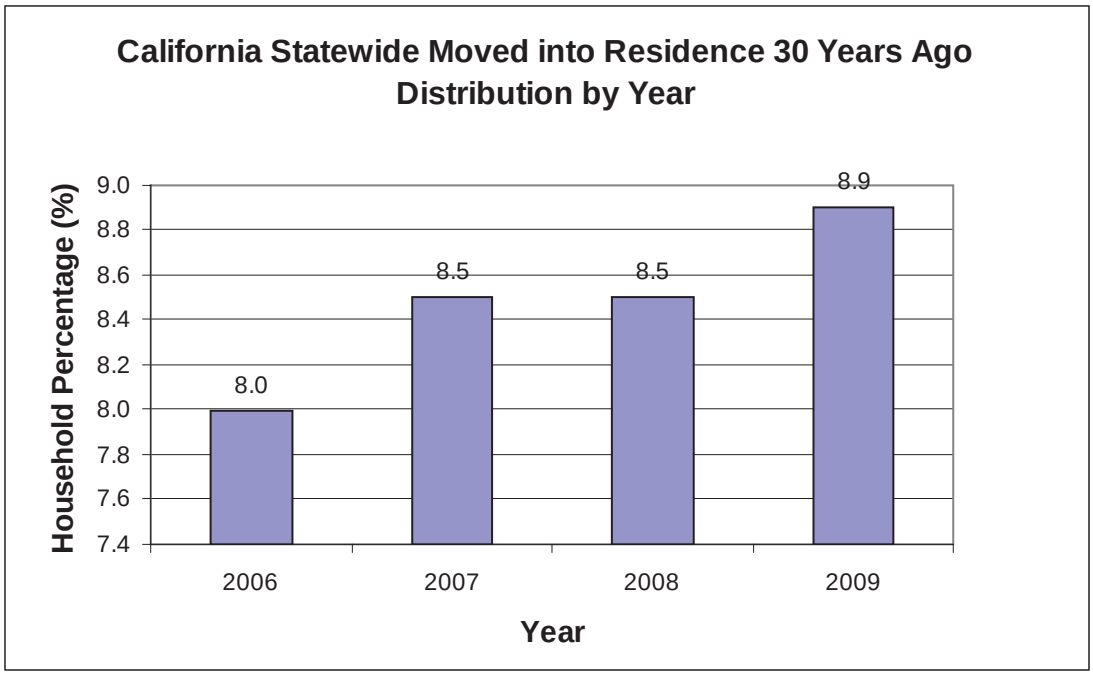
(Weighted Household Percent and Number)

Time Moved into Residence	2006	2007	2008	2006-2008 3-year Sample	2009
12 months or less	**17.2** 2,084,533.0	**15.9** 1,939,774.0	**15.4** 1,871,049.0	**16.2** 1,968,717.0	**15.7** 1923501
13 to 23 months ago	**7.5** 910,536.0	**6.9** 838,322.0	**6.5** 796,030.0	**7** 848,579.0	**6.4** 783261
2 to 4 years ago	**21.9** 2,665,547.0	**22.9** 2,795,422.0	**23.3** 2,834,921.0	**22.7** 2,768,053.0	**20.3** 2482340
5 to 9 years ago	**19.8** 2,411,057.0	**20.1** 2,449,371.0	**20.1** 2,448,160.0	**20** 2,434,099.0	**20.9** 2554979
10 to 19 years ago	**17.6** 2,141,482.0	**17.7** 2,162,519.0	**18.1** 2,208,805.0	**17.8** 2,169,353.0	**18.9** 2311981
20 to 29 years ago	**7.9** 960,926.0	**8.1** 982,699.0	**8.0** 979,208.0	**8.0** 974,196.0	**8.7** 1067833
30 years ago	**8.0** 977,136.0	**8.5** 1,032,572.0	**8.5** 1,038,566.0	**8.3** 1,014,849.0	**8.9** 1090992
TOTAL	***100.0*** *12,151,217.0*	***100.0*** *12,200,679.0*	***100.0*** *12,176,739.0*	***100.0*** *12,177,846.0*	***100.0*** *12214887*

* IPUMS-USA ACS 2006 to 2009 data with household weight applied. As of March 2011, there is no IPUMS-USA multi-year sample with ACS 2009 sample included yet.

Figure L.1 graphically depicts the 2006 to 2009 statewide householder percentages of Californians that moved into their current home location 30 years ago. From 2006 to 2009, this figure shows an increase in the percentage of statewide householders that moved into residence 30 years ago.

Figure L.1*

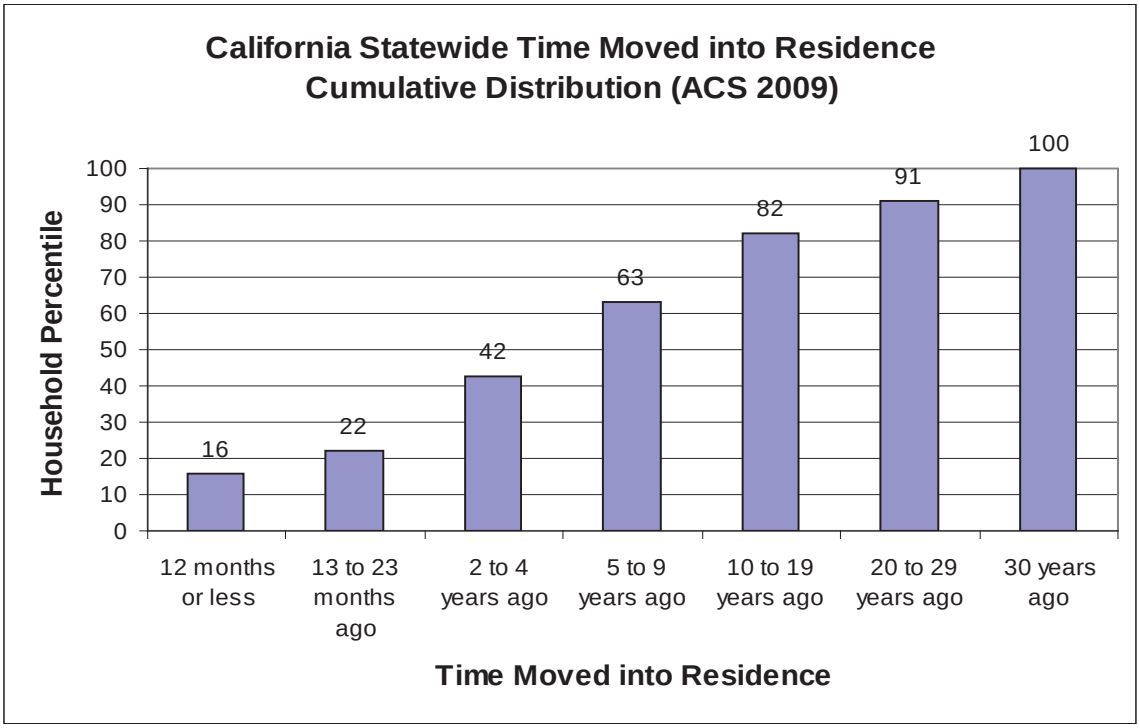


* IPUMS-USA ACS 2006, 2007, 2008, and 2009 single year samples with household weight applied.

Figure L.2 and Figure L.3, respectively, show the California statewide time moved into residence cumulative distributions using IPUMS-USA ACS 2009 sample and 2006-2008 3-year sample with household weight applied. Both of these figures show that over 90 percent of California householders had lived at their current home address for less than 30 years, and approximately 63 to 66 percent of the householders had lived at their current residency location for 9 years or less.

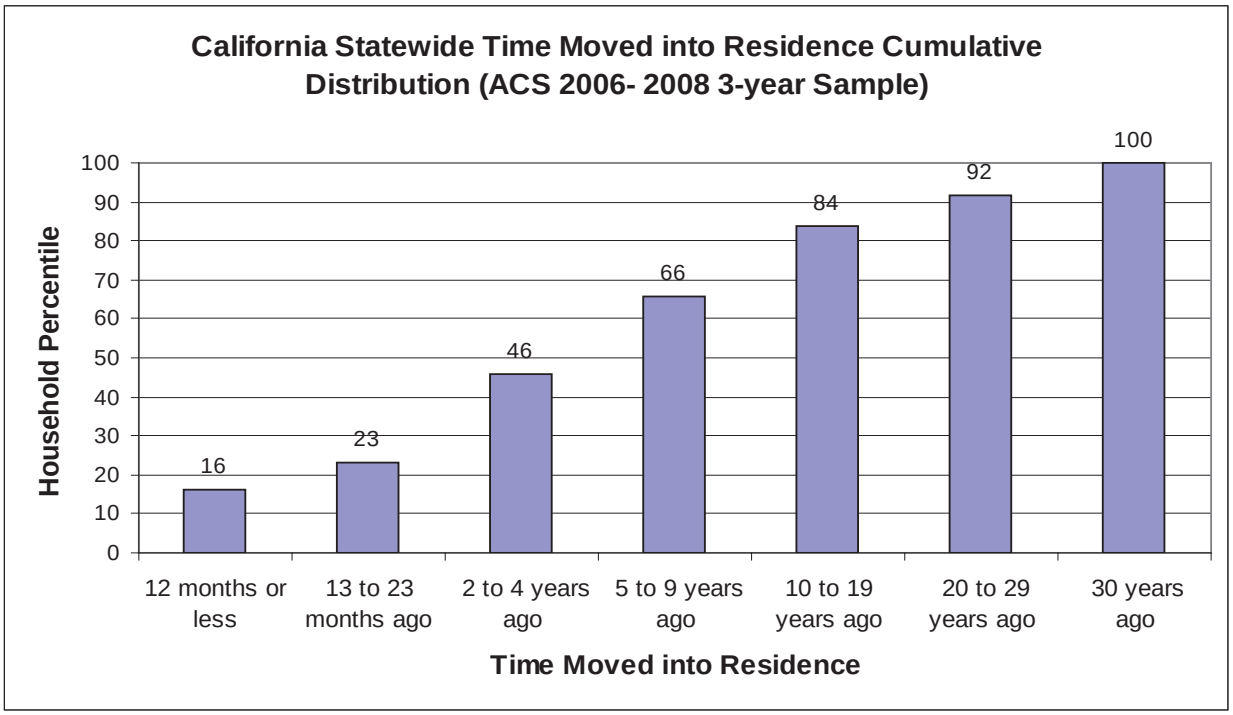
See Supplemental Information section (page 29) for additional information on time moved into residence distributions by California householder's ethnicity, age, and household income from IPUMS-USA ACS 2009 data.

Figure L.2*



* IPUMS-USA ACS 2009 data with household weight applied.

Figure L.3*



* IPUMS-USA ACS 2006-2008 3-year sample with household weight applied. As of March 2011, there is no IPUMS-USA multi-year sample with ACS 2009 sample included available yet.

L.3.1.2.2 Evaluation of Populations and Residency Duration Distributions for California Cities

Table L.3 and Figure L.4 display the populations and population changes for 8 selected California cities from IPUMS-USA ACS 2006 and ACS 2009 data with person weight applied. Person weight indicates how many persons in the U.S. population are represented by a given person in an IPUMS sample (IPUMS Weights). These 8 cities have populations over 100,000 from IPUMS-USA ACS 2006 and 2009 data, and were selected to represent the regions of California and to include an Environmental Justice community (Fresno, CA). If an area consisted of less than 100,000 persons then it was combined with another area so that the total population would be greater than 100,000 persons. The exhaustive distribution data from IPUMS-USA ACS 2006 and 2009 samples contain 41 identifiable California cities.

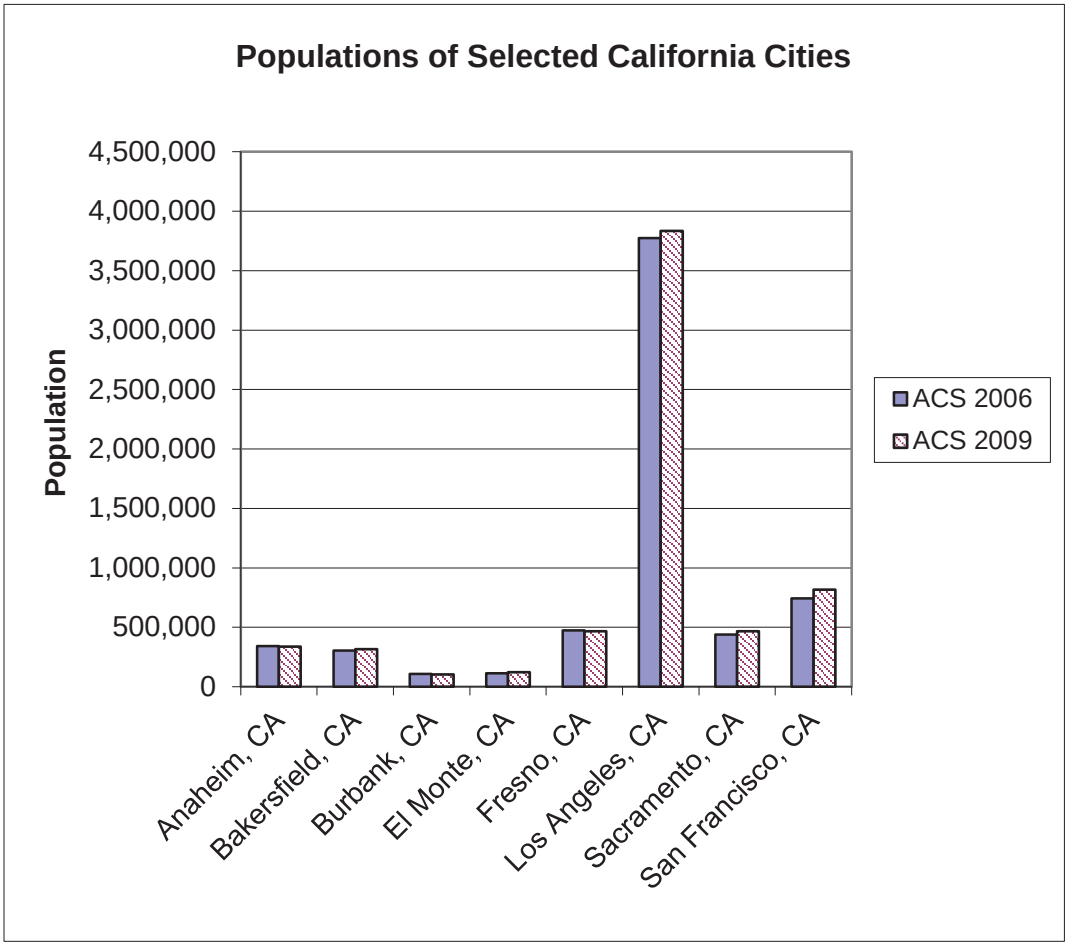
Table L.3* Comparison of Populations of Selected California Cities

(IPUMS-USA ACS 2006 and 2009)

California City	Anaheim	Bakersfield	Burbank	El Monte	Fresno	Los Angeles	Sacramento	San Francisco
2006	343,120	304,813	107,540	113,644	474,466	3,775,106	438,385	744,389
2009	337,966	316,313	103,096	121,183	466,466	3,832,554	466,492	815,575
Population Change Percent	-1.5	3.8	-4.1	6.6	-1.7	1.5	6.4	9.6

* IPUMS-USA ACS 2006 and 2009 data with person weight applied.

Figure L.4*



* IPUMS-USA ACS 2006 and 2009 data with person weight applied.

Table L.4 and L.5 display the time moved into residence distributions for the 8 selected California cities from IPUMS-USA ACS 2006 and 2009 data, respectively, with household weight applied. Both tables show that 89% to 96% of householders had moved out of their residence within 30 years. In other words, about 4% to 11% householders had lived at their current residence for 30 years or longer. The residency duration data from IPUMS-USA ACS also indicate that, for all the 41 identifiable California cities, about 1% to 15% of householders had lived at their current residence for 30 years or longer in 2006, whereas 2% to 15% of householders had lived at their current residence for 30 years or longer in 2009.

Table L.4* Time Moved into Residence Distribution for Selected California Cities Weighted Household Percent and Samples (IPUMS-USA ACS 2006)

Time Moved into Residence	Anaheim, CA	Bakersfield, CA	Burbank, CA	El Monte, CA	Fresno, CA	Los Angeles, CA	Sacramento, CA	San Francisco, CA
12 months or less	**19.1** 18,845	**23.6** 23,729	**11.3** 4,847	**11** 3,083	**22** 33,457	**15.8** 200,769	**21.9** 37,111	**15.8** 50,869
13 to 23 months ago	**8.1** 8,021	**9.1** 9,194	**9.9** 4,236	**6.1** 1,715	**7.2** 10,896	**6.4** 81,792	**9.3** 15,778	**7.9** 25,535
2 to 4 years ago	**22.9** 22,542	**25.9** 26,028	**21.8** 9,314	**23** 6,456	**24.3** 36,928	**21.8** 278,034	**23.2** 39,271	**21** 67,837
5 to 9 years ago	**21.6** 21,324	**18.9** 19,038	**23.2** 9,924	**23.1** 6,469	**19.8** 30,086	**22.3** 284,354	**17.7** 30,006	**15.6** 50,166
10 to 19 years ago	**15.6** 15,341	**13.3** 13,427	**15.5** 6,649	**18.4** 5,177	**14.9** 22,728	**18.1** 231,199	**11.2** 18,986	**20.2** 65,170
20 to 29 years ago	**4.9** 4,838	**5.3** 5,373	**7.5** 3,194	**9.9** 2,768	**5.6** 8,512	**7.3** 93,569	**7.8** 13,134	**9** 28,989
30 years ago	**7.8** 7,654	**3.8** 3,857	**10.9** 4,651	**8.5** 2,397	**6.3** 9,554	**8.2** 104,450	**8.8** 14,939	**10.5** 33,980
TOTAL	***100*** *98,565*	***100*** *100,646*	***100*** *42,815*	***100*** *28,065*	***100*** *152,161*	***100*** *1,274,167*	***100*** *169,225*	***100*** *322,546*

* IPUMS-USA ACS 2006 data with household weight applied.

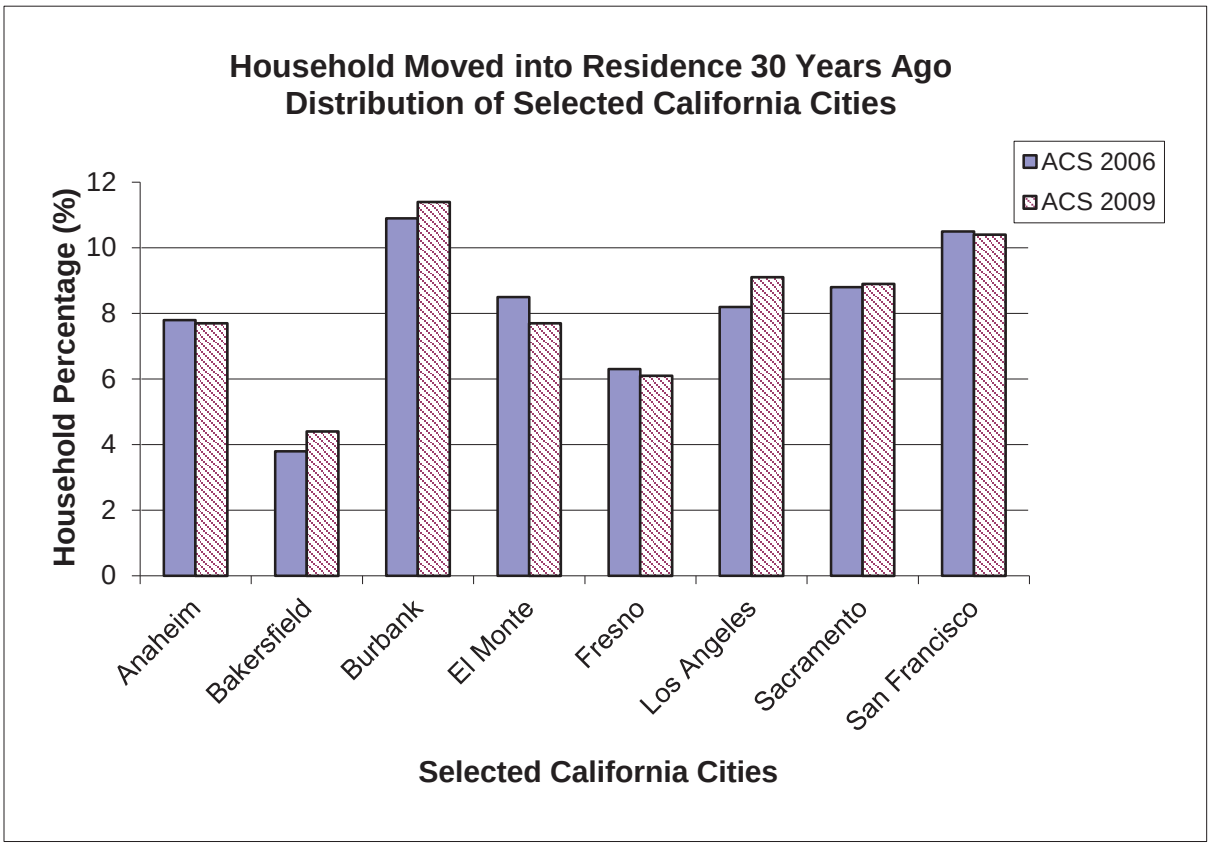
Table L.5* Time Moved into Residence Distribution for Selected California Cities Weighted Household Percent and Samples (IPUMS-USA ACS 2009)

Time Moved into Residence	Anaheim, CA	Bakersfield, CA	Burbank, CA	El Monte, CA	Fresno, CA	Los Angeles, CA	Sacramento, CA	San Francisco, CA
12 months or less	**15.8** 15,554	**21.3** 21,302	**17.5** 6,907	**11** 2,995	**21.3** 31,605	**15.5** 200,860	**23** 40,825	**14.8** 48,036
13 to 23 months ago	**6.5** 6,428	**7.9** 7,875	**6.3** 2,475	**6.9** 1,888	**8.8** 13,032	**5.7** 74,089	**8.4** 14,879	**7** 22,627
2 to 4 years ago	**22.7** 22,405	**27.1** 27,146	**19.2** 7,580	**19.7** 5,388	**19.8** 29,474	**20.3** 263,922	**22.3** 39,562	**21.9** 71,210
5 to 9 years ago	**21.1** 20,817	**20.4** 20,411	**21.5** 8,507	**26.8** 7,337	**20.2** 29,998	**21.6** 279,991	**17.4** 30,875	**18.7** 60,640
10 to 19 years ago	**19.2** 18,951	**14.6** 14,640	**18.7** 7,391	**17.2** 4,692	**16.9** 25,153	**20.2** 262,938	**13.2** 23,382	**18.6** 60,314
20 to 29 years ago	**7.1** 6,964	**4.2** 4,241	**5.5** 2,170	**10.7** 2,932	**6.9** 10,258	**7.6** 98,225	**6.7** 11,848	**8.7** 28,132
30 years ago	**7.7** 7,591	**4.4** 4,443	**11.4** 4,504	**7.7** 2,094	**6.1** 8,989	**9.1** 118,599	**8.9** 15,830	**10.4** 33,631
TOTAL	***100*** *98,710*	***100*** *100,058*	***100*** *39,534*	***100*** *27,326*	***100*** *148,509*	***100*** *1,298,624*	***100*** *177,201*	***100*** *324,590*

* IPUMS-USA ACS 2009 data with household weight applied.

Figure L.5 shows the distribution of householders with residency periods of 30 years or greater for the 8 selected California cities from IPUMS-USA ACS 2006 and ACS 2009 data with household weight applied.

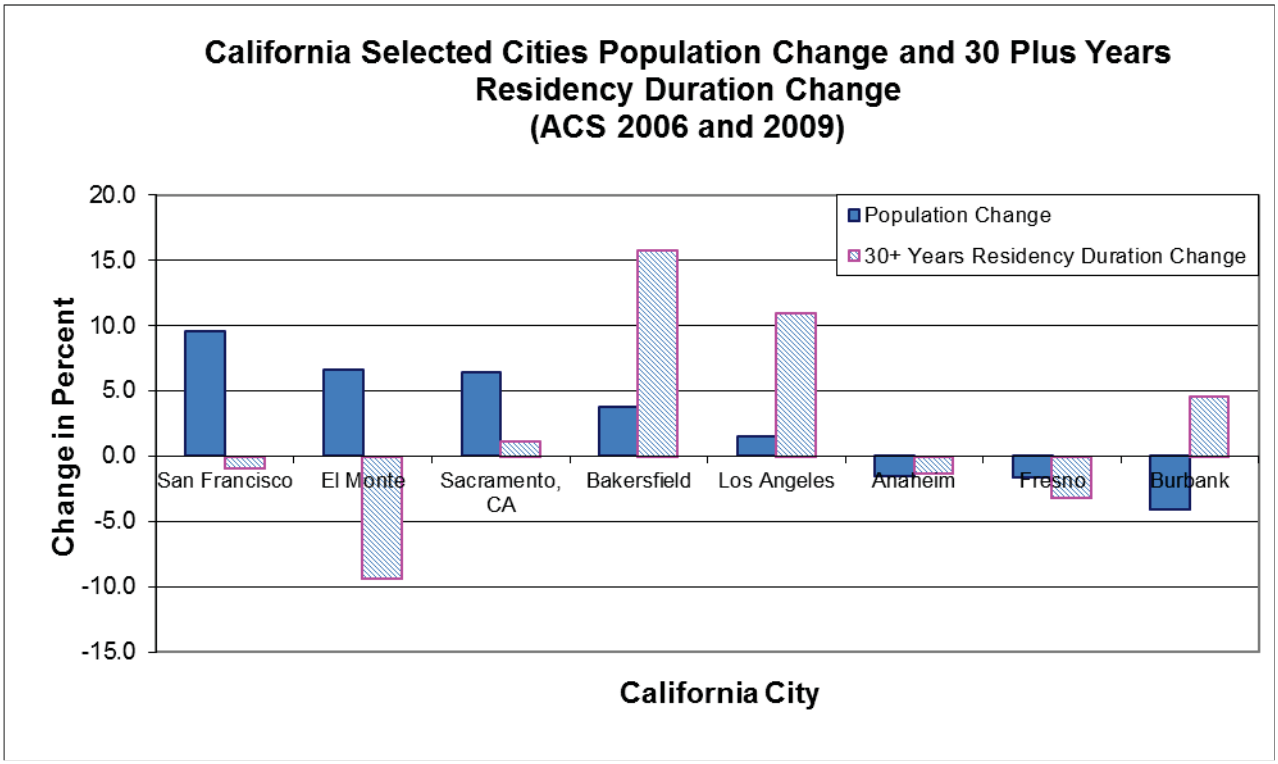
Figure L.5*



* IPUMS-USA ACS 2006 and 2009 data with household weight applied.

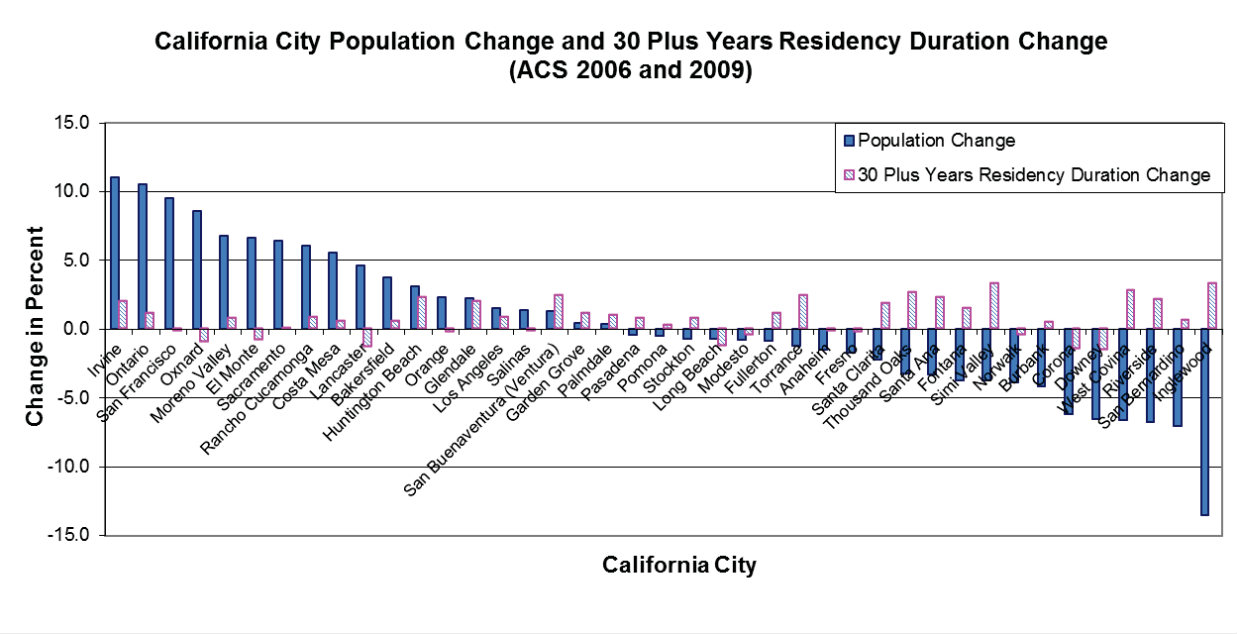
Staff also analyzed the population changes and the 30 years or greater residency duration changes for both the 8 selected cities and the 41 identifiable California cities using IPUMS-USA ACS 2006 to ACS 2009 data. The purpose of this analysis is to see if a rapidly growing city has a different pattern of residency durations. The results are illustrated in Figure L.6 and Figure L.7 respectively. There is no obvious correlation found between the population changes and the 30 years or greater residency duration changes. Figure L.7 shows that, when the population increased from 2006 and 2009, 13 cities showed an increase in 30 years or greater residency duration while 6 cities showed a decrease in 30 years or greater residency duration. And when the population decreased from 2006 to 2009, 15 cities showed an increase in 30 years or greater residency duration while 7 cities showed a decrease in 30 years or greater residency duration.

Figure L.6*



* IPUMS-USA ACS 2006 and 2009 data with household weight applied to the residency duration data, and person weight applied to population data.

Figure L.7*



* IPUMS-USA ACS 2006 and 2009 data with household weight applied to the residency duration data, and person weight applied to population data.

L.3.1.3 Limitations of the IPUMS-USA data for Our Purposes

The ideal data for our purposes would be longitudinal data on the duration of residence of individuals. The IPUMS collects information on how long the person has been in the current residence, but not previous residences. People may continue at the current residence for an indefinite period of time. Likewise people who report living in the current residence for a short period of time may have lived in the previous residence for an extended period time. This could be the case with older people who have recently moved to assisted living. Second, data on the amount of time that a person might have lived beyond thirty years were not collected. There is therefore no way of knowing the number of people who may have lived in the same residence for 40 or 50 years. Third, geographic areas with fewer than 100,000 inhabitants are not identifiable so the impact of living in a smaller community on residency time in California could not be determined. The data are binned into intervals that are as much as 9 years at the longer residency times. These data are the only California specific data that we could locate however, and are generally supportive of the nationwide data.

L.3.2 SCAG Year 2000 Post-Census Regional Household Travel Survey Data

L.3.2.1 Methodology

The survey collected demographic information about persons and households. It also captured activity and travel information for household members during a 24-hour or 48-hour timeframe. The survey coincides with 2000-2001 CHTS. According to the 2000 Census, this region had 5,386,491 households. The total number of households that participated the survey and met the criteria for a completed record was 16,939 (SCAG, 2003). In the survey report, there are some trip time and age information.

Using the SCAG survey database, a statistical analysis for the regional average time spent at home per day was performed.

L.3.2.2 Findings and Discussions

The average time at home per person per day was determined to be 17.6 hours, which is about 73% of a day. This result is based on 44,344 person day records without any weight factor applied.

The residency duration data (months lived at home location) in the database are labeled as 1 to 12, 98-unknown, and 99-refused. Label 1 to 11 represents 1 to 11 months lived at home location, whereas label 12 represents 12 plus months lived at home location. No additional data were collected on residency duration. Therefore, the residency duration data from SCAG survey are limited for long-term health risk assessment evaluations.

L.3.2.3 Limitations on the Use of SCAG Household Travel Survey Data

The limitations of SCAG travel survey data include that the time spent at home analysis does not have weight factors applied due to insufficient user information on weights for personal level analysis (SCAG Manual) and the residency duration is not further categorized for a period that is 12 months or longer, which limits the data usage for long-term health risk assessment.

L.3.3 Caltrans 2000-2001 California Statewide Household Travel Survey Data

L.3.3.1 Methodology

The Survey was “activity” based and included in-home activities and any travel to activity locations. The Survey was conducted among households in each of the 58 counties throughout the State and grouped by region to provide a snapshot of both regional and interregional travel patterns. The participating households were asked to record travel information in their diaries for a specified 24-hour or 48-hour period. The Survey produced a sample size of 17,040 randomly selected households with an overall standard error of 0.8% at the 95% confidence level with respect to household level attributes at the statewide level of analysis (CHTS, 2003).

There are statistical survey reports about income, region, trip purpose, and trip time (home-work travel time percent by five minutes intervals by region). However, no report is based on travel distance, activity duration, season, or weekend.

A statistical analysis was performed by the staff using the CHTS database for the statewide average time spent at home per person per day. The result is based on 40,696 person day respondents’ records without any population weight factor applied.

Further statistical analysis gave us the statewide time at home average by age group, income level, and ethnicity. Time at home by age group and ethnicity results are based on 40,653 person day records. Time at home by income level result is based on 40,696 person day records. These results don’t have any weight factors applied. And five percent of the person day records are weekend records.

L.3.3.2 Findings and Discussions

L.3.3.2.1 California Statewide Average Time Spent at Home and Distributions by Age, Income, and Ethnicity

The statewide average time spent at home per person per day was determined to be 17.5 hours (including weekend samples), which is 73% of a day. This statewide average time at home percentage is about the same as the SCAG's regional average time at home percentage based on its 2000 regional travel survey data.

Table L.6 and Figure L.8 demonstrate California statewide time spent at home distribution by age group. The results show that children less than 2 years old spend 85% of their time at home, which is 12% more than the statewide average of 73%. Children in the age group 2 to <16 spend 72% of their time at home, which is a little less than the statewide average time at home.

Age groups listed in Table L.6 match those used for the application of Age Specific Sensitivity Factors that are listed in OEHHA's *Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures* (May 2009).

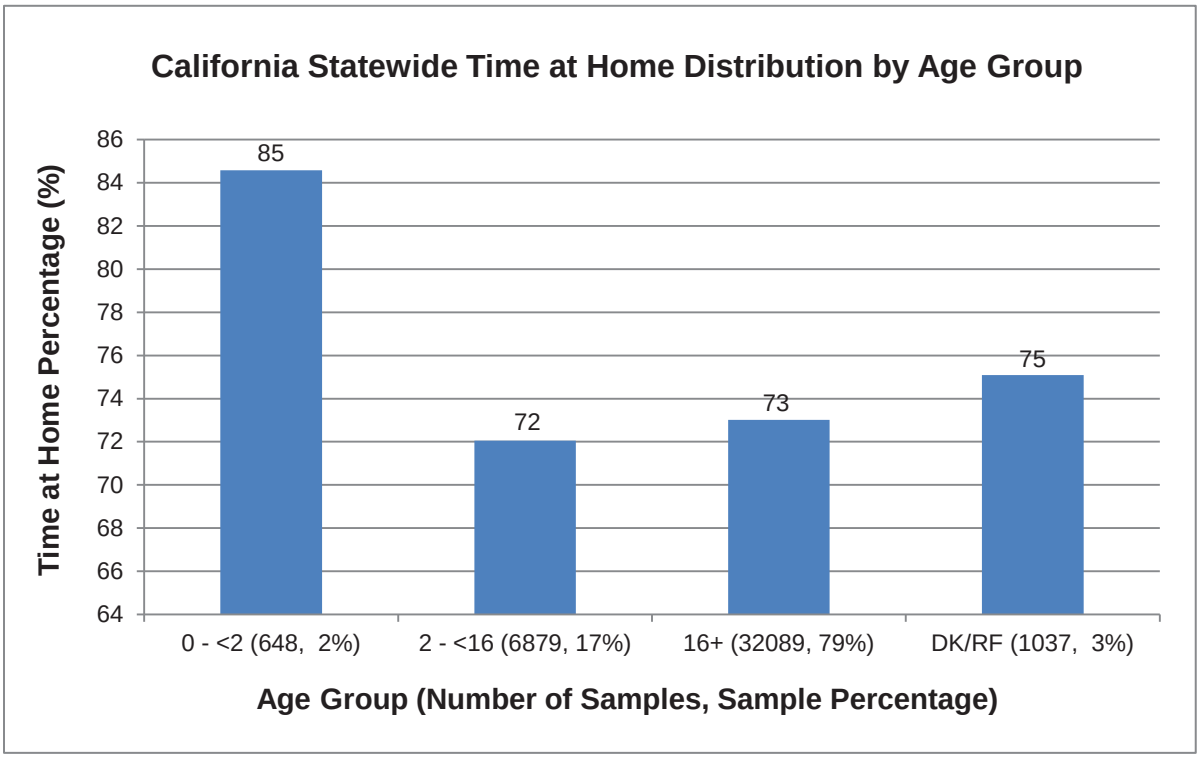
Table L.6 California Statewide Time at Home Distribution by Age Group

Age Group	Time at Home in Minute	Time at Home in Hour	Time at Home Percentage	Number of Samples	Sample Percentage
0 - <2	1218	20.3	85	648	2%
2 - <16	1037	17.3	72	6879	17%
16+	1051	17.5	73	32089	79%
DK/RF	1081	18.0	75	1037	3%
State Avg.	1052	17.5	73	40653	100%

Notes:

1. Caltrans 2000-2001 CHTS Data.
2. DK/RF means Don't Know/Refused.
3. Results don't have any weight factors applied.

Figure L.8



Notes:

1. Caltrans 2000-2001 CHTS Data.
2. DK/RF means Don't Know/Refused.
3. California statewide time at home average is 73%.
4. Total number of samples: 40,653.
5. Results don't have any weight factors applied.

Table L.7 and Figure L.9 demonstrate California statewide time spent at home distribution by household income level. They show a trend: the higher the household income is, the less time people spend at their home. The households with income level less than $10k spend most of their time at home as 81% (19.5 hr.) whereas the households with income level more than $100k but less than $150k spend the least time at home as 68% (16.2hr). The households with income level more than $35k but less than $50k spend the state average time 73% (17.5 hr) at home.

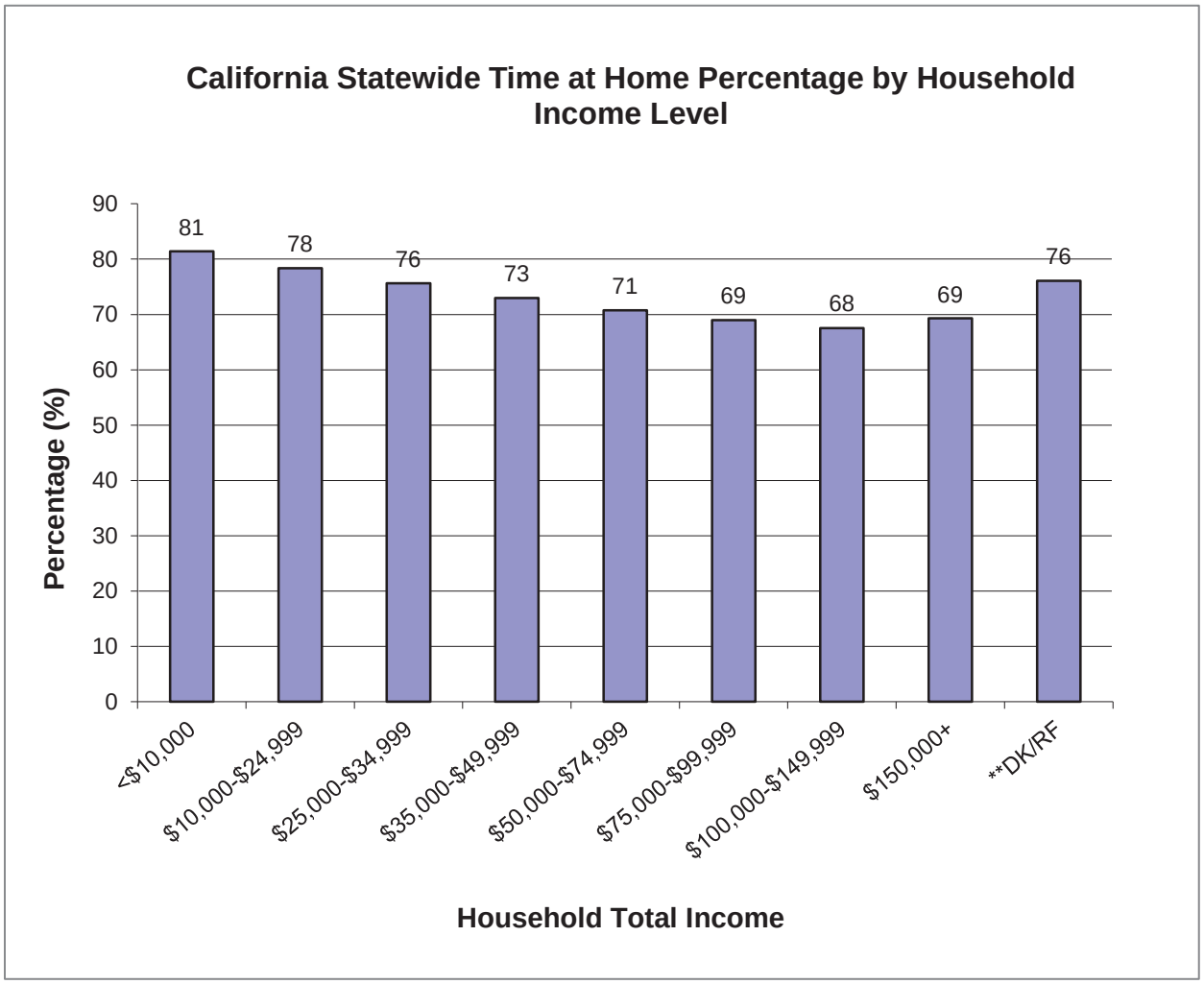
Table L.7 California Statewide Time at Home Distribution by Household Income Level

Household Total Income	Time at Home In Minute	Time at Home In Hour	Time at Home Percentage	Number of Samples	Sample Percentage
<$10,000	1172	19.5	81	1312	3%
$10,000-$24,999	1128	18.8	78	5189	13%
$25,000-$34,999	1089	18.2	76	5265	13%
$35,000-$49,999	1051	17.5	73	5568	14%
$50,000-$74,999	1019	17.0	71	8677	21%
$75,000-$99,999	994	16.6	69	5077	12%
$100,000-$149,999	973	16.2	68	3332	8%
$150,000+	998	16.6	69	1525	4%
DK/RF	1095	18.3	76	4751	12%
Total				**40696**	**100%**

Notes:

1. Caltrans 2000-2001 CHTS Data.
2. California statewide time at home average is 73%.
3. DK/RF means Don't Know/Refused.
4. Results don't have any weight factors applied.

Figure L.9



Notes:

1. Caltrans 2000-2001 CHTS Data.
2. California statewide time at home average is 73%.
3. DK/RF means Don't Know/Refused.
4. Total number of samples: 40,696.
5. Results don't have any weight factors applied.

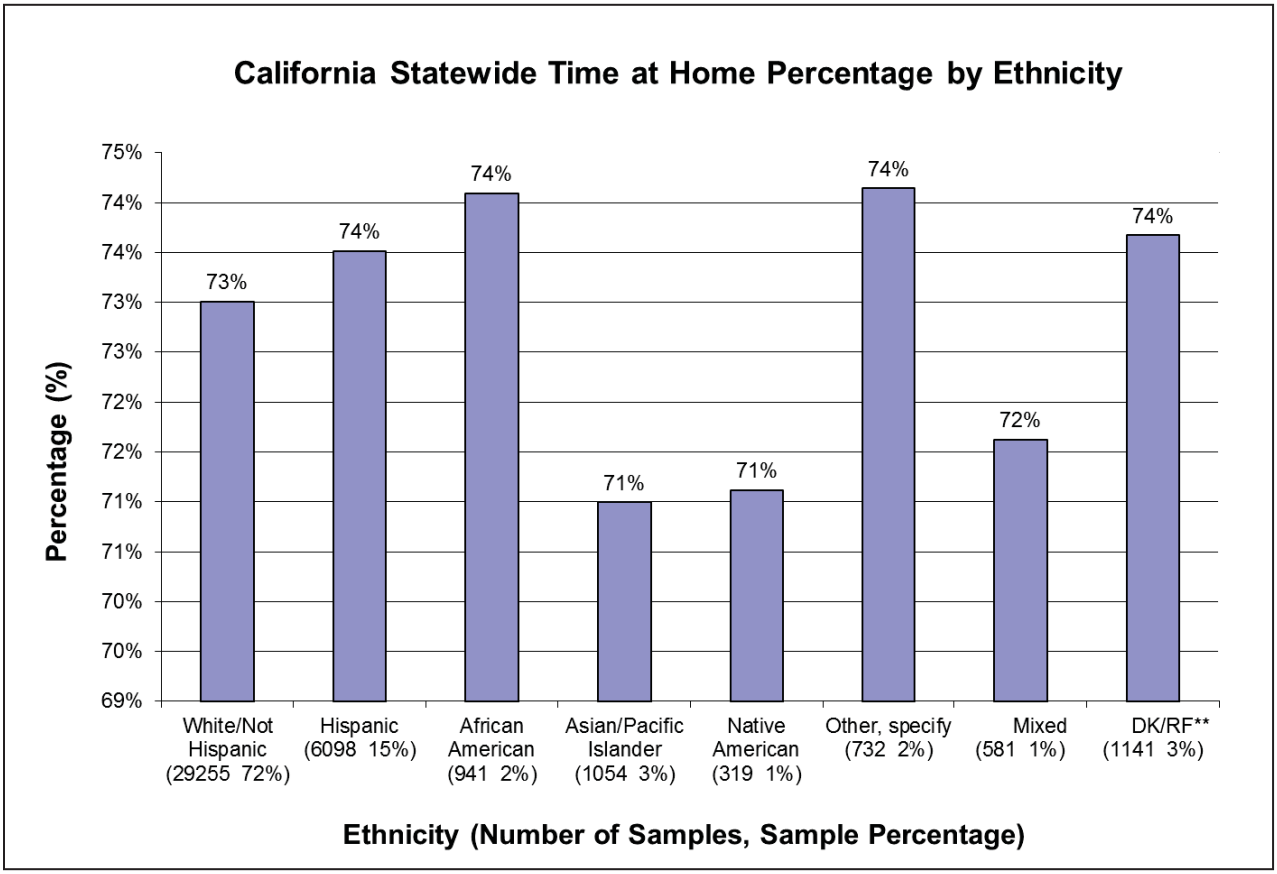
Table L.8 and Figure L.10 show California statewide time spent at home distribution by ethnicity. They depict that all the ethnic groups spend 71% to 74% time at home per day. The N/A in the ethnicity group in Table L.8 means the description of the ethnicity code 6 in the database is not available. The Caltrans survey data contact person believes that the code 6 should not have existed. This was a mistake in survey reporting. The 532 person day records (1% of the total person day records) with ethnicity code 6 may exist in error.

Table L.8 California Statewide Time at Home Average by Ethnicity

Ethnicity	Ethnicity Code	Time at Home In Minute	Time at Home In Hour	Time at Home Percentage	Number of Samples	Sample Percentage
White/Not Hispanic	1	1051	17.5	73%	29255	72%
Hispanic	2	1059	17.6	74%	6098	15%
African American	3	1067	17.8	74%	941	2%
Asian/Pacific Islander	4	1022	17.0	71%	1054	3%
Native American	5	1024	17.1	71%	319	1%
N/A	6	1077	17.9	75%	532	1%
Other, specify	7	1068	17.8	74%	732	2%
Mixed	8	1031	17.2	72%	581	1%
DK/RF	9	1061	17.7	74%	1141	3%
Total					**40653**	**100%**

Notes:
1. Caltrans 2000-2001 CHTS Data.
2. California statewide time at home average is 73%.
3. DK/RF means Don't Know/Refused.
4. N/A means the description of ethnicity code 6 is not available.
5. Results don't have any weight factors applied.

Figure L.10



Notes:

1. Caltrans 2000-2001 CHTS Data.
2. California statewide time at home average is 73%.
3. DK/RF means Don't Know/Refused.
4. Total number of samples: 40,653.
5. Results don't have any weight factors applied.

L.3.3.2.2 Comparison of Time at Home Results from CHTS Data with Time inside Home Results from ARB Activity Pattern Studies

Staff compared the time at home by age group statistical results from Caltrans 2000-2001 CHTS data and the time inside home results from 1987-1990 ARB activity pattern studies (ARB, 2005). Table L.9 and Figure L.11 show that, compared to the time spent inside home in 1987-1990, children under age of 12 spent similar amount of time at home in 2000-2001. However, teens (age 12 to17) spent 6% more time at home in 2000-2001, and adults spent 11% more time at home in 2000-2001.

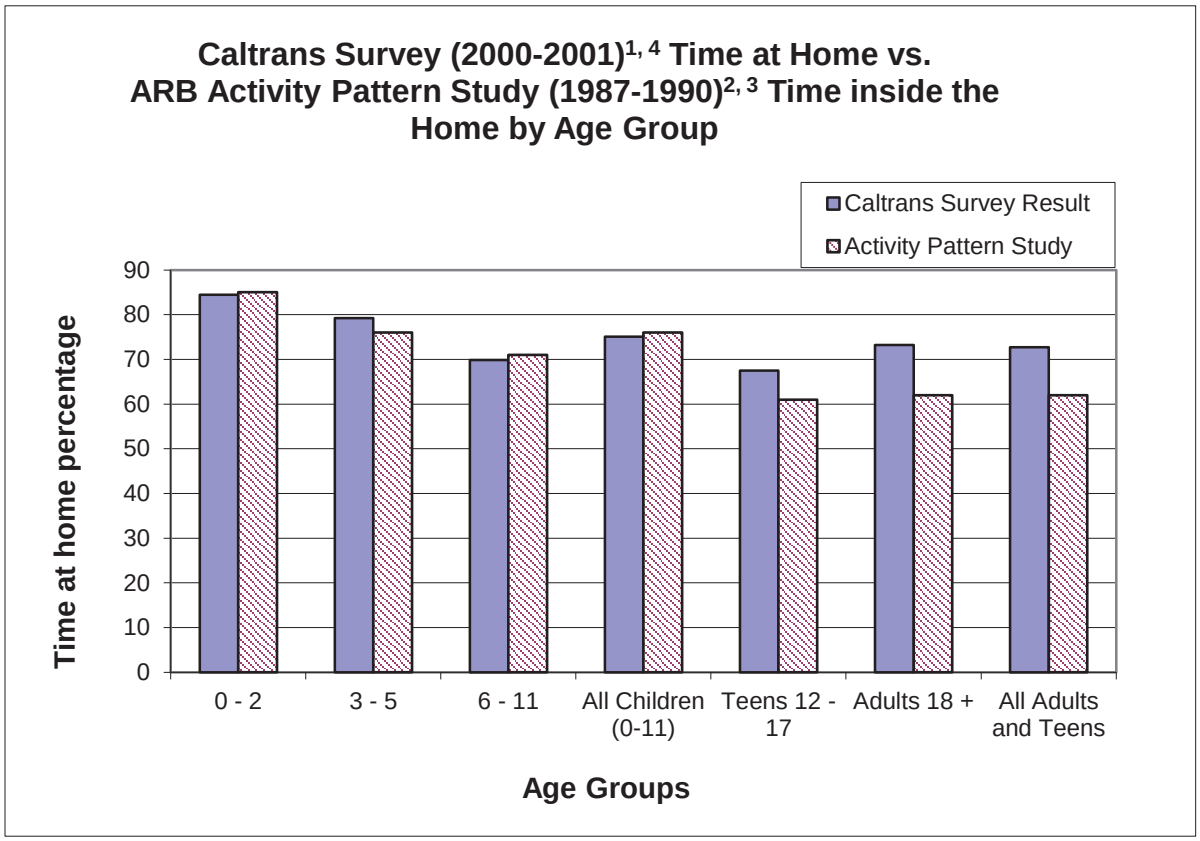
Table L.9 Caltrans Survey (2000-2001) Time at Home vs. ARB Activity Pattern Study (1987-1990) Time inside the Home by Age Group

Age Group	Caltrans[1, 4]			ARB[2, 3]	
	Number of Samples	Time at Home In Hour	Time at Home (%)	Number of Samples	Time Inside Home (%)
0 - 2	1086	20.3	84	313	85
3 - 5	1328	19.0	79	302	76
6 - 11	2985	16.8	70	585	71
All Children (0-11)	5399	18.0	75	1200	76
Teens 12 - 17	3180	16.2	67	183	61
Adults 18 +	31937	17.6	73	1579	62
All Adults and Teens	34217	17.4	73	1762	62

Notes:

1. The 2000 - 2001 California Statewide Household Travel Survey was conducted among households in each of the 58 counties throughout the State and grouped by region. Total person day records are 40,653.
2. The 1989 -1990 Children's Activity Pattern Study's samples are selected from households among three major areas: Southern Coast, S.F. Bay Area, and the rest of state. Total samples are 1,200 (ARB, 1991).
3. The 1987 – 1988 California Residents Activity Pattern Study's samples are selected from the same three major areas as for Children's Activity Pattern Study, with 1579 adult samples and 183 youth samples (ARB, 1992).
4. Results from Caltrans survey data don't have any weight factors applied, whereas the results from the activity pattern studies have the weight factors applied.

Figure L.11



Notes:

1. The 2000 - 2001 California Statewide Household Travel Survey was conducted among households in each of the 58 counties throughout the State and grouped by region. Total person day records are 40,653.
2. The 1989 -1990 Children's Activity Pattern Study's samples are selected from households among three major areas: Southern Coast, S.F. Bay Area, and the rest of state. Total samples are 1,200 (ARB, 1991).
3. The 1987 – 1988 California Residents Activity Pattern Study's samples are selected from the same three major areas as for Children's Activity Pattern Study, with 1579 adult samples and 183 youth samples (ARB, 1992).
4. Results from Caltrans survey data don't have any weight factors applied, whereas the results from the activity pattern studies have the weight factors applied.

L.3.3.3 Limitations on the Use of 2000-2001 CHTS data

The limitations of the use of the 2000-2001 CHTS data are that the analysis results do not have weight factors applied due to in-sufficient user information on weights for personal level analysis (CHTS Guide). And 2000-2001 CHTS does not have residence duration data.

L.4 Other Data Sources Not Used in This Report

L.4.1 The 2009 National Household Travel Survey

The 2009 NHTS updates information gathered in the 2001 NHTS and in prior Nationwide Personal Transportation Surveys. The data are collected on daily trips taken in a 24-hour period (NHTS, 2009). Although we may be able to analyze the 2009 NHTS data to get the time at home statistical results for Californians, the staff didn't use the data because the user manual was not ready at the time the staff was preparing this report.

L.4.2 National Human Activity Pattern Survey

NHAPS was sponsored by the U.S. Environmental Protection Agency. It was conducted between late September 1992 and September 1994, collected 24-hour activity diaries and answers of personal and exposure questions. The survey interviewed 9386 participants across the 48 contiguous states (Klepeis et al., 1995).

NHAPS has time in a residence data from California respondents. However, the staff didn't further analyze these data because the 2000-2001 CHTS provides much larger sample size and more recent California-specific data.

L.5 Conclusion

The staff has evaluated several data sources to identify the California statewide exposure duration and exposure frequency characteristics. Estimates on residence duration and time spent at home have been determined from available data on the California population. The data on residency time are similar to the available national data as discussed in Chapter 11. There is some variability in the residence duration and time spent at home by ethnicity, age, and income.

The IPUMS-USA census data show that, from 2006 to 2009, over 90% of California householders had lived at their current home address for less than 30 years, and over 63% householders had lived at their current residence for 9 years or less.

The 2000-2001 CHTS data show that, on average, Californians spend approximately 73% of their time at home per day. When looking at the data by age group, the time increases to 85% for children under 2 years old. Children that are 2 years or older but less than 16 years old spend 72% of their time at home; whereas Californians that are16 years or older spend 73% of their time at home. In addition, all ethnicity groups spend 71%-74% of their time at home. The data also demonstrate a trend where the higher the total household income is, the less time the residents spend at their home.

These data are the best available on the California population for helping to establish default recommendations for the Hot Spots program.

L.6 References

(ACS) American Community Survey: http://factfinder2.census.gov/faces/nav/jsf/pages/wc_acs.xhtml. Last visited: April, 2011.

(IPUMS-USA) Steven Ruggles, J. Trent Alexander, Katie Genadek, Ronald Goeken, Matthew B. Schroeder, and Matthew Sobek. *Integrated Public Use Microdata Series: Version 5.0* [Machine-readable database]. Minneapolis: University of Minnesota, 2010. http://usa.ipums.org/usa/index.shtml. Last visited: January, 2011.

(IPUMS Tool) IPUMS Online Data Analysis System: http://usa.ipums.org/usa/sda/. Last visited: January, 2011.

(IPUMS Weights) IPUMS-USA Sample Weights: http://usa.ipums.org/usa/intro.shtml#weights. Last visited: January, 2011.

(IPUMS Samples) Descriptions of IPUMS Samples: http://usa.ipums.org/usa/sampdesc.shtml. Last visited: January, 2011.

(IPUMS GQ) IPUMS-USA Group Quarters: http://usa.ipums.org/usa-action/variables/GQ. Last visited: April, 2011.

(SCAG, 2003) Year 2000 Post-Census Regional Travel Survey Final Report of Survey Results. SCAG, Fall 2003. http://www.scag.ca.gov/travelsurvey/pdf/MainSurveyResults.pdf

(SCAG Manual) Post Census Regional Household Travel Survey Data User's Manual. SCAG, June 2003.

(CHTS, 2003) 2000-2001 California Statewide Travel Survey Weekday Travel Report. Caltrans, June 2003. http://www.dot.ca.gov/hq/tsip/tab/documents/travelsurveys/Final2001_StwTravelSurveyWkdayRpt.pdf

(CHTS Guide) 2000-2001 California Statewide Household Travel Survey Data Users Guide. Caltrans, May 2002.

(ARB, 2005) Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant. ARB, 2005. http://www.arb.ca.gov/regact/ets2006/app3parta.pdf

(ARB, 1991) Study of Children's Activity Patterns, ARB, 1991. http://www.arb.ca.gov/research/apr/past/a733-149a.pdf

(ARB, 1992) Activity Patterns of California Residents. ARB, 1992. http://www.arb.ca.gov/research/apr/past/a6-177-33.pdf

Technical Support Document for Exposure Assessment and Stochastic Analysis, FINAL, August, 2012

(NHTS, 2009) The 2009 National Household Travel Survey: http://nhts.ornl.gov/introduction.shtml. Last visited: January, 2011.

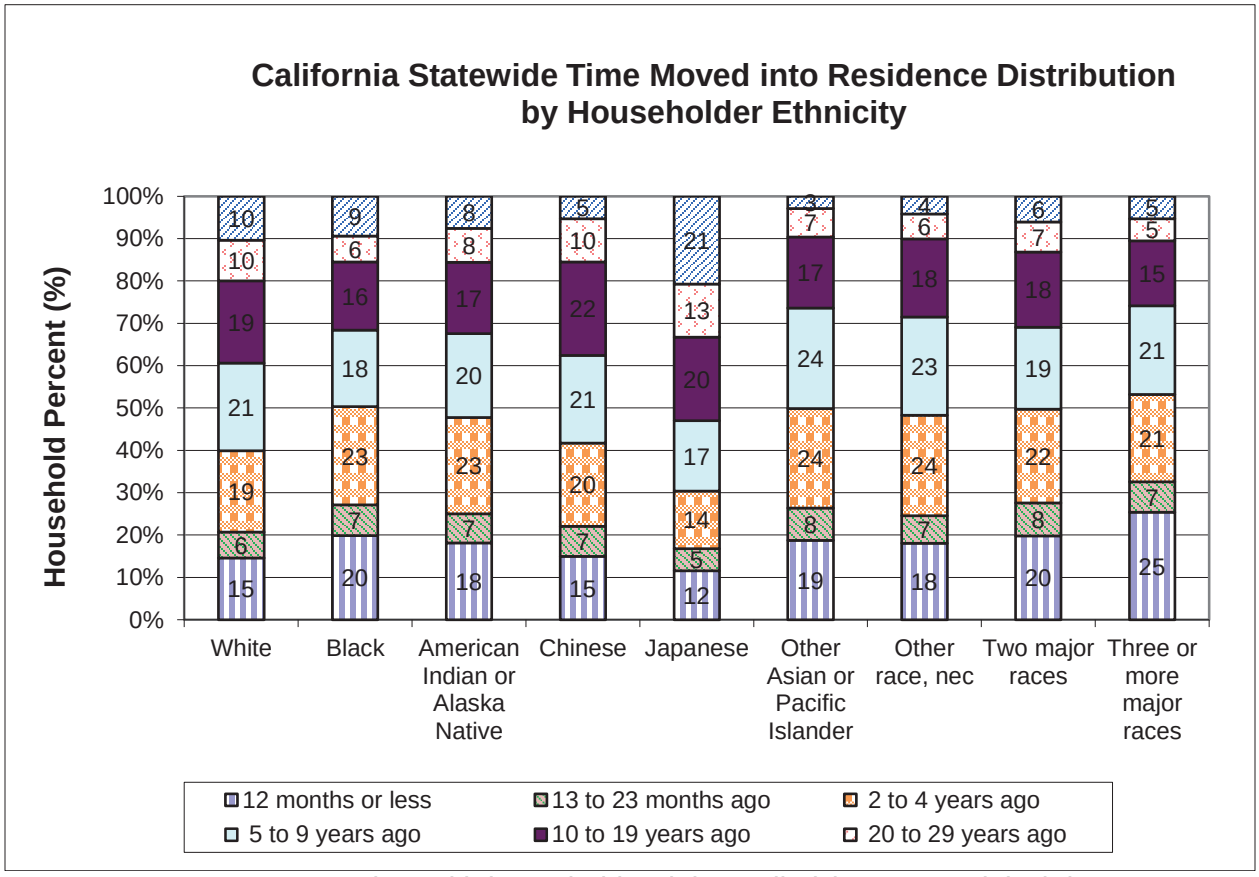
(Klepeis NE, Nelson W C, Ott W R, Robinson, J-Pm Tsang A M, Switzer P, Bhar J, Hem S C, Engelmann W H. (1995) National Human Activity Pattern Survey. Klepeis et al., 1995. http://eetd.lbl.gov/ie/viaq/pubs/LBNL-47713.pdf

A. Supplemental Information

The following figures graphically present the analysis results of California statewide time moved into residence distribution by householders' ethnicity, age, and household income respectively from IPUMS-USA ACS 2009 data (IPUMS-USA). The data are obtained by using IPUMS online analysis tool (IPUMS Tool). These data may be useful to the risk manager in considering population risk in different communities.

Figure A.1 shows California statewide time moved into residence distribution by householders' ethnicity. In general, the percentages of householders that moved into their residence 12 months or less ago, 2 to 4 years ago, 5 to 9 years ago, and 10 to 19 years ago are larger than the percentages of 13 to 23 months ago, 20 to 29 years ago, and 30 years ago.

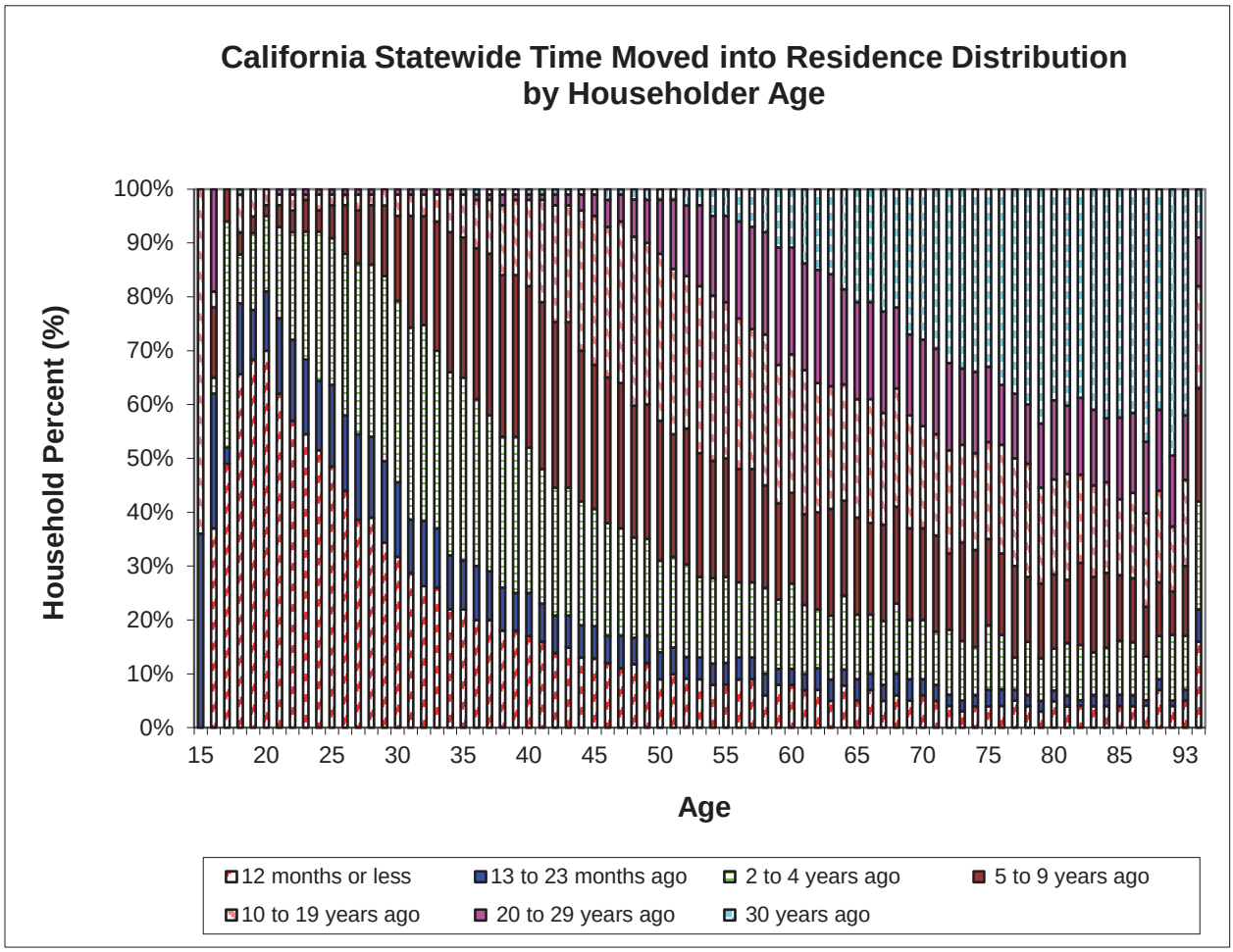
Figure A.1*



* IPUMS-USA ACS 2009 data with household weight applied (IPUMS Weights) (IPUMS Ethnicity).

Figure A.2 presents California statewide time moved into residence distribution by householders' age. It shows a general trend that the younger the householders are, the more householders moved into their residence within the last 12 months. And the older the householders are, the more householders moved into their residence 30 years ago. There are some exceptions at the both ends of the age range.

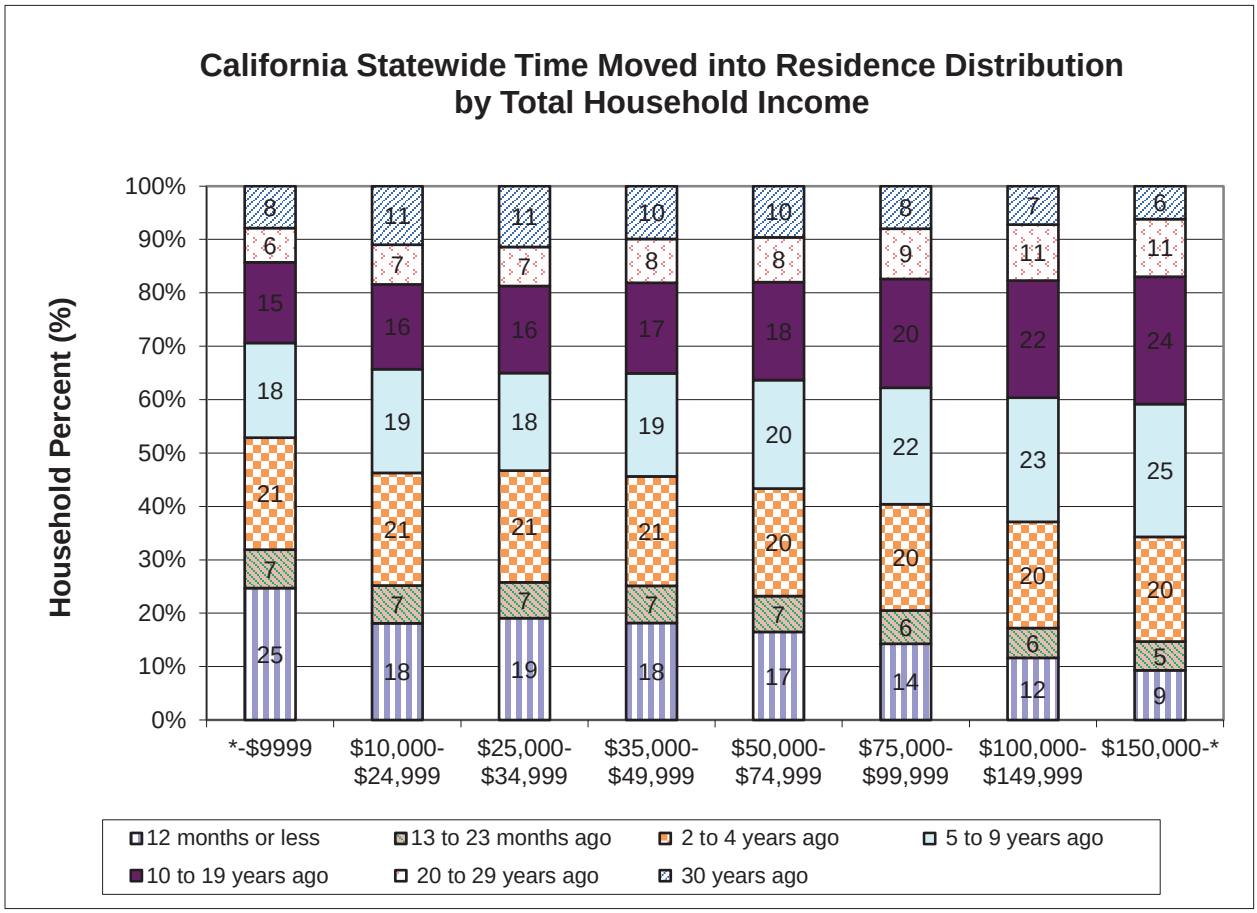
Figure A.2*



* IPUMS-USA ACS 2009 data with household weight applied (IPUMS Weights). The age categories are 15-89 and 93.

Figure A.3 shows California statewide time moved into residence distribution by total household income. It reveals a general trend that the higher the household income is, the smaller percentage of the householders moved into their residence within last the 12 months. And the households with household income of $150,000 or above not only have the smallest percentage of householders moved into their residence within the last 12 months, but also have the smallest percentage of householders moved into their residence 30 years ago.

Figure A.3*



* IPUMS-USA ACS 2009 data with household weight applied (IPUMS Weights).

A. References

(IPUMS-USA) Steven Ruggles, J. Trent Alexander, Katie Genadek, Ronald Goeken, Matthew B. Schroeder, and Matthew Sobek. *Integrated Public Use Microdata Series: Version 5.0* [Machine-readable database]. Minneapolis: University of Minnesota, 2010. http://usa.ipums.org/usa/index.shtml. Last visited: January, 2011.

(IPUMS Tool) IPUMS Online Data Analysis System: http://usa.ipums.org/usa/sda/. Last visited: January, 2011.

(IPUMS Ethnicity) IPUMS-USA Ethnicity Code: http://usa.ipums.org/usa-action/codes.do?mnemonic=RACE. Last visited: January, 2011.

(IPUMS Weights) IPUMS-USA Sample Weights: http://usa.ipums.org/usa/intro.shtml#weights. Last visited: January, 2011.

Appendix M

How to Post-Process Offsite Worker Concentrations using the Hourly Raw Results from AERMOD

This appendix describes how to calculate refined offsite worker concentrations using the hourly raw results from the AERMOD air dispersion model. In some cases, a better representation of what the offsite worker breathes during their work shift is needed for the health risk analysis. To obtain a better representation, the hourly raw results contain enough information to allow the risk assessor to evaluate the concentrations that occurs during the offsite worker's shift. However, since the hourly raw results include all the concentrations for every hour of meteorological data at each receptor for each source in the air dispersion analysis, the results must be filtered and processed to obtain the refined offsite worker concentrations. The basic steps include: 1) determining the averaging periods needed for the offsite worker analysis; 2) outputting the hourly raw results from the AERMOD air dispersion model; 3) extracting the hourly concentrations based on when the receptor is present; and 4) identifying or calculating the required concentration. The calculation methods described in this appendix can be used for assessing acute, 8-hour non-cancer chronic, and inhalation cancer health impacts.

M.1 Determine the Averaging Periods Required for the Offsite Worker Health Risk Analysis

Before any refined offsite worker concentrations can be calculated, the first step is to determine which type of refined concentrations or averaging periods are needed for the health risk analysis. The refined averaging periods needed for the analysis are based on the pollutant-specific health values emitted by the source or sources. Specifically, refined offsite worker concentrations can only be used for pollutants that have inhalation cancer potency factors, 8-hour RELs, and/or acute RELs. This section describes the refined averaging periods required for assessing acute RELs, 8-hour RELs, and inhalation cancer potency factors.

M.1.1 Averaging Period Required for Acute RELs

The maximum 1-hour concentration is typically required for the acute health hazard index calculation. AERMOD can determine and output the maximum 1-hour concentration at each receptor location for each source in the air dispersion analysis. However, if more refined concentrations for the offsite worker are needed, the maximum1-hour concentration that occurs during the offsite worker's shift may be used.

This type of refinement can be processed using the hourly raw results from the air dispersion analysis.

If there are multiple sources in the analysis, an additional refinement step is to examine the coincident acute health impacts at each receptor from all sources at each hour during the offsite worker's shift and identify the total maximum acute health impacts from all sources. For example, if there are two sources that emit a single pollutant for ten hours per day and the offsite worker's shift is from hour three to hour seven, the risk assessor may evaluate the total acute risk from all sources during the offsite worker's shift. Assuming the acute REL is 50 $\mu g/m^3$, the highest acute health impact occurs at hour three with a Health Hazard Index of 0.3 (see Table M.1). This approach is also known as a refined acute analysis.

Table M.1. Example of a Refined Acute Calculation

Hour	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
Source 1 Concentration ($\mu g/m^3$)	5	7	8	0	9	11	5	1	12	3
Source 2 Concentration($\mu g/m^3$)	4	6	7	0	2	1	3	4	5	2
Total Acute Health Hazard Index from All Sources	0.18	0.26	**0.3**	0	0.22	0.24	0.16	0.1	0.34	0.1

M.1.2 Averaging Period Required for Inhalation Cancer Potency Values

The period average is typically required for cancer risk assessments. AERMOD calculates this average by summing all the hourly concentrations and dividing it by the number of processed hours over the entire time period of the air dispersion analysis. However, the period averages calculated from AERMOD typically represent exposures for receptors (i.e., residential receptors) that are present 24 hours a day and seven days per week. For the offsite worker, the period average should represent what the worker breathes during their work shift when assessing the cancer inhalation pathway.

To estimate the offsite worker's concentration, there are two approaches. The simple approach is to obtain the period average concentration as calculated by AERMOD and approximate the worker's inhalation exposure using an adjustment factor (See Chapter 2.8.1.1. for more information). For a more representative concentration, the second approach is to calculate a refined period average using the hourly raw results from the air dispersion analysis. This refined period average should reflect only the concentrations that occur during the offsite worker's shift. It is calculated by summing all of the hourly concentrations that occurs during the offsite worker's shift and dividing it by the number of hours that occurs during the offsite worker's shift. The equation for calculating the refined offsite worker concentration is shown in Section 4.3.

M.1.3 Averaging Period Required for 8-Hour RELs

For 8-hour noncancer health impacts, we evaluate if the worker is exposed to a daily (e.g., 8-hour) average concentration that exceeds the 8-hour REL. The daily average concentration is intended to represent the long term average concentration the worker is breathing during their work shift. The long-term 8-hour daily average concentration is required for 8-hour health hazard index calculations. Specifically, this concentration represents the long-term average of repeated 8-hour daily averages that occur when the source's emission schedule and offsite worker's schedule overlap. For example, the 8-hour averages are first calculated for each day in the air dispersion analysis. The 8-hour averages should represent the eight hour sequential concentration for when the source's emission schedule and offsite worker's schedule overlap. All the 8-hour averages are then averaged over the entire time period of the air dispersion analysis.

There are two approaches for calculating the average 8-hour daily concentration. The simple approach is to obtain the long-term concentration (i.e., period average) as calculated by AERMOD and approximate the average 8-hour daily concentration using an adjustment factor (See Chapter 2.8.1.2 for more information). For a more representative concentration, the second approach is to calculate the offsite worker concentration using the hourly raw results from the air dispersion analysis.

Please note that although the duration of work shifts or period of overlap with the source's emission schedule can vary from eight hours, the calculated long-term daily average concentrations can still be applied to the 8-hour RELs. However, the risk assessor may wish to calculate the 8-hour hazard index using the adjustment factor approach as a screening assessment before proceeding with the post-processing approach. Based on the results of the screening assessment, the risk assessor can contact OEHHA for assistance in determining whether further evaluation may be necessary.

M.2 Output the Hourly Raw Results from AERMOD

The hourly raw results from the air dispersion analysis are needed to calculate the refined offsite worker concentrations as described above. AERMOD can output the hourly raw results to a file for post-processing. In order to output a file suitable for post-processing, the AERMOD input file must be modified. The AERMOD input file contains the modeling options, source location and parameter data, receptor locations, meteorological data file specifications, and output options. It is organized into five main sections that include the Control (CO), Source (SO), Receptor (RE), Meteorology (ME), and Output (OU) pathways (U.S. EPA, 2004). This section describes how to modify the pathways in the AERMOD input file to allow the hourly raw results to be saved to a file.

M.2.1 Modify the Control (CO) Pathway to Identify Calm and Missing Hours

By default, AERMOD disregards calm and missing hours when calculating the long-term and short-term averages. When calculating the refined offsite worker concentrations, the calm and missing hours must also be disregarded. However, the hourly raw results from AERMOD do not identify which hours are calm or missing. Since this is the case, an additional file from AERMOD must also be saved in order to post-process the hourly raw results correctly. The AERMOD Detailed Error Listing File will report all calm and missing hours from the air dispersion analysis. The syntax for creating a Detailed Error Listing File in the CO pathway is shown below. This modification in the CO pathway will create a file which will be used to assist with calculating the refined offsite worker concentrations. This process is described in the subsequent sections of this appendix.

Syntax for Creating the Detailed Error Listing File

CO ERRORFIL [Filename]

M.2.2 Modify the Source (SO) Pathway if Unit Emission Rates are used

In an air dispersion analysis, it is typical to use non-substance specific unit emission rates (e.g., 1 g/s) for evaluating multiple pollutants. This precludes modelers from having to run the air dispersion model for each individual pollutant that is emitted from a source. Unit emission rates allow the air dispersion modeling results to be expressed as dilution factors in $(\mu g/m^3)/(g/s)$. When these dilution factors are combined with the pollutant specific emission rate (g/s), it will yield the ground level concentrations ($\mu g/m^3$) for each pollutant in the analysis. When there are multiple sources in the air dispersion analysis and unit emission rates are used, the individual source contributions must be provided in the modeling results so the ground level concentrations can be correctly scaled for each pollutant. To do this, the air dispersion input file must be modified to create individual source groups for each source. The example below shows how individual source groups for two sources (S001 and S002) are specified in the SO pathway of an AERMOD input file. This modification in the SO pathway will allow the individual source contributions to be saved in the hourly raw results.

```
SO STARTING

**S001 and S002 location and source parameters are not shown.**

SRCGROUP SRCGP1 S001

SRCGROUP SRCGP2 S002

SO FINSHED
```

This parameter identifies the sources tied to the source group. Use only one source ID per source group.

This section specifies the name of your source group. The source group name is what is specified when you output the required concentrations files.

Please note that a separate input file is needed for evaluating acute health impacts when unit emission rates are used and the source has a variable emission schedule (e.g., emissions vary by hour-of-day and day-of-week). Acute health impacts are based on maximum hourly emissions whereas cancer and chronic health impacts are based on average hourly emissions. To correctly simulate unit emissions for the acute impacts, a duplicate source with a variable emission rate of "on" (1) or "off" (0) should be used so the maximum hourly inventory is correctly calculated separately from the emission factors placed in the annual file. The example below shows how the variable emission rates should be modified. Alternatively, a source can be duplicated in the same input file instead of rerunning the source using a separate input file.

First Run with Unmodified Emission Rate Factors for Long-Term

```
EMISFACT S002  HROFDY   0.000   0.000   0.000   0.000   0.000
         S002  HROFDY   0.000   2.667   2.667   2.667   2.667
         S002  HROFDY   2.667   2.667   1.333   1.333   1.333
         S002  HROFDY   1.333   1.333   1.333   0.000   0.000
         S002  HROFDY   0.000   0.000   0.000   0.000
```

Second Run with Modified Emission Rates Factors for Acute

```
EMISFACT S002  HROFDY   0.000   0.000   0.000   0.000   0.000
         S002  HROFDY   0.000   1.000   1.000   1.000   1.000
         S002  HROFDY   1.000   1.000   1.000   1.000   1.000
         S002  HROFDY   1.000   1.000   1.000   0.000   0.000
         S002  HROFDY   0.000   0.000   0.000   0.000
```

M.2.3 Modify the Receptor (RE) Pathway to Reduce the Processing Time

AERMOD is capable of outputting the hourly raw results from the air dispersion analysis. However, without taking appropriate precautions, outputting the hourly raw results can produce extremely large file sizes especially when evaluating multiple years of meteorological data, a large number of receptors, and short-term averaging periods (e.g., 1-hour). To minimize the amount of processing time and hard disk space, it is recommended to use only a single discrete receptor representing the off-site worker location. The proper syntax for specifying a discrete receptor is shown below.

Sample Syntax for Creating a Single Discrete Receptor

```
RE DISCCART XcoordYcoord (ZelevZhill) (Zflag)
```

M.2.4 Modify the Output (OU) Pathway to Output the Hourly Raw Results

To create a file containing the hourly raw results, modify the OU pathway to include the POSTFILE keyword and parameters. The sample below shows the syntax for outputting the hourly raw results for a single source. The POSTFILE will list in order the concentration for each receptor and for each hour of meteorological data regardless of the source's emission schedule. Use Table M.2 to help construct the proper syntax for the POSTFILE option. This step must be repeated for each source in the analysis which will result in additional files.

Please note that if the data are outputted as binary file (UNFORM), a separate computer program will be needed to read and parse the data.

Sample Syntax for Outputting the Hourly Concentrations for a Single Source

```
OU POSTFILE 1 SRCGP1PLOT PSTS001.TXT
```

Table M.2. Descriptions of the POSTFILE Parameters

Keyword	Parameters	
POSTFILE	AveperGrpid Format Filnam (Funit)	
where:	Aveper	Specifies averaging period to be output to file. Set this value to 1 to output 1-hour raw results.
	Grpid	Specifies source group to be output to file. If there are multiple sources, you will need to repeat the POSTFILE option for each source. You can combine the different outputs to a single file using the Funit parameter.
	Format	Specifies format of file, either UNFORM for binary files or PLOT for formatted files. Unformatted files offer a smaller file size; however, this file requires programming expertise in order to view and parse the data. Selecting the PLOT option will allow you to view the file in any text editor.
	Filnam	Specifies filename for output file
	Funit (optional)	The file unit is an optional parameter. If the filename and the file unit number are the same, the results for different source groups can be combined into a single file.

M.3 Extract the Hourly Concentrations when the Offsite Worker is Present

To calculate the refined offsite worker concentrations, it is necessary to extract the hourly concentrations based on the offsite worker's schedule. This section provides information on how to extract the hourly concentrations for the offsite worker including the calm and missing hours that may occur during the offsite worker's shift.

At this point, it is recommended the hourly raw results be imported into a spreadsheet or database to assist with the extraction process. Spreadsheets and database contain preprogrammed functions to assist with deciphering data. **Use the information in Section M.3.1 as a guide to help import the hourly raw results into a database or spreadsheet.**

M.3.1 Description of the POSTFILE File Format

AERMOD was created using FORTRAN, a type of programming language. When the AERMOD output files are created, it is based on a specified FORTRAN format. The variables provided on each data record in the POSTFILE include the X and Y coordinates of the receptor location, the concentration value for that location, receptor terrain elevation, hill height scale, flagpole receptor height, the averaging period, the source group ID, and the date for the end of the averaging period (in the form of YYMMDDHH) (U.S. EPA, 2004). Table M.3 shows the equivalent data types based on the POSTFILE format. The POSTFILE will list in order the concentration for each receptor and for each hour of meteorological data regardless of the source's emission schedule (see Figure M.3.1). Use the information in this section as a guide to help import the hourly raw results into a database or spreadsheet.

Table M.3. POSTFILE Variables and Equivalent Data Types

Column Name	Fortran Format	Equivalent Data Type
X	F13.5	Number/Double Precision
Y	F13.5	Number/Double Precision
AVERAGE_CONC	F13.5	Number/Double Precision
ZELEV	F8.2	Number/Double Precision
ZHILL	F8.2	Number/Double Precision
ZFLAG	F8.2	Number/Double Precision
AVE	A6	6-Character String/Text
GRP	A8	8-Character String/Text
NUM_HRS OR DATE	I8.8	8-Character String/Text
NET_ID	A8	8-Character String/Text

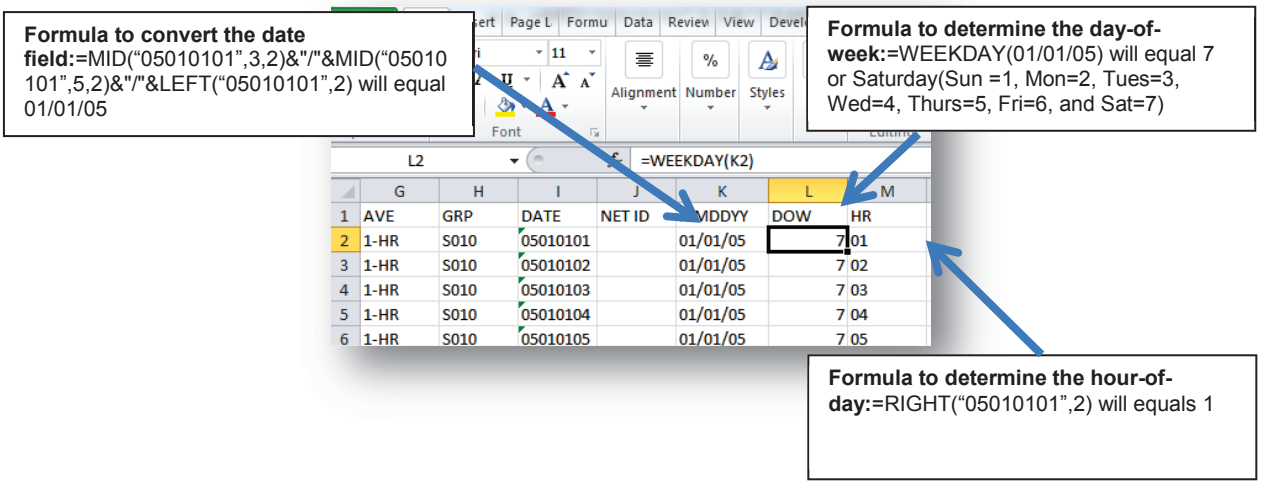
Figure M.3.1. Sample of an AERMOD POSTFILE

```
AERMOD (09292): LARGE PS                                                    08/24/10
MODELING OPTIONS USED:                                                      07:39:24
NonDFAULT CONC                          FLAT          FLGPOL
        POST/PLOT FILE OF CONCURRENT  1-HR VALUES FOR SOURCE GROUP: S010
        FOR A TOTAL OF     1 RECEPTORS.
        FORMAT: (3(1X,F13.5),3(1X,F8.2),2X,A6,2X,A8,2X,I8.8,2X,A8)
       X             Y        AVERAGE CONC    ZELEV    ZHILL    ZFLAG    AVE     GRP      DATE       NET ID
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010101
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010102
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010103
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010104
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010105
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010106
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010107
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010108
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010109
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010110
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010111
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010112
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010113
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010114
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010115
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010116
 100.00000     0.00000     0.00000    10.00    10.00     1.20    1-HR    S010    05010117
```

M.3.2 Determine the Day-of-Week and Hour-of-Day

In order to extract only the hourly concentrations that occur when an offsite worker is present, the risk assessor must first determine the day-of-week and hour-of-day for each hourly record using the date field. Since the date outputted by AERMOD cannot be directly interpreted by the day-of-week function in a database or spreadsheet, the date must be first converted. For example, the date field can be first converted using the LEFT and MID functions in Microsoft Excel (See Column K in Figure M.3.2). After which, the WEEKDAY function in Microsoft Excel can be used to determine the day-of-week (See Column L in Figure M.3.2). The hour-of-day can be extracted using the RIGHT function (See Column M in Figure M.3.2).

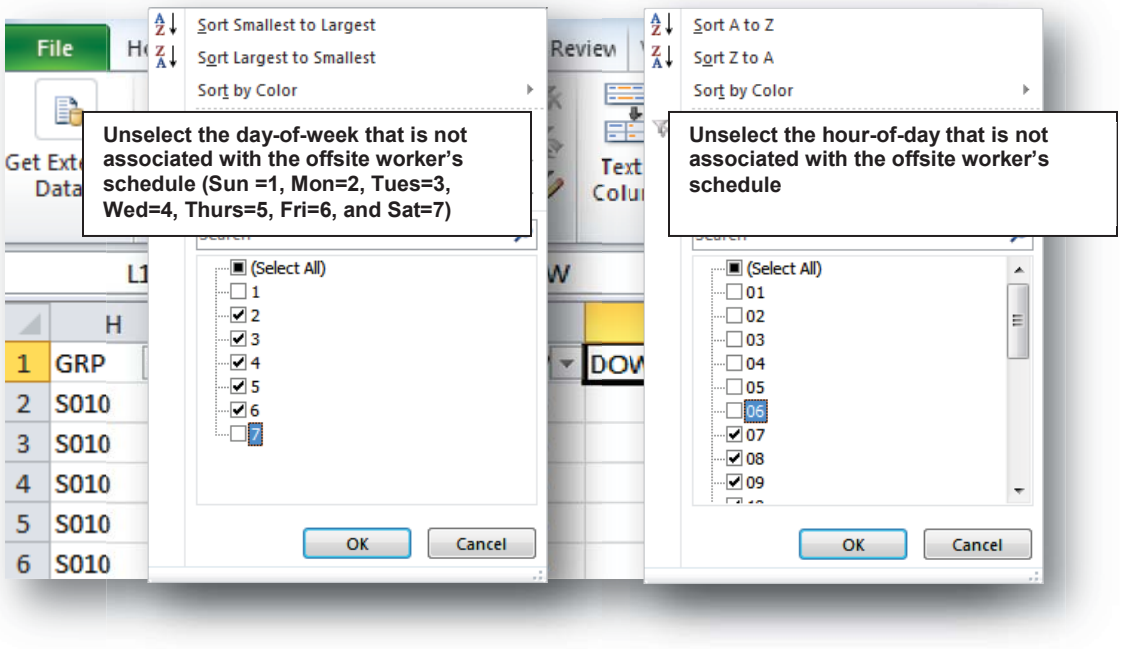
Figure M.3.2. How to Determine the Day-of-Week and Hour-of-Day in Microsoft Excel



M.3.3 Extract the Hourly Concentrations Based on the Offsite Worker's Schedule

After the day-of-week and hour-of-day have been determined, the concentrations can now be extracted or filtered. Based on the offsite worker's schedule, filter or query the hourly concentrations using a spreadsheet or database. For example, in Microsoft Excel, you can filter the data by selecting the data filter option (see Figure M.3.3). Then unselect the records that are not associated with the offsite worker's schedule using the day-of-week and hour-of-day fields that were created in previous section. If the data contains information for multiple receptors, filter the X and Y coordinates to get the concentrations that are specific to each receptor. The results from the filter will now only show hourly concentrations for times when the offsite worker is present.

Figure M.3.3. How to Filter the Data in Microsoft Excel



M.3.4 Count the Number of Calm and Missing Hours that Occur During the Offsite Worker's Schedule

If calm hour processing was used in the air dispersion analysis, then calm and missing hours must also be considered when post-processing the long-term and short-term averages for the offsite worker. To assist in this calculation, the Detailed Error Listing File that was created from the air dispersion analysis (Section 2.1) can be used to count the number of calm and missing hours that occurred during the worker's shift.

To identify the calm and missing hours, it is recommended to import the Detailed Error Listing File into a spreadsheet or database. Then follow the instructions from Sections 3.2 and 3.3 to determine the number of calm and missing hours that occur during the offsite worker's schedule. This information is needed to calculate the averaging periods for the offsite worker.

M.4 How to Identify or Calculate the Refined Concentrations for the Offsite Worker Analysis

Depending on which averaging periods are needed (as determined by Section 1.0), use Sections 4.1 through 4.3 below to identify or calculate refined concentrations for estimating the acute, 8-hour, and cancer health impacts. The equations are based on how the long-term and short-term averages are calculated in AERMOD. These equations also account for how calm and missing hours are handled by AERMOD (U.S. EPA, 2005). After calculating the appropriate averaging periods, the refined concentrations can be used to assess the health impacts for the offsite worker's inhalation pathway.

Please note that if unit emission rates were used in the air dispersion analysis, each averaging period calculated using the methods below must be combined with the pollutant specific emission rate (g/s) to yield the actual ground level concentrations (µg/m^3) for each pollutant in the analysis before the health impacts can be assessed.

M.4.1 How to Determine the Maximum 1-Hour Average for a Simple Acute Assessment

The maximum 1-hour average concentration represents the highest concentration that occurs during the offsite worker's schedule. To determine the maximum 1-hour average, sort the extracted hourly concentrations in descending order using a spreadsheet or a database. The maximum hourly concentration will be at the top of the list (Figure M.4.1). This process must be repeated at each receptor for all sources of interest.

Figure M.4.1. Identifying the Maximum 1-Hour Concentration

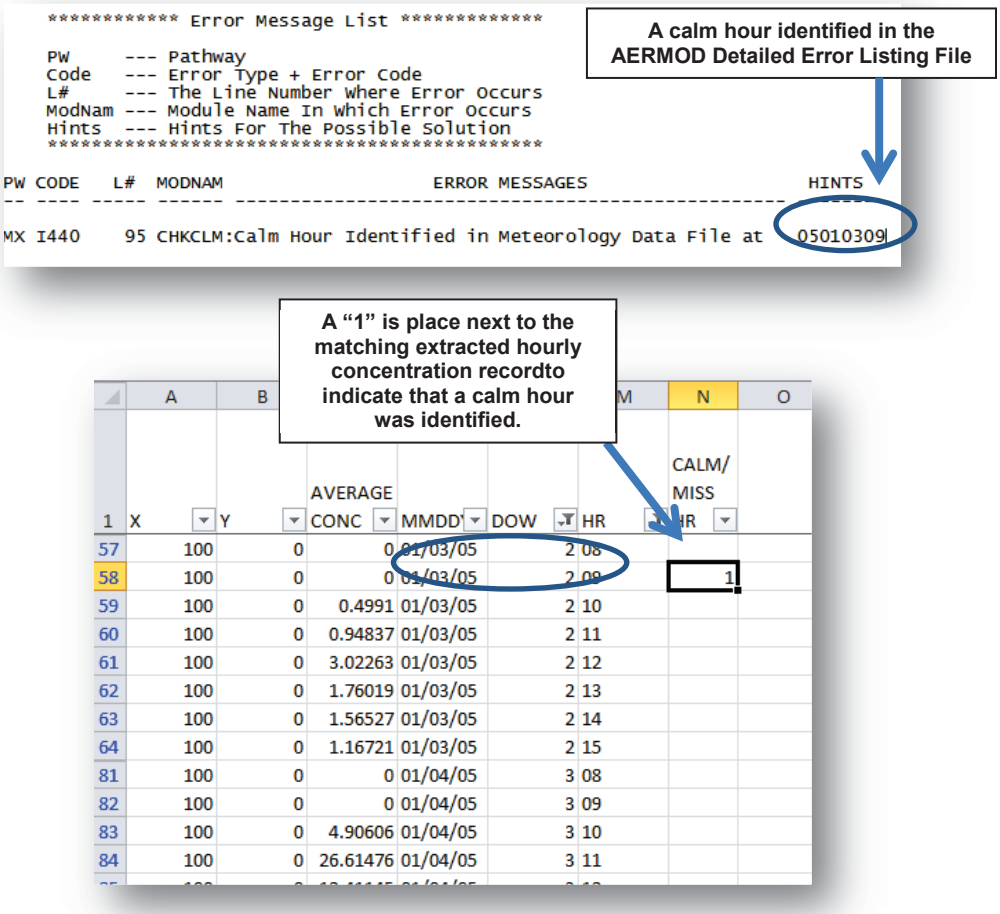
A	B	C	D	E	F	G	H	I	J	K	L
X	Y	AVERAGE CONC	ZELEV	ZHILL	ZFLAG	AVE	GRP	DATE	NET ID	MMDD	DOW
100	0	110.2656	10	10	1.2	1-HR	S010	05082610		08/26/05	
100	0	105.365	10	10	1.2	1-HR	S010	05082315		08/23/05	
100	0	105.1168	10	10	1.2	1-HR	S010	05080512		08/05/05	
100	0	103.7613	10	10	1.2	1-HR	S010	05071310		07/13/05	
100	0	103.6595	10	10	1.2	1-HR	S010	05082314		08/23/05	
100	0	103.6498	10	10	1.2	1-HR	S010	05071113		07/11/05	
100	0	103.2635	10	10	1.2	1-HR	S010	05082413		08/24/05	
100	0	103.0836	10	10	1.2	1-HR	S010	05012012		01/20/05	
100	0	102.8738	10	10	1.2	1-HR	S010	05052310		05/23/05	

M.4.2 How to Determine the Long-Term Average of 8-Hour Daily Concentrations for an 8-Hour Assessment

To calculate the long-term 8-hour daily average concentration, the 8-hour averages are first calculated for each day in the air dispersion analysis. All the 8-hour averages are then averaged over the entire time period of the air dispersion analysis. However, since the 8-hour daily average is considered a short-term average, the total number of valid hours (i.e., not calm or not missing) must be considered. The total number of valid hours should be 75% of the 8-hour average. If the total number of valid hours in an 8-hour average is less than six (6), the 8-hour total concentration should be divided by six (6) (U.S. EPA, 2005). The following steps below are an example that shows how the average of 8-hour daily concentration is calculated.

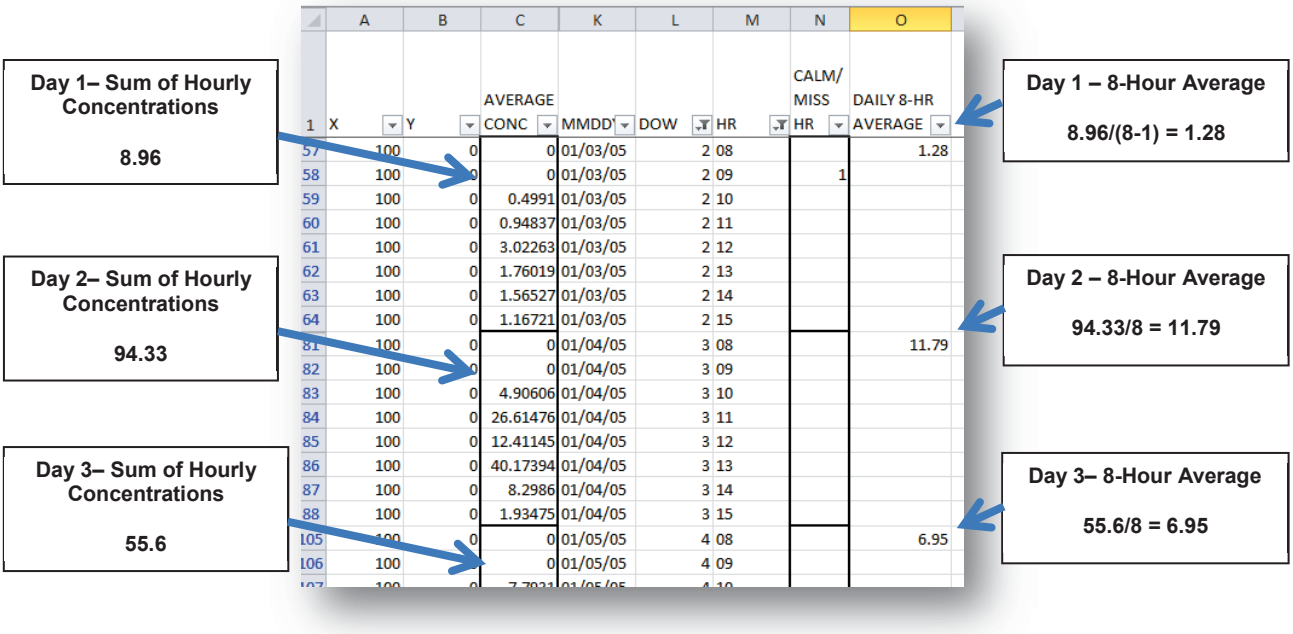
- Using the extracted hourly concentrations based on the steps from Section 3.0, identify any calm and missing hours with a “1”. To do this, use the Detailed Error Listing File that was created from the air dispersion analysis (See Section 2.1 for more information).The Detailed Error Listing File will list the calm and missing hours by date. Place a “1” where the dates match up with the extracted hourly concentrations (See Column N in Figure M.4.2.1). Please note that some of the columns are hidden in Figure M.4.2.1 for presentation purposes.

Figure M.4.2.1. Identify Calm and Missing Hours



- Then calculate the 8-hour average for each day throughout the file. The 8-hour average is the sum of the hourly concentrations in a day divided by eight (see Figure M.4.2.2). However, if there are any calm or missing hours in the time period, the sum of hourly concentrations should be divided by total number of valid hours. The total number of valid hours is eight minus the total number of calm and missing hours. If the total number of valid hours is less than six, then the sum of hourly concentrations should be divided by six.

Figure M.4.2.2. 8-Hour Daily Average Calculation



	A	B	C	K	L	M	N	O
1	X	Y	AVERAGE CONC	MMDD	DOW	HR	CALM/ MISS HR	DAILY 8-HR AVERAGE
57	100	0	0	01/03/05	2	08		1.28
58	100	0	0	01/03/05	2	09	1	
59	100	0	0.4991	01/03/05	2	10		
60	100	0	0.94837	01/03/05	2	11		
61	100	0	3.02263	01/03/05	2	12		
62	100	0	1.76019	01/03/05	2	13		
63	100	0	1.56527	01/03/05	2	14		
64	100	0	1.16721	01/03/05	2	15		
81	100	0	0	01/04/05	3	08		11.79
82	100	0	0	01/04/05	3	09		
83	100	0	4.90606	01/04/05	3	10		
84	100	0	26.61476	01/04/05	3	11		
85	100	0	12.41145	01/04/05	3	12		
86	100	0	40.17394	01/04/05	3	13		
87	100	0	8.2986	01/04/05	3	14		
88	100	0	1.93475	01/04/05	3	15		
105	100	0	0	01/05/05	4	08		6.95
106	100	0	0	01/05/05	4	09		

- Assuming that there were only three days in the entire time period of the air dispersion analysis, the average of 8-hour daily concentrations is (1.28 11.79 + 6.95) /3 = 6.78.

M.4.3 Equation for Calculating the Average Concentration for the Inhalation Cancer Pathway

Below is the equation for calculating the period average for the inhalation cancer pathway. This calculation must be repeated at each receptor for each source of interest.

$$C_{worker\_period\_average} = \frac{\sum C_{hourly}}{N_{total\_hrs} - N_{calm\_hrs} - N_{missing\_hrs}}$$

Where:

C_{hourly} = the concentration that occurs during the worker's shift. To obtain the sum of the hourly concentrations for the offsite worker, sum the extracted worker concentrations from Section 3.0.

N_{total_hrs} = the number of processed hours that occur during worker's shift. To obtain the number of processed hours, use the COUNT function to return the total number of extracted worker concentrations from Section 3.0.

N_{calm_hrs}= the number of calm hours that occur during the worker's shift. To obtain the number of calm and missing hours, use the COUNT function to return the total number of missing and calm hours from Section 3.0. Since the total will include missing hours, it is not necessary to repeat this step for the variable below.

$N_{missing\_hrs}$ = the number of missing hours that occur during worker's shift.

M.5 References

U.S. EPA (2004). User's Guide for the AMS/EPA Regulatory Model – AERMOD. EPA-454/B-03-001.U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2005). Guideline on Air Quality Models (Revised). 40 CFR 51, Appendix W.

Appendix N

Sensitivity Study of the Worker Adjustment Factor using AERMOD

N.1. Introduction

The offsite worker health risk analysis begins with estimating the pollutant concentration at a receptor location. To estimate this concentration, the typical approach is to use the residential annual concentration that is modeled based on the adjacent facility's emission schedule. However, if the facility emissions are non-continuous, the residential concentration may not represent what the worker breathes during their work shift. In lieu of conducting additional special case modeling which can be time-consuming, the residential annual concentration is adjusted upwards using a worker adjustment factor based on the facility's emission schedule with respect to the worker's schedule. For an 8-hour work shift that coincides with an adjacent facility that emits eight hours per day, a worker adjustment factor of 4.2 (24 hours / 8 hours * 7 days / 5 days) is typically used for cancer risk assessment.

A possible problem with using this approach is that wind direction, wind speed, and atmospheric stability can vary throughout the day and night and straight scaling as above may skew the results. If the diurnal variation is considerable, the 4.2 adjustment could be an under- or overestimate depending on the time of day that the offsite worker shift begins and ends. The goal of this study is to test the validity of the 4.2 adjustment using five meteorological data sets from five different locations in California and with three different size point sources. The modeling is performed with 8-hour emissions coinciding with the offsite workers' schedule. The 8-hour shifts are modeled as starting every hour around the clock.

To perform this study, the AERMOD air dispersion model, meteorological data from five locations (i.e., Kearny Mesa, Palomar, Pomona, Redlands, and San Bernardino), and three different size point sources (small, medium, and large) are used. The AERMOD-ready meteorological datasets are selected to represent a range of meteorological conditions around the state. To mirror the assumptions used in the 4.2 worker adjustment factor, the emission rate of each source is simulated for eight continuous hours with 24 different start times for five days a week (Monday through Friday). This will simulate the conditions that result during an 8-hour work schedule starting any hour of the day. In addition, the emitting source and offsite worker are assumed to have coincident schedules.

Using the AERMOD air dispersion modeling results, the Point of Maximum Impact (PMI) is identified and the hourly raw concentrations are post-processed to calculate the long-term offsite worker concentration for each scenario. To test the validity of the worker adjustment factor, the calculated long-term offsite worker concentration is divided by the long term residential average to obtain a quotient that is unique to each

meteorological data location. The quotient is then compared to the 4.2 worker adjustment factor to see which is higher or more health protective.

Although this study is primarily based on an 8-hour work schedule, the actual duration that an offsite worker is present near the emitting source may vary when considering a lunch break or a longer work shift. Thus, 10-hour scenarios are also evaluated. The worker adjustment factor for ten hours is 3.4 (24 hours / 10 hours * 7 days / 5 days).

N.2. Background on the Worker Adjustment Factor for Inhalation Cancer Assessments

There are basically two approaches that can be used to calculate the offsite worker inhalation exposure for cancer assessments. One approach is to post-process the hourly dispersion modeling results and examine the coincident hours between the source's emission schedule and the worker's schedule. The second, and more commonly used approach, is to apply a worker adjustment factor to the modeled long-term residential concentration. While post-processing the hourly modeling output will offer a more representative worker concentration, it is very time consuming and requires the management of large amounts of data. Thus, the simplistic approach of applying a worker adjustment factor to estimate the worker inhalation exposure is typically used.

The worker adjustment factor is used together with the long-term residential concentration to estimate the offsite worker's inhalation exposure. This calculation is summarized below.

a. Obtain the long-term concentrations from air dispersion modeling as is typical for residential receptors (all hours of a year or multi-year analysis are used).

b. Determine the coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule.

c. Calculate the worker adjustment factor using Equation N.1. When assessing inhalation cancer health impacts, a discount factor (*DF*) may also be applied if the offsite worker's schedule partially overlaps with the source's emission schedule. The discount factor is based on the number of coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule (see Equation N.2).

Please note that worker adjustment factor does not apply if the source's emission schedule and the offsite worker's schedule do not overlap. Since the worker is not around during the time that the source is emitting, the worker is not exposed to the source's emission (i.e., the DF in Equation N.2 becomes 0).

$$WAF = \frac{H_{residential}}{H_{source}} \times \frac{D_{residential}}{D_{source}} \times DF \qquad \textbf{Eq. N.1}$$

Where:

WAF = the worker adjustment factor

$H_{residential}$ = the number of hours per day the long-term residential concentration is based on (24)

H_{source} = the number of hours the source operates per day

$D_{residential}$ = the number of days per week the long-term residential concentration is based on (7).

D_{source} = the number of days the source operates per week.

DF = a discount factor for when the offsite worker's schedule partially overlaps the source's emission schedule. Use 1 if the offsite worker's schedule occurs within the source's emission schedule. If the offsite worker's schedule partially overlaps with the source's emission schedule, then calculate the discount factor using Equation N.2 below.

$$DF = \frac{H_{coincident}}{H_{worker}} \times \frac{D_{coincident}}{D_{worker}} \qquad \textbf{Eq. N.2}$$

Where:

DF = *the discount factor for assessing cancer impacts*

$H_{coincident}$ = *the number of hours per day the offsite worker's schedule and the source's emission schedule overlap*

$D_{coincident}$ = *the number of days per week the offsite worker's schedule and the source's emission schedule overlap.*

H_{worker} = *the number of hours the offsite worker works per day*

D_{worker} = *the number of days the offsite worker works per week.*

d. The final step is to estimate the offsite worker inhalation exposure by multiplying the worker adjustment factor with the long-term residential concentration.

N.3. Method and Modeling Parameters

For this study, all scenarios are simulated using the AERMOD (Version 09292) air dispersion model. The modeling parameters input to AERMOD and methods used to process the model outputs are discussed below.

N.3.1. Point Source Release Parameters

This study uses three different size point sources representing small, medium, and large. The point source release parameters are shown in Table N.1.

Table N.1. Point Source Modeling Parameters

Source Size	Emission Rate (g/s)	Release Ht (m)	Diameter (m)	Exit Temp (K)	Exit Vel (m/s)	Building Dimensions L (m) x W (m) x H (m)	XBADJ YBADJ [1]
Large	1	30	3	400	10	15 x 15 x 6	7.5
Medium	1	10	1	400	10	12 x 12 x 6	6
Small	1	2.15	0.1	400	10	6 x 6 x 2	3

1 – The XBADJ and YBADJ are keywords defining the along-flow and across-flow distances from the stack to the center of the upwind face of the projected building, respectively (U.S. EPA, 2004).

N.3.2. Temporal Emission Rate

Each point source (i.e., small, medium, and large) is simulated with continuous emissions for eight hours a day from Monday through Friday. In addition, all starting hour combinations (24 scenarios) are evaluated by duplicating each source 24 times with unique start times. Table N.2 shows the 8-hour operating schedule for each scenario. All emissions for Saturday and Sunday are set at zero. This process will also be repeated for the 10-hour evaluation. Table N.3 shows the 10-hour operating schedule for each scenario.

Table N.2. 8-Hour Operating Schedule

Time	Scenario																							
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
12:00 AM	ON																	ON	ON	ON	ON	ON	ON	ON
1:00 AM	ON	ON																	ON	ON	ON	ON	ON	ON
2:00 AM	ON	ON	ON																	ON	ON	ON	ON	ON
3:00 AM	ON	ON	ON	ON																	ON	ON	ON	ON
4:00 AM	ON	ON	ON	ON	ON																	ON	ON	ON
5:00 AM	ON	ON	ON	ON	ON	ON																	ON	ON
6:00 AM	ON	ON	ON	ON	ON	ON	ON																	ON
7:00 AM	ON	ON	ON	ON	ON	ON	ON	ON																
8:00 AM		ON	ON	ON	ON	ON	ON	ON	ON															
9:00 AM			ON	ON	ON	ON	ON	ON	ON	ON														
10:00 AM				ON	ON	ON	ON	ON	ON	ON	ON													
11:00 AM					ON	ON	ON	ON	ON	ON	ON	ON												
12:00 PM						ON	ON	ON	ON	ON	ON	ON	ON											
1:00 PM							ON	ON	ON	ON	ON	ON	ON	ON										
2:00 PM								ON	ON	ON	ON	ON	ON	ON	ON									
3:00 PM									ON	ON	ON	ON	ON	ON	ON	ON								
4:00 PM										ON	ON	ON	ON	ON	ON	ON	ON							
5:00 PM											ON	ON	ON	ON	ON	ON	ON	ON						
6:00 PM												ON	ON	ON	ON	ON	ON	ON	ON					
7:00 PM													ON	ON	ON	ON	ON	ON	ON	ON				
8:00 PM														ON	ON	ON	ON	ON	ON	ON	ON			
9:00 PM															ON	ON	ON	ON	ON	ON	ON	ON		
10:00 PM																ON	ON	ON	ON	ON	ON	ON	ON	
11:00 PM																	ON	ON	ON	ON	ON	ON	ON	ON

Table N.3. 10-Hour Operating Schedule

Time	Scenario																							
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
12:00 AM	ON															ON	ON	ON	ON	ON	ON	ON	ON	ON
1:00 AM	ON	ON															ON	ON	ON	ON	ON	ON	ON	ON
2:00 AM	ON	ON	ON															ON	ON	ON	ON	ON	ON	ON
3:00 AM	ON	ON	ON	ON															ON	ON	ON	ON	ON	ON
4:00 AM	ON	ON	ON	ON	ON															ON	ON	ON	ON	ON
5:00 AM	ON	ON	ON	ON	ON	ON															ON	ON	ON	ON
6:00 AM	ON	ON	ON	ON	ON	ON	ON															ON	ON	ON
7:00 AM	ON	ON	ON	ON	ON	ON	ON	ON															ON	ON
8:00 AM	ON	ON	ON	ON	ON	ON	ON	ON	ON															ON
9:00 AM	ON	ON	ON	ON	ON	ON	ON	ON	ON	ON														
10:00 AM		ON	ON	ON	ON	ON	ON	ON	ON	ON	ON													
11:00 AM			ON	ON	ON	ON	ON	ON	ON	ON	ON	ON												
12:00 PM				ON	ON	ON	ON	ON	ON	ON	ON	ON	ON											
1:00 PM					ON	ON	ON	ON	ON	ON	ON	ON	ON	ON										
2:00 PM						ON	ON	ON	ON	ON	ON	ON	ON	ON	ON									
3:00 PM							ON	ON	ON	ON	ON	ON	ON	ON	ON	ON								
4:00 PM								ON	ON	ON	ON	ON	ON	ON	ON	ON	ON							
5:00 PM									ON	ON	ON	ON	ON	ON	ON	ON	ON	ON						
6:00 PM										ON	ON	ON	ON	ON	ON	ON	ON	ON	ON					
7:00 PM											ON	ON	ON	ON	ON	ON	ON	ON	ON	ON				
8:00 PM												ON	ON	ON	ON	ON	ON	ON	ON	ON	ON			
9:00 PM													ON	ON	ON	ON	ON	ON	ON	ON	ON	ON		
10:00 PM														ON	ON	ON	ON	ON	ON	ON	ON	ON	ON	
11:00 PM															ON	ON	ON	ON	ON	ON	ON	ON	ON	ON

N.3.3. Receptor Grid Parameters

A 1000 meter by 1000 meter receptor grid is centered over each source. The receptors are spaced in 50 meter increments resulting in 441 receptor points. All receptor flagpole heights are set at 1.2 meters above ground.

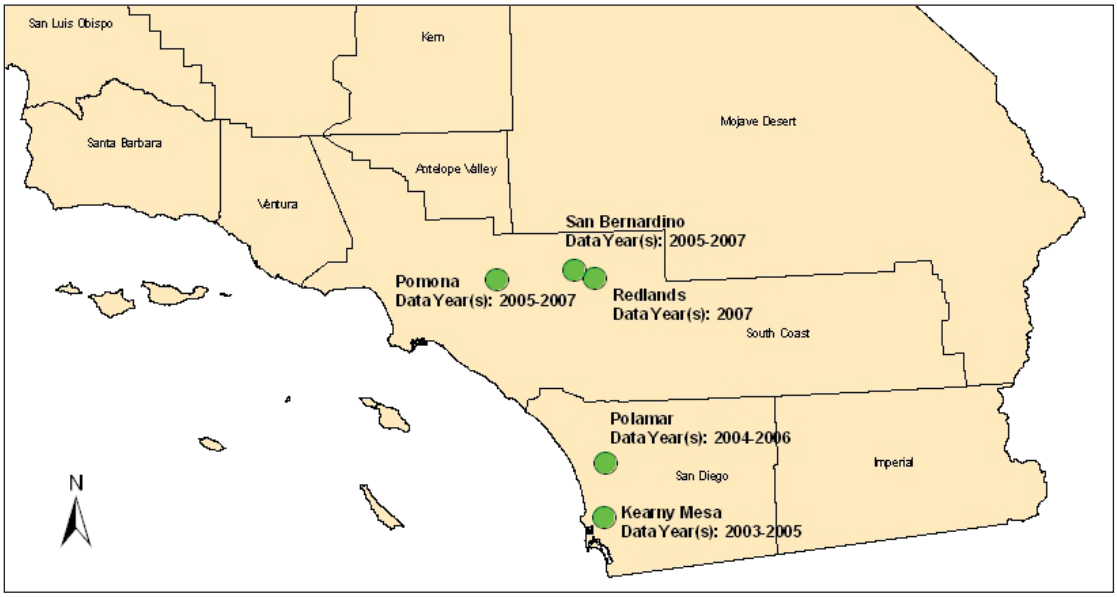
N.3.4. Meteorological Data

The meteorological data input to AERMOD were requested from two local air districts in California (ARB 2009a and ARB 2009b). The meteorological data that were provided by the Districts are, based on the Districts' observations and expertise, datasets that were likely to result in higher than average long-term impacts. The data includes four multi-year files and one single year file. Table N.4 shows the meteorological datasets used in this study. Figure N.1 shows the location of the meteorological station. The AERMOD profile base is defaulted to 10 meters above mean sea level for each meteorological file.

Table N.4. Meteorological Datasets

Data Provider	Area	Data Year(s)	Total Hours	Percent of Calm and Missing Hours	Avg. Wind Speed (m/s)
San Diego Air Pollution Control District	Kearny Mesa	2003-2005	26304	6.9	1.36
	Palomar	2004-2006	26304	8.7	1.36
South Coast Air Quality Management District	Pomona	2005-2007	26280	1.6	1.18
	Redlands	2007	8760	5.5	0.94
	San Bernardino	2005-2007	26280	4.9	1.44

Figure N.1. Meteorological Data Set Locations



N.3.5. Post-Processing the Period Average Concentrations for the Offsite Worker

The period average concentration represents the average concentration of all hours processed within the meteorological set. Equation N.3 shows how the period average is calculated in AERMOD including how calm and missing hours are processed (U.S. EPA, 2005).

$$C_{period\_average} = \frac{\sum C_{hourly}}{N_{total\_hrs} - N_{calm\_hrs} - N_{missing\_hrs}} \qquad \textbf{Eq. N.3}$$

Where:

C_{hourly} = the concentration that occurs at a given hour

N_{total_hrs} = the number of processed hours reported by AERMOD (e.g., 1 yr = 8760 hours)

N_{calm_hrs} = the number of calm hours reported by AERMOD

$N_{missing\_hrs}$ = the number of missing hours reported by AERMOD

Normally to post-process hourly data, the off-site worker hours are extracted from the hourly model output files and then averaged. However, this sensitivity study assumes the hourly emissions are coincident with the off-site worker schedule. Since this is the case, the 8-hour period average for the offsite worker can simply be scaled from the period average reported by AERMOD (see Equation N.4). To make sure this calculation is accurate, a check was performed by processing the hourly concentrations for one receptor with the Pomona data. If the emission schedule was not 100%

coincident with the offsite worker, then all post-processing would have to be completed on an hourly basis. See Appendix M for more information on how to post-process worker concentrations using hourly raw results.

$$C_{worker\_period\_average} = C_{period\_average} \times \frac{N_{total\_hrs} - N_{calm\_hrs} - N_{missing\_hrs}}{N_{worker\_hrs} - N_{worker\_calm\_hrs} - N_{worker\_missing\_hrs}} \quad \textbf{Eq. N.4}$$

Where:

$C_{period\_average}$ = the period concentration reported by AERMOD

N_{total_hrs} = the total number of processed hours reported by AERMOD

N_{calm_hrs} = the total number of calm hours reported by AERMOD

$N_{missing\_hrs}$ = the total number of missing hours reported by AERMOD

N_{worker_hrs}[a] = the total number of hours that occurred during the worker's shift

$N_{worker\_calm\_\_hrs}$[b] = the number of calm hours that occurs during the worker's shift

$N_{worker\_missing\_hrs}$[b] = the number of missing hours that occurred during the worker's shift

a. The worker hours are determined by multiplying the number of weekdays (Monday through Friday) that occurs in the meteorological data set by the work shift duration (8 hours). For example, a meteorological data set ranging from 1/1/2003 to 12/31/2005 contains 783 weekdays. If you multiply the number weekdays by the work shift duration (8 hour/day), this will equal 6264 worker hours. The number of weekdays varies depending on the day of the week January 1st starts on.

b. Calm and missing hours are reported in the AERMOD Detailed Message Listing File. To determine the number of worker calm and missing hours, the calm and missing hours that occur during the worker shift are isolated and summed.

N.4. Results

To test the validity of the worker adjustment factor, the post-processed period average concentration for the offsite worker was divided by the modeled period residential average to obtain a quotient. This calculation was performed at the PMI of each scenario. If the quotient is smaller or equal to the worker adjustment factor, the worker adjustment factor is considered a suitable health protective approximation. If the quotient is greater, the worker adjustment factor will underestimate the long-term average concentration and would not be the most conservative estimation of what the worker breathes. For these scenarios, the 8-hour and 10-hour worker adjustment factors are 4.2 and 3.4, respectively. The results for this study are summarized in the figures and tables below. To view the details for every scenario, see Appendix N-1.

Figure N.2 shows how the post-processed period averages changes over 8-hour rolling work shifts. The value at each 8-hour work shift represents the quotient average across the five meteorological data sets. Values that fall on or below the thick dashed line (i.e., the 4.2 worker adjustment factor) indicate that the worker adjustment factor would be a health protective value. Based on the five metrological data sets, the worker adjustment factor is health protective for work shifts that start approximately between 8 am and 3 pm (i.e., 8-hour work shifts starting at 8 am and ending by 11 pm).

Figure N.2. Summary of the 8-Hour Scenarios

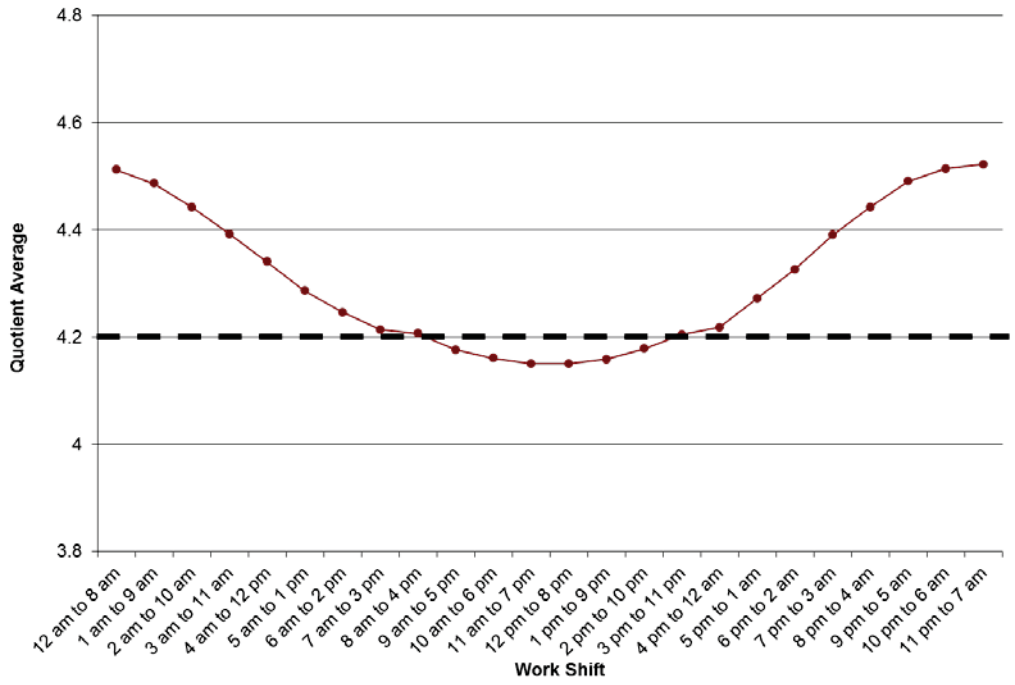


Figure N.3 shows relationship between the worker schedule and the percent of calm and missing hours that occurred during 8-hr work shifts. The figure shows the percent of calm and missing hours are higher during the early morning and evening hour start hours.

Figure N.3. Average Percent of Calm and Missing Hours for 8-Hour Work Shifts

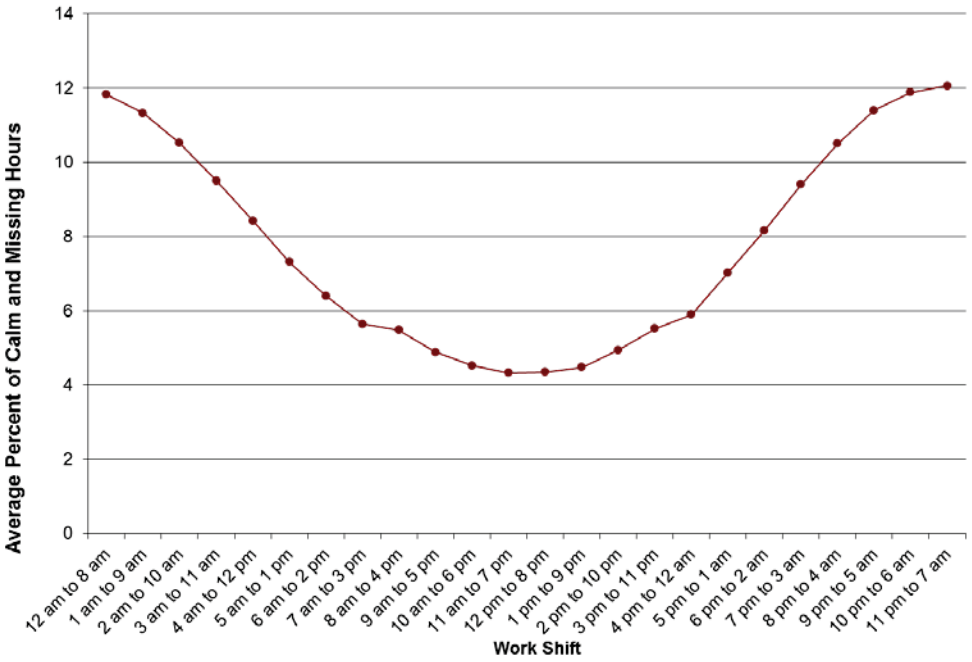


Figure N.4 shows how the post-processed period averages change over 10-hour rolling work shifts. The value at each 10-hour work shift represents the quotient average across the five meteorological data sets. Values that fall on or below the thick dashed line (i.e., the 3.4 worker adjustment factor) indicate that the worker adjustment factor would be a health protective value. Based on the five metrological data sets, the worker adjustment factor is health protective for work shifts that start approximately between 5 am and 4 pm (i.e., 10-hour work shifts starting at 5 am and ending by 2 am).

Figure N.4. Summary of the 10-Hour Scenarios

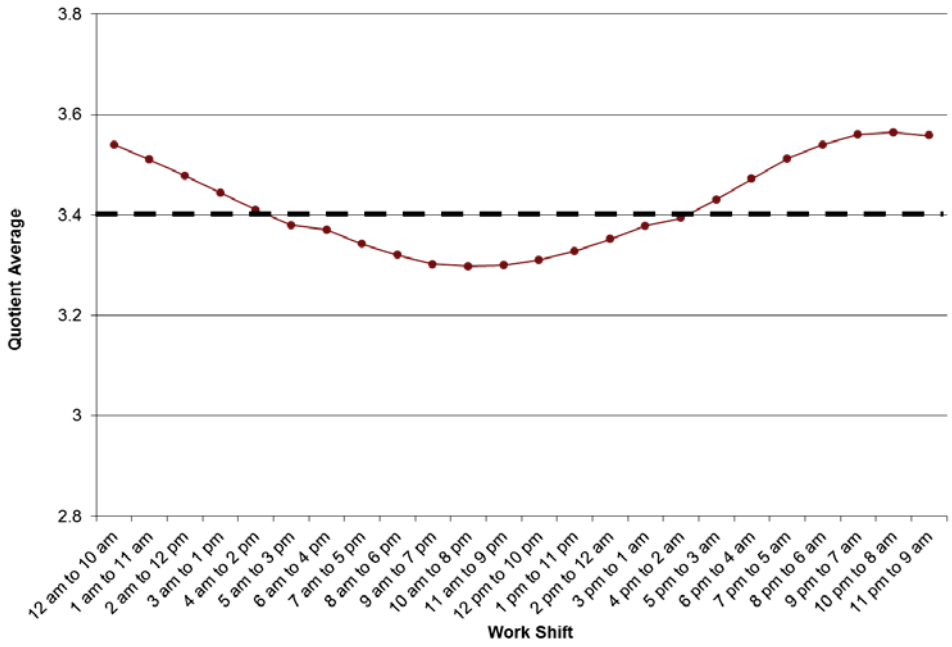


Figure N.5 shows relationship between the worker schedule and the percent of calm and missing hours that occurred during 10-hr work shifts. The figure shows the percent of calm and missing hours are higher during the early morning and evening hour start hours.

Figure N.5. Average Percent of Calm and Missing Hours for 10-Hour Work Shifts

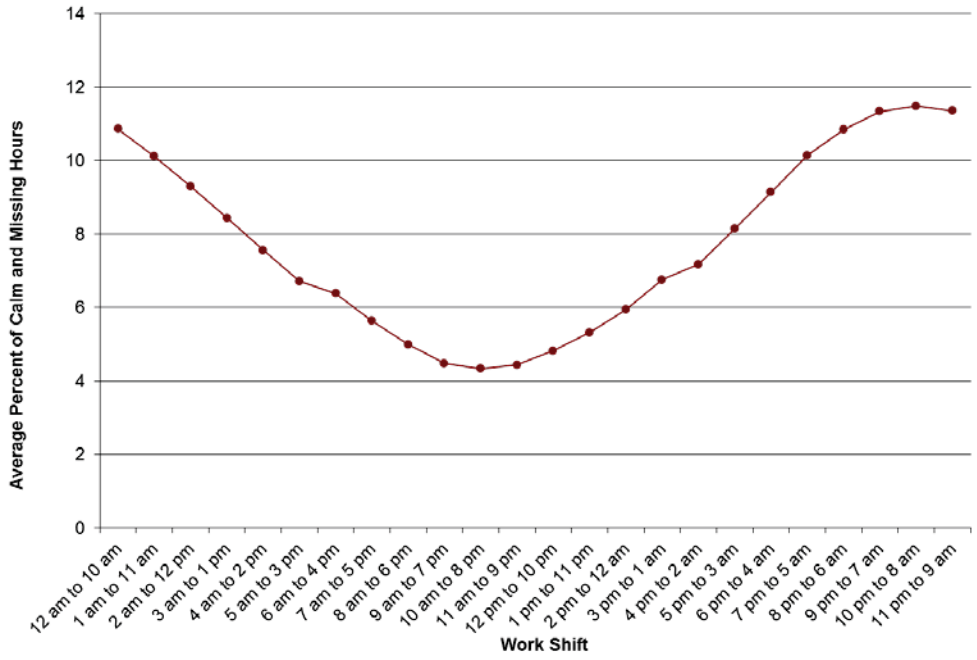


Table N.5 shows the average, minimum, and maximum quotients across all 24 8-hour work shifts for each point source size (i.e., small, medium, and large). The values in the parentheses are the range across the 24 work shifts for each meteorological data set.

Table N.5. Summary of the Average 8-Hour Scenarios by Point Source Size

Meteorological Set	Point Source Size			% Calm/Missing Hours During the Worker's Shift
	Small	Medium	Large	
Kearny Mesa	4.33 (4.19 to 4.43)	4.33 (4.19 to 4.43)	4.33 (4.19 to 4.43)	9.6 (6.8 to 11.8)
Palomar	4.38 (4.18 to 4.65)	4.38 (4.18 to 4.65)	4.38 (4.18 to 4.65)	12.2 (8.2 to 17.5)
Pomona	4.24 (4.23 to 4.25)	4.24 (4.23 to 4.25)	4.24 (4.23 to 4.25)	2.3 (2.1 to 2.5)
Redlands	4.31 (4.00 to 4.75)	4.31 (4.00 to 4.75)	4.31 (4.00 to 4.75)	7.6 (1.0 to 16.5)
San Bernardino	4.31 (4.06 to 4.65)	4.31 (4.06 to 4.65)	4.31 (4.06 to 4.65)	6.9 (1.4 to 14.1)

Table N.6 shows the average, minimum, and maximum quotients across all 24 10-hour work shifts for each point source size (i.e., small, medium, and large). The values in the parentheses are the range across the 24 work shifts for each meteorological data set.

Table N.6. Summary of the Average 10-Hour Scenarios by Point Source Size

Meteorological Set	Point Source Size			% Calm/Missing Hours During the Worker's Shift
	Small	Medium	Large	
Kearny Mesa	3.46 (3.38 to 3.54)	3.46 (3.38 to 3.54)	3.46 (3.38 to 3.54)	9.6 (7.5 to 11.6)
Palomar	3.50 (3.34 to 3.70)	3.50 (3.34 to 3.70)	3.50 (3.34 to 3.70)	12.2 (8.0 to 17.1)
Pomona	3.39 (3.38 to 3.39)	3.39 (3.38 to 3.39)	3.39 (3.38 to 3.39)	2.3 (2.2 to 2.5)
Redlands	3.45 (3.21 to 3.74)	3.45 (3.21 to 3.74)	3.45 (3.21 to 3.74)	7.6 (1.1 to 15.2)
San Bernardino	3.31 (3.12 to 3.54)	3.31 (3.12 to 3.54)	3.31 (3.12 to 3.54)	6.9 (1.5 to 13.1)

N.5. Conclusions

The goal of this study was to determine if the worker adjustment factor of 4.2 (8 hours/day, 5 days/week) or 3.4 (10 hours/day, 5 days/week) would always yield a more conservative or health protective approximation using five meteorological data sets. This study demonstrated that the worker adjustment factor does not always represent the most health protective approximation of long-term hourly model predictions. This is primarily observed during night conditions. Air Districts may wish to evaluate their meteorological data to determine an appropriate worker adjustment factor for their area using the methods described in this appendix.

Although the meteorological data used in this study are site-specific, several general conclusions and recommendations can be made. These conclusions and recommendations are summarized below.

- ***The worker adjustment factor is generally a suitable health protective approximation for daytime work shifts.***

  For the meteorological data used in this study, the results show that the worker adjustment factor is a suitable health protective approximation for work shifts that occur during the daytime hours. When comparing the 8-hour and 10-hour scenarios, the results show that the range of work shifts that were considered a more health protective approximation increased with the longer work shift duration.

- ***The size of the emitting source did not affect the long-term concentration approximated with the worker adjustment factor.***

  The size of the source was inconsequential in determining whether the worker adjustment factor is health protective. This is because the worker adjustment factor is applied to the modeling results after the air dispersion analysis has been completed. However, it should be noted that the size of the source does affect the location of the PMI during a specific time of day. This is shown in the scenario details in Appendix N-1.

- ***The worker adjustment factor may not represent the most conservative estimation of the worker's inhalation exposure for nighttime work shifts.***

  In most cases, the worker adjustment factor will represent a health protective approximation for work shifts that occur during the daytime. However, the worker adjustment factor may not represent the most conservative estimation when the source's emission schedule and offsite worker's schedules are 100% coincident at night. It is recommended that the offsite worker long-term average concentrations be post-processed using the hourly dispersion modeling results when examining work shifts occurring at night. Alternatively, a more conservative worker adjustment factor can be used to account for the calm hours (see the next bullet point below).

- ***Recommended worker adjustment factor for 8 and 10-hour work shifts***

  Based on the five meteorological data sets used in this study, the range of worker adjustment factors (WAF) was between 4.2 and 4.8. We recommend using the 4.2 WAF for most cases. In the event of predominant night time emissions and worker schedule or if only one year of meteorological data are available, then we recommend using 4.8 for the 8-hour WAF.

N.6. References

ARB (2009a). Harris, Gregory. "Aermod met data in San Diego." Email to Ralph Desina, San Diego Air Pollution Control District.

ARB (2009b). Harris, Gregory. "Aermod met data in SC." Email to Tom Chico, South Coast Air Quality Management District.

U.S. EPA (2004). User's Guide for the AMS/EPA Regulatory Model – AERMOD. EPA-454/B-03-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2005). Guideline on Air Quality Models (Revised). 40 CFR 51, Appendix W.

APPENDIX N-1 – SCENARIO DATA DETAILS

KEARNY MESA - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	500	0.02584	26304	1813	632.84744	6264	723	11.5	0.11421	**4.42**
2	0	300	0.05638	26304	1813	1380.80258	6264	739	11.8	0.24992	**4.43**
3	150	-150	0.10366	26304	1813	2538.73706	6264	729	11.6	0.45867	**4.42**
4	150	-100	0.19993	26304	1813	4896.48563	6264	718	11.5	0.88289	**4.42**
5	200	-100	0.33363	26304	1813	8170.93233	6264	700	11.2	1.46854	**4.40**
6	200	-100	0.48136	26304	1813	11788.98776	6264	688	11.0	2.11424	**4.39**
7	200	-100	0.62685	26304	1813	15352.18335	6264	684	10.9	2.75129	**4.39**
8	200	-100	0.76245	26304	1813	18673.16295	6264	681	10.9	3.34465	**4.39**
9	200	-100	0.85443	26304	1813	20925.84513	6264	665	10.6	3.73743	**4.37**
10	250	-100	0.89012	26304	1813	21799.92892	6264	618	9.9	3.86113	**4.34**
11	250	-100	0.85448	26304	1813	20927.06968	6264	568	9.1	3.67399	**4.30**
12	250	-100	0.76187	26304	1813	18658.95817	6264	517	8.3	3.24673	**4.26**
13	250	-100	0.63409	26304	1813	15529.49819	6264	488	7.8	2.68863	**4.24**
14	250	-100	0.48738	26304	1813	11936.42358	6264	467	7.5	2.05907	**4.22**
15	300	-150	0.34902	26304	1813	8547.84882	6264	454	7.2	1.47123	**4.22**
16	300	-150	0.20978	26304	1813	5137.72198	6264	433	6.9	0.88110	**4.20**
17	300	-150	0.09739	26304	1813	2385.17849	6264	425	6.8	0.40849	**4.19**
18	350	-200	0.02843	26304	1813	696.27913	6264	456	7.3	0.11988	**4.22**
19	0	500	0.00479	26304	1813	117.31189	6264	516	8.2	0.02041	**4.26**
20	-50	500	0.00491	26304	1813	120.25081	6264	578	9.2	0.02115	**4.31**
21	0	500	0.00512	26304	1813	125.39392	6264	625	10.0	0.02224	**4.34**
22	0	500	0.00513	26304	1813	125.63883	6264	658	10.5	0.02241	**4.37**
23	0	500	0.00528	26304	1813	129.31248	6264	675	10.8	0.02314	**4.38**
24	0	500	0.01002	26304	1813	245.39982	6264	699	11.2	0.04410	**4.40**

KEARNY MESA - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	100	0.48213	26304	1813	11807.84583	6264	723	11.5	2.13100	**4.42**
2	0	100	0.99949	26304	1813	24478.50959	6264	739	11.8	4.43050	**4.43**
3	50	50	1.69544	26304	1813	41523.02104	6264	729	11.6	7.50190	**4.42**
4	50	50	2.6458	26304	1813	64798.28780	6264	718	11.5	11.68379	**4.42**
5	50	50	3.51528	26304	1813	86092.72248	6264	700	11.2	15.47317	**4.40**
6	50	50	4.24949	26304	1813	104074.25959	6264	688	11.0	18.66468	**4.39**
7	100	-50	5.33685	26304	1813	130704.79335	6264	684	10.9	23.42380	**4.39**
8	100	-50	6.51541	26304	1813	159568.90631	6264	681	10.9	28.58121	**4.39**
9	100	-50	7.325	26304	1813	179396.57500	6264	665	10.6	32.04082	**4.37**
10	100	-50	7.60514	26304	1813	186257.48374	6264	618	9.9	32.98928	**4.34**
11	100	-50	7.28086	26304	1813	178315.54226	6264	568	9.1	31.30540	**4.30**
12	100	-50	6.51093	26304	1813	159459.18663	6264	517	8.3	27.74651	**4.26**
13	100	-50	5.53256	26304	1813	135497.92696	6264	488	7.8	23.45878	**4.24**
14	100	-50	4.37499	26304	1813	107147.88009	6264	467	7.5	18.48333	**4.22**
15	100	-50	3.13098	26304	1813	76680.83118	6264	454	7.2	13.19808	**4.22**
16	100	-50	1.92339	26304	1813	47105.74449	6264	433	6.9	8.07850	**4.20**
17	150	-50	0.97341	26304	1813	23839.78431	6264	425	6.8	4.08285	**4.19**
18	200	-100	0.37344	26304	1813	9145.91904	6264	456	7.3	1.57471	**4.22**
19	0	150	0.19509	26304	1813	4777.94919	6264	516	8.2	0.83124	**4.26**
20	0	150	0.18348	26304	1813	4493.60868	6264	578	9.2	0.79029	**4.31**
21	0	150	0.17623	26304	1813	4316.04893	6264	625	10.0	0.76539	**4.34**
22	0	150	0.16448	26304	1813	4028.27968	6264	658	10.5	0.71857	**4.37**
23	0	150	0.16295	26304	1813	3990.80845	6264	675	10.8	0.71405	**4.38**
24	0	150	0.22443	26304	1813	5496.51513	6264	699	11.2	0.98769	**4.40**

KEARNY MESA - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	50	56.94704	26304	1813	1394689.95664	6264	723	11.5	251.70366	**4.42**
2	0	50	63.90855	26304	1813	1565184.29805	6264	739	11.8	283.29128	**4.43**
3	0	50	72.78622	26304	1813	1782607.31402	6264	729	11.6	322.06094	**4.42**
4	0	50	80.59339	26304	1813	1973812.71449	6264	718	11.5	355.89843	**4.42**
5	0	50	86.44869	26304	1813	2117214.86679	6264	700	11.2	380.52029	**4.40**
6	50	0	96.25147	26304	1813	2357294.75177	6264	688	11.0	422.75731	**4.39**
7	50	0	117.66867	26304	1813	2881823.39697	6264	684	10.9	516.45581	**4.39**
8	50	0	138.64904	26304	1813	3395653.63864	6264	681	10.9	608.21308	**4.39**
9	50	0	156.76654	26304	1813	3839369.33114	6264	665	10.6	685.72412	**4.37**
10	50	0	172.75048	26304	1813	4230832.00568	6264	618	9.9	749.35034	**4.34**
11	50	0	184.10847	26304	1813	4509000.53877	6264	568	9.1	791.60824	**4.30**
12	50	0	190.80885	26304	1813	4673099.54535	6264	517	8.3	813.13721	**4.26**
13	50	0	183.97723	26304	1813	4505786.33993	6264	488	7.8	780.08766	**4.24**
14	50	0	168.91026	26304	1813	4136781.17766	6264	467	7.5	713.60724	**4.22**
15	50	0	150.42213	26304	1813	3683988.38583	6264	454	7.2	634.07717	**4.22**
16	50	-50	146.48297	26304	1813	3587514.41827	6264	433	6.9	615.24857	**4.20**
17	50	-50	144.08415	26304	1813	3528764.91765	6264	425	6.8	604.34405	**4.19**
18	50	-50	130.6006	26304	1813	3198539.29460	6264	456	7.3	550.71269	**4.22**
19	50	-50	111.9118	26304	1813	2740831.89380	6264	516	8.2	476.83227	**4.26**
20	50	-50	86.25428	26304	1813	2112453.57148	6264	578	9.2	371.51839	**4.31**
21	50	-50	65.37008	26304	1813	1600978.62928	6264	625	10.0	283.91180	**4.34**
22	0	50	56.60048	26304	1813	1386202.35568	6264	658	10.5	247.27120	**4.37**
23	0	50	53.20196	26304	1813	1302969.20236	6264	675	10.8	233.13101	**4.38**
24	-100	-100	54.24037	26304	1813	1328400.90167	6264	699	11.2	238.70636	**4.40**

PALOMAR - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	250	0.02363	26304	2291	567.42719	6256	1096	17.5	0.10997	**4.65**
2	100	150	0.0631	26304	2291	1515.22030	6256	1090	17.4	0.29331	**4.65**
3	150	50	0.14317	26304	2291	3437.94121	6256	1050	16.8	0.66038	**4.61**
4	150	50	0.27432	26304	2291	6587.24616	6256	971	15.5	1.24640	**4.54**
5	200	50	0.42859	26304	2291	10291.73167	6256	879	14.1	1.91403	**4.47**
6	200	50	0.58751	26304	2291	14107.87763	6256	788	12.6	2.58008	**4.39**
7	200	0	0.73867	26304	2291	17737.68271	6256	701	11.2	3.19310	**4.32**
8	200	0	0.87304	26304	2291	20964.30952	6256	628	10.0	3.72500	**4.27**
9	250	0	0.96493	26304	2291	23170.86409	6256	679	10.9	4.15472	**4.31**
10	250	0	0.99791	26304	2291	23962.81283	6256	589	9.4	4.22848	**4.24**
11	250	0	0.9484	26304	2291	22773.92920	6256	540	8.6	3.98424	**4.20**
12	250	0	0.83614	26304	2291	20078.22982	6256	518	8.3	3.49917	**4.18**
13	250	0	0.68595	26304	2291	16471.71735	6256	517	8.3	2.87014	**4.18**
14	250	0	0.51501	26304	2291	12366.93513	6256	523	8.4	2.15715	**4.19**
15	300	0	0.34888	26304	2291	8377.65544	6256	550	8.8	1.46822	**4.21**
16	300	-50	0.20229	26304	2291	4857.58977	6256	596	9.5	0.85823	**4.24**
17	300	-100	0.10109	26304	2291	2427.47417	6256	516	8.2	0.42290	**4.18**
18	300	-150	0.0311	26304	2291	746.80430	6256	612	9.8	0.13232	**4.25**
19	-450	-200	0.00583	26304	2291	139.99579	6256	701	11.2	0.02520	**4.32**
20	-400	-150	0.00576	26304	2291	138.31488	6256	802	12.8	0.02536	**4.40**
21	-400	-200	0.00503	26304	2291	120.78539	6256	895	14.3	0.02253	**4.48**
22	-400	-200	0.00427	26304	2291	102.53551	6256	980	15.7	0.01943	**4.55**
23	-400	-200	0.00323	26304	2291	77.56199	6256	1040	16.6	0.01487	**4.60**
24	-500	-500	0.0081	26304	2291	194.50530	6256	1067	17.1	0.03748	**4.63**

PALOMAR - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	50	0.39916	26304	2291	9585.02908	6256	1096	17.5	1.85756	**4.65**
2	50	50	1.1355	26304	2291	27266.76150	6256	1090	17.4	5.27812	**4.65**
3	50	50	2.23922	26304	2291	53770.38986	6256	1050	16.8	10.32854	**4.61**
4	50	50	3.46481	26304	2291	83200.48253	6256	971	15.5	15.74276	**4.54**
5	100	0	5.01511	26304	2291	120427.83643	6256	879	14.1	22.39685	**4.47**
6	100	0	7.1387	26304	2291	171421.60310	6256	788	12.6	31.34996	**4.39**
7	100	0	9.3361	26304	2291	224187.76930	6256	701	11.2	40.35783	**4.32**
8	100	0	11.30065	26304	2291	271362.50845	6256	628	10.0	48.21651	**4.27**
9	100	0	12.55274	26304	2291	301428.94562	6256	679	10.9	54.04858	**4.31**
10	100	0	12.9907	26304	2291	311945.67910	6256	589	9.4	55.04600	**4.24**
11	100	0	12.32253	26304	2291	295900.91289	6256	540	8.6	51.76713	**4.20**
12	100	0	10.99232	26304	2291	263958.58016	6256	518	8.3	46.00184	**4.18**
13	100	0	9.16435	26304	2291	220063.53655	6256	517	8.3	38.34528	**4.18**
14	100	0	7.04288	26304	2291	169120.67744	6256	523	8.4	29.49951	**4.19**
15	100	0	4.85232	26304	2291	116518.76016	6256	550	8.8	20.42039	**4.21**
16	100	0	2.83666	26304	2291	68116.71658	6256	596	9.5	12.03476	**4.24**
17	150	0	1.4789	26304	2291	35512.82570	6256	516	8.2	6.18690	**4.18**
18	150	0	0.51952	26304	2291	12475.23376	6256	612	9.8	2.21035	**4.25**
19	500	100	0.16252	26304	2291	3902.59276	6256	701	11.2	0.70254	**4.32**
20	-100	-50	0.13578	26304	2291	3260.48514	6256	802	12.8	0.59782	**4.40**
21	-100	-50	0.12284	26304	2291	2949.75692	6256	895	14.3	0.55023	**4.48**
22	-100	-50	0.10491	26304	2291	2519.20383	6256	980	15.7	0.47748	**4.55**
23	-150	-50	0.08895	26304	2291	2135.95635	6256	1040	16.6	0.40950	**4.60**
24	-100	0	0.15313	26304	2291	3677.11069	6256	1067	17.1	0.70864	**4.63**

PALOMAR - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	0	62.23758	26304	2291	1494511.00854	6256	1096	17.5	289.63392	**4.65**
2	-50	0	67.07392	26304	2291	1610646.04096	6256	1090	17.4	311.77817	**4.65**
3	-50	0	69.58692	26304	2291	1670990.70996	6256	1050	16.8	320.97401	**4.61**
4	50	0	76.6273	26304	2291	1840051.35490	6256	971	15.5	348.16487	**4.54**
5	50	0	101.35151	26304	2291	2433753.80963	6256	879	14.1	452.62299	**4.47**
6	50	0	132.881	26304	2291	3190871.45300	6256	788	12.6	583.55367	**4.39**
7	50	0	166.85749	26304	2291	4006748.90737	6256	701	11.2	721.28693	**4.32**
8	50	0	199.35655	26304	2291	4787148.83515	6256	628	10.0	850.59503	**4.27**
9	50	0	227.0465	26304	2291	5452067.60450	6256	679	10.9	977.59864	**4.31**
10	50	0	258.20597	26304	2291	6200299.95761	6256	589	9.4	1094.10622	**4.24**
11	50	0	284.95975	26304	2291	6842738.47675	6256	540	8.6	1197.12010	**4.20**
12	50	0	306.84919	26304	2291	7368369.59947	6256	518	8.3	1284.13552	**4.18**
13	50	0	305.48615	26304	2291	7335638.91995	6256	517	8.3	1278.20856	**4.18**
14	50	0	284.9321	26304	2291	6842074.51730	6256	523	8.4	1193.45448	**4.19**
15	50	0	255.29701	26304	2291	6130447.10113	6256	550	8.8	1074.38610	**4.21**
16	50	0	222.46841	26304	2291	5342133.92933	6256	596	9.5	943.83992	**4.24**
17	50	0	190.65477	26304	2291	4578192.99201	6256	516	8.2	797.59460	**4.18**
18	50	0	149.99496	26304	2291	3601828.97448	6256	612	9.8	638.16956	**4.25**
19	50	0	109.43689	26304	2291	2627908.03957	6256	701	11.2	473.07075	**4.32**
20	50	0	71.34752	26304	2291	1713267.99776	6256	802	12.8	314.13055	**4.40**
21	50	0	47.98635	26304	2291	1152296.22255	6256	895	14.3	214.94054	**4.48**
22	-50	50	46.33971	26304	2291	1112755.45623	6256	980	15.7	210.90892	**4.55**
23	-50	0	48.61618	26304	2291	1167420.33034	6256	1040	16.6	223.81525	**4.60**
24	-50	0	55.01306	26304	2291	1321028.60978	6256	1067	17.1	254.58250	**4.63**

POMONA - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	300	-100	0.0378	26280	432	977.05440	6248	138	2.2	0.15991	**4.23**
2	200	-50	0.08941	26280	432	2311.06968	6248	140	2.2	0.37837	**4.23**
3	200	-50	0.18145	26280	432	4690.11960	6248	142	2.3	0.76812	**4.23**
4	200	-50	0.30538	26280	432	7893.46224	6248	145	2.3	1.29337	**4.24**
5	200	-50	0.4489	26280	432	11603.16720	6248	147	2.4	1.90185	**4.24**
6	200	0	0.59344	26280	432	15339.23712	6248	152	2.4	2.51628	**4.24**
7	200	0	0.72765	26280	432	18808.29720	6248	154	2.5	3.08636	**4.24**
8	250	0	0.84968	26280	432	21962.52864	6248	157	2.5	3.60573	**4.24**
9	250	0	0.93127	26280	432	24071.46696	6248	159	2.5	3.95327	**4.25**
10	250	0	0.9478	26280	432	24498.73440	6248	158	2.5	4.02278	**4.24**
11	250	0	0.89255	26280	432	23070.63240	6248	157	2.5	3.78766	**4.24**
12	250	0	0.7753	26280	432	20039.95440	6248	154	2.5	3.28847	**4.24**
13	300	0	0.63398	26280	432	16387.11504	6248	149	2.4	2.68685	**4.24**
14	300	0	0.49462	26280	432	12784.93776	6248	145	2.3	2.09486	**4.24**
15	300	50	0.35974	26280	432	9298.55952	6248	142	2.3	1.52286	**4.23**
16	350	50	0.22753	26280	432	5881.19544	6248	139	2.2	0.96271	**4.23**
17	350	50	0.11619	26280	432	3003.27912	6248	135	2.2	0.49129	**4.23**
18	400	0	0.03912	26280	432	1011.17376	6248	134	2.1	0.16539	**4.23**
19	0	-50	0.0042	26280	432	108.56160	6248	133	2.1	0.01775	**4.23**
20	0	-50	0.00468	26280	432	120.96864	6248	133	2.1	0.01978	**4.23**
21	0	-50	0.0052	26280	432	134.40960	6248	136	2.2	0.02199	**4.23**
22	0	-50	0.00567	26280	432	146.55816	6248	135	2.2	0.02397	**4.23**
23	0	-50	0.00623	26280	432	161.03304	6248	136	2.2	0.02635	**4.23**
24	500	-250	0.01616	26280	432	417.70368	6248	136	2.2	0.06834	**4.23**

POMONA - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	-50	0.59146	26280	432	15288.05808	6248	138	2.2	2.50214	**4.23**
2	100	0	1.20437	26280	432	31130.55576	6248	140	2.2	5.09669	**4.23**
3	100	0	2.08811	26280	432	53973.46728	6248	142	2.3	8.83941	**4.23**
4	100	0	3.14746	26280	432	81355.54608	6248	145	2.3	13.33042	**4.24**
5	100	0	4.34608	26280	432	112337.47584	6248	147	2.4	18.41296	**4.24**
6	100	0	5.57952	26280	432	144219.43296	6248	152	2.4	23.65804	**4.24**
7	100	0	6.79151	26280	432	175546.95048	6248	154	2.5	28.80652	**4.24**
8	100	0	7.82163	26280	432	202173.49224	6248	157	2.5	33.19217	**4.24**
9	100	0	8.41525	26280	432	217517.38200	6248	159	2.5	35.72301	**4.25**
10	100	0	8.44758	26280	432	218353.04784	6248	158	2.5	35.85436	**4.24**
11	100	0	7.8987	26280	432	204165.59760	6248	157	2.5	33.51922	**4.24**
12	100	0	6.84909	26280	432	177035.27832	6248	154	2.5	29.05075	**4.24**
13	100	0	5.65066	26280	432	146058.25968	6248	149	2.4	23.94790	**4.24**
14	100	0	4.41875	26280	432	114215.85000	6248	145	2.3	18.71471	**4.24**
15	100	0	3.20379	26280	432	82811.56392	6248	142	2.3	13.56233	**4.23**
16	150	0	2.10868	26280	432	54505.16064	6248	139	2.2	8.92211	**4.23**
17	150	0	1.168	26280	432	30190.46400	6248	135	2.2	4.93873	**4.23**
18	200	0	0.48016	26280	432	12411.17568	6248	134	2.1	2.02996	**4.23**
19	500	-200	0.19471	26280	432	5032.86408	6248	133	2.1	0.82304	**4.23**
20	500	0	0.07366	26280	432	1903.96368	6248	133	2.1	0.31136	**4.23**
21	0	-50	0.04644	26280	432	1200.38112	6248	136	2.2	0.19640	**4.23**
22	0	-50	0.05041	26280	432	1302.99768	6248	135	2.2	0.21315	**4.23**
23	0	-50	0.05369	26280	432	1387.77912	6248	136	2.2	0.22706	**4.23**
24	100	-50	0.21115	26280	432	5457.80520	6248	136	2.2	0.89297	**4.23**

POMONA - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	-50	65.9476	26280	432	1704613.56480	6248	138	2.2	278.98749	**4.23**
2	50	0	58.23568	26280	432	1505275.85664	6248	140	2.2	246.44333	**4.23**
3	50	0	70.24739	26280	432	1815754.53672	6248	142	2.3	297.37218	**4.23**
4	50	0	88.80241	26280	432	2295364.69368	6248	145	2.3	376.10432	**4.24**
5	50	0	111.03137	26280	432	2869938.85176	6248	147	2.4	470.40466	**4.24**
6	50	0	135.13711	26280	432	3493024.01928	6248	152	2.4	573.00263	**4.24**
7	50	0	158.47651	26280	432	4096300.83048	6248	154	2.5	672.18589	**4.24**
8	50	0	179.27428	26280	432	4633881.58944	6248	157	2.5	760.77517	**4.24**
9	50	0	197.23857	26280	432	5098222.55736	6248	159	2.5	837.28405	**4.25**
10	50	0	218.81575	26280	432	5655949.50600	6248	158	2.5	928.72734	**4.24**
11	50	0	244.03622	26280	432	6307848.21456	6248	157	2.5	1035.60141	**4.24**
12	50	0	270.93265	26280	432	7003067.13720	6248	154	2.5	1149.17413	**4.24**
13	50	0	285.34864	26280	432	7375691.64672	6248	149	2.4	1209.32803	**4.24**
14	50	0	285.77704	26280	432	7386764.92992	6248	145	2.3	1210.34982	**4.24**
15	50	0	275.07823	26280	432	7110222.08904	6248	142	2.3	1164.46480	**4.23**
16	50	0	256.69684	26280	432	6635099.92032	6248	139	2.2	1086.11883	**4.23**
17	50	0	236.76058	26280	432	6119787.47184	6248	135	2.2	1001.11033	**4.23**
18	50	0	207.98698	26280	432	5376047.45904	6248	134	2.1	879.30119	**4.23**
19	50	0	170.7548	26280	432	4413670.07040	6248	133	2.1	721.77761	**4.23**
20	100	-50	154.35448	26280	432	3989754.59904	6248	133	2.1	652.45374	**4.23**
21	100	-50	130.80712	26280	432	3381102.43776	6248	136	2.2	553.19084	**4.23**
22	100	-50	109.58201	26280	432	2832475.79448	6248	135	2.2	463.35282	**4.23**
23	100	-50	93.63298	26280	432	2420225.26704	6248	136	2.2	395.97926	**4.23**
24	100	-50	78.6095	26280	432	2031898.35600	6248	136	2.2	332.44410	**4.23**

REDLANDS - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-500	0	0.04181	8760	478	346.27042	2088	291	13.9	0.19269	**4.61**
2	150	-100	0.08511	8760	478	704.88102	2088	250	12.0	0.38350	**4.51**
3	150	-100	0.18241	8760	478	1510.71962	2088	209	10.0	0.80400	**4.41**
4	150	-100	0.31173	8760	478	2581.74786	2088	167	8.0	1.34396	**4.31**
5	150	-100	0.45602	8760	478	3776.75764	2088	125	6.0	1.92397	**4.22**
6	200	-100	0.60555	8760	478	5015.16510	2088	84	4.0	2.50258	**4.13**
7	200	-50	0.75634	8760	478	6264.00788	2088	51	2.4	3.07511	**4.07**
8	200	-100	0.88379	8760	478	7319.54878	2088	31	1.5	3.55836	**4.03**
9	200	-50	0.9679	8760	478	8016.14780	2088	25	1.2	3.88568	**4.01**
10	250	-50	0.99231	8760	478	8218.31142	2088	20	1.0	3.97404	**4.00**
11	250	-50	0.94769	8760	478	7848.76858	2088	20	1.0	3.79534	**4.00**
12	250	-50	0.83365	8760	478	6904.28930	2088	21	1.0	3.34025	**4.01**
13	250	-50	0.69935	8760	478	5792.01670	2088	35	1.7	2.82125	**4.03**
14	300	-50	0.54905	8760	478	4547.23210	2088	53	2.5	2.23451	**4.07**
15	300	-50	0.40803	8760	478	3379.30446	2088	83	4.0	1.68544	**4.13**
16	300	-50	0.27569	8760	478	2283.26458	2088	120	5.7	1.16020	**4.21**
17	350	-50	0.15386	8760	478	1274.26852	2088	162	7.8	0.66161	**4.30**
18	400	-50	0.05645	8760	478	467.51890	2088	208	10.0	0.24868	**4.41**
19	-50	0	0.00342	8760	478	28.32444	2088	249	11.9	0.01540	**4.50**
20	-50	0	0.00391	8760	478	32.38262	2088	290	13.9	0.01801	**4.61**
21	-50	0	0.0043	8760	478	35.61260	2088	318	15.2	0.02012	**4.68**
22	-50	0	0.0046	8760	478	38.09720	2088	341	16.3	0.02181	**4.74**
23	-50	0	0.00521	8760	478	43.14922	2088	344	16.5	0.02474	**4.75**
24	-500	50	0.01975	8760	478	163.56950	2088	327	15.7	0.09288	**4.70**

REDLANDS - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	WORKER PERIOD AVE CONC	% WORKER CALM & MISSING HRS	QUOTIENT (FACTOR)
1	-50	0	0.52894	8760	478	4380.68108	2088	291	2.43777	13.9	**4.61**
2	50	-50	1.22841	8760	478	10173.69162	2088	250	5.53520	12.0	**4.51**
3	50	-50	2.14057	8760	478	17728.20074	2088	209	9.43491	10.0	**4.41**
4	50	-50	3.12441	8760	478	25876.36362	2088	167	13.47026	8.0	**4.31**
5	100	-50	4.19282	8760	478	34724.93524	2088	125	17.68973	6.0	**4.22**
6	100	-50	5.31036	8760	478	43980.40152	2088	84	21.94631	4.0	**4.13**
7	100	-50	6.45196	8760	478	53435.13272	2088	51	26.23227	2.4	**4.07**
8	100	-50	7.43242	8760	478	61555.30244	2088	31	29.92479	1.5	**4.03**
9	100	-50	7.96745	8760	478	65986.42090	2088	25	31.98566	1.2	**4.01**
10	100	-50	7.90056	8760	478	65432.43792	2088	20	31.64044	1.0	**4.00**
11	100	-50	7.20298	8760	478	59655.08036	2088	20	28.84675	1.0	**4.00**
12	100	-50	6.14084	8760	478	50858.43688	2088	21	24.60495	1.0	**4.01**
13	100	0	5.07104	8760	478	41998.35328	2088	35	20.45706	1.7	**4.03**
14	150	-50	4.07763	8760	478	33770.93166	2088	53	16.59505	2.5	**4.07**
15	150	0	3.14168	8760	478	26019.39376	2088	83	12.97725	4.0	**4.13**
16	150	0	2.23696	8760	478	18526.50272	2088	120	9.41387	5.7	**4.21**
17	150	0	1.32077	8760	478	10938.61714	2088	162	5.67945	7.8	**4.30**
18	150	0	0.517	8760	478	4281.79400	2088	208	2.27755	10.0	**4.41**
19	500	-100	0.07352	8760	478	608.89264	2088	249	0.33110	11.9	**4.50**
20	-50	0	0.04779	8760	478	395.79678	2088	290	0.22013	13.9	**4.61**
21	-50	0	0.05202	8760	478	430.82964	2088	318	0.24341	15.2	**4.68**
22	-50	0	0.05512	8760	478	456.50384	2088	341	0.26131	16.3	**4.74**
23	-50	0	0.05897	8760	478	488.38954	2088	344	0.28004	16.5	**4.75**
24	-50	0	0.18742	8760	478	1552.21244	2088	327	0.88144	15.7	**4.70**

REDLANDS - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-300	50	45.47894	8760	478	376656.58108	2088	291	13.9	209.60299	**4.61**
2	-50	0	45.80464	8760	478	379354.02848	2088	250	12.0	206.39501	**4.51**
3	-50	0	53.94402	8760	478	446764.37364	2088	209	10.0	237.76710	**4.41**
4	50	0	74.29323	8760	478	615296.53086	2088	167	8.0	320.30012	**4.31**
5	50	0	96.44381	8760	478	798747.63442	2088	125	6.0	406.90149	**4.22**
6	50	0	123.94464	8760	478	1026509.50848	2088	84	4.0	512.23029	**4.13**
7	50	0	151.19332	8760	478	1252183.07624	2088	51	2.4	614.71923	**4.07**
8	50	0	175.86202	8760	478	1456489.24964	2088	31	1.5	708.06478	**4.03**
9	50	0	200.54185	8760	478	1660887.60170	2088	25	1.2	805.08367	**4.01**
10	50	0	230.43001	8760	478	1908421.34282	2088	20	1.0	922.83431	**4.00**
11	50	0	263.81094	8760	478	2184882.20508	2088	20	1.0	1056.51944	**4.00**
12	50	0	299.22627	8760	478	2478191.96814	2088	21	1.0	1198.93177	**4.01**
13	50	0	298.91289	8760	478	2475596.55498	2088	35	1.7	1205.84343	**4.03**
14	50	0	277.77399	8760	478	2300524.18518	2088	53	2.5	1130.47872	**4.07**
15	50	0	252.24911	8760	478	2089127.12902	2088	83	4.0	1041.95867	**4.13**
16	50	0	224.21967	8760	478	1856987.30694	2088	120	5.7	943.59111	**4.21**
17	50	0	190.84881	8760	478	1580609.84442	2088	162	7.8	820.66970	**4.30**
18	50	0	147.20039	8760	478	1219113.62998	2088	208	10.0	648.46470	**4.41**
19	50	0	96.70574	8760	478	800916.93868	2088	249	11.9	435.51764	**4.50**
20	100	-50	65.67926	8760	478	543955.63132	2088	290	13.9	302.53372	**4.61**
21	100	-50	44.74535	8760	478	370580.98870	2088	318	15.2	209.36779	**4.68**
22	-300	50	46.41385	8760	478	384399.50570	2088	341	16.3	220.03406	**4.74**
23	-300	50	48.26296	8760	478	399713.83472	2088	344	16.5	229.19371	**4.75**
24	-300	50	48.06504	8760	478	398074.66128	2088	327	15.7	226.05035	**4.70**

SAN BERNARDINO - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	200	350	0.04085	26280	1292	1020.75980	6248	872	14.0	0.18987	**4.65**
2	100	200	0.09946	26280	1292	2485.30648	6248	823	13.2	0.45812	**4.61**
3	100	150	0.20057	26280	1292	5011.84316	6248	744	11.9	0.91058	**4.54**
4	100	150	0.33332	26280	1292	8329.00016	6248	636	10.2	1.48414	**4.45**
5	150	150	0.48464	26280	1292	12110.18432	6248	526	8.4	2.11643	**4.37**
6	150	150	0.64456	26280	1292	16106.26528	6248	414	6.6	2.76076	**4.28**
7	150	150	0.79252	26280	1292	19803.48976	6248	312	5.0	3.33617	**4.21**
8	150	150	0.92034	26280	1292	22997.45592	6248	206	3.3	3.80627	**4.14**
9	200	200	1.02323	26280	1292	25568.47124	6248	138	2.2	4.18469	**4.09**
10	200	200	1.0794	26280	1292	26972.04720	6248	99	1.6	4.38641	**4.06**
11	200	200	1.04725	26280	1292	26168.68300	6248	87	1.4	4.24747	**4.06**
12	200	200	0.92541	26280	1292	23124.14508	6248	91	1.5	3.75575	**4.06**
13	200	200	0.78218	26280	1292	19545.11384	6248	92	1.5	3.17497	**4.06**
14	250	250	0.6348	26280	1292	15862.38240	6248	109	1.7	2.58387	**4.07**
15	250	250	0.49254	26280	1292	12307.58952	6248	150	2.4	2.01830	**4.10**
16	250	250	0.34312	26280	1292	8573.88256	6248	208	3.3	1.41952	**4.14**
17	300	300	0.19921	26280	1292	4977.85948	6248	282	4.5	0.83437	**4.19**
18	300	300	0.08024	26280	1292	2005.03712	6248	370	5.9	0.34111	**4.25**
19	500	500	0.0042	26280	1292	104.94960	6248	461	7.4	0.01814	**4.32**
20	500	-400	0.00275	26280	1292	68.71700	6248	565	9.0	0.01209	**4.40**
21	-50	0	0.00279	26280	1292	69.71652	6248	674	10.8	0.01251	**4.48**
22	-50	0	0.00305	26280	1292	76.21340	6248	769	12.3	0.01391	**4.56**
23	500	-450	0.00363	26280	1292	90.70644	6248	830	13.3	0.01674	**4.61**
24	500	-400	0.01549	26280	1292	387.06412	6248	878	14.1	0.07208	**4.65**

SAN BERNARDINO - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	100	0.61923	26280	1292	15473.31924	6248	872	14.0	2.87822	**4.65**
2	50	50	1.30694	26280	1292	32657.81672	6248	823	13.2	6.01987	**4.61**
3	50	50	2.2765	26280	1292	56885.18200	6248	744	11.9	10.33524	**4.54**
4	50	50	3.33493	26280	1292	83333.23084	6248	636	10.2	14.84911	**4.45**
5	50	50	4.37187	26280	1292	109244.28756	6248	526	8.4	19.09198	**4.37**
6	50	50	5.37512	26280	1292	134313.49856	6248	414	6.6	23.02254	**4.28**
7	50	100	6.31892	26280	1292	157897.17296	6248	312	5.0	26.59993	**4.21**
8	100	100	7.24372	26280	1292	181006.07536	6248	206	3.3	29.95797	**4.14**
9	100	100	8.1813	26280	1292	204434.32440	6248	138	2.2	33.45897	**4.09**
10	100	100	8.82249	26280	1292	220456.38012	6248	99	1.6	35.85240	**4.06**
11	100	100	8.99277	26280	1292	224711.33676	6248	87	1.4	36.47319	**4.06**
12	100	100	8.30546	26280	1292	207536.83448	6248	91	1.5	33.70746	**4.06**
13	100	100	7.26975	26280	1292	181656.51300	6248	92	1.5	29.50886	**4.06**
14	100	100	6.13035	26280	1292	153185.18580	6248	109	1.7	24.95279	**4.07**
15	100	100	4.96832	26280	1292	124148.38016	6248	150	2.4	20.35887	**4.10**
16	100	100	3.72613	26280	1292	93108.53644	6248	208	3.3	15.41532	**4.14**
17	100	100	2.45722	26280	1292	61401.01336	6248	282	4.5	10.29182	**4.19**
18	150	150	1.45646	26280	1292	36394.02248	6248	370	5.9	6.19157	**4.25**
19	250	300	0.78676	26280	1292	19659.55888	6248	461	7.4	3.39719	**4.32**
20	400	500	0.34453	26280	1292	8609.11564	6248	565	9.0	1.51489	**4.40**
21	400	500	0.1543	26280	1292	3855.64840	6248	674	10.8	0.69172	**4.48**
22	150	-100	0.09964	26280	1292	2489.80432	6248	769	12.3	0.45443	**4.56**
23	150	-100	0.1332	26280	1292	3328.40160	6248	830	13.3	0.61432	**4.61**
24	150	-100	0.22779	26280	1292	5692.01652	6248	878	14.1	1.05997	**4.65**

SAN BERNARDINO - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	100	63.46595	26280	1292	1585887.15860	6248	872	14.0	294.99389	**4.65**
2	0	50	55.96467	26280	1292	1398445.17396	6248	823	13.2	257.77791	**4.61**
3	0	50	65.81835	26280	1292	1644668.92980	6248	744	11.9	298.81340	**4.54**
4	0	50	76.94855	26280	1292	1922790.36740	6248	636	10.2	342.62123	**4.45**
5	0	50	88.11255	26280	1292	2201756.39940	6248	526	8.4	384.78791	**4.37**
6	0	50	98.59945	26280	1292	2463803.05660	6248	414	6.6	422.31797	**4.28**
7	0	50	107.32754	26280	1292	2681900.56952	6248	312	5.0	451.80266	**4.21**
8	0	50	112.73519	26280	1292	2817026.92772	6248	206	3.3	466.24080	**4.14**
9	50	50	120.54293	26280	1292	3012126.73484	6248	138	2.2	492.98310	**4.09**
10	50	50	141.77071	26280	1292	3542566.50148	6248	99	1.6	576.12075	**4.06**
11	50	50	169.40463	26280	1292	4233082.89444	6248	87	1.4	687.07724	**4.06**
12	50	50	207.02118	26280	1292	5173045.24584	6248	91	1.5	840.18926	**4.06**
13	50	50	237.14305	26280	1292	5925730.53340	6248	92	1.5	962.59430	**4.06**
14	50	50	260.28953	26280	1292	6504114.77564	6248	109	1.7	1059.47463	**4.07**
15	50	50	274.82077	26280	1292	6867221.40076	6248	150	2.4	1126.14323	**4.10**
16	50	50	274.32052	26280	1292	6854721.15376	6248	208	3.3	1134.88761	**4.14**
17	50	50	267.24594	26280	1292	6677941.54872	6248	282	4.5	1119.33315	**4.19**
18	50	50	247.00929	26280	1292	6172268.13852	6248	370	5.9	1050.06263	**4.25**
19	50	50	216.76584	26280	1292	5416544.80992	6248	461	7.4	935.98493	**4.32**
20	50	100	173.1904	26280	1292	4327681.71520	6248	565	9.0	761.51359	**4.40**
21	50	100	149.39248	26280	1292	3733019.29024	6248	674	10.8	669.72000	**4.48**
22	50	100	121.76981	26280	1292	3042784.01228	6248	769	12.3	555.35390	**4.56**
23	50	100	100.07427	26280	1292	2500655.85876	6248	830	13.3	461.54593	**4.61**
24	50	100	79.55709	26280	1292	1987972.56492	6248	878	14.1	370.19973	**4.65**

KEARNY MESA - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	150	-150	0.08297	26304	1813	2032.01827	7830	910	11.6	0.29364	**3.54**
2	150	-100	0.15998	26304	1813	3918.07018	7830	907	11.6	0.56595	**3.54**
3	200	-100	0.26694	26304	1813	6537.62754	7830	886	11.3	0.94148	**3.53**
4	200	-100	0.38512	26304	1813	9431.97392	7830	872	11.1	1.35556	**3.52**
5	200	-100	0.50152	26304	1813	12282.72632	7830	856	10.9	1.76122	**3.51**
6	200	-100	0.61064	26304	1813	14955.18424	7830	848	10.8	2.14196	**3.51**
7	200	-100	0.69021	26304	1813	16903.93311	7830	849	10.8	2.42142	**3.51**
8	250	-100	0.73932	26304	1813	18106.68612	7830	817	10.4	2.58187	**3.49**
9	250	-100	0.75042	26304	1813	18378.53622	7830	755	9.6	2.59767	**3.46**
10	250	-100	0.72932	26304	1813	17861.77612	7830	685	8.7	2.49990	**3.43**
11	250	-100	0.68371	26304	1813	16744.74161	7830	645	8.2	2.33051	**3.41**
12	250	-100	0.60961	26304	1813	14929.95851	7830	621	7.9	2.07102	**3.40**
13	250	-100	0.50731	26304	1813	12424.52921	7830	610	7.8	1.72085	**3.39**
14	250	-100	0.38994	26304	1813	9550.02054	7830	593	7.6	1.31961	**3.38**
15	300	-150	0.27924	26304	1813	6838.86684	7830	590	7.5	0.94459	**3.38**
16	300	-150	0.16786	26304	1813	4111.05926	7830	592	7.6	0.56798	**3.38**
17	300	-150	0.07795	26304	1813	1909.07345	7830	606	7.7	0.26427	**3.39**
18	350	-200	0.02278	26304	1813	557.90498	7830	645	8.2	0.07765	**3.41**
19	0	500	0.00482	26304	1813	118.04662	7830	702	9.0	0.01656	**3.44**
20	0	500	0.00483	26304	1813	118.29153	7830	762	9.7	0.01674	**3.47**
21	0	500	0.00496	26304	1813	121.47536	7830	797	10.2	0.01727	**3.48**
22	-50	500	0.00874	26304	1813	214.05134	7830	825	10.5	0.03056	**3.50**
23	-50	500	0.02154	26304	1813	527.53614	7830	859	11.0	0.07568	**3.51**
24	0	300	0.04544	26304	1813	1112.87104	7830	898	11.5	0.16054	**3.53**

KEARNY MESA - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	50	1.35817	26304	1813	33262.94147	7830	910	11.6	4.80678	**3.54**
2	50	50	2.11813	26304	1813	51875.12183	7830	907	11.6	7.49316	**3.54**
3	50	50	2.81323	26304	1813	68898.81593	7830	886	11.3	9.92206	**3.53**
4	50	50	3.40099	26304	1813	83293.64609	7830	872	11.1	11.97092	**3.52**
5	100	-50	4.27704	26304	1813	104748.98664	7830	856	10.9	15.01993	**3.51**
6	100	-50	5.2404	26304	1813	128342.63640	7830	848	10.8	18.38193	**3.51**
7	100	-50	6.03015	26304	1813	147684.40365	7830	849	10.8	21.15519	**3.51**
8	100	-50	6.5101	26304	1813	159438.85910	7830	817	10.4	22.73476	**3.49**
9	100	-50	6.57622	26304	1813	161058.20402	7830	755	9.6	22.76441	**3.46**
10	100	-50	6.3076	26304	1813	154479.43160	7830	685	8.7	21.62063	**3.43**
11	100	-50	5.84464	26304	1813	143141.07824	7830	645	8.2	19.92221	**3.41**
12	100	-50	5.22149	26304	1813	127879.51159	7830	621	7.9	17.73887	**3.40**
13	100	-50	4.43399	26304	1813	108592.84909	7830	610	7.8	15.04056	**3.39**
14	100	-50	3.50471	26304	1813	85833.85261	7830	593	7.6	11.86042	**3.38**
15	100	-50	2.50936	26304	1813	61456.73576	7830	590	7.5	8.48850	**3.38**
16	100	-50	1.54547	26304	1813	37850.10577	7830	592	7.6	5.22936	**3.38**
17	150	-50	0.78926	26304	1813	19329.76666	7830	606	7.7	2.67577	**3.39**
18	200	-100	0.30774	26304	1813	7536.86034	7830	645	8.2	1.04897	**3.41**
19	0	150	0.18342	26304	1813	4492.13922	7830	702	9.0	0.63021	**3.44**
20	0	150	0.16993	26304	1813	4161.75563	7830	762	9.7	0.58882	**3.47**
21	0	150	0.16545	26304	1813	4052.03595	7830	797	10.2	0.57615	**3.48**
22	0	150	0.21125	26304	1813	5173.72375	7830	825	10.5	0.73858	**3.50**
23	0	100	0.41536	26304	1813	10172.58176	7830	859	11.0	1.45927	**3.51**
24	0	100	0.83705	26304	1813	20500.19155	7830	898	11.5	2.95733	**3.53**

KEARNY MESA - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	50	68.76835	26304	1813	1684205.65985	7830	910	11.6	243.38232	**3.54**
2	0	50	74.07187	26304	1813	1814094.16817	7830	907	11.6	262.03874	**3.54**
3	0	50	78.4778	26304	1813	1921999.79980	7830	886	11.3	276.78569	**3.53**
4	50	0	81.98311	26304	1813	2007848.34701	7830	872	11.1	288.56688	**3.52**
5	50	0	99.45639	26304	1813	2435786.44749	7830	856	10.9	349.26677	**3.51**
6	50	0	117.63254	26304	1813	2880938.53714	7830	848	10.8	412.62368	**3.51**
7	50	0	134.71148	26304	1813	3299218.85668	7830	849	10.8	472.59975	**3.51**
8	50	0	151.26253	26304	1813	3704570.62223	7830	817	10.4	528.24335	**3.49**
9	50	0	164.57775	26304	1813	4030673.67525	7830	755	9.6	569.70653	**3.46**
10	50	0	175.05832	26304	1813	4287353.31512	7830	685	8.7	600.04945	**3.43**
11	50	0	176.15086	26304	1813	4314110.71226	7830	645	8.2	600.43295	**3.41**
12	50	0	169.94269	26304	1813	4162066.42079	7830	621	7.9	577.34310	**3.40**
13	50	0	158.91434	26304	1813	3891971.10094	7830	610	7.8	539.05417	**3.39**
14	50	0	144.4592	26304	1813	3537950.26720	7830	593	7.6	488.86973	**3.38**
15	50	-50	129.79889	26304	1813	3178904.61499	7830	590	7.5	439.07522	**3.38**
16	50	-50	127.14583	26304	1813	3113928.52253	7830	592	7.6	430.21947	**3.38**
17	50	-50	122.72119	26304	1813	3005564.66429	7830	606	7.7	416.05269	**3.39**
18	50	-50	111.89165	26304	1813	2740338.40015	7830	645	8.2	381.39713	**3.41**
19	50	-50	97.37192	26304	1813	2384735.69272	7830	702	9.0	334.55888	**3.44**
20	50	-50	76.25987	26304	1813	1867680.47617	7830	762	9.7	264.24455	**3.47**
21	0	50	59.92054	26304	1813	1467513.94514	7830	797	10.2	208.66116	**3.48**
22	0	50	56.81233	26304	1813	1391390.77403	7830	825	10.5	198.62823	**3.50**
23	0	50	58.33987	26304	1813	1428801.75617	7830	859	11.0	204.96367	**3.51**
24	0	50	63.14546	26304	1813	1546495.46086	7830	898	11.5	223.09513	**3.53**

PALOMAR - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	150	50	0.11461	26304	2291	2752.12993	7820	1313	16.8	0.42295	**3.69**
2	150	50	0.21952	26304	2291	5271.33376	7820	1235	15.8	0.80051	**3.65**
3	200	50	0.34291	26304	2291	8234.29783	7820	1156	14.8	1.23564	**3.60**
4	200	50	0.47006	26304	2291	11287.55078	7820	1071	13.7	1.67248	**3.56**
5	200	0	0.59099	26304	2291	14191.44287	7820	985	12.6	2.07629	**3.51**
6	200	0	0.70014	26304	2291	16812.46182	7820	902	11.5	2.43025	**3.47**
7	250	0	0.78328	26304	2291	18808.90264	7820	951	12.2	2.73823	**3.50**
8	250	0	0.83593	26304	2291	20073.18709	7820	858	11.0	2.88325	**3.45**
9	250	0	0.84409	26304	2291	20269.13317	7820	757	9.7	2.86976	**3.40**
10	250	0	0.8161	26304	2291	19597.00930	7820	663	8.5	2.73816	**3.36**
11	250	0	0.75885	26304	2291	18222.26505	7820	623	8.0	2.53193	**3.34**
12	250	0	0.66899	26304	2291	16064.45687	7820	623	8.0	2.23210	**3.34**
13	250	0	0.54882	26304	2291	13178.81466	7820	656	8.4	1.83959	**3.35**
14	250	0	0.41206	26304	2291	9894.79678	7820	710	9.1	1.39167	**3.38**
15	300	0	0.27978	26304	2291	6718.35714	7820	766	9.8	0.95242	**3.40**
16	300	-50	0.16245	26304	2291	3900.91185	7820	842	10.8	0.55903	**3.44**
17	300	-100	0.08094	26304	2291	1943.61222	7820	779	10.0	0.27604	**3.41**
18	300	-150	0.02496	26304	2291	599.36448	7820	876	11.2	0.08631	**3.46**
19	-450	-200	0.00494	26304	2291	118.62422	7820	978	12.5	0.01734	**3.51**
20	-400	-150	0.00466	26304	2291	111.90058	7820	1085	13.9	0.01661	**3.57**
21	-400	-200	0.00408	26304	2291	97.97304	7820	1179	15.1	0.01475	**3.62**
22	-500	-250	0.00734	26304	2291	176.25542	7820	1254	16.0	0.02684	**3.66**
23	-50	250	0.01896	26304	2291	455.28648	7820	1312	16.8	0.06996	**3.69**
24	100	150	0.05053	26304	2291	1213.37689	7820	1336	17.1	0.18713	**3.70**

PALOMAR - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	50	1.79401	26304	2291	43079.56213	7820	1313	16.8	6.62050	**3.69**
2	50	50	2.7745	26304	2291	66624.06850	7820	1235	15.8	10.11755	**3.65**
3	100	0	4.02097	26304	2291	96555.55261	7820	1156	14.8	14.48913	**3.60**
4	100	0	5.71297	26304	2291	137185.54861	7820	1071	13.7	20.32680	**3.56**
5	100	0	7.47105	26304	2291	179402.32365	7820	985	12.6	26.24760	**3.51**
6	100	0	9.08402	26304	2291	218134.57226	7820	902	11.5	31.53145	**3.47**
7	100	0	10.25315	26304	2291	246208.89095	7820	951	12.2	35.84348	**3.50**
8	100	0	10.98429	26304	2291	263765.75577	7820	858	11.0	37.88649	**3.45**
9	100	0	11.11226	26304	2291	266838.69938	7820	757	9.7	37.77980	**3.40**
10	100	0	10.70486	26304	2291	257055.80318	7820	663	8.5	35.91670	**3.36**
11	100	0	9.8762	26304	2291	237157.19060	7820	623	8.0	32.95223	**3.34**
12	100	0	8.79903	26304	2291	211291.10739	7820	623	8.0	29.35822	**3.34**
13	100	0	7.34081	26304	2291	176274.87053	7820	656	8.4	24.60565	**3.35**
14	100	0	5.64239	26304	2291	135490.71107	7820	710	9.1	19.05636	**3.38**
15	100	0	3.89019	26304	2291	93415.13247	7820	766	9.8	13.24286	**3.40**
16	100	0	2.28302	26304	2291	54822.15926	7820	842	10.8	7.85643	**3.44**
17	150	0	1.19218	26304	2291	28627.81834	7820	779	10.0	4.06587	**3.41**
18	150	0	0.42743	26304	2291	10263.87659	7820	876	11.2	1.47809	**3.46**
19	500	100	0.13519	26304	2291	3246.31747	7820	978	12.5	0.47447	**3.51**
20	-100	-50	0.11603	26304	2291	2786.22839	7820	1085	13.9	0.41369	**3.57**
21	-100	-50	0.1019	26304	2291	2446.92470	7820	1179	15.1	0.36846	**3.62**
22	-100	0	0.13253	26304	2291	3182.44289	7820	1254	16.0	0.48469	**3.66**
23	-50	50	0.32155	26304	2291	7721.38015	7820	1312	16.8	1.18644	**3.69**
24	50	50	0.91054	26304	2291	21864.79702	7820	1336	17.1	3.37212	**3.70**

PALOMAR - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	0	64.60191	26304	2291	1551285.66483	7820	1313	16.8	238.40259	**3.69**
2	50	0	67.16566	26304	2291	1612848.99358	7820	1235	15.8	244.92771	**3.65**
3	50	0	86.7754	26304	2291	2083737.68020	7820	1156	14.8	312.68573	**3.60**
4	50	0	111.35187	26304	2291	2673892.45431	7820	1071	13.7	396.19091	**3.56**
5	50	0	139.09175	26304	2291	3340010.19275	7820	985	12.6	488.66279	**3.51**
6	50	0	167.58523	26304	2291	4024224.12799	7820	902	11.5	581.70340	**3.47**
7	50	0	194.22411	26304	2291	4663903.55343	7820	951	12.2	678.97853	**3.50**
8	50	0	224.85236	26304	2291	5399379.72068	7820	858	11.0	775.55009	**3.45**
9	50	0	252.42285	26304	2291	6061429.89705	7820	757	9.7	858.19480	**3.40**
10	50	0	275.34655	26304	2291	6611896.70515	7820	663	8.5	923.83634	**3.36**
11	50	0	282.82242	26304	2291	6791414.77146	7820	623	8.0	943.64524	**3.34**
12	50	0	277.9957	26304	2291	6675510.74410	7820	623	8.0	927.54075	**3.34**
13	50	0	262.24815	26304	2291	6297364.82595	7820	656	8.4	879.02915	**3.35**
14	50	0	239.25516	26304	2291	5745234.15708	7820	710	9.1	808.04981	**3.38**
15	50	0	213.26193	26304	2291	5121058.72509	7820	766	9.8	725.97941	**3.40**
16	50	0	185.3631	26304	2291	4451124.12030	7820	842	10.8	637.87964	**3.44**
17	50	0	158.33517	26304	2291	3802102.43721	7820	779	10.0	539.99467	**3.41**
18	50	0	125.85979	26304	2291	3022271.13727	7820	876	11.2	435.23490	**3.46**
19	50	0	93.2437	26304	2291	2239060.96810	7820	978	12.5	327.25241	**3.51**
20	50	0	62.12509	26304	2291	1491809.78617	7820	1085	13.9	221.50108	**3.57**
21	-50	0	47.17899	26304	2291	1132909.08687	7820	1179	15.1	170.59315	**3.62**
22	-50	0	51.9114	26304	2291	1246548.44820	7820	1254	16.0	189.84899	**3.66**
23	-50	0	57.95502	26304	2291	1391673.89526	7820	1312	16.8	213.84049	**3.69**
24	-50	0	62.2143	26304	2291	1493951.98590	7820	1336	17.1	230.40592	**3.70**

POMONA - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	0	1.67498	26280	432	43294.88304	7810	175	2.2	5.67058	**3.39**
2	100	0	2.52254	26280	432	65202.61392	7810	179	2.3	8.54444	**3.39**
3	100	0	3.48087	26280	432	89973.52776	7810	183	2.3	11.79671	**3.39**
4	100	0	4.46874	26280	432	115507.99152	7810	188	2.4	15.15455	**3.39**
5	100	0	5.44049	26280	432	140625.78552	7810	189	2.4	18.45241	**3.39**
6	100	0	6.37933	26280	432	164892.92184	7810	192	2.5	21.64517	**3.39**
7	100	0	7.16963	26280	432	185320.59624	7810	193	2.5	24.32987	**3.39**
8	100	0	7.58985	26280	432	196182.44280	7810	193	2.5	25.75587	**3.39**
9	100	0	7.54073	26280	432	194912.78904	7810	194	2.5	25.59254	**3.39**
10	100	0	7.03831	26280	432	181926.23688	7810	193	2.5	23.88424	**3.39**
11	100	0	6.33091	26280	432	163641.36168	7810	190	2.4	21.47524	**3.39**
12	100	0	5.48577	26280	432	141796.18296	7810	188	2.4	18.60354	**3.39**
13	100	0	4.52666	26280	432	117005.10768	7810	184	2.4	15.34292	**3.39**
14	100	0	3.53869	26280	432	91468.05912	7810	179	2.3	11.98638	**3.39**
15	100	0	2.56683	26280	432	66347.42184	7810	174	2.2	8.68877	**3.39**
16	150	0	1.68973	26280	432	43676.14104	7810	170	2.2	5.71677	**3.38**
17	150	0	0.93943	26280	432	24282.38664	7810	168	2.2	3.17749	**3.38**
18	200	0	0.38972	26280	432	10073.48256	7810	168	2.2	1.31817	**3.38**
19	500	-200	0.15933	26280	432	4118.36184	7810	169	2.2	0.53898	**3.38**
20	500	0	0.06427	26280	432	1661.25096	7810	169	2.2	0.21741	**3.38**
21	0	-50	0.04922	26280	432	1272.23856	7810	171	2.2	0.16655	**3.38**
22	100	-50	0.17372	26280	432	4490.31456	7810	170	2.2	0.58774	**3.38**
23	100	-50	0.47768	26280	432	12347.07264	7810	170	2.2	1.61611	**3.38**
24	100	0	0.96732	26280	432	25003.28736	7810	171	2.2	3.27311	**3.38**

POMONA - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	200	-50	0.14539	26280	432	3758.04072	7810	175	2.2	0.49221	**3.39**
2	200	-50	0.24454	26280	432	6320.86992	7810	179	2.3	0.82831	**3.39**
3	200	-50	0.35936	26280	432	9288.73728	7810	183	2.3	1.21788	**3.39**
4	200	0	0.475	26280	432	12277.80000	7810	188	2.4	1.61084	**3.39**
5	200	0	0.58245	26280	432	15055.16760	7810	189	2.4	1.97548	**3.39**
6	250	0	0.68649	26280	432	17744.39352	7810	192	2.5	2.32927	**3.39**
7	250	0	0.77125	26280	432	19935.27000	7810	193	2.5	2.61721	**3.39**
8	250	0	0.81936	26280	432	21178.81728	7810	193	2.5	2.78047	**3.39**
9	250	0	0.82376	26280	432	21292.54848	7810	194	2.5	2.79577	**3.39**
10	250	0	0.78241	26280	432	20223.73368	7810	193	2.5	2.65508	**3.39**
11	250	0	0.7142	26280	432	18460.64160	7810	190	2.4	2.42266	**3.39**
12	250	0	0.62035	26280	432	16034.80680	7810	188	2.4	2.10375	**3.39**
13	300	0	0.50729	26280	432	13112.43192	7810	184	2.4	1.71944	**3.39**
14	300	0	0.39583	26280	432	10231.41384	7810	179	2.3	1.34077	**3.39**
15	300	50	0.28793	26280	432	7442.41464	7810	174	2.2	0.97465	**3.39**
16	350	50	0.18215	26280	432	4708.21320	7810	170	2.2	0.61626	**3.38**
17	350	50	0.09308	26280	432	2405.93184	7810	168	2.2	0.31483	**3.38**
18	400	0	0.03142	26280	432	812.14416	7810	168	2.2	0.10627	**3.38**
19	0	-50	0.00464	26280	432	119.93472	7810	169	2.2	0.01570	**3.38**
20	0	-50	0.00508	26280	432	131.30784	7810	169	2.2	0.01718	**3.38**
21	0	-50	0.00569	26280	432	147.07512	7810	171	2.2	0.01925	**3.38**
22	500	-250	0.01302	26280	432	336.54096	7810	170	2.2	0.04405	**3.38**
23	300	-100	0.0304	26280	432	785.77920	7810	170	2.2	0.10285	**3.38**
24	200	-50	0.07176	26280	432	1854.85248	7810	171	2.2	0.24281	**3.38**

POMONA - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKE HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	0	66.88293	26280	432	1728789.97464	7810	175	2.2	226.42960	**3.39**
2	50	0	78.93616	26280	432	2040341.86368	7810	179	2.3	267.37542	**3.39**
3	50	0	94.94525	26280	432	2454144.82200	7810	183	2.3	321.77066	**3.39**
4	50	0	113.62804	26280	432	2937057.57792	7810	188	2.4	385.33949	**3.39**
5	50	0	133.76259	26280	432	3457495.42632	7810	189	2.4	453.68002	**3.39**
6	50	0	155.21512	26280	432	4012000.42176	7810	192	2.5	526.64747	**3.39**
7	50	0	174.83572	26280	432	4519153.69056	7810	193	2.5	593.29837	**3.39**
8	50	0	196.43289	26280	432	5077397.34072	7810	193	2.5	666.58755	**3.39**
9	50	0	221.2805	26280	432	5719658.36400	7810	194	2.5	751.00556	**3.39**
10	50	0	249.09373	26280	432	6438574.73304	7810	193	2.5	845.29011	**3.39**
11	50	0	267.02625	26280	432	6902094.51000	7810	190	2.4	905.78668	**3.39**
12	50	0	271.20773	26280	432	7010177.40504	7810	188	2.4	919.72939	**3.39**
13	50	0	265.00007	26280	432	6849721.80936	7810	184	2.4	898.20637	**3.39**
14	50	0	252.4629	26280	432	6525661.03920	7810	179	2.3	855.15149	**3.39**
15	50	0	237.46298	26280	432	6137943.10704	7810	174	2.2	803.81654	**3.39**
16	50	0	219.40304	26280	432	5671129.77792	7810	170	2.2	742.29447	**3.38**
17	50	0	200.09348	26280	432	5172016.27104	7810	168	2.2	676.78831	**3.38**
18	50	0	174.28381	26280	432	4504887.92088	7810	168	2.2	589.49070	**3.38**
19	100	-50	148.72624	26280	432	3844275.85152	7810	169	2.2	503.11162	**3.38**
20	100	-50	136.06151	26280	432	3516917.91048	7810	169	2.2	460.26932	**3.38**
21	100	-50	116.42089	26280	432	3009247.16472	7810	171	2.2	393.93208	**3.38**
22	100	-50	95.89973	26280	432	2478816.22104	7810	170	2.2	324.45238	**3.38**
23	100	-50	79.98215	26280	432	2067378.61320	7810	170	2.2	270.59929	**3.38**
24	100	-50	67.81091	26280	432	1752776.40168	7810	171	2.2	229.45103	**3.38**

REDLANDS - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	150	-100	0.14613	8760	478	1210.24866	2610	303	11.6	0.52460	**3.59**
2	150	-100	0.24958	8760	478	2067.02156	2610	258	9.9	0.87884	**3.52**
3	150	-100	0.36502	8760	478	3023.09564	2610	216	8.3	1.26278	**3.46**
4	200	-100	0.4846	8760	478	4013.45720	2610	172	6.6	1.64621	**3.40**
5	200	-50	0.6053	8760	478	5013.09460	2610	128	4.9	2.01978	**3.34**
6	200	-100	0.71152	8760	478	5892.80864	2610	86	3.3	2.33471	**3.28**
7	200	-50	0.79696	8760	478	6600.42272	2610	54	2.1	2.58233	**3.24**
8	250	-50	0.85358	8760	478	7069.34956	2610	36	1.4	2.74645	**3.22**
9	250	-50	0.87022	8760	478	7207.16204	2610	32	1.2	2.79564	**3.21**
10	250	-50	0.82892	8760	478	6865.11544	2610	29	1.1	2.65987	**3.21**
11	250	-50	0.75826	8760	478	6279.90932	2610	42	1.6	2.44545	**3.23**
12	250	-50	0.66701	8760	478	5524.17682	2610	58	2.2	2.16465	**3.25**
13	250	-50	0.55959	8760	478	4634.52438	2610	86	3.3	1.83618	**3.28**
14	300	-50	0.43933	8760	478	3638.53106	2610	122	4.7	1.46243	**3.33**
15	300	-50	0.32652	8760	478	2704.23864	2610	165	6.3	1.10603	**3.39**
16	300	-50	0.22066	8760	478	1827.50612	2610	213	8.2	0.76241	**3.46**
17	350	-50	0.12319	8760	478	1020.25958	2610	256	9.8	0.43342	**3.52**
18	400	-50	0.04524	8760	478	374.67768	2610	299	11.5	0.16213	**3.58**
19	-50	0	0.0038	8760	478	31.47160	2610	340	13.0	0.01386	**3.65**
20	-50	0	0.00417	8760	478	34.53594	2610	378	14.5	0.01547	**3.71**
21	-50	0	0.00479	8760	478	39.67078	2610	395	15.1	0.01791	**3.74**
22	-500	50	0.01591	8760	478	131.76662	2610	396	15.2	0.05952	**3.74**
23	-500	0	0.03356	8760	478	277.94392	2610	373	14.3	0.12425	**3.70**
24	150	-100	0.06827	8760	478	565.41214	2610	343	13.1	0.24941	**3.65**

REDLANDS - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	-50	1.71658	8760	478	14216.71556	2610	303	11.6	6.16243	**3.59**
2	50	-50	2.50366	8760	478	20735.31212	2610	258	9.9	8.81603	**3.52**
3	100	-50	3.35706	8760	478	27803.17092	2610	216	8.3	11.61369	**3.46**
4	100	-50	4.25095	8760	478	35206.36790	2610	172	6.6	14.44068	**3.40**
5	100	-50	5.1653	8760	478	42779.01460	2610	128	4.9	17.23570	**3.34**
6	100	-50	6.01292	8760	478	49799.00344	2610	86	3.3	19.73019	**3.28**
7	100	-50	6.72041	8760	478	55658.43562	2610	54	2.1	21.77560	**3.24**
8	100	-50	7.11772	8760	478	58948.95704	2610	36	1.4	22.90169	**3.22**
9	100	-50	7.01506	8760	478	58098.72692	2610	32	1.2	22.53636	**3.21**
10	100	-50	6.50262	8760	478	53854.69884	2610	29	1.1	20.86583	**3.21**
11	100	-50	5.76643	8760	478	47757.57326	2610	42	1.6	18.59719	**3.23**
12	100	-50	4.91534	8760	478	40708.84588	2610	58	2.2	15.95174	**3.25**
13	100	0	4.05934	8760	478	33619.45388	2610	86	3.3	13.31991	**3.28**
14	150	-50	3.26436	8760	478	27035.42952	2610	122	4.7	10.86633	**3.33**
15	150	0	2.51516	8760	478	20830.55512	2610	165	6.3	8.51965	**3.39**
16	150	0	1.79145	8760	478	14836.78890	2610	213	8.2	6.18973	**3.46**
17	150	0	1.05852	8760	478	8766.66264	2610	256	9.8	3.72416	**3.52**
18	150	0	0.41545	8760	478	3440.75690	2610	299	11.5	1.48886	**3.58**
19	500	-100	0.05953	8760	478	493.02746	2610	340	13.0	0.21719	**3.65**
20	-50	0	0.05022	8760	478	415.92204	2610	378	14.5	0.18635	**3.71**
21	-50	0	0.05482	8760	478	454.01924	2610	395	15.1	0.20497	**3.74**
22	-50	0	0.15882	8760	478	1315.34724	2610	396	15.2	0.59410	**3.74**
23	-50	0	0.43321	8760	478	3587.84522	2610	373	14.3	1.60386	**3.70**
24	50	-50	0.98664	8760	478	8171.35248	2610	343	13.1	3.60448	**3.65**

REDLANDS - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	0	45.3508	8760	478	375595.32560	2610	303	11.6	162.80682	**3.59**
2	50	0	60.52773	8760	478	501290.65986	2610	258	9.9	213.13378	**3.52**
3	50	0	78.2791	8760	478	648307.50620	2610	216	8.3	270.80514	**3.46**
4	50	0	100.35242	8760	478	831118.74244	2610	172	6.6	340.90186	**3.40**
5	50	0	123.30279	8760	478	1021193.70678	2610	128	4.9	411.43985	**3.34**
6	50	0	147.25117	8760	478	1219534.18994	2610	86	3.3	483.17519	**3.28**
7	50	0	173.53484	8760	478	1437215.54488	2610	54	2.1	562.29090	**3.24**
8	50	0	204.41071	8760	478	1692929.50022	2610	36	1.4	657.70377	**3.22**
9	50	0	237.08429	8760	478	1963532.08978	2610	32	1.2	761.64938	**3.21**
10	50	0	270.99063	8760	478	2244344.39766	2610	29	1.1	869.56389	**3.21**
11	50	0	274.80034	8760	478	2275896.41588	2610	42	1.6	886.25250	**3.23**
12	50	0	263.13703	8760	478	2179300.88246	2610	58	2.2	853.95803	**3.25**
13	50	0	247.94703	8760	478	2053497.30246	2610	86	3.3	813.58847	**3.28**
14	50	0	227.47119	8760	478	1883916.39558	2610	122	4.7	757.20112	**3.33**
15	50	0	205.25923	8760	478	1699956.94286	2610	165	6.3	695.27891	**3.39**
16	50	0	181.48141	8760	478	1503029.03762	2610	213	8.2	627.04591	**3.46**
17	50	0	154.0154	8760	478	1275555.54280	2610	256	9.8	541.86727	**3.52**
18	50	0	118.85346	8760	478	984344.35572	2610	299	11.5	425.93871	**3.58**
19	50	0	78.48865	8760	478	650042.99930	2610	340	13.0	286.36255	**3.65**
20	100	-50	55.02469	8760	478	455714.48258	2610	378	14.5	204.17316	**3.71**
21	-300	50	46.19985	8760	478	382627.15770	2610	395	15.1	172.74364	**3.74**
22	-300	50	45.56241	8760	478	377347.87962	2610	396	15.2	170.43716	**3.74**
23	-300	50	43.32203	8760	478	358793.05246	2610	373	14.3	160.39028	**3.70**
24	-300	50	40.49639	8760	478	335391.10198	2610	343	13.1	147.94491	**3.65**

SAN BERNARDINO - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	150	0.16062	26280	2291	3853.11318	7810	945	12.1	0.56127	**3.49**
2	100	150	0.26681	26280	2291	6400.50509	7810	857	11.0	0.92054	**3.45**
3	150	150	0.38784	26280	2291	9303.89376	7810	768	9.8	1.32120	**3.41**
4	150	150	0.51578	26280	2291	12373.04642	7810	659	8.4	1.73025	**3.35**
5	150	150	0.63431	26280	2291	15216.46259	7810	547	7.0	2.09507	**3.30**
6	150	150	0.74255	26280	2291	17813.03195	7810	433	5.5	2.41467	**3.25**
7	200	200	0.84805	26280	2291	20343.87145	7810	332	4.3	2.72050	**3.21**
8	200	200	0.92818	26280	2291	22266.11002	7810	229	2.9	2.93709	**3.16**
9	200	200	0.95389	26280	2291	22882.86721	7810	160	2.0	2.99122	**3.14**
10	200	200	0.91165	26280	2291	21869.57185	7810	125	1.6	2.84575	**3.12**
11	200	200	0.83833	26280	2291	20110.69837	7810	116	1.5	2.61382	**3.12**
12	200	200	0.74042	26280	2291	17761.93538	7810	132	1.7	2.31335	**3.12**
13	200	200	0.6259	26280	2291	15014.71510	7810	171	2.2	1.96553	**3.14**
14	250	250	0.50812	26280	2291	12189.29068	7810	227	2.9	1.60745	**3.16**
15	250	250	0.39411	26280	2291	9454.30479	7810	302	3.9	1.25923	**3.20**
16	250	250	0.27457	26280	2291	6586.65973	7810	393	5.0	0.88805	**3.23**
17	300	300	0.15944	26280	2291	3824.80616	7810	483	6.2	0.52202	**3.27**
18	300	300	0.06426	26280	2291	1541.53314	7810	591	7.6	0.21354	**3.32**
19	500	500	0.00341	26280	2291	81.80249	7810	703	9.0	0.01151	**3.38**
20	-50	0	0.00273	26280	2291	65.48997	7810	810	10.4	0.00936	**3.43**
21	500	-400	0.00355	26280	2291	85.16095	7810	909	11.6	0.01234	**3.48**
22	500	-400	0.01276	26280	2291	306.09964	7810	996	12.8	0.04492	**3.52**
23	200	350	0.03276	26280	2291	785.87964	7810	1024	13.1	0.11581	**3.54**
24	100	200	0.07971	26280	2291	1912.16319	7810	1008	12.9	0.28112	**3.53**

SAN BERNARDINO - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	50	1.82487	26280	2291	43776.80643	7810	945	12.1	6.37681	**3.49**
2	50	50	2.67182	26280	2291	64094.28998	7810	857	11.0	9.21822	**3.45**
3	50	50	3.50148	26280	2291	83997.00372	7810	768	9.8	11.92800	**3.41**
4	50	50	4.30472	26280	2291	103265.92808	7810	659	8.4	14.44077	**3.35**
5	50	100	5.06866	26280	2291	121592.08474	7810	547	7.0	16.74130	**3.30**
6	100	100	5.91978	26280	2291	142009.60242	7810	433	5.5	19.25032	**3.25**
7	100	100	6.91876	26280	2291	165974.13364	7810	332	4.3	22.19499	**3.21**
8	100	100	7.75458	26280	2291	186024.61962	7810	229	2.9	24.53827	**3.16**
9	100	100	8.257	26280	2291	198077.17300	7810	160	2.0	25.89244	**3.14**
10	100	100	8.0456	26280	2291	193005.89840	7810	125	1.6	25.11463	**3.12**
11	100	100	7.431	26280	2291	178262.25900	7810	116	1.5	23.16900	**3.12**
12	100	100	6.66787	26280	2291	159955.53343	7810	132	1.7	20.83297	**3.12**
13	100	100	5.82847	26280	2291	139819.16683	7810	171	2.2	18.30333	**3.14**
14	100	100	4.91446	26280	2291	117892.98094	7810	227	2.9	15.54701	**3.16**
15	100	100	3.97902	26280	2291	95452.71078	7810	302	3.9	12.71347	**3.20**
16	100	100	2.9845	26280	2291	71595.17050	7810	393	5.0	9.65285	**3.23**
17	100	100	1.96987	26280	2291	47255.21143	7810	483	6.2	6.44946	**3.27**
18	150	150	1.16932	26280	2291	28050.81748	7810	591	7.6	3.88569	**3.32**
19	250	300	0.63256	26280	2291	15174.48184	7810	703	9.0	2.13515	**3.38**
20	400	500	0.28079	26280	2291	6735.87131	7810	810	10.4	0.96227	**3.43**
21	400	500	0.14007	26280	2291	3360.13923	7810	909	11.6	0.48691	**3.48**
22	150	-100	0.19283	26280	2291	4625.79887	7810	996	12.8	0.67887	**3.52**
23	50	100	0.50387	26280	2291	12087.33743	7810	1024	13.1	1.78122	**3.54**
24	50	50	1.0492	26280	2291	25169.25880	7810	1008	12.9	3.70027	**3.53**

SAN BERNARDINO - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	50	60.43292	26280	2291	1449725.31788	7810	945	12.1	211.17630	**3.49**
2	0	50	69.41259	26280	2291	1665138.62151	7810	857	11.0	239.48492	**3.45**
3	0	50	77.69048	26280	2291	1863716.92472	7810	768	9.8	264.65733	**3.41**
4	0	50	85.534	26280	2291	2051875.12600	7810	659	8.4	286.93541	**3.35**
5	0	50	93.35436	26280	2291	2239477.74204	7810	547	7.0	308.34060	**3.30**
6	0	50	100.18756	26280	2291	2403399.37684	7810	433	5.5	325.79631	**3.25**
7	50	50	106.42361	26280	2291	2552995.98029	7810	332	4.3	341.40091	**3.21**
8	50	50	125.22838	26280	2291	3004103.60782	7810	229	2.9	396.26746	**3.16**
9	50	50	150.67387	26280	2291	3614515.46743	7810	160	2.0	472.48568	**3.14**
10	50	50	184.43774	26280	2291	4424476.94486	7810	125	1.6	575.72895	**3.12**
11	50	50	211.62126	26280	2291	5076582.40614	7810	116	1.5	659.81055	**3.12**
12	50	50	232.56731	26280	2291	5579057.19959	7810	132	1.7	726.62897	**3.12**
13	50	50	246.19103	26280	2291	5905876.61867	7810	171	2.2	773.12169	**3.14**
14	50	50	248.55743	26280	2291	5962644.18827	7810	227	2.9	786.31731	**3.16**
15	50	50	246.83969	26280	2291	5921437.32341	7810	302	3.9	788.68371	**3.20**
16	50	50	238.7665	26280	2291	5727769.56850	7810	393	5.0	772.24883	**3.23**
17	50	50	227.65219	26280	2291	5461148.38591	7810	483	6.2	745.34576	**3.27**
18	50	50	209.04015	26280	2291	5014664.15835	7810	591	7.6	694.64803	**3.32**
19	50	50	182.12183	26280	2291	4368920.57987	7810	703	9.0	614.73485	**3.38**
20	50	100	150.39433	26280	2291	3607809.58237	7810	810	10.4	515.40137	**3.43**
21	50	100	130.14718	26280	2291	3122100.70102	7810	909	11.6	452.41280	**3.48**
22	50	100	105.33813	26280	2291	2526956.40057	7810	996	12.8	370.84773	**3.52**
23	50	100	85.36188	26280	2291	2047746.13932	7810	1024	13.1	301.76041	**3.54**
24	50	100	68.96638	26280	2291	1654434.48982	7810	1008	12.9	243.22765	**3.53**

EXHIBIT 5

Technical Support Document for Cancer Potency Factors:

Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures.

May 2009

California Environmental Protection Agency

Office of Environmental Health Hazard Assessment

Air Toxicology and Epidemiology Branch

Prepared by:

John D. Budroe, Ph.D.

Joseph P. Brown, Ph.D.

James F. Collins, Ph.D.

Melanie A. Marty, Ph.D.

Andrew G. Salmon, M.A., D.Phil.

Air Toxicology and Epidemiology Branch

and

Martha S. Sandy, Ph.D., M.P.H.

Claire D. Sherman, Ph.D.

Rajpal S. Tomar, Ph.D.

Lauren Zeise, Ph.D.

Reproductive and Cancer Hazard Assessment Branch,

Office of Environmental Health Hazard Assessment

Reviewed By

George V. Alexeeff, Ph.D., Deputy Director

Melanie A. Marty, Ph.D., Chief, Air Toxicology and Epidemiology Branch

Lauren Zeise, Ph.D., Chief, Reproductive and Cancer Hazard Assessment Branch

EXECUTIVE SUMMARY

The Air Toxics "Hot Spots" Information and Assessment Act (AB 2588, Connelly) was enacted in September 1987. Under this Act, stationary sources of air pollution are required to report the types and quantities of certain substances their facilities routinely release into the air. The goals of the Air Toxics "Hot Spots" Act are to collect emission data, identify facilities having localized impacts, ascertain health risks posed by those facilities, notify nearby residents of significant risks and reduce emissions from significant sources.

The Technical Support Document for Cancer Potency Factors (TSD) contains cancer unit risks and potency factors for 107 of the 201 carcinogenic substances or groups of substances for which emissions must be quantified in the Air Toxics Hot Spots program. These unit risks are used in the cancer risk assessment of facility emissions.

The purpose of this revision to the TSD is to provide updated calculation procedures used to derive the estimated unit risk and cancer potency factors, and to describe the procedures used to consider the increased susceptibility of infants and children compared to adults to carcinogens. This updates cancer risk assessment methods originally laid out in the California Department of Health Services' Guidelines for Chemical Carcinogen Risk Assessment (CDHS, 1985), and more recently summarized in the previous Hot Spots technical support document Part II (OEHHA, 2005a). Summaries of cancer potency factors and the underlying data are provided in Appendices A and B, which are subject to ongoing updates but were not changed as part of the revision process which created this TSD.

The procedures used to consider the increased susceptibility to carcinogens of infants and children as compared to adults include the use of age-specific weighting factors in calculating cancer risks from exposures of infants, children and adolescents, to reflect their anticipated special sensitivity to carcinogens

This document is one part of the Air Toxics Hot Spots Program Risk Assessment Guidelines. The other documents originally included in the Guidelines are Part I: Technical Support Document for the Determination of Acute Toxicity Reference Exposure Levels for Airborne Toxicants; Part III: Technical Support Document for Determination of Noncancer Chronic Reference Exposure Levels; Part IV: Technical Support Document for Exposure Assessment and Stochastic Analysis; Part V: Air Toxic Hot Spots Program Risk Assessment Guidelines. As a part of the same revision process which led to production of this revised TSD on cancer potencies, the original TSDs for Acute and Chronic Reference Exposure Levels have been replaced with a new unified TSD for Acute, 8-hour and Chronic Reference Exposure Levels.

The major changes to the TSD include the following:

- Based on the OEHHA analysis of the potency by lifestage at exposure, OEHHA proposes weighting cancer risk by a factor of 10 for exposures that occur from the third trimester of pregnancy to 2 years of age, and by a factor of 3 for exposures that occur from 2 years through 15 years of age. We intend to apply this weighting factor to all carcinogens,

regardless of purported mechanism of action, unless chemical-specific data exist to the contrary. In cases where there are adequate data for a specific carcinogen of potency by age, we would use the data to make any adjustments to risk.

- OEHHA proposes to use the Benchmark Dose method to compute potency factors rather than the more traditional linearized multistage model (LMS), although the LMS will still be used in some instances. The BMDL model essentially uses an empirical fit to the data (usually best with the multistage model), and then extrapolates with a straight line from the 95% lower confidence limit of the BMD (BMDL) to zero. This method is simpler and does not assume any underlying theoretical mechanisms at the low dose range. The BMDL method results in estimates of potency very similar to those obtained using the LMS method.

- OEHHA will use scaling based on body weight to the ¾ power, rather than to the ⅔ power.

- OEHHA's evaluations of the carcinogenicity of chemicals generally follow the guidelines laid out by IARC for identification and classification of potential human carcinogens, which are described in detail in the most recent revision of the *Preamble* to the IARC monographs series (IARC, 2006).

PREFACE

The Air Toxics "Hot Spots" Information and Assessment Act (AB 2588, Connelly) was enacted in September 1987. Under this Act, stationary sources are required to report the types and quantities of certain substances their facilities routinely release into the air. The goals of the Air Toxics "Hot Spots" Act are to collect emission data, identify facilities having localized impacts, ascertain health risks posed by those facilities, notify nearby residents of significant risks and reduce emissions from significant sources.

The Technical Support Document for Cancer Potency Factors (TSD) contains cancer unit risks and potency factors for 107 of the 201 carcinogenic substances or groups of substances for which emissions must be quantified in the Air Toxics Hot Spots program. These unit risks are used in risk assessment of facility emissions. The TSD provides updated calculation procedures used to derive the estimated unit risk and cancer potency factors, and procedures to consider early-life susceptibility to carcinogens. Summaries of cancer potency factors and the underlying data are provided in Appendices A and B.

In this document, OEHHA is responding to the requirements of the 1999 Children's Environmental Health Protection Act (SB25, Escutia) by revising the procedures for derivation and application of cancer potency factors to take account of general or chemical-specific information which suggests that children may be especially susceptible to certain carcinogens (OEHHA, 2001a). The revised cancer potency derivation procedures described will not be used to impose any overall revisions of the existing cancer potencies, although they do reflect updated methods of derivation. However, individual cancer potency values will be reviewed as part of the ongoing re-evaluation of health values mandated by SB 25, and revised values will be listed in updated versions of the appendices to this document as necessary. The revisions also include the use of weighting factors in calculating cancer risks from exposures of infants, children and adolescents, to reflect their anticipated special sensitivity to carcinogens. Similar legal mandates to update risk assessment methodology and cancer potencies apply to the OEHHA program for development of Public Health Goals (PHGs) for chemicals in drinking water, and Proposition 65 No Significant Risk Levels (NSRLs). The NSRLs may also be revised to reflect concerns for children's health. Revising these numbers will require the originating program to reconsider the value in an open public process. For example, OEHHA would need to release any revised potency factors for public comment and review by the Scientific Review Panel on Toxic Air Contaminants (SRP) prior to adoption under the TAC program. The procedures for outside parties to request reevaluation of cancer potency values by the programs which originated those values are listed in Appendix G.

Appendices A and B provide previously adopted Cal/EPA values which were included in the previous version of the TSD for Cancer Potency Factors (OEHHA, 2005a). Cal/EPA values were developed under the Toxic Air Contaminant (TAC) program, the PHG program, the Proposition 65 program, or in some cases specifically for the Air Toxics Hot Spots program. All the Cal/EPA values are submitted for public comments and external peer review prior to adoption by the program of origin. In the future, new values developed by the Toxic Air Contaminants or Hot Spots programs or other suitable sources will be added as these are approved.

Some U.S. EPA IRIS cancer unit risk values were adopted under the previous versions of these guidelines, and these values will continue to be used unless and until revised by Cal/EPA. U.S. EPA has recently revised its cancer risk assessment guidelines (U.S. EPA, 2005a). Some of the recommended changes in methodology could result in slightly different potency values compared to those calculated by the previous methodology, although in practice a number of the recommendations (for example, the use of ¾ power of the body weight ratio rather than ⅔ power for interspecies scaling) have been available in draft versions of the revised policy for some time and appear in many more recent assessments. U.S. EPA has stated that cancer potency values listed in IRIS will not be revisited solely for the purpose of incorporating changes in cancer potency value calculation methods contained in the revised cancer risk assessment guidelines. U.S. EPA has also issued supplementary guidelines on assessing cancer risk from early-life exposure (U.S. EPA, 2005b).

OEHHA uses a toxic equivalency factor procedure for dioxin-like compounds, including polychlorinated dibenzo-*p*-dioxins, dibenzofurans and polychlorinated biphenyls (PCBs). The Toxicity Equivalency Factor scheme (TEF_{WHO-97}) developed by the World Health Organization/European Center for Environmental Health (WHO-ECEH) is used for determining cancer unit risk and potency values for these chemicals where individual congener emissions are available (Appendix C).

This document is one part of the Air Toxics Hot Spots Program Risk Assessment Guidelines. The other documents originally included in the Guidelines are Part I: Technical Support Document for the Determination of Acute Toxicity Reference Exposure Levels for Airborne Toxicants; Part III: Technical Support Document for Determination of Noncancer Chronic Reference Exposure Levels; Part IV: Technical Support Document for Exposure Assessment and Stochastic Analysis; Part V: Air Toxic Hot Spots Program Risk Assessment Guidelines. As a part of the same revision process which led to production of this revised TSD on cancer potencies, the original TSDs for Acute and Chronic Reference Exposure Levels have been replaced with a new unified TSD for Acute, 8-hour and Chronic Reference Exposure Levels.

TABLE OF CONTENTS

APPENDICES

Appendix A. A lookup table containing unit risk and cancer potency values.

Appendix B. Chemical-specific summaries of the information used to derive unit risk and cancer potency values.

Appendix C. A description of the use of toxicity equivalency factors for determining unit risk and cancer potency factors for polychlorinated dibenzo-*p*-dioxins, dibenzofurans and dioxin-like polychlorinated biphenyls.

Appendix D. A listing of Toxic Air Contaminants identified by the California Air Resources Board.

Appendix E. Descriptions of the International Agency for Research on Cancer (IARC) and U.S. Environmental Protection Agency (U.S. EPA) carcinogen classifications.

Appendix F. An asbestos quantity conversion factor for calculating asbestos concentrations expressed as 100 fibers/m^3 from asbestos concentrations expressed as µg/m^3.

Appendix G. Procedures for revisiting or delisting cancer potency factors by the program of origin.

Appendix H. Exposure routes and studies used to derive cancer unit risks and slope factors.

Appendix I. "Assessing susceptibility from early-life exposure to carcinogens": Barton *et al*., 2005 (from *Environmental Health Perspectives)*.

Appendix J. "In Utero and Early Life Susceptibility to Carcinogens: The Derivation of Age-at-Exposure Sensitivity Measures" – conducted by OEHHA's Reproductive and Cancer Hazard Assessment Branch.

INTRODUCTION

The Technical Support Document (TSD) for Describing Available Cancer Potency Factors provides technical information support for the Air Toxics Hot Spots Program Risk Assessment Guidelines. The TSD consists of 12 sections:

1. The TSD introduction.

2. A description of the methodologies used to derive the unit risk and cancer potency values listed in the lookup table.

3. A lookup table containing unit risk and cancer potency values. (Appendix A)

4. Chemical-specific summaries of the information used to derive unit risk and cancer potency values. (Appendix B).

5. A description of the use of toxicity equivalency factors for determining unit risk and cancer potency factors for polychlorinated dibenzo-*p*-dioxins, dibenzofurans and dioxin-like polychlorinated biphenyls (Appendix C).

6. A listing of Toxic Air Contaminants identified by the California Air Resources Board (Appendix D).

7. Descriptions of the International Agency for Research on Cancer (IARC) and U.S. Environmental Protection Agency (U.S. EPA) carcinogen classifications (Appendix E).

8. An asbestos quantity conversion factor for calculating asbestos concentrations expressed as 100 fibers/m^3 from asbestos concentrations expressed as µg/m^3 (Appendix F).

9. Procedures for revisiting or delisting cancer potency factors by the program of origin (Appendix G).

10. Exposure routes and studies used to derive cancer unit risks and slope factors (Appendix H).

11. "Assessing susceptibility from early-life exposure to carcinogens": Barton *et al*., 2005 (from *Environmental Health Perspectives)* (Appendix I).

12. "In Utero and Early Life Susceptibility to Carcinogens: The Derivation of Age-at-Exposure Sensitivity Measures" – conducted by OEHHA's Reproductive and Cancer Hazard Assessment Branch (Appendix J)

SELECTION OF CANCER POTENCY VALUES

The Office of Environmental Health Hazard Assessment (OEHHA) has developed a number of cancer potencies for use in the Toxic Air Contaminants and Air Toxics Hot Spots programs. This document also provides summaries of cancer potency factors which were originally developed for other California Environmental Protection Agency (Cal/EPA) programs, or by the U.S. EPA. These were reviewed for accuracy, reliance on up-to-date data and methodology, and applicability in the context of the Air Toxics Hot Spots program. Values found appropriate were adopted after public and peer review rather than devoting the resources necessary for a full *de novo* assessment. Thus, cancer potency values (CPF) included in the Technical Support Document (TSD) for Cancer Potency Factors were from the following sources:

1. Toxic Air Contaminant documents

2. Standard Proposition 65 documents

3. U.S.EPA Integrated Risk Information Systems (Office of Health and Environmental Assessment, U.S.EPA)

4. Expedited Proposition 65 documents

5. Other OEHHA assessments , for example for the drinking water program.

All the cancer potency value sources used generally follow the recommendations of the National Research Council on cancer risk assessment (NRC, 1983, 1994). All Cal/EPA program documents undergo a process of public comment and scientific peer review prior to adoption, although the procedures used vary according to the program. The publication procedure for Toxic Air Contaminant documents includes a public comment period and review by the Scientific Review Panel on Toxic Air Contaminants (SRP) before identification of a Toxic Air Contaminant by the Air Resources Board of the California Environmental Protection Agency (Cal/EPA). Furthermore, a petition procedure is available to initiate TAC document review and revision if appropriate because of new toxicity data. Documents developed for the Air Toxics Hot Spots program similarly undergo public comment and peer review by the SRP before adoption by the Director of OEHHA. The standard Proposition 65 document adoption procedure includes a public comment and external peer review by the Proposition 65 Carcinogen Identification Committee. The expedited Proposition 65 document adoption procedure included a public comment period. Risk assessments prepared for development of Public Health Goals (PHGs) for chemicals in drinking water are subject to two public comment periods before the final versions and responses to comments are published on the OEHHA Web site. PHG documents may also receive external peer review. Documents from U.S. EPA's Integrated Risk Information System (IRIS) receive external peer review and are posted on the Internet for public viewing during the external peer review period, and any public comments submitted are considered by the originating office. Additionally, public comment may be solicited during the document posting period. Future preference for use of developed cancer potency factors/unit risks will be done on a case by case basis. Preference will be given to those assessments most relevant to inhalation exposures of the California population, to the most recent derivations using the latest data sets and scientific methodology, and to those having undergone the most open and extensive peer review process.

CANCER RISK ASSESSMENT METHODOLOGIES

This section describes in general the methodologies used to derive the cancer unit risk and potency factors listed in this document. As noted in the Preface to this document, no new cancer unit risks or potency factors were developed for this document. All of the values contained here were previously developed in documents by Cal/EPA or U.S. EPA. Following the recommendations of the National Academy of Sciences (NRC, 1983), Cal/EPA and U.S. EPA have both used formalized cancer risk assessment guidelines, the original versions of which (California Department of Health Services, 1985; U.S. EPA, 1986) were published some time ago. Both these guidelines followed similar methodologies.

In the twenty years since these original guidelines were published there have been a number of advances in the methodology of cancer risk assessment. There have additionally been considerable advances in the quantity of data available not only from animal carcinogenesis bioassays and epidemiological studies, but also from mechanistic studies of carcinogenesis and related phenomena. Some of these advances have been incorporated into newer risk assessments by both agencies on a more or less *ad hoc* basis. There has also been an ongoing effort to provide updated risk assessment guidance documents. In 1995, U.S. EPA released for public comment the "Proposed and Interim Guidelines for Carcinogen Risk Assessment", which was the first of several drafts released for public comment. Many risk assessments appearing since then have used elements of the recommendations contained in that document, in spite of its draft status. A final version of the U.S. EPA's revised cancer risk assessment guidelines has now been released (U.S. EPA, 2005a). Although these new guidelines incorporate a number of substantial changes from their predecessors (U.S. EPA, 1986; 1995), U.S. EPA has stated that cancer potency values listed in IRIS will not be revisited solely for the purpose of incorporating changes in cancer potency value calculation methods.

Cal/EPA has not produced a revised cancer risk assessment guideline document to replace the original version (DHS, 1985). Rather, Cal/EPA has relied on incorporating new data and methodologies as these became available, and described the methods used on a case by case basis in the individual risk assessment documents where these went beyond the original guidance. However, this revision of the TSD for cancer potencies provides a convenient opportunity to summarize the current status of the methodology used by OEHHA for the air toxics programs, and also to highlight points of similarity to, and difference from, the recommendations of U.S. EPA (2005a).

In this document, OEHHA intends to follow the recommendations of the NRC (1994) in describing a set of clear and consistent principles for choosing and departing from default cancer risk assessment options. NRC identified a number of objectives that should be taken into account when considering principles for choosing and departing from default options. These include, "protecting the public health, ensuring scientific validity, minimizing serious errors in estimating risks, maximizing incentives for research, creating an orderly and predictable process, and fostering openness and trustworthiness". The OEHHA cancer risk methodologies discussed in this document are intended to generally meet those objectives cited above.

Hazard Identification

This section will describe: 1) how weight of evidence evaluations are used in hazard evaluation; 2) guidelines for inferring causality of effect; 3) the use of human and animal carcinogenicity data, as well as supporting evidence (*e.g.*, genetic toxicity and mechanistic data); 4) examples of carcinogen identification schemes.

Evaluation of Weight of Evidence

In evaluating the range of evidence on the toxicity and carcinogenicity of a compound, mixture or other agent, a "weight-of-evidence" approach is generally used to describe the body of evidence on whether or not exposure to the agent causes a particular effect. Under this approach, the number and quality of toxicological and epidemiological studies, as well as the consistency of study results and other sources of data on biological plausibility, are considered. Diverse and sometimes conflicting data need to be evaluated with respect to possible explanations of differing results. Consideration of methodological issues in the review of the toxicological and epidemiological literature is important in evaluating associations between exposure to an agent and animal or human health effects. This aspect of the evaluation process has received particular emphasis with respect to epidemiological data, where concerns as to the statistical and biological significance and reliability of the data and the impacts of confounding and misclassification are pressing. Such concerns are also relevant to some extent in the interpretation of animal bioassay data and mechanistic studies. Although the test animals, laboratory environment and characterization of the test agent are usually much better controlled than the equivalent parameters in an epidemiological study, the small sample size can be problematic. In addition, there are uncertainties associated with extrapolation of biological responses from test animal species to humans.

Criteria for Causality

There has been extensive discussion over the last two centuries on causal inference. This has particularly related to epidemiological data, but is also relevant to interpretation of animal studies. Most epidemiologists utilize causal inference guidelines based on those proposed by Bradford Hill (1971). OEHHA has relied on these and on recommendations by IARC (2006), the Institute of Medicine (2004), the Surgeon General's Reports on Smoking (U.S. DHHS, 2004) and standard epidemiologic texts (*e.g.*, Lilienfeld and Lilienfeld, 1980; Rothman and Greenland, 1998). The criteria for determination of causality used by OEHHA have been laid out in various risk assessment documents. The summary below is adapted from the Health Effects section of the document prepared to support the identification of environmental tobacco smoke (ETS) as a Toxic Air Contaminant (OEHHA, 2005b).

1. *Strength of Association*. A statistically significant strong association, which is easier to detect if there is a high relative risk, between a factor and a disease is often viewed as an important criterion for inferring causality because, all other things being equal, a strong and statistically significant association makes alternative explanations for the disease less likely. However, as discussed in Rothman and Greenland (1998), the fact that a relative risk is small in magnitude does not exclude a casual association between the risk factor

and the outcome in question. Since it is more difficult to detect (*i.e.*, reach statistical significance) a small magnitude risk, it is just as likely to indicate causality as a larger magnitude risk.

When assessing all evidence, it is important to consider the strength of the study design (particularly controlling for confounding variables, obtaining an unbiased sample, measurement error) and the level of statistical significance (*i.e.*, the ability to exclude a Type I [false positive] error). The power of the study to detect biologically meaningful effects (*i.e.*, the risk of a Type II [false negative] error) is important in considering studies that do not reach traditional (*i.e.*, $P < 0.05$) statistical significance, particularly if the biological endpoint is serious. If the outcome is serious and the study small (*i.e.*, low power), a larger P value (*e.g.*, $P < 0.10$) may be adequate evidence for identifying an effect.

There are a number of examples of statistically significant, small magnitude associations that are widely accepted as causal, such as causal links between air pollution and cardiovascular/pulmonary mortality and between second-hand smoke exposure and various cancers and heart disease. From a public health perspective, even a small magnitude increase in risk for a common disease can mean large numbers of people affected by the health outcome when exposure is frequent and widespread, as measured by the population attributable risk or attributable fraction. Small magnitude of association must not be confused with statistical significance, which is much more important.

2. *Consistency of Association.* If several investigations find an association between a factor and a disease across a range of populations, geographic locations, times, and under different circumstances, then the factor is more likely to be causal. Consistency argues against hypotheses that the association is caused by some other factor(s) that varies across studies. Unmeasured confounding is an unlikely explanation when the effect is observed consistently across a number of studies in different populations.

   Associations that are replicated in several studies of the same design or using different epidemiological approaches or considering different sources of exposure and in a number of geographical regions are more likely to represent a causal relationship than isolated observations from single studies (IARC, 2006). If there are inconsistent results among investigations, possible reasons are sought, such as adequacy of sample size or control group, methods used to assess exposure, or range in levels of exposure. The results of studies judged to be rigorous are emphasized over those of studies judged to be methodologically less rigorous. For example, studies with the best exposure assessment are more informative for assessing the association between ETS and breast cancer than studies with limited exposure assessment, all else being equal.

3. *Temporality.* Temporality means that the factor associated with causing the disease occurs in time prior to development of the disease. The adverse health effect should occur at a time following exposure that is consistent with the nature of the effect. For example, respiratory irritation immediately following exposure to an irritant vapor is temporally consistent, whereas irritation noted only years later may not be. On the other

hand, tumors, noted immediately following exposure, might be temporally inconsistent with a causal relationship, but tumors arising after a latency period of months (in rodents) or years (in rodents or humans) would be temporally consistent.

4. *Coherence and Biological Plausibility.* A causal interpretation cannot conflict with what is known about the biology of the disease. The availability of experimental data or mechanistic theories consistent with epidemiological observations strengthens conclusions of causation. For example, the presence of known carcinogens in tobacco smoke supports the concept that exposure to tobacco smoke could cause increased cancer risk. Similarly, if the mechanism of action for a toxicant is consistent with development of a specific disease, then coherence and biological plausibility can be invoked. It should be noted that our understanding of the biology of disease, and therefore biological plausibility, changes in light of new information which is constantly emerging from molecular biology (including epigenetics), and from new clinical and epidemiological investigations revealing effects influenced by genetic polymorphisms, pre-existing disease, and so forth.

5. *Dose-Response.* A basic tenet of toxicology is that increasing exposure or dose generally increases the response to the toxicant. While dose-response curves vary in shape and are not necessarily always monotonic, an increased gradient of response with increased exposure makes it difficult to argue that the factor is not associated with the disease. To argue otherwise necessitates that an unknown factor varies consistently with the dose of the substance and the response under question. While increased risk with increasing levels of exposure is considered to be a strong indication of causality, absence of a graded response does not exclude a causal relationship (IARC, 2006).

   The dose-response curves for specific toxic effects may be non-monotonic. Under appropriate circumstances, where the dose response shows saturation, the effect of exposures could be nearly maximal, with any additional exposure having little or no effect. In some instances, a response is seen strongly in susceptible subpopulations, and the dose-response is masked by mixing susceptible and non-susceptible individuals in a sample. Further, there are examples of U-shaped or inverted U-shaped dose-response curves, (*e.g.*, for endocrine disrupters) (Almstrup et al., 2002; Lehmann et al., 2004). Finally, timing of exposure during development may mask an overall increase in risk with increasing dose.

6. *Specificity.* Specificity is generally interpreted to mean that a single cause is associated with a single effect. It may be useful for determining which microorganism is responsible for a particular disease, or associating a single carcinogenic chemical with a rare and characteristic tumor (*e.g.*, liver angiosarcoma and vinyl chloride, or mesothelioma and asbestos). However, the concept of specificity is not helpful when studying diseases that are multifactorial, or toxic substances that contain a number of individual constituents, each of which may have several effects and/or target sites.

7. *Experimental evidence.* While experiments are often conducted over a short period of time or under artificial conditions (compared to real-life exposures), experiments offer the opportunity to collect data under highly controlled conditions that allow strong causal

conclusions to be drawn. Experimental data that are consistent with epidemiological results strongly support conclusions of causality. There are also "natural experiments" that can be studied with epidemiological methods, such as when exposure of a human population to a substance declines or ceases; if the effect attributed to that exposure decreases, then there is evidence of causality. One example of this is the drop in heart disease death and lung cancer risk after smoking cessation.

It should be noted that the causal criteria are guidelines for judging whether a causal association exists between a factor and a disease, rather than hard-and-fast rules. Lilienfeld and Lilienfeld (1980) note that "*In medicine and public health, it would appear reasonable to adopt a pragmatic concept of causality. A causal relationship would be recognized to exist whenever evidence indicates that the factors form part of the complex of circumstances that increases the probability of the occurrence of disease and that a diminution of one or more of these factors decreases the frequency of that disease. After all, the reason for determining the etiological factors of a disease is to apply this knowledge to prevent the disease.*" Rothman and Greenland (2005) discuss the complexities of causation and the use of rules and deductive methods in causal inference. They also concur with Bradford Hill and others that a determination of causality is a pragmatic conclusion rather than an absolute verdict, and advocate that these criteria should be seen as "*deductive tests of causal hypotheses".*

Data Sources

Human studies: epidemiology, ecological studies and case reports

The aim of a risk assessment for the California Air Toxics programs is to determine potential impact on human health. Ideally therefore, the hazard identification would rely on studies in humans to demonstrate the nature and extent of the hazard. However, apart from clinical trials of drugs, experimental studies of toxic effects in human subjects are rarely undertaken or justifiable. Pharmacokinetic studies using doses below the threshold for any toxic effect have been undertaken for various environmental and occupational agents, but are not usually regarded as appropriate for suspected carcinogens.

The human data on carcinogens available to the risk assessor therefore mostly consist of epidemiological studies of existing occupational or environmental exposures. It is easier to draw reliable inferences in situations where both the exposures and the population are substantial and well-defined, and accessible to direct measurement rather than recall. Thus, many important findings of carcinogenicity to humans are based on analysis of occupational exposures. Problems in interpretation of occupational epidemiological data include simultaneous exposure to several different known or suspected carcinogens, imprecise quantification of exposures and confounding exposures such as active or passive tobacco smoking. The historical database of occupational data has a bias towards healthy white adult males. Thus, the hazard analysis of these studies may not accurately characterize effects on women, infants, children or the elderly, or on members of minority ethnic groups. Nevertheless, the analysis of occupational epidemiological studies, including meta-analyses, has proved an important source for unequivocal identification of human carcinogens.

Epidemiological evidence may also be obtained where a substantial segment of a general population is exposed to the material of interest in air, drinking water or food sources. Rigorous cohort and case-control studies may sometimes be possible, in which exposed individuals are identified, their exposure and morbidity or mortality evaluated, and compared to less exposed but otherwise similar controls. More often at least the initial investigation is a cross-sectional study, where prevalence of exposures and outcomes is compared in relatively unexposed and exposed populations. Such studies are hypothesis-generating, but are important sources of information nevertheless, and can often also justify more costly and labor-intensive follow-up cohort and/or case-control studies.

The clinical medical literature contains many case reports where a particular health outcome is reported along with unusual exposures that might have contributed to its occurrence. These reports typically describe a single patient or a small group, and have no statistical significance. They are nevertheless useful as indications of possible associations that deserve follow-up using epidemiological methods, and as supporting evidence, addressing the plausibility of associations measured in larger studies.

Animal studies

Although the observation of human disease in an exposed population can provide definitive hazard identification, adequate data of this type are not always available. More often, risk estimates have to be based on studies in experimental animals, and extrapolation of these results to predict human toxicity. The animals used are mostly rodents, typically the common laboratory strains of rat and mouse.

Rats and mice have many similarities to humans. Physiology and biochemistry are similar for all mammals, especially at the fundamental levels of xenobiotic metabolism, DNA replication and DNA repair that are of concern in identifying carcinogens. However, there are also several important differences between rodents and humans. Rodents, with a short life span, have differences in cell growth regulation compared to longer-lived species such as the human. For instance, whereas laboratory investigations have suggested that mutations in two regulatory genes (*e.g.*, H-ras and p-53) are sometimes sufficient to convert a rodent cell to a tumorigenic state, many human cancers observed clinically have seven or eight such mutations. In addition, cultured normal human cells have a very stable karyotype, whereas cultured rodent cells facilely undergo tetraploidization and then aneuploidization in cell culture. Further, cultured human cells senesce and rarely undergo spontaneous immortalization (frequency is 10^{-7} or less), whereas cultured rodent cells facilely undergo immortalization at frequencies on the order of 10^{-3}. The use of genomics to study chemical carcinogenesis is relatively new, but the differences at present appear to be a matter of degree rather than kind.

Differences in regulation of cell division are another likely reason for variation between species in the site of action of a carcinogen, or its potency at a particular site. A finding of carcinogenesis in the mouse liver, for instance, is a reasonably good indicator of potential for carcinogenesis at some site in the human, but not usually in human liver (Huff, 1999). The mouse liver (and to a lesser extent that of the rat) is a common site of spontaneous tumors. It is also relatively sensitive to chemical carcinogenesis. The human liver is apparently more resistant to carcinogenesis; human liver tumors are unusual except when associated with

additional predisposing disease, such as hepatitis B or alcoholic cirrhosis, or exposure to aflatoxin B1, or simultaneous exposure to hepatitis B virus and aflatoxin B1. Conversely, other tumor sites are more sensitive in the human than in experimental animals. Interspecies variation in site and sensitivity to carcinogenesis may also arise from differences in pharmacokinetics and metabolism, especially for carcinogens where metabolic activation or detoxification is important. This variability may cause important differences in sensitivity between individuals in a diverse population such as humans. Variability between individuals in both susceptibility and pharmacokinetics or metabolism is probably less in experimental animal strains that are bred for genetic homogeneity.

Animal carcinogenesis studies are often designed to maximize the chances of detecting a positive effect, and do not necessarily mimic realistic human exposure scenarios. Thus extrapolation from an experimentally accessible route to that of interest for a risk assessment may be necessary. Even for studies by realistic routes such as oral or inhalation, doses may be large compared to those commonly encountered in the environment, in order to counter the limitation in statistical power caused by the relatively small size of an animal experiment. Whereas the exposed population of an epidemiological study might number in the thousands, a typical animal study might have fifty individuals per exposure group. With this group size any phenomenon with an incidence of less than about 5% is likely to be undetectable. Statistically significant results may be obtained even with groups as small as ten animals per dose group, when incidence of a tumor that is rare in the controls approached 100% in a treated group. The consensus experimental design for animal carcinogenesis studies, which has evolved over the last 50 years of investigation, is represented by the protocol used by the U.S. National Toxicology Program (NTP) for studies using oral routes (diet, gavage or drinking water) or inhalation. These carcinogenesis bioassays usually involve both sexes of an experimental species, and most often two species. NTP has standardized the use of the C57BlxC3H F_1 hybrid mouse, and the Fischer 344 rat as the standard test species, although NTP has announced plans to substitute use of the Wistar Han rat for the Fischer 344 rat. There is now an extensive database of background tumor incidences, normal physiology, biochemistry, histology and anatomy for these strains, which aids in the interpretation of pathological changes observed in experiments. Nevertheless, there is enough variation in background rates of common tumors that the use of concurrent controls is essential for hazard identification or dose-response assessment. "Historical control" data are mainly used to reveal anomalous outcomes in the concurrent controls. The fact that a significantly elevated incidence of a tumor relative to the concurrent control group is within the range of historical controls at that site for the test sex and strain is not necessarily grounds for dismissing the biological significance of the finding.

Groups of fifty animals of each sex and species are used, with control groups, and several dose groups, the highest receiving the maximum tolerated dose (MTD). Recent study designs have emphasized the desirability of at least three dose levels covering a decade with "logarithmic" spacing (*i.e.* MTD, 1/2 MTD or 1/3 MTD, and 1/10 MTD). This extended design is aimed at providing better dose-response information, and may contribute important additional information, such as mechanistic insights, for the hazard identification phase.

Supporting evidence: genetic toxicity, mechanistic studies

Investigators have developed additional data sources that can support or modify the conclusions of animal carcinogenesis bioassays, and provide information on mechanisms of action of agents suspected of being carcinogenic based on epidemiological studies or animal bioassays.

Genetic damage in exposed organisms includes both gene mutations (point or frameshift), and larger scale effects such as deletions, gene amplification, sister-chromatid exchanges, translocations and loss or duplication of segments or whole chromosomes. These genetic effects of chemical exposures are deleterious in their own right. In addition, since carcinogenesis results from somatic mutations and similar genetic alterations, agents that cause genetic damage generally have carcinogenic potential. Conversely, many known carcinogens are also known to be genotoxic, although there is also a significant class of carcinogens that are not directly genotoxic according to the usual tests. These latter agents presumably work by some other mechanism, such as methylation of tumor suppressor genes or demethylation of cellular proto-oncogenes, although recent genetic studies have shown that even tumors induced by these agents may show mutations, deletions or amplification of growth regulatory genes.

Experimental procedures to demonstrate and measure genetic toxicity may involve exposure of intact animals, and examination of genetic changes in, for example, bone marrow cells (or cells descended from these, *e.g.*, the micronucleus test, which detects remnants of chromosomal fragments in immature erythrocytes), mutations in flies (*Drosophila)*, or appearance of color spots in the coat of mice. However, many tests have employed single celled organisms or mammalian cells in culture. The best known of these tests is the *Salmonella* reverse mutation assay, popularly known as the Ames test after its inventor. This is representative of a larger class of tests for mutagenic activity in prokaryotic organisms (bacteria), which necessarily only look at gene-level mutations. Similar tests in eukaryotic microorganisms (yeasts, *Aspergillus*) and cultured mammalian cells also detect chromosomal effects. Many tests using microorganisms *in vitro* involve addition of activating enzymes (*e.g.*, liver postmitochondrial supernatant – "S9") to mimic the metabolism of promutagenic chemicals *in vivo*. Another type of test examines the induction in mammalian cells of morphological transformation or anchorage-independent growth. These two chemically induced, *in vitro* changes are considered two of the many changes that fibroblastic cells must undergo on their route to neoplastic transformation (tumorigenicity). These various genetic tests contribute different information, which may be used to amplify and confirm conclusions drawn from human studies or animal bioassays, or to draw conclusions in the absence of epidemiological or bioassay data. In the latter case they have also been used in prioritizing agents for further evaluation by means of bioassays.

Carcinogen Identification Schemes

Some regulatory programs, such as California's Safe Drinking Water and Toxics Enforcement Act ("Proposition 65") and various activities of the U.S. EPA, require that explicit lists of substances having the potential to act as human carcinogens be maintained. Other such lists are developed by non-regulatory research organizations, such as the U.S. National Toxicology Program and the International Agency for Research on Cancer (IARC), an international program of the World Health Organization. The California air toxics programs do not have any statutory requirement to "identify" carcinogens. The requirement instead is to identify hazardous

substances as Toxic Air Contaminants, and to determine whether or not a threshold concentration, below which no adverse effects are expected, is likely to exist:

> ***HEALTH AND SAFETY CODE, Division 26 (Air Resources), § 39660.***
>
> *(2) The evaluation shall also contain an estimate of the levels of exposure that may cause or contribute to adverse health effects. If it can be established that a threshold of adverse health effects exists, the estimate shall include both of the following factors:*
>
> *(A) The exposure level below which no adverse health effects are anticipated.*
>
> *(B) An ample margin of safety that accounts for the variable effects that heterogeneous human populations exposed to the substance under evaluation may experience, the uncertainties associated with the applicability of the data to human beings, and the completeness and quality of the information available on potential human exposure to the substance. In cases in which there is no threshold of significant adverse health effects, the office shall determine the range of risk to humans resulting from current or anticipated exposure to the substance.*

In practice however this requirement amounts to the need to establish whether or not a substance is carcinogenic. Any such effects are clearly harmful. Whereas the great majority of non-cancer health effects of chemicals are regarded as having a threshold, the default assumption for carcinogens is that there is no threshold (as described below). OEHHA follows the guidelines laid out by IARC for identification and classification of potential human carcinogens, which are described in detail in the most recent revision of the *Preamble* to the IARC monographs series (IARC, 2006). The IARC Monograph series provides evaluations of the carcinogenicity of individual substances or commonly occurring mixtures. The evaluation guidelines used are similar to those used by other scientific or regulatory authorities, including U.S.EPA.

The data inputs to hazard identification for carcinogens are human epidemiological studies, animal bioassays, along with supporting evidence such as mechanistic and genotoxicity data and structure-activity comparisons. IARC also assembles data on the structure and identity of the agent. The list of agents considered includes specific chemicals and also complex mixtures, occupational and lifestyle factors, physical and biological agents, and other potentially carcinogenic exposures.

IARC evaluations determine the quality of evidence for both animal and human evidence as falling into one of four categories: sufficient evidence of carcinogenicity, limited evidence of carcinogenicity, inadequate evidence of carcinogenicity and evidence suggesting lack of carcinogenicity. Stringent requirements for data quality are imposed. In view of their crucial importance, these definitions are quoted directly from the *Preamble* (IARC 2006):

> **"(a) Carcinogenicity in humans**
>
> ***Sufficient evidence of carcinogenicity:*** The Working Group considers that a causal relationship has been established between exposure to the agent and human cancer. That is, a positive relationship has been observed between the exposure and cancer in studies in which chance, bias and confounding could be ruled out with reasonable confidence. A statement that there is *sufficient evidence* is followed by a separate sentence that

identifies the target organ(s) or tissue(s) where an increased risk of cancer was observed in humans. Identification of a specific target organ or tissue does not preclude the possibility that the agent may cause cancer at other sites.

***Limited evidence of carcinogenicity*:** A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.

Inadequate evidence of carcinogenicity: The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association between exposure and cancer, or no data on cancer in humans are available.

Evidence suggesting lack of carcinogenicity: There are several adequate studies covering the full range of levels of exposure that humans are known to encounter, which are mutually consistent in not showing a positive association between exposure to the agent and any studied cancer at any observed level of exposure. The results from these studies alone or combined should have narrow confidence intervals with an upper limit close to the null value (*e.g.*, a relative risk of 1.0). Bias and confounding should be ruled out with reasonable confidence, and the studies should have an adequate length of follow-up. A conclusion of *evidence suggesting lack of carcinogenicity* is inevitably limited to the cancer sites, conditions and levels of exposure, and length of observation covered by the available studies. In addition, the possibility of a very small risk at the levels of exposure studied can never be excluded.

(b) Carcinogenicity in experimental animals

Carcinogenicity in experimental animals can be evaluated using conventional bioassays, bioassays that employ genetically modified animals, and other in-vivo bioassays that focus on one or more of the critical stages of carcinogenesis. In the absence of data from conventional long-term bioassays or from assays with neoplasia as the end-point, consistently positive results in several models that address several stages in the multistage process of carcinogenesis should be considered in evaluating the degree of evidence of carcinogenicity in experimental animals.

The evidence relevant to carcinogenicity in experimental animals is classified into one of the following categories:

***Sufficient evidence of carcinogenicity*:** The Working Group considers that a causal relationship has been established between the agent and an increased incidence of malignant neoplasms or of an appropriate combination of benign and malignant neoplasms in (a) two or more species of animals or (b) two or more independent studies in one species carried out at different times or in different laboratories or under different protocols. An increased incidence of tumours in both sexes of a single species in a well-conducted study, ideally conducted under Good Laboratory Practices, can also provide *sufficient evidence*.

A single study in one species and sex might be considered to provide *sufficient evidence of carcinogenicity* when malignant neoplasms occur to an unusual degree with regard to

incidence, site, type of tumour or age at onset, or when there are strong findings of tumours at multiple sites.

***Limited evidence of carcinogenicity*:** The data suggest a carcinogenic effect but are limited for making a definitive evaluation because, *e.g.*, (a) the evidence of carcinogenicity is restricted to a single experiment; (b) there are unresolved questions regarding the adequacy of the design, conduct or interpretation of the studies; (c) the agent increases the incidence only of benign neoplasms or lesions of uncertain neoplastic potential; or (d) the evidence of carcinogenicity is restricted to studies that demonstrate only promoting activity in a narrow range of tissues or organs.

***Inadequate evidence of carcinogenicity*:** The studies cannot be interpreted as showing either the presence or absence of a carcinogenic effect because of major qualitative or quantitative limitations, or no data on cancer in experimental animals are available.

***Evidence suggesting lack of carcinogenicity*:** Adequate studies involving at least two species are available which show that, within the limits of the tests used, the agent is not carcinogenic. A conclusion of *evidence suggesting lack of carcinogenicity* is inevitably limited to the species, tumour sites, age at exposure, and conditions and levels of exposure studied."

IARC utilizes the evaluations of animal and human data, along with supporting evidence including genotoxicity, structure-activity relationships, and identified mechanisms, to reach an overall evaluation of the potential for carcinogenicity in humans. The revised *Preamble* (IARC, 2006) includes a description of the data evaluation criteria for this supporting evidence, and indications as to the situations where the availability of supporting evidence may be used to modify the overall conclusion from that which would be reached on the basis of bioassay and/or epidemiological evidence alone. The overall evaluation is expressed as a numerical grouping, the categories of which are described below, as before by directly quoting IARC (2006):

"Group 1: The agent is *carcinogenic to humans*.

This category is used when there is *sufficient evidence of carcinogenicity* in humans. Exceptionally, an agent may be placed in this category when evidence of carcinogenicity in humans is less than *sufficient* but there is *sufficient evidence of carcinogenicity* in experimental animals and strong evidence in exposed humans that the agent acts through a relevant mechanism of carcinogenicity.

Group 2.

This category includes agents for which, at one extreme, the degree of evidence of carcinogenicity in humans is almost *sufficient*, as well as those for which, at the other extreme, there are no human data but for which there is evidence of carcinogenicity in experimental animals. Agents are assigned to either Group 2A (*probably carcinogenic to humans*) or Group 2B (*possibly carcinogenic to humans*) on the basis of epidemiological and experimental evidence of carcinogenicity and mechanistic and other relevant data. The terms *probably carcinogenic* and *possibly carcinogenic* have no quantitative significance and are used simply as descriptors of different levels of evidence of human

carcinogenicity, with *probably carcinogenic* signifying a higher level of evidence than *possibly carcinogenic*.

Group 2A: The agent is *probably carcinogenic to humans*.

This category is used when there is *limited evidence of carcinogenicity* in humans and *sufficient evidence of carcinogenicity* in experimental animals. In some cases, an agent may be classified in this category when there is *inadequate evidence of carcinogenicity* in humans and *sufficient evidence of carcinogenicity* in experimental animals and strong evidence that the carcinogenesis is mediated by a mechanism that also operates in humans. Exceptionally, an agent may be classified in this category solely on the basis of *limited evidence of carcinogenicity* in humans. An agent may be assigned to this category if it clearly belongs, based on mechanistic considerations, to a class of agents for which one or more members have been classified in Group 1 or Group 2A.

Group 2B: The agent is *possibly carcinogenic to humans*.

This category is used for agents for which there is *limited evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals. It may also be used when there is *inadequate evidence of carcinogenicity* in humans but there is *sufficient evidence of carcinogenicity* in experimental animals. In some instances, an agent for which there is *inadequate evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals together with supporting evidence from mechanistic and other relevant data may be placed in this group. An agent may be classified in this category solely on the basis of strong evidence from mechanistic and other relevant data.

Group 3: The agent is *not classifiable as to its carcinogenicity to humans*.

This category is used most commonly for agents for which the evidence of carcinogenicity is *inadequate* in humans and *inadequate* or *limited* in experimental animals.

Exceptionally, agents for which the evidence of carcinogenicity is *inadequate* in humans but *sufficient* in experimental animals may be placed in this category when there is strong evidence that the mechanism of carcinogenicity in experimental animals does not operate in humans.

Agents that do not fall into any other group are also placed in this category.

An evaluation in Group 3 is not a determination of non-carcinogenicity or overall safety. It often means that further research is needed, especially when exposures are widespread or the cancer data are consistent with differing interpretations.

Group 4: The agent is *probably not carcinogenic to humans*.

This category is used for agents for which there is *evidence suggesting lack of carcinogenicity* in humans and in experimental animals. In some instances, agents for

> which there is *inadequate evidence of carcinogenicity* in humans but *evidence suggesting lack of carcinogenicity* in experimental animals, consistently and strongly supported by a broad range of mechanistic and other relevant data, may be classified in this group."

The IARC hazard evaluation system provides a detailed and generally accepted scheme to classify the strength of evidence as to the possible human carcinogenicity of chemicals and other agents. This includes careful consideration of mechanistic data and other supporting evidence, the evaluation of which is also important to inform selection of models or defaults used in dose response assessment, as is described below. The extended consideration of supporting evidence is in fact the primary difference between more recent versions of the guidance from IARC, and also by other organizations including U.S. EPA, and the original versions of that guidance. In fact, the basic criteria for hazard identification based on bioassay and epidemiological data have not changed substantially in other respects from earlier guidance documents, including that originally published by California (DHS, 1985). Although as noted earlier the California Air Toxics programs do not categorize identified carcinogens, it has generally been the practice to regard any agent with an IARC overall classification in Group 1 or Group 2 as a known or potential human carcinogen. This implies the selection of various policy-based default options, including absence of a threshold in the dose-response curve, unless specific data are available to indicate otherwise. The same basic identification criteria are used by OEHHA scientific staff to determine the appropriate treatment of agents not evaluated by IARC, or for which newer data or revised interpretations suggest that an earlier IARC determination is no longer appropriate.

U.S. EPA has also proposed a scheme for carcinogen hazard identification and strength of evidence classification in their recently finalized Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005). These principally differ from the IARC guidance in recommending a more extensive narrative description rather than simply a numerical identifier for the identified level of evidence, and also to some degree in the weight accorded to various types of supporting evidence. However, for most purposes they may be regarded as broadly equivalent to the scheme used by IARC, and OEHHA has chosen to cite the IARC (2006) *Preamble* as representing the most up-to-date and generally accepted guidance on this issue.

Dose Response Assessment

The dose-response phase of a cancer risk assessment aims to characterize the relationship between an applied dose of a carcinogen and the risk of tumor appearance in a human. This is usually expressed as a cancer slope factor ["potency" – in units of reciprocal dose - usually $(mg/kg\text{-}body\ weight.day)^{-1}$ or "unit risk" – reciprocal air concentration – usually $(\mu g/m^3)^{-1}$] for the lifetime tumor risk associated with lifetime continuous exposure to the carcinogen at low doses. Cancer potency factors may also be referred to as "cancer slope factors". (As will be described later, additional algorithms may need to be applied to determine risk for specific age groups, or at higher doses where toxicokinetic factors have significant effect.) The basic methodologies recommended in this document are similar to those described by U.S. EPA (2005a) in their Carcinogen Risk Assessment Guidelines. This document therefore refers to U.S. EPA (2005a) for explanation of detailed procedures, and will provide only a brief summary except in cases where OEHHA recommendations are different from or more explicit than those of U.S. EPA.

The following descriptions of methods for dose response assessment, and considerations in their application, apply in principle to the analysis of both animal and human (epidemiological) cancer incidence data. Indeed, the original formulation of the multistage model (Armitage and Doll, 1954) described below was developed based on human cancer incidence. Nevertheless, the number and quality of human cancer incidence datasets are limited. The more complex analyses have usually only been possible for animal experimental data, where the interindividual variability and the exposure conditions can be both measured and controlled. Most commonly, epidemiological studies have necessarily used a form of multivariate analysis to separate the effects of several different variables relating to exposure, demographics and behaviors (*e.g.*, smoking). In these analyses it is usually assumed that the effect measure(s) vary linearly with the exposure: any more complex variance assumptions might exceed the power of the data to determine the required model parameters. However, there are exceptions, especially for occupational studies where the critical exposure is measured as a continuous variable (rather than just categorical) and where the effect of this exposure is substantial relative to other confounding factors. For example, OEHHA (1998) used a multistage model dealing with both exposure intensity and duration in the analysis of cancer incidence in railroad workers exposure to diesel exhaust (Garshick et al., 1988)

Interspecies Extrapolation

The procedures used to extrapolate low-dose human cancer risk from epidemiological or animal carcinogenicity data are generally health-protective in that they determine an upper confidence bound on the risk experienced by an exposed population. As statistical estimates they cannot be regarded as definite predictions of the risk faced by any one specific individual, who might for a variety of reasons, including individual exposure and susceptibility, experience a risk different from the estimate. The risk assessment procedures used aim to include the majority of variability in the general human population within the confidence bounds of the estimate, although the possibility that some individuals might experience either lower or even no risk, or a considerably higher risk, cannot be excluded. Additionally, differences may exist between the characteristics of the general public and those of studied populations. For example, healthy workers, the subject of most epidemiological studies, are often found to have lower rates of morbidity and mortality than the general population (Wen et al., 1983; Monson, 1986; Rothman and Greenland, 1998). Most human data are derived from studies of largely male adult workers and risk estimates cannot take into account specific physiological factors of women, children, and older populations that may affect the potency of a carcinogen, including early age-at-exposure.

Dose-response assessment based on environmental epidemiological studies may involve evaluation of health impacts at exposure levels within the range of those measured in the study population. However, more usually the source data are studies of occupationally exposed humans or of animals, in which case the exposures in the study are likely to be much higher than those of concern for risk assessments relating to community or ambient exposures. Further, even when extrapolation from animal species to humans is not required, the general population to which the URF is applied may differ in characteristics relative to the occupational population studied. It is therefore necessary to extrapolate from the available data to the population and exposure range of concern, which is done by using a dose-response model derived from the source data. The models used fall into three main classes: mechanistically based models, empirical models and (where data are lacking to support a true data-based model) default

assumptions. The factors affecting the dose-response relationships for carcinogenesis may also be divided into those relating to absorption, distribution, metabolism and excretion on the one hand (*i.e.* toxicokinetics), and those relating to the underlying dose-response characteristics of carcinogenesis at the tissue or cellular level (*i.e.* toxicodynamics). In this sense the problem of dose response assessment for carcinogens is similar to that for non-cancer toxic effects. The toxicokinetic models used may in fact be similar for both situations, but the toxicodynamic models are generally different.

Intraspecies Extrapolation and Inter-individual Variability

In estimating the impact of a particular level of exposure to a carcinogen on a target human population, it is necessary to consider the range of susceptibility in the target population. In the present case this is typically defined as the general population of the State of California, including of course women (some of whom are pregnant), infants and children, the elderly, the sick, and those with genetic polymorphisms or acquired differences which affect their susceptibility to carcinogens. In general it has been assumed that the upper-bound risk estimates obtained from the standard toxicodynamic models described below are sufficiently health-protective to cover the intrinsic variability of the adult human target population, in spite of the fact that these models do not explicitly address this type of variability, except in the few cases where an estimate is based on epidemiological data from a large and unselected study group (U.S. EPA, 2005a). However, various analyses (Drew et al., 1983; Barton et al., 2005; Appendix J) have suggested that this assumption is inadequate to cover the expected variability within a human population that includes infants and children. Accordingly both U.S. EPA (2005b) and this document now offer guidance on the use of age-specific adjustment factors to allow for the potentially greater sensitivity of infants and children to chemical carcinogenesis.

The ability to accommodate human variability with regard to the toxicokinetic factors affecting susceptibility to carcinogens varies with the level of detail used in the particular assessment. If the generic interspecies extrapolation approach based on body weight is used without any explicit toxicokinetic model, then the assumption is made, as in the case of toxicodynamic variability, that the overall health-protective assumptions made are sufficient to cover the toxicokinetic variability. On the other hand if explicit models such as those referenced in the following paragraph are used, this variability may be more explicitly accommodated by using parameter values which are taken as point estimates from measured distributions of population values, or by using Monte Carlo techniques to include those distributions in the model (Bois et al., 1996; OEHHA, 1992; 2001b).

Toxicokinetic Models

Considerable literature exists showing the importance of understanding the toxicokinetics of carcinogens in understanding their mechanism of action, sites of impact and dose-response relationships. U.S. EPA (2005) in Section 3.1 refers to the importance of identifying an appropriate dose metric for the dose-response analysis. Early cancer risk assessments typically used applied dose as the dose metric, which is adequate in simple cases provided appropriate correction factors are applied for interspecies extrapolation. However, it is often observed that the uptake, metabolism and elimination of the carcinogenic substance (and/or a procarcinogen and metabolites) is non-linear, especially at the higher doses employed in experimental animal

studies (Hoel *et al*., 1983, Gaylor *et al*., 1994). Extrapolation to lower doses where such relationships tend to linearity (Hattis, 1990) is aided by the use of toxicokinetic models. These may be relatively simple compartment models, or sophisticated "physiologically based pharmacokinetic (PBPK) models" which to a greater or lesser degree model the actual biochemical and physiological events of toxicokinetic importance. Applications of both types of model may be found in various risk assessment documents prepared for the Toxic Air Contaminants program (and other OEHHA risk assessments). Since the details vary widely according to the nature of the chemical and the availability of appropriate kinetic data these general guidelines will defer to those examples rather than attempt a fuller exposition here. Further analysis of the use of toxicokinetic modeling in extrapolation from animals to humans, and in accounting for interindividual variability among adult humans, infants and children is presented in the Air Toxics Hot Spots *Technical Support Document for the Derivation of Noncancer Reference Exposure Levels* (OEHHA, 2008). Although this refers to the use of toxicokinetic modeling in non-cancer risk assessment, the primary considerations are similar for cancer risk assessment.

Toxicodynamic Models

An early use of mechanistic analysis to support risk assessment was the development of the Armitage-Doll multistage model of dose-response for carcinogenesis. The multistage model was initially developed on theoretical grounds, and by examination of epidemiological and animal data on time to tumor incidence. Subsequent discovery of the molecular biology of proto-oncogenes has provided a basis for explaining the model in terms of actual biological events and systems (Barrett and Wiseman, 1987). This model was developed by Crump and others into the "linearized multistage model", which has been extensively used for carcinogen risk assessment. It leads to a number of partially verifiable predictions, including linearity of the dose-response relationship at low doses, which is observed for many genotoxic carcinogens. It also predicts the form of the dose-response relationship at higher doses, which generally follow a polynomial form (subject to sampling and background corrections) except where other identifiable factors such as pharmacokinetics intervene.

It has been argued that the simple linearized form of the multistage model has limitations as a description of carcinogenic mechanisms, which detract from its usefulness and generality. Cell proliferation is known to be important in the progression of cancer. It may actually be the primary mechanism of action for a few carcinogens, as opposed to the direct modification of DNA by the carcinogen or a metabolite which is assumed to cause the mutational event at each stage in the original multistage description. A cell proliferation model has been developed (Moolgavkar and Knudson, 1981), which retains the concept of an initiating mutational event (in most cases caused by interaction of the chemical with DNA, although it could also be a spontaneous mutation) as in the original multistage model, but also considers proliferation, death or terminal differentiation of both normal and initiated cells. This model is thought to better describe the biological events in carcinogenesis. However, it has not been used extensively in risk assessment because it requires many parameters that are difficult to define and measure (such as proliferation and death rates for various classes of cell). If these cannot be accurately determined, the model has too many free parameters and is not helpful in defining extrapolated values for risk assessment purposes. This highlights a general problem in using mechanistic models in carcinogen risk assessment, which is that the carcinogenesis data themselves are

generally insufficient to define fully the dose response curve shape at low doses or provide much mechanistic information. The analysis is therefore supplemented with policy-based assumptions (such as the expectation of linearity at low doses) and, wherever possible, additional experimental measurements relating to the mechanism of action, in order to make meaningful prediction of risk from environmental exposures to humans.

Because of the difficulties in validating simplified mechanistic models such as the basic multistage model, and the additional difficulty of parameter estimation with more complex mechanistic models, the new U.S. EPA guidelines (U.S. EPA, 2005a) and some recent California risk assessments have chosen instead to use a less overtly mechanistic approach. This approach combines benchmark dose methodology (described below) with an explicit choice of the method for low-dose extrapolation, either assuming low-dose linearity or, for certain carcinogens where data indicate that this is appropriate, a "margin of exposure" or safety/uncertainty factor based approach. This benchmark method is now normally recommended for carcinogen dose response analysis, and the results generally differ little from those derived by the linearized multistage model. Although the linearized multistage method is no longer recommended as the default approach for cancer potency estimation it remains a plausible alternative in many cases, and still has useful applications, such as for time-to-tumor analyses for which benchmark methods are not yet widely available. Additionally, a considerable number of existing cancer potencies in Appendices A and B, and used in the Air Toxics Hot Spots program were derived by this method. Many of these would not be significantly different if calculated by the benchmark approach, and are unlikely to be replaced soon by newly calculated values. The linearized multistage method will therefore also be briefly described here.

Benchmark Dose Methodologies

The use of benchmark dose methodology has been explored by various investigators [including Gaylor et al. (1998); van Landingham et al. (2001) and Crump (1984, 1995, 2002)] as a tool for dose response extrapolation. This has been recommended in regulatory guidelines for both carcinogenic (U.S. EPA, 2005a) and non-carcinogenic (U.S. EPA, 1995) endpoints. The basic approach is to fit an arbitrary function to the observed incidence data, and to select a "point of departure" (POD) (benchmark dose) *within the range of the observed data.* From this a low dose risk estimate or assumed safe level may be obtained by extrapolation, using an assumed function (usually linear) or by application of uncertainty factors. The critical issue here is that no assumptions are made about the nature of the underlying process in fitting the data. The assumptions about the shape of the dose response curve (linear, threshold, etc.) are explicitly confined to the second step of the estimation process, and are chosen on the basis of policy, mechanistic evidence or other supporting considerations. The benchmark chosen is a point at the low end of the observable dose-response curve. Usually a dose at which the incidence of the tumor is 10% is chosen for animal studies, although lower effect levels may be appropriate for large epidemiological data sets. Because real experimental data include variability in the response of individual subjects, and measurement errors, likelihood methodology is applied in fitting the data. A lower confidence bound (usually 95%) of the effective dose (LED_{10}), rather than its maximum likelihood estimate (MLE), is used as the point of departure. This properly reflects the uncertainty in the estimate, taking a cautious interpretation of highly variable or error-prone data. It also reflects the instability of MLE values from complex curve-fitting routines, which has been recognized as a problem also with the linearized multistage model.

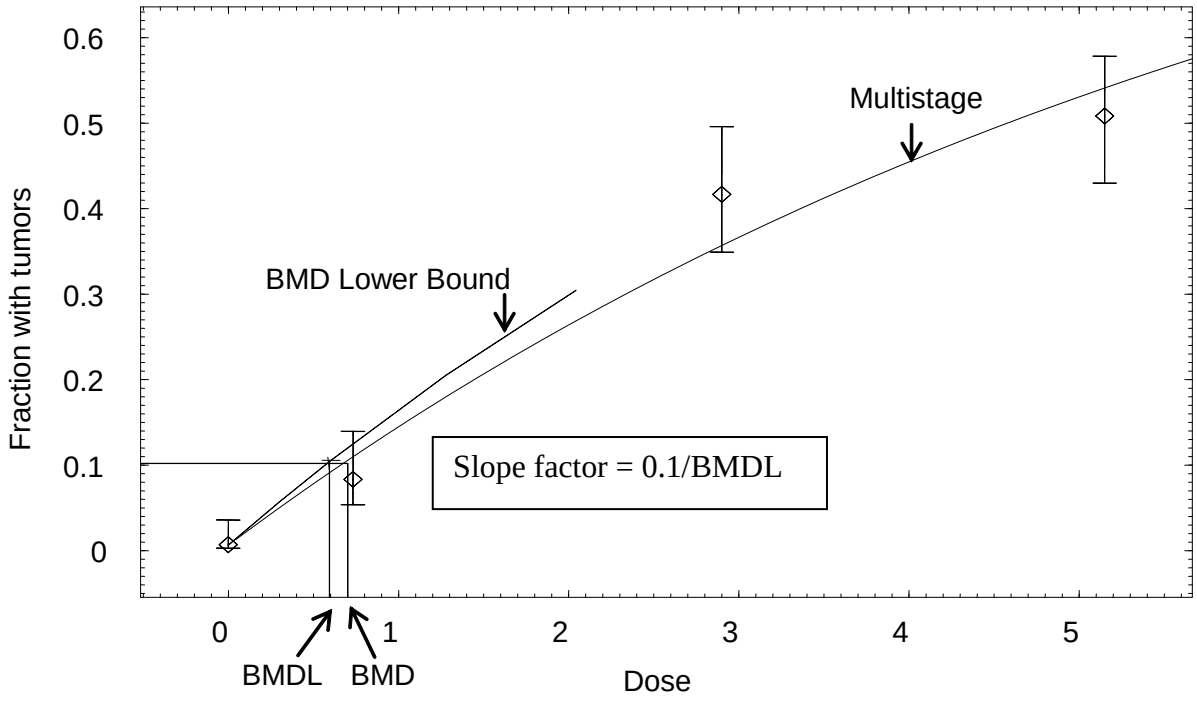
For cancer dose-response estimation using the benchmark dose method, either animal bioassay data or epidemiological data provide a suitable basis. In the absence of a pharmacokinetic model (which could provide tissue-specific dose metrics), the potency would ordinarily be based on the time-weighted average exposure during the exposure or dosing period. The model used to fit the data can be chosen from a range of available alternative quantal models, depending on which provides the best fit to the data in the observable range. In practice, the multistage polynomial fit developed for the linearized multistage model works well for most tumor data sets. Here it is being used merely as a mathematical curve-fitting tool, where the model well fits the data set, without making assumptions about its validity as a biological model of carcinogenesis.

Suitable polynomial fits and estimates of the benchmark may be obtained using U.S. EPA's BMDS software. The benchmark often used is the 95% lower confidence bound on the dose producing 10% tumor incidence. However, if data are available which include a significant dose-response at less than 10% tumor incidence, then that lower benchmark should be used (*e.g.*, LED_{05} or LED_{01}). Other software such as Tox_Risk, which was used for the linearized multistage model, has been used successfully, although the earlier GLOBAL program and its relatives are less suitable as curve-fitting tools for benchmark dose analysis.

Since it is usually assumed in cancer risk estimation that the low-dose response relationship is linear, risk estimates and a potency value (slope factor) may be obtained by linear extrapolation from an appropriate benchmark dose. The potency is the slope of that line ($0.1/LED_{10}$). The low dose linearity assumption is a general default for any carcinogen, and it is unlikely to be altered for genotoxic carcinogens.

A calculation using the benchmark dose approach (using a polynomial model with exponents restricted to zero or positive values), and linear extrapolation from the LED_{10} to obtain a potency estimate is shown in Figure 1 (the figure was generated by the U.S. EPA's BMDS program). This is based on tumor incidence data from an actual experiment with vinyl bromide in rats (Benya *et al*., 1982), with metabolized dose calculated by means of a pharmacokinetic model (Salmon *et al.*, 1992). The value of q_1* obtained by this calculation would then be corrected for the duration of the experiment if it had lasted for less than the standard rat lifetime, and for bodyweight and route-specific pharmacokinetic factors as described below. This is in addition to the correction for exposure duration that would be necessary if the study had not lasted for 105 weeks, and the interspecies correction, both of which are described below.

Figure 1. Benchmark dose calculation for tumor data in rats exposed to vinyl bromide



From Salmon *et al*. (1992), based on data from Benya *et al*. (1982)

Linearized Multistage Model

Quantal Analyses

A "multistage" polynomial (U.S. EPA, 1986, 2005a; Anderson *et al.*, 1983), based on the mechanistic insights of the original Armitage and Doll model of cancer induction and progression, has been used extensively by U.S. EPA, OEHHA and other risk assessors to model the dose response for lifetime risk of cancer. It usually is used for analysis of animal bioassay data, although related approaches have occasionally been used with epidemiological data. In mathematical terms, the probability of dying with a tumor (P) induced by an average daily dose (d) is:

$$P(d) = 1 - \exp[-(q_0 + q_1 d + q_2 d^2 + ... + q_j d^j)]$$

with constraints

$$q_i \geq 0 \text{ for all i.}$$

Equivalently, $A(d) = 1 - \exp [- (q_1 d + q_2 d^2 + \cdots + q_k d^k)]$,

where $A(d) = \dfrac{P(d) - P(0)}{1 - P(0)}$ is the extra risk over background at dose d.

The q_i model parameters are constants that can be estimated by fitting the polynomial to the data from the bioassay, *i.e.* the number of tumor bearing animals (as a fraction of the total at risk) at each dose level, including the controls. The fit is optimized using likelihood methodology, assuming that the deviations from expected values follow a χ^2 distribution, with the number of degrees of freedom (and hence the maximum number of terms allowed in the polynomial) determined by the number of points in the data set. All the coefficients of the terms are constrained to be zero or positive, so the curve is required to be straight or upward curving, with no maxima, minima or other points of inflection. In addition to the maximum likelihood estimates of the parameters, the upper 95% confidence limits on these parameters are calculated.

The parameter q_0 represents the background lifetime incidence of the tumor. The 95% upper confidence limit of the slope factor q_1 (q_1^*), is termed the cancer potency. The maximum likelihood estimate (MLE) of q_1 is not usually regarded as a reliable estimate for several reasons. First, it fails to reflect the uncertainty and variability in the data which affect the value of the estimate. This is an important issue for protection of public health, which is emphasized by current regulatory guidelines. Secondly, due to the variable order of the polynomial and the effect of some terms being zero as opposed to having a small but finite value, the MLE is unstable, and may show large and unpredictable changes in response to very slight changes in the input data. It may also erratically have a zero value, even when the data imply a significant positive dose-response relationship. The MLE is not a measure of central tendency for this estimate distribution (which is always asymmetrical and often multi-peaked). For small doses, the cancer potency is the ratio of excess lifetime cancer risk to the average daily dose received. Details of the estimation procedure are given in Crump (1981) and Crump, Guess, and Deal (1977). Several software programs are available to perform the necessary calculations, including U.S. EPA's BMDS, Tox_Risk and the earlier GLOBAL programs by Crump and colleagues, and Mstage, written by Crouch (1987).

When dose is expressed in units of mg/kg-d, the potency is given in units of $(\text{mg/kg-d})^{-1}$. Likewise, when the model input is in units of concentration ($\mu g/m^3$, ppb), the potency is given in units of $\mu g/m^3)^{-1}$ pr $(\text{ppb})^{-1}$. As in the case of potencies obtained by the benchmark approach, the experiment-based potency value needs to be corrected for less-than lifetime or intermittent exposure, and extrapolated from the test species to humans. Risk calculations using potency value estimated using the linearized multistage model predict the cancer risk at low doses only, with the higher order terms of the fitted polynomial being ignored since their contribution is negligible at low doses.

Selection of Site and Tumor Type

In developing cancer potency estimates from animal data, standard practice has been to use dose-response data for the most sensitive tumor site as the basis of the estimate (CDHS, 1985). Where tumors of more than one histological type (*e.g.*, adenomas and carcinomas) are observed at a single site, the combined incidence, *i.e.* proportion of animals affected with at least one tumor of any of the relevant types, is used for dose-response assessment. The same rules for combining

tumor types are generally applied in determining statistical significance for carcinogen identification (IARC, 2006). Tumor types considered to represent different stages of progression following initiation of a common original normal cell type are combined, whereas tumor types having different cellular origins are generally not combined by this procedure. Other considerations that may influence choice of site for dose response estimation include the quality of the data (especially, the statistical impact of a high or variable rate of a particular tumor type and site in control animals), and biological relevance to humans. However, it is an important principle that, just as for the hazard identification phase, concordance of site or tumor type between animal models and human health effects may occur but is not assumed or required.

Carcinogens Inducing Tumors at Multiple Sites

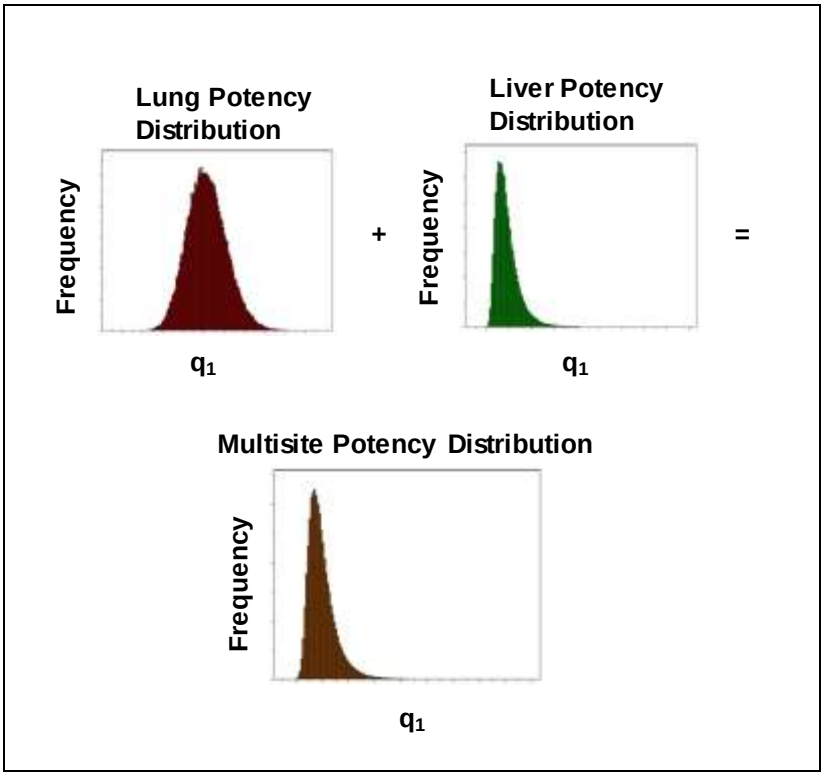
For most carcinogens, the selection of the most sensitive site in the animal studies is recognized as providing a risk estimate which is appropriate to protect human health. However, for chemicals that induce tumors at multiple sites, the single-site approach may underestimate the true carcinogenic potential. For example, the overall assessment of cancer risk from cigarette smoking (U.S. DHHS, 1982) or ionizing radiation (NRC, 1990) is not based on risk at one site, such as lung cancer. Instead, total cancer risk is estimated from all the sites at which agent-induced tumors are observed (lung, bladder, leukemia, etc), combined.

For carcinogens that induce tumors at multiple sites and/or with different cell types in a particular species and sex, OEHHA derives the animal cancer potency by probabilistically summing the potencies from the different sites and/or cell types. Using the combined potency distribution takes into account the multisite tumorigenicity and provides a basis for estimating the cumulative risk of all treatment-related tumors.

The linear term (q_1) of either the multistage model or the multistage-in-dose, Weibull-in-time model is first estimated based on the dose-response data for each of the treatment-related tumor sites. Statistical distributions, rather than point estimates, are generated at each site by tracing the profile likelihood of the linear term (q_1) (Zeise et al., 1991). The distributions of q_1 for each of the treatment-related sites are then statistically summed using a Monte Carlo approach and assuming independence (Figure 2). The sum is created by adding the linear term for each tumor site, according to its distribution, through random sampling. The upper 95 percent confidence limit on the summed distribution is taken as the multisite animal cancer potency estimate (McDonald et al., 2003, McDonald and Komulainen, 2005).

OEHHA has applied this approach in several recent dose-response analyses, including that for naphthalene presented in Appendix B of this document.

Figure 2. Addition of potency distributions for multi-site cancer potency derivations.



Early-Lifestage Cancer Potency Adjustments

In recent years, there have been growing concerns regarding the exposure of children to environmental chemicals, including the possibility that they may be more susceptible than adults to injury caused by those chemicals. The California Legislature passed the Children's Environmental Health Protection Act (Senate Bill 25, Escutia; Chapter 731, Statutes of 1999; "SB 25") to help address these concerns. Under SB25, OEHHA is mandated to consider infants and children specifically, where data permit, in evaluating the health effects of Toxic Air Contaminants (TACs).

The development of cancer is one of the adverse health effects that may occur in children as a result of exposure to environmental chemicals. The document "Prioritization of Toxic Air Contaminants under the Children's Environmental Health Protection Act" (OEHHA, 2001a) noted that risks of cancer from exposures to carcinogens occurring from conception through puberty can be different than those from exposures occurring in adulthood. Exposure to a carcinogen early in life may result in a greater lifetime risk of cancer for several reasons:

1. Cancer is a multistage process and the occurrence of the first stages in childhood increases the chance that the entire process will be completed, and a cancer produced, within an individual's lifetime.
2. Tissues undergoing rapid growth and development may be especially vulnerable to carcinogenic agents. During periods of increased cell proliferation there is rapid turnover of DNA, and more opportunity for misrepair of damage (*e.g.*, DNA breaks, crosslinks, adducts) or alterations to result in permanent changes to the DNA (*e.g.*, mutations, altered DNA methylation) that may ultimately lead to cancer.
3. During early development, a greater proportion of the body's cells are relatively undifferentiated stem cells, and as such represent a large target population of somatic cells capable of passing along permanent changes to the DNA during future cell divisions.
4. There may be greater sensitivity to hormonal carcinogens early in life since the development of many organ systems is under hormonal control (*e.g.*, male and female reproductive systems, thyroid control of CNS development).
5. Other factors that may play a role in increased cancer risk from exposures during critical developmental periods include differences in immunological activity, intestinal absorption, biliary and kidney excretion, blood and fat distribution, and expression of enzyme systems that activate or detoxify carcinogens.

Data in humans and animals for a variety of carcinogens suggest that exposures to such carcinogens early in life may result in a greater lifetime risk of cancer compared to exposures later in life. Examples of this effect in humans are carcinogenicity due to ionizing radiation, diethylstilbestrol (DES), chemotherapeutic agents, and tobacco smoke.

Ionizing radiation exposure carries an increased risk of cancer when exposures occur early in life compared to adult exposures for a number of tumor types. Children exposed to ionizing radiation (diagnostic X-rays) *in utero* demonstrate a larger excess of leukemia cases than

children exposed to ionizing radiation postnatally (NRC, 1990). Exposure to radioisotopes (^{131}I, ^{137}Cs, ^{134}Cs, ^{90}Sr) as a consequence of the 1986 Chernobyl nuclear accident resulted in an elevated thyroid cancer incidence in children but not adults (Moysich, 2002). Treatment of children for Hodgkin's lymphoma with both chemotherapeutic agents and irradiation has been shown to increase the risk of secondary tumors (Swerdlow et al., 2000; Franklin et al., 2006). Age at irradiation in Hodgkin's disease patients treated with radiotherapy strongly influenced the risk of developing breast cancer. The relative risk (RR) of developing breast cancer was 136 for women treated before 15 years of age, 19 for women 15-24 years of age, and 7 for those 24-29 years of age. In women above 30 years of age, the risk was not increased (Hancock *et al*., 1993).

DES was administered to pregnant women in the 1940s-1960s for the purpose of preventing pregnancy loss. In 1970, Herbst and Scully described 7 cases of vaginal adenocarcinoma (6 cases of the clear-cell type) in women aged 15-22 years. This type of cancer is extremely rare in that age range. A follow-up epidemiological study included an additional case, and noted the fact that the mothers of 7 of the 8 patients had been treated with DES during their pregnancy (Herbst *et al*., 1971). Reports by other investigators confirmed the association between maternal use of DES during pregnancy and the development of vaginal adenocarcinoma in their female offspring (Preston-Martin, 1989). It was observed that *in utero* DES exposure resulted in female genital tract morphological changes which correlated with both dose and duration of exposure, and those changes were not related to the maternal conditions which were the reason for the DES administration. Additionally, the risk of occurrence of those morphological changes declined with increasing gestational age at first exposure (O'Brien *et al*., 1979; Preston-Martin, 1989). In contrast, vaginal adenocarcinoma incidence did not increase in the exposed mothers themselves, indicating an increased early-life susceptibility to the carcinogenic effects of DES.

There is evidence in the epidemiological literature indicating that exposure to tobacco smoke during puberty may increase risk of breast cancer later in life, particularly among women who are NAT2 slow deacetylators (Marcus *et al*., 2000; Morabia *et al*., 2000; Lash and Aschengrau, 1999). Wiencke *et al*. (1999) report that early age at initiation of smoking is associated with a higher level of DNA adducts in lung tissue of former-smokers with lung cancer.

It has also been observed by Smith *et al*. (2006) that human *in utero* or early childhood exposure to arsenic in drinking water results in significantly increased lung cancer incidences during adult life.

Data from animal studies provide additional examples of increased sensitivity to early life (typically postnatal and juvenile) exposures. These effects span a range of target tissues, including the liver (vinyl chloride, safrole), brain (methylnitrosourea), reproductive tract (DES, tamoxifen), and lung (urethane) (OEHHA, 2001a).

In the following sections we summarize two efforts to evaluate quantitatively the effect of lifestage at exposure on carcinogenic response in experimental animal studies. The first section provides a description of OEHHA's analysis of data on the effect of age at exposure on carcinogenic potency. (Details of this analysis are in Appendix J.) The second section describes U.S. EPA's work in this area. (We also provide the published paper in Appendix I that presents the U.S. EPA analyses.) Both analyses used extant data available in the published literature. U.S. EPA used their analysis to modify the procedures they have used to estimate cancer risk by

weighting risk by specific factors for childhood exposures. The weighting factors are a policy choice supported by U.S. EPA's data analysis. The results of OEHHA's analysis, summarized below and described in detail in Appendix J, support the decision to modify policy to weight risk when exposure occurs during childhood.

OEHHA Analysis of the Effect of Age at Exposure on Cancer Potency

The analysis of animal cancer studies which include early life exposure by the Reproductive and Cancer Hazard Assessment Branch (RCHAB) of OEHHA also supports the application of lifestage-specific cancer potency factor adjustments. This analysis is provided in detail as Appendix J of this document.

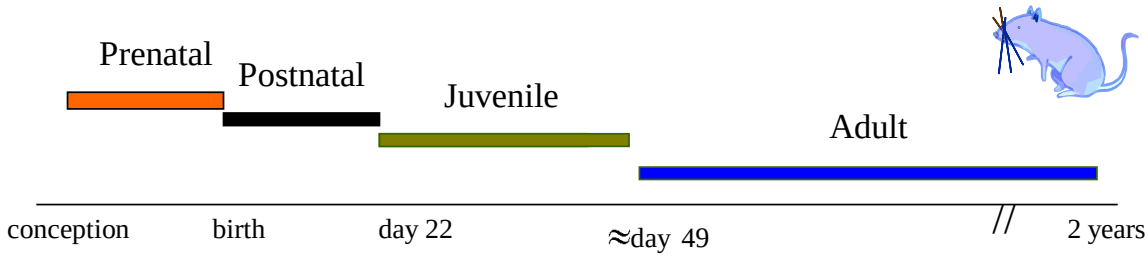
Early-in-life susceptibility to carcinogens has long been recognized by the scientific community and clinicians as a public health concern. Numerous scientific publications and symposia have addressed this issue over the years and the scientific literature contains a number of human clinical findings and epidemiological studies of early life cancer susceptibility. While there are many indications of increased human cancer susceptibility in early life, the magnitude of the impact has been difficult to gauge. Until recently risk assessment procedures have not in general addressed the issue. As described in the next section, in 2005 the U.S. EPA adopted an approach to weight carcinogens by age at exposure if they act via a mutagenic mode of action. The California legislature in 2000 directed OEHHA to assess methodologies used in addressing early-in-life risk, compile animal data to evaluate those methods, and develop methods to adequately address carcinogenic exposures to the fetus, infants, and children (Children's Environmental Health Initiative [AB 2872, Shelly]; California Health and Safety Code [HSC] section 901 [a] through [e]).

OEHHA assessed cancer risk assessment methodologies, and found that the existing risk assessment approaches did not adequately address the possibility that risk from early-in-life and adult exposures may differ. OEHHA further concluded that there was a need to address early-in-life cancer risk, and undertook studies to develop methods for doing so. Age-related cancer susceptibility data were identified from published animal cancer bioassays in which these issues were addressed. Two types of studies with early-in-life exposures were compiled. The first type are "multi-lifestage exposure studies." These studies have at least two groups exposed during different lifestages: One dose group is exposed to a chemical only during one of the following lifestages (Figure 3):

- prenatal (from conception to birth),
- postnatal (from birth to weaning),
- juvenile (from weaning to sexual maturity).

The second dose group is exposed for some period of time at an older age, preferably during the adult lifestage, that is, after sexual maturity. This group served as the reference group. In some cases where there was no adult exposure group, animals exposed as juveniles served as the reference group. Multi-lifestage exposure studies are available for many chemicals, enabling the exploration of patterns in early-life susceptibility across chemicals.

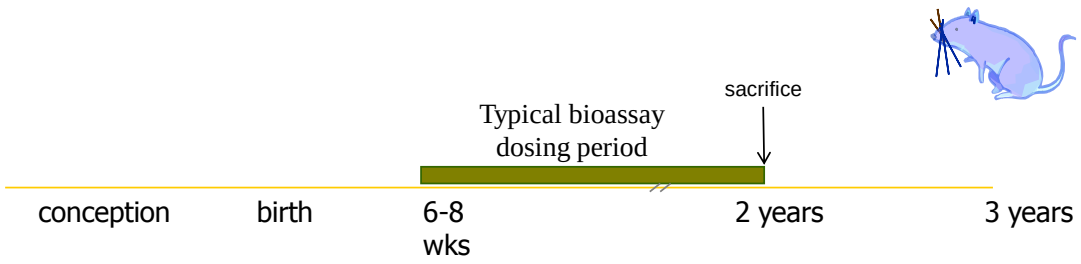
Figure 3. Definition of Rodent Lifestage Adopted in the OEHHA Analyses



OEHHA also conducted "chemical-specific case studies" of early-life sensitivity for two carcinogens, ethyl-N-nitrosoamine (DEN) and N-ethyl-N-nitrosourea (ENU) that combine data from a number of studies. These "chemical-specific case studies" were conducted to explore the feasibility of analyzing chemical-specific data on age susceptibility from single-lifestage exposure experiments. For these chemicals, OEHHA compiled from the literature a second type of study, "single-lifestage exposure experiments." In these experiments dose groups were exposed only during a particular lifestage and, unlike the "multi-lifestage exposure studies," there was no requirement that the same study also include groups exposed during a different lifestage. Thus, single-lifestage exposure experiments were identified as being either prenatal, postnatal, juvenile, or adult exposure studies. For each of the two chemicals, there were many prenatal studies conducted that were compiled, analyzed, and grouped together. Postnatal studies from different publications were similarly compiled, analyzed and grouped together, as were juvenile studies. Adult studies were not available for either DEN or ENU, thus for both chemicals juvenile exposure studies served as the referent for prenatal studies, and for postnatal studies.

Typical cancer bioassays such as those conducted in rats and mice by NTP involve exposing animals starting at six to eight weeks of age, which is the time at which these animals reach sexual maturity (late teenagers relative to humans). The experiments are run for two years, ending when the animal is in late middle age. Thus, early and very late life exposures are not included in the typical rodent bioassay (see Figure 4). If the NTP bioassay is used as a basis for estimating cancer potency, the potency and resulting risk estimates may be too low. Thus OEHHA focused on finding studies that evaluated early in life exposures.

Figure 4. Dosing Period for Typical Rodent Bioassays.



Since bioassays examining the effect of age at exposure on carcinogenesis were conducted by various investigators for different purposes, there is a great deal of variation across studies in terms of dose selection, duration of exposure, number of animals, and length of study duration. To be included in the compilation of studies with early life exposure, a study or an experimental group in a study had to meet minimum requirements.

The criteria for study inclusion are as follows:

- Treated groups were exposed to a single chemical carcinogen or a single carcinogenic chemical mixture.
- Study groups were not compromised by severe treatment-related non-cancer toxicity.
- Overall the duration of exposure period plus observation period exceeded 40 weeks, unless animals died of tumor.
- For included dose groups, the study must report age at dosing, age at sacrifice, and site-specific tumor incidence.
- Each lifestage exposure treatment group has an appropriate concurrent control group, or, for rare tumors only, an appropriate historical control.
- The studies were on mammals.
- Each treatment and control group consists of at least ten animals, unless the conduct and design of the study was well done in all other aspects (*e.g.*, the length of the study was sufficiently long to observe treatment-related tumors) and tumor incidence was high in treated groups and very low in controls.
- Site specific tumor data were reported, not only total number of tumor bearing animals.
- The test compound was administered in the diet, water, via gavage, or by intraperitoneal (i.p.), intravenous (i.v.), or subcutaneous (s.c.) injection. For dermal and subcutaneous injection studies, distal tumor findings are utilized (for dermal, other than skin tumors; for injection, non-injection site tumors).

- While studies designed to histopathologically examine tumors at multiple sites were preferred, studies that examined only a select set of organ/tissue sites were not excluded if the sites examined were known with confidence to be the only target tissues for the chemical and lifestage in question in that particular strain of animal.

Different approaches were taken to identify animal cancer studies that included groups of animals exposed during early life stages. First, MEDLINE and TOXLINE (National Library of Medicine) databases were searched using combinations of various key words for cancer (*e.g.*, tumor(s), neoplasm(s), cancer, neoplasia, cancerous, neoplasms-chemically induced) and for early-life exposure (*e.g.*, age, age-at-exposure, development (al), prenatal, *in utero*, gestation (al), postnatal, neonatal, juvenile, weaning, weanling, adolescent, adolescence, young). Second, the extensive compilation of bioassays in the *Survey of Compounds which have been Tested for Carcinogenic Activity*, was reviewed. This survey, formerly maintained by the National Cancer Institute as Public Health Service Publication Number 149, or PHS 149, is now available from a private source electronically as CancerChem, 2000. Third, from bibliographies from relevant published papers additional studies were identified. Finally the Single Dose Database developed by Calabrese and Blain (1999) was obtained and utilized to identify additional publications that appeared to contain potentially useful data. All of these publications were evaluated to determine if the study dosed separate groups of animals early in life and at or near adulthood. A total of 145 publications, providing data on 84 chemicals, were identified as meeting the criteria for study inclusion. A subset of these met the criteria for inclusion in the multi-lifestage exposure analysis.

Finally, for the OEHHA multi-lifestage analyses, we define "experiment" as a study component consisting of a control group as well as a treated group(s) exposed during the same lifestage (*i.e.*, prenatal, postnatal, juvenile or adult), and using the same experimental protocol (*e.g.*, route of exposure, strain, species, laboratory). Thus, by our definition one publication may report multiple experiments.

In the OEHHA analysis, data from studies on 23 unique carcinogens, 20 of which are considered to act via primarily genotoxic modes of action, were analyzed. Of these 20 carcinogens, 15 are thought to require metabolic activation to the ultimate carcinogenic species (Table 1). Fourteen carcinogens, including one thought to act via primarily nongenotoxic modes of action, were included in the prenatal multi-lifestage exposure studies. Eighteen carcinogens, including two thought to act via primarily nongenotoxic modes of action, were included in the postnatal multi-lifestage exposure studies. Five carcinogens were included in the juvenile multi-lifestage exposure studies. The case study chemicals, DEN and ENU, are both genotoxic. ENU is a direct acting alkylating agent, while DEN requires metabolic activation.

Table 1. Carcinogens for which studies with multi-lifestage exposures in animal studies are available

Genotoxic carcinogens requiring metabolic activation
Benzidine
Benzo[a]pyrene
Dibutylnitrosamine
Diethylnitrosamine (DEN)
7,12-Dimethylbenz[a]anthracene (DMBA)
Dimethylnitrosamine (DMN)
Di-n-propylnitrosamine (DPN)
1 -Ethyl-nitrosobiuret
2-Hydroxypropylnitrosamine
3-Hydroxyxanthine
3-Methylcholanthrene (3-MC)
4-(Methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK)
Safrole
Urethane
Vinyl chloride
Genotoxic carcinogens not requiring metabolic activation
Butylnitrosourea
1,2-Dimethylhydrazine
Ethylnitrosourea (ENU)
Methylnitrosourea (MNU)
ß-Propiolactone
Nongenotoxic carcinogens
1,1-Bis(p-chlorophenol)-2,2,2-trichloroethane (DDT)
Diethylstilbestrol (DES)
2,3,7,8-Tetrachlorodibenzodioxin (TCDD)

Cancer Potency Estimation

Statistical methods were developed and used to analyze the data and derive measures of early-life susceptibility. These are described in detail in Appendix J. In brief, a cancer potency (the slope of the dose response curve) was developed for each of the experiments selected using the linearized multistage model. This model was chosen because of widespread use in risk assessment, and its flexibility in being able to fit many different data sets needed to evaluate the effect of lifestage-at-exposure on cancer potency. The dose metric used for the potency analyses is cumulative dose normalized to body weight. The cancer potency is thus expressed as the increase in tumor probability with increasing cumulative dose in units of mg/kg body weight.

To take into account uncertainty in potency estimation, cancer potencies are depicted by a statistical distribution, rather than by a single, fixed value, using methods described in Appendix J. While these methods have typically been used to obtain and report the 95th percentile of the cancer slope parameter for cancer risk assessment purposes, here OEHHA utilized the full distribution of the cancer slope parameter to derive measures of early-life susceptibility to carcinogens. This was done to systematically take into account uncertainty in the analysis.

For experiments where treatment related tumors were observed at multiple sites or at the same site but arising from different cell types, slopes from these sites were statistically combined by summing across the potency distributions (assuming independence across the sites that were observed) to create an overall multisite cancer potency. It is not uncommon that a carcinogen causes more than one type of cancer or causes tumors at different sites depending on lifestage at exposure. For example, in humans tobacco smoke causes cancers of the lung, bladder, and certain other organs. This multi-site carcinogenicity is frequently observed in animal experiments as well. In order to account for this, all treatment-related tumors that were observed in a given lifestage were taken into account in estimating cancer potency from that particular experiment.

Addressing Early-Age Sensitivity in Estimating Cancer Risk: Age Sensitivity Factors

Inherent Sensitivity of Lifestages – Lifestage Potency Ratios

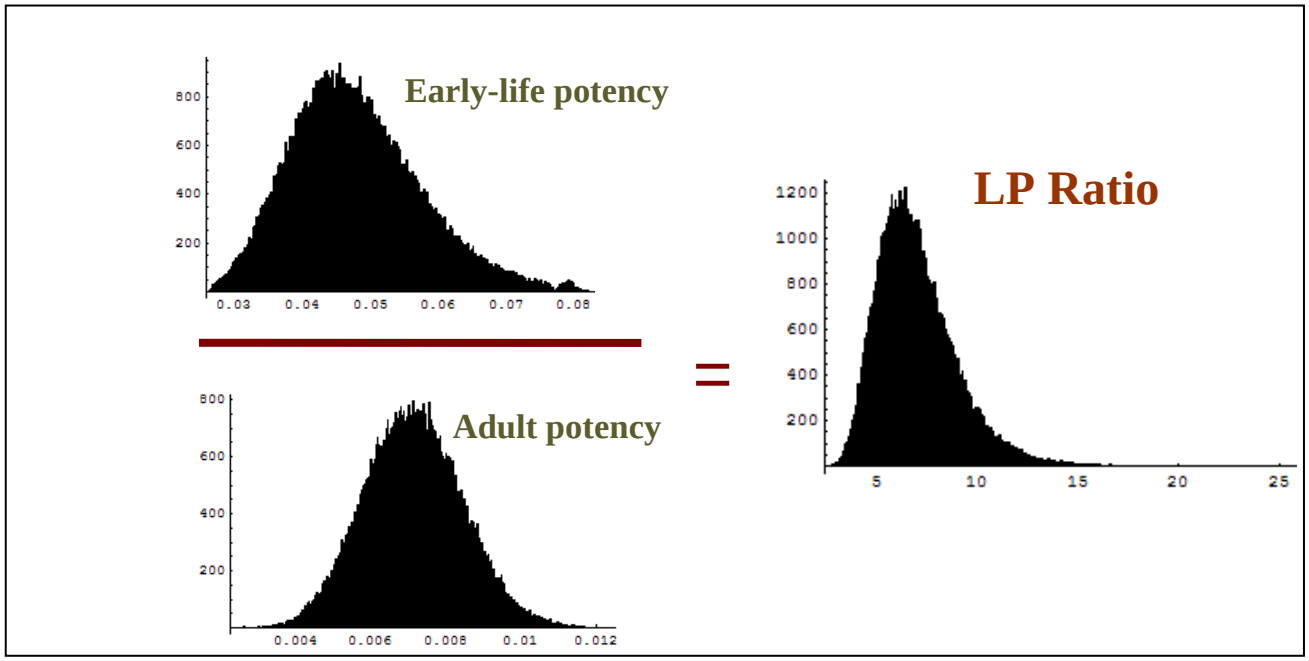
For this analysis, OEHHA calculates the ratio of cancer potency derived from an early lifestage exposure experiment(s) to that derived from an experiment(s) conducted in adult animals. OEHHA used the potency distributions for the individual lifestage exposures, rather than a point estimate, to derive the ratios. The lifestage cancer potency ratio is then described as a distribution and one can select specific percentiles from the distribution to better understand and bound the uncertainty (Figure 5). Of particular importance is the location of the ratio distribution in relation to the reference value of 1.0, which would mean no difference in risk from exposures at early versus adult lifestages. A lifestage cancer potency ratio distribution that primarily lies above the value of 1.0 indicates early life exposures to a carcinogen result in a stronger tumor response relative to adult exposure. Conversely, a lifestage cancer potency ratio distribution that mainly lies below the value of 1.0 indicates early life exposure to a carcinogen results in a weaker tumor response relative to adult exposure.

A lifestage potency (LP) ratio distribution was derived for each multi-lifestage study, resulting in 22 prenatal ratio distributions representing 14 unique carcinogens, 55 postnatal LP ratio distributions representing 18 unique carcinogens, and seven juvenile LP ratio distributions representing five unique carcinogens. The LP ratio distributions for a given early lifestage were combined into a single "LP ratio mixture distribution," in order to show the range of susceptibilities of that lifestage to the carcinogens studied.

LP ratio mixture distributions for a given early lifestage were developed by (1) obtaining a single LP ratio distribution for each chemical (when a chemical is represented by more than one study) and then (2) equally sampling across all chemicals. When a chemical is represented by more than one study, then the LP ratio distributions from all studies of that chemical were combined by equally sampling from each LP ratio distribution via Monte Carlo methods to obtain a single

LP ratio distribution for that chemical. (Appendix J describes this in more detail, as well as a sensitivity analysis that included two alternative sampling methods.) Once each chemical is represented by a single LP ratio distribution, then the LP ratio mixture distribution for each early lifestage (prenatal, postnatal, and juvenile) is obtained by equally sampling across all of the chemicals via Monte Carlo methods.

Figure 5. Lifestage Potency Ratio (LPR) distribution.

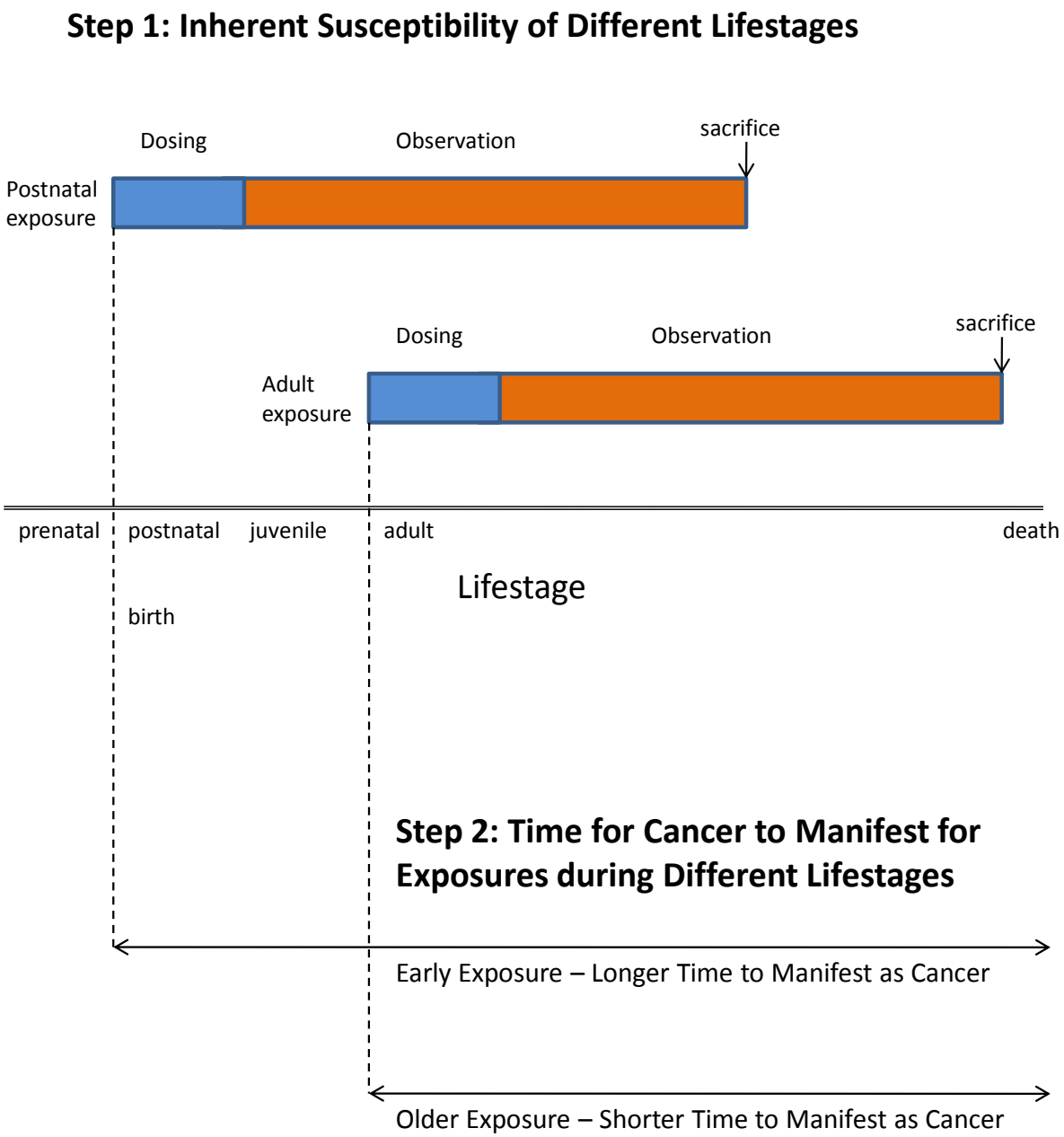


Effect of Longer Time Period for Cancer to Manifest

The LP ratios described above characterize the inherent susceptibility of early lifestages to carcinogen exposure, by comparing potencies for individuals followed for similar periods of time and similarly exposed, but exposed during different lifestages. Age-specific adjustments to the cancer potency must also take into account the longer period of time that carcinogen exposure to the young has to manifest as cancer. Empirical data from studies of both humans and animals demonstrate that, for many cancers, cancer risk increases with age, or time since first exposure. While some cancers have been seen to increase by as much as the sixth power of age, a general approach taken for example by the National Toxicology Program in analyzing tumor incidences in its chronic bioassays is to assume that cancer risk increases by the third power of age. Thus, consistent with the approach used by the NTP in analyzing rodent cancer bioassay data, the longer period of time that exposed young have to develop tumors is addressed by taking into account time-of-dosing. This was done by multiplying the LP ratio by a time-of-dosing factor, to yield an age sensitivity factor (ASF). Specifically, the prenatal LP ratio is multiplied by a factor of 3.0, the postnatal LP ratio is multiplied by a factor of 2.9, and the juvenile LP ratio is multiplied by 2.7. Thus, ASFs were developed for each experiment, by first calculating the LP ratio to address inherent susceptibility of early lifestages relative to adults, and then accounting for the effect of years available to manifest a tumor following carcinogen exposure. (see Figure

6). Note that we are not using the term "sensitivity" in the immunologic sense (*e.g.*, sensitization), but rather are using the term more generically.

Figure 6. Issues addressed by the Age-Sensitivity Factor (ASF)



Application of this approach for risk associated with lifetime exposures would include an ASF of less than 1 for exposures during the latter part of adult life for carcinogens that act on early stages. Therefore, the addition of this adjustment to the younger lifestages but not to the later part of the adult period could overestimate the risk of whole-life exposures. On the other hand, the 70 year "lifetime" used in estimating lifetime cancer risk does not reflect the longer lifespan of the U.S. population. Further, as noted above, the animal bioassays on which potency was based typically exclude pre-weaning dosing and sacrifice animals during their late middle-age. Use of cancer potencies calculated from standard assays can therefore underestimate lifetime cancer risk. The ASF calculated for carcinogens includes both inherent sensitivity of developing animals and the available time since exposure to develop cancer.

Results of OEHHA Analysis

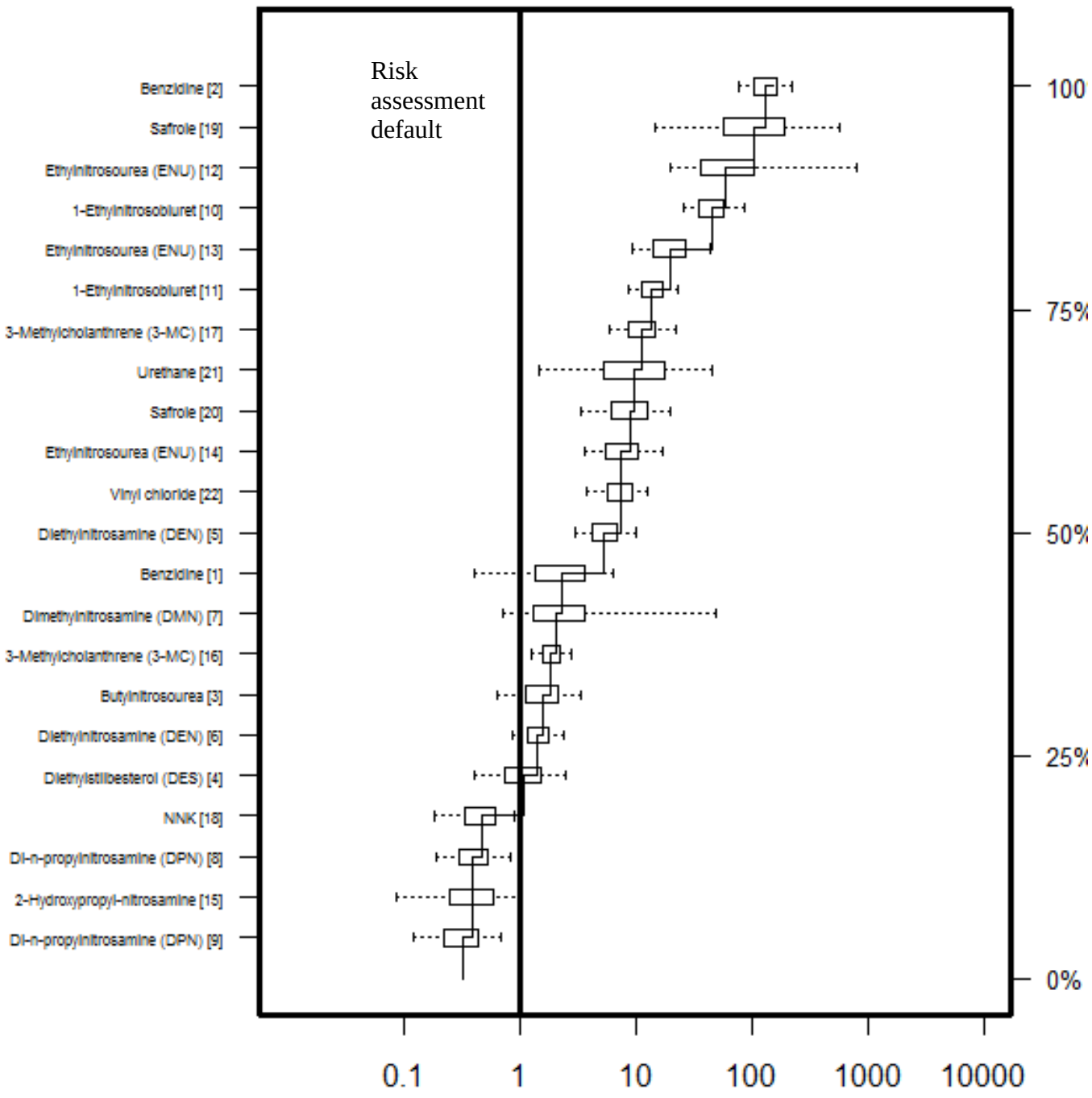
The analyses indicate that both the prenatal and postnatal lifestages can be, but are not always, much more susceptible to developing cancer than the adult lifestage. The analyses also indicated that the ASFs for these age windows vary by chemical, gender and species.

Regarding prenatal lifestage exposure, few cases were indicative of equal inherent adult and prenatal susceptibility, with an LP ratio of unity. The LP ratio distribution was roughly bimodal, with LP ratios for several studies significantly greater than unity and several others significantly less than unity. Figure 7 below shows the ASFs from each of the prenatal multi-lifestage exposure studies, displayed as a cumulative frequency profile. The median of the prenatal ASF mixture distribution was 2.9 (see also Table 6 in Appendix J),

The modality in the prenatal LP ratio distribution was reflected in the DEN and ENU case studies, with results for DEN suggesting inherently less sensitivity than older animals from exposure *in utero*, and for ENU just the opposite. For the DEN and ENU case studies, the referent groups were juvenile rather than adult animals, and the results may have underestimated the LP ratio and ASF, to the extent that some of the apparent sensitivity for DEN and ENU in the prenatal period carries through to the juvenile period. ENU is a direct acting carcinogen that does not require metabolic activation, whereas DEN can not be metabolized to any significant extent by fetal tissues until relatively late in gestation. This may explain the lower fetal susceptibility of DEN. However, prenatal metabolic status is not the sole determinant of prenatal susceptibility; *e.g.*, benzidine and safrole require metabolic activation and exhibit greater susceptibility from prenatal exposure.

The median of the postnatal ASF mixture distribution was 13.5 (see Table 7 in Appendix J). Figure 8 below shows the ASFs from each of the postnatal multi-lifestage exposure studies, displayed as a cumulative frequency profile. Thus, for the chemicals studied, there was generally greater susceptibility to carcinogens during the early postnatal compared to the adult period, particularly when the ASF accounts for the longer period cancer has to manifest when exposure occurs early in life. The DEN and ENU case studies also exhibited substantial extra susceptibility during the postnatal period. To summarize, for most of the carcinogens studied here, rodents are inherently more sensitive in the postnatal period, as indicated by Figure 8.

Figure 7. Prenatal ASF Cumulative Frequency Profile

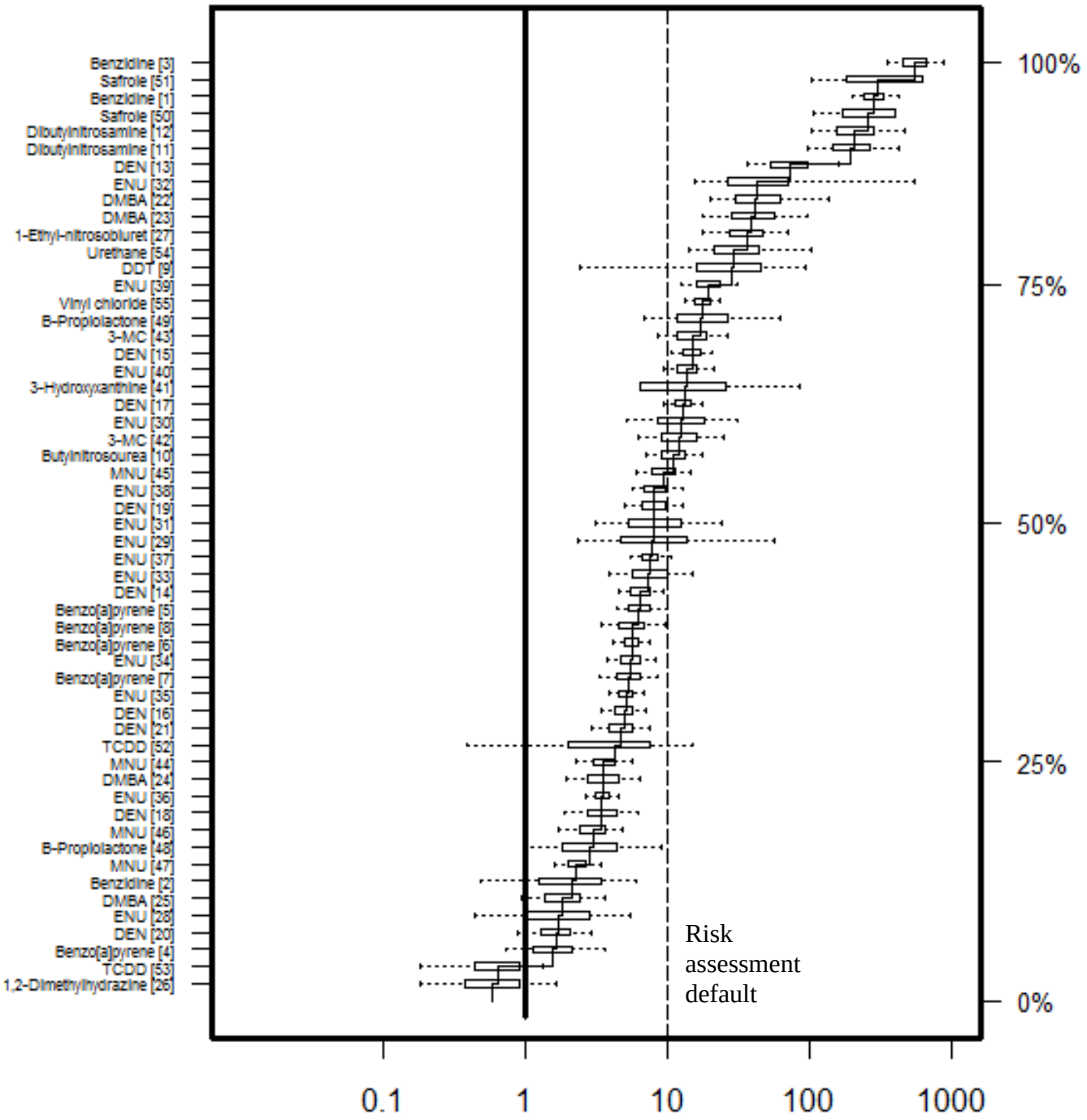


The median of the prenatal ASF mixture distribution was 2.9 (see also Table 6 in Appendix J). References are given in the legend on the next page

Figure 7 Legend (References as in Appendix J)

1. Vesselinovitch *et al*. (1979a), mouse, B6C3F_1, F, day -9 to 0
2. Ibid, M, day -9 to 0
3. Zeller *et al*. (1978), rat, Sprague Dawley, M/F day -2
4. Turusov *et al.* (1992), mouse, CBA, F, day -2
5. Mohr et al. (1975), hamster, Syrian Golden, day -15 to -1
6. Mohr *et al*. (1995), hamster, Syrian Golden, F, day -3
7. Althoff *et al*. (1977), hamster, Syrian Golden, M/F, day -9 to -3
8. Ibid, day -9 to -3
9. Althoff and Grandjean (1979), hamster, Syrian Golden, F, day -9 to -3
10. Druckrey and Landschutz (1971), rat, BD IX, M/F, day -10
11. Ibid, day -3
12. Naito *et al*. (1981), rat, Wistar, day -9
13. Ibid, day -9
14. Tomatis *et al*. (1977), rat, BDVi, F, day -5
15. Althoff and Grandjean (1979), hamster, Syrian Golden, M/F, day -9 to -3
16. Tomatis *et al*. (1971), mouse, CF-1, F day -4 to -1
17. Turusov *et a*l. (1973), mouse, CF-1, F, day -2
18. Anderson *et al.* (1989), mouse, C3H & B6C3 F_1,M/F day -8 to -4
19. Vesselnovitch *et al.* (1979a), mouse, B6C3 F_1, M, day -9 to -3
20. Vesselnovitch *et al.* (1979b), mouse, B6C3 F_1, F day -9 to -3
21. Choudari Kommineni *et al*. (1970), rat, MRC, M/F, day -4
22. Maltoni *et al.* (1981), rat, Sprague Dawley, M/F day -13 to -7

Figure 8. Postnatal ASF Cumulative Frequency Profile



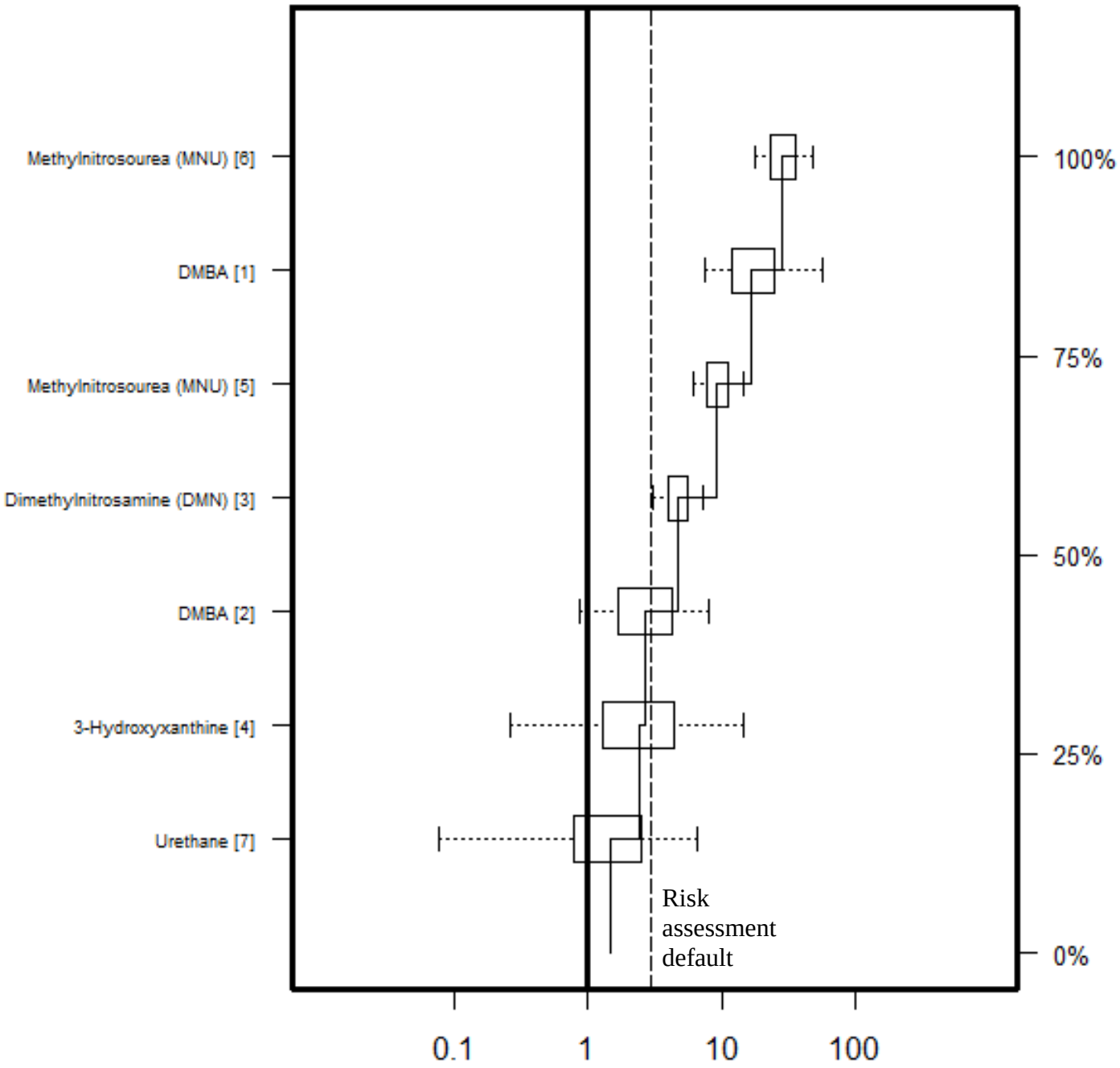
The median of the postnatal ASF mixture distribution is 13.5. The dotted line represents the default ASF for weighting risk for carcinogen exposures to humans between the third trimester and 2 years of age (see next section). References are given in the legend on the next page.

Figure 8 Legend (References as in Appendix J)

1 Vesselinovitch *et al.* (1975b), mouse, B6C3F$_1$, M, day 7-27
2 Vesselinovitch *et al.* (1979), mouse, B6C3F$_1$, F, day 1-21
3 Ibid, M, day 1-21
4 Truhaut *et al.* (1966), mouse, swiss, M/F, day 1
5 Vesselinovitch *et al.* (1975a), mouse, B6C3F$_1$, F, day 1
6 Ibid, M, day 1
7 Ibid, C3A F$_1$, F, day 1
8 Ibid, M, day 1
9 Vesselinovitch *et al.* (1979a), mouse, B6C3F$_1$, M, day 1-28
10 Zeller *et al.* (1978), rat, Sprague Dawley, M/F, day 2
11 Wood *et al.* (1970), mouse, IF x C57, F, day 1-15
12 Ibid, M, day 1-15
13 Rao and Vesselinovitch (1973), mouse, B6C3F$_1$, M, day 15
14 Vesselinovitch *et al.* (1984), mouse, B6C3F$_1$, F, day 1
15 Ibid, M, day 1
16 Ibid, F, day 15
17 Ibid, M, day 15
18 Ibid, C3A F$_1$, F, day 1
19 Ibid, M, day 1
20 Ibid, F, day 15
21 Ibid, M, day 15
22 Meranze *et al.* (1969), rat, Fels-Wistar, F, day 10
23 Ibid, M, day 10
24 Walters (1966), mouse, BALB/c, F, day 17
25 Ibid, M, day 17
26 Martin *et al.* (1974), rat, BDIX, M/F, day 10
27 Druckrey and Landschutz (1971), rat, BDIX, M/F, day 10
28 Naito *et al.* (1985), gerbil, mongolian, F, day 1
29 Ibid, M, day 1
30 Bosch (1977), rat, WAG, F, day 8
31 Ibid, M, day 8
32 Naito *et al.* (1981), rat, Wistar, F, day 7
33 Ibid, M, day 7
34 Vesselinovitch *et al.* (1974), mouse, B6C3F$_1$, F, day 1
35 Ibid, M, day 1
36 Ibid, F, day 15
37 Ibid, M, day 15
38 Ibid, C3A F$_1$, F, day 1
39 Ibid, M, day 1
40 Ibid, M, day 15
41 Anderson *et al.* (1978), rat, Wistar, F, day 9
42 Klein (1959), mouse, A/He, F, day 8-31
43 Ibid, M, day 8-31
44 Terracini and Testa (1970), mouse, B6C3F$_1$, F, day 1
45 Ibid, M, day 1
46 Terracini *et al*. (1976), mouse, C3Hf/Dp, F, day 1
47 Ibid, M, day 1
48 Chernozemski and Warwick (1970), mouse, B6A F$_1$, F, day 9
49 Ibid, M, day 9
50 Vesselinovitch *et al*. (1979a), mouse, B6C3F$_1$, M, day 1-21
51 Vesselinovitch *et al*. (1979b), mouse, B6C3F$_1$, M, day 1-21
52 Della Porta *et al*. (1987), mouse, B6C3F$_1$, F, day 10-45
53 Ibid, M, day 10-45
54 Choudari Kommineni *et al*. (1970), rat, MRC, M/F, day 1-17
55 Maltoni *et al*. (1981), rat, Sprague Dawley, M/F, day 1-35

There were only five chemicals and seven studies, two of which were not independent, available to examine susceptibility in the juvenile period. The juvenile LP ratios indicated significantly greater susceptibility in this period for three independent studies, with the remaining studies consistent with equal inherent susceptibility to adult animals (see Figure 16 in Appendix J). Figure 9 below shows the ASFs from each of the juvenile multi-lifestage exposure studies, displayed as a cumulative frequency profile. The median of the juvenile ASF mixture distribution was 4.5 (see Table 8 in Appendix J).

Figure 9. Juvenile ASF Cumulative Frequency Profile



The median of the juvenile ASF mixture distribution is 4.5. The dotted line represents the default value for weighting risk for carcinogen exposures between 2 and 15 years of age (see next section).

Figure 9 Legend (References as in Appendix J)

1. Meranze *et al.* (1969), rat, Fels-Wistar, F, day 45
2. Ibid, M, day 45
3. Noronha and Goodall (1984), rat, CRL/CDF, M, day 46
4. Anderson *et al*. (1978), rat, Wistar, F, day 28
5. Grubbs *et al*. (1983), rat, Sprague Dawley, F, day 50-57; adult comparison group dosed on days 80-87
6. Ibid, F, day 50-57; adult comparison group dosed on days 140-147
7. Choudari Kommineni *et al*. (1970), rat, MRC, M/F, day 28-43

The studies that comprise the set of multi-lifestage exposure studies available for these analyses were not homogeneous. That is, they do not represent observations from the same distribution. Sensitivity analyses were conducted to test the robustness of the findings to different procedures for analyzing data and combining results. Of the methods used to combine the LC ratio distributions for underlying studies within each lifestage, the method of equally weighting studies within a chemical appeared to best represent the available data.

In calculating the ASF, to take into account the longer period of time for early carcinogen exposures to result in tumors, the hazard function was assumed to increase with the third power of age. This assumption is standard and has been borne out by a number of observations (Bailer and Portier, 1988). If the true rate of increase with age is greater than that, then the use of these ASFs may result in underestimates of the true sensitivity of these early life stages.

As the multi-lifestage exposure and case studies show, there appears to be considerable variability in age-at-exposure related susceptibility across carcinogens. There is also variability in age-at-exposure related susceptibility among studies of the same carcinogen. The sources of variability evident in the analyzed studies include timing of exposure within a given age window, and gender, strain, and species differences in tumor response. The set of studies identified and analyzed was not sufficiently robust to fully describe the variability quantitatively. This variability raises concerns that selection of the median (the 50^{th} percentile) estimates may considerably underestimate effects for certain agents or population groups. Relatively large variability in humans in response to carcinogens is expected to be common (Finkel, 1995). On the other hand, the numbers of carcinogens represented in the available data are limited and may not be representative of the population of carcinogens to which we are exposed (*e.g.*, greater than 500 on the Proposition 65 list alone). Thus, the size of the weighting factors used to weight risk by age at exposure is a policy decision.

Several of the carcinogens studied induced tumors at multiple sites in the same experiment, and at different sites, depending upon the lifestage during which exposure occurred. For these cases the combined multisite potency distribution referred to above was the basis for the lifestage comparison. This approach differs from other researchers investigating early vs. late in life differences who focused on tumor site-specific measures of carcinogenic activity (*e.g.*, Barton *et al*., 2005; Hattis *et al*., 2004, 2005). OEHHA believes that use of combined multisite potency distributions provides a more complete approach for considering age specific differences in carcinogenic activity. However, the observation that early life is generally a period of increased

susceptibility was similarly found using the tumor site-specific approach by these other researchers.

One limitation of the approach was the focus on lifestages, without attempting to describe changes in susceptibility that occur within a lifestage. Timing of carcinogen exposure within a given age window can affect the cancer outcome. For example, experiments with 1-ethyl-1-nitroso-biuret in prenatal and adult rats showed a three-fold difference in activity between groups exposed on prenatal day -10 versus prenatal day -3. In a second example, female rats exposed early in the adult period were more than three times as sensitive to the breast cancer effects of MNU as females exposed six weeks later. In general, the adult comparison groups in the multi-lifestage exposure studies were fairly young. The extent to which this may result in an overall bias of the results presented here is unclear. Also, for several cases, juvenile animals were used as the later life exposure group. In these cases the ASFs are likely underestimates of the relative sensitivity of the prenatal and postnatal lifestages, compared to that of the adult lifestage.

Excluded from the analysis were early in life studies in which the period of exposure for a specific exposure group crossed multiple lifestages. An example of results from studies of this type is provided by mouse studies for two non-genotoxic carcinogens, diphenylhydantoin (Chhabra *et al*., 1993a) and polybrominated biphenyls (PBBs) (Chhabra *et al*., 1993b), in which exposures began prior to conception, and continued throughout the prenatal, postnatal, and post-weaning period, up to the age of eight weeks. The data demonstrate an increased sensitivity of the early life period. Some studies that crossed multiple lifestages were included in the analyses of Barton *et al*. (2005) (Appendix I), which are consistent with the general conclusions discussed above.

Selection of Default Age-Sensitivity Factors (ASF)

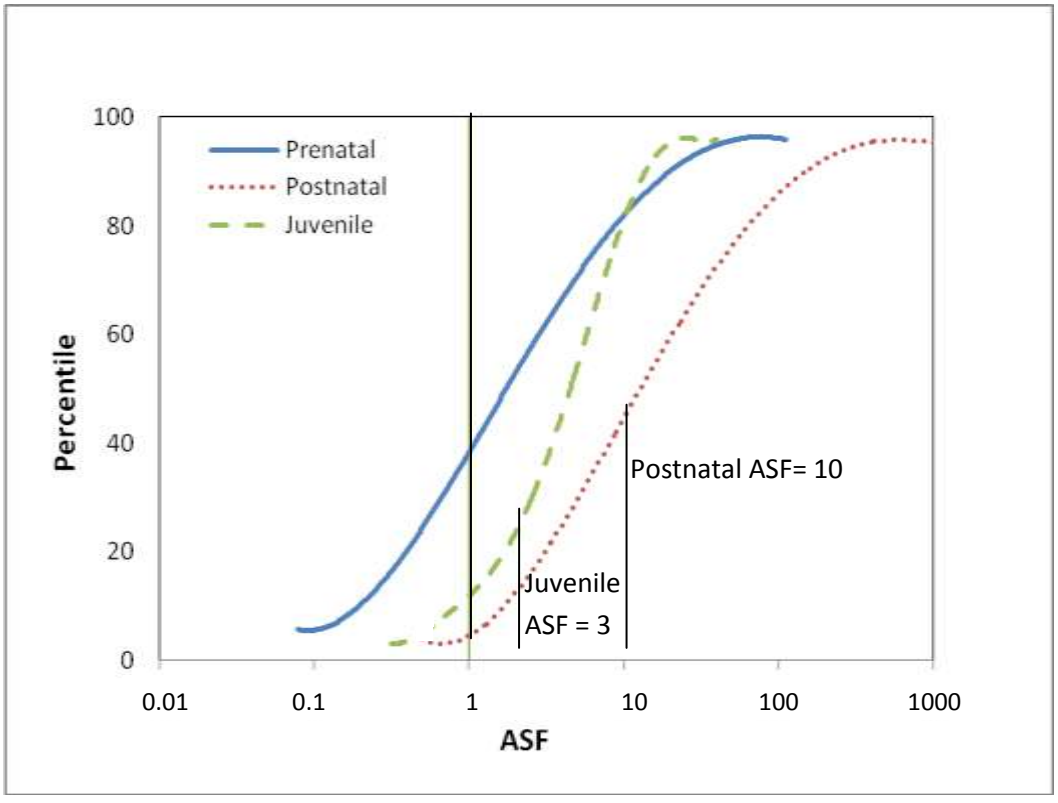
Selection of appropriate values to use to weight exposures that occur early in life using default ASFs for prenatal, postnatal and juvenile exposures is complicated by the limited database of chemicals and studies available for analysis, and the broad distribution of results for different chemicals as is shown in Figure 7, Figure 8, and Figure 9 (see also Appendix J). In view of the variability thus shown, and the considerable uncertainty in applying conclusions from this relatively small set of chemicals to the much larger number of chemicals of concern, it is probably unreasonable to specify a default ASF with greater than half-log precision (*i.e.* values of 1, 3, 10, 30 etc.). Further, rodents are born at a stage of maturity that approximates a third trimester human. Therefore, in the absence of chemical-specific data, OEHHA proposes to apply a default ASF of 10 for the third trimester to age 2 years, and a factor of 3 for ages 2 through 15 years to account for potential increased sensitivity to carcinogens during childhood. A factor of 10 falls just below the median estimate of the ASF for postnatal studies. This is also the value selected by U.S. EPA; while it is consistent with the OEHHA analysis, it may underestimate risk for some chemicals. The broad distribution of observed chemical-specific sensitivity ratios clearly indicates that there are some chemicals for which the sensitivity ratio is much larger than 10. Further research is needed to develop criteria for identifying these cases. Similarly, a factor of 3 for juvenile exposures is consistent with the range of estimates derived from the multi-lifestage exposure studies, and falls close to the median juvenile ASF estimate. It is acknowledged that there are few data available on which to base an estimate for the juvenile period. A factor of 3 adjusts for the longer time available for cancer to manifest, but may not

fully account for some inherent differences in susceptibility to cancer, for example the observed susceptibility of breast tissue of pubescent girls exposed to radiation. For specific carcinogens where data indicate enhanced sensitivity during lifestages other than the immediate postnatal and juvenile periods, or demonstrate ASFs different from the default ASFs, the chemical-specific data should be used in order to adequately protect public health.

The ASFs will be applied to all carcinogens, regardless of the theorized mode of action. While U.S. EPA currently intends to apply weighting factors only to those carcinogens with "a mutagenic mode of action" (U.S.EPA, 2005), OEHHA notes that there is evidence that early life is a susceptible time for carcinogens that are thought to act via non-mutagenic mode of action (DES is a prime example). Defining a mutagenic mode of action may be problematic if approached narrowly (ERG, 2008). Further, carcinogens may have multiple modes of action and one mode may predominate over other modes at different lifestages. The complexity of carcinogenesis argues against restricting the ASF to chemicals acting via a mutagenic mode of action.

Figure 10 provides a visual comparison of the ASF mixture distributions for the three early-life stages, prenatal, postnatal, and juvenile. In this figure, which is in log space, the policy choice of an ASF of 10 for exposures during the third trimester to age 2 years and 3 for the period of life from 2 to 15 years of age are indicated as vertical lines. It is apparent from this figure that weighting risk from exposures to carcinogens early in life is well-supported.

Figure 10. Prenatal, Postnatal, and Juvenile ASF Mixture Distributions and relation to default ASFs



OEHHA recognizes the limitations in the data and analyses presented, as discussed above. However, the analyses do provide some guidance on the extent to which risk may be over or underestimated by current approaches. While there is a great deal of variability across chemicals in the prenatal ASFs, the data indicate that the potency associated with prenatal carcinogen exposure is not zero. A factor of 3 is close to the median ASF, while a factor of 10 falls roughly at the 70th percentile of the prenatal ASF estimate. An ASF could be applied as a default when calculating lifetime cancer risk in humans arising from carcinogen exposures that occur *in utero*. In view of the considerable variability in the data for different carcinogens and the limited database available for analysis, OEHHA is not proposing the application of a specific factor to cancer potency estimates for prenatal exposures in the first and second trimesters as a default position in these Guidelines. However, given that the rodent is born at a stage of maturation similar to a third trimester fetus, it is reasonable to include the third trimester in the 10X potency weighting proposed up to age 2 years. The applicability of a cancer potency adjustment factor for first and second trimester prenatal exposure will be evaluated on a case-by-case basis, and may be used as evidence develops that supports such use. The consideration of prenatal exposures, including application of an appropriate susceptibility factor, would not make a large difference for risk estimates based on continuous lifetime exposures, due to the relatively short duration of gestation. However, risk estimates for short-term or intermittent exposures would be slightly increased by inclusion of the risks to the fetus during the prenatal period. Thus, risk may be underestimated when the first and second trimesters are excluded from the analysis.

Age Bins for Application of ASFs

The choice of human ages to which the ASFs apply is based on toxicodynamic and toxicokinetic considerations. Important toxicodynamic factors related to susceptibility to carcinogens include the rate of cellular proliferation and differentiation, which is quite high during organ maturation. In addition, toxicokinetic differences by age are important, due to impacts on detoxification and clearance of carcinogens (see following section). OEHHA's analysis of the influence of age-at-exposure on carcinogenesis broke the experimental rodent data into age bins that we termed "lifestages" including prenatal, "postnatal" (birth to weaning, about day 21) and "juvenile" (weaning to sexual maturation, or about day 22 to about day 49). Experiments were placed into the lifestage bins if exposure occurred at some time during the experimental rodent age bin.

There is no simple way to compare the rodent age groups used in the OEHHA analysis of available data to equivalent age groups in humans. Complicating factors include variations in organ system structural and functional maturation both within and between species. Further, the rodent age bins were chosen by gross indicators of development namely birth, weaning and sexual maturation, not on the basis of known susceptibility to carcinogenesis. Thus, critical factors relating to carcinogen susceptibility by age are the focus of the choice of human age bins to which the ASFs of 10 and 3 apply, rather than an attempt at exact correlation of rodent lifestage bin with human age.

The investigations used by OEHHA to evaluate the relationship between age at exposure and cancer potency were not conducted by standardized protocol. Further, the windows of susceptibility are quite varied by chemical and organ system, even within the lifestages defined in the OEHHA analysis. This complicates choosing a default ASF and the human age bin to which it applies. Examples from animal studies provided in Appendix J include the chemical

diethylnitrosamine (DEN). The cancer potency varied over several orders of magnitude depending on when during gestation and postnatal life the exposure occurred. A three-fold difference in potency between exposure on prenatal day -3 and prenatal day -10 is noted for 1-ethyl-1-nitrosobiuret in rats. There are also human examples of extensive variation of potency by age at exposure, including radiation, DES, and chemotherapeutic agents. The diversity of responses to different agents obviously underscores uncertainty in the choice of age bins to apply the default ASFs. However, the ASFs are a ***default*** to use when you have no chemical-specific data on influence of age-at-exposure on potency in order to protect public health. There will always be specific chemical examples where the ASF for either the third trimester-<2 yrs or 2-<16 yrs age bin is quite a bit larger or quite a bit smaller than the default.

In the following sections, we discuss our logic in proposing age bins of third trimester to age 2 years, and 2 to age <16 years to which the ASFs of 10 and 3 apply, respectively, and indicate the impact on risk estimates of these age bins.

Toxicokinetic Factors Relevant to Age Bins

Choice of the age-bins to which the default ASFs are applied is based on our understanding of the two primary drivers of age-related sensitivity to carcinogens, namely age-related toxicokinetic factors and toxicodynamic factors. In the case of toxicokinetics, the largest postnatal differences in xenobiotic metabolic capability occur between infants and adults. As noted in OEHHA (2001) and reviewed in detail elsewhere (*e.g.*, Cresteil et al., 1998; Ginsberg et al., 2004), hepatic drug metabolism by the cytochrome P-450 family of enzymes and the Phase II conjugating enzymes undergoes a maturation process during the first few years of life. The hepatic cytochrome P-450 enzymes exist in fetal isoforms at birth, and progressively change to adult isoforms at a relatively early stage of postnatal development. Thus, in humans the metabolic capability towards prototypical substrates develops over the first year of life towards adult levels. Similarly, the largest differences in metabolic capability of Phase II enzymes (conjugation of xenobiotic metabolites prior to excretion) tend to be between infants and adults. Other factors such as renal capability also are most different between neonates and adults. Thus, the first 2 years of life would encompass the increased sensitivity of early life stages due to toxicokinetic differences between early life and adulthood.

Ontogeny of Cytochrome P-450 Enzymes in Humans.

Cresteil (1998) describes three groups of neonatal cytochrome P-450: Cyp3A7 and Cyp4A1 present in fetal liver and active on endogenous substrates; an early neonatal group including Cyp2D6 and 2E1 which surge within hours of birth; and a later developing group, Cyp3A4, Cyp2Cs, and Cyp1A2. Total Cyp 3A protein, a major cytochrome P-450 enzyme responsible for biotransformation of many xenobiotics, is relatively constant in neonates and adults. However, Cyp3A7 is the primary fetal form (Hakkola et al., 1998), while Cyp3A4 is the primary adult hepatic form of the 3A series. At one month Cyp3A4 activity is about one-third of that in the adult liver (Lacroix et al., 1997; Hakkola et al., 1998). Allegaert *et al*. (2007) stated that Cyp3A4 (testosterone-6ß-hydroxylase) activity equaled or exceeded adult activity after 1 year of age. Cyp2E1, which metabolizes benzene, trichloroethylene and toluene, among others, increases gradually postnatally, reaching about one-third of adult levels by one year of age and attains adult levels by 10 years of age (Vieira et al., 1996; Cresteil, 1998). Cyp1A2, and Cyp2C9 and

2C19, the most abundant Cyp2 enzymes in adult human liver, appear in the weeks after birth, and reach 30% to 50% of adult levels at about 1 year of age (Treluyer et al., 1997; Hines and McCarver, 2002). Cyp1A1 is expressed in fetal liver where it can activate such xenobiotics as benzo[a]pyrene and aflatoxin B1 (Shimada et al., 1996), but is less important in adult liver (Hakkola et al., 1998).

Ontogeny of Cytochrome P-450 Enzymes in Rodents.

Hart et al. (2009) report developmental profiles of a number of cytochrome P-450 enzymes (measured as levels of mRNA transcripts of the specific genes) in mice. They identified three groups of isoforms. Group 1 (Cyp3A16 in both sexes; Cyp3A41b in males) appeared rapidly after birth but declined to essentially zero at 15-20 days, which is the period of weaning in mice. A second group (Cyp2E1, Cyp3A11 and Cyp4A10 in both sexes; Cyp3A41b in females) also increased rapidly after birth, but reached a stable maximal level by postnatal day 5. The third group (Cyp1A2, Cyp2A4, Cyp2B10, Cyp2C29, Cyp2D22, Cyp2F2, Cyp3A13 and Cyp3A25) were expressed only at low levels until days 10 to 15, but reached high stable levels by day 20.

ElBarbry et al. (2007) examined the developmental profiles of two toxicologically significant cytochrome P-450 enzymes, Cyp1A2 and Cyp2E1 in rats. mRNA transcripts of these genes were very low postnatally, but thereafter increased to reach a peak at or shortly after weaning (postnatal day 21 - 28 for rats). Immunoreactive Cyp1A2 and Cyp2E1 proteins were first detectable at postnatal day 3 and reached 50% of adult levels at weaning and adult levels at puberty. Differences in profiles between gene expression as mRNA and appearance of specific proteins as determined by immunoassay may reflect changes in the relative importance of transcription and translation control processes at various phases in development. Enzyme activities characteristic of Cyp1A2 and Cyp2E1 were found to parallel gene expression levels (ElBarbry et al., 2007) rather than immunodetectable protein levels, so there may also be issues of cross-reactivity between these two isoenzymes and others for which gene expression was not measured in these experiments.

In summary, the gene expression data in rats and mice show differences in details, but broadly resemble one another in that the main changes occur in the early postnatal period, with the major adjustments completed at or around the time of weaning, although the adult pattern may not be completely established until puberty. There do not appear to be substantive data for experimental species other than rats and mice, although the situation in humans appears similar in general outline and one may conclude that this pattern or some variant of it is characteristic of mammalian species in general.

Ontogeny of Phase II Enzymes

Phase II conjugating enzymes are generally less active in the neonate than the adult (Milsap and Jusko, 1994). Hence, there is concern that detoxification and elimination of chemicals is slower in infants. In humans, expression of some of the UGT enzymes matures to adult levels in two months after birth, although glucuronidation of some drugs by the UGT1A subfamily does not reach adult levels until puberty (Levy et al., 1975; Snodgrass, 1992; McCarver and Hines, 2002). Reduced glucuronidation in neonates slows the clearance of *N*-hydroxyarylamines, phenol, and benzene metabolites. Acetylation by the N-acetyltransferases and sulfation by sulfotransferases

are generally somewhat comparable to adult levels, although it varies by tissue and by specific sulfotransferase (McCarver and Hines, 2002). Human glutathione sulfotransferase (GST) is present as a fetal isoform which decreases postnatally, while GST-alpha and GST-mu increase over the first few years of life to adult levels (McCarver and Hines, 2002). Epoxide hydrolase, important in detoxifying reactive epoxide metabolites, is present in neonatal liver although at much reduced activity relative to adults (McCarver and Hines, 2002).

Clearances of Drugs in Infants and Children *vs.* Adults

Several investigators have evaluated age-related drug disposition (Renwick, 1998; Renwick et al., 2000; Ginsberg et al., 2002; Hattis et al., 2003). Renwick et al. (2000) noted higher internal doses in neonates and young infants versus adults for seven drugs that are substrates for glucuronidation, one with substrate specificity for CYP1A2, and four with substrate specificity for CYP3A4 metabolism. Ginsberg et al (2002) evaluated toxicokinetic information on 45 drugs in children and adults metabolized by different cytochrome P-450 pathways, by Phase II conjugations, or eliminated unchanged by the kidney. These authors noted half-lives 3-9-fold longer in infants than those in adults. It was also shown that the bulk of the elevated child/adult half-life ratios occurred primarily in the 0 to 6 month age range, and that for some compounds the clearance is actually higher in the 6 month to 2 year age grouping. In evaluating the interindividual variability by age, Hattis et al (2003) note that the largest interindividual variability occurs in the youngest children, apparently due to variability in development of critical metabolism and elimination pathways. Anderson and Holford (2008) noted that a comparison of three early-life drug clearance models (surface area, allometric ¾ power and per kilogram scaling) all demonstrated an increase in clearance over the first year of life due to the maturation of metabolic capacity.

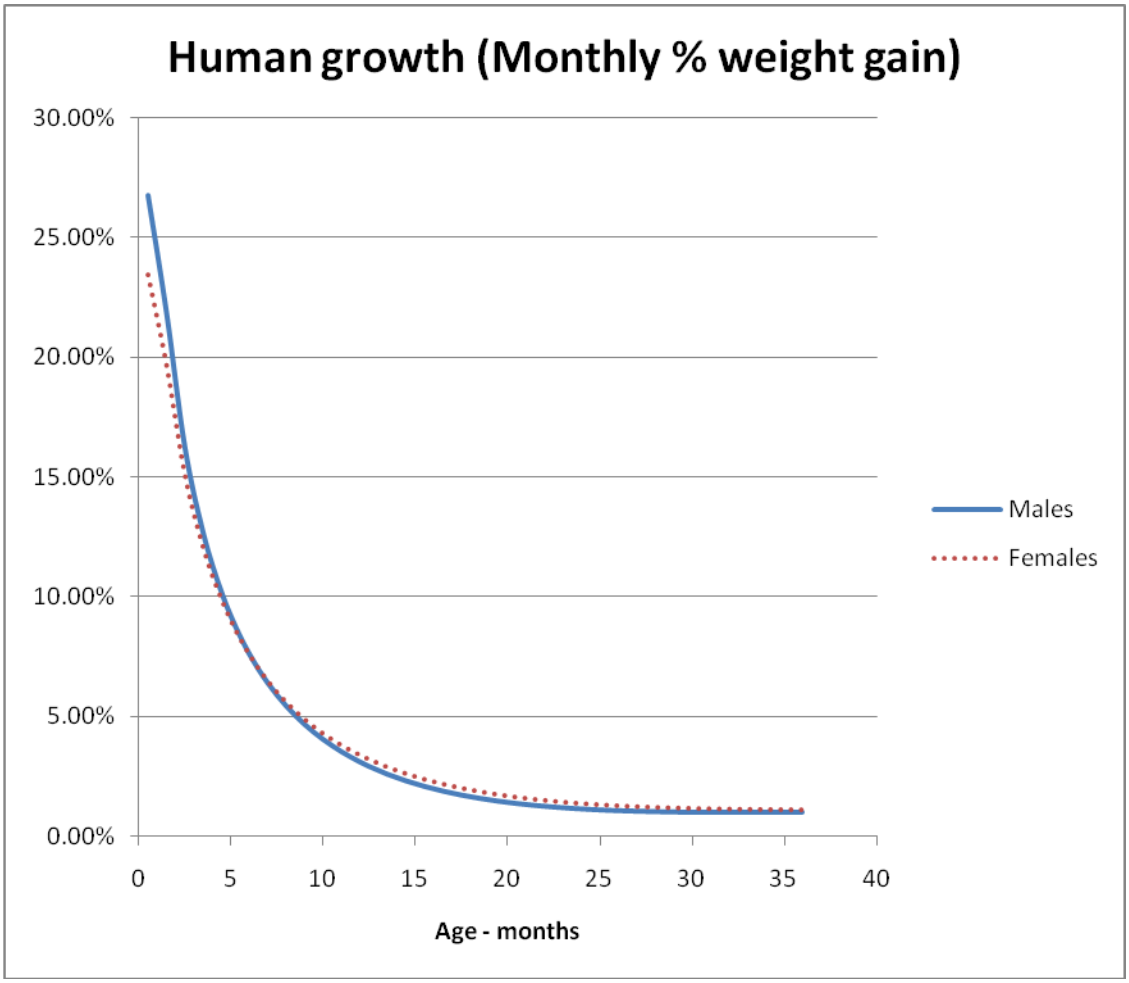
Renal elimination depends on maturity of processes related to tubular reabsorption and secretion, and glomerular filtration rates. At birth, the glomerular filtration rate (GFR) is low (2-4 ml/min), increases in the first few days (8-20 ml/min) and slowly increases to adult values in 8-12 month old infants (Plunkett et al., 1992; Kearns et al, 2003).

Newborn and young animals have less capacity to excrete chemicals into the bile than do adult animals. A number of chemicals are excreted more slowly via bile in neonates than adult rats, including ouabain, the glucuronide conjugate of sulfobromophthalein (Klaassen, 1973), and methyl mercury (Ballatori and Clarkson, 1982), resulting in a longer half-life in neonates.

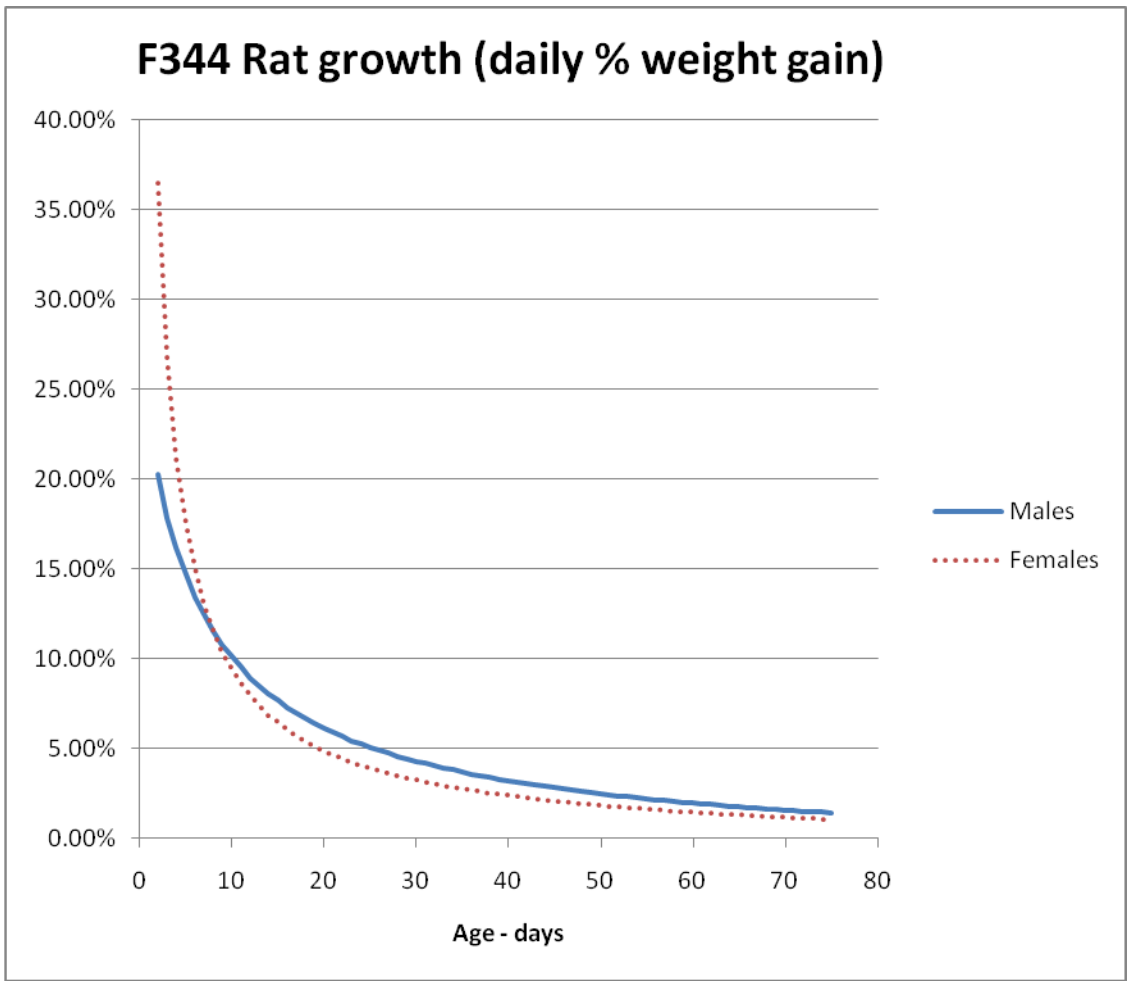
Toxicodynamic Factors Relevant to Age Bins

Important as the developmental changes in toxicokinetics are in determining sensitivity to carcinogens and other toxicants, it is likely that the toxicodynamic differences, *i.e.* intrinsic differences in susceptibility to carcinogenesis at the tissue or cellular level, are even more influential. Changes in cell division rates and differentiation, which are thought to be important toxicodynamic determinants of susceptibility to carcinogenesis, peak in the first 2 years of life for most major organ systems. Cell division continues to accommodate growth throughout childhood and adolescence, extending in some cases even into the young adult period in both humans and experimental animals. Adolescence is an important period for organ cell division and differentiation for the mammary gland and reproductive organs.

As noted above, one of the key factors influencing susceptibility to carcinogenesis is believed to be cell division rate, which acts both by forcing error-prone repair which fixes DNA damage as mutated gene sequences (McLean et al, 1982) and by promoting expansion of mutated clones (Moolgavkar and Knudson, 1981). Actual cell division rates as a function of age are hard to determine for practical and (in the human case) ethical reasons. However, growth curves expressed as the proportional increment in body weight with time may be regarded as a reasonable although not perfect surrogate since for most tissues of the body cell size does not change markedly during growth. Both humans and rodents show remarkably high growth rates in infancy, which then drop steeply to a lower but still significant rate during childhood. A growth spurt at the beginning of adolescence is noticeable in its absolute magnitude, especially in males, but does not approach the proportional growth rate seen in infancy. The time intervals proposed to reflect the period of highest sensitivity to carcinogenesis (up to about 21 days in rodents, up to 24 months in humans) encompass the period of highest growth rate and thus it is assumed the highest cell division rates, as show in the following charts:



Data from CDC NHANES 2000:
http://www.cdc.gov/nchs/about/major/nhanes/growthcharts/datafiles.htm



Data from Tables A3 and A4 of Appendix J

Cell division rates in adult rodents and humans are harder to relate to growth curves since at least some tissues retain active cell division as part of their ongoing functionality and repair. In humans growth in body weight slows to essentially zero at the end of adolescence (and any later increments represent tissue specific changes such as increase in muscle or adipose tissue mass rather than overall growth). On the other hand, rodents continue to increase in body size (at a modest rate compared to that seen in earlier lifestages) throughout the adult period. However, it appears reasonable to conclude from the body weight data that an essentially adult pattern of overall cell division is established by the late adolescent period (age six weeks in rodents; 16 years in humans). However, increased cell division and cell differentiation are seen in the reproductive system and its accessories during puberty.

Organ Development

The age intervals chosen for the ASFs are generally supported by human organ system development data. Examples of supporting data are available for the lung, brain, immune system and liver. Zeltner and Burri (1987) stated that postnatal lung development consists of an alveolar stage, which lasts to about 1-1.5 years of age, and a stage of microvascular maturation, which exists from the first months after birth to the age of 2-3 years. Pinkerton and Joad (2006)

describe alveolar proliferation as occurring most prominently in the 0-2 year age range, with alveolar expansion continuing in the 2-8 year age range. Ballinoti et al. (2008) demonstrated that addition of alveoli rather than expansion is a major mode of lung growth in infants and toddlers by measuring a constant carbon monoxide diffusion capacity to lung volume from 3 through 23 months of age. Kajekar (2007) also considered the 0-2 age range to be the primary period of alveolar development, although there is continued cellular proliferation resulting in lung growth and expansion up to approximately 18 years of age.

Rice and Barone (2000) note that most of the cell proliferation phase of human radial glia and neuronal growth is finished by 2 years of age, based on evidence in Bayer et al. (1993). They note further that numerous studies have shown actively proliferating brain regions are more susceptible to anti-mitotic agents than the same structures after active proliferation ceases. Peak brain growth as a percentage of body weight occurs at birth and around post-natal day (PND) 7-8 in humans and rats, respectively (Watson *et al*., 2006). De Graaf-Peters and Hadders-Algra (2006) reviewed the ontogeny of the human central nervous system and found that a large amount of axon and dendrite sprouting and synapse formation and the major part of telencephalic myelination take place during the first year after birth. While the brain continues to remodel itself throughout life, cellular proliferation in the whole brain peaks by about one year of age and is relatively complete by age 2. Development of the blood-brain barrier (BBB) appears to continue in humans until approximately 6 months of age. Rat BBB functionality is essentially complete by approximately two weeks after birth (Watson *et al*., 2006).

The immune system development occurs in stages, primarily prenatally in primates and both pre- and post-natally in rodents (Dietert et al., 2000). Formation and expansion of hematopoetic stem cells is followed by expansion of lineage-specific stem cells, colonization of bone marrow and thymus, and maturation of cells to immunocompetence. In the primate, this is largely complete by 1 to 2 years of age (Holsapple et al., 2003), although establishment of immune memory develops throughout childhood and beyond. In the rodent, maturation to immunocompetence occurs postnatally from birth to about 30 days of age. In terms of carcinogenesis, perhaps one of the more important immune cells is the NK cell, thought to be responsible for immune surveillance and killing of circulating transformed cells. Based on immunohistochemistry, the principal cell lines including NK cells are present at gestation day 100 in the monkey and are at about 60% of adult values at birth (Holladay and Smialowicz, 2000).

As noted above, renal and hepatic clearance are both lower in humans at birth than in adults. Nephrogenesis is complete by 35 weeks gestation in humans and before birth in the mouse (but after birth in the rat). The ability to concentrate urine and the development of acid-base equilibrium appear in the first few months after birth (Zoetis and Hurtt, 2003). Renal clearance of drugs, a function of a number of processes in the kidney, appears to be comparable to adults within the first few months of life (Hattis et al., 2003; Ginsberg et al., 2002), while glomerular filtration, which rises rapidly over the first few postnatal months, is at adult values by two years of age (Zoetis and Hurtt, 2003). While complete anatomic maturity of the human liver is noted by 5 years of age (Walthall et al, 2005), liver function also appears to mature within the first year of life as seen by drug clearance studies cited above.

Critical Windows of Susceptibility to Carcinogens

It has been shown that there are critical windows during development both pre-and postnatally where enhanced susceptibility to carcinogenesis occurs (Anderson et al, 2000). Some of these observations relate to factors affecting the incidence of cancers in childhood, resulting from prenatal or preconception mutational events. For example, prenatal exposure to ionizing radiation and DES can result in leukemia and vaginal carcinoma, respectively, in childhood. Although obviously a source of great concern, these cancers appearing during childhood are relatively rare compared to cancers appearing later in life. Thus the concern in risk assessment for early in life exposures is to address the lifetime cancer incidence as a result of these exposures, including both cancers appearing during childhood and those appearing later.

OEHHA (see Appendix J) and other investigators (U.S. EPA, 2005; Barton et al, 2005; Hattis et al., 2004) have examined the available rodent data on sensitivity to carcinogenic exposures early in life. All these investigators found substantial increases in sensitivity to carcinogens in animal studies where exposures to young animals were compared to similar exposures to adults. Hattis et al. (2004) reported maximum likelihood estimates for the ratio of carcinogenic potency during the period from birth to weaning to the adult potency of between 8.7 and 10.5, whereas Barton et al (2005) reported a weighted geometric mean of 10.4 for the ratio of juvenile (less than 6-8 weeks) to adult potency in rodents. However, the number of experiments which provide information of this type, and the carcinogenic agents which have been studied, are relatively limited. Hattis examined several different datasets and study designs, but these covered only 13 different chemicals, while the mean value reported by Barton et al. was based on only six of the 18 chemicals which they examined. OEHHA's analysis included data in rodents on 23 chemicals, and found median potency ratios of 13.5 for the postnatal period (birth to day 22) and 4.5 for the juvenile period (postnatal days 22 to ~49) relative to adults (day ~49 to 2 years). These potency ratios include the adjustment for time to manifest tumor (*e.g.*, age to the power of three), unlike the earlier investigations. All these investigations identified variations in the observed lifetime potency ratio depending on the type of experimental design, the sex of the animals, the time of exposure and especially between chemicals. Nevertheless these analyses, although falling far short of a comprehensive evaluation of the age dependence of carcinogenic potency for all the chemicals of interest, do show a consistent overall trend of increasing potency for exposures early in life, especially soon after birth.

An evaluation of cancer induction by ionizing radiation also provides support for the concept of enhanced sensitivity to carcinogenesis at younger ages. Various studies of this phenomenon have been undertaken in animal models, but the important point for the present discussion is that epidemiological data exist which indicate age-dependent sensitivity in humans (U.S. EPA, 1994; 1999). The most extensive data set showing age-dependent effects is that for Japanese survivors of the atomic bomb explosions at Hiroshima and Nagasaki. Analysis of these data shows linear increases in tumor incidence at a number of sites with increasing radiation dose and younger age at exposure. There are other data suggesting humans are more susceptible to chemical carcinogens when exposure occurs in childhood. These data exist for tobacco smoke (Marcus et al., 2000; Wiencke et al., 1999) and chemotherapy and radiation (Mauch et al., 1996; Swerdlow et al., 2000; Franklin et al., 2006).

Proposed Age Bins for Application of Default Age Sensitivity Factors

In developing a default science-based risk assessment policy to address this general conclusion, one key variable to define is the age interval or intervals over which age-dependent sensitivity factors should be applied. Different investigators have considered different age ranges, but in general the more sensitive period has at least been defined as including the time from birth up to mid-adolescence when the major phases of growth and hormonal change are complete. It is also recognized that, apart from the dramatic prenatal developmental events, the earliest postnatal stages represent the greatest differences in physiology and biochemistry from the adult. This reflects the immaturity of many organ systems, extremely rapid growth, and the incomplete maturation of various metabolic capabilities. As noted earlier, the rodent age bins in OEHHA's analysis were based on gross developmental milestones (birth, weaning, sexual maturity). OEHHA's analysis of studies that included exposure sometime between birth and weaning indicated this period as having the highest sensitivity to carcinogenesis. The data for the later juvenile period (postnatal days 22 to ~ 49) are somewhat sparse, covering only three carcinogens and only one where there are corresponding data for both postnatal and juvenile lifestages. However, it appears based on the overall range of potency ratios observed for the juvenile period that sensitivity to many carcinogens is elevated in this period also, but to a lesser extent than during the first 22 days. [Hattis et al. (2005) and Barton et al. (2005) report analyses for exposures at any time during the juvenile period, i.e. up to 6-8 weeks, and do not separate by additional age bins].

Weaning is not such an obvious or consistently timed transition for humans, being subject to a wide range of cultural and economic variables. However, it is generally considered that the human infant period encompasses the first two years of life. This period includes the most rapid periods of cellular division and differentiation for the major organ systems (excluding the breast and reproductive organs). Although there is linear growth between 2 and 8 years of age, the organ development is largely although not entirely complete.

Thus, considering both the development of major organ systems and the associated differences in toxicodynamic and toxicokinetic factors, OEHHA initially proposed to apply the postnatal ASF derived from rodent studies (birth to 21 days) to the human age intervals of birth - < 2 years. Similarly, OEHHA chose to apply the "juvenile" ASF derived from rodent studies (22 - ~49 days) to the human ages 2 - < 16 years. This timetable was also selected by U.S. EPA (2005) in their supplemental guidance for assessing early-life susceptibility to carcinogens. They describe their choice of critical periods as follows:

> "The adjustments described below reflect the potential for early-life exposure to make a greater contribution to cancers appearing later in life. The 10-fold adjustment represents an approximation of the weighted geometric mean tumor incidence ratio from juvenile or adult exposures in the repeated dosing studies (see Table 8). This adjustment is applied for the first 2 years of life, when toxicokinetic and toxicodynamic differences between children and adults are greatest (Ginsberg et al., 2002; Renwick, 1998). Toxicokinetic differences from adults, which are greatest at birth, resolve by approximately 6 months to 1 year, while higher growth rates extend for longer periods. The 3-fold adjustment represents an intermediate level of adjustment that is applied after 2 years of age through <16 years of age. This upper age limit represents middle adolescence following the

period of rapid developmental changes in puberty and the conclusion of growth in body height in NHANES data (Hattis et al., 2005). Efforts to map the approximate start of mouse and rat bioassays (i.e., 60 days) to equivalent ages in humans ranged from 10.6 to 15.1 years (Hattis et al., 2005)."

There is general agreement that rodents are born at a maturational stage approximately equivalent to a third trimester human fetus. Thus, there is good rationale to include the third trimester of pregnancy in the age bin for application of the ASF of 10. Therefore, OEHHA is applying the ASF of 10 for exposures during the third trimester of pregnancy to age 2. The default ASF values used by OEHHA are summarized in Table 2.

While there is strong evidence that growth and therefore cell proliferation rates and cell differentiation are extremely high prior to age 2, and lower (although still elevated relative to the adult) thereafter, there is still residual uncertainty with respect to the cut point for application of the ASFs of 10 and 3. Thus, another possible approach would be to move the cut point for the application of the ASF of 10 to a later age to account for this uncertainty. We present the effect on risk estimates of varying cut points in Table 3 and Table 4.

Table 2. Default Age Sensitivity Factors to be used to estimate cancer risks to infants and children

R (third trimester to age 2yrs)	10
R (age 2 to age 16 yrs)	3
R (age 16 to 70 yrs)	1

Application of ASFs in Risk Assessment

The effect of using the proposed default ASFs in calculating cancer risk over a 70 year lifetime, and for a 9 year exposure common in the Hot Spots program risk assessments is demonstrated in Table 3 and Table 4 below. Ignoring for the moment the increased exposures to carcinogens that children experience, the effect of the weighting factors is to increase the lifetime cancer risk by about 2. For risks from shorter exposures, such as the commonly used 9 year exposure scenario, OEHHA proposes to evaluate risk from exposures starting at the third trimester in the surrounding general population. The weighting factors in this case increase the risk to a larger extent. Depending on the exposure scenario, the use of age-specific distributions for uptake rates for air, food and water would also increase the risk estimates significantly independent of any application of ASFs. This is because the uptake rates for all these media per unit of body weight are higher in children and, especially, infants.

Assessing risks to short-term exposures to carcinogens involves additional uncertainties. The cancer potency factors are generally based on long-term exposures. However, in reality, the local air districts in California are frequently assessing risk from short term activities related to construction, mitigation of contaminated soils, and so forth. OEHHA recommends that when assessing such shorter term projects, the districts assume a minimum of 2 years of exposure and apply the slope factors and the 10 fold ASF to such assessments. Exposure durations longer than 2 years would use the method for the remaining years as noted above.

Table 3. Example of default ASF use for a lifetime exposure (not adjusted for age-specific exposure)

Carcinogen Potency = 1 $(mg/kg\text{-}d)^{-1}$
Exposure = 0.0001 mg/kg-d
No consideration of differences of exposure

No adjustment: Lifetime Risk = potency × dose			Risk
70 year Lifetime risk = 1 × 0.0001			**1.0×10^{-4}**

With proposed default ASF of 10 for third trimester to age 2, and 3 for ages 2 to 16 years: LR = Σ (potency x dose x ASF x fraction of lifetime)	ASF	Duration	Risk
R (third trimester to age 2yrs)	10	2.25/70	0.321×10^{-4}
R (age 2 to age 16 yrs)	3	14/70	0.600×10^{-4}
R (age 16 to 70 yrs)	1	54/70	0.771×10^{-4}
70 year Lifetime Risk			**1.7×10^{-4}**

For comparison, if ASF of 10 were applied to age 5, and ASF of 3 for the ages 5 to 16 years: LR = Σ (potency x dose x ASF x fraction of lifetime)	ASF	Duration	Risk
R (birth to age 5)	10	5.25/70	0.750×10^{-4}
R (age 5 to 16 yrs)	3	11/70	0.471×10^{-4}
R (age 16 to 70 yrs)	1	54/70	0.771×10^{-4}
70 year Lifetime Risk			**2.0×10^{-4}**

Table 4. Example of default ASF use for a 9-year exposure

Carcinogen Potency = 1 $(mg/kg\text{-}d)^{-1}$
Exposure = 0.0001 mg/kg-d
No consideration of differences of exposure

No adjustment: Total Risk = potency × dose x fraction of lifetime		Duration	Risk
9-year Total Risk		9/70	**0.13×10^{-4}**
With default ASF of 10 for third trimester to age 2 and 3 thereafter: LR = Σ (potency x dose x ASF x fraction of lifetime)	ASF	Duration	Risk
R (third trimester to age 2yrs)	10	2.25/70	0.321×10^{-4}
R (age 2 to 9 yrs)	3	7/70	0.300×10^{-4}
9 year Total Risk			**0.62×10^{-4}**
For comparison, if ASF of 10 applied to age 5, and ASF of 3 thereafter: LR = Σ (potency x dose x ASF x fraction of lifetime)	ASF	Duration	Risk
R (birth to age 5 yrs)	10	5/70	0.750×10^{-4}
R (age 5 to 9 yrs)	3	4/70	0.171×10^{-4}
9 year Total Risk			**0.92×10^{-4}**

Special Consideration of Puberty

In addition to the general concerns over increased sensitivity to carcinogenesis during infancy and childhood, there are specific concerns for exposure during the period when hormonal and developmental changes associated with puberty are in process, especially for carcinogens with hormonal modes of action or with impacts on the reproductive system and its accessory organs. At puberty, there is increased development of breast and reproductive organs that clearly involves rapid cellular division and differentiation. Thus, for carcinogens that induce mammary and reproductive organ cancers, puberty represents a time of increased sensitivity. As noted in the section on Selection of Default Age-Sensitivity Factors (page 50), if the risk assessor is evaluating a chemical with the potential for more than usually enhanced potency during this period, such as those which induce mammary or reproductive organ tumors (*e.g.*, a polycyclic aromatic hydrocarbon), then the risk assessment may use a larger ASF to calculate risk from exposure during puberty. OEHHA may recommend chemical-specific ASFs for puberty to the local air quality management districts for use in the Air Toxics Hot Spots program.

U.S.EPA Analysis of the Effect of Age at Exposure on Cancer Potency

U.S. EPA addressed the potential for increased susceptibility to cancer caused by environmental chemicals when the exposure occurs during an early lifestage in "Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens" (U.S. EPA, 2005b) (referred to henceforth as the Supplemental Guidance). This document is intended to be a companion to the revised "Guidelines for Carcinogen Risk Assessment" (U.S. EPA, 2005a). We present a summary of their analysis, which supports the policy decision to weight cancer potency and therefore risk by age-at-exposure. As previously noted, there are several methodological differences between the U.S. EPA analysis and the OEHHA analysis. Of note, in the OEHHA analysis all treatment-related tumors that were observed in a given lifestage exposure experiment were taken into account in estimating cancer potency. Thus in comparing cancer potencies associated with early life vs. adult exposure, OEHHA compared the total cancer risk associated with exposure during a given lifestage, rather than comparing the risk for cancers at one single site in each lifestage, as the U.S. EPA did. In addition, the age groupings are somewhat different in the U.S. EPA analysis from those used by OEHHA in their analysis (described above). For example, prenatal (*in utero*) exposures were not part of the analysis performed by U.S. EPA, and that Agency's analyses did not distinguish between postnatal and juvenile exposures.

U.S. EPA oral exposure cancer risk methodology relies on estimation of the lifetime average daily dose, which can account for exposure factor differences between adults and children (*e.g.*, eating habits and body weight). However, early lifestage susceptibility differences have not been taken into consideration when cancer potency factors were calculated. The Supplemental Guidance document focused on studies that define the potential duration and degree of increased susceptibility that may arise from early-life exposures. An analysis of those studies including a detailed description of the procedures used was published in Barton *et al*. (2005) (included as Appendix I). The criteria used to decide if a study could be included in the quantitative analysis are as follows (excerpted from U.S. EPA, 2005b):

1. Exposure groups at different post-natal ages in the same study or same laboratory, if not concurrent (to control for a large number of potential cross-laboratory experimental variables including pathological examinations),
2. Same strain/species (to eliminate strain-specific responses confounding age-dependent responses),
3. Approximately the same dose within the limits of diets and drinking water intakes that obviously can vary with age (to eliminate dose-dependent responses confounding age-dependent responses),
4. Similar latency period following exposures of different ages (to control for confounding latency period for tumor expression with age-dependent responses), arising from sacrifice at >1 year for all groups exposed at different ages, where early-life exposure can occur up to about 7 weeks. Variations of around 10 to 20% in latency period are acceptable,
5. Postnatal exposure for juvenile rats and mice at ages younger than the standard 6 to 8 week start for bioassays; prenatal (*in utero*) exposures are not part of the current analysis. Studies that have postnatal exposure were included (without adjustment) even if they also involved prenatal exposure,

6. "Adult" rats and mice exposure beginning at approximately 6 to 8 weeks old or older, *i.e.* comparable to the age at initiation of a standard cancer bioassay (McConnell, 1992). Studies with animals only at young ages do not provide appropriate comparisons to evaluate age-dependency of response (*e.g.*, the many neonatal mouse cancer studies). Studies in other species were used as supporting evidence, because they are relatively rare and the determination of the appropriate comparison ages across species is not simple, and
7. Number of affected animals and total number of animals examined are available or reasonably reconstructed for control, young, and adult groups (i.e., studies reporting only percent response or not including a control group would be excluded unless a reasonable estimate of historical background for the strain was obtainable).

Cancer potencies were estimated from a one-hit model (a restricted form of the Weibull time-to-tumor model), which estimates cumulative incidence for tumor onset. U.S. EPA (2005b) compared the estimated ratio of the cancer potency from early-life exposure to the estimated cancer potency from adult exposure. The general form of the equation for the tumor incidence at a particular dose, [P(dose)] is:

$$P(dose) = 1-[1-P(0)]\exp(-\text{cancer potency}*\text{dose})$$

where P(0) is the incidence of the tumor in controls. The ratio of juvenile to adult cancer potencies at a single site were calculated by fitting this model to the data for each age group. The model fit depended upon the design of the experiment that generated the data. Studies evaluated by U.S. EPA had two basic design types: experiments in which animals were exposed either as juveniles or as adults (with either a single or multiple dose in each period), and experiments in which exposure began either in the juvenile or in the adult period, but once started, continued through life.

The model equations for the first study type are:

$$P_A = P_0 + (1-P_0)\,(1-e^{-m_A\delta_A})$$

$$P_J = P_0 + (1-P_0)\,(1-e^{-m_A e^{\lambda}\,\delta_J})$$

where *A* and *J* refer to the adult and juvenile period, respectively, λ is the natural logarithm of the juvenile:adult cancer potency ratio, P_0 is the fraction of control animals with the particular tumor type being modeled, P_x is the fraction of animals exposed in age period *x* with the tumor, m_A is the cancer potency, and δ_x is the duration or number of exposures during age period *x*.

The goal of the model is to determine λ, which is the logarithm of the estimated ratio of juvenile to adult cancer potencies. This serves as a measure of potential susceptibility for early-life exposure.

For the second study type, the model equations take into account that exposures that were initiated in the juvenile period continue through the adult period. The model equations for the fraction of animals exposed only as adults with tumors in this design are the same as in the first study type, but the fraction of animals whose first exposure occurred in the juvenile period is:

$$P_J = P_0 + (1-P_0)\,(1-e^{-m_A e^{\lambda}(\delta_J - \delta_A) - m_A \delta_A})$$

δ_J includes the duration of exposure during the juvenile period and the subsequent adult period.

Parameters in these models were estimated using Bayesian methods and all inferences about the ratios were based on the marginal posterior distribution of λ. A complete description of these procedures (including the potential effect of alternative Bayesian priors that were examined) was published in Barton *et al*. (2005) (Appendix I). This method produced a posterior mean ratio of the early-life to adult cancer potency, which is an estimate of the potential susceptibility of early-life exposure to carcinogens. Ratios of greater or less than one indicate greater or less susceptibility from early-life exposure, respectively.

U.S. EPA reviewed several hundred studies reporting information on 67 chemicals or complex mixtures that are carcinogenic via perinatal exposure. Eighteen chemicals were identified which had animal study designs involving early-life and adult exposures in the same experiment. Of those 18 chemicals, there were overlapping subsets of 11 chemicals involving repeated exposures during early postnatal and adult lifestages and 8 chemicals using acute exposures (usually single doses) at different ages. Those chemicals are listed in Table 5.

Table 5 Chemicals having animal cancer study data available with early-life and adult exposures in the same experiment.

Chemical	Study Type
Amitrole	repeat dosing
Benzidine	repeat dosing
Benzo[a]pyrene (BaP)	acute exposure
Dibenzanthracene (DBA)	acute exposure
Dichlorodiphenyltrichloroethane (DDT)	lifetime exposure, repeat dosing
Dieldrin	lifetime exposure, repeat dosing
Diethylnitrosamine (DEN)	acute exposure, lifetime exposure
Dimethylbenz[a]anthracene (DMBA)	acute exposure
Dimethylnitrosamine (DMN)	acute exposure
Diphenylhydantoin, 5,5-(DPH)	lifetime exposure, repeat dosing
Ethylnitrosourea (ENU)	acute exposure
Ethylene thiourea (ETU)	lifetime exposure, repeat dosing
3-Methylcholanthrene (3-MC)	repeat dosing
Methylnitrosourea (NMU)	acute exposure
Polybrominated biphenyls (PBBs)	lifetime exposure, repeat dosing
Safrole	lifetime exposure, repeat dosing
Urethane	acute exposure, lifetime exposure
Vinyl chloride (VC)	repeat dosing

U.S. EPA calculated the difference in susceptibility between early-life and adult exposure as the estimated ratio of cancer potency at specific sites from early-life exposure over the cancer potency from adult exposure for each of the studies that were determined qualitatively to have appropriate study designs and adequate data. The results were grouped into four categories: 1) mutagenic chemicals administered by a chronic dosing regimen to adults and repeated dosing in the early postnatal period (benzidine, diethylnitrosamine, 3-methylcholanthrene, safrole, urethane and vinyl chloride); 2) chemicals without positive mutagenicity data administered by a chronic dosing regimen to adults and repeated dosing in the early postnatal period (amitrole, dichlorodiphenyltrichloroethane (DDT), dieldrin, ethylene thiourea, diphenylhydantoin, polybrominated biphenyls); 3) mutagenic chemicals administered by an acute dosing regimen (benzo[*a*]pyrene, dibenzanthracene, diethylnitrosamine, dimethylbenzanthracene, dimethyl-nitrosamine, ethylnitrosourea, methylnitrosourea and urethane); 4) chemicals with or without positive mutagenicity data with chronic adult dosing and repeated early postnatal dosing.

The acute dosing animal cancer studies were considered qualitatively useful by U.S. EPA because they involve identical exposures with defined doses and time periods demonstrating that differential tumor incidences arise exclusively from age-dependent susceptibility. However, they

were not used to derive a quantitative cancer potency factor age adjustment, primarily because most of the studies used subcutaneous or intraperitoneal injection as a route of exposure. These methods have not been considered quantitatively relevant routes of environmental exposure for human cancer risk assessment by U.S. EPA, for reasons including the fact that these routes of exposure are expected to have a partial or complete absence of first pass metabolism which could affect potency estimates. Additionally, U.S. EPA decided that cancer potency estimates are usually derived from chronic exposures, and therefore, any adjustment to those potencies should be from similar exposures.

The repeated dosing studies with mutagenic chemicals using exposures during early postnatal and adult lifestages were used to develop a quantitative cancer potency factor age adjustment. Studies with repeated early postnatal exposure were included in the analysis even if they also involved earlier maternal and/or prenatal exposure, while studies addressing only prenatal exposure were not used in the analysis. The weighted geometric mean susceptibility ratio (juvenile to adult) for repeated and lifetime exposures in this case was 10.4 (range 0.12 – 111, 42% of ratios greater than 1).

USEPA suggests the use of age-dependent-adjustment factors (ADAF) for chemicals acting through a mutagenic mode of action., based on the results of the preceding analysis, which concluded that cancer risks generally are higher from early-life exposure than from similar exposure doses and durations later in life:

1. For exposures before 2 years of age (i.e., spanning a 2-year time interval from the first day of birth until a child's second birthday), a 10-fold ADAF.

2. For exposures between 2 and <16 years of age (i.e., spanning a 14-year time interval from a child's second birthday until their sixteenth birthday), a 3-fold ADAF.

3. For exposures after turning 16 years of age, no adjustment (ADAF=1).

The ADAF of 10 used for the 0 – 2 years of age range is approximately the weighted geometric mean cancer potency ratio from juvenile versus adult exposures in the repeated dosing studies. U.S. EPA considered this period to display the greatest toxicokinetic and toxicodynamic differences between children and adults. Data were not available to calculate a specific dose-response adjustment factor for the 2 to <16-year age range, so EPA selected an ADAF of 3 because it was half the logarithmic scale difference between the 10-fold adjustment for the first two years of life and no adjustment (*i.e.*, 1-fold) for adult exposure. The ADAF of 3 represents an intermediate level of adjustment applied after 2 years of age through <16 years of age. The upper age limit (16 years of age) reflects the end of puberty and the attainment of a final body height. U.S. EPA recognizes that the use of a weighted geometric mean of the available study data to develop an ADAF for cancer potencies may either overestimate or underestimate the actual early-life cancer potency for specific chemicals, and therefore emphasizes in the Supplemental Guidance that chemical-specific data should be used in preference to these default adjustment factors whenever such data are available.

U.S. EPA is recommending the ADAFs described above only for mutagenic carcinogens, because the data for non-mutagenic carcinogens were considered to be too limited and the modes

of action too diverse to use this as a category for which a general default adjustment factor approach can be applied. OEHHA considers this approach to be insufficiently health protective. There is no obvious reason to suppose that the toxicokinetics of non-mutagens would be systematically different from those of mutagens. It would also be inappropriate to assume by default that non-mutagenic carcinogens are assumed to need a toxicodynamic correction factor of 1. Most if not all of the factors that make individuals exposed to carcinogens during an early-lifestage potentially more susceptible than those individuals exposed during adulthood also apply to non-mutagenic carcinogen exposures (*e.g.*, rapid growth and development of target tissues, potentially greater sensitivity to hormonal carcinogens, differences in metabolism). It should also be noted that carcinogens that do not cause gene mutations may still be genotoxic by virtue of causing chromosomal damage. Additionally, many carcinogens do not have adequate data available for deciding on a specific mode of action, or do not necessarily have a single mode of action. For these reasons, OEHHA will apply the default cancer potency factor age adjustments described above to all carcinogens unless data are available which allow for the development of chemical-specific cancer potency factor age adjustments. In those cases, an agent-specific model of age dependence (based on observational or experimental data) might be used, or alternative (larger or smaller) adjustment factors and age ranges may be applied where understanding of the mechanism of action and target tissues makes this appropriate.

Other Source Documents for Cancer Risk Assessment Guidance

As noted previously, the cancer potencies and unit risks tabulated in this technical support document have been developed by various programs over a number of years. The methods used therefore necessarily varied according to the date of the assessment and the program responsible. The following section summarizes the sources and procedures most commonly applied, and their historical context where this is apposite.

United States Environmental Protection Agency (U.S. EPA)

The U.S. EPA was one of the first regulatory agencies to develop and apply cancer risk assessment methodology. Their guidance documents and technical publications have been influential for many programs, including the California Air Toxics (Toxic Air Contaminants and Hot Spots) programs.

Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1986)

Prior to the more recent guidelines updating project which, after nearly ten years of internal and public review drafts culminated in the 2005 final revision (see below), U.S. EPA carcinogen risk assessment procedures were generally as described in Anderson *et al*. (1983) and "Guidelines for Carcinogen Risk Assessment" (U.S. EPA, 1986). These methods, which are outlined below, were used to calculate the Integrated Risk Information System (IRIS) cancer potency values, some of which are cited in this document. U.S. EPA has always indicated that cancer risk estimates based on adequate human epidemiologic data are preferred if available over estimates based on animal data. Although the newer guidelines offer alternative methods for dose-response analysis of animal bioassays, and updated consideration of specific topics such as lifestage-related differences in sensitivity, and mechanism of action for some types of carcinogen, the underlying principles and many of the specific procedures developed in these original guidelines are still applicable and in use.

U.S. EPA Calculation of Carcinogenic Potency Based on Animal Data

In extrapolating low-dose human cancer risk from animal carcinogenicity data, it is generally assumed that most agents that cause cancer also damage DNA, and that the quantal type of biological response characteristic of mutagenesis is associated with a linear non-threshold dose-response relationship. U.S. EPA stated that the risk assessments made with this model should be regarded as conservative, representing the most plausible upper limit for the risk. The mathematical expression used by U.S. EPA in the 1986 guidelines to describe the linear non-threshold dose-response relationship at low doses is the linearized multistage procedure developed by Crump (1980). This model is capable of fitting almost any monotonically increasing dose-response data, and incorporates a procedure for estimating the largest possible linear slope at low extrapolated doses that is consistent with the data at all experimental dose levels. A description of the linearized multistage procedure has been provided above (page 29). U.S. EPA used an updated version (GLOBAL86, Howe *et al*., 1986) of the computer program GLOBAL79 developed by Crump and Watson (1979) to calculate the point estimate and the 95% upper confidence limit of the extra risk $A(d)$.

U.S. EPA separated tumor incidence data according to organ sites or tumor types. The incidence of benign and malignant tumors was combined whenever scientifically defensible. U.S. EPA considered this incidence combination scientifically defensible unless the benign tumors are not considered to have the potential to progress to the associated malignancies of the same histogenic origin. The primary comparison in carcinogenicity evaluation is tumor response in dosed animals as compared to contemporary matched control animals. However, U.S. EPA stated that historical control data could be used along with concurrent control data in the evaluation of carcinogenic responses, and notes that for the evaluation of rare tumors, even small tumor responses may be significant compared to historical data. If several data sets (dose and tumor incidence) are available (different animal species, strains, sexes, exposure levels, exposure routes) for a particular chemical, the data set used in the model was the set where the incidence is statistically significantly higher than the control for at least one test dose level and/or where the tumor incidence rate shows a statistically significant trend with respect to dose level. The data set generating the highest lifetime cancer risk estimate (q_1^*) was chosen where appropriate. An example of an inappropriate data set would be a set which generates an artifactually high risk estimate because of a very small number of animals used. If there are 2 or more data sets of comparable size for a particular chemical that are identical with respect to species, strain, sex and tumor sites, the geometric mean of q_1^* estimated from each of those data sets was used for risk estimation. U.S. EPA assumed that mg/surface area/day is an equivalent dose between species. Surface area was further assumed to be proportional to the 2/3 power of the weight of the animal in question. Equivalent dose was therefore computed using the following relationship:

$$\mathrm{d} = \frac{1_e * m}{L_e * W^{2/3}}$$

where L_e = experimental duration, l_e = exposure duration, m = average dose (mg/day) and W = average animal weight. Default average body weights for humans, rats and mice are 70, 0.35 and 0.03 kg, respectively.

Exposure data expressed as ppm in the diet were generally converted to mg/day using the relationship m = ppm * F * r, where ppm is parts per million of the chemical in the diet, F is the weight of the food consumed per day in kg, and r is the absorption fraction (assumed to be 1 in the absence of data indicating otherwise). The weight of food consumed, calories required, and animal surface area were generally all considered to be proportional to the 2/3 power of the animal weight, so:

$$m \propto \text{ppm} * W^{2/3} * r, \text{ or } \quad \frac{m}{rW^{2/3}} \propto \text{ppm}$$

The relationship could lead to the assumption that dietary ppm is an equivalent exposure between species. However, U.S. EPA did not believe that this assumption is justified, since the calories/kg food consumed by humans is significantly different from that consumed by laboratory animals (primarily due to differences in moisture content). An empirically derived food factor, $f = F/W$ was used, which is the fraction of a species' body weight consumed per day as food. U.S. EPA (1986) gave the f values for humans, rats and mice as 0.028, 0.05 and 0.13, respectively.

Dietary exposures expressed as concentrations in ppm were converted to mg/surface area using the following relationship:

$$\frac{m}{r * W^{2/3}} = \frac{\text{ppm} * F}{W^{2/3}} = \frac{\text{ppm} * f * W}{W^{2/3}} = \text{ppm} * f * W^{2/3}$$

Exposures expressed as mg/kg/day ($m/Wr = s$) were converted to mg/surface area using the relationship:

$$\frac{m}{rW^{2/3}} = s * W^{2/3}$$

The calculation of dose when exposure is via inhalation was performed for cases where 1) the chemical is either a completely water-soluble gas or aerosol and is absorbed proportionally to the amount of inspired air, or 2) where the chemical is a partly water-soluble gas which reaches an equilibrium between the inspired air and body compartments. After equilibrium is attained, the rate of absorption is proportional to metabolic rate, which is proportional to the rate of oxygen consumption, which is related to surface area.

Exposure expressed as mg/day to completely water-soluble gas or aerosols can be calculated using the expression $m = I * v * r$, where I is the inspiration rate/day in m^3, v is the concentration of the chemical in air (mg/m^3), and r is the absorption fraction (assumed to be the same for all species in the absence of data to the contrary; usually 1). For humans, the default inspiration rate of 20 m^3 has been adopted. Inspiration rates for 113 g rats and 25 g mice have been reported to be 105 and 34.5 liters/day, respectively. Surface area proportionality can be used to determine inspiration rate for rats and mice of other weights; for mice, $I = 0.0345\ (W / 0.025)^{2/3}$ m^3/day; for rats, $I = 0.105\ (W / 0.113)^{2/3}$ m^3/day. The empirical factors for air intake/kg/day (i) for humans, rats and mice are 0.29, 0.64 and 1.3, respectively. Equivalent exposures in mg/surface area can be calculated using the relationship:

$$\frac{m}{W^{2/3}} = \frac{Ivr}{W^{2/3}} = \frac{iWvr}{W^{2/3}} = iW^{1/3}vr$$

Exposure expressed as mg/day to partly water-soluble gases is proportional to surface area and to the solubility of the gas in body fluids (expressed as an absorption coefficient r for that gas). Equivalent exposures in mg/surface area can be calculated using the relationships $m = kW^{2/3} * v * r$, and $d = m/W^{2/3} = kvr$. The further assumption is made that in the case of route-to-route extrapolations (*e.g.*, where animal exposure is via the oral route, and human exposure is via inhalation, or vice versa), unless pharmacokinetic data to the contrary exist, absorption is equal by either exposure route.

Adjustments were made for experimental exposure durations shorter than the lifetime of the test animal; the slope q_1^* was increased by the factor $(L/L_e)^3$, where L is the normal lifespan of the experimental animal and L_e is the duration of the experiment. This assumed that if the average dose d is continued, the age-specific rate of cancer will continue to increase as a constant function of the background rate. Since age-specific rates for humans increase by at least the 2nd power of the age, and often by a considerably higher power (Doll, 1971), there is an expectation

that the cumulative tumor rate, and therefore q_1^*, will increase by at least the 3rd power of age. If the slope q_1^* is calculated at age L_e, it would be expected that if the experiment was continued for the full lifespan L at the same average dose, the slope q_1^* would have been increased by at least $(L/L_e)^3$.

U.S. EPA Calculation of Carcinogenic Potency Based on Human Data

U.S. EPA stated that existing human epidemiologic studies with sufficiently valid exposure characterization are always used in evaluating the cancer potency of a chemical. If they showed a carcinogenic effect, the data were analyzed to provide an estimate of the linear dependence of cancer rates on lifetime cancer dose (equivalent to the factor q_1^*). If no carcinogenic effect was demonstrated and carcinogenicity had been demonstrated in animals, then it was assumed that a risk does exist, but it is smaller than could have been observed in the epidemiologic study. An upper limit of cancer incidence was calculated assuming that the true incidence is just below the level of detection in the cohort studied, which is largely determined by the cohort size. Whenever possible, human data are used in preference to animal data. In human epidemiologic studies, the response is measured as the relative risk of the exposed cohort of individuals compared to the control group. The excess risk ($R(X)$ - 1, where $R(X)$ is relative risk) was assumed to be proportional to the lifetime average exposure X, and to be the same for all ages. The carcinogenic potency is then equal to $[R(X) - 1]/X$ multiplied by the lifetime risk at that site in the general population. According to this original procedure, the confidence limit for the excess risk was not usually calculated. This decision was ascribed to the difficulty in accounting for inherent uncertainty in the exposure and cancer response data. More recent assessments have taken the opposite view and attempted to calculate and characterize this uncertainty by determining confidence limits, *inter alia*.

Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005a)

U.S. EPA revised its "Guidelines for Carcinogen Risk Assessment" (referred to henceforth as the "U.S. EPA Guidelines") in 2005. Compared to the 1986 version of this document, more emphasis is placed on establishing a "mode of action" (MOA). The following excerpt provides a definition of this term:

> "The term "mode of action" is defined as a sequence of key events and processes, starting with interaction of an agent with a cell, proceeding through operational and anatomical changes, and resulting in cancer formation. A "key event" is an empirically observable precursor step that is itself a necessary element of the mode of action or is a biologically based marker for such an element. Mode of action is contrasted with "mechanism of action," which implies a more detailed understanding and description of events, often at the molecular level, than is meant by mode of action".

Cancer risk assessments performed under the prior U.S. EPA Guidelines sometimes included a MOA description. However, the 1986 U.S. EPA Guidelines did not explicitly mandate the development of a MOA description in cancer risk assessments.

The MOA information is then used to govern how a cancer risk assessment shall proceed. Tumor incidence data sets arising from a MOA judged to be not relevant to humans are not used

to extrapolate a cancer potency factor. If an MOA cannot be determined or is determined to have a low-dose linear dose-response and a nonmutagenic MOA, then a linear extrapolation method is used to develop a cancer potency factor. The same linear extrapolation is used for all lifestages, unless chemical specific information on lifestage or population sensitivity is available. Carcinogens that act via an MOA judged to have a nonlinear low-dose dose response are modeled using MOA data, or the RfD/RfC risk assessment method is used as a default. Adjustments for susceptible lifestages or populations are to be performed as part of the risk assessment process.

If a carcinogen is deemed to act via a mutagenic MOA, then the data from the MOA analysis is evaluated to determine if chemical-specific differences between adults and juveniles exist and can be used to develop a chemical-specific risk estimate incorporating lifestage susceptibility. If this cannot be done, then early-life susceptibility is assumed, and age-dependent adjustment factors (ADAFs) are applied as appropriate to develop risk estimates. In cases where it is not possible to develop a toxicokinetic model to perform cross-species scaling of animal tumor data sets which arise from oral exposures, the U.S. EPA Guidelines state that administered doses should be scaled from animals to humans on the basis of equivalence of $mg/kg^{3/4}$-d (milligrams of the agent normalized by the 3/4 power of body weight per day). This is a departure from the 1986 U.S. EPA guidelines, which used a 2/3 power of body weight normalization factor. Other adjustments for dose timing, duration and route are generally assumed to be handled in similar fashion to that described for the 1986 guidelines, although of course updated parameter values would be used where available.

The 2005 U.S. EPA Guidelines also use benchmark dose methodology (described above, page 27) to develop a "point-of departure" (POD) from tumor incidence data. For linear extrapolation, the POD is used to calculate a cancer potency factor, and for nonlinear extrapolation the POD is used in the calculation of a reference dose (RfD) or reference concentration (RfC).

It should be noted that none of the cancer potency factors listed in this document were obtained from U.S. EPA risk assessments performed under the 2005 U.S. EPA Guidelines. All U.S. EPA IRIS cancer potency values contained in this document were obtained from risk assessments using the 1986 U.S. EPA Guidelines.

Office of Environmental Health Hazard Assessment (OEHHA), California Environmental Protection Agency

The cancer risk assessment procedures originally used by the Office of Environmental Health Hazard Assessment (OEHHA) are outlined in "Guidelines for Chemical Carcinogen Risk Assessments and their Scientific Rationale" (referred to below as the Guidelines) (CDHS, 1985). These procedures were generally used in generating Toxic Air Contaminant (TAC) cancer potency values, standard Proposition 65 cancer potency values and Public Health Goal (PHG) cancer potency values. Expedited Proposition 65 cancer potency values depart somewhat from those procedures and are discussed separately below.

OEHHA cancer risk assessment methodology as described by CDHS (1985) generally resembled that used at that time by U.S. EPA (Anderson *et al*., 1983; U.S. EPA, 1986). OEHHA risk

assessment practice similarly reflects the evolution of the technical methodology (*e.g.*, as described in U.S. EPA, 2005a) since the original guidelines were published. The basic principles and procedures described below are still considered applicable. More recent additions to OEHHA cancer risk assessment methods such as the use of benchmark dose methodologies and early-lifestage cancer potency adjustments are discussed above. The Guidelines state that both animal and human data, when available, should be part of the dose-response assessment.

OEHHA Calculation of Carcinogenic Potency Based on Animal Data

The procedures used to extrapolate low-dose human cancer risk from animal carcinogenicity data assumed that a carcinogenic change induced in a cell is transmitted to successive generations of cell descendants, and that the initial change in the cell is an alteration (*e.g.*, mutation, rearrangement, etc.) in the cellular DNA. Non-threshold models are used to extrapolate to low-dose human cancer risk from animal carcinogenicity data.

Several models were proposed for extrapolating low-dose human cancer risk from animal carcinogenicity data in the original Guidelines. These models include the Mantel-Bryan method (log-probit model), the one-hit model, the linearized multistage procedure, the gamma multihit model, and a number of time-to-tumor models. The Guidelines stated that time-to-tumor models (i.e., a Weibull-in-time model) should be used for low-dose extrapolation in all cases where supporting data are available, particularly when survival is poor due to competing toxicity. However, the Guidelines also noted the difficulty of determining the actual response times in an experiment. Internal tumors are generally difficult to detect in live animals and their presence is usually detected only at necropsy. Additionally, use of these models often requires making the determination of whether a tumor was the cause of death, or was found only coincidentally at necropsy when death was due to other causes. Further, competing causes of death, such as chemical toxicity, may decrease the observed time-to-tumor for nonlethal cancers by allowing earlier necropsy of animals in higher dose groups. The linearized multistage (LMS) procedure was noted as being an appropriate method for dose extrapolation in most cases, with the primary exception being a situation in which sufficient empirical data are available to indicate a dose-response curve of a "quasi-threshold" type (*e.g.*, flat for two or three dose levels, then curving sharply upwards). In this case, the LMS procedure may underestimate the number of stages and overestimate the low-dose risks. In this case, the gamma multihit model was suggested as being a potential alternative. The Mantel-Bryan model was described as having little biological basis as applied to carcinogenesis, and being likely to underestimate risks at low doses. The Guidelines stated that this model should not be used for low dose extrapolation. More recent practice has departed from these original guidelines in some respects, for instance by experimenting with cell-proliferation based models in a few cases. However, the LMS model remained the preferred extrapolation model for most purposes. Some of the difficulties in achieving a satisfactory fit to tumor incidence data were found to be alleviated by application of toxicokinetic models and use of an internal rather than applied dose metric with the LMS model. This has resulted in the alternative models originally advocated (Gamma multihit, Mantel-Bryan) being mostly abandoned. As noted above (Dose-Response Assessment, page 23), the use of allegedly biologically based statistical models such as LMS has fallen from favor in recent years, and benchmark dose methodology has become the preferred method for extrapolating cancer potency values from animal cancer incidence data. However, it should also be noted that results

generated by the LMS model and benchmark dose methodology from the same data set are often quite similar.

The 1985 Guidelines stated that both animal and human data, when available, should be part of the dose-response assessment. Although preference was given to human data when these were of adequate quality, animal studies may provide important supporting evidence. Low-dose extrapolation of human cancer risk from animal carcinogenicity data was generally based on the most sensitive site, species and study demonstrating carcinogenicity of a particular chemical, unless other evidence indicates that the data set in question is not appropriate for use. Where both benign and malignant tumors are induced at the same site and the benign tumors are considered to have the potential to progress to malignant tumors, the incidence data for both types of tumors could be combined to form the basis for risk assessment. Pharmacokinetic data on chemical metabolism, effective dose at target site, or species differences between laboratory test animals and humans were considered in dose-response assessments when available. In performing exposure scaling from animals to humans, the "surface area" correction (correcting by the 2/3 power of body weight) was used unless specific data indicate that this should not be done. The Guidelines assumed that in the absence of evidence to the contrary, chemicals that cause cancer after exposure by ingestion will also cause cancer after exposure by inhalation, and vice versa. These original proposals have continued in use with little change except that currently, TAC and PHG cancer potency factor calculations use a 3/4 power of body weight correction for interspecies scaling, in line with current U.S. EPA practice. The standard Proposition 65 cancer potency factor calculations still use a 2/3 power correction because the cancer potency calculation method is specified in regulation (California Health and Safety Code 25249.5 *et seq.*).

Cancer unit risk factors [in units of $(\mu g/m^3)^{-1}$] have been calculated from cancer potency factors [in units of $(mg/kg\text{-}day)^{-1}$] using the following relationship:

$$UR = \frac{CPF * 20\ m^3}{70\ kg\ *\ CV}$$

where UR is the cancer unit risk, CPF is the cancer potency factor, 70 kg is the reference human body weight, 20 m^3 is the reference human inspiration rate/day, and CV is the conversion factor from mg to µg (= 1000). The cancer unit risk describes the excess cancer risk associated with an inhalation exposure to a concentration of 1 $\mu g/m^3$ of a given chemical; the cancer potency factor describes the excess cancer risk associated with exposure to 1 mg of a given chemical per kilogram of body weight.

It should be noted that although this default method is still used in deriving published cancer unit risk values, for site-specific risk assessments age-appropriate distributions and percentile values are used in the current version of the Hot Spots exposure assessment document. Where exposure to children occurs (as it does in most exposures to the general population surrounding a source site) it is also necessary to apply the age-specific adjustment factors for the appropriate durations in accordance with the guidance offered above (Page 30 *et seq.*).

OEHHA Calculation of Carcinogenic Potency Based on Human Data

Human epidemiologic studies with adequate exposure characterization are used to evaluate the cancer potency of a chemical. If they show a carcinogenic effect, the data are analyzed to provide an estimate of the linear dependence of cancer rates on lifetime cancer dose. The 1985 Guidelines stated that with continuous exposure, age-specific incidence continues to increase as a power function (*e.g.*, t^3 or t^4) of the elapsed time since initial exposure. Lifetime risks can be estimated by applying such a power function to the observed data and extrapolating beyond the actual followup period. OEHHA has generally undertaken the calculation of study power and confidence bounds on the potency estimate as important tools to establish the credibility of the estimate obtained and in comparing this with other estimates (from other human studies or from animal data). Due to the diversity in quality and type of epidemiological data, the specific approaches used in OEHHA risk assessments based on human epidemiologic studies vary on a case by case basis rather than following explicit general guidelines. Examples of the methods used can be observed in the Toxic Air Contaminant documents (these documents are listed in Appendix D: the methods used are described in the compound summaries provided in Appendix B).

Expedited Proposition 65 Cancer Risk Assessment Methodology

Expedited cancer potency values developed for several agents listed as carcinogens under Proposition 65 (California Health and Safety Code 25249.5 *et seq*.) were derived from selected animal carcinogenicity data sets of the Carcinogenic Potency Database (CPDB) of Gold *et al.* (1984, 1986, 1987, 1989, 1990, 1997) using default procedures specified in the administrative regulations for Proposition 65 (Title 22 California Code of Regulations [CCR] 12703). OEHHA hazard assessments usually describe all relevant data on the carcinogenicity (including dose-response characteristics) of the chemical under examination, followed by an evaluation of any pharmacokinetic and mechanistic (*e.g.*, genotoxicity) data. An evaluation of the data set for the chemical may indicate that adjustments in target dose estimates or use of a dose response model different from the default are appropriate. The procedure used to derive expedited Proposition 65 cancer potency values differs from the usual methodology in two ways. First, it relies on cancer dose response data evaluated and extracted from the original literature by Gold *et al.* Second, the choice of a linearized multistage procedure for generating cancer potency values is automatic, and pharmacokinetic adjustments are not performed. The methods used to develop expedited cancer potency values incorporate the following assumptions:

1. The dose response relationship for carcinogenic effects in the most sensitive species tested is representative of that in humans.
2. Observed experimental results can be extrapolated across species by use of the interspecies factor based on "surface area scaling."
3. The dose to the tissue giving rise to a tumor is assumed to be proportional to the administered dose.
4. The linearized multistage polynomial procedure can be used to extrapolate potency outside the range of experimental observations to yield estimates of "low" dose potency.
5. Cancer risk increases with the third power of age.

The Carcinogenic Potency Database of Gold *et al.* (1984, 1986, 1987, 1989, 1990) contains the results of more than 4000 chronic laboratory animal experiments on 1050 chemicals by combining published literature with the results of Federal chemical testing programs (Technical Reports from the Carcinogenesis Bioassay Program of the National Cancer Institute (NCI)/National Toxicology Program (NTP) published prior to June 1987). The published literature was searched (Gold *et al*., 1984) through the period December 1986 for carcinogenicity bioassays; the search included the Public Health Service publication "Survey of Compounds Which Have Been Tested for Carcinogenic Activity" (1948-1973 and 1978), monographs on chemical carcinogens prepared by the International Agency for Research on Cancer (IARC) and Current Contents. Also searched were Carcinogenesis Abstracts and the following journals: British Journal of Cancer, Cancer Letters, Cancer Research, Carcinogenesis, Chemosphere, Environmental Health Perspectives, European Journal of Cancer, Food and Chemical Toxicology, Gann, International Journal of Cancer, Journal of Cancer Research and Clinical Oncology (formerly Zeitschrift fur Krebsforschung und Klinische Onkologie), Journal of Environmental Pathology and Toxicology, Journal of Toxicology and Environmental Health, Journal of the National Cancer Institute, and Toxicology and Applied Pharmacology. Studies were included in the database if they met the following conditions:

1. The test animals were mammals.
2. Chemical exposure was started early in life (100 days of age or less for hamsters, mice and rats).
3. Route of administration was via the diet, drinking water, gavage, inhalation, intravenous injection or intraperitoneal injection.
4. The test chemical was administered alone (not in combination with other chemicals).
5. Chemical exposure was chronic (*i.e*. duration of exposure was at least one-fourth the standard lifespan for that species), with not more than 7 days between exposures.
6. The experiment duration was at least half the standard lifespan for the species used.
7. The study design included a control group and at least 5 animals/exposure group.
8. No surgical interventions were performed.
9. Pathology data were reported for the number of animals with tumors (not total number of tumors).
10. All results reported were original data (not analysis of data reported by other authors).

Included in their data set tabulations are estimates of average doses used in the bioassay, resulting tumor incidences for each of the dose levels employed for sites where significant responses were observed, dosing period, length of study and histopathology. Average daily dose levels were calculated assuming 100% absorption. Dose calculations follow procedures similar to those of Cal/EPA and U.S. EPA; details on methods used and standard values for animal lifespans, body weights, and diet, water and air intake are listed in Gold *et al.* (1984). OEHHA (1992) reviewed the quality assurance, literature review, and control procedures used in compiling the data and found them to be sufficient for use in an expedited procedure. Cancer potency estimates were derived by applying the mathematical approach described in the section below to dose response data in the Gold *et al.* database.

The following criteria were used for data selection:

1. Data sets with statistically significant increases in cancer incidence with dose ($p \leq 0.05$) were used. (If the authors of the bioassay report considered a statistically significant result to be unrelated to the exposure to the carcinogen, the associated data set was not used.)
2. Data sets were not selected if the endpoint was specified as "all tumor-bearing animals" or results were from a combination of unrelated tissues and tumors.
3. When several studies were available, and one study stood out as being of higher quality due to numbers of dose groups, magnitude of the dose applied, duration of study, or other factors, the higher quality study was chosen as the basis for potency calculation if study results were consistent with those of the other bioassays listed.
4. When there were multiple studies of similar quality in the sensitive species, the geometric mean of potencies derived from these studies was taken. If the same experimentalists tested two sexes of the same species/strain under the same laboratory conditions, and no other adequate studies were available for that species, the data set for the more sensitive sex was selected.
5. Potency was derived from data sets that tabulate malignant tumors, combined malignant and benign tumors, or tumors that would have likely progressed to malignancy.

Cancer potency was defined as the slope of the dose response curve at low doses. Following the default approach, this slope was estimated from the dose response data collected at high doses and assumed to hold at very low doses. The Crump linearized multistage polynomial (Crump *et al.*, 1977) was fit to animal bioassay data:

$$\text{Probability of cancer} = 1 - \exp[-(q_0 + q_1 d + q_2 d^2 + ...)]$$

Cancer potency was estimated from the upper 95% confidence bound on the linear coefficient q_1, which is termed q_1^*.

For a given chemical, the model was fit to a number of data sets. As discussed in the section above, the default was to select the data for the most sensitive target organ in the most sensitive species and sex, unless data indicated that this was inappropriate. Deviations from this default occur, for example, when there are several bioassays or large differences exist between potency values calculated from available data sets.

Carcinogenicity bioassays using mice and/or rats will often use an exposure duration of approximately two years. For standard risk assessments, this is the assumed lifespan for these species. Animals in experiments of shorter duration are at a lower risk of developing tumors than those in the standard bioassay; thus potency is underestimated unless an adjustment for experimental duration is made. In estimating potency, short duration of an experiment was taken into account by multiplying q_1^* by a correction factor equal to the cube of the ratio of the assumed standard lifespan of the animal to the duration of the experiment (T_e). This assumes that the cancer hazard would have increased with the third power of the age of the animals had they lived longer:

$$q_{animal} = q_1^* * (104 \text{ weeks}/T_e)^3$$

In some cases excess mortality may occur during a bioassay, and the number of initial animals subject to late occurring tumors may be significantly reduced. In such situations, the above described procedure can, at times, significantly underestimate potency. A time-dependent model fit to individual animal data (i.e., the data set with the tumor status and time of death for each animal under study) may provide better potency estimates. When Gold *et al.* indicated that survival was poor for a selected data set, a time-dependent analysis was attempted if the required data were available in the Tox Risk (Crump *et al.*, 1991) data base. The Weibull multistage model (Weibull-in-time; multistage-in-dose) was fit to the individual animal data.

To estimate human cancer potency, q_{animal} values derived from bioassay data were multiplied by an interspecies scaling factor (K; the ratio of human body weight (bw_h) to test animal body weight (bw_a), taken to the 1/3 power (Anderson *et al.*, 1983)):

$$K = (bw_h/bw_a)^{1/3}$$

Thus, cancer potency = $q_{human} = K * q_{animal}$

Chemical-specific Descriptions of Cancer Potency Value Derivations

Unit Risk and potency values for chemicals whose cancer potency values were obtained from Toxic Air Contaminant documents, standard or expedited Proposition 65 documents, U.S. EPA's Integrated Risk Information System (IRIS) documents and Health Effects Assessment Summary Table (HEAST) entries, or from other documents prepared by OEHHA's Air Toxicology and Epidemiology Branch or Pesticide and Environmental Toxicology Branch are presented in Appendix A. Information summaries for these chemicals are presented in Appendix B.

REFERENCES

Allegaert K, Verbesselt R, Rayyan M, Debeer A, de Hoon J (2007). Urinary metabolites to assess in vivo ontogeny of hepatic drug metabolism in early neonatal life. Methods Find Exp Clin Pharmacol 29(4):251-6.

Anderson EL and the Carcinogen Assessment Group of the U.S. Environmental Protection Agency (1983). Quantitative approaches in use to assess cancer risk. Risk Anal 3:277-295.

Anderson LM, Diwan BA, Fear NT, Roman E. (2000). Critical windows of exposure for children's health: Cancer in human epidemiological studies and neoplasms in animal models. Environ Health Perspect 108 (Suppl3):573-94.

Armitage P, Doll R. (1954). The age distribution of cancer and a multistage theory of carcinogenesis. Br J Cancer 8(1): 1-12.

Barone S Jr, Das KP, Lassiter LT, White LD. (2000). Vulnerable processes of nervous system development: a review of markers and methods. NeuroTox 21:15-36.

Barrett JC, Wiseman RW (1987). Cellular and molecular mechanisms of multistep carcinogenesis: relevance to carcinogen risk assessment. Environ Health Perspect 76:65-70.

Barton HA, Cogliano VJ, Flowers L, Valcovic L, Setzer RW, Woodruff TJ (2005). Assessing susceptibility from early-life exposure to carcinogens. Environ Health Perspect 113:1125-1133.

Bayer SA, Altman J, Russo RJ, Zhang X (1993) Timetables of neurogenesis in the human brain based on experimentally determined patterns in the rat. Neurotoxicology 14:83-144.

Benya TJ, Busey WM, Dorato MA, Berteau PE (1982). Inhalation carcinogenicity bioassay of vinyl bromide in rats. Toxicol Appl Pharmacol 64:367-379.

Bogen KT, Spear RC (1987). Integrating uncertainty and inter-individual variability in environmental risk assessment. Risk Anal 7:427-436.

Bogen KT, Witschi HP (2002). Lung tumors in A/J mice exposed to environmental tobacco smoke: estimated potency and implied human risk. Carcinogenesis 23:511-519.

Bogen KT (1994). Cancer potencies of heterocyclic amines found in cooked foods. Food Chem Toxicol 32: 505-515.

Bois FY, Gelman A, Jiang J, Maszle DR, Zeise L, Alexeeff G (1996). Population toxicokinetics of tetrachloroethylene. Arch Toxicol 70:347-55.

Bradford Hill A. 1971. Statistical evidence and inference. In: Principles of Medical Statistics, 9th ed., pp. 309-323. Oxford University Press, New York, NY.

California Department of Health Services (CDHS) (1985). Guidelines for Chemical Carcinogen Risk Assessments and Their Scientific Rationale. CDHS, Health and Welfare Agency, Sacramento, CA.

California Environmental Protection Agency (Cal/EPA) (1992). Expedited Cancer Potency Values and Proposed Regulatory Levels for Certain Proposition 65 Carcinogens. Office of Environmental Health Hazard Assessment, Reproductive and Cancer Hazard Assessment Section, Berkeley, CA.

Cresteil T (1998). Onset of xenobiotic metabolism in children: toxicological implications. Food Addit Contam 15 Suppl:45-51.

Crouch E, Wilson R (1979). Interspecies comparison of carcinogenic potency. J Toxicol Environ Health 5:1095-1118.

Crouch E (1992). MSTAGE (Version 1.1). E.A.C. Crouch, Cambridge Environmental Inc., 58 Buena Vista Road, Arlington, Massachusetts 02141.

Crump KS, Watson WW (1979). GLOBAL79: A FORTRAN program to extrapolate dichotomous animal carcinogenicity data to low doses. National Institute of Environmental Health Sciences, Contract No. 1-ES-2123.

Crump KS, Guess HA, Deal LL (1987). Confidence intervals and test of hypotheses concerning dose response relations inferred from animal carcinogenicity data. Biometrics 33:437-451.

Crump KS, Howe RB, Van Landingham C, Fuller WG (1991). TOXRISK Version 3. TOXicology RISK Assessment Program. KS Crump Division, Clement International Division, 1201 Gaines Street, Ruston LA 71270.

Crump KS (1980). An improved procedure for low-dose carcinogenic risk assessment from animal data. J Environ Pathol Toxicol 5:675-684.

Crump KS (1984). A new method for determining allowable daily intakes. Fundam Appl Toxicol 4:854-871.

Crump KS (1995). Calculation of benchmark doses from continuous data. Risk Anal 15:78-89.

Crump KS (2002). Critical issues in benchmark calculations from continuous data. Crit Rev Toxicol 32:133-153.

Dietert RR, Etzel RA, Chen D, Halonen M, Holladay SD, Jarabek AM, Landreth K, Peden DB, Pinkerton K, Smialowicz RJ, Zoetis T (2000). Workshop to identify critical windows of exposure for children's health: immune and respiratory systems work group summary. Environ Health Perspect (Suppl 3):483-90.

Doll R (1971). Weibull distribution of cancer: implications for models of carcinogenesis. J Royal Stat Soc A 13:133-166.

Drew RT, Boorman GA, Haseman JK, McConnell EE, Busey WM, Moore JA (1983). The effect of age and exposure duration on cancer induction by a known carcinogen in rats, mice, and hamsters. Toxicol Appl Pharmacol 68:120-130.

Elbarbry FA, McNamara PJ, Alcorn J (2007). Ontogeny of hepatic Cyp1A2 and Cyp2E1 expression in rat. J Biochem Mol Toxicol 21(1):41-50.

ERG (2008) Summary Report of the Peer Review Meeting: EPA's Draft Framework for Determining a Mutagenic Mode of Action for Carcinogenicity. Final Report. Submitted to Risk Assessment Forum, Office of the Science Advisor, U.S. Environmental Protection Agency, Washington D.C., by Eastern Research Group. May 23, 2008.

Finkel AM (1995). Toward less misleading comparisons of uncertain risks: the example of aflatoxin and alar. Environ Health Perspect 103:376-385

Franklin J, Pluetschow A, Paus M, et al. (2006). Secondary malignancy risk associated with treatment of Hodgkin's lymphoma: meta-analysis of the randomized trials. Annals of Oncology 17:1749-60.

Freireich EJ, Gehan EA, Rall DP, Schmidt LH, Skipper HE (1966). Quantitative comparison of toxicity of anticancer agents in mouse, rat, hamster, dog, monkey, and man. Cancer Chemother Rep 50:219-244.

Garshick E, Schenker MB, Munoz A, Segal M, Smith TJ, Woskie SR, Hammond SK, Speizer FE. (1988). A retrospective cohort study of lung cancer and diesel exhaust exposure in railroad workers. Am Rev Respir Dis 137: 820-825.

Gaylor D, Ryan L, Krewski D, Zhu Y (1998). Procedures for calculating benchmark doses for health risk assessment. Regul Toxicol Pharmacol 28:150-164.

Gaylor DW, Gold LS (1994). Quick estimate of the regulatory virtually safe dose based on the maximum tolerated dose for rodent bioassays. Regul Toxicol Pharmacol 22:57-63.

Gold L, de Veciana M, Backman G, Magaw R, Lopipero P, Smith M, Blumenthal M, Levinson R, Bernstein L, Ames B (1986). Chronological supplement to the Carcinogenic Potency Database: Standardized results of animal bioassays published through December 1984 and by the National Toxicology Program through May 1986. Environ Health Perspect 74:237-329.

Gold L, Sawyer C, Magaw R, Backman G, de Veciana M, Levinson R, Hooper N, Havender W, Bernstein L, Peto R, Pike M, Ames B (1984). A Carcinogenic Potency Database of the standardized results of animal bioassays. Environ Health Perspect 58:9-319.

Gold L, Slone T, Bernstein L (1989). Summary of carcinogenic potency and positivity for 492 rodent carcinogens in the Carcinogenic Potency Database. Environ Health Perspect 79:259-272.

Gold L, Slone T, Backman G, Eisenberg S, Da Costa M, Wong M, Manley N, Ames B (1990). Third chronological supplement to the Carcinogenic Potency Database; Standardized results of animal bioassays published through December 1986 and by the National Toxicology Program through June 1987. Environ Health Perspect 84:215-285.

Gold L, Slone T, Backman G, Magaw R, Da Costa M, Ames B (1987). Second chronological supplement to the Carcinogenic Potency Database; Standardized results of animal bioassays published through December 1984 and by the National Toxicology Program through May 1986. Environ Health Perspect 74:237-329.

Gold LS, Slone TH, Manley NB, Garfinkel GB, Rohrbach L, Ames BN (1997). Carcinogenic Potency Database. In: Handbook of Carcinogenic Potency and Genotoxicity Databases, Gold LS and Zeiger E, eds. CRC Press, Boca Raton, FL, pp. 1-605.

Hakkola J, Tanaka E, Pelkonen O (1998). Developmental expression of cytochrome P450 enzymes in human liver. Pharmacol Toxicol 82(5):209-17.

Hancock SL, Tucker MA, Hoppe RT (1993). Breast cancer after treatment of Hodgkin's disease. J Natl Cancer Inst 85:25-31.

Hattis D, Goble R, Chu M (2005). Age-related differences in susceptibility to carcinogenesis. II. Approaches for application and uncerta1inty analyses for individual genetically acting carcinogens. Environ Health Perspect 113:509-16.

Hattis D, Goble R, Russ A, Chu M, Ericson J (2004). Age-related differences in susceptibility to carcinogenesis: a quantitative analysis of empirical animal bioassay data. Environ Health Perspect 112:1152-1158.

Hattis D (1990). Pharmacokinetic principles for dose-rate extrapolation of carcinogenic risk from genetically active agents. Risk Anal 10:303-16.

Herbst AL, Scully RE (1970). Adenocarcinoma of the vagina in adolescence. A report of 7 cases including 6 clear-cell carcinomas (so-called mesonephromas). Cancer 25:745-757.

Herbst AL, Ulfelder H, Poskanzer DC (1971). Adenocarcinoma of the vagina. Association of maternal stilbestrol therapy with tumor appearance in young women. N Engl J Med 284:878-881.

Hines RN, McCarver DG (2002). The ontogeny of human drug-metabolizing enzymes: Phase I oxidative enzymes. J Pharmacol Exp Ther 300(2):355-60.

Hoel DG, Kaplan NL, Anderson MW (1983). Implication of nonlinear kinetics on risk estimation in carcinogenesis. Science 219:1032-1037.

Holsapple MP, West LJ, Landreth KS (2003). Species comparison of anatomical and functional immune system development. Birth Defects Research (Part B) 68:321-34.

Howe RB, Crump KS, Van Landingham C (1986). GLOBAL86: A computer program to extrapolate quantal animal toxicity data to low doses. Clement Associates, Inc., Ruston, LA.

Huff J (1999). Long-term chemical carcinogenesis bioassays predict human cancer hazards. Issues, controversies, and uncertainties. Ann N Y Acad Sci 895:56-79.

IARC (2006). Monographs on the Evaluation of Carcinogenic Risks to Humans: Preamble. International Agency for Research on Cancer, Lyon, France. Available at: http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf

Institute of Medicine (2004). Gulf War and Health: Updated literature review of Sarin. The National Academy of Sciences, National Academy Press, Washington, DC, pp 20-22. www.nap.edu

Lacroix D, Sonnier M, Moncion A, Cheron G, Cresteil T (1997). Expression of Cyp3A in the human liver - evidence that the shift between Cyp3A7 and Cyp3A4 occurs immediately after birth. Eur J Biochem. 247(2):625-34.

Lash TL, Aschengrau A (1999). Active and passive cigarette smoking and the occurrence of breast cancer. Am J Epidemiol 149:5-12.

Lilienfeld AM, Lilienfeld DE (1980). Foundations of Epidemiology. Oxford University Press, Oxford, England.

Marcus PM, Newman B, Millikan RC, Moorman PG, Baird DD, Oaguish B (2000). The association of adolescent cigarette smoking, alcoholic beverage consumption, environmental tobacco smoke, and ionizing radiation with subsequent breast cancer (United States). Cancer Causes Control 11:271-8.

Mauch PM, Kalish LA, Marcus KC, Coleman CN, Shulman LN, Krill E, Come S, Silver B, Canellos GP, Tarbell NJ (1996). Second malignancies after treatment for laporotomy staged IA-IIIB Hodgkin's disease: long-term analysis of risk factors and outcome. Blood 87:3625-32.

McConnell EE (1992). Comparative response in carcinogenesis bioassay as a function of age at first exposure. In: Guzelian P, Henry CJ, Olin SS, eds. Similarities and difference between children and adults: implications for risk assessment. ILSI Press, Washington, DC, pp. 66−67.

McDonald T, Komulainen H (2005). Carcinogenicity of the chlorination disinfection by-product MX. J Environ Sci Health Part C, 23:163–214.

McDonald T, Hoover S, Faust J, Rabovsky J, MacGregor MK, Sherman C, Sandy M, Zeise L (2003). Development of cancer potency estimates for California's Proposition 65. Poster at Society of Toxicology Annual Meeting. March 2003, Salt Lake City, UT. Abstract No. 687, Toxicol Sci 72, S-1, 142.

Monson RR (1986). Observations on the healthy worker effect. J Occup Med 28: 425-433.

Moolgavkar SH, Knudson AG Jr. (1981). Mutation and cancer: a model for human carcinogenesis. J Natl Cancer Inst 66:1037-1052.

Morabia A, Bernstein MS, Bouchardy I, Kurtz J, Morris MA (2000). Breast cancer and active and passive smoking: the role of the N-acetyltransferase 2 genotype. Am J Epidemiol 152:226-232.

Moysich KB, Menezes RJ, Michalek AM (2002). Chernobyl-related ionising radiation exposure and cancer risk: an epidemiological review. Lancet Oncol 3:269-279.

National Research Council (NRC) (1983). Risk Assessment in the Federal Government: Managing the Process. Committee on the Institutional Means for Assessment of Risks to Public Health. National Academy Press, Washington, DC.

National Research Council (NRC) (1990). Health Effects of exposure to low levels of ionizing radiation. BEIR V. Committee on the Biological Effects of Ionizing Radiation. National Academy Press, Washington, DC

National Research Council (NRC) (1994). Science and Judgment in Risk Assessment. Committee on Risk Assessment of Hazardous Air Pollutants, Board on Environmental Studies and Toxicology, Commission on Life Sciences. National Academy Press, Washington, DC.

O'Brien PC, Noller KL, Robboy SJ, Barnes AB, Kaufman RH, Tilley BC, Townsend DE (1979). Vaginal epithelial changes in young women enrolled in the National Cooperative Diethylstilbestrol Adenosis (DESAD) project. Obstet Gynecol 53:300-308.

Office of Environmental Health Hazard Assessment (OEHHA) (2001a). Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act. California Environmental Protection Agency, Sacramento, CA.

Office of Environmental Health Hazard Assessment (OEHHA) (2001b). Public Health Goals for chemicals in drinking water: Tetrachloroethylene. California Environmental Protection Agency, Sacramento, CA.

Office of Environmental Health Hazard Assessment (OEHHA) (1998). Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Part B: Health Effects. (Approved by the Scientific Review Panel April 22, 1998). California Environmental Protection Agency, Sacramento, CA.

Office of Environmental Health Hazard Assessment (OEHHA) (1992). Proposed Identification of Perchloroethylene as a Toxic Air Contaminant. Part B: Health Effects. (Approved by the Scientific Review Panel, 1991: revised 1992). California Environmental Protection Agency, Sacramento, CA.

Office of Environmental Health Hazard Assessment (OEHHA) (2005a). Air Toxics Hot Spots Program Risk Assessment Guidelines. Part II: Technical Support Document for Describing Available Cancer Potency Factors. California Environmental Protection Agency, Sacramento, CA.

Office of Environmental Health Hazard Assessment (OEHHA) (2005b). Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant. Part B: Health Effects. As approved by the Scientific Review Panel, June 24, 2005. California Environmental Protection Agency, Sacramento, CA.

Pinkerton KE, Joad JP (2000). The mammalian respiratory system and critical windows of exposure for children's health. Environ Health Perspect 108 (Suppl3):457-62.

Preston-Martin S (1989). Epidemiological studies of perinatal carcinogenesis. IARC Sci Publ 96:289-314.

Rice D, Barone S Jr. (2000). Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models. Environ Health Perspect 108 (suppl3):511-33.

Rothman K, Greenland S (1998). Modern Epidemiology. 2nd edition. Lippincott–Raven, Philadelphia, PA, pp. 133-134.

Rothman KJ, Greenland S (2005). Causation and causal inference in epidemiology. Am J Public Health 95 Suppl1:S144-S150.

Salmon AG, Monserrat L, Brown JP (1992). Use of a pharmacokinetic model in cancer risk assessment for vinyl bromide. Presented at the Society of Toxicology Annual Meeting, Seattle, WA, February 1992. Abstract: The Toxicologist 12(1): 96.

Shimada T, Yamazaki H, Mimura M, Wakamiya N, Ueng YF, Guengerich FP, Inui Y (1996). Characterization of microsomal cytochrome P-450 enzymes involved in the oxidation of xenobiotic chemicals in human fetal liver and adult lungs. Drug Metab Dispos 24(5):515-22.

Smith AH, Marshall G, Yuan Y, Ferreccio C, Liaw J, von Ehrenstein O, Steinmaus C, Bates MN, Selvin S (2006). Increased mortality from lung cancer and bronchiectasis in young adults after exposure to arsenic in utero and in early childhood. Environ Health Perspect 114:1293-1296.

Swerdlow AJ, Barber JA, Vaughan Hudson G, Cunningham D, Gupta RK, Hancock BW, Horwich A, Lister TA, Linch DC (2000). Risk of second malignancy after Hodgkin's disease in a collaborative British cohort: the relation to age at treatment. J Clin Oncology 18:498-509.

Travis CC,White RK (1988). Interspecific scaling of toxicity data. Risk Anal 8:119-125.

Treluyer JM, Gueret G, Cheron G, Sonnier M, Cresteil T (1997). Developmental expression of Cyp2C and Cyp2C-dependent activities in the human liver: in-vivo/in-vitro correlation and inducibility. Pharmacogenetics 7(6):441-52.

U.S. Dept. of Health and Human Services (U.S. DHHS) (1982). The health consequences of smoking: Cancer. A Report of the Surgeon General. United States Department of Health and Human Services. Pub No (PHS) 82-50179. Washington DC.

U.S. Dept. of Health and Human Services (U.S. DHHS) (1994). The health consequences of smoking: a report of the Surgeon General. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, Washington, DC.

U.S. Environmental Protection Agency (U.S. EPA) (1986). Guidelines for Carcinogen Risk Assessment. Federal Register 51:33992-34003.

U.S. Environmental Protection Agency (U.S. EPA) (1994). Estimating Radiogenic Cancer Risks. EPA 402-R-93-076. U.S. Environmental Protection Agency Washington, DC, June 1994.

U.S. Environmental Protection Agency (U.S. EPA) (1999). Cancer Risk Coefficients for Environmental Exposure to Radionuclides. Federal Guidance Report No. 13. EPA 402-R-99-001. Environmental Protection Agency, Office of Radiation and Indoor Air. Washington, DC, September 1999.

U.S. Environmental Protection Agency (U.S. EPA) (2002). A review of the reference dose and reference concentration process. Risk Assessment Forum, Washington, DC. EPA/630/P-02/002F. Available from: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=55365.

U.S. Environmental Protection Agency (U.S. EPA) (2005a). Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum, Washington, DC. EPA/630/P-03/001F.

U.S. Environmental Protection Agency (U.S. EPA) (2005b). Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. EPA/630/R-03/003F. Available from: http://www.epa.gov/iris/children032505.pdf.

Van Landingham CB, Allen BC, Shipp AM, Crump KS (2001). Comparison of the EU T25 single point estimate method with benchmark dose response modeling for estimating potency of carcinogens. Risk Anal 21:641-56.

Vieira I, Sonnier M, Cresteil T (1996). Developmental expression of Cyp2E1 in the human liver. Hypermethylation control of gene expression during the neonatal period. Eur J Biochem. 238(2):476-83.

Walthall K, Cappon GD, Hurtt ME, Zoetis T (2005). Postnatal development of the gastrointestinal system: a species comparison. Birth Defects Res Part B 74:132-56.

Watson RE, DeSesso JM, Hurtt ME, Cappon GD (2006). Postnatal growth and morphological development of the brain: a species comparison. Birth Defects Res Part B. 77:471-484.

Wen CP, Tsai SP, Gibson RL (1983). Anatomy of the healthy worker effect: A critical review. J Occup Med 25: 283-289.

Wiencke JK, Thurston SW, Kelsey KT, Varkonyi A, Wain JC, Mark EJ, Christiani DC (1999). Early age at smoking and tobacco carcinogen DNA damage in the lung. J Natl Cancer Inst 91:614-9.

Zeise L, Salmon AG, McDonald T, Painter P (1991). Cancer potency estimation. In: Risks of carcinogenesis from urethane exposure. Salmon AG and Zeise L, eds, CRC Press, Boca Raton, FL, pp 97-112.

Zoetis T, Hurtt ME (2003). Species comparison of anatomical and functional renal development. Birth Defects Res PartB 68:111-120.

EXHIBIT 6

In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines

Contract #11612

Prepared for:

Mr. Adewale Oshinuga
South Coast Air Quality Management District
21865 Copley Drive
Diamond Bar, CA. 91765

September 2013

Submitted by:

Drs. Wayne Miller, Kent C. Johnson, Thomas Durbin, and Ms. Poornima Dixit
University of California
CE-CERT
Riverside, CA 92521
951-781-7586
951-781-5790 (fax)

Disclaimer

The statements and conclusions in this report are those of the contractor and not necessarily those of the South Coast Air Quality Management District or other participating organizations and their employees. The mention of commercial products, their source, or their use in connection with material reported herein is not to be construed as actual or implied endorsement of such products.

Acknowledgments

This report was prepared at the University of California, Riverside, and Bourns College of Engineering-Center for Environmental Research and Technology (CE-CERT). The primary contributors to this report include Drs. Wayne Miller, Kent C. Johnson, Thomas D. Durbin, and Ms. Poornima Dixit. The authors thank the following organizations and individuals for their valuable contributions to this project. We acknowledge Messrs. Don Pacocha, Edward O'Neil, and Joe Valdez of CE-CERT for their assistance in carrying out the experimental program.

Also, we would like to thank the following: Mr. Scott Monday, Alfredo Ortiz, and the rest of the support team at the Air Resources Board for N_2O analysis and technical guidance. Scott was very helpful in coordinating samples and Alfredo was patient in performing the analysis with short notice.

Also, we would like to thank the following: T. J. Rafel at Western Truck Center and Steve South at EDCO disposal for the use of their SCR equipped diesel refuse haulers. Patrick McGuruk at Container Freight/EIT., LLC for the use of two class 8 diesel powered vehicles. John Vondriska at Moreno Valley Unified School District for the use of a propane powered school bus. David Chiu at Krisda Inc for the use of a Class 8 LPG vehicle. Doug Kollmyer from A to Z Bus Sales for the use of a DPF equipped diesel school bus. Ed Hardiman, Rudy Raya and their support team at Caltrans for the use of two diesel Navistar refuse haulers. Jonathan Evans and Mark Yragui at Cummins Cal Pacific for their assistance in locating SCR equipped diesel refuse haulers in California. Bennie Anselmo for his assistance in locating an SCR equipped diesel refuse hauler in Northern California.

Table of Contents

List of Figures

List of Tables

Acronyms and Abbreviations

40 CFR 1065 or 1065	Part 1065 of Title 40 of the Code of Federal Regulations
ARB	Air Resources Board
bs	brake specific
ACES	Advanced Collaborative Emissions Study
ARB	California Air Resources Board
ATS	aftertreatment system
BTEX	benzene, toluene, ethylbenzene, and xylenes
CBD	central business district
CE-CERT	College of Engineering-Center for Environmental Research and Technology (University of California, Riverside)
CFO	critical flow orifice
CFR	Code of Federal Regulations
CO	carbon monoxide
COV	coefficient of variation
CO_2	carbon dioxide
CVS	constant volume sampling
DOC	diesel oxidation catalyst
DPF	diesel particulate filter
DR	dilution ratio
ECM	engine control module
efuel	ECM fuel consumption rate
EGR	exhaust gas recirculation
EPA	United States Environmental Protection Agency
FEL	family emission limit
FID	flame ionization detector
GFM	gravimetric filter module
g/bhp-h	grams per brake horsepower hour
HD-UDDS	heavy duty urban dynamometer driving schedule
HPDI	high pressure direct injection
lpm	liters per minute
LDL	lower detection limit
MDL	minimum detection limit
MEL	CE-CERT's Mobile Emissions Laboratory
MFC	mass flow controller
MY	model year
NMHC	non-methane hydrocarbons
NTE	Not-to-exceed
NO_x	nitrogen oxides
OC	organic carbon
OCTA	Orange County Transit Authority
OEM	original equipment manufacturer
PEMS	portable emissions measurement systems
PM	particulate matter

RPM .....revolutions per minute
SCAQMD.....South Coast Air Quality Management District
SCR .....selective catalytic reduction
scfm.....standard cubic feet per minute
Tier 2, 3, or 4 .....federal emissions standards levels for off-road diesel engines
THC.....total hydrocarbons
TWC.....three way catalyst
UCR.....University of California at Riverside
ULSD .....ultralow sulfur diesel
WVU .....West Virginia University

Executive Summary

Heavy-duty diesel vehicles are a major contributor to diesel emissions in the South Coast Air Basin. While emission measurements of these vehicles in engine dynamometer certification laboratories are showing nitrogen oxides (NO_x) and particulate matter (PM) emissions meeting the U.S. Environmental Protection Agency's (EPA's) and California Air Resources Board's (CARB's) emissions standards, some values from in-use conditions are showing increased emissions of ammonia from liquefied natural gas (LNG) trucks and of NO_x from diesel trucks. As such, additional studies are required to assess the impact of technology on emissions from heavy-duty engines used in variety of heavy-duty applications. The objective of this study was to carry out chassis dynamometer testing of heavy-duty natural gas and diesel vehicles using near-certification and in-use driving cycles while measuring: 1) regulated emissions; 2) unregulated emissions such as ammonia and formaldehyde; 3) greenhouse gas levels of carbon dioxide (CO_2) and nitrous oxide (N_2O); and 4) ultrafine PM emissions.

In December 2010 and October 2011, the SCAQMD Board awarded contracts to University of California, Riverside (UCR) and West Virginia University (WVU) to conduct chassis dynamometer testing of twenty-four model year (MY) 2007-2012 heavy-duty vehicles from different vocations and fueling technologies, and if necessary, to evaluate emission-reduction potential of retrofit technology for ammonia emissions from a natural gas heavy-duty engine. The test vehicle vocations included goods movement, refuse, transit and school bus applications, and the test cycles used for the specific vocations were port drayage truck cycles for goods movement, SCAQMD refuse truck cycles for the refuse applications, and Orange County Transportation Authority (OCTA) and Central Business District (CBD) cycles for transit applications. The Heavy Duty Urban Dynamometer Driving Schedule (HD-UDDS) was a common cycle for all vocations. The test matrix involved five natural gas and four dual-fuel vehicles to be tested on a chassis dynamometer by WVU, eight diesel and two propane vehicles tested by UCR, and five diesel vehicles tested by both WVU and UCR for inter-laboratory comparison. The heavy-duty natural gas engines were both stoichiometric fueled and three-way catalytic converter (TWC) equipped; lean burn high-pressure direct injection (HPDI) engines were equipped with diesel particulate filters DPFs and selective catalytic reduction (SCR) technology. Diesel engines tested in were either U.S. EPA 2007 emissions compliant or U.S. EPA 2010 emissions compliant. The U.S. EPA 2007 emissions compliant engines were equipped with exhaust gas recirculation (EGR) technology and DPFs, while the U.S. EPA 2010 emissions compliant engines were of two types: a) with EGR and DPF only b) with DPF and SCR.

The emission results for PM and NO_x are summarized below:

- PM emissions from the diesel test vehicles were below 0.01 grams per brake horsepower-hour (g/bhp-h) measured over port drayage, CBD, and UDDS drive cycles. Cold start PM emissions were relatively high for two diesel vehicles; one was a port SCR equipped vehicle and the other was a refuse SCR equipped vehicle. The port vehicle was 17 times higher (22.9 mg/mi vs 1.33 mg/mi) and the refuse vehicle was 8 times higher (18.4 mg/mi vs 2.75 mg/mi). In both cases the high cold start emission factors were below the certification standard. PM emissions were well below the certification for all diesel tests, thus suggesting DPF-based solutions are robust and reliable in meeting targeted standards. In addition, PM emissions from a liquefied petroleum gas (LPG) test vehicle was approximately 0.14 g/bhp-hr measured over the UDDS cycle, which is above the certification standard.

- NO_x results covered a wide range of emission factors, where the emissions depended on the certification standard, vehicle application, driving cycle, and manufacturer. For example, NO_x emissions were lowest for goods movement vehicles powered by diesel engines equipped with SCR technology; however, increases from 0.112 g/mi (0.028 g/bhp-h) during high speed cruise operation to 5.36 g/mi (1.34 g/bhp-h) for low speed transient operation were measured. Unique to the high NO_x emissions was a condition in which the temperature of the SCR was less than 250°C. Advanced EGR 2010 certified engines showed higher NO_x emissions compared to SCR equipped engines, and pre-2010 certified engines were higher than the 2010 certified engines.
- The NO_x impact of SCR equipped diesel engines depends on the vehicles' duty cycles and manufacturers' implementation for low temperature SCR performance. For the near dock port cycle, the SCR was below 250°C approximately 80% of the time, 65% of the time for the local port cycle, and approximately 45% of the time for the regional port cycle. The percentage of time below 250°C varied significantly between manufacturers, from 8% to 30% for the near dock cycle, and from 41% to 64% for the regional cycle. The difference in time below 250°C suggests some manufacturers have better strategies for maintaining high exhaust temperatures than others.
- The SCR equipped engines were within their certification standards and were typically below 0.2 g/bhp-h. Only during low SCR temperature were the emissions found to be higher than the certification standard. In-use compliance testing does not enforce the emissions standards when the SCR is below 250 °C, thus the SCR equipped vehicles were typically compliant based on the results presented in this report.

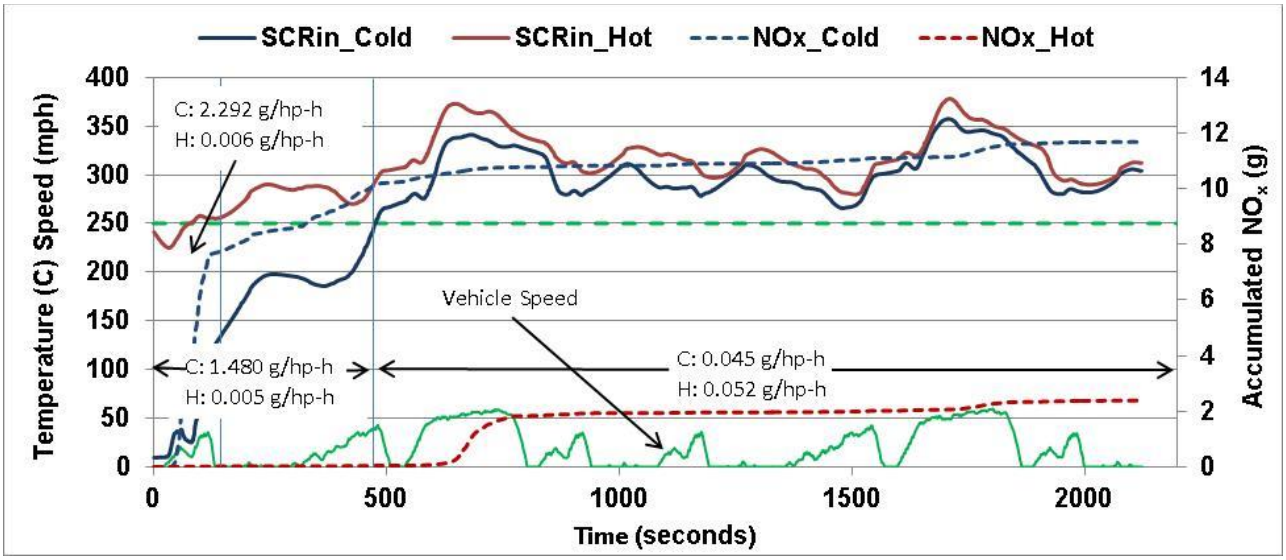


Figure ES-1: Accumulated NOx emissions during hot and cold start UDDS testing

- Figure ES-1 shows the cumulative NO_x emissions, instantaneous SCR inlet temperature and vehicle speed for a class 8 Freightliner equipped with a Cummins 11.9 liter 2011 engine. The figure is typical for SCR equipped diesel engines, where cold start NO_x emissions can be as high as 2.3 g/bhp-hr compared to an equivalent warm test of 0.006 g/bhp-h. Although cold start emissions do not contribute to the inventory, it is important to consider the extreme nature of cold start emissions if vehicles are allowed to cool frequently. The NO_x emissions

accumulated in 1 mile after a cold start were equivalent to emissions accumulated during 32 miles of running hot.

- The 2010 certified diesel engines with advanced cooled EGR and no SCR were tested. These vehicles operated utilizing a lug curve with peak torque starting as low as 1000 revolutions per minute (RPM), where the driver was instructed to operate the vehicle down to 900 RPM before shifting. The truck behavior was unusual, and both UCR and WVU trained drivers commented on the strange operation. Additionally, the certified emissions had a family emission limit (FEL) of 0.5 g/bhp-hr for 2010 MY, but the measured NO_x emissions were around 1 g/bhp-hr (0.25 g/mi) for the UDDS cycle, which represents a certification-like cycle. Even the port cycles showed brake specific emissions higher than 1 g/bhp-hr and as high as 2 g/bhp-hr for the near dock cycle.
- Pre-2010 certified diesel engines exhibited regulated emissions that were very close to the standard and were found to be repeatable for randomly selected models tested. This suggests that pre-2010 emissions inventories may be more reliable than SCR-equipped diesel engines due to SCR performance variability.
- Most NO_x emissions from SCR equipped diesel refuse vehicles were produced during the compaction portion of the in-use test cycle. The high NO_x emissions corresponded with a low SCR exhaust temperature, where the emissions increased from 0.27 g/bhp-hr NO_x for the transient and curbside cycles to 3.8 g/bhp-hr NO_x for the compaction cycle.
- The percentage of NO_x as NO_2 ranged from 10% to near 90%, with the highest levels of NO_2 emissions from non-SCR-equipped diesel vehicles. NO_2 was highest for the pre-2010 certified engines (averaging 1.15 ± 0.48 g/mi for the UDDS cycle). In general NO_2 ratios were similar for all tests at around 45%±8%, except for the SCR equipped diesel vehicles, which showed high variability with a NO_2 ratio of 47%±36%.

The emission results for ammonia, hydrocarbons, toxics, and fine particles are summarized below:

- Ammonia (NH_3) emissions from the vehicles tested ranged from about 0.01 to 0.1 g/mi. The diesel vehicles' NH_3 emissions averaged 0.04±0.03 g/mi (0.01±0.01 g/hp-h), where the port vehicle emissions were similar (0.03±0.02 g/mi), but the propane school bus had relatively higher NH_3 emissions (0.48±0.04 g/mi) over the CBD test cycle. All the diesel vehicles showed cycle averaged raw NH_3 emission concentrations less than 10ppm. Of the 54 diesel tests conducted, only 2 vehicles had NH_3 emissions over 5 parts per million (ppm). Half of the tests were below 2 ppm. Five of seven propane vehicle tests had NH_3 emissions greater than 5 ppm and two were over 50 ppm, suggesting that relatively higher NH_3 emissions exist for the propane vehicles compared to the diesel vehicles.
- The emission factors for total hydrocarbon (THC), methane (CH_4), non-methane hydrocarbon (NMHC) and toxics were very low for all diesel vehicles tested. This agrees with other research from the Advanced Collaborative Emissions Study (ACES) project that showed a 98% reduction from diesel engines with catalytic exhaust systems. THC, NMHC, and CH_4 emissions were at or below 0.09 g/mi, 0.06 g/mi, and 0.04 g/mi, respectively, for all vehicles (except the LPG vehicle) for both the UDDS and port regional cycles. Slightly higher THC, CH_4, and NMHC emissions were found for the lower power near dock port cycle (0.36 g/mi, 0.10 g/mi, and 0.29 g/mi, respectively). Toxic emissions were low and near the

detection limits of the method where 75% of the measured carcinogenetic species (benzene, toluene, ethylbenzene, and xylenes - BTEX) were below the average ambient background concentration pulse one standard deviation (< 10 mg/mi and typically < 2 mg/mi background corrected). Carbonyl emissions were also low relative to the measurement method, where more than 75% of the measured species were below the same threshold except for formaldehyde. Formaldehyde showed a relatively higher emission concentration, with 75% of the measurements above the threshold. Even though the formaldehyde samples were relatively high, their absolute contribution were below 72 mg/mi, with an average of 18±19 mg/mi. Acetaldehyde was the next largest carbonyl with maximum emissions of 18 mg/mi and an average of 1.5±4 mg/mi. The rest of the carbonyls were below 2 mg/mi. Cold start UDDS emissions were similar to the hot start UDDS emissions for THC, CH_4, NMHC, and toxics (note the UDDS was performed as a 2xUDDS cycle, which may have minimized the cold start effect for the HCs and toxics).

- The LPG goods movement vehicle showed higher THC, NMHC, CH_4, and toxic emissions than the diesel vehicles tested. THC, NMHC and CH_4 were 22.4 g/mi, 1.43 g/mi, and 21.4 g/mi respectively for the UDDS hot cycle. BTEX and formaldehyde samples were more than 10 times the average ambient background concentration plus one standard deviation. The propane vehicle averaged 6.5±9.3 mg/mi, 9.7±12 mg/mi, and 22.4±19 mg/mi for 1,3-butadiene, n-butane, and benzene respectively for the BTEX sample. The Carbonyls were high for formaldehyde and acetaldehyde (241±253 mg/mi and 42±48 mg/mi respectively) with the remaining aldehydes below 2 mg/mi. These results should be confirmed with additional testing on LPG port vehicles.

- Real-time PM measurements suggest the reported reference PM emission rate may be lower due to low filter weights for DPF equipped vehicles. The PM mass of the gravimetric method averaged 0.78±1.57 mg/bhp-hr for selected diesel vehicles. The average PM mass from the real-time measurement method averaged 0.05±0.09 mg/bhp-hr for the same vehicles. The average filter weight for these selected vehicles ranged from 10-20 µg, where UCR's CVS tunnel blank averages were 5µg with a 5µg single standard deviation. Thus, there is speculation that some of the uncertainty may be artifacts on the filter. As such, real-time PM measurements are useful for identifying low level PM mass in addition to real-time analysis.

- Elemental carbon (EC) and organic carbon (OC) PM was very low for all the vehicles tested and was typically below 0.2 mg/mi and 2.2 mg/mi respectively. More than half (69%) of the measured EC and OC emissions were below the average ambient background concentration plus one standard deviation. The propane vehicles had the highest organic PM contribution (>10 mg/mi for the near dock port cycle).

- Fine-particle emissions were typically higher during the first 200 seconds of the cold start UDDS cycle compared to the hot stabilized UDDS cycle ($5x10^5$ #/cc vs $1x10^3$ #/cc, respectively). The fine particle emissions appear to be higher for the regional port cycle compared to the near dock, local, and UDDS cycles ($8x10^4$ #/cc vs $1x10^3$ #/cc, respectively). The higher concentration of the regional port cycle may be a result of higher ATS temperatures and possible passive regenerations.

The results for greenhouse gas emissions and fuel economy are summarized below:

- The greenhouse gases (GHG) and fuel economy are characterized by CO_2 emissions for the diesel vehicle, but with the LPG truck, methane emissions represented approximately 8% of the GHG. The diesel fuel economy averaged 3.5 mi/gal (Port 1, 2 and UDDS) to 5.06 mi/gal (Port 3) for the port vehicles, 7.0 mi/gal for the school buses, and 4.2 mi/gal (UDDS) to 2.0 mi/gal (RTC) for the refuse haulers. The regional cycle (Port 3) showed 20% higher fuel economy than the more transient Port 1, 2, and UDDS cycles. The fuel economy from the refuse trash cycle (with integrated compaction phase) was about 50% lower than the transient UDDS cycle. The propane port vehicle showed 19% lower fuel economy than the diesel vehicles (3.3 mi/gal).
- The project measured N_2O greenhouse gases on selected tests. For those vehicles measured more than half (64%) of the N_2O emissions were below 0.4 ppm, which is the average ambient background concentration plus one standard deviation. The emission factors averaged 3.6±1.9 mg/mi with a maximum of 18 mg/mi (Cum_11.9 near dock port cycle).

The results for cross laboratory check are summarized below:

- The work comparison averaged around 3% negative bias (-3%), where UCR's laboratory was slightly lower than WVU's, with a spread of -9% to +4% on average. Both WVU and UCR show very low test-to-test variability, with a coefficient of variation (COV) less than 2% for all tests.
- The bsCO_2 was close and averaged around 5% positive bias, where UCR's laboratory was slightly higher than WVU's with a spread of 0% to 10% overall. Both WVU and UCR show very low test-to-test variability, with a COV less than 3% for all tests.
- The bsNO_x correlation was also good, but the comparison varied for the SCR equipped vehicles due to the low emission levels and the variable conditions of the SCR. For the non-SCR equipped vehicles, the deviation averaged about 3% positive bias, where UCR's laboratory was slightly higher than WVU's, with an average of -2% to 8%. The NO_x correlation was poor for the cold start SCR equipped vehicles and for two refuse haulers due to variability in the aftertreatment systems.

In summary, the data from this study suggests that 2010 compliant SCR-equipped HDD vehicles are exhibiting high in-use NO_x emissions that can be as high as 2 g/hp-h under low load conditions represented by short trips or frequent stops. The cause of the high NO_x emissions appears to be low load exhaust temperatures and, thus, low SCR aftertreatment temperatures. For SCR-equipped diesel engines, some accounting of vehicle duty cycle and SCR exhaust temperature is needed to properly characterize NO_x inventories. Additionally, there were differences in SCR performance that varied between manufacturers, suggesting future performance will continue to vary. The ratio of NO_2 in the NO_x has been demonstrated to be about 45% for all diesel vehicles tested, where there is more variability with the SCR equipped diesels. Both NO_x emission factors and NO_2 ratios suggest NO_x emissions are more variable for SCR equipped diesels compared to non-SCR equipped diesel vehicles. This also suggests activity studies are needed to assess the impact of SCR performance on NO_x inventories. Other results showed the diesel PM, CO, THC, and selected toxics were all very low, well below certification limits, and near the limits of the measurement method for all the tests performed. The low PM, CO, THC, and selected toxics for all the diesel vehicles tested suggest these emissions are well controlled. Looking ahead, the overall results suggest NO_x emissions are still a concern for selected activities, and SCR performance needs to be investigated during wide in-use, on-road operation to characterize its impact on local inventories.

1 Introduction

1.1 Background

Emissions from heavy-duty trucks and buses accounted for about one-third of NOx emissions and one-quarter of PM emissions from mobile sources when stringent emission standards were introduced by the EPA on December 21, 2000 and by CARB in October 2001. The new standards, shown below, further reduced PM by 90% and NOx by 95% from existing standards.

- PM—0.01 g/bhp-hr
- NOx—0.20 g/bhp-hr
- NMHC—0.14 g/bhp-hr

The PM emission standard took effect in the 2007. However, the NO_x and NMHC standards were phased in for diesel engines between 2007 and 2010 based on the percent-of-sales basis: 50% from 2007 to 2009 and 100% in 2010. The regulation contained other provisions for meeting the NOx requirement so very few engines actually met the stringent standard of 0.20 g/bhp-hr before 2010. In addition to transient Federal Test Procedure (FTP) testing, the emission certification requirements included: 1) the 13-mode steady-state engine dynamometer test Supplemental Emissions Test (SET) test, with limits equal to the FTP standards, and 2) the not-to-exceed (NTE) emission testing with limits of 1.5 × FTP standards for engines meeting a NOx FEL of 1.5 g/bhp-hr or less and 1.25 × FTP standards for engines with a NOx FEL higher than 1.5 g/bhp-hr.

The implementation of the more stringent standards for heavy-duty highway engines was a key strategic element of the plan for improving air quality in the South Coast Air Quality Management District (AQMD). While measurements in laboratories were showing NOx and PM emissions meeting the stringent certification standards, some values from in-use conditions were showing increased emissions of ammonia from LNG trucks and of NOx from diesel trucks. Since there was a question about whether the in-use engines were meeting the stringent emission standards the AQMD Board authorized issuance of RFP #P2011-06 to assess the in-use emissions.

The RFP's objectives were to carry out chassis dynamometer testing of heavy-duty natural gas and diesel vehicles using near-certification and in-use driving cycles while measuring: 1) regulated emissions; 2) unregulated emissions such as ammonia and formaldehyde; 3) greenhouse gas levels of CO_2 and N_2O and 4) ultrafine PM emissions. The study would test about twenty-five heavy-duty vehicles used for transit, refuse and goods movement applications with engines fueled with natural gas, propane, diesel, and a combination of diesel and natural gas fuels. The engine fleet was sub-divided by emission standards and technology.

1.2 Objectives

The University of California, Riverside (UCR) was contracted to test 16 heavy-duty vehicles, mainly diesel fueled engines, used for goods movement, refuse and for transient applications. The testing protocol involved measuring the emissions identified in the RFP while the vehicles operated following driving cycles that better represented the in-use conditions rather than just certification conditions. For example, the trucks used in goods movement were tested on three port driving cycles; refuse haulers tested on the AQMD refuse hauler cycle and buses were tested on the central business district cycle. The contract involved cross-laboratory testing of some common vehicles with West Virginia University as part of the quality assurance program.

1.3 Technology Used for Meeting Low-Emission Limits

Meeting the very strict emission standards was a challenge for engine manufacturers and required them to develop technology solutions that looked at the integrated system of engine and after treatment. Furthermore the solutions for a diesel engine were not the same for an engine fueled by natural gas.

For control of PM from diesel engines, the engine manufacturers relied on Diesel Particulate Filters (DPF). In general, DPF control system consists of four sections: 1) an inlet, 2) a Diesel Oxidation Catalyst (DOC), 3) a DPF and 4) an outlet. Exhaust flows out of the engine and through a DOC before entering the DPF where PM is collected on the walls of the DPF. The collected carbon is oxidized to remove it from the DPF during the regeneration process. When operating conditions maintain high exhaust temperatures, the DPF is self-regenerating. Otherwise, an active regeneration is required to remove a build-up of PM and pressure drop in the DPF by adding diesel fuel upstream of the DOC. The chemical reaction over the DOC raises the exhaust gas temperature high enough to oxidize the carbon from the filter.

The control of NOx from diesel engines from 2007 to 2009 was met with the use of cooled exhaust gas recirculation (EGR) and a redesign of engine operating conditions. For the 2010 engines, EGR was continued for all manufacturers and all but one manufacturer, Navistar, adopted the use of Selective Catalytic Reduction (SCR)[1]. In the SCR process NOx is converted into nitrogen by the reaction with ammonia over a special catalyst. When operating temperatures are >250°C, an aqueous solution of urea is injected into the exhaust upstream of the SCR catalyst. The heat converts the urea into ammonia and water, which is the reactant to convert NOx to nitrogen. At temperatures <250°C, urea is not injected so the full engine out NOx emissions are emitted. SCR technology has a long history of successful operation in stationary sources.

Another path for meeting the stringent 2010 emissions limits was to design engines based on either natural gas or liquefied propane gas (LPG). Gaseous fueled engines meet the strict PM limits without a DPF. However, gaseous-fueled engines require technology for control of NOx. When designed and operated at stoichiometric conditions, then the engine can use three-way catalyst (TWC) technology, like that on gasoline vehicles. However, many engines operate as lean burn so NOx is higher than the 2010 limit and must be controlled with EGR and SCR technologies as used in the diesel engines.

1.4 Vehicle/Engine

The overall project included twenty-five on-road heavy-duty vehicles (test vehicles) used in the goods movement, refuse, and transit applications. Some are powered by diesel fuel and others by gaseous fuels. Some vehicles were added later to the matrix. The complete vehicle matrix is shown in Table 1-1 with a summarized view by technology in Table 1-2. The “Test Lab” column in Table 1-1 and the shaded portion of the matrix of Table 1-2 identify the vehicles contracted to UCR. The total vehicles contracted to UCR were 16 vehicles, nine port vehicles, five refuse haulers, and two school busses.

[1] On October 23, 2012 Navistar and Cummins announced deal on SCR emissions technology.

Table 1-1 Complete SCAQMD emission testing program vehicle list

Group	Test Lab	Vehicle Vocation	Fleet Name	Fuel	Engine						Vehicle				Cert. Level g NOX
					Family	OEM	MY	Model	Disp. (L)	Max Power HP@RPM	MY	GVWR	ODO miles	Test Wt.	
I	WVU	Transit Bus	OCTA	CNG	8CEXH054.0LBD	Cummins	2008	ISLG280	8.9	280@2200	2008	42540	116232	35000	0.2
I	WVU	Refuse Truck	LA Sanitation Bureau	LNG	8CEXH054.6LBL	Cummins	2008	ISLG320	8.9	320@2100	2008	58000	21465.2	56000	0.2
I	WVU	Goods Movement	Ryder Truck Rental	CNG	BCEXH054.0LBH	Cummins	2011	ISLG320	8.9	320@2100	2011	52000	191.9	69500	0.2
I	WVU	Goods Movement	TTSI Drayage Company	LNG	BCEXH054.0LBH	Cummins	2008	ISLG320	8.9	320@2100	2008	52000	45563	69500	0.2
I	WVU	Goods Movement	TTSI Drayage Company	LNG	BCEXH054.0LBH	Cummins	2009	ISLG320	8.9	320@2100	2010	50000	63256	69500	0.2
II	WVU	Goods Movement	Border Valley	LNG & ULSD	8WFSH0912XAL	Westport Innovations	2008	ISXG 450	14.9	450@1800	2008	48000	196562	69000	0.8
II	WVU	Goods Movement	HayDay	LNG & ULSD	8WFSH0912XAL	Westport Innovations	2008	ISXG 450	14.9	450@1800	2009	48000	368080	69000	0.8
II	WVU	Goods Movement	HayDay	LNG & ULSD	8WFSH0912XAL	Westport Innovations	2008	ISXG 450	14.9	450@1800	2008	48000	379860	69000	0.8
III	WVU	Goods Movement	UPS	LNG & ULSD	BWFSH0912XAL	Westport Innovations	2011	GX 450	14.9	450@1800	2011	34700	12300	69000	0.2
IV	WVU UCR	Goods Movement	Ryder Truck Rental	ULSD	9NVXH0757AGA	Navistar Inc.	2009	MAXX FORCE13	12.4	430@1700	2010	52000	80412	69500	1.2
IV	UCR	Goods Movement	Container Freight Port	ULSD	8DDXH14.0ELC	DDC	2008	DDC/60	14	425@1800	2009	52000	129815	69500	1.07
IV	UCR	Goods Movement	Container Freight Port	ULSD	8DDXH14.0ELC	DDC	2008	DDC/60	14	425@1800	2009	52000	121766	69500	1.07

Group	Test Lab	Vehicle Vocation	Fleet Name	Fuel	Engine						Vehicle				Cert. Level g NOX
					Family	OEM	MY	Model	Disp. (L)	Max Power HP@RPM	MY	GVWR	ODO miles	Test Wt.	
IV	UCR	Refuse	District 11 CalTrans	ULSD	BNVXH04666AGC	Navistar Inc.	2008	GDT260	7.6	260@2200	2009	33000	9754	56000	0.82
V	UCR	School Bus	Moreno Valley SD	LPG	8GMXH08.1502	GM	2008	LPI	8.1	330@1800	2009	30280	55570	20000	*
V	UCR	School Bus	A-Z Bus Sales	ULSD	7CEXH0408BAC	Cummins	2007	IS	6.7	220@1800	2008	31000	3357	20000	2.0*
VI	UCR	Goods [1] Movement	Port/China Shipping	LPG	9BPTE08.1601	GM	2009	P	8.1	325@4000	2005	52000	103608	69500	0.2
VII	UCR	Goods Movement	Ryder	ULSD	ANVXH0757AGA	Navistar Inc.	2010	12WZJ/B	12.4	430@1700	2011	52000	80651	69500	0.46
VII*	UCR	Refuse	***Not Tested-***												
VII	WVU UCR	Goods Movement	Idealease of Los Angeles	ULSD	BNVXH07570GB	Navistar Inc.	2011	MAXX FORCE13	12.4	475@1700	2011	52350	67373	69500	0.5
VII	WVU UCR	Refuse	CalTrans	ULSD	BNVZH0466AGA	Navistar Inc.	2011	MAXX FORCE A260	7.6	260@2200	2012	33000	10014	56000	0.5
VIII	UCR	Refuse	Waste Connection	ULSD	BCEXH0540LAQ	Cummins	2011	ISL9 370	8.9	370@2100	2012	36000	2500	56000	0.2
VIII	WVU UCR	Refuse	EDCO	ULSD	BCEXH0505CAC	Cummins	2011	ISC 8.3 300	8.3	300@2000	2011	60000	14269.4	56000	0.2
VIII	UCR	Goods Movement	Pac Lease	ULSD	BCEXH0729XAC	Cummins	2011	ISX11.9-425	11.9	425@1800	2011	80000	4769	69500	0.12
VIII	UCR	Goods Movement	Coca Cola	ULSD	ACEXH0505CAC	Cummins	2010	ISC-300	8.3	300@2100	2011	52000	13918	65000	0.2
VIII	WVU UCR	Goods Movement	Worldwide Rentals	ULSD	BVPTH12.8S01	Mack	2011	MP8-445C	12.8	445@1500	2011	52000	36982	69500	0.2

[1] Note…LPG truck odometer was 103,608 but the engine was <20,000 miles

Table 1-2 Overall Vehicle/Engine Test Matrix for AQMD Project (UCR Matrix is Shaded)

Engine/Technology	Number of Vehicles			
	Transit	School Bus	Refuse	Goods Movement
I. 8.9L 0.2g natural gas engine with 3-way cat	1		1	3
II. 15L 0.8 HPDI engine with EGR & DPF				3
III. 15L 0.2g HPDI engine with EGR, DPF & SCR				2
IV. Diesel Engine at 1.2 g NOx (2007-09)			1	3
V. Propane & Diesel School bus (2007-09)		2		
VI. LPG Engine >0.2 NOx w/o SCR				1
VII. Diesel Engine > 0.2 g NOx w/o SCR			2	2
VII. Diesel Engine ≤0.2 g NOx w/SCR			2	3
VIII Natural gas engine with 3-way cat + AFD			1	1
Total	1	2	7	18

1.5 Test Cycles

Five driving cycles were chosen for this project and details are provided in Appendix A. While certification of an engine is carried out with an engine dynamometer these cycles were run with the engine installed in a chassis; hence a chassis dynamometer was used. Furthermore some of the selected driving cycles were to be more representative of in-use activity rather than certification. The matrix of selected driving schedules for each engine application is shown in Table 1-3. Table 1-4 summarizes the test matrix and unique vehicle ID for quick reference.

Table 1-3 Test cycles

Application	Test Drive Cycle				
	CBD	UDDS	OCTA	AQMD Refuse	Drayage Truck Port (DTP)
Transit	X	X	X		
Refuse truck		X		X	
Goods movement		X			X
School bus	X				

Table 1-4 Summarized test matrix, fuel, cycle, and unique ID name

Unique ID	Group	Vehicle Vocation	Fuel	Test Cycle	Engine									Cert. Level
					Family	OEM	MY	Model	Disp. (L)	Max Power HP@RPM	GVWR	ODO miles	Test Wt.	
N12.4a	IV	Goods Movement	ULSD	UDDS, DTP	9NVXH0757AGA	Navistar Inc.	2009	MAXX FORCE13	12.4	430@1700	52000	80412	69500	1.2
D14a	IV	Goods Movement	ULSD	UDDS, DTP	8DDXH14.0ELC	DDC	2008	DDC/60	14	425@1800	52000	129815	69500	1.07
D14b	IV	Goods Movement	ULSD	UDDS, DTP	8DDXH14.0ELC	DDC	2008	DDC/60	14	425@1800	52000	121766	69500	1.07
N7.6	IV	Refuse	ULSD	UDDS, REF	BNVXH04666AGC	Navistar Inc.	2008	GDT260	7.6	260@2200	33000	9754	56000	0.82
GM8.1a	V	School Bus	LPG	CBD	8GMXH08.1502	GM	2008	LPI	8.1	330@1800	30280	55570	20000	*
C6.7	V	School Bus	ULSD	CBD	7CEXH0408BAC	Cummins	2007	IS	6.7	220@1800	31000	3357	20000	2.0*
GM8.1b	VI	Goods [1] Movement	LPG	UDDS, DTP	9BPTE08.1601	GM	2009	P	8.1	325@4000	52000	103608	69500	0.2
N12.4b	VII	Goods Movement	ULSD	UDDS, DTP	ANVXH0757AGA	Navistar Inc.	2010	12WZJ/B	12.4	430@1700	52000	80651	69500	0.46
N12.4c	VII	Goods Movement	ULSD	UDDS, DTP	BNVXH07570GB	Navistar Inc.	2011	MAXX FORCE13	12.4	475@1700	52350	67373	69500	0.5
N7.6	VII	Refuse	ULSD	UDDS, REF	BNVZH0466AGA	Navistar Inc.	2011	MAXX FORCE A260	7.6	260@2200	33000	10014	56000	0.5
C8.9	VIII	Refuse	ULSD	UDDS, REF	BCEXH0540LAQ	Cummins	2011	ISL9 370	8.9	370@2100	36000	2500	56000	0.2
C8.3r	VIII	Refuse	ULSD	UDDS, REF	BCEXH0505CAC	Cummins	2011	ISC 8.3 300	8.3	300@2000	60000	14269.4	56000	0.2
C11.9	VIII	Goods Movement	ULSD	UDDS, DTP	BCEXH0729XAC	Cummins	2011	ISX15-485	11.9	425@1800	80000	4769	69500	0.12
C8.3p	VIII	Goods Movement	ULSD	UDDS, DTP	ACEXH0505CAC	Cummins	2010	ISC-300	8.3	300@2100	52000	13918	65000	0.2
V12.8	VIII	Goods Movement	ULSD	UDDS, DTP	BVPTH12.8S01	Mack	2011	MP8-445C	12.8	445@1500	52000	36982	69500	0.2

[1] Grey sections are shared vehicles between UCR and WVU

1.5.1 EPA Urban Dynamometer Driving Schedule (UDDS)

The EPA Urban Dynamometer Driving Schedule (UDDS) was a basis for the development of the Federal Test Procedure (FTP) transient engine dynamometer cycle for heavy-duty engines. While not the FTP, values from the UDDS on a chassis dynamometer are often compared with the values from a “certification test” run on an engine dynamometer. A comparison of the two test cycles is shown in Table 1-5. In this study the values from the UDDS were used to confirm that the selected engine was representative of the emission values for that technology.

The AQMD test program also included a cold-start UDDS as that is similar to the cold start FTP used in the certification testing. In a final certification procedure, the cold start values are weighted at 14% of the final number.

Table 1-5 Basic Parameters of the Cycle

	UDDS	FTP
Duration, seconds	**1060**	**1200**
Distance, km	**8.9**	**10.3**
Average speed, km/h	**30.4**	**30**
Dynamometer	**Chassis**	**Engine**

1.5.2 Port Drayage Cycles

Three port cycles were developed by TIAX for the Ports of Long Beach and Los Angeles based on the analysis of activity for over 1,000 Class 8 drayage trucks. Five characteristic operating parameters -- average speed, maximum speed, energy per mile, distance, and number of stops – were mapped to driver behavior. The driving behaviors are associated with specific activities such as queuing or on-dock movement, near-dock, local or regional movement, and highway movements. The final driving schedules, called the drayage port tuck (DPT) cycle, is represented by three distinct driving cycles, each composed of three phases. Some details are provided in Table 1-6.

Table 1-6 Drayage Truck Port Cycles

Drayage Truck Port cycles	Phase 1	Phase 2	Phase 3
Near-dock (2 to 6 miles)	Creep	Low Speed Transient	Short High Speed Transient
Local (6 to 20 miles)	Creep	Low Speed Transient	Long High Speed Transient
Regional (20+ miles)	Creep	Low Speed Transient	High Speed Cruise

1.5.3 AQMD refuse truck cycle (AQMD-RTC)

The refuse haulers will be tested using the AQMD refuse truck cycle (AQMD-RTC) that was developed by West Virginia University to simulate waste hauler operation in the AQMD District. The AQMD cycle is a modification of the William H. Martin Refuse Truck Cycle consisting of a transport segment (Phase 1), a curbside pickup segment (Phase 2), and a compaction segment (Phase 3).

1.5.4 Buses: Central Business District and Orange County Cycles

The Central Business District (CBD) Cycle is a chassis dynamometer testing procedure for heavy-duty vehicles. The graph of CBD cycle looks like a “sawtooth” driving pattern that is composed of idle, acceleration, cruise, and deceleration modes. The CBD is representative of the activity of in-use bus service.

The Orange County Bus Cycle (OCTA) is a chassis dynamometer test for heavy-duty vehicles developed by the West Virginia University (WVU) based on the driving patterns of urban transit buses in the Los Angeles, California area.

1.6 Fuel Selection

Commercial grade #2, ultra-low sulfur diesel fuel was used for the testing rather than fuel used for certification. Street fuel was more representative of what in-use vehicles would be using. Similarly, for the propane vehicles, locally supplied propane fuel was used in the testing as this was more representative of an in-use fuel.

For automotive propane usage Autogas is used for propane, Autogas is a mixture of propane with various contributions from other gasses. As such, locally supplied propane fuel meets the HD-5 specification for propane. As such it consist of at least 90% propane, no more than 5% propylene, and 5% other gases, primarily butane and butylene[2].

1.7 Emission Measurements

The contract specified the measurements of certain properties of the exhaust stream. These included:

- Regulated emissions: nitric oxides (NOx), particulate mass (PM), carbon monoxide (CO) and non-methane hydrocarbons (NMHC).
- Non-regulated emissions: ammonia (NH_3), benzene, toluene, butadiene, carbonyls (like formaldehyde).
- Greenhouse gases: nitrous oxide (N_2O), methane (CH_4), and carbon dioxide (CO_2)
- Ultrafine PM concentration and particle size distribution.

1.8 Engine Power Measurement

Engine break power was calculated using ECM broadcast J1939 standardized information. These signals are the same signals used for in-use compliance testing for the not-to-exceed standards in the 40 CFR Part 1065. A brief description is provided to describe the calculation and the results from the calculation. Equation 1 below shows the formula to calculate brake power:

$$bhp = \frac{RPM*(T_{actual}-T_{friction})*T_{reference}}{5252} \qquad \text{Eq 1}$$

Where:

bhp – is the brake power in units of (hp)

RPM – engine speed in revolutions per minute (rpm)

T_{actual} – ECM broadcast actual torque in (%)

[2] Alternative Fuels Data Center, US Department of Energy "Propane Fuel Basics, Office of Energy Efficiency and Renewable Energy, http://www.afdc.energy.gov/fuels/propane_basics.html

$T_{friction}$ – ECM broadcast friction torque in (%)

$T_{reference}$ – ECM broadcast reference torque in (ft-lb)

The engine speed, actual torque, and friction torque are real time second by second signals. The reference torque is a constant value and is fixed for each engine under test. Sometimes the reference torques is provided from the OEM and other times they can be downloaded from the ECM. Table 1-7 below lists all the reference torques used for this testing program. Two of the vehicles did not report a reference torque due to the ECM not supporting the latest J1939 data signals. The older format may be a result of the two vehicles being propane. As such, an estimated power was determined for these from the chassis dynamometer and previous tests for similar sized vehicles.

Table 1-7 Reference Torques Used for the Various Test Vehicles

Vocation	Mfg/Model/Yr	SN	Catalyst Type	RefTorque ftlb
Port	CUM/ISC300/2010	73058723	Active_DPF/SCR	1201.0
Port	Mack/MP8445C/2011	953695	Active_DPF/SCR	1888.0
Port	Navistar/12WZJ-B/2009	3006726C1	Active DPF	1689.0
School Bus	GM/8.1/2008	10XB11804020020	TWC	n/a [1]
Port	Navistar/A475/2011	1S125HM2Y4111072	DOC/DPF	1879.0
Port	Cummins/ISX11.9/2011	75002469	DOC/SCR/DPF	2050.0
School Bus	Cummins/ISB 220/2007	46789175	DPF+FBC	685.9
Port	Bi-Phase/8.1l GM/2009	81ELHHE	TWC	n/a [1]
Refuse	Navistar/GDT260/2008	1882496C1	Active DPF	906.5
Port	Navistar/A430/2011	1s125hm2y4115928	EGR	1633.7
Port	DDC/60 14L/2008	06R1019704	DOC/DPF	1615.3
Port	Cummins/ISL9 370/2011	73276566	DOC/SCR/DPF	1250.0
Refuse	Navistar/A260/2011	466HM2U3319545	DOC/DPF	775.2
Refuse	Cummins/ISC 8.3/2012	73268934	Active_DPF/SCR	1201.0

[1] this vehicle did not report a reference torque because it utilized an older ECM interface since it was powered by propane fuel.

2 Vehicle and Chassis Testing Procedures

This section describes the vehicle inspection that covers safety, maintenance, emissions, and fault codes. UCR worked with WVU and the AQMD project manager to generate a common vehicle acceptance program, as described in the following section.

2.1 Vehicle Selection

Selecting a vehicle meeting the emissions and control technology shown on Table 1-2 was the first step in starting a vehicle on the process of evaluating its emissions from near in-use conditions. The vehicle selection process included a number of steps, shown below. All the vehicles selected by UCR were discussed with the AQMD project manager before any testing was initiated.

2.1.1 Safety inspection

Vehicles were first inspected for safety as part of UCR's routine vehicle inspection and acceptance practices. This involved reviewing recent vehicle maintenance records for brakes, steering, fluids, and tires. These records were usually in good shape with fleets due to California Highway Patrol requirements. In addition to the records of the vehicle owner, UCR independently inspected brakes, tires, and other items as shown in Appendix D.

2.1.2 Maintenance and usage history

The maintenance and vehicle usage history were documented in this study. Appendix D shows the list of maintenance and usage history information that were recorded. This includes mileage, rebuilds information, oil maintenance records and other details. A list of some ECM downloads and other records can be found in Appendix C.

2.1.3 On-site emissions inspection

On site emissions inspections were performed to ensure that the vehicles emission systems were operating as designed. These inspections included a snap and idle test to look for visible smoke, wiping the tail pipe for diesel soot with a clean cloth, and visually inspecting the EGR, aftertreatment system components and other details that could affect the emissions.

2.1.4 Fault codes

The final inspection was to connect an electronic tool to the engine ECM and identify active fault codes. If fault codes were active then the AQMD project manager and the owner were contacted before testing the vehicle. ECM readings and fault codes were documented at the end of the testing program to capture both the as-received and as-left condition of the vehicles. There were a few vehicles that UCR did not have the appropriate vehicle interface tools to download the ECM information. For these vehicles the leasing agency or other parties were utilized for the vehicle ECM downloading. In addition to ECM downloads, all vehicle dashboards were monitored for visual vehicle/engine faults. A list of all the downloaded and observed information for each vehicle tested is summarized in Appendix D.

There were no fault codes for any of the vehicles tested. The LPG vehicle also did not generate a fault code, but UCR felt the vehicle ran with a higher than usual coolant temperature and the

engine size seemed small relative to the chassis application. Unfortunately there was only one LPG port vehicle identified and available at the time of the study.

2.2 Chassis Dynamometer

In 2010, UCR installed a state-of-art heavy-duty chassis dynamometer that is capable of performing all of the cycles listed in the RFP. The new dynamometer handles buses and trucks at on-road driving conditions. The dynamometer includes a 48" Electric AC Chassis Dynamometer with dual, direct connected, 300 horsepower motors attached to each roll set. The dynamometer applies appropriate loads to a vehicle to simulate factors such as the friction of the roadway and wind resistance, as would be experienced under typical in-use driving conditions. A driver accelerates and decelerates following a driving trace while on the dynamometer. As the on-road driving conditions are simulated, emissions measurements will be collected with UCR's Center for Environmental College of Engineering Research and Technology's (CE-CERT's) Mobile Emissions Laboratory (MEL) that is described in subsequent sections.

2.3 Chassis Test Procedures

Testing on a chassis dynamometer was ideal for evaluating the effect of the in-use driving cycle on tail pipe emissions. To improve measurement accuracy, a repeatable test procedure was developed to reduce variability in the data due to: 1) the engine, 2) aftertreatment system (ATS), 3) the Mobile Emissions Laboratory (MEL) and 4) dynamometer conditions. For example, it was important to define an amount of time between tests, the so-called soak time. This section describes UCR's approach to pre-test conditioning to minimize variability and to maximize the quality of the comparison test data.

2.3.1 Setting up the dynamometer

The first activity with the dynamometer was adjustments for the coast down coefficients and for the load. The road load coefficients were calculated based on parameters; for example, the frontal area of the vehicle and a factor accounting for is general shape. The road load and associated coast down coefficients were verified with chassis dynamometer coast downs prior to testing. The targeted vehicle weights for each application varied for each application, as listed in Table 2-1.

Table 2-1 Test vehicle application weight selections

Vehicle Weights (lb)			
Transit	School Bus	Refuse	Goods Movement
34,500 [1]	20,000 [2]	56,000 [3]	69,500

[1] Typical weight of an average transit bus with passengers of 150 lb

[2] A school bus with a capacity of 64 passengers at 100 lb. The weight accounted here is the sum of the vehicle weight with school kids.

[3] Typically loaded refuse hauler in the SC AQMD district

2.3.2 Vehicle and dynamometer preconditioning

After adjusting the loads on the dynamometer, it was ready for the warm-up cycle. For test days with cold starts of either the UDDS or CBD tests, the cold start test was used as the

conditioning and warm-up cycle for the dyno. For test days without planned cold starts; for example the port and refuse cycles, the vehicle was warmed up using a preconditioning drive cycle representative of the application. Thus the preconditioning cycle was the AQMD Refuse for the refuse truck cycle, the OCTA for the OCTA cycle, and a modified version of the DTP cycle for the DTP cycle. Due to the length of the DTP cycles only the final phase of the DTP cycles was used and this is about 30 minutes.

The preconditioning cycles warm up both the vehicle and dynamometer to the conditions of the test configuration, thus reducing emissions variability between tests. This approach is commonly used for certification testing, fuels evaluations, and other repeatable test evaluations.

2.3.3 After treatment system (ATS) preconditioning (regenerations)

There were several discussions with committee members about whether or not to control regenerations from the DPF equipped test vehicles. Regenerations are known to cause variability in measured emission levels due to systems that are statistically not under control; for example, back pressure on the DPF, after treatment catalyst temperature, and DPF cleaning that involved adding and combusting raw diesel fuel in the exhaust line. If uncontrolled, each of these parameters will significantly affect the measured emissions so in this work regenerations were performed on a regular basis prior to or in between test to improve the repeatability of the test data.

Given that the nature of this study is targeting in-use emissions, it was decided to allow regenerations to occur while testing following the in-use cycles. If DPF cleaning/regeneration occurred during the test, that test was repeated and the data were treated as a unique sample and not averaged with the other non-regeneration data. The regeneration data were analyzed and reported separately to characterize emissions with regeneration. Several regenerations occurred during the test project. In the case of one refuse hauler, regeneration occurred on every test cycle so those emission results include regeneration since that was the norm.

2.3.4 Soak time between tests

Soak time or the time between tests is known as an important factor that needs to be standardized to ensure test repeatability. Practically speaking, time is needed between each test to load new filter and sorption media, check instrument calibrations, and give the driver a break. For reference, EPA protocol certification tests use a 20 minute engine off soak period to return the engine to stabile operating condition prior to the next test. As the EPA's recommended 20 minute interval proved sufficient to prepare all media and checks before subsequent tests, UCR used 20 minutes as the standard soak period for all cycles during this project.

3 Emissions Laboratory Setup and Checks

This section describes the measures performed for the mobile emissions laboratory (MEL) to ensure accuracy (trueness and precision) of the emissions data.

3.1 Emissions Measurement Laboratory

As the on-road driving conditions are simulated, emissions measurements were collected with *UCR's* Mobile Emissions Laboratory (MEL). UCR's Mobile Emission Lab[3,4] (MEL) measures criteria pollutants, particulate matter (PM), and toxics with a CVS system, all meeting federal requirements. As discussed in the previous section, MEL will be located next to the UCR heavy-duty chassis dynamometer and measure emissions from vehicles on the dynamometer. The MEL was the second HDD lab in the United States to meet 40CFR Part 1065 specifications and has successfully carried out cross laboratory comparisons of both gaseous and PM emissions with Southwest Research Institute in 2007 and 2009. Earlier cross correlation measurements were carried out with NREL in Denver in 2005, as well as with the ARB lab in Los Angeles. Results from UCR's mobile lab are recognized by the engine manufacturers and regulatory groups, including the US EPA and CARB, and the data are often used to support regulation.

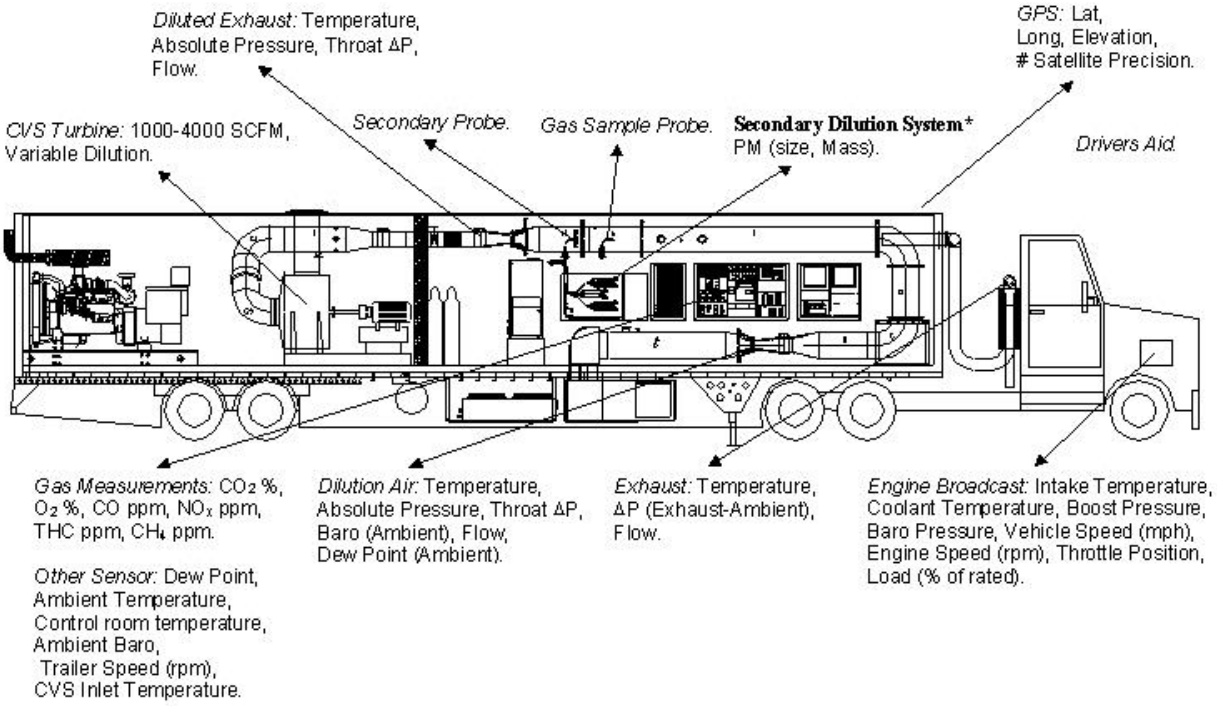


Figure 3-1 UCR's Mobile Emissions Lab (MEL)

---

[3] Cocker III, D. R., Shah, S., Johnson, K., Miller, J. W., Norbeck, J., *Development and Application of a Mobile Laboratory for Measuring Emissions from Diesel Engines. I Regulated Gaseous Emissions*, Environ. Sci. & Technology.,**2004**, 38,2182-2189

[4] Cocker, D.R.; Shah, S.D.; Johnson, K.J.; Zhu, X; Miller, J.W.; Norbeck, J.M., *Development and Application of a Mobile Laboratory for Measuring Emissions from Diesel Engines. 2. Sampling for Toxics and Particulate Matter,* Environ. Sci. & Technology, **2004**, 38, 6809-6816

The first research carried out in the new combined HDD chassis-MEL facility involved a comparison of emissions from federally mandated diesel fuel with those from the stricter California formulation. The project successfully tested 15 heavy-duty trucks over a 75-day period, and so can easily handle the 22 vehicles within the time period specified for this RFP.

Instruments within MEL continuously measure emissions of NOx, CO, CO_2, NMHC, and $PM_{2.5}$ with one-second resolution. The Dekati Mass Monitor (DMM) was used for real-time PM sampling. Ultrafines were monitored using UCR's unique fast-Scanning Mobility Particle Spectrometer (f-SMPS) analyzer[5], which is specifically designed for ultrafines. Integrated PM samples, such as PM mass and speciated PM, were collected on Teflo® and Quartz filters respectively.

The DMM measures PM mass concentrations through a combination of an electrical mobility diameter via particle charging and an aerodynamic diameter via inertial impaction over six stages of electrometers[6]. The combination of mobility diameter and number averaged aerodynamic particle diameter allows estimation of particle mass with the assumption of a log normal distribution. The aerodynamic diameters are estimated from six impactor electrometers that range from 0.030 um to 0.532 µm. The mobility diameter estimates the sub 30 nm particle diameters. If the distribution is bimodal, the DMM assumes an average density of 1 g/cm3. The DMM also has an inlet precut classifier set around 1.32 µm. The DMM was operated on the faster response option, as opposed to the lower detection option. The faster response setting is more typical for transient emission testing.

The f-SMPS is a key tool for measuring particle size distribution; however, use for transient cycles was limited to gathering data from steady state cycles as use was limited by the time resolution of the SMPS. For this study a unique instrument designed at UCR[7] was used to measure the near real time particle size distribution (PSD) with an emphasis on the ultrafine (<100nm) mass. The f-SMPS instrument utilizes a Radial Differential Mobility Analyzer (rDMA) and a Mixing Condensation Particle Counter (mCPC). The combination of these two components allowed the acquisition of particle size distributions range of 5–98 nm at rates of up to 0.4 Hz. For this research program the f-SMPS was setup for a 9 second scan time where typical SMPS's utilize a 90 second scan time.

Ammonia (NH_3) concentration was continuously measured using a Tunable Diode Laser (TDL) unit that is part of MEL. Data in Figure 3-2 show the very rapid release of NH_3 on acceleration and the quick response of the TDL used in UCR's earlier research. Other measurement

---

[5] S.D. Shah, D.R. Cocker*, ''*A Fast Scanning Mobility Particle Spectrometer for Monitoring Particle Size Distributions From Vehicles,*" Aerosol Science and Technology, 39, 519-526, 2005.

[6] Lehmann, U, V. Niemela, and Mohr, (2004) New Method for time-Resolved Diesel Engine Exhaust Particle Mass Measurement, Environ. Sci. Technol. 38 (21), 5704-5711, 2004

[7] Sandip D. Shah and David R. Cocker III, *A Fast Scanning Mobility Particle Spectrometer for Monitoring Transient Particle Size Distributions,* Aerosol Science and Technology, 39:519–526, (2005)

methods for ammonia, such as the dilute FTIR or a chemiluminescent analyzer, do not provide the needed response and accuracy during transient operation, as seen in other research[8].

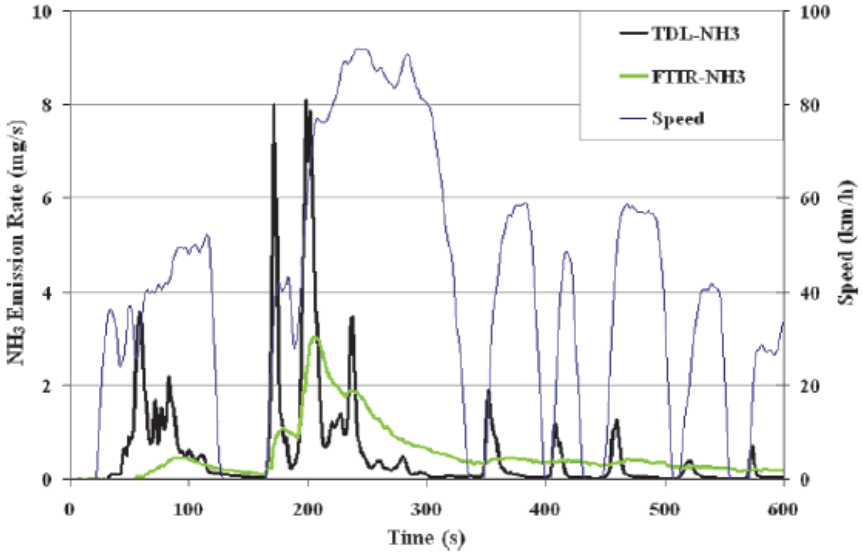


Figure 3-2 Comparison of FTIR & TDL Measurements of Ammonia Concentrations

3.2 Laboratory Setup for the Emissions Bench (MEL)

Prior to testing a number of steps were undertaken for the emissions lab (MEL) to ensure the testing was carried out according to CFR Part 1065 protocols. In addition to the real-time analyses in MEL, some samples, like PM Teflon filters, were moved to UCR labs for subsequent analysis. Those steps are described in this section

3.2.1 MEL dilution tunnel cleaning

Due to the low level of PM mass emissions expected from DPF equipped vehicles, the dilution tunnel in MEL was cleaned using a burn-out procedure developed at UCR. This procedure is used prior to testing any vehicle expected to have <5 mg/bhp-hr PM mass emissions and ensures that the measured PM is from the engine and not from materials desorbed from the tunnel walls. UCR's studies showed the procedure allowed PM measurements at the sub 1 mg/bhp-hr level and the main source of error is filter handling and not tunnel contamination.

In addition to reliance on experience of the burn-out procedure, UCR has carried out a routine check of the effectiveness by taking tunnel blanks. Summary data are available for MEL's tunnel blank and other blank reference checks as part of this research project.

3.2.2 MEL laboratory steps prior to test

This section summarizes the MEL's analyzers and support systems as per 40 CFR Part 1065 prior to, in between, and after testing for this program. The results from the pre-tests show successful linearity for all the MEL analyzers, chassis load cell, micro balance, humidity, and

[8] Huai, T., T.D. Durbin, J.W. Miller, J.T. Pisano, C.G. Sauer, S.H. Rhee, and J.M. Norbeck. **2003**. *Investigation of NH_3 Emissions from New Technology Vehicles as a Function of Vehicle Operating Condition*. Environ. Sci. & Technol., vol. 37, 4841-4847.

other integrated systems. Steps prior to test and startup are included in a checklist as part of the SOP for MEL.

3.3 Laboratory Setup for the Off-line Analyses

This section provides information about the various analyses that were carried out in the UCR labs after the chassis test. Each of these test methods follows a Standing Operating Procedure created according to the EPA protocol guidance document[9].

3.3.1 Filter weighting for PM mass

The mass concentrations of $PM_{2.5}$, metals and ions were acquired by analysis of particulates collected on 47mm diameter 2µm pore Teflo filters (Whatman brand). The filters were measured for net gains using a UMX2 ultra precision microbalance with buoyancy correction following the weighing procedure guidelines of the Code of Federal Regulations (CFR) (2). Before and after collection, the filters were conditioned for 24 hours in an environmentally controlled room (*Tdry* = 22 C and *Tdew* = 9.5C or 45%RH at *Tdry* 22C) and weighed daily until two consecutive weight measurements were within 3µg.

3.3.2 Measurement of Elemental and Organic Carbon (EC-OC)

OC/EC analysis was performed on samples collected on 2500 QAT-UP Tissuquartz Pall (Ann Arbor, MI) 47 mm filters that were preconditioned at 600°C for 5 h. A 1.5 cm_2 punch is cut out from the quartz filter and analyzed with a Sunset Laboratory (Forest Grove, OR) Thermal/Optical Carbon Aerosol Analyzer according to the NIOSH 5040 reference method.

3.3.3 Measuring Carbonyls

Carbonyls are collected on 2,4-dinitrophenylhydrazine (DNPH) coated silica cartridges (Waters Corp., Milford, MA) after a Teflon filter. A critical flow orifice controls the flow to 1.0 LPM through the cartridge and the sample time is adjusted to draw a known volume of exhaust sample through the DNPH cartridge so that the amount of formaldehyde on the cartridge is at the mass level recommended by Waters. Sampled cartridges are extracted using 5 mL of acetonitrile and injected into an Agilent 1100 series high performance liquid chromatograph (HPLC) equipped with a diode array detector. The column is a 5µm Deltabond AK resolution (200cm x 4.6mm ID) with upstream guard column. The HPLC sample injection and operating conditions are set up according to the specifications of the SAE 930142HP protocol. Samples from the dilution air are collected for background correction.

3.3.4 Measuring volatile toxic compounds

Traditional air monitoring methods for direct measurement of very-volatile and volatile organic compounds (VVOC/VOC) are insensitive at the low levels found in exhaust from lean burn engines. Accordingly, UCR uses selective adsorbents for concentrating the molecules of interest after diluted exhaust gas passes through a Teflon filter. After collection, adsorbents are returned to the laboratory where the adsorbed molecules are flashed into a

[9] United States Office of Environmental Protection Agency Information Bulleting EPA/600/B-07/*001 Guidance for Preparing Standard Operating Procedures (SOPs)* EPA QA/G-6, April 2007

concentrator/reservoir at low temperature, and then controllably vaporized into a gas chromatograph with a field ionization detector (GC/FID).

Molecules starting at approximately C_4 (butadiene) to C_6 (benzene) to C_{12} are effectively collected and concentrated on an adsorbent column composed with a multi-bed carbon bed including molecular sieve, activated charcoal, and carbotrap resin; each adsorbent has a specific selectivity towards certain boiling ranges or polarity. The adsorbent material first contacted in the column adsorbs the most volatile compounds, while the remaining compounds adsorb sequentially in relation to their volatility. The GC sample injection, columns, and operating conditions are set up according to the specifications of SAE 930142HP Method-2 for C_4-C_{12} hydrocarbons. Samples from the dilution air are collected for background correction

3.3.5 Measuring nitrous oxide (N_2O)

N_2O emissions were collected in a Tedlar bags and analyzed using an outside laboratory equipped with a MKS Fourier Transform Infrared (FTIR) system. The absorption cell for the FTIR has a volume of 5 liters, and the residence time in the cell is approximately 10 seconds. UCR's N_2O FTIR was not available at the time of testing, thus, off-site analyses were carried out by the California Air resources Board and West Virginia University. Since the analysis of N_2O was performed off-site UCR did not measure all vehicles in the test program due to logistics. Thus the analysis of N_20 only reflects those vehicles sampled.

4 Quality Control/Quality Assurance

This section covers some of the quality control/assurance planning that was taken to assure the accuracy of the data.

4.1 Cross-laboratory Correlation

Five diesel vehicles were tested at both UCR and WVU. Although six laboratories are required for a statistically significant comparison, the data obtained from this study still allow a comparison of values from two independent laboratories and create a measure of the confidence in the accuracy of the data since the two laboratories would presumably not have the same bias in the data sets. Three port vehicles and two refuse haulers were jointly tested and comparative data for engine work and brake specific emissions are presented. The vehicles tested represent three different emission level categories from less than 0.2 g/bhp-hr to 1.2 g/bhp-hr NO_x emission levels.

This cross-laboratory correlation task serves as a quality check for the emissions data that were collected independently by each laboratory. This correlation attempts to compare the emissions testing procedure of both laboratories that will include both the chassis dynamometer loading of the vehicle and the associated emissions measurement system. Although both WVU and UCR may adopt different procedures to conduct an emissions measurement campaign, the resulting data should be within an acceptable tolerance for real-world representativeness in each laboratory. Both UCR and WVU conducted the emissions measurement within immediate succession to prevent test vehicles going back into service. This procedure ensured the vehicle condition remained the same between WVU and UCR with no engine faults or maintenance conducted between the test intervals. Both laboratories tested the vehicle during day time conditions in Riverside Ca (as WVU was located only 5 miles away from UCR with their mobile laboratory setup)

Table 4-1 through Table 4-5 show the UCR and WVU engine work and selected emissions for five different shared vehicles including the cycle to cycle averaged coefficient of variation (COV). The emissions comparison is on a brake specific basis and includes $bsCO_2$, $bsNO_x$, bsPM, and $bsNH_3$ with units of g/bhp-hr for all species. Chassis dynamometer data is traditionally reported as distance-specific. However, for laboratory comparison purposes, changes in vehicle loading procedure and dynamometer setup can result in differences in distance-specific emissions. Therefore, brake-specific emissions were chosen as metric for comparison as components such as CO_2 are linear with work done by the engine.

Engine Work:

Five diesel vehicles were tested by both UCR and WVU for cross-laboratory comparison. Although six laboratories are required for a statistically significant comparison, the data obtained from this study still allow a comparison of values from two independent laboratories and create a measure of confidence in the accuracy of the data since the two laboratories would presumably not have the same bias in the data sets. Three port vehicles and two refuse haulers were jointly tested and comparative data for engine work and brake specific emissions are presented. The vehicles tested represent three different emission level categories from less than 0.2 g/bhp-hr to 1.2 g/bhp-hr NOx emission levels.

This cross-laboratory correlation task serves as a quality check for the emissions data that were collected independently by each laboratory. This correlation attempts to compare the emissions testing procedures of both laboratories, including the chassis dynamometer loading of the test vehicle and the associated emissions measurement system. Although both WVU and UCR may adopt different procedures to conduct an emissions measurement campaign, the resulting data should be within an acceptable tolerance for real-world representativeness in each laboratory. Both UCR and WVU conducted the emissions measurement within immediate succession before returning test vehicles back into their regular revenue service. This procedure ensured the vehicle condition remained the same between WVU and UCR with no engine faults or maintenance conducted between the test intervals. Both laboratories tested the vehicle during day time conditions in Riverside CA (as WVU was located only 5 miles away from UCR with their mobile laboratory setup).

Tables 1-5 show the UCR and WVU engine work and selected emissions for five different shared vehicles including the cycle to cycle averaged coefficient of variation (COV). The emissions comparison is on a brake specific basis and includes bsCO2, bsNOx, bsPM, and bsNH3 in g/bhp-hr. Although chassis dynamometer data is traditionally reported as distance-specific, for laboratory comparison purposes, changes in vehicle loading procedure and dynamometer setup can result in differences in distance-specific emissions. Therefore, brake-specific emissions were chosen as metric for comparison as components such as CO2 are linear with work done by the engine.

ENGINE WORK

Engine work was calculated from ECU reported actual engine percent torque, nominal friction torque, engine speed and reference torque. Although the design of the two chassis dynamometers are vastly different, with, WVU absorbing power directly at the wheel and hub and UCR absorbing power using rollers, the work comparison averaged around 3% negative bias (-3%) where UCR's laboratory was slightly lower than WVU's with a spread of -9% to +4% on average. Both WVU and UCR show very low test to test variability with coefficient of variation (COV) less than 2% for all tests.

There were a few test vehicles that showed small absolute work biases and others with relatively large biases. Typically the work differences were around ±5% (5 hp), but for two port regional cycles the power difference was as higher with one at 9 hp difference (#2 vehicle) and another at 14 hp difference (#3 vehicle). Both UCR and WVU investigated their power numbers with chassis dyno wheel torque and other power metrics and found all the measurements presented were valid. Interesting for both UCR and WVU most of the vehicles on the port cycles generated the same amount of work (107 bhp-hr) except for these two vehicles. On these two vehicles, UCR was high by 13 bhp-hr for the #2 vehicle and WVU was low for the by 25 bhp-hr for the #3 vehicle.

CARBON DIOXIDE

The $bsCO_2$ is the most suitable metric for cross-laboratory comparison, since CO_2 is an accurate indicator of both fueling and work. Fueling of the engine is highly liner with engine work, and therefore a similar work between the two laboratories should result in a similar $bsCO_2$. This

metric will provide the comparison of the emissions measurement system of the two laboratories. This comparison also normalizes chassis dynamometer setup differences to evaluate the ability to measure engine conditions. The bsCO_2 was very close and averaged around 5% positive bias where UCR's laboratory was slightly higher than WVU's with a spread of 0% to 10% overall. Both WVU and UCR show very low test to test variability with COV less than 3% for all tests.

OXIDES OF NITROGEN

For SCR equipped diesel engines the efficiency of control is highly dependent on temperature; in fact, conversion of NOx increases exponentially with temperature. As a consequence, small temperature differences during a test will lead to different NOx emissions from one laboratory to another. The importance of temperature is evident in the test data in that the COV results for CO_2 can be approximately 1% and can be as high as 10% for NOx. Given this backdrop the observed differences between the two laboratories in the NOx levels for the SCR-equipped vehicles are reasonable.

The cold start NOx variability between UCR and WVU is expected due to different catalyst conditions for the testing. Differences at low emission levels for the SCR-equipped vehicles are not a significant difference, but represent an expected variability for aftertreatment systems and NOx emissions.

The Navistar engine in Vehicle #2 was 0.7 g/bhp-hr different in brake specific NOx emissions for UCR and WVU during the regional port cycle. Since both showed very good agreement for bsCO2 (0% difference) the higher NOx may be a result of higher sustained loads for the UCR test compared to the WVU test. The Navistar engine utilized an advanced NOx system to approach a 0.5 g/bhp-hr certification level. If UCR had a slightly higher load then Vehicle #2 results could be related to the DPF regeneration and NO_2 used in that process.

Test vehicle #4 (advanced EGR refuse vehicle) shows a significant difference in NOx emissions measured over the SCAQMD-RTC cycle and not the UDDS cycle. The two laboratories showed a NOx emission factor ranging from 0.25 g/bhp-hr to 0.29 g/bhp-hr for the UDDS cycle, but 0.28 g/bhp-hr to 1.56 g/bhp-hr for the SCAQMD-RTC cycle. Figure 4-1 shows the accumulated NOx for two of UCR refuse cycles. The NOx emissions, from 0 to 2000 seconds, were around 0.29 g/bhp-hr (almost a perfect match with WVU). After 2000 seconds, the UCR measured NOx emission increases dramatically to 3.6 g/bhp-hr for the end of the curbside portion of the cycle and all of the compaction part of the cycle. WVU did not measure the same high NOx during the compaction part of the cycle.

To further understand the NOx emissions from these higher EGR engines during partial regeneration and non-regeneration operation, WVU had instrumented the vehicle with pre DPF and post DPF tailpipe NOx sensors. These sensors are installed by WVU for internal sanity check of the measured data. Figure 4-2 and Figure 4-3 show the pre and post DPF NOx concentrations during a test in which no DPF regeneration was detected and during a test in which a partial regeneration was detected. It can be observed that the DPF in these vehicles are contributing to a significant reduction in NOx concentrations during vehicle operation. This can be attributed to the continuous passive regeneration of the catalyzed DPF to utilize NO_2 to light-off soot

accumulation. On an average the DPF contributes to 68% reduction in engine-out NOx during normal vehicle operation. However, in some instances when passive soot light-off is insufficient, the engine strategy employs one or more different approaches to improve soot light off. The approaches included an in-cylinder increase in NOx concentration together with exhaust fuel injection. Figure 4-3 shows a partial active regeneration event during which a significant increase in NOx emissions is observed followed by a return to normal vehicle operation towards the end of the test.

UCR data for the refuse truck cycle could be characterized by such an event, which is beyond the control of the test laboratory and hence could have resulted in a significant difference in brake-specific NOx emissions.

Test vehicle #5 (SCR equipped refuse vehicle) showed a difference in NOx between 0.18 g/bhp-hr and 0.25 g/bhp-hr on the UDDS cycle. This difference is small considering the test to test variably was high. The high variability is again related to stability of the SCR.

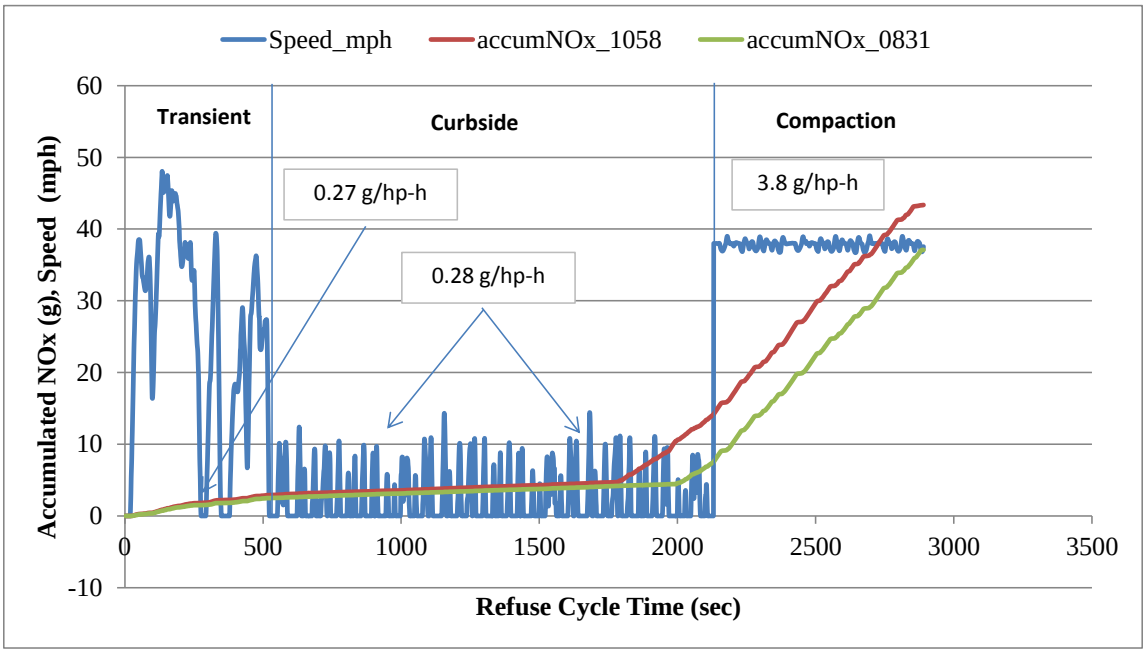


Figure 4-1 Refuse hauler shared vehicle #4 (Navistar A260 2011)

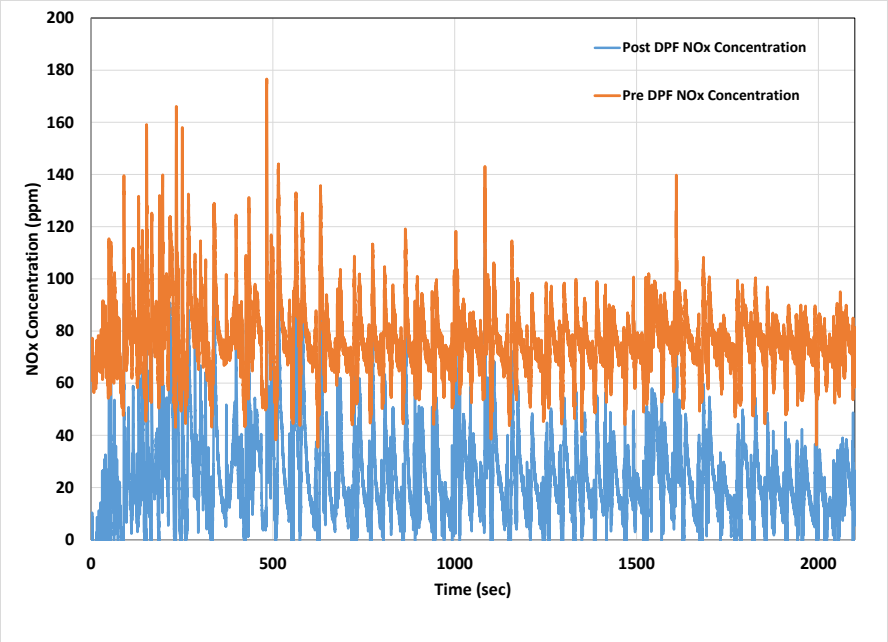


Figure 4-2 Pre and Post DPF NO_x concentration for a non-regeneration vehicle operation

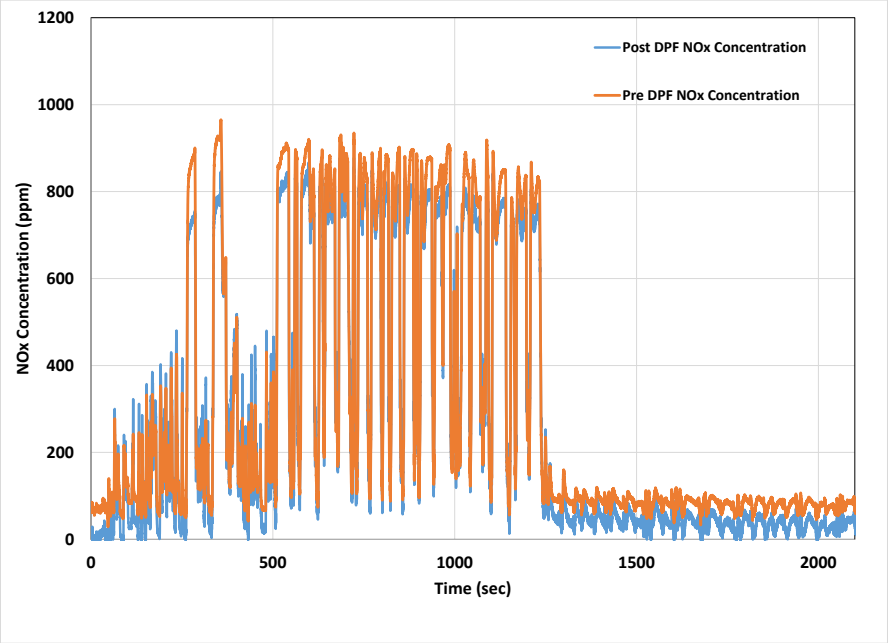


Figure 4-3 Pre and Post DPF NO_x during partial active regeneration during refuse truck cycle

Particulate Matter:

The bsPM emission levels were low for both UCR and WVU and were below the PM certification value for all tests and typically around 10% of the standard (< 1 mg/bhp-h) as expected for a properly functioning DPF. The PM emissions were thus similar between both laboratories and no significant outliers were identified.

Ammonia:

The bsNH_3 emissions were very low where there was no statistical difference between the different vehicles. As suggested for UCR, see Section 8.6, most of the NH_3 measurements were at or just above the lower detection limits of UCR's NH_3 measurement method. WVU also suggested several of the vehicles showed no quantifiably NH_3 emissions. The NH_3 emissions were thus similar between both laboratories and no significant outliers were identified.

Report Summary:

4.1.1 Port vehicle #1 (Mack MP8445C 2011)

Table 4-1 Port vehicle #1 comparative bsCO2, Engine Work & Emissions (g/bhp-h)

		Engine Work	Average Emissions g/bhp-h				COV Emissions [1,2] Work	COV Emissions [1,2] g/bhp-h			
Results	Cycle	bhp-h	CO_2	NOx	PM	NH_3	hp-hr	CO_2	NOx	PM	NH_3
UCR	CS_UDDS	29.0	555	0.40	0.0007	0.002					
UCR	UDDS	25.8	525	0.27	0.0003	0.003	1.2%	0.9%	14.9%	0.9%	1.1%
UCR	Near Dock	26.5	561	1.80	0.0004	0.001	1.3%	1.1%	2.7%	2.0%	1.5%
UCR	Local	40.1	556	1.10	0.0004	0.001	0.4%	1.8%	1.4%	0.9%	2.6%
UCR	Regional	107.2	513	0.36	0.0011	0.005	0.9%	0.7%	27.6%	4.3%	1.0%
WVU	CS_UDDS	29.0	506	0.51	0.0010	0.036					
WVU	UDDS	26.5	493	0.40	0.0020	<0.003	1.4%	0.6%	8.9%	1.8%	-
WVU	Near Dock	28.3	544	1.79	0.0011	<0.003	0.3%	0.8%	5.6%	0.6%	-
WVU	Local	40.8	532	1.26	0.0021	<0.003	0.6%	0.7%	4.5%	0.9%	-
WVU	Regional	98.4	520	0.36	0.0006	<0.003	0.4%	0.4%	7.4%	0.4%	-

[1] The COV is the coefficient of variation defined as one standard deviation divided by the averaged measured value. For PM and NH_3 the measurements were small and thus the COV was calculated as Stdev/10mg/bhp-hr for PM was used and Stdev/60mg/bhp-hr for NH_3.PM = 10 mg/bhp-hr was used based on the 10 mg/bhp-hr certification standard and 60 mg/bhp-hr is used based on an average of 10 ppm flow weighted limit for the raw exhaust.

[2] Blank values represent only one value or no data available. For example there were only single cold start tests and thus no COV was calculated. The dashes for NH_3 indicate no COV was practical.

4.1.2 Port vehicle #2 (Navistar MAXX-FORCE13 2009)

Table 4-2 Port vehicle #2 comparative bsCO2, Engine Work & Emissions (g/bhp-h)

		Engine Work	Average Emissions g/bhp-h				COV Emissions [1,2] Work	COV Emissions [1,2] g/bhp-h			
Results	Cycle	bhp-h	CO_2	NOx	PM	NH_3	hp-hr	CO_2	NOx	PM	NH_3
UCR	CS_UDDS	29.5	584	1.69	0.0005	0.005					
UCR	UDDS	29.4	557	1.56	0.0002	0.003	2.8%	1.1%	0.4%	0.3%	4.6%
UCR	Near Dock	23.5	760	2.16	0.0002	0.004	1.8%	1.4%	3.4%	1.3%	4.0%
UCR	Local	41.0	657	2.00	0.0004	0.005	1.0%	2.9%	2.3%	3.5%	10.3%
UCR	Regional	120.8	531	2.23	0.0001	0.006	0.6%	0.8%	2.0%	1.1%	3.1%
WVU	CS_UDDS	31.8	591	1.58	-	<0.003					
WVU	UDDS	28.8	591	1.42	0.0124	<0.003	1.3%	2.4%	5.4%	6.7%	-
WVU	Near Dock	27.9	617	1.84	0.0016	<0.003	0.3%	2.3%	1.6%	0.3%	-
WVU	Local	43.7	589	1.84	0.0008	<0.003	1.2%	0.9%	1.4%	0.1%	-
WVU	Regional	106.7	528	1.50	0.0008	<0.003	2.0%	1.9%	1.7%	0.1%	

[1] The COV is the coefficient of variation defined as one standard deviation divided by the averaged measured value. For PM and NH_3 the measurements were small and thus the COV was calculated as Stdev/10mg/bhp-hr for PM was used and Stdev/60mg/bhp-hr for NH_3.PM = 10 mg/bhp-hr was used based on the 10 mg/bhp-hr certification standard and 60 mg/bhp-hr is used based on an average of 10 ppm flow weighted limit for the raw exhaust.

[2] Blank values represent only one value or no data available. For example there were only single cold start tests and thus no COV was calculated. The dashes for NH_3 indicate no COV was practical.

4.1.3 Port vehicle #3 (Navistar MAXX-FORCE12 2011)

Table 4-3 Port vehicle #3 comparative bsCO2, Engine Work & Emissions (g/bhp-h)

Results	Cycle	Engine Work bhp-h	Average Emissions g/bhp-h CO_2	NOx	PM	NH_3	COV Emissions [1,2] Work hp-hr	g/bhp-h CO_2	NOx	PM	NH_3
UCR	CS_UDDS	25.6	564	1.49	0.0002	0.009					
UCR	UDDS	26.4	516	1.15	0.0001	0.004	1.4%	0.9%	5.8%	0.7%	2.5%
UCR	Near Dock	19.1	749	1.85	0.0004	0.012	1.2%	1.8%	2.2%	0.2%	3.6%
UCR	Local	33.2	636	1.59	0.0000	0.006	0.5%	1.8%	7.0%	0.3%	4.6%
UCR	Regional	107.1	506	1.04	0.0002	0.009	0.9%	0.3%	3.7%	0.5%	1.3%
WVU	CS_UDDS	23.5	565	1.83	0.0012	<0.003					
WVU	UDDS	23.6	487	1.27	0.0009	<0.003	2.1%	1.8%	2.0%	0.2%	-
WVU	Near Dock	-	-	-	-	-	-	-	-		-
WVU	Local	34.6	500	1.38	0.0020	<0.003	2.0%	0.5%	0.9%	0.2%	-
WVU	Regional	82.3	498	1.28	0.0019	<0.003	0.6%	0.8%	2.6%	0.5%	-

[1] The COV is the coefficient of variation defined as one standard deviation divided by the averaged measured value. For PM and NH_3 the measurements were small and thus the COV was calculated as Stdev/10mg/bhp-hr for PM was used and Stdev/60mg/bhp-hr for NH_3.PM = 10 mg/bhp-hr was used based on the 10 mg/bhp-hr certification standard and 60 mg/bhp-hr is used based on an average of 10 ppm flow weighted limit for the raw exhaust.
[2] Blank values represent only one value or no data available. For example there were only single cold start tests and thus no COV was calculated. The dashes for NH_3 indicate no COV was practical.

4.1.4 Refuse vehicle #4 (Navistar A260 2011)

Table 4-4 Refuse vehicle #4 comparative bsCO2, Engine Work & Emissions (g/bhp-h)

Results	Cycle	Engine Work bhp-h	Average Emissions g/bhp-h CO_2	NOx	PM	NH_3	COV Emissions [1,2] Work hp-hr	g/bhp-h CO_2	NOx	PM	NH_3
UCR	CS_UDDS	17.5	608	0.36	0.0008	0.004					
UCR	UDDS	17.4	612	0.25	0.0004	0.007	2.7%	1.0%	1.7%	1.5%	4.0%
UCR	RTC	26.9	816	1.56	0.0003	0.004	1.8%	1.3%	6.9%	2.4%	6.1%
WVU	CS_UDDS	18.6	663	2.09	-	<0.003					
WVU	UDDS	18.5	569	0.29	0.0026	<0.003	0.9%	0.0%	2.7%	0.7%	-
WVU	RTC	37.4	556	0.28	0.0020	<0.003	0.9%	1.3%	0.5%	0.1%	-

[1] The COV is the coefficient of variation defined as one standard deviation divided by the averaged measured value. For PM and NH_3 the measurements were small and thus the COV was calculated as Stdev/10mg/bhp-hr for PM was used and Stdev/60mg/bhp-hr for NH_3.PM = 10 mg/bhp-hr was used based on the 10 mg/bhp-hr certification standard and 60 mg/bhp-hr is used based on an average of 10 ppm flow weighted limit for the raw exhaust.
[2] Blank values represent only one value or no data available. For example there were only single cold start tests and thus no COV was calculated. The dashes for NH_3 indicate no COV was practical.

4.1.5 Refuse vehicle #5 (Cummins ISC 8.3 2012)

Table 4-5 Refuse vehicle #5 comparative bsCO2, Engine Work & Emissions (g/bhp-h)

Results	Cycle	Engine Work bhp-h	Average Emissions g/bhp-h CO_2	NOx	PM	NH_3	COV Emissions [1,2] Work hp-hr	CO_2	NOx	PM	NH_3
UCR	CS-UDDs	29.1	584	0.36	0.0035	0.023					
UCR	UDDS	26.6	607	0.18	0.0006	0.010	2.1%	1.5%	4.3%	1.0%	4.7%
UCR	RTC	43.6	612	0.32	0.0003	0.012	0.6%	0.4%	16.6%	0.6%	2.9%
WVU	CS_UDDS	-	-	-	-	-					
WVU	UDDS	26.7	672	0.25	0.0020	<0.003	1.3%	1.4%	9.4%	1.9%	-
WVU	RTC	50.4	654	0.11	0.0013	<0.003	1.8%	1.0%	39.0%	4.9%	-

[1] The COV is the coefficient of variation defined as one standard deviation divided by the averaged measured value. For PM and NH_3 the measurements were small and thus the COV was calculated as Stdev/10mg/bhp-hr for PM was used and Stdev/60mg/bhp-hr for NH_3.PM = 10 mg/bhp-hr was used based on the 10 mg/bhp-hr certification standard and 60 mg/bhp-hr is used based on an average of 10 ppm flow weighted limit for the raw exhaust.

[2] Blank values represent only one value or no data available. For example there were only single cold start tests and thus no COV was calculated. The dashes for NH_3 indicate no COV was practical. WVU did not have a cold start test on this vehicle due to vehicle availability.

4.2 Post-test QC Procedures

4.2.1 MEL carbon balance

Mass balances are a standard engineering check. For selected vehicles, UCR compared the carbon balance between the fuel flow rate reported by the ECM and the carbon measured in MEL's analytical instruments. An example of this comparison is shown in Figure 4-4.

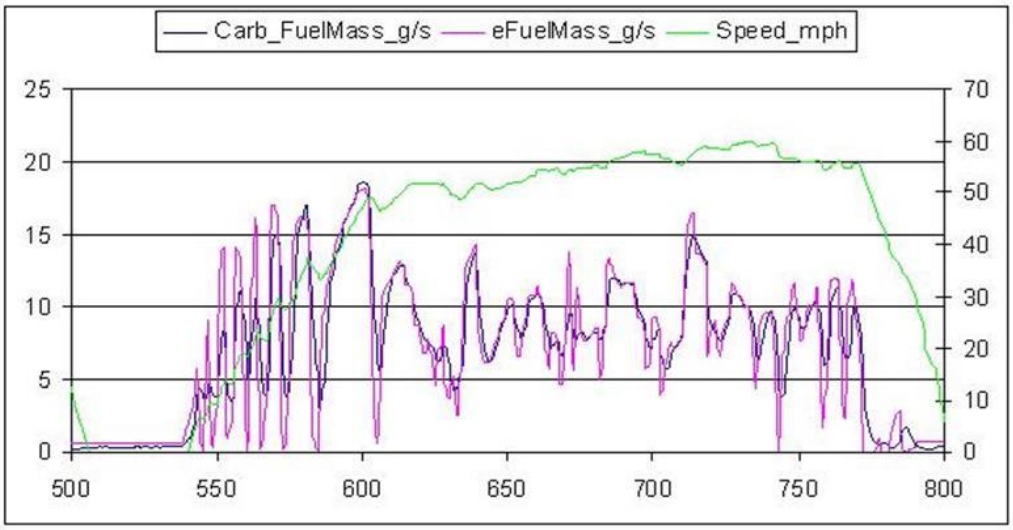


Figure 4-4: Real time second by second ECM and carbon balance fuel rate (port cycle)

While the visual agreement between ECM and measured fuel flow is good, a more quantitative measure of the closeness of fit is a parity chart of all data for a typical regional port cycle, as shown in Figure 4-5. The coefficient of determination, denoted R^2, is 91% and while generally considered quite acceptable, there appeared to be a greater deviation at the low values for

carbon flow. It appeared as though the ECM flow was less than that measured. Further investigation was warranted.

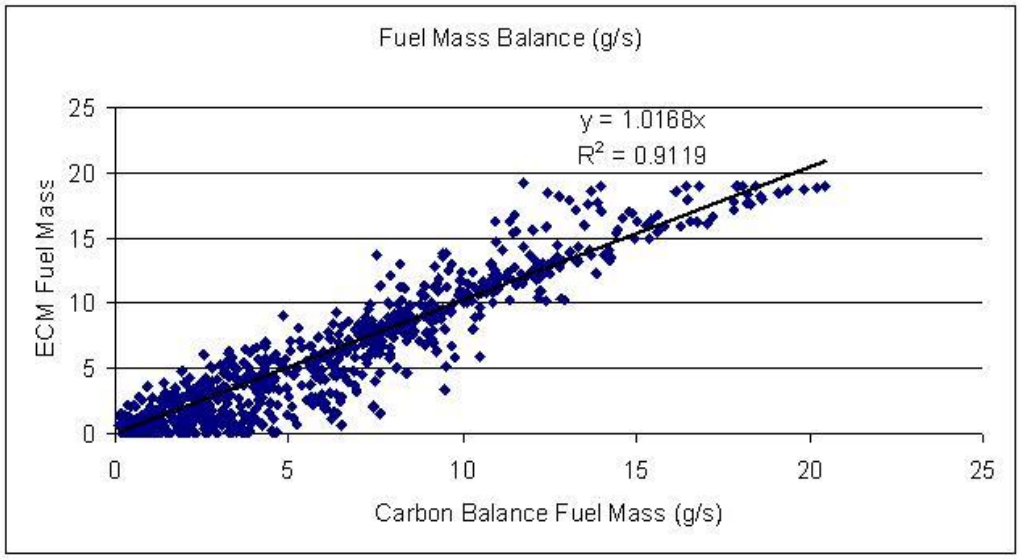


Figure 4-5: Parity Plot of Data from ECM and Exhaust Measurements (port cycle)

The further investigation compared the mass balances of selected engines for various engine manufacturers and test cycles. For example, Figure 4-6 shows plots of the UDDS, near dock, local port, and regional port cycles for a Navistar, Cummins and Volvo engine. For high loads and fuel flow rates, the coefficient of determination was 99% for all three manufacturers and the relative error ranged from +6% to -2%, excellent agreement. The same data were re-plotted in Figure 4-7 with the parameter being the test cycle. Phase 1 of the port cycle has the lowest load/fuel rates and this portion of the port cycle showed the highest uncertainty, ranging from -20% to + 60%, or 10-times that found at high flow/power rates. On the other hand, data for the UDDS cycle with relatively high power and fuel flow rates showed an excellent mass balance comparing MEL and the ECM. Data from this research confirms the findings of previous research that showed fuel rate is inaccurate below 30% load

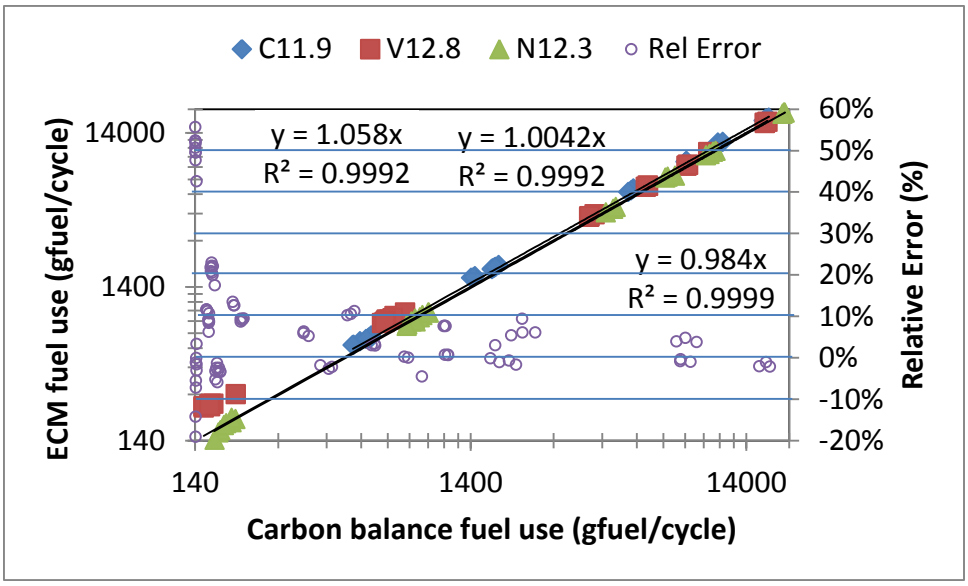


Figure 4-6: Carbon balance for all three port cycles and the UDDS cycle

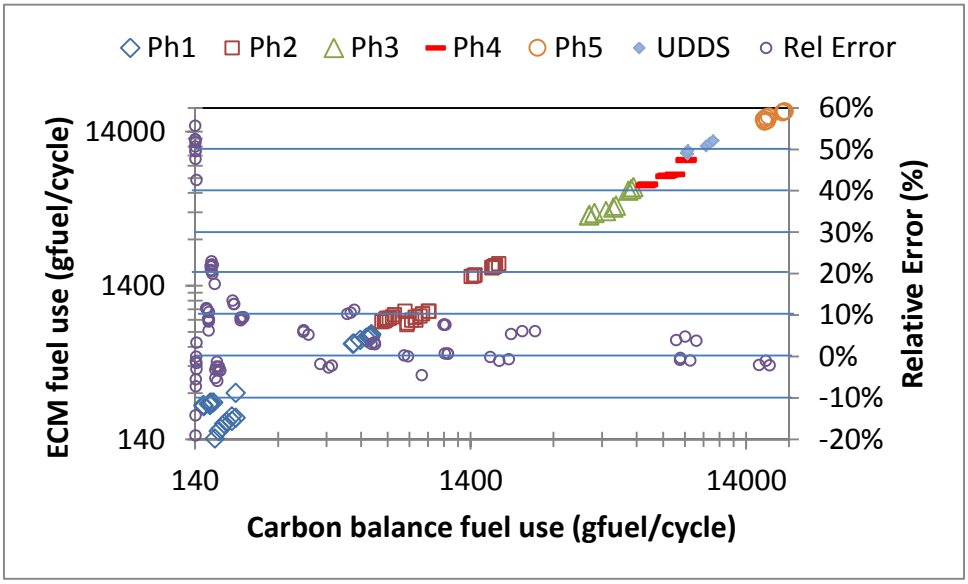


Figure 4-7 Carbon Balance Correlation on a test cycle basis

4.3 MEL quality control NTE

NTE data was calculated for selected vehicles to show representativeness of the cycles relative to in-use compliance NTE calculation methods. The NTE data presented does not include the measurement allowance since a reference laboratory was used and not PEMS. The following results provide perspective on the emissions generated by the vehicles and the type of cycle selected for this in-use study. The true emissions impact for this project should be drawn from the emissions results section.

Figure 4-8, Figure 4-9, and Figure 4-10 show the bsNOx NTE emissions for a 2009 Navistar, 2011 Navistar, and a 2010 SCR equipped Cummins engine. All three NTE method calculations are provided in the figures and show that all NTE data pass the in-use requirements for both the Navistar 2011 and Cummins 2010 vehicles. Only the 2009 Navistar vehicle showed bsNOx emissions that exceeded the in-use NTE standard during the regional port cycle.

For the Cummins SCR equipped engine, only one point exceeded the in-use NTE standard and it was excluded due to temperature exclusions as per 1065. No NTE's were generated for the near dock and local port cycles except for two with the 8.3 liter Cummins engine. Only the UDDS and regional port cycles generated NTE values as defined by 1065.

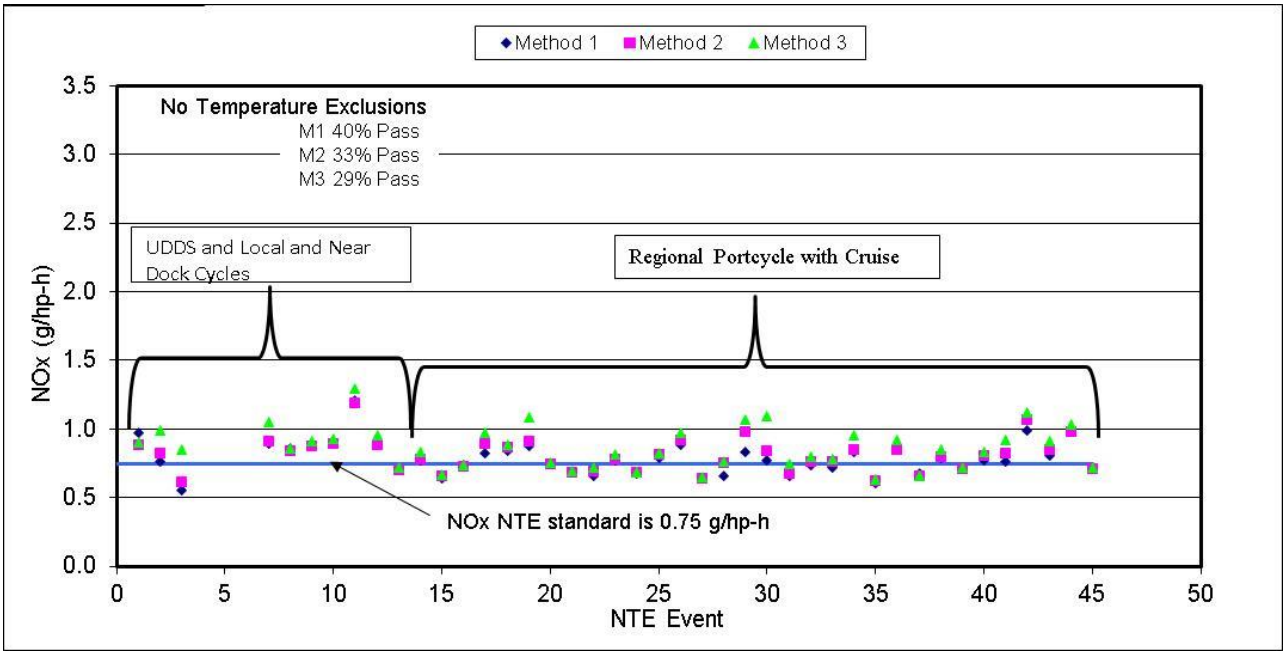


Figure 4-8: NOx NTE standard 1.8 g/bhp-hr Navistar 2009 MaxForce M13[1,2]

[1] Brake specific emissions are based on ECM reference torque.
[2] Lug curve based on **estimated** manufacturer's **2010** lug curve

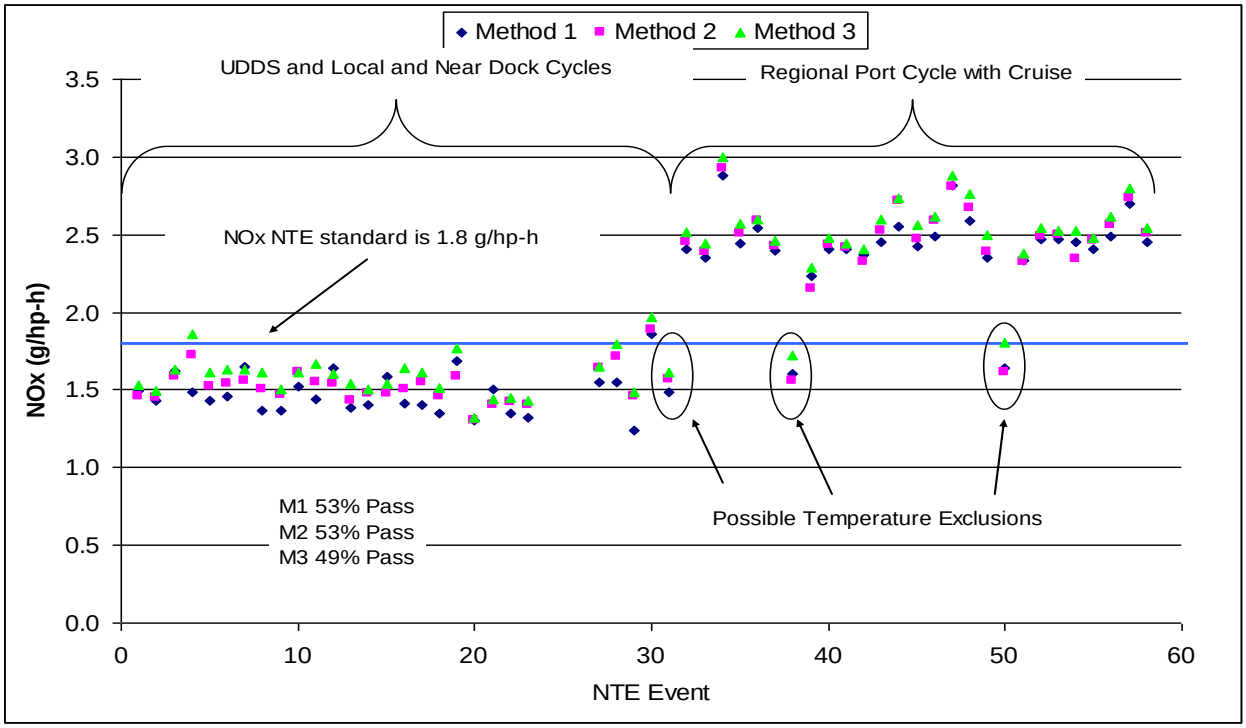


Figure 4-9: NOx NTE standard 0.75 g/bhp-hr Navistar 2011 MaxForce M13[1,2]

[1] Brake specific emissions are based on ECM reference torque.
[2] Lug curve based on **estimated** manufacturer's **2010** lug curve

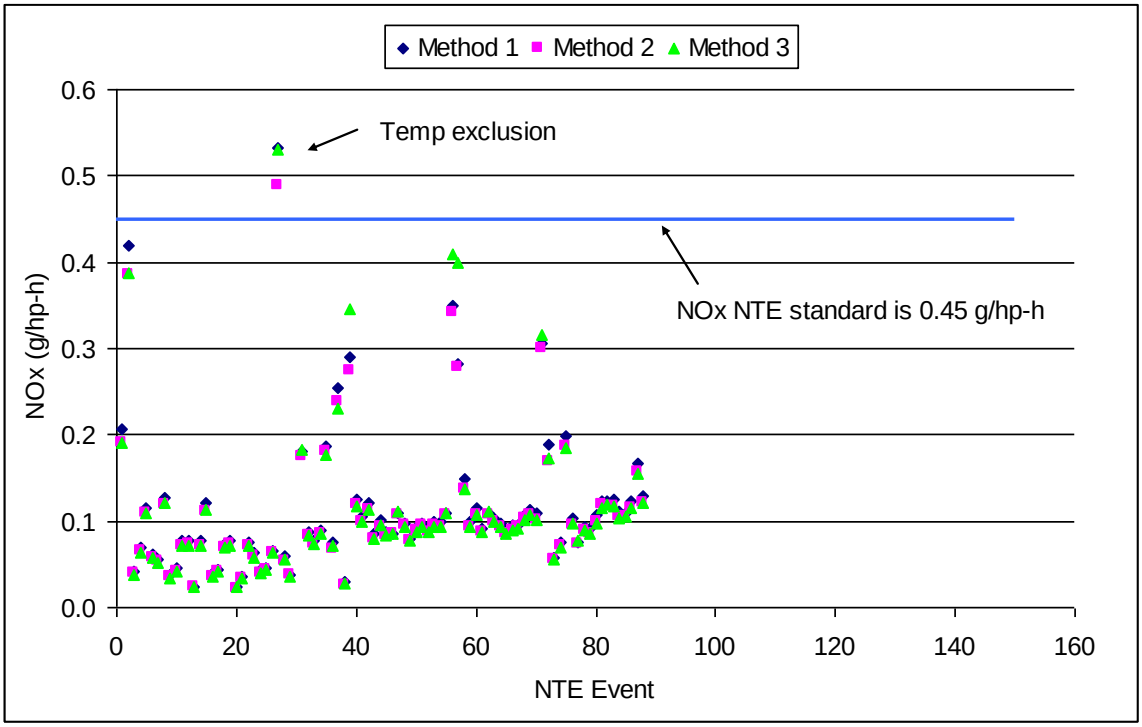


Figure 4-10: NOx NTE Standard 0.45 g/bhp-hr for the 2010 Cummins ISC 8.3 vehicle [1,2]

[1] Brake specific emissions are based on ECM reference torque.
[2] Lug curve based on **estimated** manufacturer's **2010** lug curve

4.4 MEL quality control checks

During the data analysis phase of this project the repeated tests were evaluated for consistency. This includes analyzing the variability between replicates by plotting the single standard deviation. All tests identified as greater than 2-3 times the standard deviation were viewed as outliers and investigated. Below are the following test points that were investigated for this project.

Table 4-6: Tests investigated for repeatability and consistency

ID	Cycle	Species	Issue	Action
Cummins/ISX11.9/2011	PDT_1	PM	stdev	valid
Navistar/12WZJ-B/2009	PDT_2	PM	stdev	PM spike for #1 tests see Appendix
Navistar/A430/2011	PDT_1	CO	stdev	valid
Bi-Phase/8.1l GM/2009	PDT_1	PM	stdev	valid
Navistar/12WZJ-B/2009	PDT_1	CO2	stdev	drivability issue from vehicle
Bi-Phase/8.1l GM/2009	PDT_2	PM	high value	fixed PM typo
Cummins/M2/2010	PDT_3	PM	stdev	valid
Mack/MP8445C/2011	PDT_3	PM	stdev	valid
Bi-Phase/8.1l GM/2009	PDT_3	NOx and PM	stdev	valid
Navistar/A430/2011	UDDS	CO	stdev	valid
Bi-Phase/8.1l GM/2009	UDDS	NOx and PM	stdev	valid
Cummins/ISB 220/2007	CS-CB	PM	stdev	2 tests at 4 and 2 at 12 g/mi
Navistar/A260/2011	UDDS	NOx	stdev	valid

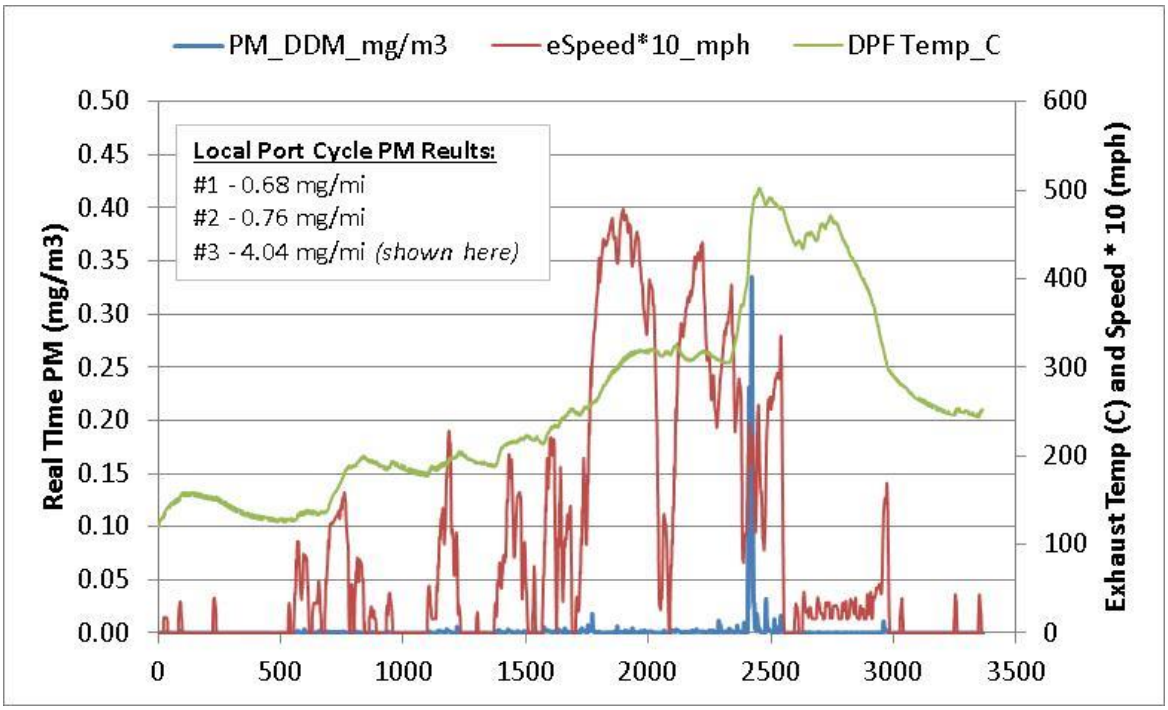


Figure 4-11: Navistar (12WZJ-B) real-time PM, vehicle speed, and DPF temp for local port cycle

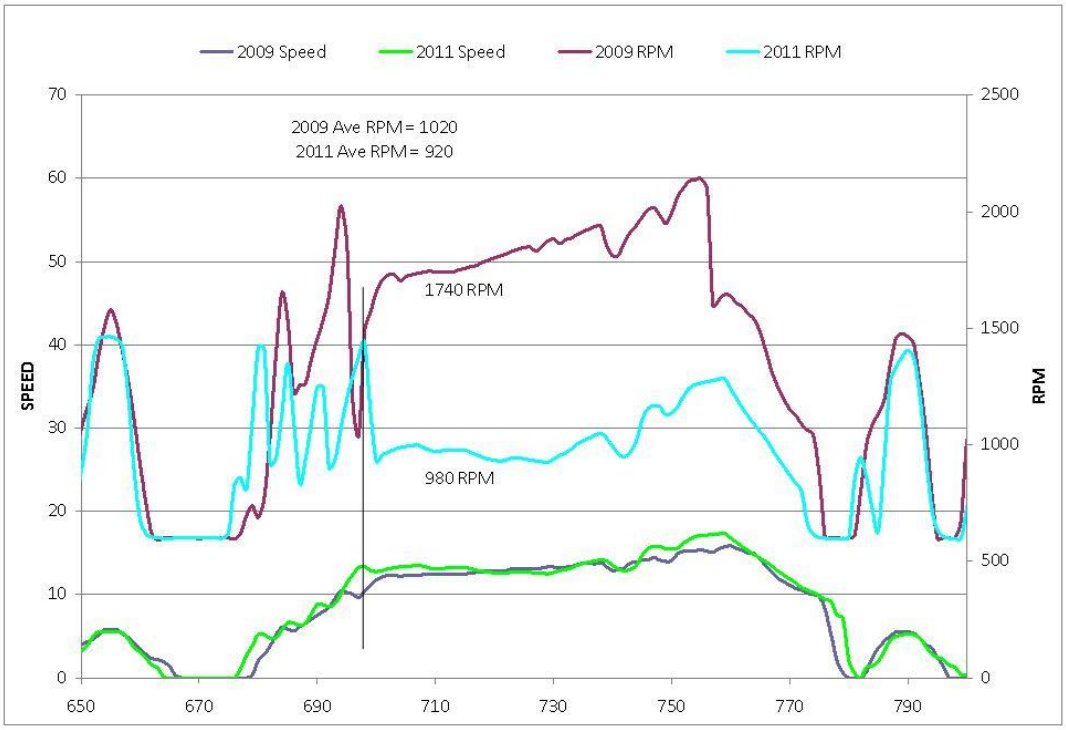


Figure 4-12: Navistar (12WZJ-B) engine speed repeatability while following the port cycle.

5 Results and Discussion for Goods Movement Vehicles

The results are reported in several sections, according to application. This first section focuses on the heavy, heavy-duty gas trucks and is followed by buses. This order can be justified from data in SCAQMD's 2012 AQMP as show in Table 5-1 where the HHD trucks are about 80% of truck/bus emissions in 2014 and about 70% in 2023. Focusing on the vehicle category with the greatest emissions contribution to the inventory provides the AQMD with the most likely path to achieving their goal of reducing NO_x.

Table 5-1 Data from the SCAQMD's 2012 AQMP (tons NO_x/day)

Code	Source Category	2014	2023
736	Heavy Heavy Duty Gas Trucks ((HHD)	1.02	0.96
746	**Heavy Heavy Duty Diesel Trucks (HHD)**	**76.43**	**32.63**
760	Diesel Urban Bus (UB)	13.4	11.03
762	Gas Urban Bus (UB)	0.76	0.70
772	Diesel School Buses (SB)	2.15	1.81
777	Gas Other Buses (OB)	0.86	0.53
	Total	94.62	47.66

Emission factors for all cycles are presented on the basis of grams per mile and for the UDDS cycle as grams per brake-hp-hr in order to compare with FTP values and. The emission factor in grams/mile is more useful for inventory purposes.

5.1 Test Trucks

Nine trucks used for good movement were tested on a number of chassis cycles. Selected information for the trucks were identified and listed in Table 1-1. The LPG vehicle in Category VI was the only one found to be available in the Los Angeles area so we presume the market share is very small for such vehicles. LPG truck odometer reading was 103,608 but the truck owner said the engine was installed in an existing chassis and the engine mileage was <20,000 miles.

5.2 Test Conditions

Vehicles were tested on the UDDS cycle and on three port cycles that more closely represented in-use activities for a goods movement vehicle. The loads for the goods movement vehicles were set at 69,500 lb. load and street #2 CARB diesel fuel was used. Both load and fuel matched in-use conditions. The emission values represent the average of triplicate runs and the graphs show the confidence limits to one standard deviation.

5.3 Emissions from the UDDS Cycle

As mentioned earlier, the brake specific emissions values from the UDDS chassis dyno test are often compared with the values measured in the heavy-duty FTP certification test on an engine dyno. This comparison provides some indication that the selected vehicle is representative of

the desired FEL and technology. This section focuses on NOx emissions given the interest in the original RFP.

5.3.1 Brake-specific emissions from Hot UDDS Cycle

Figure 5-1 shows the UDDS values for NOx in Category IV ranged from 1.6 to 2.7 g/bhp-hr versus a certification standard of about 1.2 so values for the hot UDDS are on the high side. Category VI has the only LPG Class 8 truck that we were able to find in the Los Angeles area. Although there were no fault codes on the vehicle, it was difficult to test (engine near overheating) and the emissions were higher than anticipated. The engine did not appear to be sized properly for the chassis and perhaps was leaner than expected. Future evaluations of LPG vehicles are needed to confirm these high results. For Category VII, emissions for the non-SCR or Navistar trucks were >0.2 g/bhp-h as expected as they were using solely using EGR, a unique shifting strategy and NOx emission credits. Values were about 1g/bhp-hr for the Navistar. Finally the Category VII with SCR had the lowest NOx emissions. The UDDS values ranged from 0.06 to 0.27 and were close to the certification values.

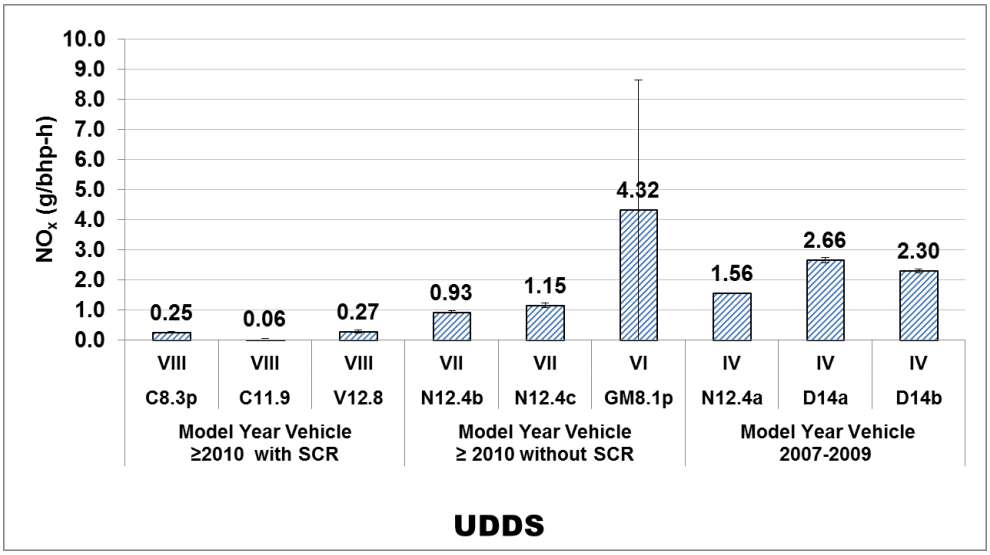


Figure 5-1 Brake Specific NOx Emissions for UDDS Cycle

5.3.2 Brake-specific emissions from Cold UDDS Cycle

Brake-specific emissions from cold UDDS cycle are shown in Figure 5-2 and values for the emission factors are increased significantly, about double for Category VII with SCR. Smaller increases were observed for Category IV with just EGR technology but then the emissions levels are about 10x those of systems with an SCR. For example the Navistar increased from about 1.6 to 1.7 and the DDC from 2.7 to 3.0. When the SCR is cold, raw engine out emissions are headed to the atmosphere.

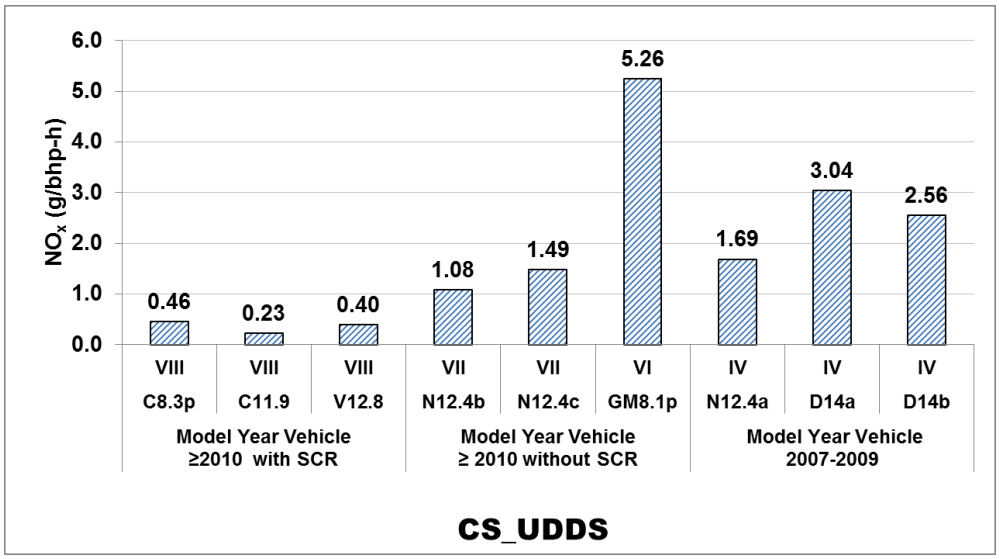


Figure 5-2 Brake Specific NOx Emissions for a Cold Start UDDS Cycle

[1] No error bars for the cold start tests because on only one test was performed

5.3.3 Emissions in g/mile for the UDDS cycle

Results were also analyzed and calculated on the basis of the emissions being expressed in grams per mile, the figure needed for calculating the inventory. These data are shown in Figure 5-6.

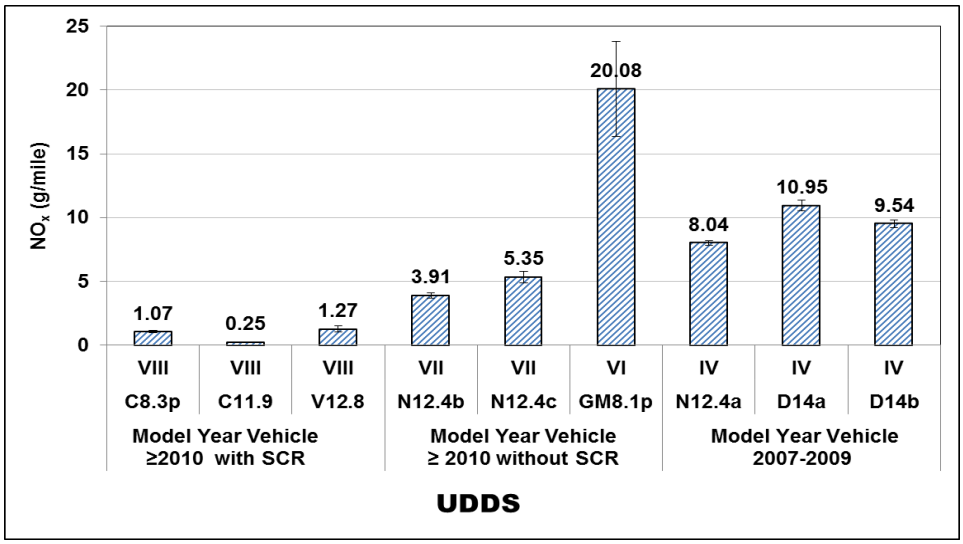


Figure 5-3: NO_x Emission Factors for hot UDDS cycle (g/mile)

Many have asked whether there is a relationship and a single factor to convert g/bhp-hr to g/mile. The answer to this question is presented in Table 5-2. The average factor is 4.45 with a coefficient of variation of 7.9%. This seems like a rather good fit considering the emission control technology varies widely. The value 4.5 compares with 3.5 used in earlier work.

Table 5-2 Relationship Between g/mile & g/bhp-hr for the Hot UDDS

Units									
g/mi	1.07	0.25	1.27	3.91	5.35	20.08	8.04	10.95	9.54
g/bkhp-hr	0.25	0.06	0.27	0.93	1.15	4.36	1.56	2.66	2.3
ratio	4.28	4.17	4.70	4.20	4.65	4.61	5.15	4.12	4.15

NOx emissions in g/mile for a cold UDDS cycle are presented in Figure 5-4. Values were a multiple of the values for the hot UDDS cycle, as expected. Note that NOx emissions 1 mile after cold start equal 2 miles after hot start.

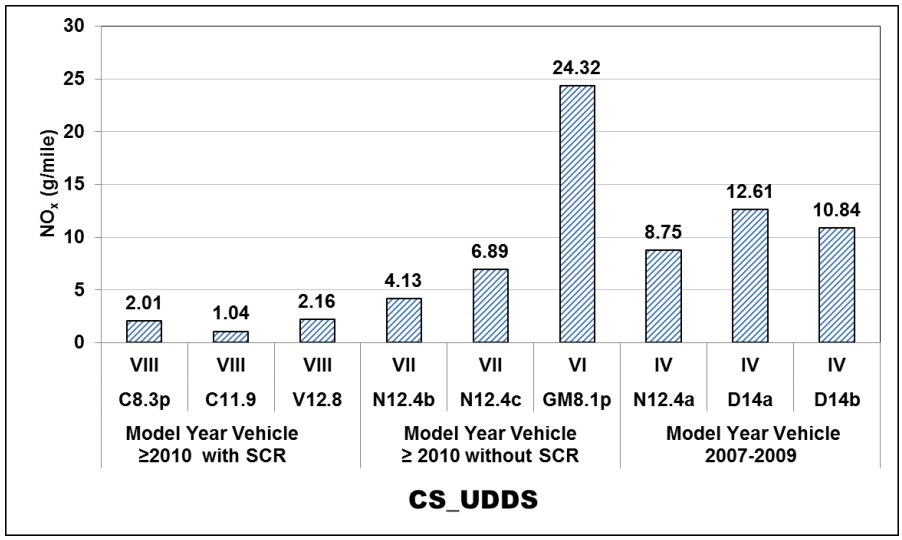


Figure 5-4: NO_x Emission factors for Cold Start UDDS Cycle (g/mile)

[1] No error bars for the cold start tests because on only one test was performed

Apportioning the NOx emissions into NO and NO_2 was part of the analysis. These data are shown in Table 5-3. Excluding the LPG truck, the percentage of NO_2 ranges from 24% to 59% and most values are >50%. These values are reminiscent of the early retrofit data and the subsequent rule that limited the increase above baseline to a 20% increase. Thus if the baseline was 10%, then the control technology was limited to 30%,

Table 5-3: Fraction of NO_2 to total NO_x for the Vehicles on the UDDS cycles

Vehicle			Port UDDS			Port UDDS_CS		
Category	Engine	MY	NO_2	NO_x	$\%NO_2$	NO_2	NO_x	$\%NO_2$
VIII	C8.3p	2010	0.54	1.07	50%	0.51	2.01	25%
VIII	C11.9	2011	0.15	0.25	58%	0.24	1.04	23%
VIII	V12.8	2011	0.30	1.27	24%	0.17	2.16	8%
VII	N12.4b	2011	2.19	3.91	56%	1.82	4.13	44%
VII	N12.4c	2011	2.18	5.35	41%	2.16	6.89	31%
VI	GM8.1	2009	0.05	20.08	0%	0.07	24.32	0%
IV	N12.4a	2009	3.08	8.04	38%	2.83	8.75	32%
IV	D14a	2008	6.13	10.95	56%	6.25	12.61	50%
IV	D14b	2008	5.60	9.54	59%	5.33	10.84	49%

Figure 5-5 shows the NOx emissions for the cold and hot start UDDS cycles for the port vehicles. In general NOx increased for the cold start and varied by a factor of 3 higher for the Cummins 11.9 SCR equipped vehicle (C11.9) to only a 6% increase for the Navistar 12.4 liter non SCR equipped vehicle (N12.4b). The cold start NOx emissions were much higher (160%) for the SCR equipped vehicles compared to the non-SCR equipped vehicles (only 15% higher). This shows that non-SCR cold start NOx emissions are not as big an impact as SCR equipped engines.

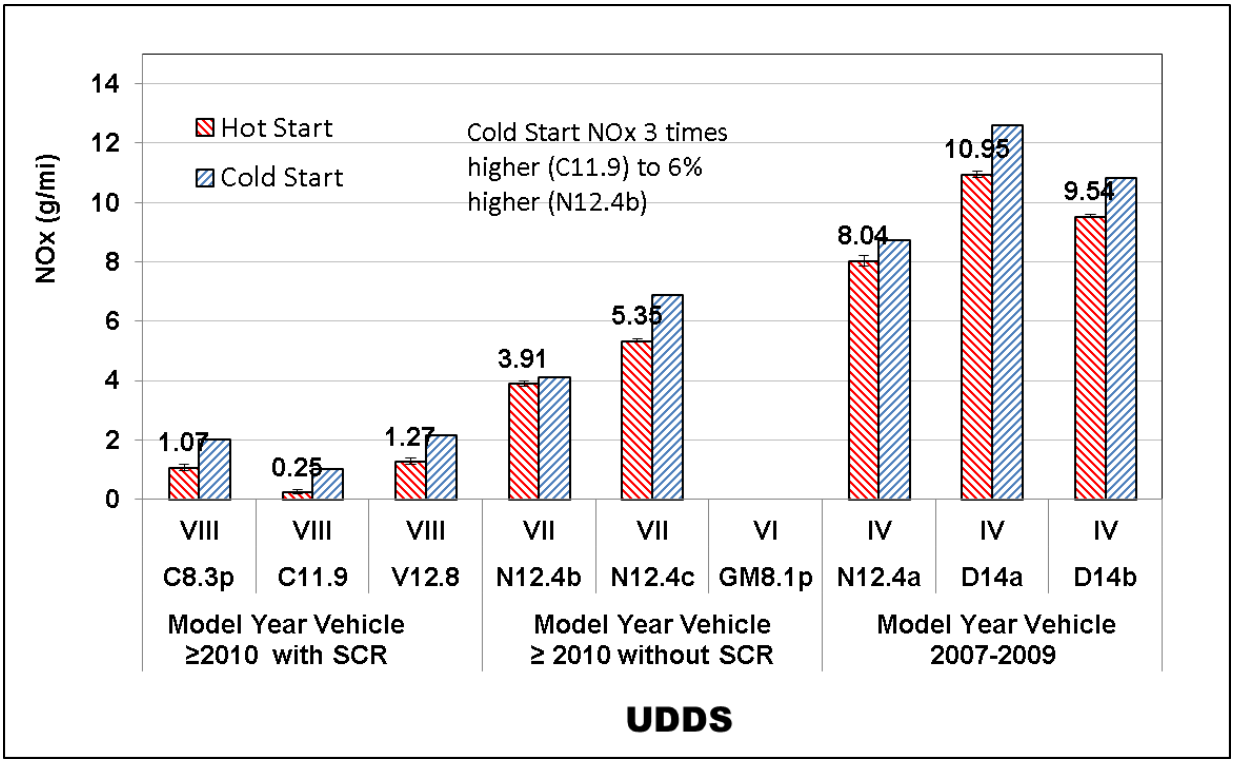


Figure 5-5 NOx emissions compared between cold and hot start UDDS cycles

5.4 Regulated Emissions from Port Cycles in grams/mile

Goods leaving the ports on HDD trucks travel over many routes and distances, few of which resemble the federal FTP driving schedule. Accordingly, the ports contracted TIAX to data log

and create driving schedules that better represented the in-use activity of trucks entering and leaving the ports. Based on travel distance, TIAX developed three driving schedules: 1) near dock; 2) local and 3) regional. Data are presented for each of the driving schedules.

5.4.1 NO_x emissions

The NO_x emission results in g/mile for the different port cycles are presented in Figure 5-6, Figure 5-7 and Figure 5-8. Clearly the single LPG truck remains an outlier and data suggest the engine is running lean or not properly configured for the chassis. Additional LPG vehicle testing is needed to confirm these results. That truck is not further discussed.

Trucks with SCR and EGR technology had the lowest NOx emissions with the observation that the longer the truck drove; that is the regional cycle, the lower the overall emissions. Presumably longer distances raise average catalyst temperature resulting in lower overall emissions.

Trucks with EGR and an active DPF showed a similar pattern with longest driving times resulting in the lowest emissions. Since nitrogen and oxygen in air react to form NOx, one suspects that the concentration of oxygen is higher for the near dock cycle. This hypothesis is being confirmed. In-use data did show excellent repeatability of the DDC product in the field.

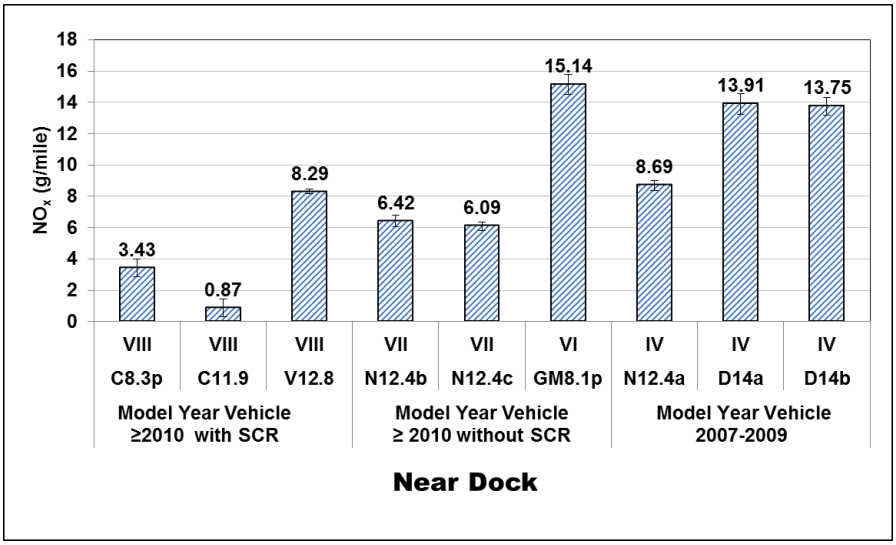


Figure 5-6: NO_x Emission factors for Near Dock Cycle (g/mile)

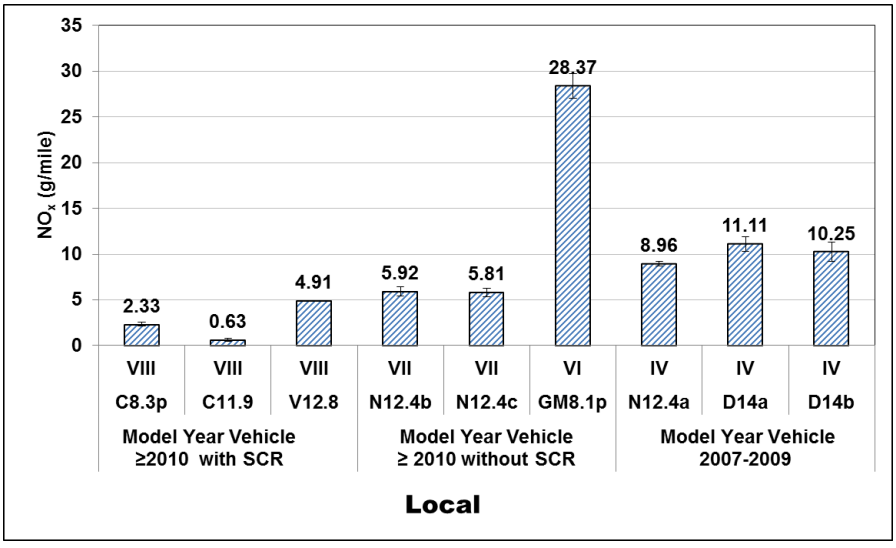


Figure 5-7: NOx Emission factors for Local Cycle (g/mile)

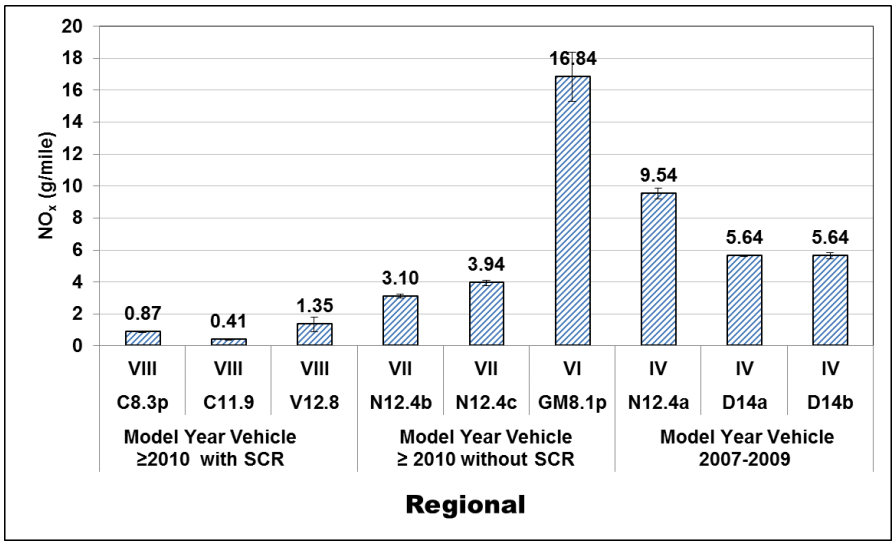


Figure 5-8: NOx Emission factors for Regional cycle (g/mile)

Category VII trucks had similar emissions for different driving cycles. These were the Navistar technology of cooled EGR and DPF. As mentioned in the background section, a discussion of these data is not warranted as this technology was pulled from the market and no longer offered.

5.4.2 Percentage of NOx emissions as NO_2

Apportioning the NOx emissions into NO and NO_2 for the port cycles was part of the analysis. These data are shown in Table 5-4. Excluding the LPG truck, the percentage of NO_2 ranged from 5% to 91%. Not surprising, the highest NO_2 level was observed for the vehicle with the lowest NOx level. Literature studies reveal that NO_2 reacts slower than NO over the SCR catalyst. Similar to the observation with the UDDS cycle, levels of NO_2 are high as compared with the ARB retrofit rule of 20% over baseline.

Table 5-4: Fraction of NO_2 to total NO_x for the Port Cycles

Vehicle			Near Dock			Local			Regional		
Category	Engine	MY	NO_2	NO_x	$\%NO_2$	NO_2	NO_x	$\%NO_2$	NO_2	NO_x	$\%NO_2$
VIII	C8.3p	2010	1.65	3.43	48%	1.18	2.33	51%	0.39	0.87	45%
VIII	C11.9	2011	0.79	0.87	91%	0.58	0.63	92%	0.36	0.41	86%
VIII	V12.8	2011	0.45	8.29	5%	0.28	4.91	6%	0.14	1.35	10%
VII	N12.4b	2011	2.13	6.42	33%	2.95	5.92	50%	1.55	3.10	50%
VII	N12.4c	2011	1.63	6.09	27%	1.99	5.81	34%	1.68	3.94	43%
VI	GM8.1p	2009	0.27	15.14	2%	-0.20	28.37	~0%	1.04	16.84	6%
IV	N12.4a	2009	3.18	8.69	37%	3.47	8.96	39%	4.17	9.54	44%
IV	D14a	2008	6.26	13.91	45%	6.59	11.11	59%	3.13	5.64	55%
IV	D14b	2008	5.12	13.75	37%	5.17	10.25	50%	3.04	5.64	54%

5.4.3 PM emissions

PM emission results for the different port cycles are presented in Figure 5-9, Figure 5-10, and Figure 5-11. Except for the LPG truck, PM emissions were ≤2 mg/mi for most vehicle/cycle combinations. There were a few vehicle/cycle combinations above 2 mg/mi for some of the 2010+ vehicles on the Regional and near dock cycles.

Figure 5-12 shows the PM emissions for the cold start and hot start UDDS cycles with the propane vehicle results removed. The cold start PM emissions for the Cummins 2010 SCR equipped 8.3 liter engine was 17 times higher than the hot start emissions (22.9 mg/mi vs 1.33 mg/mi respectively). After closer investigation it appears that a passive regeneration may have occurred as indicated by a high exhaust temperature, but no regeneration illumination lamp from the engine, see Real-Time analysis section. The cold-start PM was slightly higher for the other port vehicles tested, but the emission factors were still very low were the difference was not statistically significant based on the uncertainty of the measurement method and the low filter weight obtained.

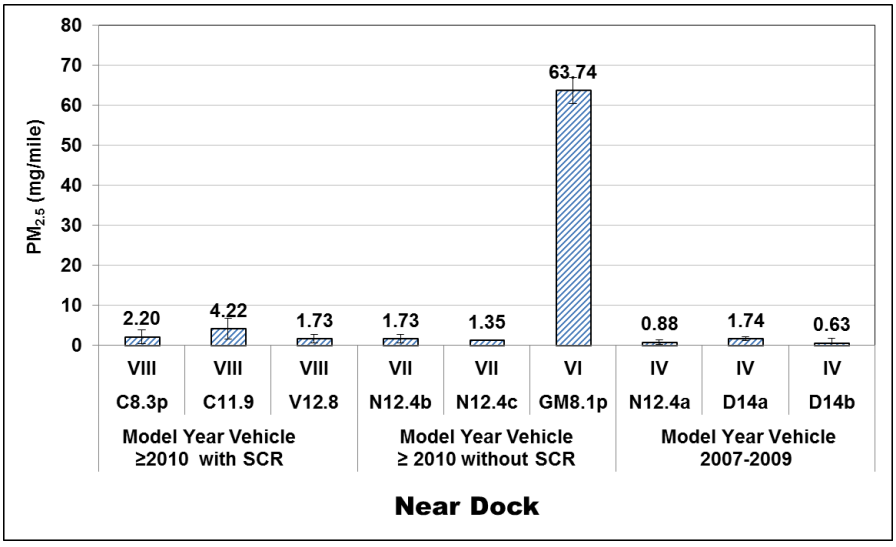


Figure 5-9: PM Emission factors for Near Dock cycle (g/mile)

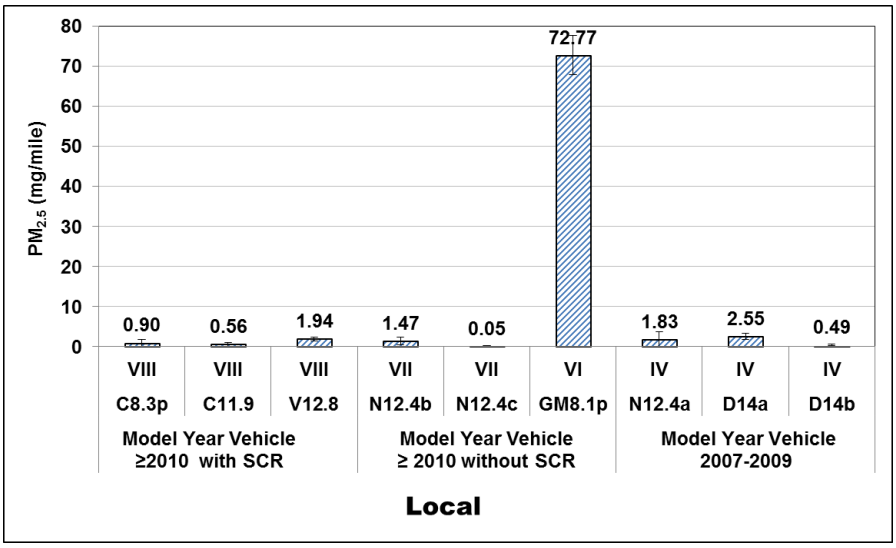


Figure 5-10: PM Emission factors for Local cycle (g/mile)

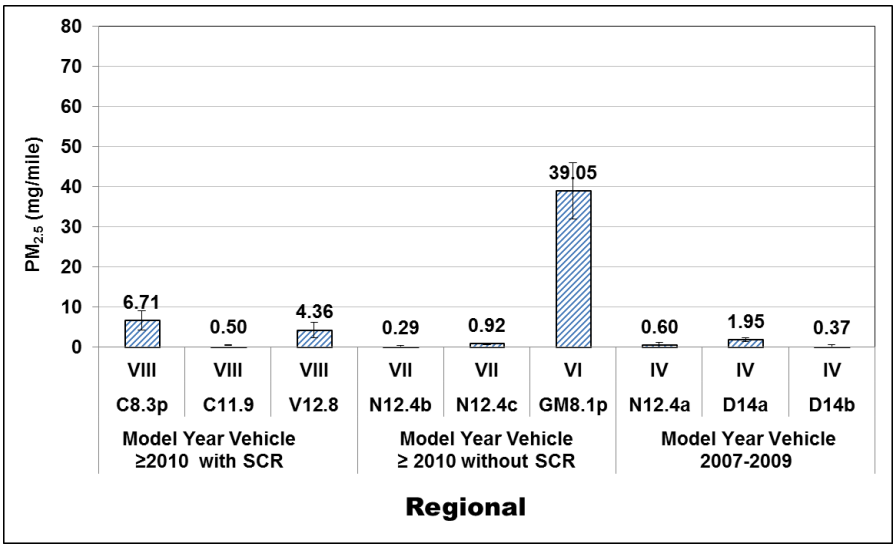


Figure 5-11: PM Emission factors for Regional cycle (g/mile)

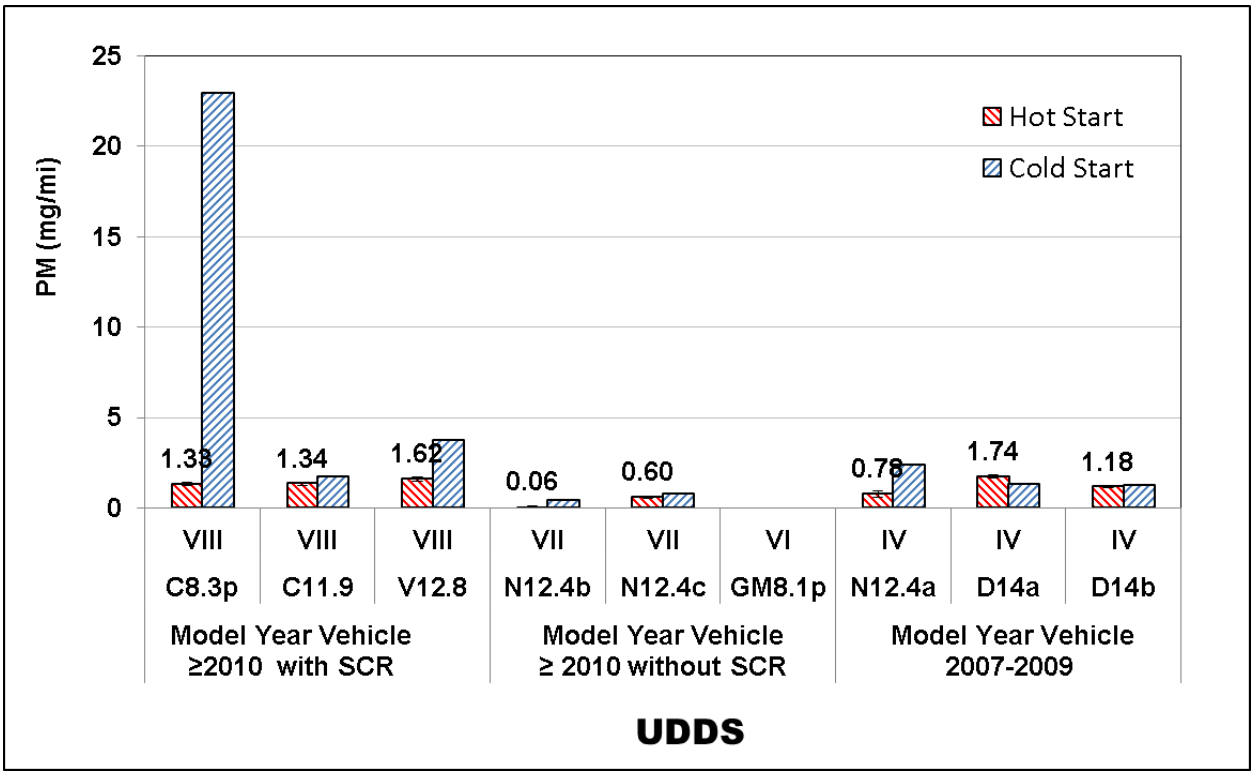


Figure 5-12 PM emissions for the cold and hot start UDDS cycles (port vehicles)

5.4.4 THC/NMHC/CH_4 and CO emissions—UDDS cycle

Table 5-5 and Table 5-6 show the emission factors for THC, CH_4, NMHC and CO for the hot and cold UDDS cycles. Except for the LPG truck, values are very low. This finding is not surprising given that the exhaust passes over a diesel oxidation catalyst (DOC) with noble metals, a catalyst that is well known to efficiently convert hydrocarbon and carbon monoxide to water and carbon dioxide.

Table 5-5: THC, CH4, NMHC, and CO emissions for the UDDS cycle

Vehicle			Emission Factor (g/mi)			
Category	Engine	MY	THC	CH4	NMHC	CO
VIII	C8.3p	2010	0.00	0.01	-0.01	-0.18
VIII	C11.9	2011	0.01	0.02	-0.01	-0.12
VIII	V12.8	2011	0.00	0.02	-0.01	-0.10
VII	N12.4b	2011	0.24	0.14	0.13	6.12
VII	N12.4c	2011	0.03	0.02	0.01	0.69
VI	GM8.1p	2009	22.41	1.43	21.38	74.40
IV	N12.4a	2009	0.02	0.02	0.00	-0.19
IV	D14a	2008	0.03	0.04	-0.01	0.27
IV	D14b	2008	0.02	0.04	-0.01	-0.14

Table 5-6: THC, CH_4, NMHC, and CO emissions for the Cold Start UDDS Cycle

Cycle	Category	Vehicle Engine	MY	Emission Factor (g/mi) THC	CH4	NMHC	
CS-UDDS	VIII	C8.3p	2010	0.02	0.01	0.02	-0.07
CS-UDDS	VIII	C11.9	2011	0.03	0.03	0.01	-0.03
CS-UDDS	VIII	V12.8	2011	0.06	0.04	0.02	0.27
CS-UDDS	VII	N12.4b	2011	0.08	0.05	0.04	1.44
CS-UDDS	VII	N12.4c	2011	0.08	0.04	0.05	0.46
CS-UDDS	VI	GM8.1p	2009	14.89	0.76	14.38	77.16
CS-UDDS	IV	N12.4a	2009	0.03	0.02	0.01	-0.10
CS-UDDS	IV	D14a	2008	0.05	0.04	0.01	0.85
CS-UDDS	IV	D14b	2008	-0.02	0.03	-0.04	0.54

5.4.5 THC/NMHC/CH_4 and CO emissions—In-use port cycles

Table 5-7, Table 5-8 and Table 5-9 show the emission factors for THC, CH_4, NMHC and CO for all three port cycles. Except for the LPG truck, values are very low. As stated earlier this finding is not surprising given that the exhaust passes over a diesel oxidation catalyst (DOC) with noble metals, a catalyst that is well known to efficiently convert hydrocarbon and carbon monoxide to water and carbon dioxide. While truck emissions need to meet a carbon monoxide limit, the data show that NOx is more important as the measured values are <10% of the CO standard.

Table 5-7: THC, CH_4, NMHC, and CO emissions for the Near Dock port cycle

Category	Vehicle Engine	MY	Emission Factor (g/mi) THC	CH4	NMHC	CO
VIII	C8.3p	2010	0.04	0.03	0.02	-0.41
VIII	C11.9	2011	0.06	0.09	-0.02	-0.50
VIII	V12.8	2011	0.34	0.06	0.29	0.65
VII	N12.4b	2011	0.36	0.10	0.28	3.21
VII	N12.4c	2011	0.24	0.07	0.18	2.06
VI	GM8.1p	2009	33.79	1.61	32.73	157.34
IV	N12.4a	2009	0.10	0.07	0.04	-0.15
IV	D14a	2008	0.08	0.09	0.00	2.83
IV	D14b	2008	0.19	0.07	0.13	0.16

Table 5-8: THC, CH_4, NMHC, and CO emissions for the Local port cycle (g/mile)

Vehicle			Emission Factor (g/mi)			
Category	Engine	MY	THC	CH4	NMHC	CO
VIII	C8.3p	2010	0.03	0.04	0.00	-0.03
VIII	C11.9	2011	0.45	0.17	0.30	5.13
VIII	V12.8	2011	0.19	0.04	0.15	1.07
VII	N12.4b	2011	27.88	1.50	26.86	117.82
VII	N12.4c	2011	0.06	0.04	0.03	-0.31
VI	GM8.1p	2009	-0.02	0.07	-0.08	0.45
IV	N12.4a	2009	0.07	0.05	0.03	0.10
IV	D14a	2008	0.00	0.00	0.00	0.00
IV	D14b	2008	-0.01	0.01	-0.02	-0.26

Table 5-9: THC, CH_4, NMHC, and CO emissions for the Regional port cycle

Vehicle			Emission Factor (g/mi)			
Category	Engine	MY	THC	CH4	NMHC	CO
VIII	C8.3p	2010	-0.01	0.01	-0.02	-0.26
VIII	C11.9	2011	0.01	0.02	-0.01	-0.18
VIII	V12.8	2011	0.00	0.02	-0.02	-0.15
VII	N12.4b	2011	0.09	0.06	0.04	1.76
VII	N12.4c	2011	0.05	0.02	0.04	0.19
VI	GM8.1p	2009	11.91	1.02	11.14	60.08
IV	N12.4a	2009	0.02	0.02	0.00	-0.16
IV	D14a	2008	0.02	0.03	0.00	0.23
IV	D14b	2008	0.01	0.02	-0.01	-0.07

5.5 Non-regulated Gaseous Emissions

5.5.1 NH_3 emissions

Ammonia emissions were of interest for the trucks with the latest technology. Ammonia can be released from diesel trucks with SCRs if excess urea is added, so called ammonia slip. Ammonia is also created and released with trucks using natural gas as the three-way catalyst used for after treatment can produce ammonia by a complicated series of reactions on the catalyst surface. Results are shown from Figure 5-13 to Figure 5-17. NH_3 emissions ranged from approximately 10 to 100 mg/mi over all combinations of vehicle and hot cycles. Looking at the UDDS, the results show that vehicles had similar low ammonia releases with and without the SCR catalyst. This finding was true even with the lowest level of NOx measured when a SCR catalyst was used. The CS-UDDS cycle with SCR showed slightly higher NH_3 emissions than the other cycles suggesting a timing issue with the introduction of the urea.

Table 8-26 shows of the 54 diesel tests conducted, only 2 vehicles were 5 times the lower detection limit (LDL) (i.e. greater than 5ppm), and 26 tests were above 2 time the LDL (2 ppm), see Section 8.4.4 for discussion of the LDL's used in this report. Of the 2 tests above 5*LDL,

both were for a cold start SCR equipped diesel vehicle. For the 26 tests above 2* LDL these were both SCR and non-SCR equipped vehicles. It is not expected that a non-SCR equipped vehicle had more NH_3 emissions than an SCR equipped vehicle. Five of seven tests for the propane vehicle also had NH_3 greater than 5 ppm and 2 were over 50 ppm suggesting very high relative NH_3 emissions for the propane vehicles.

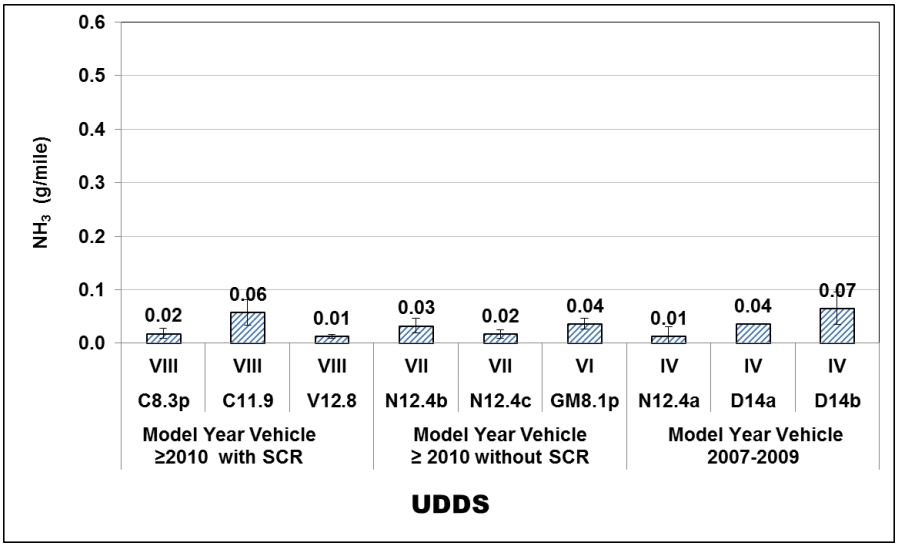


Figure 5-13: NH_3 Emission Factors for UDDS cycle (g/mile)[1]

[1] NH_3 scale is based on 10 ppm raw exhaust concentration

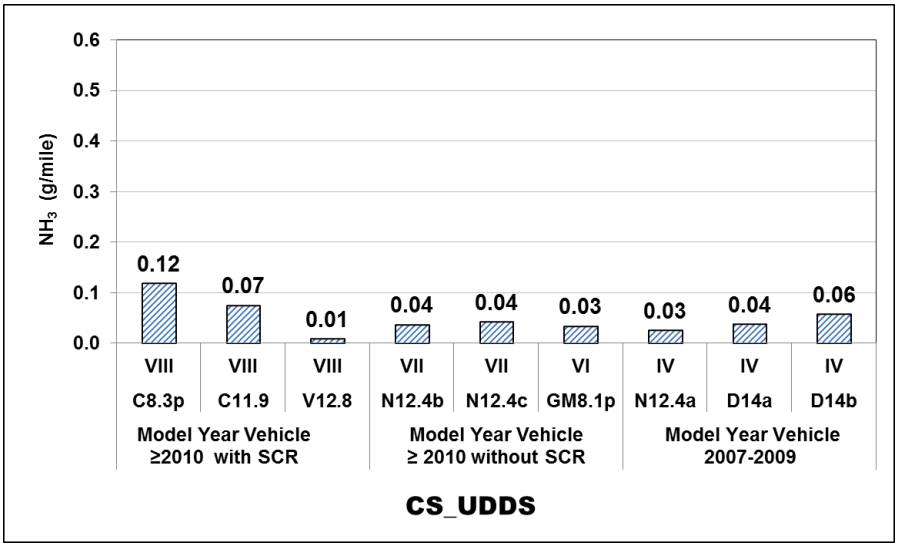


Figure 5-14: NH_3 Emission Factors for Cold Start UDDS cycle (g/mile)[1]

[1] NH_3 scale is based on 10 ppm raw exhaust concentration, thus 10 ppm NH3 in the raw exhaust will be approximately 0.6 g/mi (full scale) for perspective.

[2] No error bars for the cold start tests because on only one test was performed

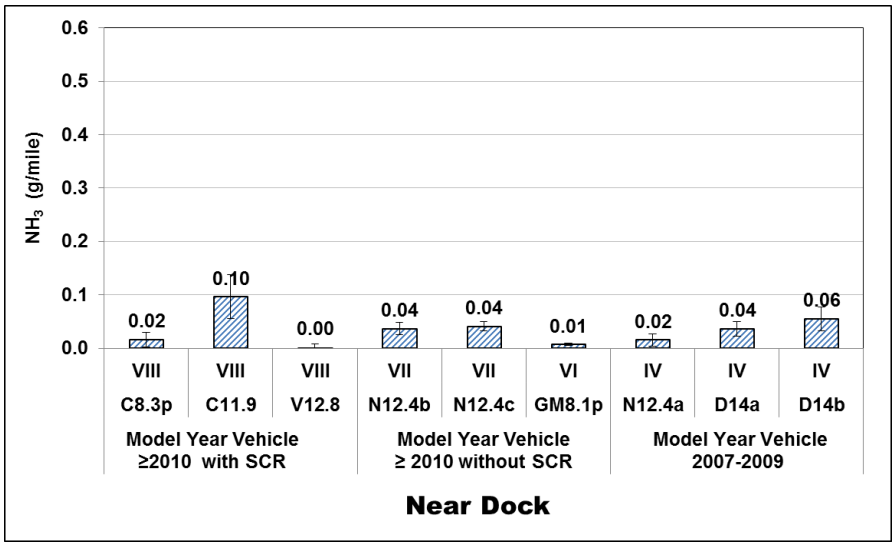


Figure 5-15: NH_3 Emission factors for Near Dock Cycle (g/mile)

[1] NH_3 scale is based on 10 ppm raw exhaust concentration

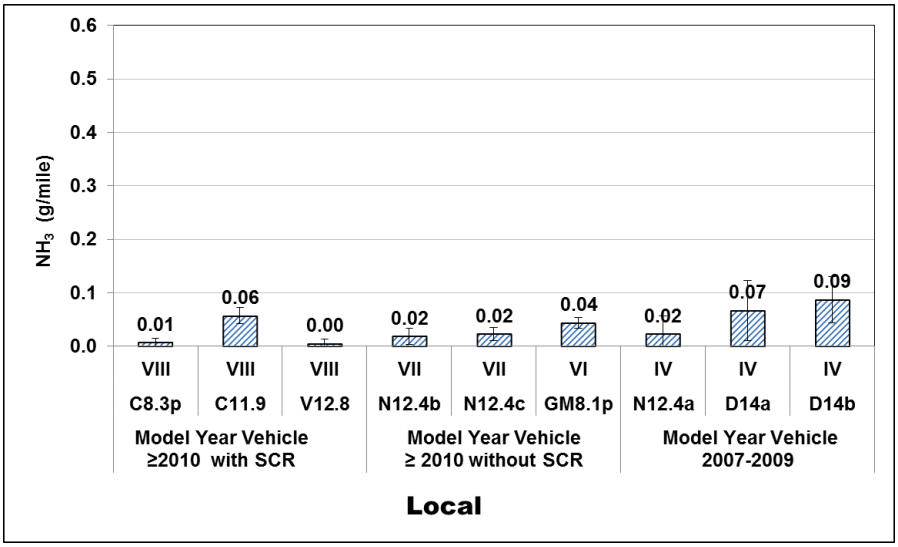


Figure 5-16: NH_3 Emission factors for Local Cycle (g/mile)

[1] NH_3 scale is based on 10 ppm raw exhaust concentration

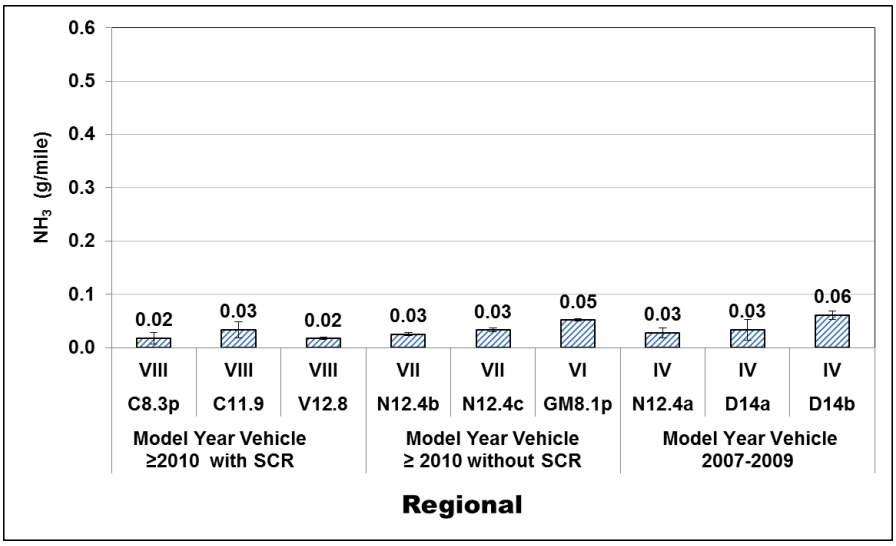


Figure 5-17: NH_3 Emission factors for Regional Cycle (g/mile)[1]

[1] NH_3 scale is based on 10 ppm raw exhaust concentration

5.5.2 Selected Toxic Emissions (1,3-butadiene and BTEX)

A slip stream of the exhaust was passed through tubes containing three beds of materials where hydrocarbon gases are adsorbed. Subsequent off-line analysis focused on the measurement of 1,3-butadiene and BTEX. Some early results for benzene were confounded with the co-elution of the butanol used in the CPC but those corrected results did not affect the conclusions. Basically with the DOC catalyst associated with the DPF the ACES project showed that all hydrocarbon emissions would be very low. Results are shown in Figure 5-18 to Figure 5-21. Except for the Navistar vehicles and a couple of apparent outliers, the rest of the values are <10mg/mile, at levels that were near the detection limit of the method. In some cases when exhaust emissions are compared with the ambient values, results are negative values showing the vehicle levels were below ambient levels. High uncertainty levels are one consequence of being near the low detection level.

The propane vehicle showed high BTEX emissions where the vehicles tested averaged 6.5±9.3 mg/mi, 9.7±12 mg/mi, and 22.4±19 mg/mi of 1,3-Butadiene, n-butane, and Benzene emissions respectively. The remaining BTEX species were below 2 mg/mi and were not statistically significant.

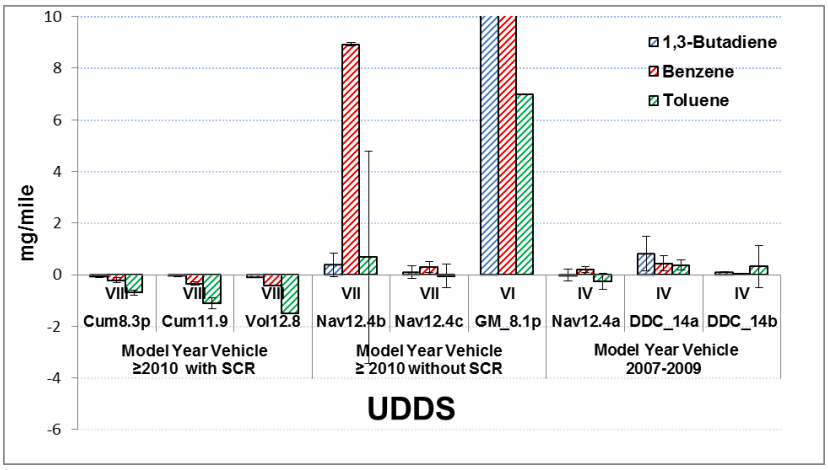


Figure 5-18 Emissions in mg/mile for Butadiene & BTEX for the UDDS Cycle

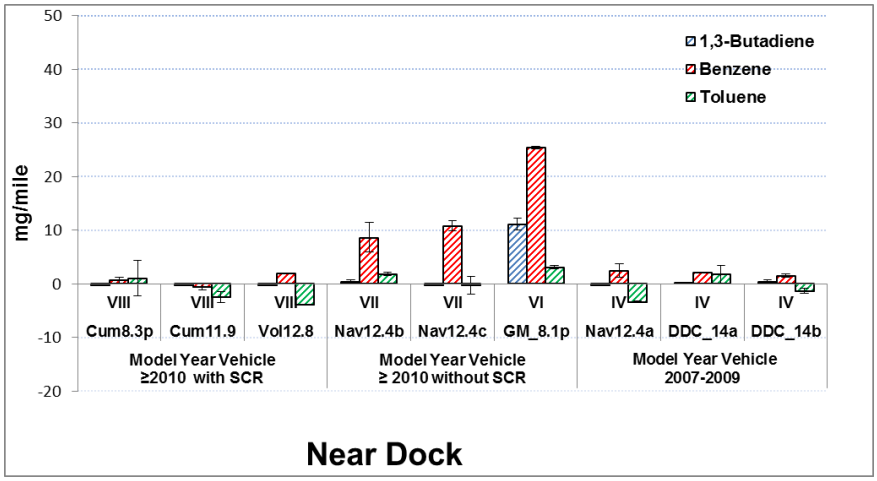


Figure 5-19 Emissions in mg/mile for Butadiene & BTEX for the Near Port Cycle

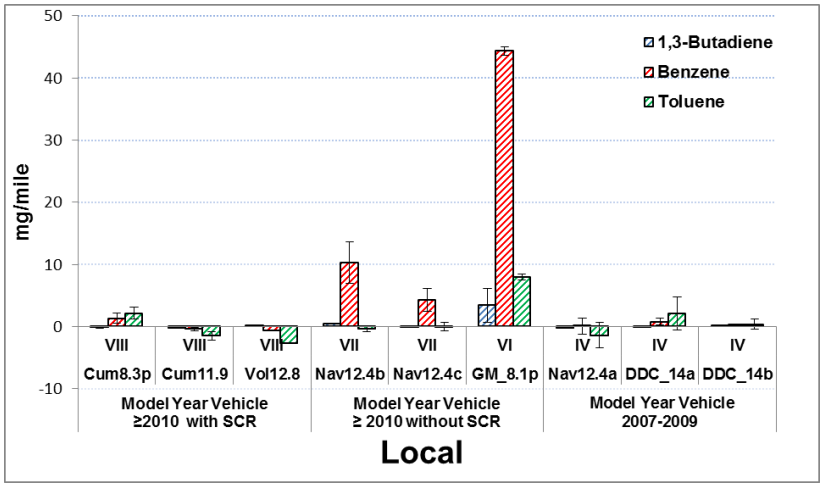


Figure 5-20 Emissions in mg/mile for Butadiene & BTEX for the Local Port Cycle

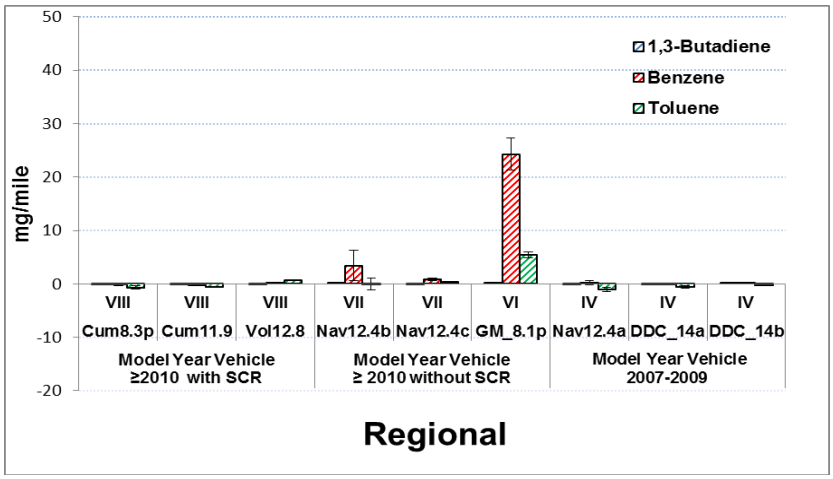


Figure 5-21 Emissions in mg/mile for Butadiene & BTEX for the Regional Port Cycle

5.5.3 Selected Toxic Emissions (carbonyls)

A slip stream of the exhaust was passed through tubes of silica gel with DNPH at controlled rates to adsorb the carbonyls and ketones in the exhaust stream. Subsequent off-line analysis focused on the measurement of aldehydes and ketones. However, as mentioned previously, the ACES project showed that all hydrocarbon emissions would be very low due to the DOC catalyst containing noble metals that is associated with the DPF.

Results are shown in Figure 5-22 to Figure 5-26. As expected, formaldehyde had by far the highest emissions. Except for the LPG vehicle with the three-way catalyst and one acetone outlier with the C11.9 vehicle for the hot UDDS cycle all values are low. We suspect the outlier sample picked up laboratory acetone during the handling. Otherwise the values are <50mg/mile, levels near the detection limit of the method. In some cases when exhaust emissions are compared with the ambient values, results are negative values showing the vehicle levels were below ambient levels. Confidence levels with this method were better than those for the volatile toxics.

The Carbonyls were high for the propane vehicle where formaldehyde and acetaldehyde emissions averaged 241±253 mg/mi and 42±48 mg/mi respectively (one standard deviation error bars). The remaining Carbonyls species were below 2 mg/mi and were not statistically significant.

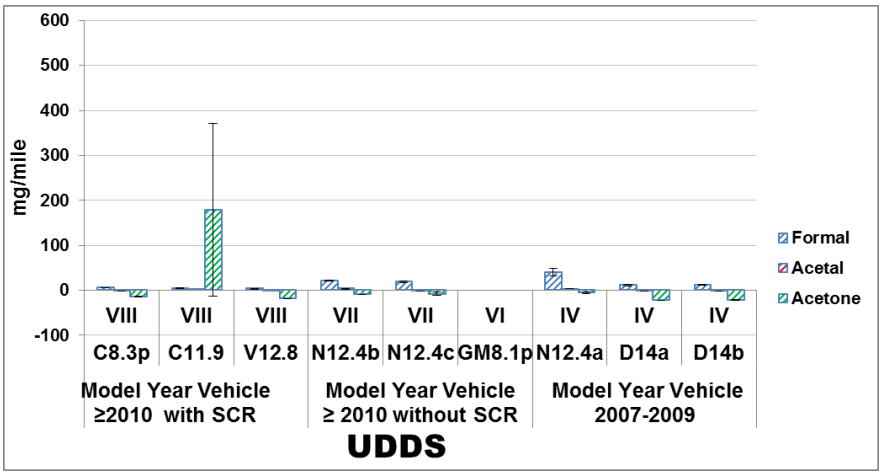


Figure 5-22 Emissions in mg/mile for Carbonyls & Ketones for the UDDS Cycle

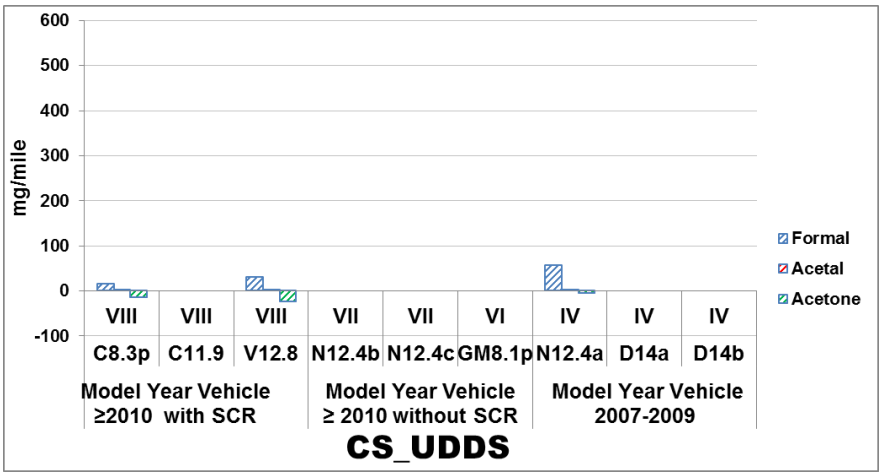


Figure 5-23 Emissions in mg/mile for Carbonyls & Ketones for cold- UDDS Cycle

[1] No error bars for the cold start tests because on only one test was performed

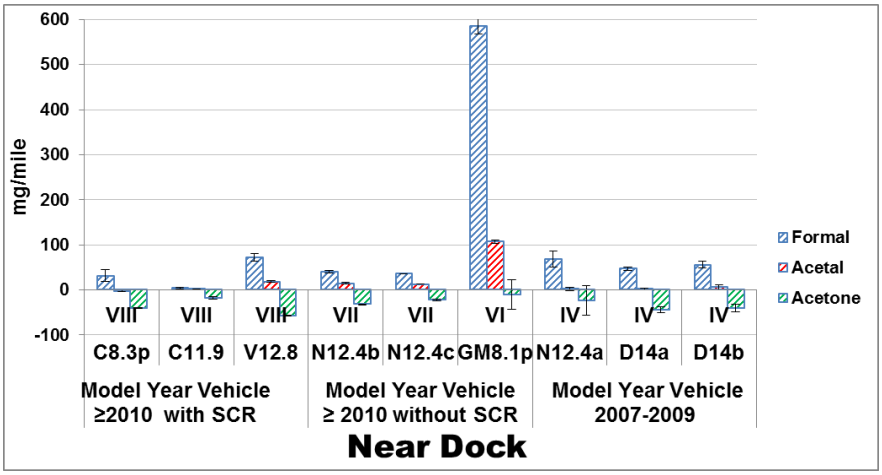


Figure 5-24 Emissions in mg/mile for Carbonyls & Ketones for the Near Port Cycle

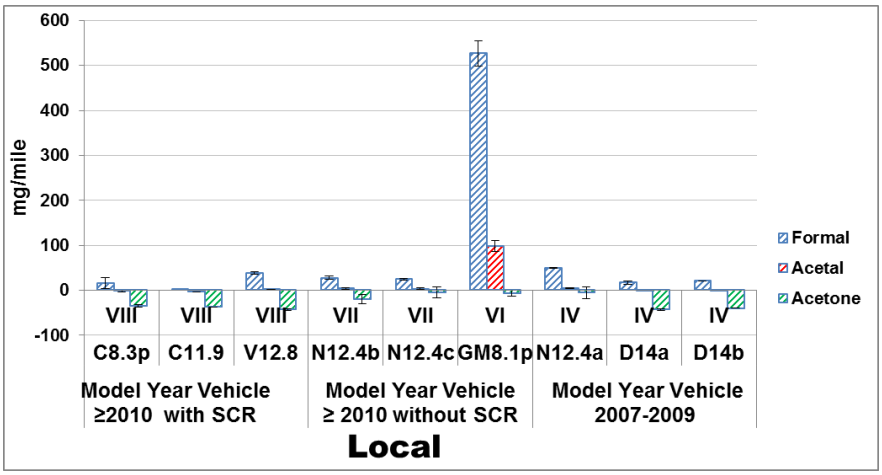


Figure 5-25 Emissions in mg/mile for Carbonyls & Ketones for the Local Port Cycle

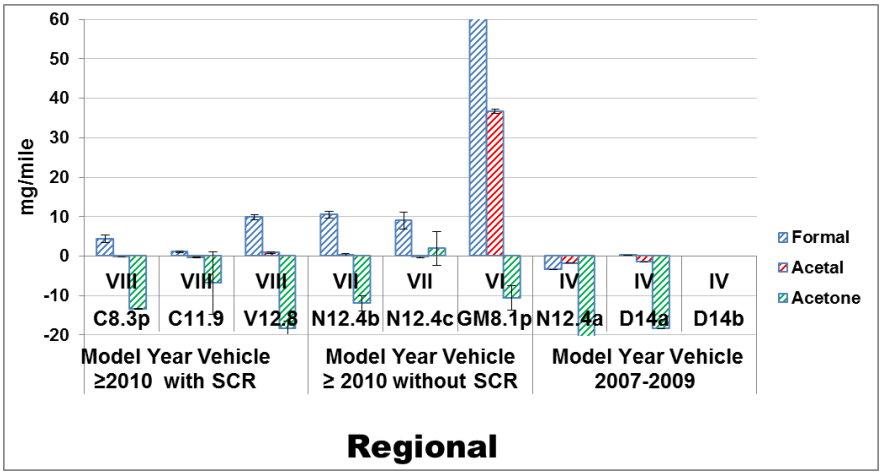


Figure 5-26 Emissions in mg/mile for Carbonyls & Ketones for the Regional Port Cycle

5.6 Non-regulated PM Emission Data

This section covers some of the non-regulated PM data that was included in the contract. These data include: 1) the fractioning of the PM mass emissions into organic and elemental carbon (OC & EC) and 2) the particle size distribution with a focus on ultra-fines (<100nm).

5.6.1 Fractionation of the PM mass into OC and EC

As described earlier, samples of exhaust were filtered with quartz media and subsequently processed to measure the amount of OC and EC. Results are shown in Figure 5-27 to Figure 5-31. The results include background subtraction for both EC and CO PM emissions at 0.5 ug/filter and 10 ug/filter respectively (see discussion in Section 8.4.4). For all samples, the level of EC is <2mg/mile as DPF have very high filtration efficiencies for EC. The organic PM emissions were higher where the propane vehicle showed a large OC fraction for a cold start at 78 mg/mi and less than 7 mg/mi for the warm tests. The diesels were much lower at 6 mg/mi which is slightly higher than the EC, but not statistically significant. More OC was observed in the cold start UDDS for the diesels and propane vehicle than after the engine and catalysts were warm.

It is as expected that the precursors to OC are in the vapor phase and pass through the DPF. Findings in this project are similar to those of the Advanced Collaborative Emissions Study[10] which found that for DPF technology engine the PM was composed mainly of sulfate and organic carbon. In both studies, elemental carbon and metals were a small fraction of the PM mass.

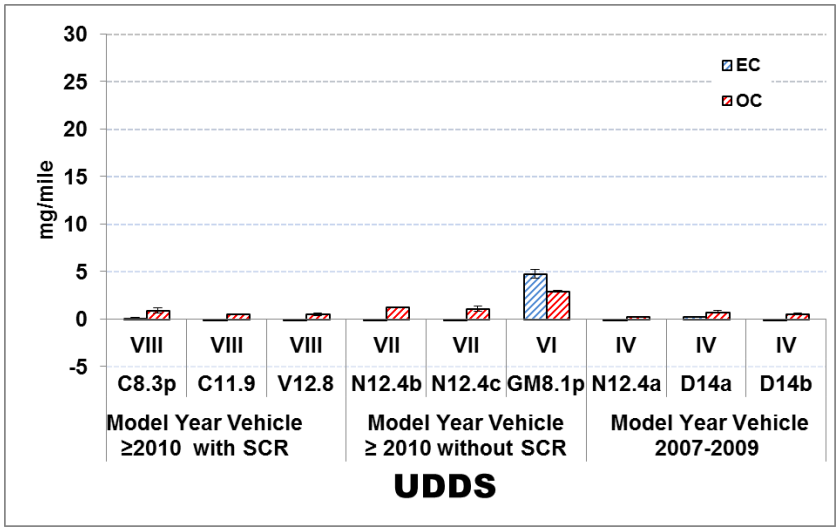


Figure 5-27 Emissions in grams/mile for the PM as OC & EC for the UDDS Cycle

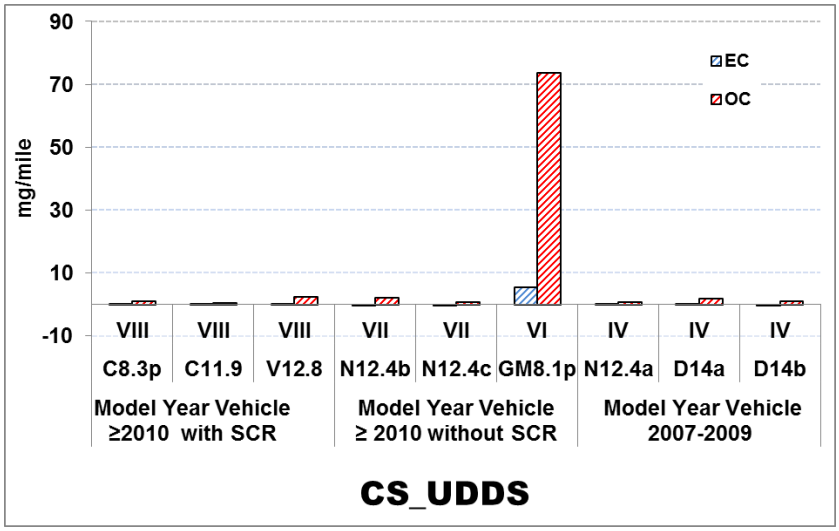


Figure 5-28 Emissions in grams/mile for the PM as OC & EC for cold- UDDS Cycle

[1] No error bars for the cold start tests because on only one test was performed

[10] CRC Report: ACES Phase 1 of *the Advanced Collaborative Emissions Study*, June 2009

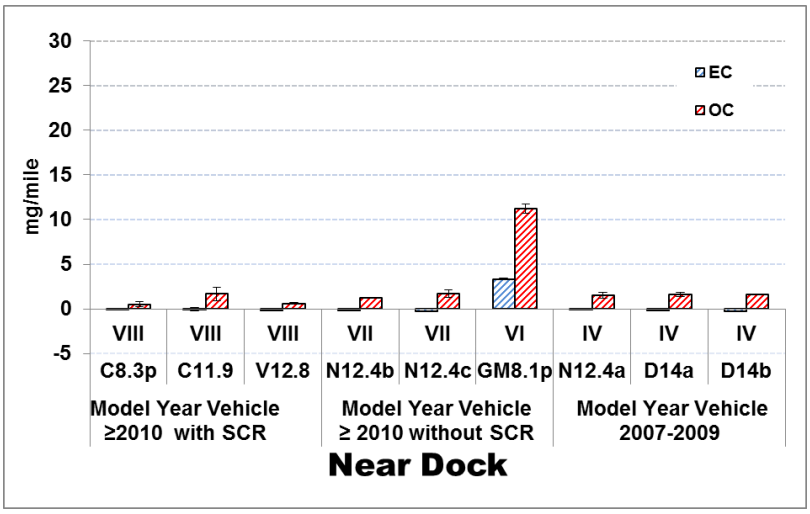


Figure 5-29 Emissions in grams/mile for the PM as OC & EC for the Near Port Cycle

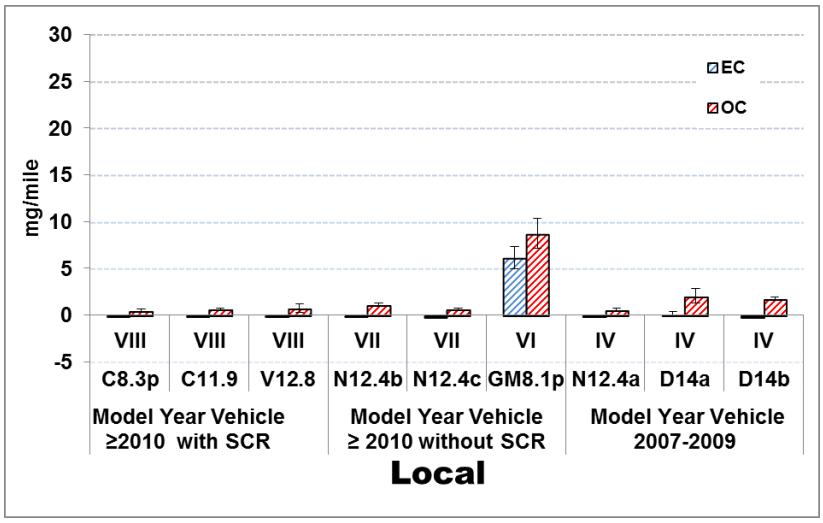


Figure 5-30 Emissions in grams/mile for the PM as OC & EC for the Local Port Cycle

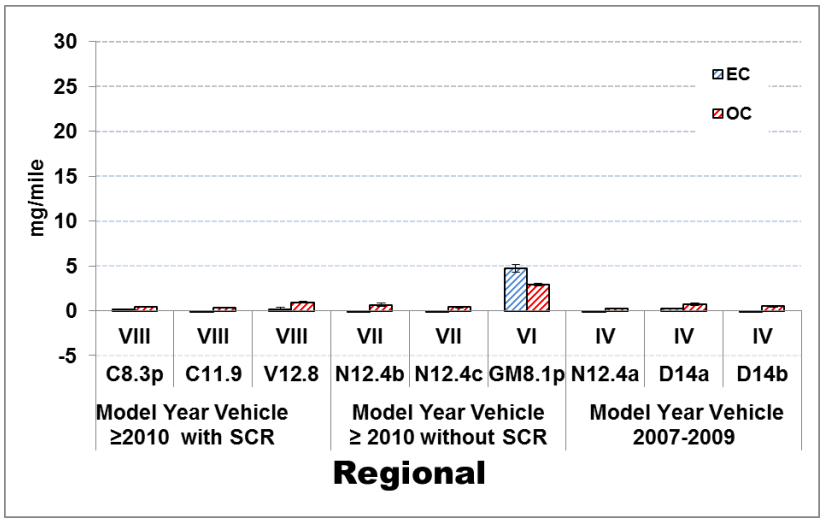


Figure 5-31 Emissions in grams/mile for the PM as OC & EC for the Regional Port Cycle

5.6.2 Measurement of the real-time and ultrafine PM emissions

Two instruments were used for the real time PM analysis as described earlier. These are the Dekati DMM and the f-SMPS. The DMM was used to characterize the real time PM mass concentration and the f-SMPS was used for the ultrafine PM emissions characterization.

Real-time PM mass DMM

As presented earlier the PM mass of the gravimetric method were very low and typically around 1.4 mg/mi or 0.4 gm/bhp-hr for most port vehicles tested. The average PM mass from the DMM measurement method averaged 0.5 mg/mi and 0.1 mg/bhp-hr for the same vehicles.

The lower real-time PM emission rate compared to the gravimetric method is not surprising as there is less confidence in the gravimetric method at filter weights below 40 µg. During the testing the actual filter weights ranged from 10-20 µg where UCR's CVS tunnel blank averages 5µg with a 5µg single standard deviation. As such many of the PM gravimetric measurements were at the detection limit of the method.

The DMM results suggest the actual PM mass at these low filter weights may be four times lower. The real-time instrument do have a lower detection limit, but that lower detection capability is not perfect and may have a poor mass correlation to the gravimetric mass method. As such, it is hard to quantify the true mass emission rate of DPF equipped vehicles and the actual PM mass may be lower than reported.

The real-time PM instrument is also useful for diagnosing PM anomalies and outliers. The Navistar vehicle showed a total PM mass of 0.68, 0.76 and 4.04 mg/mi on the local port cycle as reported earlier. Figure 5-32 shows the DMM PM mass concentration on a second by second basis for the 4.04 mg/mi test case. At 2500 seconds there was a large PM spike, as denoted with the blue line, which was not present in the other two tests. After closer investigation it appears that a passive regeneration occurred as can be seen by the high exhaust temperature, but no regeneration illumination lamp from the engine on this test.

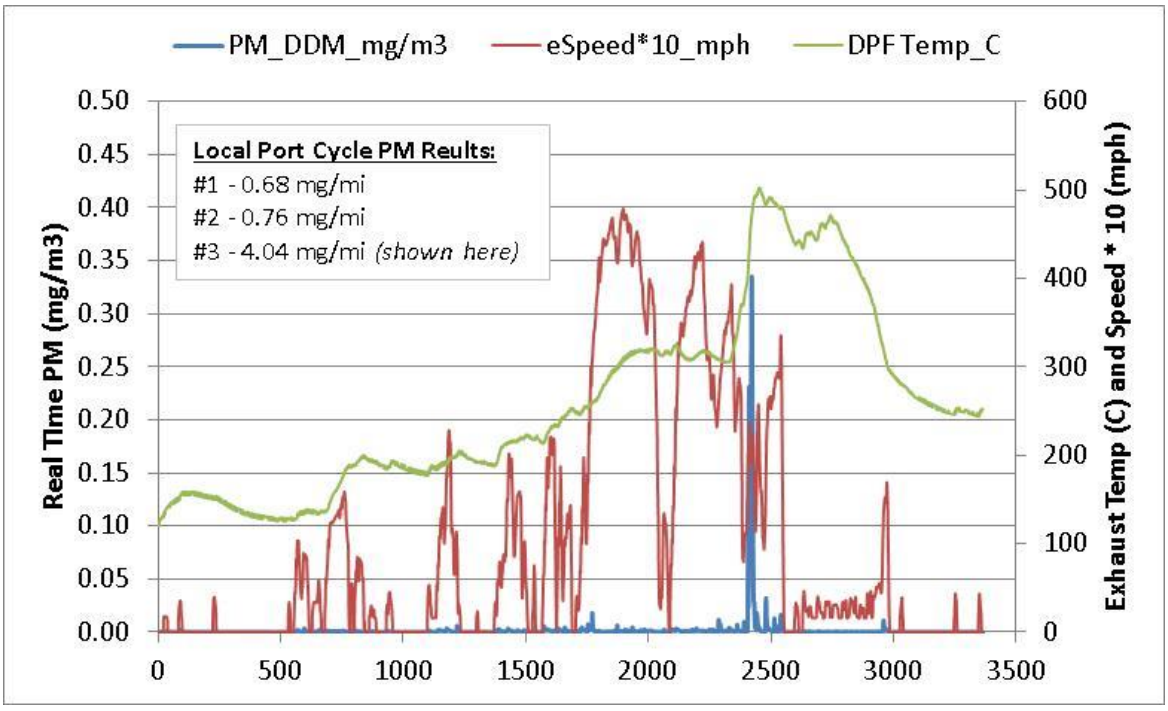


Figure 5-32: Navistar (12WZJ-B) real-time PM, vehicle speed, and DPF temp for local port cycle

In summary the low reading for the DMM suggests the actual PM mass is lower than reported by the filter mass method. Additionally, the real-time PM mass measurement method is useful for identifying test outliers and anomalies. Real-time PM is recommended with most source emissions research studies.

Ultra-fine PM emissions

In this sub section we investigate the size distribution nature of the particles. This analysis looks at particle diameters ranging from 7 nm to 200 nm, as described previously. High particle concentration at low particle diameters does not imply, necessarily, high PM mass. The calculation from particle size to mass is based on the particle diameter to the 3rd power and assumptions on density which is a strong function of particle size.

The ultra-fine PM emissions showed three unique cases, 1) the effect of cold start conditions, 2) the effect of the cycle, and 3) the difference between after treatment technologies. This section is broken down into those three categories.

Impact of cold start conditions

The cold start conditions are creating higher ultra-fine PM emissions compared to equivalent hot tests. Figure 5-33 - Figure 5-36 show the size distribution data for the UDDS hot and cold start test cycles. Figure 5-33 shows the average triplicate scans and single standard deviation error bars for the hot UDDS with the cold start UDDS from 0-200 seconds and from 200 seconds to the end of the cycle. The 0-200 seconds represents the average concentration for the first 200 seconds or the first 23 scans. Figure 5-33 is showing a very large concentration spike for the full size range from 7 to 200 nm compared to the hot UDDS. Figure 5-34 shows additional details with scan averaging from 0-100 seconds, 100-200 seconds, 0-end, and 200 – to the end.

The data in Figure 5-34 suggests the high concentration at the beginning of the UDDS cold start is occurring from the first 200 seconds since there is no change in scan from 0-100 and 100-200.

Figure 5-35 shows the data for a single particle diameter (60 nm) as a function of time to investigate the reason for the high concentration at the beginning of the scan. The results are suggesting the particles are produced at the beginning of the test and are well represented by the first 23 scans or the first 200 seconds (9 sec/scan setup time).

Figure 5-36 shows the same details as Figure 5-33, but for a different port vehicle. The results appear to be consistent where there is more PM concentration at the beginning of the cold start test. The higher concentration is also corroborated by higher PM filter masses as described previously during the $PM_{2.5}$ analysis and the real-time PM analysis sections.

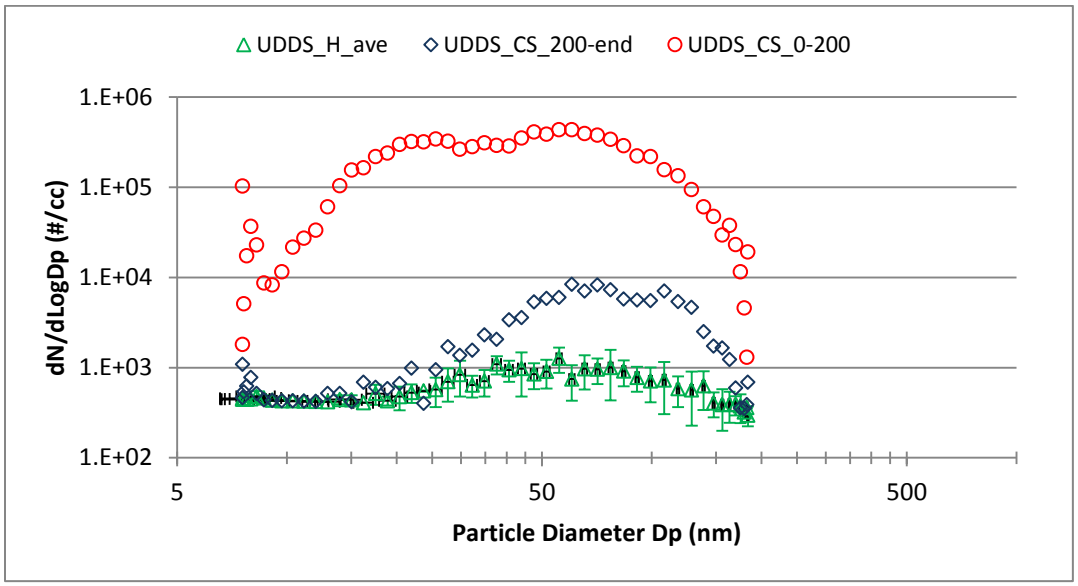


Figure 5-33Average size distributions for the V12.8 SCR equipped vehicle

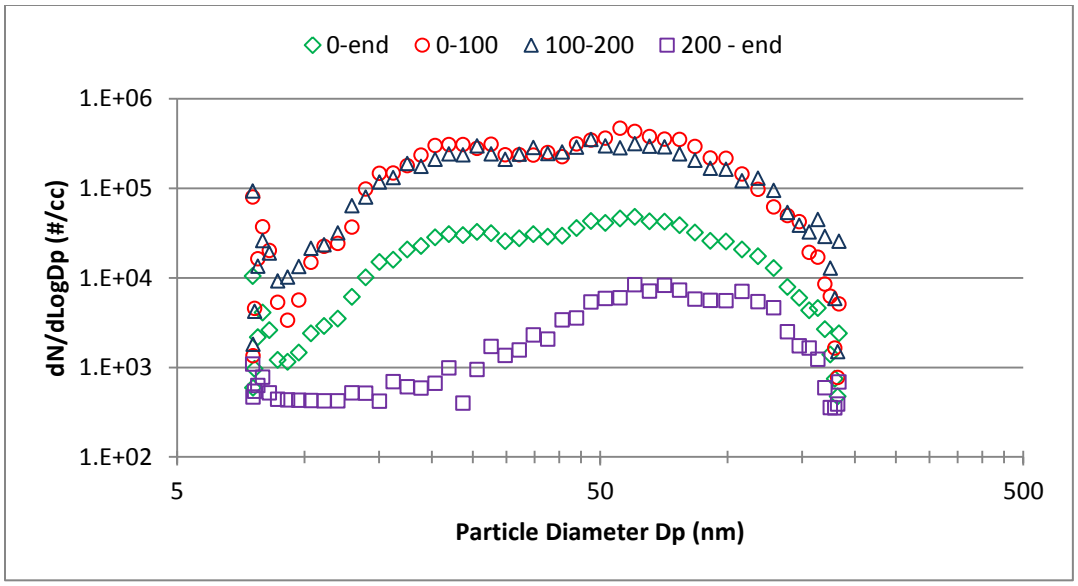


Figure 5-34 Average size distributions for the V12.8 SCR equipped vehicle selected times

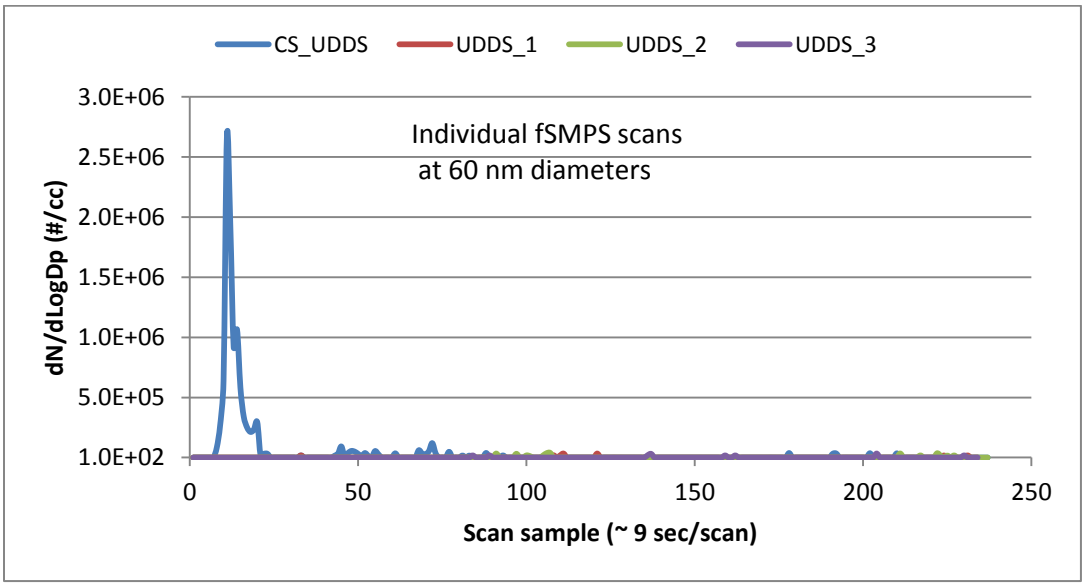


Figure 5-35 Real time scan at 60nm for the V12.8 SCR equipped vehicle

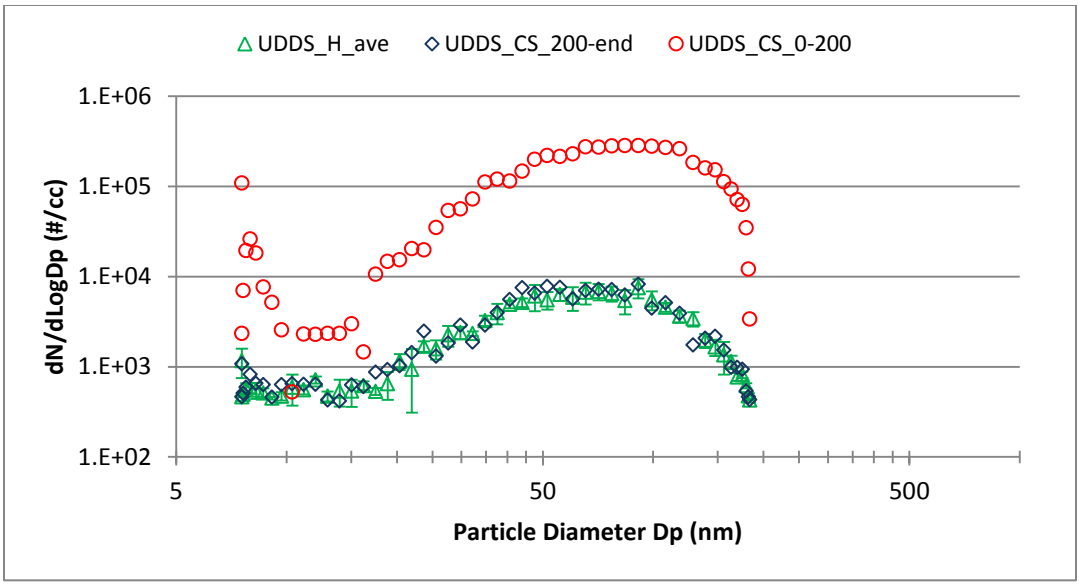


Figure 5-36 Average size distributions for the N12.3 non-SCR vehicle

Impact of cycle

The high speed regional port cycle appears to have a higher fine-particle mass impact compared to the other port cycles and the UDDS cycle. In some cases the high concentrations of the port cycle are similar to the cold start UDDS cycle, but with twice the work output of the UDDS cycle. Figure 5-37 and Figure 5-38 show the average size distributions compared between the UDDS, near dock, local, and regional. Figure 5-37 shows the comparison for an SCR equipped vehicle and Figure 5-38 shows the comparison for a non-SCR equipped vehicle. In both cases the PM concentration was much higher for the regional (Port3) cycle compared to the other cycles. For the Navistar vehicle in Figure 5-38 it appears most of the increase is for the lower size concentrations where for the Volvo vehicle in Figure 5-38 the increase was over the full size range.

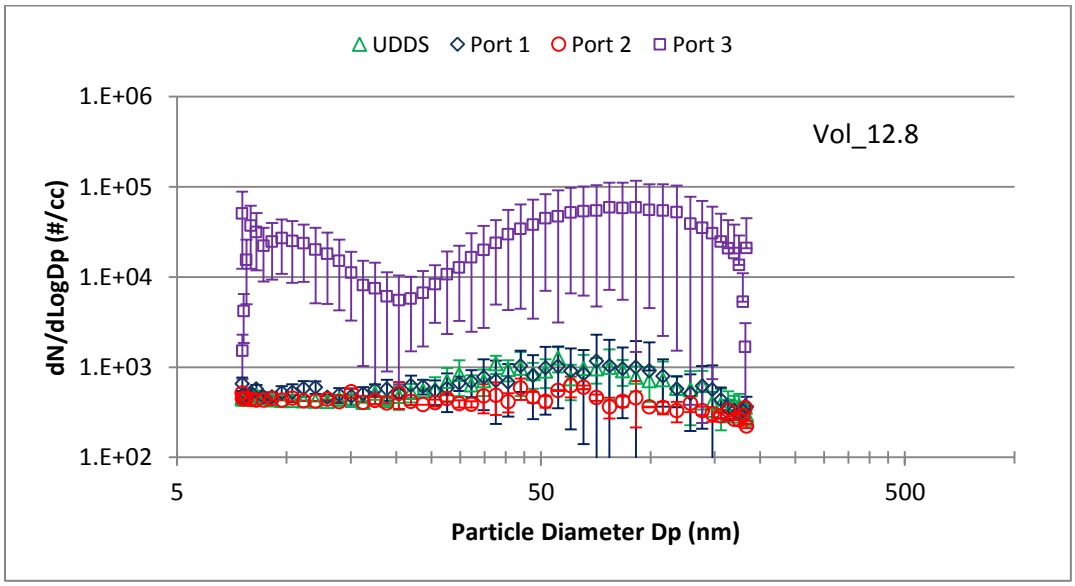


Figure 5-37 Average size distributions for the V12.8 SCR equipped vehicle by cycle

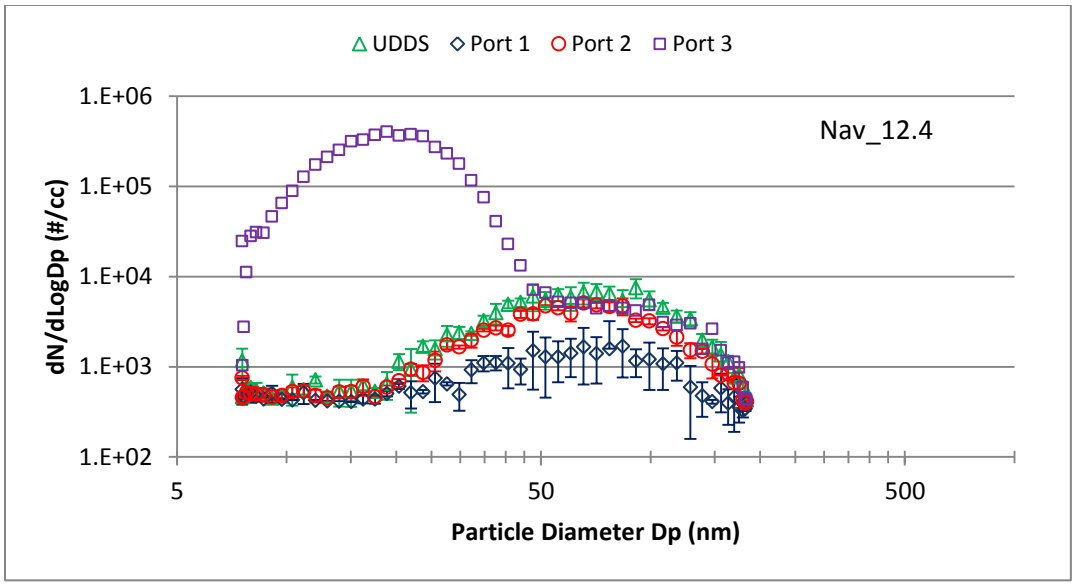


Figure 5-38 Average size distributions for the N12.3 non-SCR vehicle by cycle

Impact of after treatment technology

The basis of this research study was to consider different emission categories. The categories include with SCR, without SCR (two emission levels), high EGR, and alternative fuels. This section investigates the difference between emission control technologies. Figure 5-39 show the average size distributions for the UDDS test cycle compared between an SCR equipped truck and a non-SCR equipped truck. The two vehicles show similar size distributions where the non-SCR equipped vehicles (Nav_12.4) is slightly higher at the higher particle diameters near 50nm

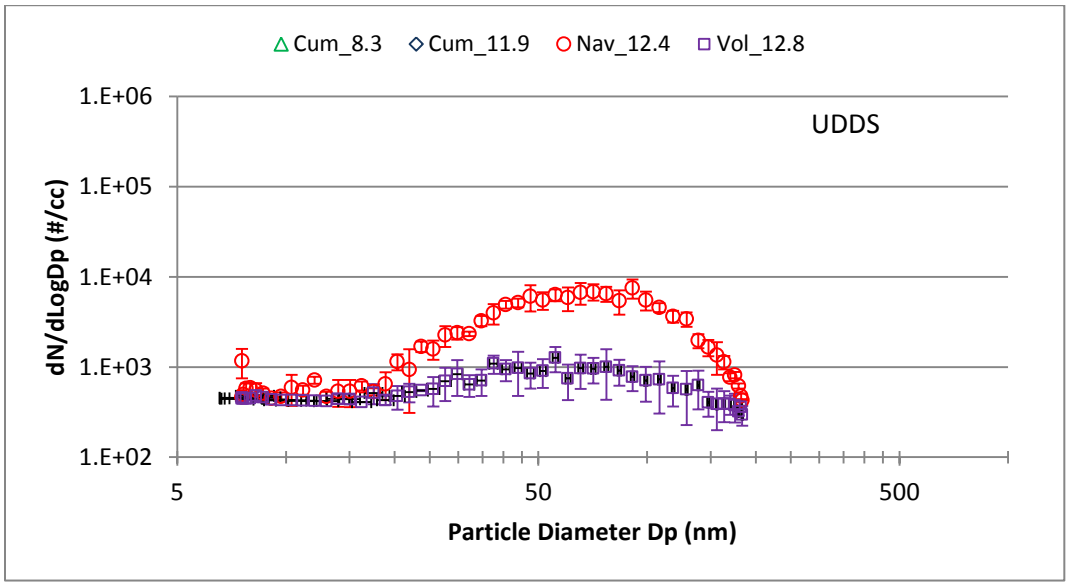


Figure 5-39 Average size distributions for various vehicles: UDDS cycle

5.7 Greenhouse Gas (N_2O, CH_4 & CO_2) Emissions and Fuel Economy

For greenhouse gases, UCR measured emissions factors of methane and carbon dioxide in real-time for all vehicles and N_2O with off-site analyses for selected vehicles. The off-site analyses were carried out by the California Air resources Board and West Virginia University. Results showed the measured values were close to ambient levels, as expected for diesel vehicles. Literature indicates N_2O is observed when vehicles rely on three way catalyst and UCR did not have any included in their fleet of test vehicles.

5.7.1 Emissions of nitrous oxide (N_2O)

N_2O emissions were measured by the IR methods described earlier and found to be near the detection limits. A literature review showed that Huai et alia[11] only found nitrous oxide when a three way catalyst was warming. Thus we only expected N_2O for the LPG truck with the three way catalyst. Unfortunately the LPG port vehicle equipped with a TWC did not have N_20 analysis available at the time of testing so no results for vehicles with TWC are reported in this section.

The N_2O measurements were very close to the ambient concentrations were negative numbers were reported. The reason for negative numbers is based on the correction of the ambient measured concentration exceeding the sample measurement, as described in a later section. It is expected that many of the measurements are near the detection limits of the N_2O measuring method. See Section 8.4.3 for analysis and summary of N_20 measurements and detection limits.

The general observations of the N_2O emissions from the vehicles tested can be summarized as:

- N_2O Analysis was done offsite when facilities were available. As such, not all vehicles or cycles were tested for N_2O. Only selected vehicles were tested for N_2O analysis.
- During the refuse and school bus testing there were no facilities for N_2O analysis thus they were not performed. Similar results are expected for all the vehicle categories.
- More than half (64%) of the measured toxic emissions were below the defined threshold (0.4 ppm), the average ambient background concentration plus one standard deviation.
- Only the SCR equipped vehicles showed signs of N_2O emissions not the non-SCR equipped vehicles.
- The cold start UDDS did not show higher integrated N_2O emissions compared with hot start UDDS tests (with or with/out SCR). It is not clear from the testing if higher N_20 emissions were created for a short duration at the cold start of the cold test cycles. Additional real time N_2O data would be necessary to evaluate the first 100 seconds of the cold start UDDS N_2O emissions.
- Of the values greater than the threshold, the average vehicle sample concentration was 1.06 ppm (only 2.6 times the threshold) and the single standard deviation was 0.44 ppm.

[11] T. Huai, Durbin, T.D., J.W. Miller, and J.M. Norbeck, *Estimates of the Emission Rates of Nitrous Oxide from Light-Duty Vehicles using Different Chassis Dynamometer Test Cycles*. Atmospheric Environment, vol. 38, 6621-6629 (2004)

- The N_2O emission rate in mg/mi for port vehicles with higher than threshold concentrations ranged from 1.5 mg/mi to 17 mg/mi where the highest concentrations were for the shorter test cycle.
- N_2O emissions appear to be below or near detection limit for diesel and propane vehicles appear operated on the UDDS and port related test cycles.

5.7.2 Emissions of methane (CH_4)

Vehicles emit methane, a greenhouse gas, with a global warming potential (GWP) over 20 years of 72. This factor means that methane will trap 72 times more heat than an equal mass of carbon dioxide over the next 20 years. There are factors for 100 and 500 years but the 20 year factor is used in this analysis. From results of this project, the CH_4 contribution to greenhouse gases with diesel trucks can be ignored given that the emissions rate for CO_2 was about >2,000 gram/mile and that of CH4 was ~0.02 grams/mile. Thus emissions of CO_2 predominate for the greenhouse calculation, even after adjusting the methane rate by a factor of 72.

The CH_4 contribution was considered with the LPG truck. In this case, the CO_2 emissions were about 1,500 grams/mile and that of methane was ~1.5 grams/mile. Thus for this case, multiplying by 72, the contribution to the greenhouse gases will be ~105/1,500 = 7% so significant and more important to consider.

5.7.3 CO_2 and Fuel Economy emissions

Emissions of CO_2 for the goods movement vehicles are provided in Table 5-10 for the different test cycles and ranges from 1,489 grams/mile with the LPG fuel to 3,904 grams/mile.

Table 5-10: CO_2 Emissions for the Goods Movement Vehicles.

Vehicle			CO_2 Emission Factor (g/mi)				
Category	Engine	MY	Near Dock	Local	Regional	UDDS	UDDS-CS
VIII	C8.3p	2010	2958	2874	2170	2672	2671
VIII	C11.9	2011	3904	3795	2135	3089	3117
VIII	V12.8	2011	2578	2473	1953	2426	3019
VII	N12.4b	2011	2580	2656	1690	2565	2518
VII	N12.4c	2011	2466	2323	1922	2401	2611
VI	GM8.1p	2009	1742	2031	1489	1709	1577
IV	N12.4a	2009	3064	2943	2274	2868	3032
IV	D14a	2008	2640	2525	1850	2373	2379
IV	D14b	2008	2696	2426	1821	2506	2455

Fuel economy for the goods movement vehicles in different driving cycles are provided in Figure 5-40 to Figure 5-44. Looking first at the hot-UDDS cycle, the fuel economy ranged from 3.31 to 4.25 miles per gallon with an average 3.48 miles/gallon and coefficient of variation of 1%. Thus the values were statistically the same, even though some paired values did show a significant difference. While we expected the vehicles with SCR to have advanced fuel injection, more NOx and better fuel economy, the results did not show that finding.

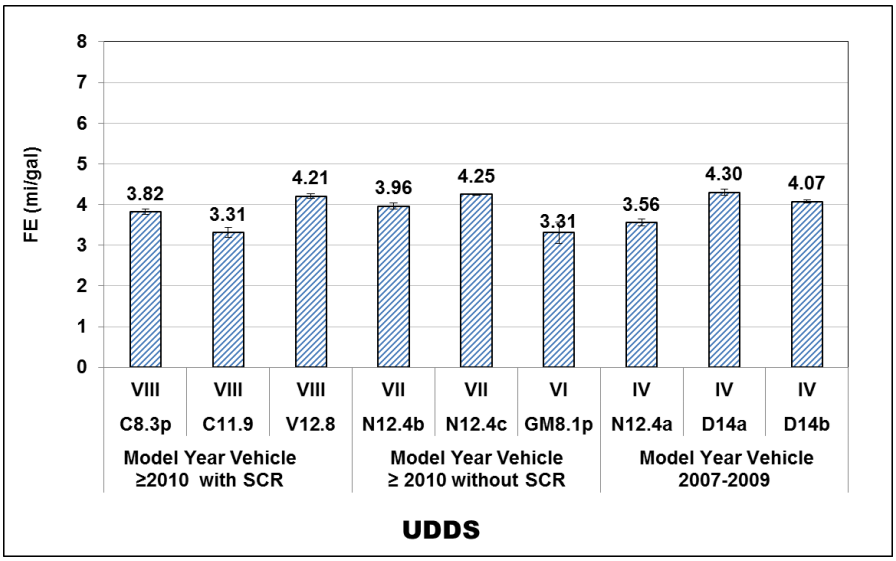


Figure 5-40: Fuel Economy in miles/gallon of Fuel for the UDDS Cycle.

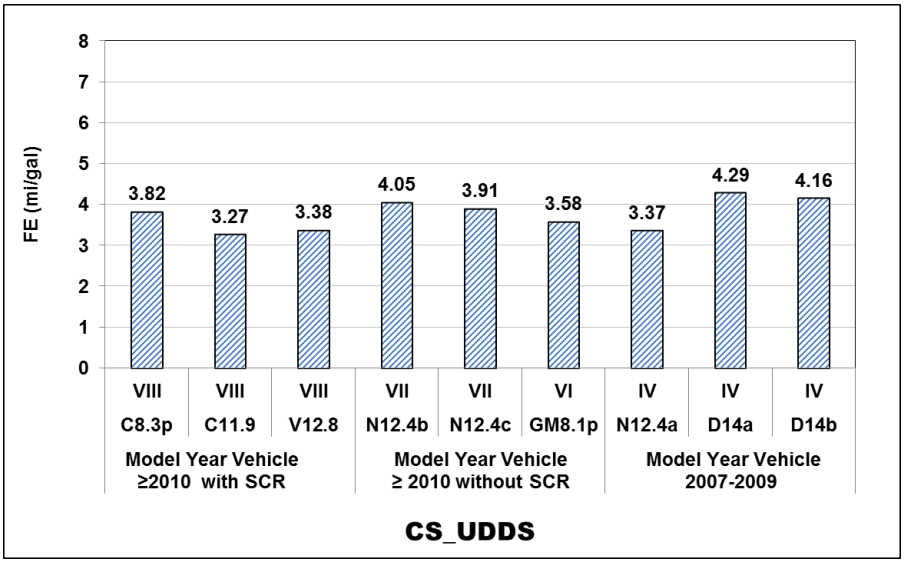


Figure 5-41: Fuel Economy in miles/gallon of Fuel for Cold start UDDS cycle.

[1] No error bars for the cold start tests because on only one test was performed

Reviewing the fuel economy for the three port cycles representing in-use activities shows that the fuel economy ranged from 2.62 to 6.02 miles per gallon. The lowest and highest fuel economy values were found for the engine with the lowest NOx emissions. Temperature appears to play a role in that finding as in the regional cycle the truck achieved the lowest NOx emissions with comparable fuel economy. Other trends showed that the lowest fuel economy was during the Near Dock driving schedule which is not surprising given the low power and creep cycles. The 2010+ Navistar vehicles without SCR had the highest fuel economy but as mentioned earlier, they failed to meet the NOx standard and were withdrawn from the market.

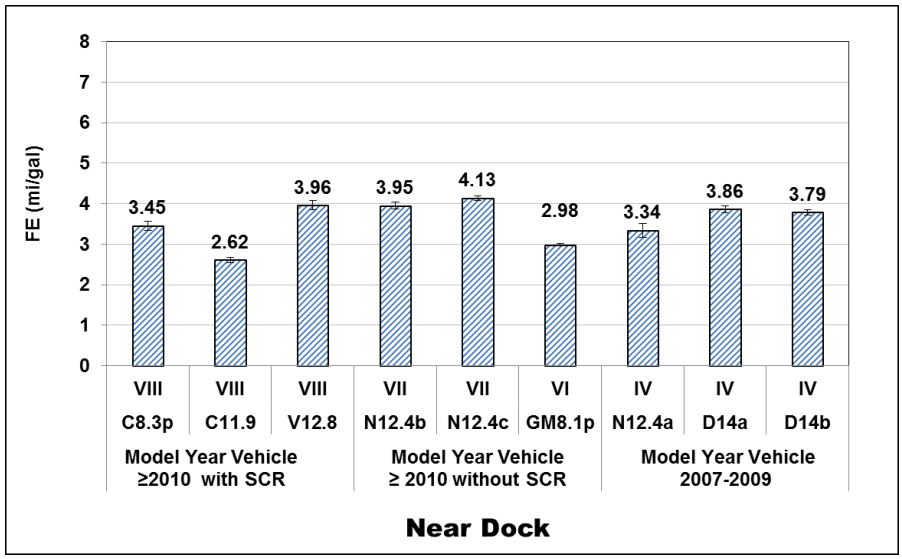


Figure 5-42: Fuel Economy in miles/gallon of Fuel for the Near Port Cycle

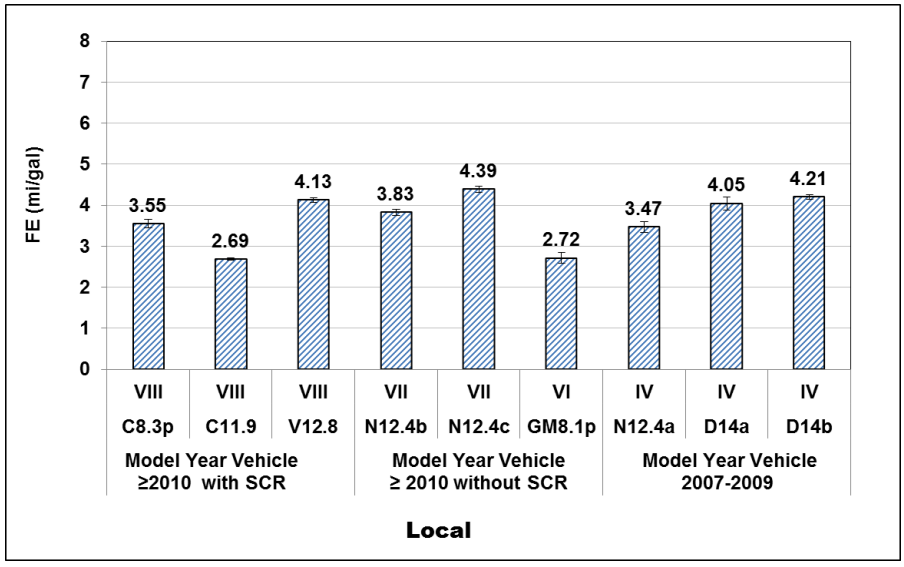


Figure 5-43: Fuel Economy in miles/gallon of Fuel for the Local Port Cycle

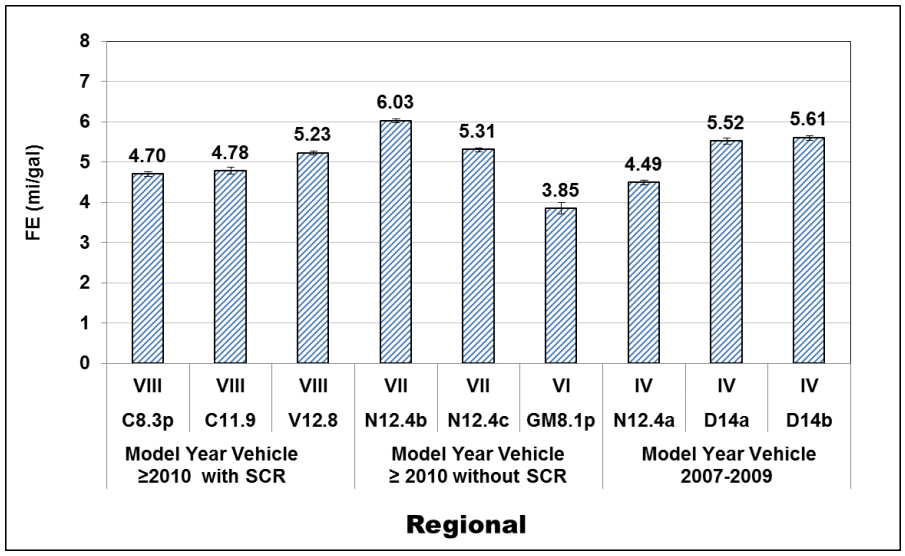


Figure 5-44: Fuel Economy in miles/gallon of Fuel for the Regional Port Cycle

6 Results and Discussion for Refuse Haulers

This section covers the emissions for refuse vehicles that were tested on the UDDS and the AQMD refuse truck cycles. Most emissions are based on grams per mile for inventory purposes and the UDDS cycle values are in grams/bhp-hr so as to compare with the certification values.

6.1 Test Trucks

Four trucks used as refuse haulers were tested on a number of chassis cycles. Selected information for the trucks is identified in Table 1-1. All of these vehicles were a challenge to find within the AQMD District as most trucks in the District use natural gas. Some were shipped in from Northern California where the air conforms to federal standards and diesel vehicles are still used.

6.2 Test Conditions

Vehicles were tested on the UDDS cycle and on the AQMD refuse hauler cycle as that cycle more closely represented in-use activities for a refuse hauler. The load for the refuse haulers was set at 56,000 lb. load as that value represents the typical load of a refuse hauler in the SCAQMD District. Commercially available CARB #2 diesel fuel was used rather than a certification fuel. Both load and fuel matched in-use conditions. The emission values represent the average of triplicate runs and the graphs show the confidence limits to one standard deviation.

6.3 NOx Emissions from the UDDS Cycle

As mentioned earlier, the brake specific emissions values from the UDDS chassis dyno test are often compared with the values measured in the heavy-duty FTP certification test on an engine dyno. This comparison provides some indication that the selected vehicle is representative of the desired FEL and technology. This section focuses on NOx emissions given the interest in the original RFP.

6.3.1 Brake-specific emissions for the UDDS Cycle

Figure 6-1 shows the UDDS values for NOx ranged from 0.13 to 2.0 g/bhp-hr versus certification standards of 0.2 and 1.2 g/bhp-h. The Value for one vehicle is on the high side; perhaps the manufacturer was using credits for that family of engines, but we do not know. Category VII with SCR had the lowest NOx emissions. The UDDS values ranged from 0.13 to 0.17 and were close to the certification values.

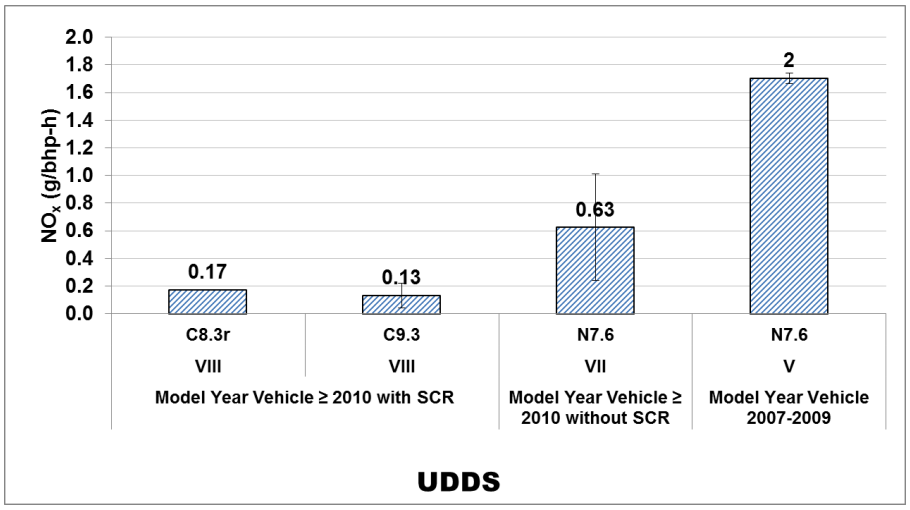


Figure 6-1 Brake Specific NOx Emissions for Hot & Cold UDDS Cycles

6.3.2 Emissions in g/mile for the UDDS cycle

Results were also analyzed and calculated on the basis of the emissions being expressed in grams per mile, the figure needed for calculating the inventory. These data are shown in Figure 6-2 and Figure 5-6.

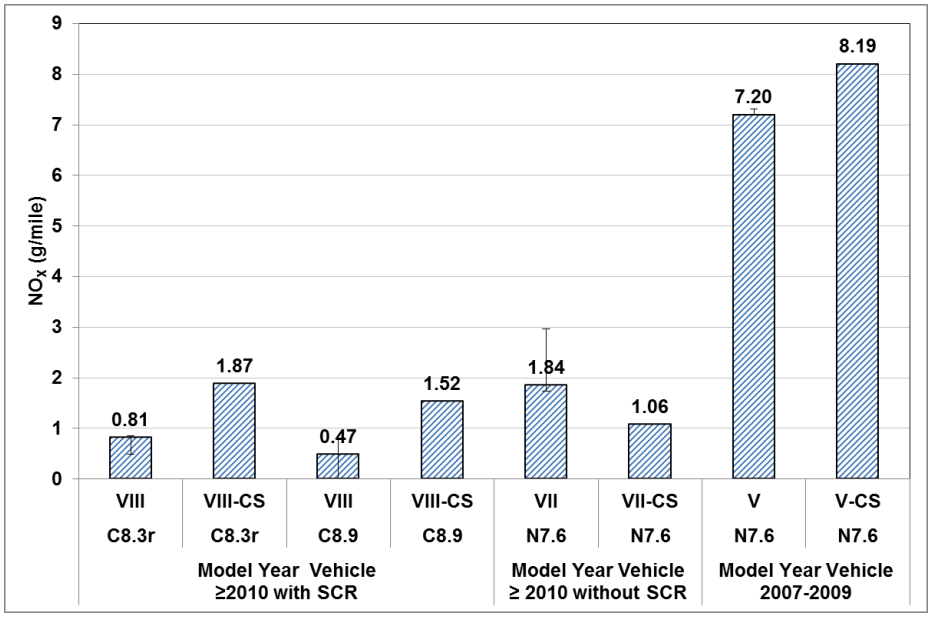


Figure 6-2: NO_x Emission Factors for Hot & Cold UDDS Cycles (g/mile)

[1] No error bars for the cold start tests because on only one test was performed

Many have asked whether there is a single factor to convert g/bhp-hr to g/mile. The answer to this question is presented in Table 6-1. The average factor is 3.73 with a coefficient of variation of 21%. This value is closer to the traditional value of 3.5. Also the coefficient of variation near 21% indicated the ratio is more dependent on technology than found with the goods movement vehicles.

Table 6-1 Relationship Between g/mile & g/bhp-hr for the Hot UDDS

Units				
g/mile	7.2	1.84	0.47	0.81
g/bkhp-hr	2	0.63	0.13	0.17
ratio	3.60	2.92	3.62	4.76

6.3.3 Percentage of NOx emissions as NO_2

NO_2 emissions are a health concern and values for the refuse trucks are presented in Table 6-2. These tables show the percentage of the total NO_x that is NO_2. On the UDDS cycle, values range from 18% to 53% and for the AQMD refuse cycle the values range from 23 to 69%. One finding is the AQMD refuse cycle increased the percentage of NO_2 significantly for the vehicles with an SCR technology. This is an important finding that should be further investigated. Not surprising the NO_2 percentage was high for the vehicles with the SCR technology. Similar to the observation with the goods movement vehicles, levels of NO_2 are high as compared with the ARB retrofit rule of 20% over baseline.

Table 6-2: Fraction of NOx (g/mile) as NO_2 for the Refuse Trucks

Vehicle			AQMD RTC			UDDS			CS_UDDS RTC		
Category	Engine	MY	NO_2	NO_x	$\%NO_2$	NO_2	NO_x	$\%NO_2$	NO_2	NO_x	$\%NO_2$
VIII	C8.3r	2012	0.67	1.22	55%	0.21	0.81	26%	0.41	1.87	22%
VIII	C9.3	2011	0.35	0.51	69%	0.08	0.47	18%	0.41	1.52	27%
VII	N7.6	2011	0.75	3.28	23%	0.51	1.84	27%	0.35	1.06	33%
V	N7.6	2008	3.63	6.31	58%	3.83	7.20	53%	3.81	8.19	47%

6.4 Regulated Emissions from the AQMD Cycle in g/mile

6.4.1 NO_x emissions for the UDDS (grams/mile)

The NO_x emission results in grams/mile for the refuse trucks are presented in for the Refuse Truck cycle, the UDDS, and the CS-UDDS, respectively. The refuse trucks show a clear trend of NO_x emissions reductions with advancing technology. For the refuse truck cycle, the 2010+ vehicles with SCR show significant reductions relative to both the 2010+ refuse truck without SCR and the 2007-2009 vehicle. For the UDDS cycle, the 2010+ vehicles with SCR also show reductions relative to both the 2010+ refuse truck without SCR and the 2007-2009 vehicle, although the differences between the 2010+ refuse trucks with and without SCR was smaller than for the refuse truck cycle. For the CS-UDDS, the 2010+ vehicles showed significant reductions relative to the 2007-2009 vehicles, but the 2010+ vehicles with SCR actually showed higher emissions than those for the 2010+ vehicles without SCR. In comparing emissions between the refuse trucks and the goods movement vehicles, the trends depended on the specific vehicle and cycle, with the refuse trucks showing lower emissions for come cycle vehicle combinations and higher emissions for others.

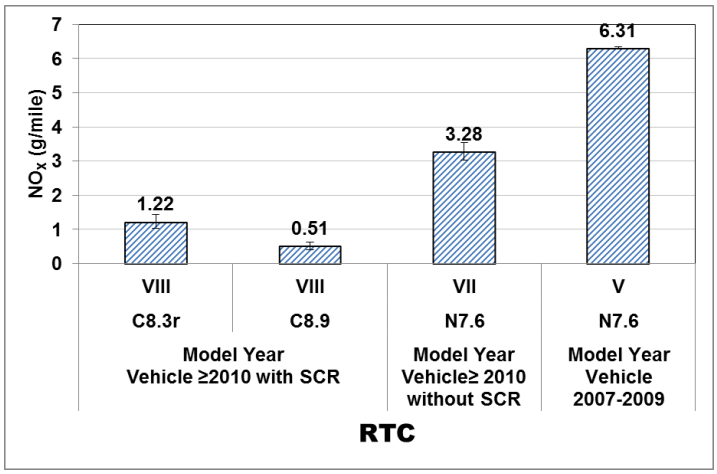


Figure 6-3: NOx Emission Factors in g/mile for AQMD Refuse Truck Cycle

6.4.2 PM emissions

The PM emission results for the refuse trucks are presented Figure 6-4 and Figure 6-5 for the, the UDDS, and the AQMD Refuse Truck cycle. The PM emissions were relatively low and were around 2 mg/mi or less for most of the hot start vehicle/cycle combinations, with only the 2010+ vehicle with SCR being slightly above 2 mg/mi. The emissions for the refuse trucks were slightly higher for the CS-CBD for each of the vehicles, and the 2010+ C8.3 vehicle showed a larger increase to 18.4 mg/mi.

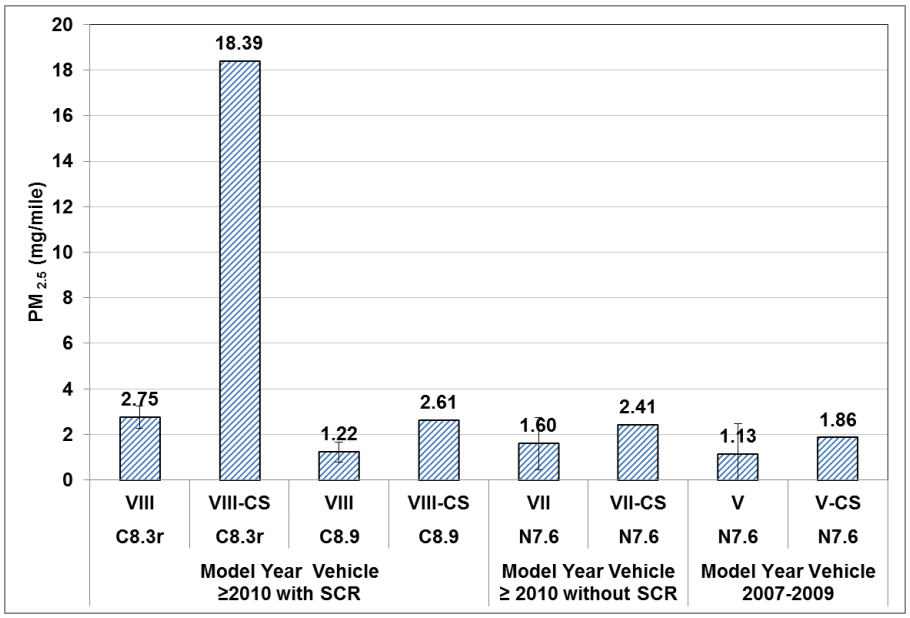


Figure 6-4 Emission factors for PM UDDS and cold start UDDS cycles (mg/mile)

[1] No error bars for the cold start tests because on only one test was performed

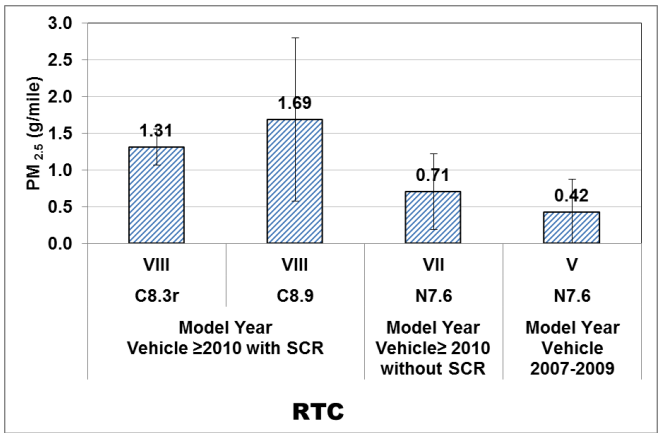


Figure 6-5: Emission factors for PM AQMD Refuse Truck Cycle (g/mile)

6.4.3 THC/NMHC/CH_4 and CO emissions

Table 6-3 through Table 6-5 show the emission factors for THC, CH4, NMHC and CO in g/mile for the hot/cold UDDS Cycle and the AQMD Refuse Truck Cycle. The emission factors for THC, CH_4, and NMHC were low for nearly all vehicle/cycle combinations. THC emissions were at or below 0.45 g/mi for nearly all vehicle/cycle combinations. NMHC emissions were at or below 0.30 g/mi for nearly all vehicle/cycle combinations. CH_4 emissions were at or below 0.20 g/mi for nearly all vehicle/cycle combinations. The 2010+ N7.6 refuse truck also had a slightly higher emissions ranging from 1.11 to 1.13 g/mi for THC, from 0.70 to 0.74 g/mi for NMHC, and from 0.45 to 0.48 g/mi for CH_4. Cold start emissions were also low for most vehicle/cycle combinations, except the 2010+ N7.6 refuse truck showed somewhat higher cold start THC, NMHC, and CH_4 emissions.

Table 6-3: THC, CH4, NMHC, and CO Emissions for UDDS Cycle for Refuse Trucks (g/mile)

Vehicle			Emission Factor (g/mi)			
Category	Engine	MY	THC	CH4	NMHC	CO
VIII	C8.3r	2012	-0.03	0.01	-0.05	-0.23
VIII	C9.3	2011	-0.03	0.01	-0.04	-0.13
VII	N7.6	2011	1.13	0.45	0.74	1.86
V	N7.6	2008	0.00	0.01	-0.01	0.06

Table 6-4: THC, CH4, NMHC, and CO Emissions for the Cold Start UDDS Cycle (g/mile)

		Vehicle		Emission Factor (g/mi)			
Cycle	Category	Engine	MY	THC	CH4	NMHC	CO
CS-RTC	VIII	C8.3r	2012	0.28	0.02	0.27	-0.11
CS-RTC	VIII	C9.3	2011	0.00	0.01	-0.01	-0.19
CS-RTC	VII	N7.6	2011	0.36	0.37	0.04	1.90
CS-RTC	V	N7.6	2008	0.01	0.01	0.00	0.00

Table 6-5: THC, CH4, NMHC, and CO Emissions for AQMD Refuse Truck Cycle (g/mile)

Vehicle			Emission Factor (g/mi)			
Category	Engine	MY	THC	CH4	NMHC	CO
VIII	C8.3r	2012	-0.06	0.02	-0.08	-0.13
VIII	C9.3	2011	-0.01	0.01	-0.02	-0.25
VII	N7.6	2011	1.11	0.48	0.70	3.36
V	N7.6	2008	0.01	0.02	-0.01	-0.10

6.5 Non-regulated Gaseous Emissions

6.5.1 NH_3 emissions

The NH_3 emission results for the refuse trucks are presented in

Figure 6-7 and Figure 6-6 for the Refuse Truck. NH_3 emissions for all of the refuse trucks were in the range of 10 to 50 mg/mi for most of the cycle combinations, with the exception of the 2010+ C8.3 vehicle for the CS-UDDS being slightly higher at 120 mg/mi. This is roughly the same range seen for the good movement vehicles.

Table 8-26 shows of the 54 diesel tests conducted, only 2 vehicles were 5 times the LDL (i.e. greater than 5ppm), and 26 tests were above 2 time the LDL (2 ppm), see Section 8.4.4. Of the 2 tests above 5*LDL, both were for a cold start SCR equipped diesel vehicle. For the 26 tests above 2* LDL these were both SCR and non-SCR equipped vehicles. It is not expected that a non-SCR equipped vehicle had more NH_3 emissions than an SCR equipped vehicle. Five of seven tests for the propane vehicle also had NH_3 greater than 5 ppm and 2 were over 50 ppm suggesting very high relative NH_3 emissions for the propane vehicles.

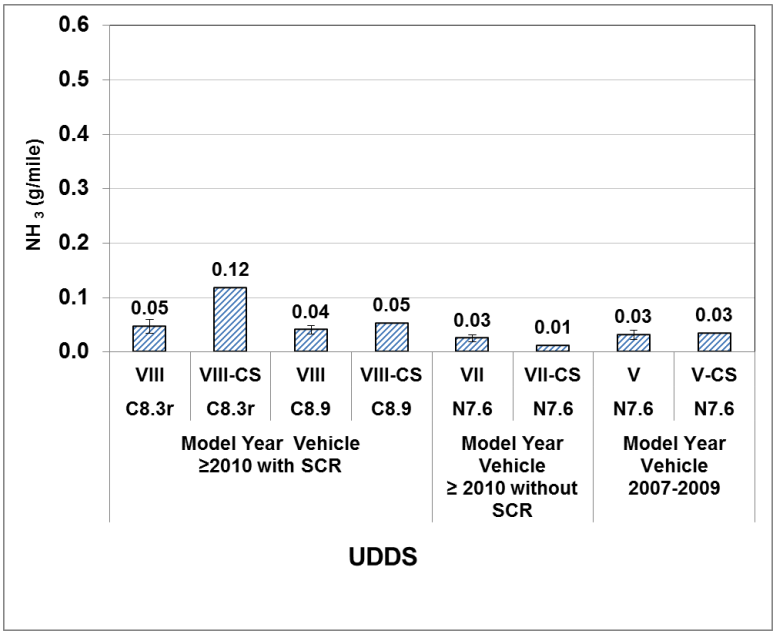


Figure 6-6: Emission of NH_3 in the cold/hot UDDS Cycle (g/mile)[1]

[1] NH_3 scale is based on 10 ppm raw exhaust concentration

[2] No error bars for the cold start tests because on only one test was performed

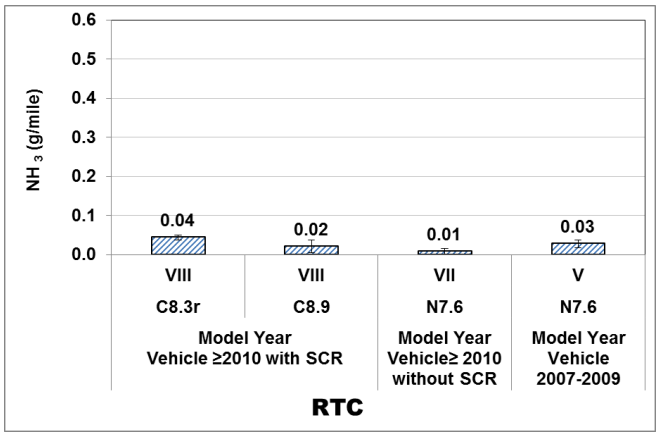


Figure 6-7: Emission of NH_3 in the AQMD Refuse Truck Cycle (g/mile)[1]

6.5.2 Selected Toxic Emissions (1,3butadience & BTEX)

The 1,3 butadiene, benzene, and toluene results for the refuse haulers are shown in Figure 6-8 and Figure 6-9. All values are low as expected based on the ACES study and that the exhaust passes over a DOC catalyst containing noble metals. Only the 2010+ N7.6 showed measureable levels of these species for both cycles. The 2007-2009 N7.6 vehicle also showed measureable levels for 1,3 butadiene and toluene for the UDDS. These findings match the NMHC results.

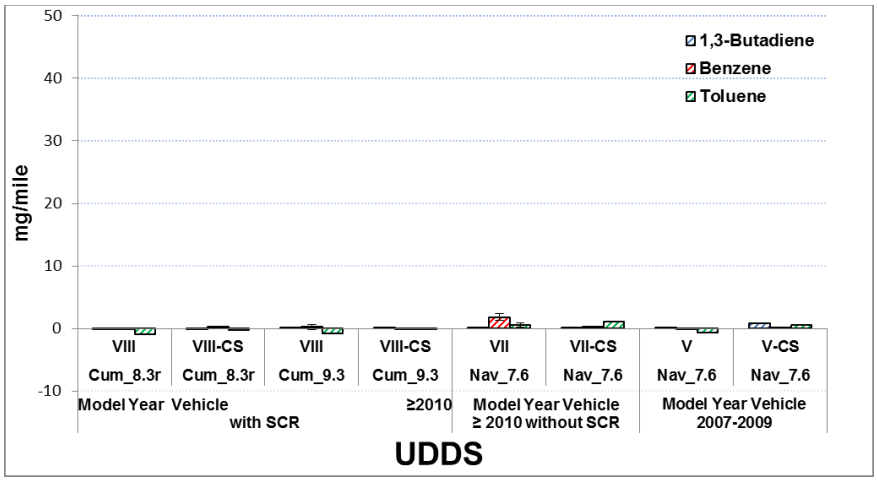


Figure 6-8 Emissions of Selected Toxics in mg/mile for UDDS Cycle

[1] No error bars for the cold start tests because on only one test was performed

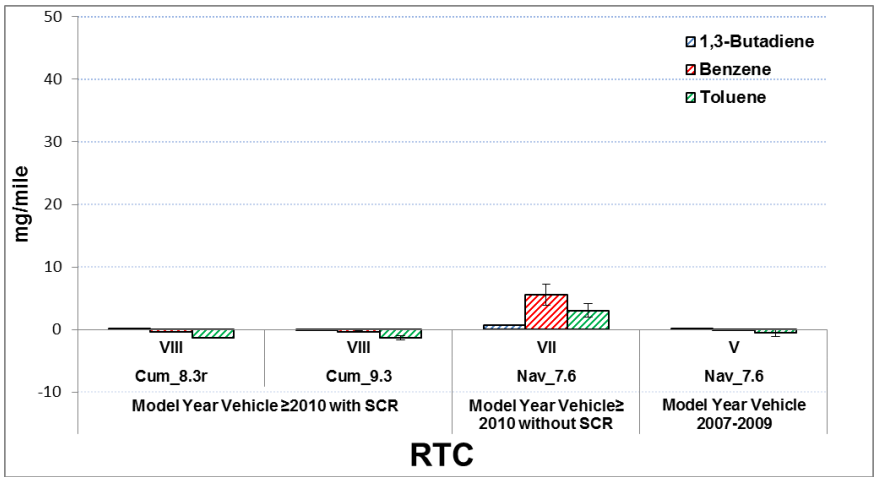


Figure 6-9 Emissions of Selected Toxics in mg/mile for AQMD Refuse Cycle

6.5.3 Selected Toxic Emissions (carbonyls & ketones)

The formaldehyde, acetaldehyde, and acetone carbonyl results for the refuse haulers are shown in Figure 6-10 and Figure 6-11. Emissions are very low, as expected. Formaldehyde emissions were the highest of the carbonyl species, which were measureable for most vehicles on both cycles. The highest formaldehyde emissions were seen for the 2007-2009 N7.6 for both cycles. Acetaldehyde emissions were measureable for several vehicles for both cycles. Acetone emissions were not measureable for any of the vehicle/cycle combinations.

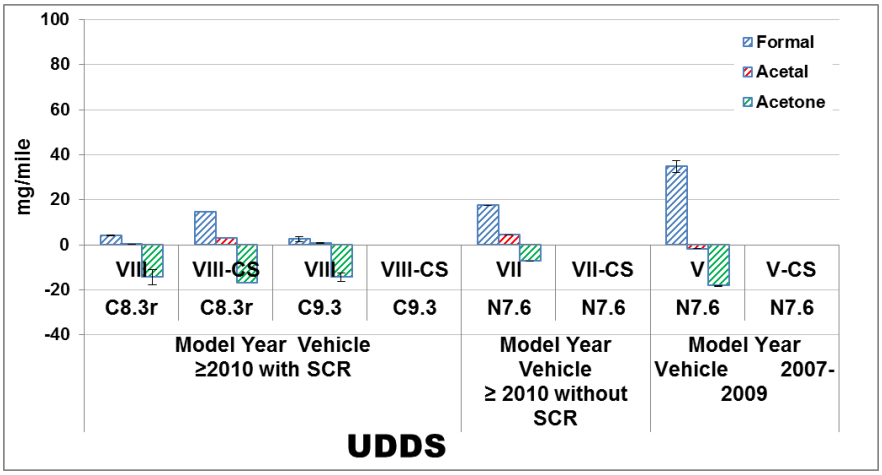


Figure 6-10 Emissions of Carbonyls & Ketones in mg/mile for UDDS Cycle

[1] No error bars for the cold start tests because on only one test was performed

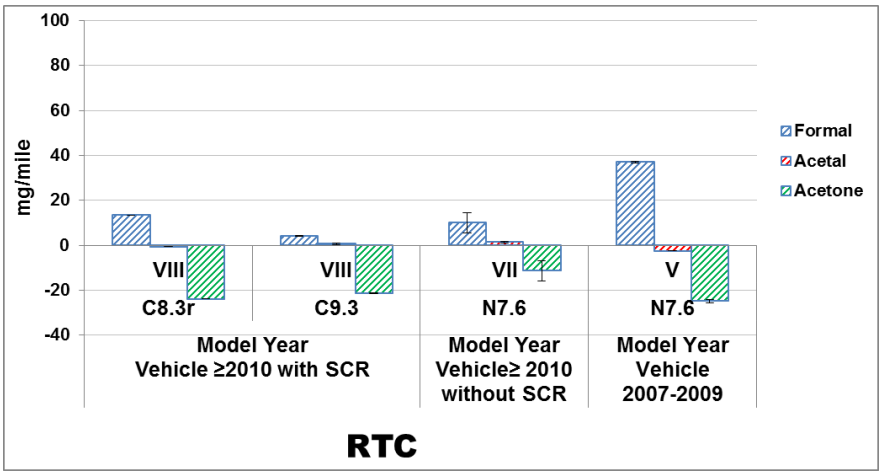


Figure 6-11 Emissions of Carbonyls & Ketones in mg/mile for AQMD Refuse Cycle

6.6 Non-regulated PM Emissions

6.6.1 Fractionation of the PM mass into OC and EC

Fractioning the PM into elemental and organic carbon was carried out by analysis of the quartz filter media collected at the test site. Results for the refuse haulers are shown in Figure 6-12 and Figure 6-13. For the Refuse Truck Cycle, the elemental and organic carbon emissions were essentially at the background levels. See Section 8.4.4 for a discussion on EC and OC detection limits. For the UDDS only the cold start emissions for the C8.3 showed organic carbon emissions measurably above the background levels, consistent with the higher $PM_{2.5}$ emissions for that vehicle/cycle combination. Elemental carbon emissions were not measurably above the background levels for the UDDS, as expected due to the high filter efficiency of the DPF.

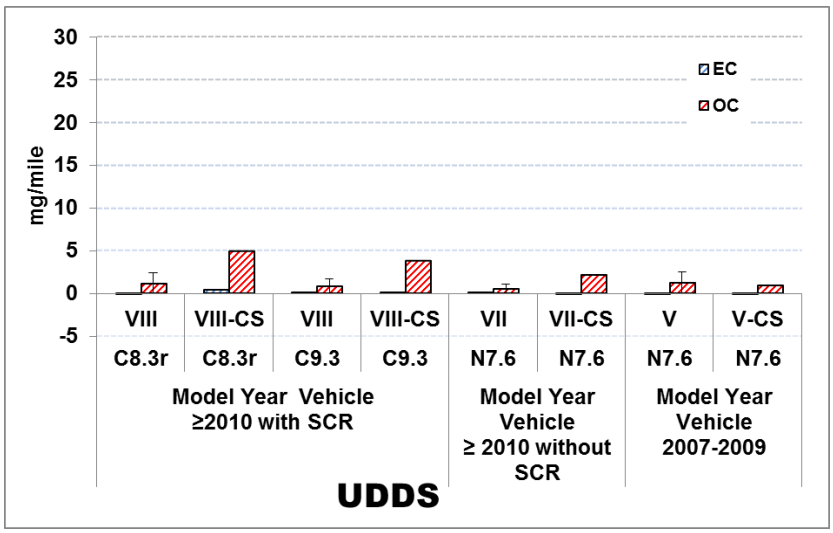


Figure 6-12 Emissions in grams/mile for the PM as OC & EC for the UDDS Cycle

[1] Error bars for the cold start tests were available for this vehicle because multiple tests were performed

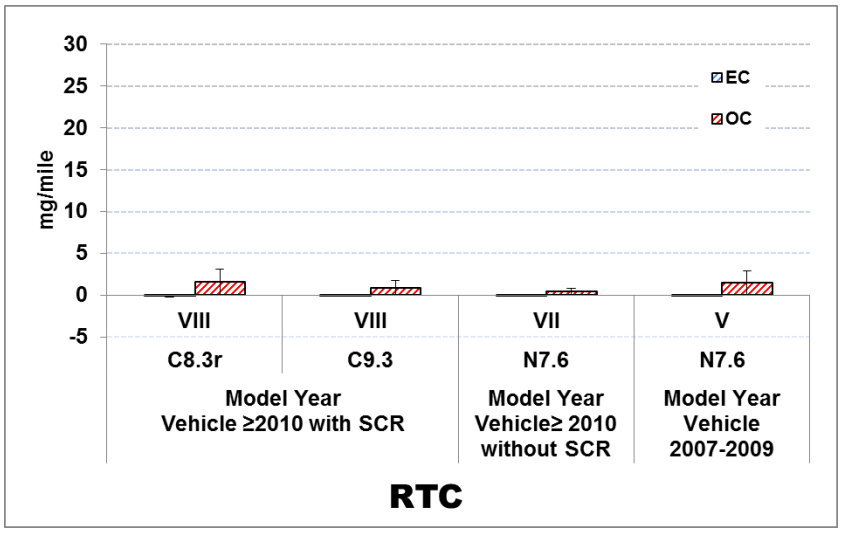


Figure 6-13 Emissions in grams/mile for the PM as OC & EC for the Refuse Cycle

6.6.2 Measurement of the real-time and ultrafine PM emissions

As described previously, The DMM was used to characterize the real time PM mass concentration and the f-SMPS was used for the ultrafine PM emissions characterization.

Real-time PM mass DMM

As presented earlier the PM mass of the gravimetric method were very low and typically around 2.1 mg/mi or 0.5 gm/bhp-hr for all the refuse vehicles tested. The average PM mass from the DMM measurement method averaged 0.3 mg/mi and 0.1 mg/bhp-hr for the same vehicles. The low PM mass emission factors were near the detection limits of the measurement method as discussed previously in Section 5.6.2.

Figure 6-14 shows the DMM PM mass concentration on a second by second basis for the Cummins 8.3 liter SCR equipped refuse hauler on the cold start UDDS cycle. At 600 seconds, the beginning of the large hill, there was a large PM spike, as denoted with the blue line, which was not present in the hot UDDS tests. The total PM from the gravimetric method was 18.4 mg/mi for the CS UDDS and between 3.2 to 2.2 mg/mi for the hot UDDS's. The cold start UDDS PM was 6 times higher than the hot UDDS cycles. ThisAfter closer investigation it appears that a passive regeneration occurred as can be seen by the high exhaust temperature, but no regeneration illumination lamp from the engine on this test.

Figure 5-32 shows the DMM PM mass concentration on a second by second basis for the 4.04 mg/mi test case. At 2500 seconds there was a large PM spike, as denoted with the blue line, which was not present in the other two tests. After closer investigation it appears that a passive regeneration occurred as can be seen by the high exhaust temperature, but no regeneration illumination lamp from the engine on this test.

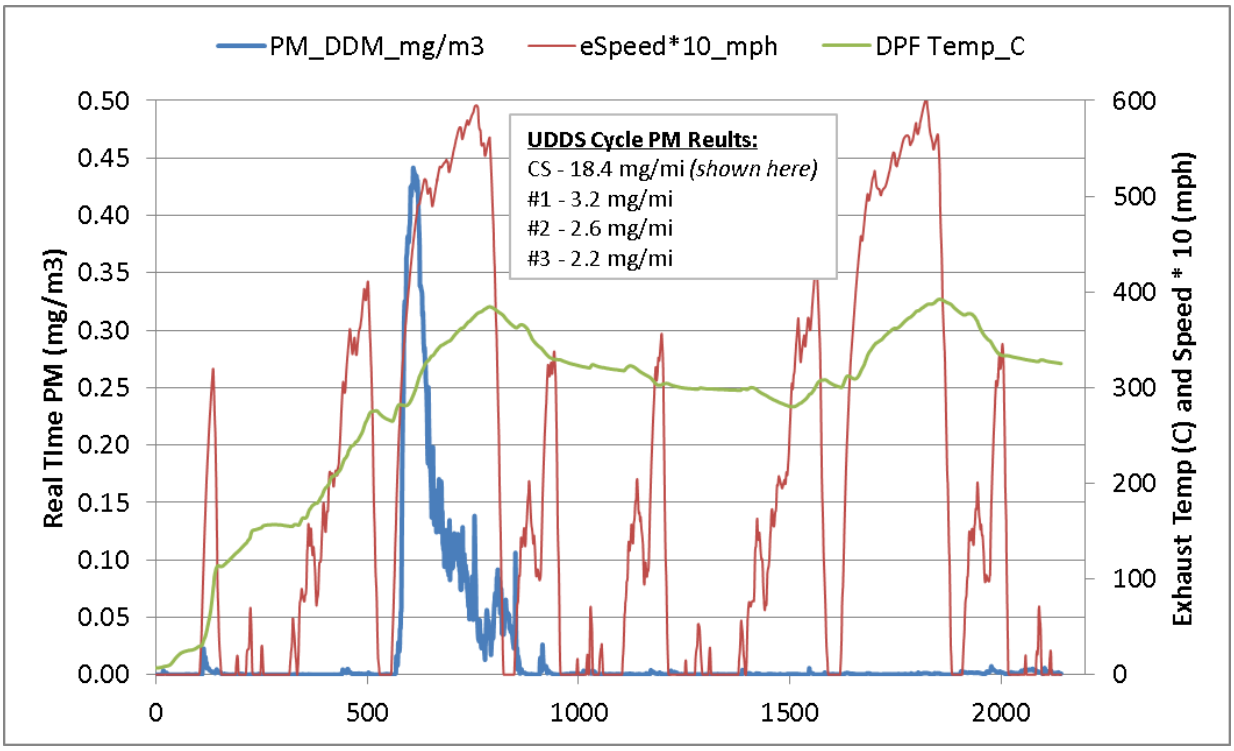


Figure 6-14 Refuse vehicle real-time PM emissions for the cold start UDDS cycle

Ultra-fine PM emissions

The cold start UDDS fine particles was also high for the refuse vehicle as compared to the port vehicles. Figure 6-15 through Figure 6-17 show the size distribution results for a selected SCR equipped refuse hauler. The results show a higher fine particle concentration for the first 200 seconds that cover most of the size range sampled. Figure 6-16 shows the real time scan at 60 nm which supports the idea that there is a burst of fine particles at the cold start then after about 200 seconds (23 scans) the high concentration is gone. The size distribution continues to drop as time progresses for the refuse vehicle as seen by the still high concentration at 200 seconds to the end, see Figure 6-15.

Figure 6-17 shows the comparison between the UDDS (hot and cold) compared to the AQMD refuse cycle. The UDDS and refuse cycle show similar fine particles were the small peak at 7-30 nm may be measurement error for the SMPS. Additional data is needed to confirm this response.

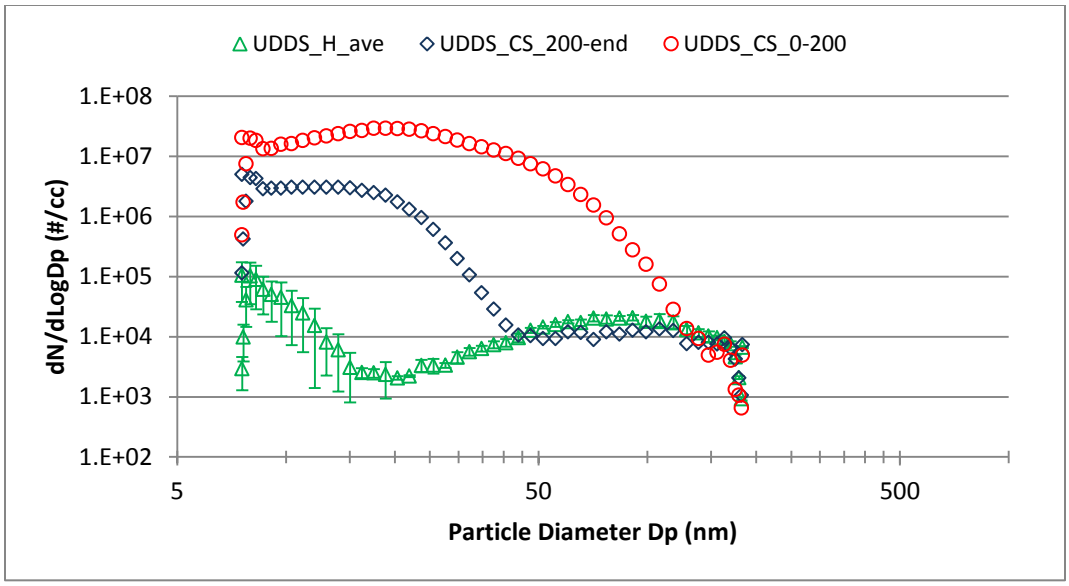


Figure 6-15 Average size distributions for the an SCR equipped refuse vehicle: UDDS

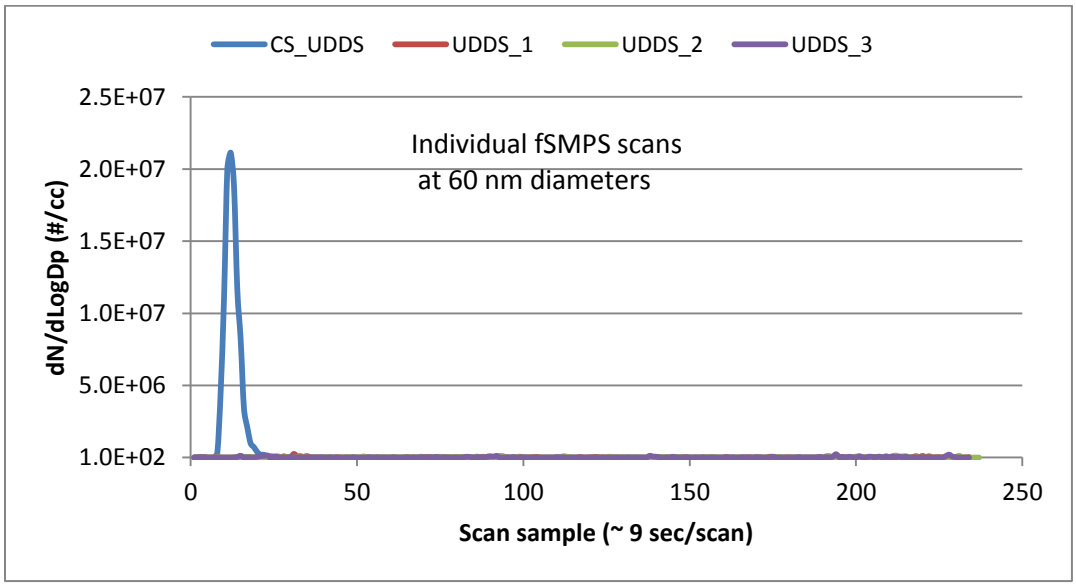


Figure 6-16 Selected scan particle size (60 nm) for the an SCR equipped refuse vehicle

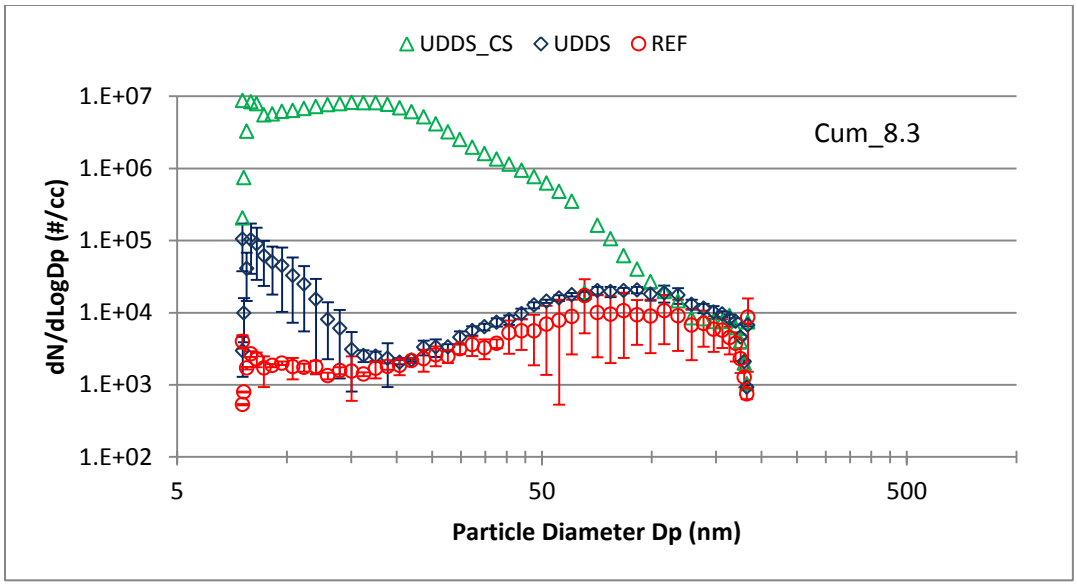


Figure 6-17 Average size distributions for an SCR equipped refuse vehicle: by cycle

In summary the refusle hauler PM mass was higher for the cold start than for the warm vehicles. This agrees with the higher measured size concentration and a similar behavior for the port vehicles.

6.7 Greenhouse Gas (N_2O, CH_4 & CO_2) Emissions and Fuel Economy

For greenhouse gases, UCR measured emissions factors of methane and carbon dioxide in real-time for all vehicles and N_2O with off-site analyses for selected vehicles. The off-site analyses were carried out by the California Air resources Board and West Virginia University. Results showed the measured values were close to ambient levels, as expected for diesel vehicles. Literature indicates N_2O is observed when vehicles rely on three way catalyst and UCR did not have any included in their fleet of test vehicles.

6.7.1 Emissions of nitrous oxide (N_2O)

N_2O emissions were measured on selected refuse haulers by the IR methods described earlier. The N_20 concentrations were found to be near the detection limits for those vehicles sampled for N_20. A literature review showed that Huai et alia[12] only found nitrous oxide when a three way catalyst was warming. Thus we only expected N_2O for the LPG school bus and LPG port truck as the after treatment was a three way catalyst. There were no refuse hauler LPG vehicles tested.

Of the vehicles tested for N_20, the N_2O measurements were very close to the ambient concentrations were negative numbers were reported for the refuse haulers sampled for N_2O. The reason for negative numbers is based on the correction of the ambient measured concentration exceeding the sample measurement, as described in a later section. It is

[12] T. Huai, Durbin, T.D., J.W. Miller, and J.M. Norbeck, *Estimates of the Emission Rates of Nitrous Oxide from Light-Duty Vehicles using Different Chassis Dynamometer Test Cycles*. Atmospheric Environment, vol. 38, 6621-6629 (2004)

expected that many of the measurements are near the detection limits of the N_2O measuring method. See Section 8.4.3 for analysis and summary of N_20 measurements and detection limits.

The general observations of the N_2O emissions from the vehicles tested can be summarized as:

- N_2O Analysis was done offsite when facilities were available. As such, not all vehicles or cycles were tested for N_2O. Only selected vehicles were tested for N_2O analysis.
- During the refuse and school bus testing there were no facilities for N_2O analysis thus they were not performed. Similar results are expected for all the vehicle categories.
- More than half (64%) of the measured toxic emissions were below the defined threshold (0.4 ppm), the average ambient background concentration plus one standard deviation.

6.7.2 Emissions of methane (CH_4)

Vehicles emit methane, a greenhouse gas, with a global warming potential (GWP) over 20 years of 72. This factor means that methane will trap 72 times more heat than an equal mass of carbon dioxide over the next 20 years. There are factors for 100 and 500 years but the 20 year factor is used in this analysis. From results of this project, the CH_4 contribution to greenhouse gases with diesel trucks can be ignored given that the emissions rate for CO_2 was about >2,000 gram/mile and that of CH4 was ~0.02 grams/mile. Thus emissions of CO_2 predominate for the greenhouse calculation, even after adjusting the methane rate by a factor of 72.

6.7.3 CO_2 and Fuel Economy emissions

CO_2 emissions for the refuse trucks are shown in Table 6-6 for the different test cycles. CO_2 emissions varied from 1,717 to 3,035 for the refuse trucks. The CO_2 emissions follow the same trends as for the fuel economy, since CO_2 is the predominant product of the combustion of the fuel.

Table 6-6: CO_2 Emissions for the Refuse Haulers in g/mile.

Vehicle			CO_2 (g/mi)		
Category	Engine	MY	RTC	UDDS	UDDS-CS
VIII	C8.3r	2012	2313	2818	3035
VIII	C9.3	2011	2016	2825	2590
VII	N7.6	2011	1717	1941	1811
V	N7.6	2008	2014	2356	2412

Fuel economy for the refuse haulers in different driving cycles are provided in Figure 6-18 to Figure 6-19. The refuse trucks showed slightly higher fuel economy values for the RTC compared to the UDDS. The 2010+ N7.6 refuse truck showed the highest fuel economy for the refuse trucks, while the lowest fuel economy for the refuse trucks was found for the 2010+ refuse trucks with SCR over the UDDS. There were no consistent trends between the UDDS and CS_UDDS cycles for the refuse haulers.

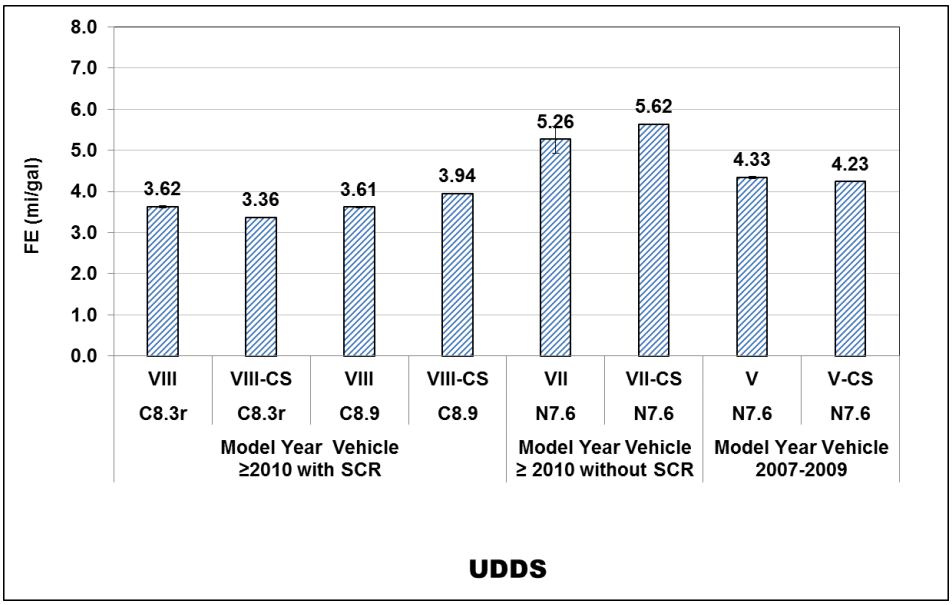


Figure 6-18: Fuel economy in miles/gallon of fuel for the UDDS cycle on the Refuse haulers.

[1] No error bars for the cold start tests because on only one test was performed

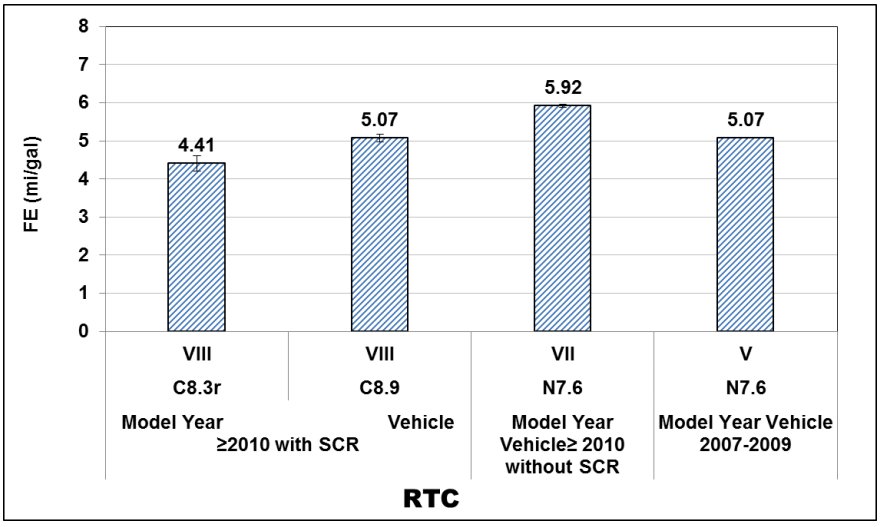


Figure 6-19: Fuel economy in miles/gallon of fuel for the AQMD Refuse Truck Cycle.

7 Results and Discussion for School Buses

This section covers the emissions for school buses for the Central Business District (CBD) cycle. The emissions are primarily reported on a grams per mile basis and where needed on a work basis to relate back to emission standards. The results represent the average from triplicate runs with one standard deviation error bars.

7.1 Test Buses

Two vehicles used as school buses were tested on a number of chassis cycles. Selected information for the school buses is identified in Table 1-1. One of the buses was fueled by LPG and the other was fueled by diesel and the aftertreatment included a DPF.

7.2 Test Conditions

Vehicles were tested on the CBC cycle, both cold and hot as these better represented what in-use values would look like. The loads for the goods movement vehicles were set at 20,000 lb., a value representative of a school bus with a capacity of 64 passengers at 100 lb. The weight accounted for the sum of the vehicle weight with school kids. Street fuels were used so both load and fuel matched in-use conditions. The emission values represent the average of triplicate runs and the graphs show the confidence limits to one standard deviation.

7.3 Regulated emissions

7.3.1 NO_x emissions

The NO_x emission results for the school buses are presented in Figure 7-1 for the CBD and the CS-CBD, respectively. The school buses showed significant differences between the two vehicles tested, with the 2007-2009 GM8.1 showing much lower emissions compared to the 2007-2009 C6.7 vehicle. These reductions were greater for the UDDS than the CS-UDDS, although the difference between the vehicles was still significant for the CS-UDDS. The emissions of the 2007-2009 C6.7 vehicles were comparable to those of the 2007-2009 vehicles in the other categories. The 2007-2009 GM8.1 had emissions that were lower than those of other vehicles in other vehicle categories, including the 2010+ vehicles with SCR.

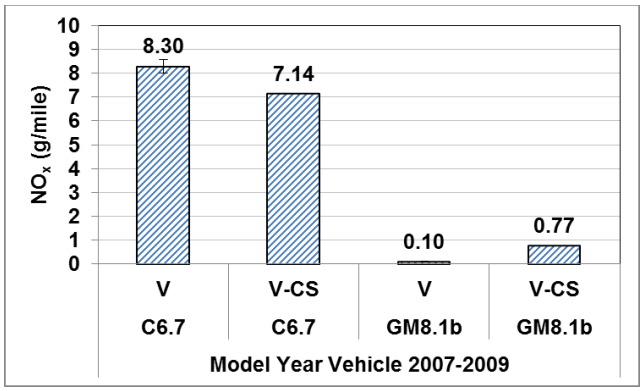


Figure 7-1: Emission factors for NOx CBD and cold start CBD cycles (g/mile)

[1] No error bars for the cold start tests because on only one test was performed

7.3.2 Percentage of NOx emissions as NO_2

NO_2 emissions are a health concern and values for the school buses are presented in Table 7-1. These tables show the percentage of the total NO_x that is NO_2 in g/mile. Interestingly the LPG vehicle did not have NOx or NO_2 while the diesel vehicle with the DPF did have up to 40% as NO_2. Similar to the observation with the goods movement vehicles, levels of NO_2 are high as compared with the ARB retrofit rule of 20% over baseline

Table 7-1: NO_2, NO_x and fraction of NO_2 to total NO_x for the bus cycles (g/mi)

Vehicle			CBD			CS_CBD		
Category	Engine	MY	NO_2	NO_x	$\%NO_2$	NO_2	NO_x	$\%NO_2$
V	C6.7	2007	2.86	7.14	40%	3.91	8.30	47%
V	GM8.1b	2008	-0.01	-0.01	n/a	-0.03	0.77	n/a

7.3.3 PM emissions

The PM emission results for the school buses are presented in Figure 7-2 for the CBD and the CS-CBD, respectively. The school buses showed differences in baseline PM emissions, which were approximately 2 mg/mi for the LPG fueled 2007-2009 GM8.1 vehicle and 6 mg/mi for the 2007-2009 C6.7 diesel vehicles. While measureable, these values are very low. The PM emissions for the CS-CBD for the 2007-2009 C6.7 were similar to those for that vehicle for the regular CBD, while the CS-CBD emissions for the 2007-2009 GM8.1 vehicle were at the limits of the measurement capability.

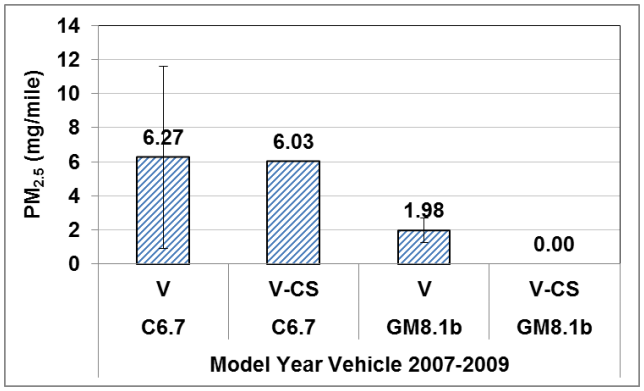


Figure 7-2: PM Emission factors for hot/cold CBD cycles (mg/mile)

[1] No error bars for the cold start tests because on only one test was performed

7.3.4 THC/NMHC/CH_4 and CO emissions

Table 7-2 and Table 7-3 show the emission factors for THC, CH_4, NMHC and CO for the CBD for buses. The emission factors for the THC, CH_4, and NMHC were low for all vehicle/cycle combinations. THC emissions were at or below 0.45 g/mi for most vehicle/cycle combinations. NMHC emissions were at or below 0.30 g/mi for nearly all vehicle/cycle combinations. CH_4 emissions were at or below 0.20 g/mi for nearly all vehicle/cycle combinations. Cold start

emissions were low for most vehicle/cycle combinations, with the 2007-2009 GM8.1 bus showing somewhat higher cold start THC, NMHC, and CH_4 emissions.

CO emissions were below 1 g/mi for most vehicle/cycle combinations, except the 2007-2009 LPG-fueled GM8.1 school buses. Cold start emissions were below 2 g/mi for all but the 2007-2009 GM8.1 school bus, which showed considerably higher CO emissions compared to the other vehicles of 16.0 g/mi.

Table 7-2: THC, CH_4, NMHC, and CO emissions for the Bus cycles

Vehicle			Emission Factor (g/mi)			
Category	Engine	MY	THC	CH4	NMHC	CO
V	C6.7	2007	0.04	0.02	0.03	0.20
V	GM8.1b	2008	0.30	0.20	0.13	9.82

Table 7-3 THC, CH_4, NMHC, and CO emissions for the Cold Start test cycles

		Vehicle		Emission Factor (g/mi)			
Cycle	Category	Engine	MY	THC	CH4	NMHC	CO
CS-CBD	V	C6.7	2007	-0.19	0.02	-0.21	-0.04
CS_CBD	V	GM8.1b	2008	0.77	0.25	0.56	16.03

7.4 Non-regulated Gaseous Emissions

7.4.1 NH_3 Emissions in g/mile

The NH_3 emission results for the school buses are presented in Figure 7-3 for the CBD and the CS-CBD. The NH_3 for the LPG-fueled 2007-2009 GM8.1 school bus was the highest among the vehicles being tested, in the range of 0.45 to 0.5 g/mi. The NH_3 emissions for the 2007-2009 C6.7 diesel-fueled vehicle were on the order of 10 to 20 mg/mi, which is near the lower end of the range of the vehicles tested for the this study.

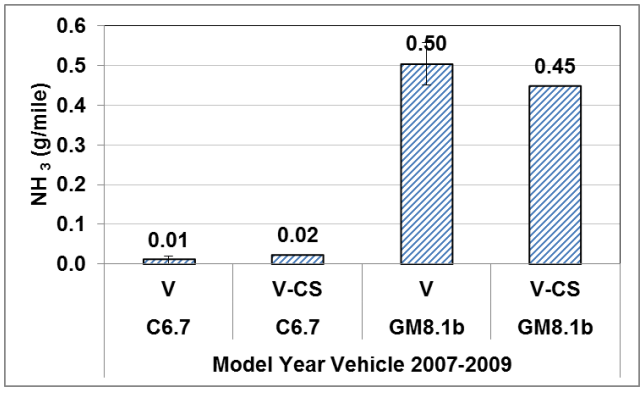


Figure 7-3: Emission factors for NH_3 CBD cycle (g/mile)[1]

[1] NH_3 scale is based on 10 ppm raw exhaust concentration

[2] No error bars for the cold start tests because on only one test was performed

7.4.2 Selected toxic emissions (1,3-butadiene & BTEX)

The 1,3 butadiene, benzene, and toluene results for the school buses are shown in Figure 7-4. Measureable levels for benzene were found for both vehicles for the cold start CBD, and measureable levels of toluene were found for the c6.7 for the CBDx2 and for the GM8.1 for the cold start CBD.

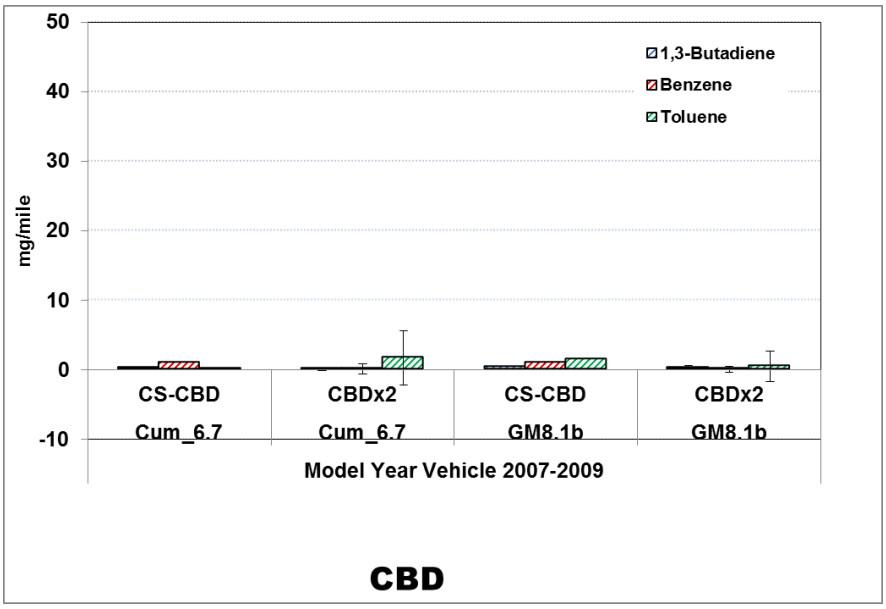


Figure 7-4 Emissions of Selected Toxic in mg/mile

[1] No error bars for the cold start tests because on only one test was performed

7.4.3 Selected toxic emissions (aldehydes & ketones)

The formaldehyde, acetaldehyde, and acetone carbonyl results for the school buses are shown in Figure 7-5. The GM 8.1 showed the highest levels of acetone and formaldehyde for cold start CBD, with the emissions of acetone being higher than those of formaldehyde. This is not surprising that there was partial oxidation of the LPG fuel on startup. The carbonyl emissions were very low for the other vehicle/cycle combinations.

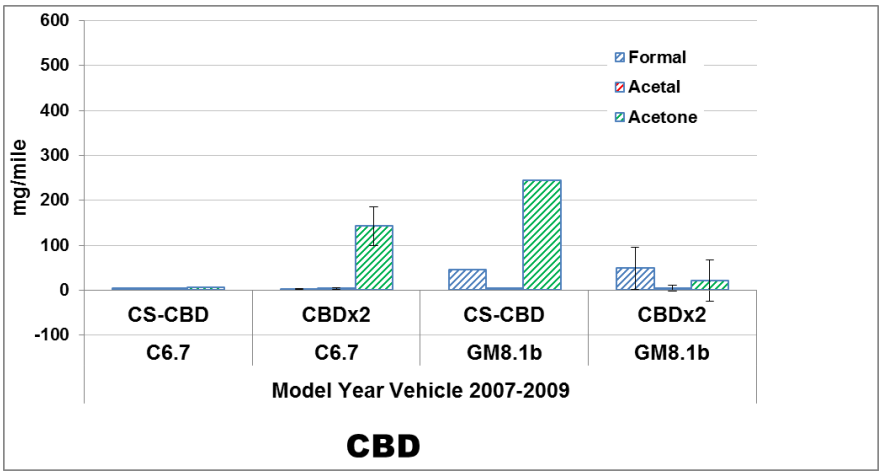


Figure 7-5 Emissions of Carbonyls & Ketones in mg/mile

[1] No error bars for the cold start tests because on only one test was performed

7.5 Non-regulated PM emissions

7.5.1 Fractionation of PM mass into OC and EC

The elemental and organic PM results for the school buses are shown in Figure 7-6. The GM8.1 showed elemental and organic carbon emissions that were essentially at the background levels. Not surprising the DPF captured all of the PM and elemental carbon. The C6.7 and GM8.1 school bus shows that OC was the primary PM for the cold start and the warm tests. Deeper analysis on the detection limits of the method used suggest the result may not be statistically significant since the OC measurement was very low and at detection limits of the method. See Section 8.4.4 for a discussion on EC and OC detection limits.

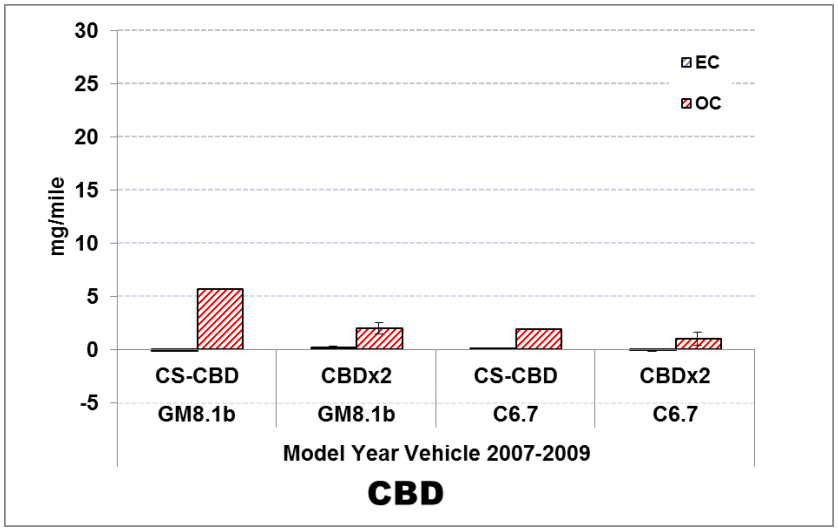


Figure 7-6 Fractionation of PM mass into OC and EC (mg/mile)

[1] No error bars for the cold start tests because on only one test was performed

7.5.2 Measurement of real-time and ultrafine PM

As described previously, The DMM was used to characterize the real time PM mass concentration and the f-SMPS was used for the ultrafine PM emissions characterization.

Real-time PM mass DMM

As presented earlier the PM mass of the gravimetric method were very low and typically around 3.8 mg/mi or 3.1 gm/bhp-hr for all the school bus vehicles tested. The average PM mass from the DMM measurement method averaged 0.5 mg/mi and 0.1 mg/bhp-hr for the same vehicles.

Ultra-fine PM emissions

There was no significant difference between cold start emissions and vehicle technology for the school bus tests. Figure 7-7 shows the size distribution for the propane and disel school bus tests for hot CBD tests cycles. The propane total PM mass was lower than the diesel PM mass on a g/mi basis which is supported by the lower size concentration at the 50 to 200 nm size range (ie most of the PM mass due to the diameter to the 3rd power). Additionally there is not a large cold start fine particle concentration for either the diesel or LNG school bus.

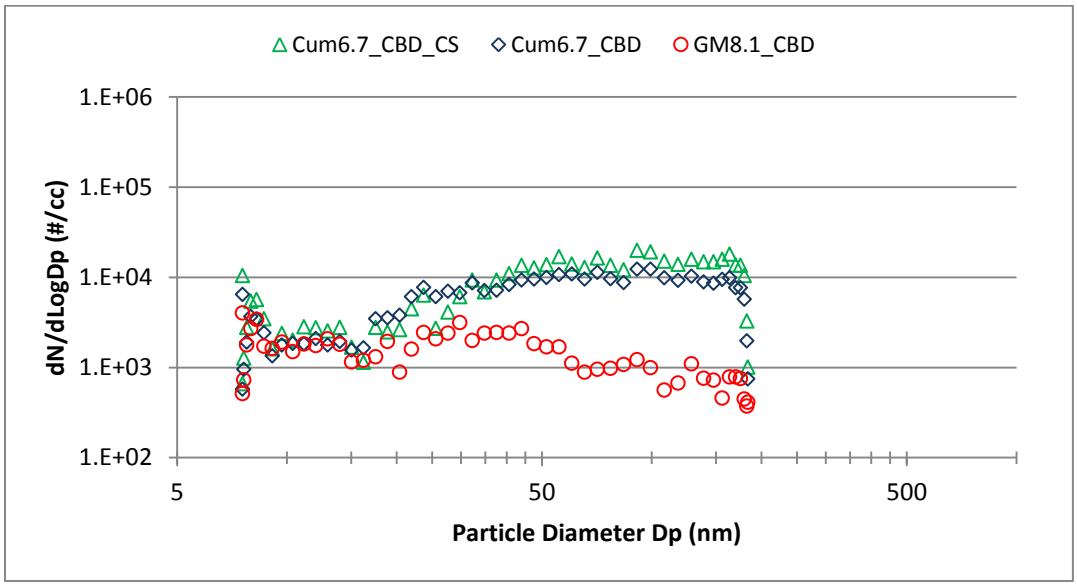


Figure 7-7 Average size distributions for the two school bus vehicles: CBD

7.6 Greenhouse Gas (N_2O, CH_4 & CO_2) Emissions and Fuel Economy

For greenhouse gases, UCR measured emissions factors of methane and carbon dioxide in real-time for all vehicles and N_2O with off-site analyses for selected vehicles. The off-site analyses were carried out by the California Air resources Board and West Virginia University. Results showed the measured values were close to ambient levels, as expected for diesel vehicles. Literature indicates N_2O is observed when vehicles rely on three way catalyst and UCR did not have any included in their fleet of test vehicles.

7.6.1 Emissions of nitrous oxide (N_2O)

N_2O emissions were measured by the IR methods described earlier and found to be near the detection limits. A literature review showed that Huai et alia[13] only found nitrous oxide when a three way catalyst was warming. Thus we only expected N_2O for the LPG truck as the after treatment was a three way catalyst.

The N_2O measurements were very close to the ambient concentrations were negative numbers were reported. The reason for negative numbers is based on the correction of the ambient measured concentration exceeding the sample measurement, as described in a later section. It is expected that many of the measurements are near the detection limits of the N_2O measuring method. See Section 8.4.3 for analysis and summary of N_20 measurements and detection limits.

The general observations of the N_2O emissions from the vehicles tested can be summarized as:

- N_2O Analysis was done offsite when facilities were available. As such, not all vehicles or cycles were tested for N_2O. Only selected vehicles were tested for N_2O analysis.
- During the refuse and school bus testing there were no facilities for N_2O analysis thus they were not performed. Similar results are expected for all the vehicle categories.
- More than half (64%) of the measured toxic emissions were below the defined threshold (0.4 ppm), the average ambient background concentration plus one standard deviation.

7.6.2 Emissions of methane (CH_4)

Vehicles emit methane, a greenhouse gas, with a global warming potential (GWP) over 20 years of 72. This factor means that methane will trap 72 times more heat than an equal mass of carbon dioxide over the next 20 years. There are factors for 100 and 500 years but the 20 year factor is used in this analysis. From results of this project, the CH_4 contribution to greenhouse gases with diesel trucks can be ignored given that the emissions rate for CO_2 was about >2,000 gram/mile and that of CH4 was ~0.02 grams/mile. Thus emissions of CO_2 predominate for the greenhouse calculation, even after adjusting the methane rate by a factor of 72.

7.6.3 CO_2 and Fuel Economy emissions

CO_2 emissions for the school busses are shown in Table 7-4 for the CBD cycle. CO_2 emissions varied from 1,354 to 1,516 for the school busses. The CO_2 emissions follow the same trends as for the fuel economy, since CO_2 is the predominant product of the combustion of the fuel. Fuel economy for the school busses is provided in Table 7-5 for the CBD cycle.

Table 7-4: CO_2 Emissions for School Buses.

[13] T. Huai, Durbin, T.D., J.W. Miller, and J.M. Norbeck, *Estimates of the Emission Rates of Nitrous Oxide from Light-Duty Vehicles using Different Chassis Dynamometer Test Cycles*. Atmospheric Environment, vol. 38, 6621-6629 (2004)

Vehicle			CO_2 (g/mi)	
Category	Engine	MY	CBD	CBD-CS
V	C6.7	2007	1354	1443
V	GM8.1b	2008	1516	1728

Table 7-5 Fuel Economy Data for School Buses (miles/gallon)

Vehicle			miles/gallon	
Category	Engine	MY	CBD	CBD-CS
V	C6.7	2007	7.07	7.56
V	GM8.1b	2008	4.07	3.55

8 Deeper Analysis of the NOx, NH_3, Toxic Emissions, and N_2O

This section was written to provide more detail on topics that the authors thought would provide better insight to the results section because of the interest in the SCAQMD District in learning more about the differences between the certification values for NOx and the values measured for near in-use conditions. As stated in the introduction, having emissions levels at certification values is assumed in the AQMP so knowing why in-use emission are higher is an important question.

8.1 NOx Emissions Control Technology & Results

8.1.1 Cooled exhaust gas recirculation (EGR)

Cooled exhaust gas recirculation (EGR) was an early solution to meet lower NOx standards. This project produced a surprising finding when the emissions from the UDDS emissions were compared with those of the three in-use port cycles. Results showed the NOx emissions for the near port cycle were 250% greater than those of the regional cycle. Furthermore the near port emissions were about 35% higher than the UDDS suggesting that the emissions from the in-use driving near the port will be greater than assumed in the AQMP. On the other hand, emissions from travel to regional distribution centers is about 55% lower so the final impact on inventory will depend on the activity-weighted mix of the driving cycles.

8.1.2 Three way catalysts (TWC)

Some vehicles have switched from diesel fuel to gaseous fuels, such as LPG or natural gas. In those cases, the NOx starts with precise metering of the air-fuel ratio so combustion is at stoichiometric conditions and then passing the exhaust over a three way catalyst. In cases where the combustion is lean, then NOx is high. The cases of the LPG truck showed higher NOx; however, the school bus had a lower NOx level.

8.1.3 Selective Catalytic reduction (SCR)

Figure 8-1 illustrates the after treatment system found on a typical exhaust after 2010 in order to meet the strict NOx standards. With Selective Catalytic Reduction (SCR) NOx is converted into nitrogen by reaction with ammonia over a special catalyst. When operating temperatures are >250°C, an aqueous solution of urea is injected into the exhaust upstream of the SCR catalyst. The heat converts the urea into ammonia and water which is the reactant to convert NOx to nitrogen. At temperatures <250°C, urea is not injected so the full engine out NOx emissions are emitted.

In actual operation catalyst temperatures are not simply either less/greater than 250°C. Instead the exhaust temperature is highly dynamic and follows the dynamic nature of the actual driving schedule. Figure 8-2 shows the temperature trace of the temperatures in three places in the exhaust as a function of time in seconds for the port cycle. Note for a significant portion of the beginning that the temperature is <250°C so urea is not added and there is no NOx control. Even after 250°C is reached, there are times that the temperature goes below the desired temperature.

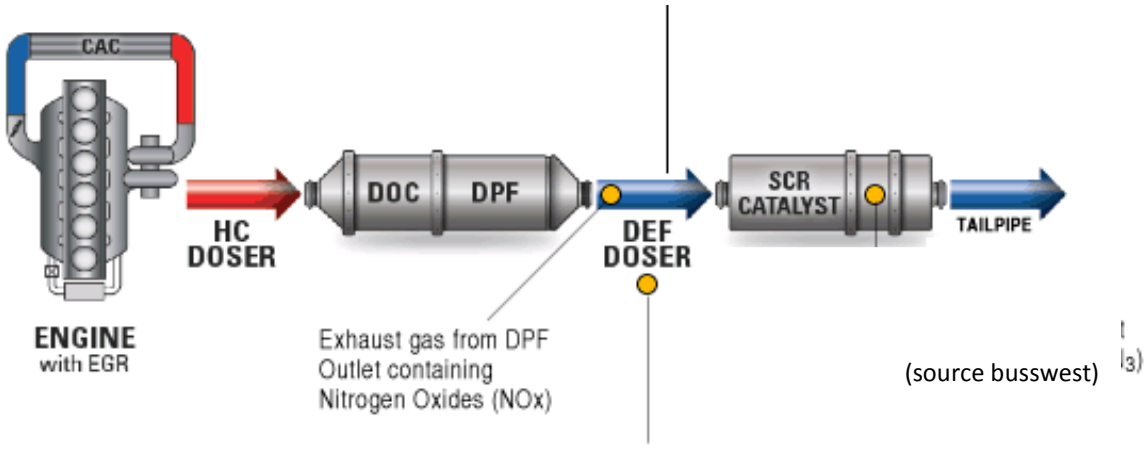


Figure 8-1: Figure of diesel DOC, DPF, and SCR after treatment system arrangement

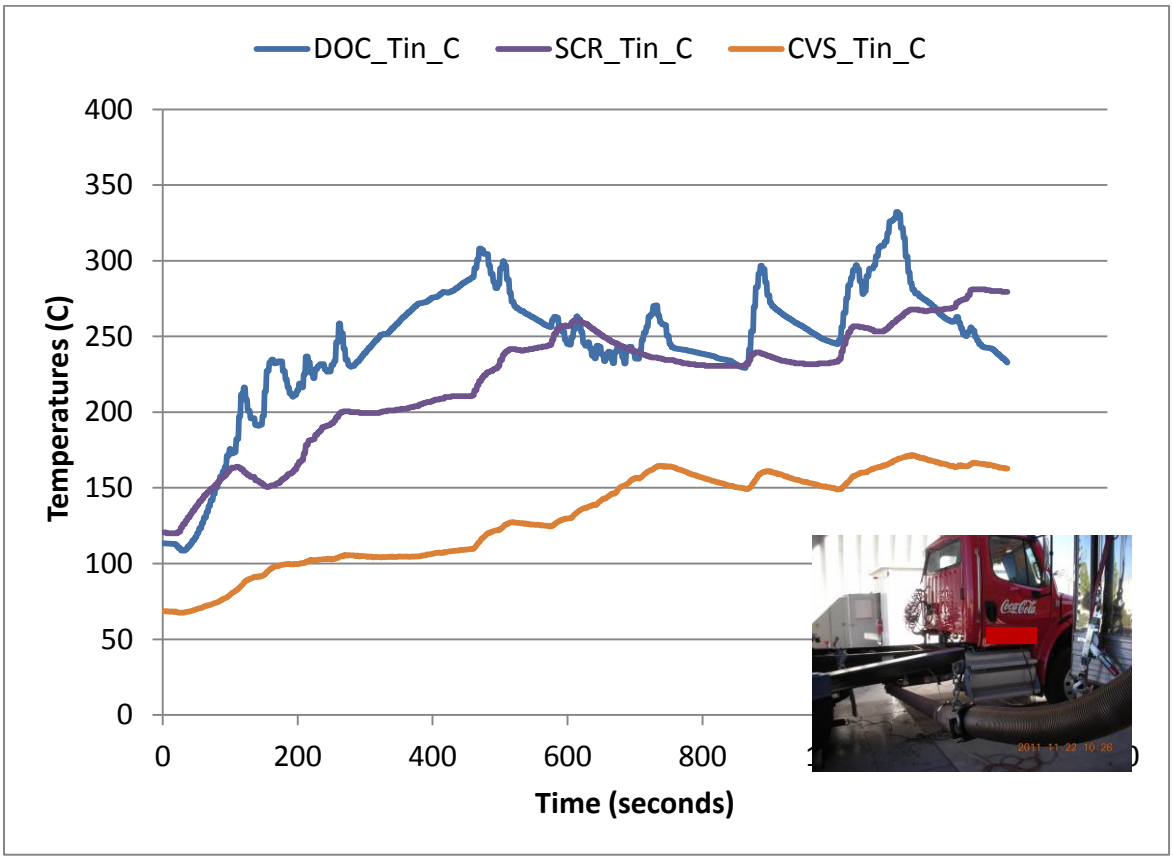


Figure 8-2: Typical engine catalyst temperatures as measured during this project

8.2 NO_x from Goods Movement Vehicles

Figure 8-3 shows how the cumulative NO_x rate varies over the regional port cycle for a SCR equipped goods movement vehicle as a function of time. Superimposed on the figure is the driving schedule with targeted vehicle speed. The results show that 2/3rd of the NOx accumulate in 1/3rd of the cycle time as the exhaust temperature at the SCR inlet is below approximately 250°C.

For the first 1750 seconds of the cycle the average NO_x emission rate is 1.34 g/bhp-h. After that time the vehicle is cruising at ~50mph and the SCR inlet temperature is above 325°C when relatively little NO_x is emitted. The average NO_x emission rate during the cruise portion of the cycle is 0.028 g/bhp-h, a value that shows the catalyst efficiency is 98%. Additional NO_x is emitted near the end of the cycle, as the temperature of the SCR inlet cools on the deceleration from the cruise. The average NO_x emission rate for the last portion of the cycle is 0.128 g/bhp-h.

The same run results are plotted as function of accumulated power in Figure 8-4, again showing that NO_x is predominately emitted during the initial period of the cycle where there is very little accumulated power and the SCR inlet temperature remains below 325°C.

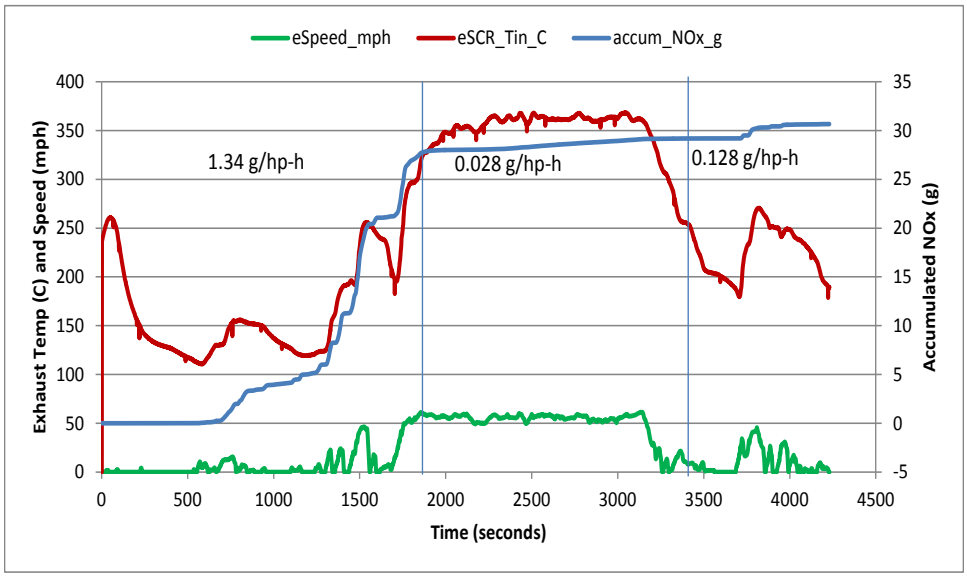


Figure 8-3: Brake specific NO_x Emissions for Regional Port Cycle versus Time

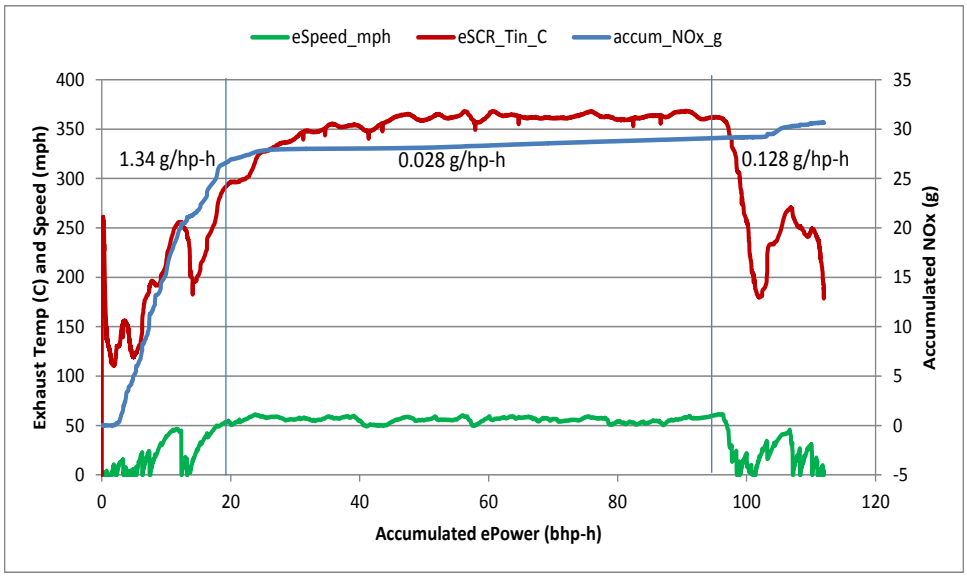


Figure 8-4: Brake specific NO_x emissions for the Regional port cycle as a function of work

The cold start catalyst temperatures were lower than the hot start catalyst temperatures and thus, showed much higher NO_x emissions. Figure 8-6 shows the Cummins ISX 11.9 liter engine's NO_x accumulated mass emissions for a cold and hot start UDDS. The cold start catalyst temperatures started at 10C and 230C for the hot start tests. The $bsNO_x$ for the first ½ mile, 1 mile, and from 1 to 11miles are computed and shown in the figure. The cold and hot start $bsNO_x$ emissions for the first ½ mile were 2.29 g/bhp-hr and 0.006 g/bhp-hr respectively. Similarly, the cold and hot start $bsNO_x$ emissions for the first 1 mile of the test were 1.48 g/bhp-hr and 0.005 g/bhp-hr respectively. The amount of emissions accumulated in 1 mile of the cold start UDDS are equivalent to 32 miles of the hot start UDDS for the Cummins ISX 11.9 engine.

Figure 8-5 show profiles of NO_x emissions in comparison with after treatment system (ATS) temperature for goods movement vehicles. For the goods movement vehicles, the highest NO_x emissions and corresponding lowest percentage of operation with the ATS >250°C were found for the Near Dock cycle. The lowest NO_x emissions and the highest percentage of operation with the ATS >250°C were found for the Regional cycle. Interestingly, for the 2010+ V12.8 vehicle, a relatively large portion of the NO_x emissions were produced when the ATS temperature was >250°C for the near dock and local cycles compared to the percentage of operation when the ATS temperature was >250°C.

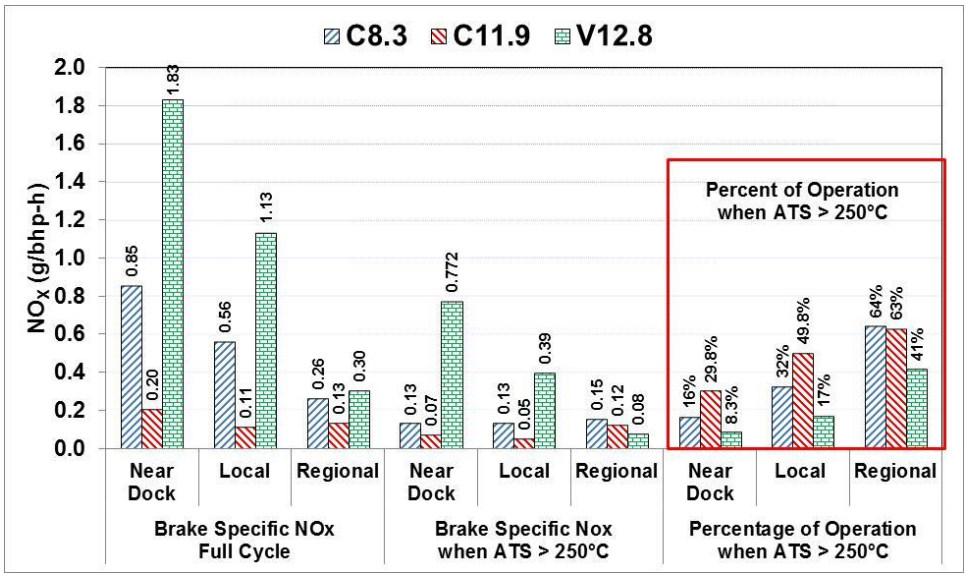


Figure 8-5: NO_x emissions in g/bhp-hr for the whole port cycle

[1] NO_x emissions only when the ATS temperature was >250^{0}C.

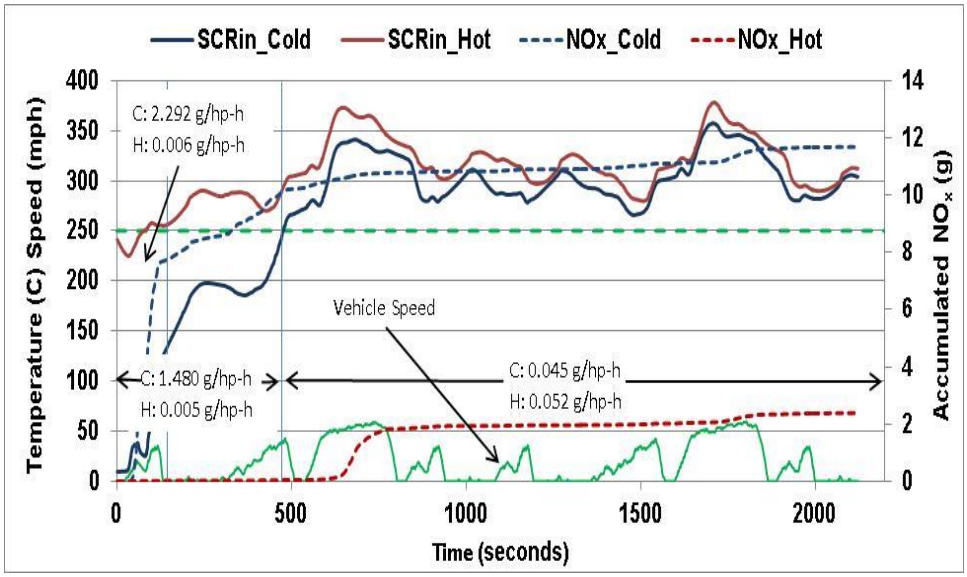


Figure 8-6: Accumulated NOx emissions for the C11.9 during hot and cold start UDDS

8.3 NO_x from Refuse Haulers

Figure 8-7 shows profiles of NO_x emissions in comparison with after treatment system (ATS) temperature for the refuse trucks. For the two refuse trucks, there was a higher percentage of operation with the ATS >250°C, with most combination have over 70% of operation with the ATS >250°C. Of the two vehicles, the 2010+ C9.3 refuse trucks showed strongest trends in NO_x emissions as a function of temperature. In particular, a relatively small percentage of NO_x emissions were formed when the ATS temperature was >250°C for the near dock and RTC cycles, even though 70+% of the operation was at these higher temperatures. For the 2010+

C8.3 refuse truck and the 2010+ C9.3 refuse truck for the UDDS, the percentage of NO_x produced when ATS was >250°C was more similar to the percentage of operation at the higher temperature operation.

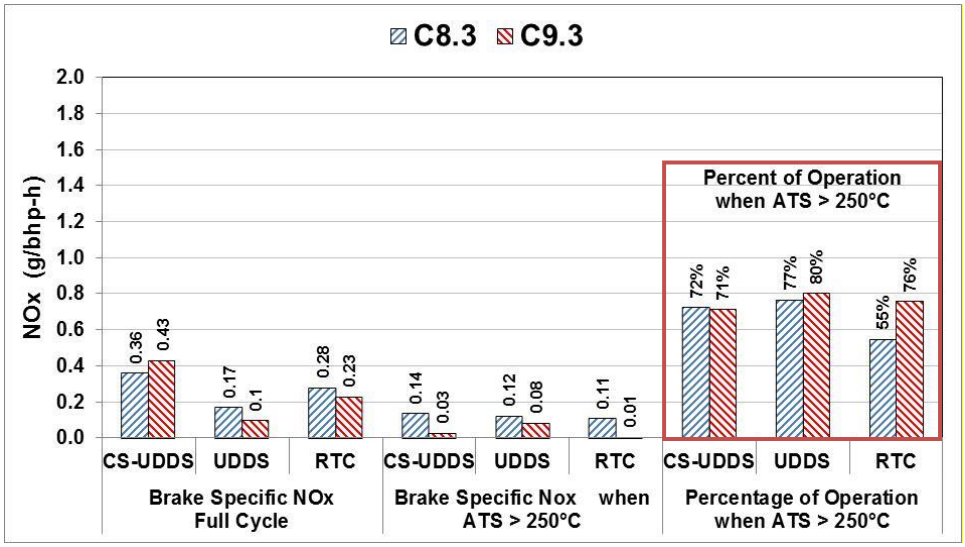


Figure 8-7: NOx emissions in g/bhp-hr for the whole AQMD Refuse Truck Cycle

[1] NO_x emissions only when the ATS temperature was >250^{0}C.

Figure 8-8 how the temperature profiles for the Refuse Truck cycle for one of the refuse trucks. The plot includes several of the different temperatures that were measurements were available for, including the exhaust temperature, pre- and post DOC temperatures, and the post-DPF temperature. The temperatures all show the same trends, where the temperature peaks after the first main double peak of the transit portion of the cycle and then slowly declines throughout the remainder of the transit portion and during the curbside portion. Temperatures during the compaction portion of the cycle show a slight increase, but overall are similar to those near the end of the curbside segment.

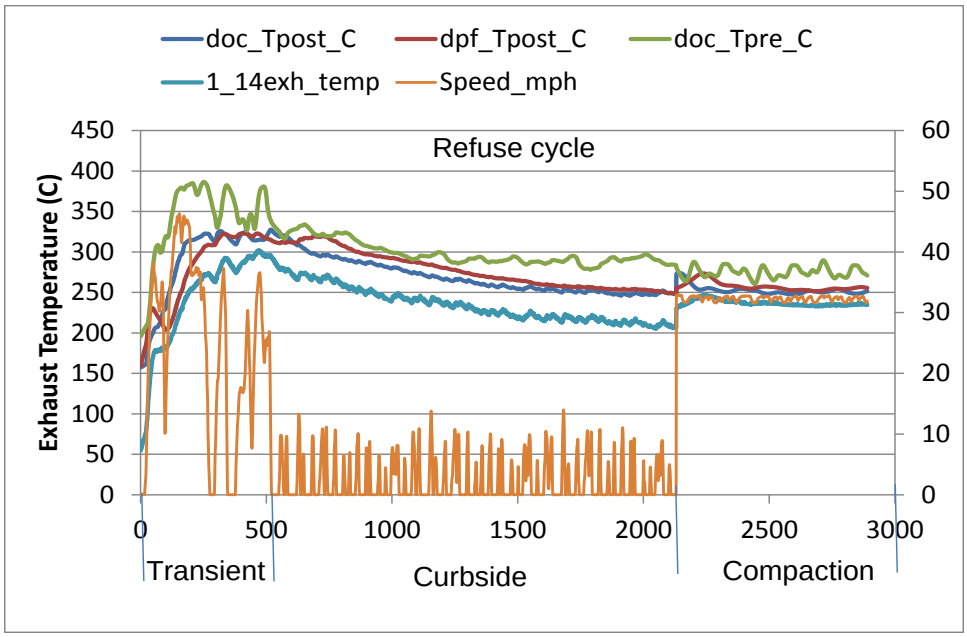


Figure 8-8: Example of SCR equipped refuse hauler exhaust temperatures

Figure 8-9 shows how cumulative NO_x varies over a refuse truck cycle for one of the SCR equipped refuse trucks as a function of cycle time. NO_x emissions over the refuse truck cycle showed some trends similar to the goods movement vehicle, but also showed a stronger dependency on the driving operation. Approximately 1/3rd of the cumulative NO_x emissions were from the first 200 seconds of operation when the post-DPF temperature was below 250˚C, with an average emission rate of 0.72 g/bhp-h. For the main part of the cycle, after the initial peak and including the curbside pickup portion of the cycle, relatively little NO_x is produced, with an average emission rate of 0.11 g/bhp-h. The greatest percentage of NO_x was formed during the latter stages of the cycle, when the compaction portion of the cycle was conducted. The average post-DPF temperature was around 250˚C during the compaction portion of the cycle and the average emission rate was 0.99 g/bhp-h. These same results for the refuse truck are plotted as function of accumulated power in Figure 8-10. The results show that the majority of the work is performed during the middle portion of the cycle, where the post-DPF temperatures are steadily above 250˚C. The initial segment of the cycle represents a relatively small portion of the overall cycle work. The compaction portion of the cycle represents only about 25% of the total work, but over 50% of the total accumulated NO_x.

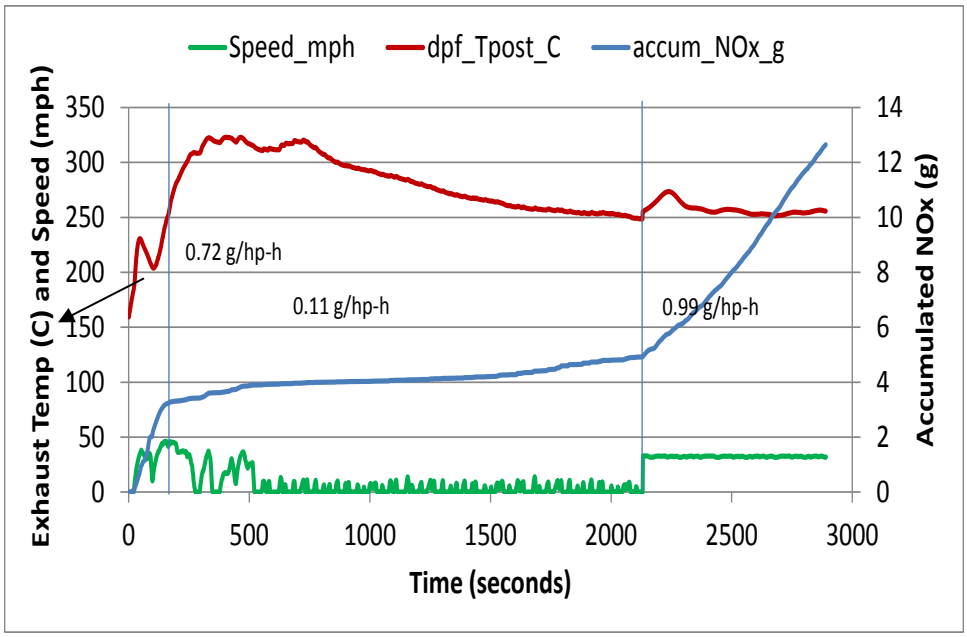


Figure 8-9: Brake specific NO_x emissions for the Refuse Truck Cycle as a function of time

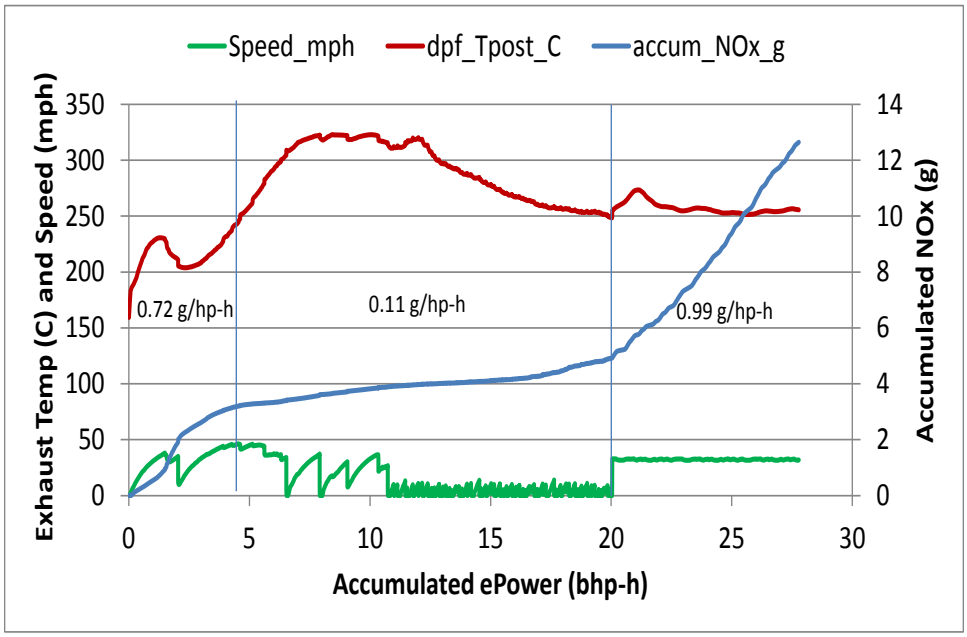


Figure 8-10: Brake specific NO_x emissions for the Refuse Truck Cycle as a function of work

The cold start catalyst temperatures were lower than the hot start catalyst temperatures and thus, showed much higher NO_x emissions. Figure 8-11 shows the Cummins ISX 11.9 liter engine's NO_x accumulated mass emissions for a cold and hot start UDDS. The cold start catalyst temperatures started at 10°C and 230°C for the hot start tests. The $bsNO_x$ for the first ½ mile, 1 mile, and from 1 to 11miles are computed and shown in the figure. The cold and hot start $bsNO_x$ emissions for the first ½ mile were 2.29 g/bhp-hr and 0.006 g/bhp-hr respectively. Similarly, the cold and hot start $bsNO_x$ emissions for the first 1 mile of the test were 1.48 g/bhp-

hr and 0.005 g/bhp-hr respectively. The amount of emissions accumulated in 1 mile of the cold start UDDS are equivalent to 32 miles of the hot start UDDS for the Cummins ISX 11.9 engine.

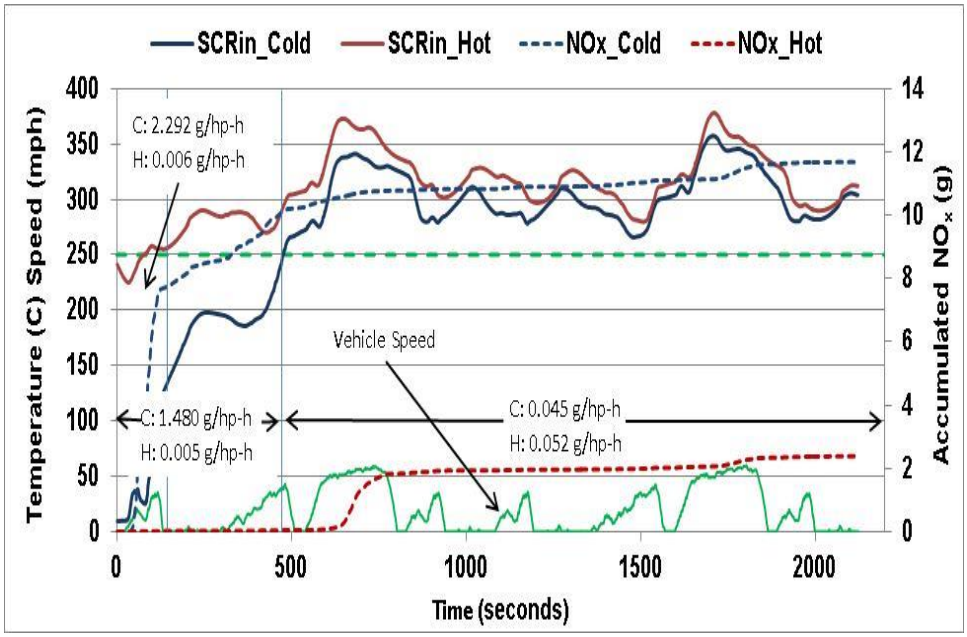


Figure 8-11: Accumulated NOx emissions for the C11.9 during hot and cold start UDDS

8.4 Discussion of detection limits

Results in this study showed that emissions of gaseous toxics were typically at or below detection level. In fact, some data showed that the exhaust values were less than ambient. A suitable reference for the discussion of hydrocarbons in diesel engines with DFPs is the Advanced Collaborative Emissions Study (ACES) which showed that hydrocarbons were reduced by up to 98% over a diesel engine without a DPF. The reason for the reduction is the DOC is an active catalyst for converting hydrocarbons to water and carbon dioxide.

8.4.1 Discussion of 1,3-butadiene & BTEX

As discussed previously many of the measurements for the non-regulated emissions were very low and sometimes negative. This section describes the BETEX concentration in relationship to measurement detection limits to help understand the limitations in making non-regulated emission measurements.

Table 8-1 through Table 8-8 shows the toxic average concentrations for the ambient and vehicle samples. The ambient concentration for the toxics ranged from 0.5 ppbv to 5 ppbv at one standard deviation. The ambient measured concentration single standard deviation is about equal to the measured value. If we establish a lower threshold for the toxic results to be equal to the average concentration pulse one standard deviation then we can visual display the data above and below this threshold. The sample concentrations in Table 8-1 through Table 8-8 are presented with this threshold in mind. If the cycle average measured concentration is less than the established threshold no value is displayed and if it is larger than the threshold than a value

will be displayed. Cells with no color are less than two times the threshold, green is less five times, orange less than ten times, and red is more than ten times.

As one can see many of the data points are not visible and are thus, below the established threshold. Additionally of the values shown in the tables most of these are still less than twice the established threshold and represent measurement at or near ambient detection limits.

The general observations about the toxic emissions from the vehicles tested can be summarized as:

- More than half (75%) of the measured toxic emissions were below a defined threshold of the average ambient background concentration pulse one standard deviation.
- More than half (55%) of the remaining values were less than two times the threshold. About 31% were between two and five times, 9% between five and ten times and only 5% were above ten times the threshold.
- Benzene appears to be the most dominate species measured for all the vehicles tested.
- More toxics emissions appear to be present for the port vehicles compared to the bus and refuse vehicles.
- The propane powered GM port vehicle showed the highest Benzene emissions. The regional cycle showed the highest Benzene emissions at more than 30 times the threshold. The Benzene emissions were highest for the port cycles compared to the UDDS cycle.
- One of the cold start port vehicles showed high ethyl benzene, m,p-xylene, and 0-xylene emissions. These measurements were only single samples (no duplicates were taken). Additional samples may be needed to confirm.
- The Advanced EGR vehicles appear to show more benzene emissions compared to the SCR equipped diesel vehicles. Additional testing would be needed to confirm this observation.

Table 8-1 Ambient Concentration and Confidence Limits

Average Toxic Ambient Background Concentration ppbv						
1,3-Butadiene	n-butane	benzene	toluene	ethyl benzene	m,p-xylene	o-xylene
0.50 ± 0.73	3.51 ± 2.01	1.46 ± 0.52	5.20 ± 4.65	0.52 ± 0.35	1.22 ± 0.86	0.41 ± 0.33

Table 8-2 Port vehicle Near dock (PDT1) cycle averaged concentrations

Test Article				Average Toxic Concentration ppbv						
Make	Engine MY	Disp. L	ATS Type	1,3-Butadiene	n-butane	benzene	toluene	ethyl benzene	m,p-xylene	o-xylene
Cummins	2010	8.3	SCR		9.32 ± 5.52	8.44 ± 3.14	22.95 ± 15.13	2.98 ± 0.87	6.79 ± 2.08	2.04 ± 1.51
Cummins	2011	11.9	SCR							
Volvo/Mack	2011	12.8	SCR			12.63 ± 1.98				
Navistar	2011	7.6	Adv EGR			5.28 ± 2.08				
Navistar	2011	12.4	Adv EGR			21.62 ± 1.98		2.23 ± 0.87	8.33 ± 6.24	3.05 ± 2.77
GM	2009	8.1	Propane	5.68 ± 1.23	13.78 ± 5.52	13.66 ± 1.98				
Navistar	2009	12.3	Adv EGR			7.08 ± 2.67				
DDC	2008	14.0	DOC/DPF							
DDC	2008	14.0	DOC/DPF							

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-3Port vehicle Local (PDT2) cycle averaged concentrations

Test Article				Average Toxic Concentration ppbv						
Make	Engine MY	Disp. L	ATS Type	1,3-Butadiene	n-butane	benzene	toluene	ethyl benzene	m,p-xylene	o-xylene
Cummins	2010	8.3	SCR			14.08 ± 11.93	27.07 ± 9.86	1.99 ± 0.87	3.05 ± 2.08	2.14 ± 1.37
Cummins	2011	11.9	SCR					1.03 ± 0.87	2.09 ± 2.08	1.12 ± 0.73
Volvo/Mack	2011	12.8	SCR	1.97 ± 1.75		2.14 ± 1.98				
Navistar	2011	7.6	Adv EGR			9.07 ± 3.62				
Navistar	2011	12.4	Adv EGR			17.49 ± 7.71	10.04 ± 9.86	1.99 ± 1.11	4.42 ± 2.08	1.86 ± 0.97
GM	2009	8.1	Propane	2.84 ± 2.99	12.76 ± 13.54	36.05 ± 1.98				
Navistar	2009	12.3	Adv EGR			3.36 ± 5.30		1.14 ± 1.86	2.30 ± 3.68	
DDC	2008	14.0	DOC/DPF							
DDC	2008	14.0	DOC/DPF							

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-4 Port vehicle Regional (PDT3) cycle averaged concentrations

Test Article				Average Toxic Concentration ppbv						
Make	Engine MY	Disp. L	ATS Type	1,3-Butadiene	n-butane	benzene	toluene	ethyl benzene	m,p-xylene	o-xylene
Cummins	2010	8.3	SCR			4.09 ± 4.58			2.38 ± 3.35	
Cummins	2011	11.9	SCR					1.13 ± 0.87	2.49 ± 2.08	1.21 ± 0.73
Volvo/Mack	2011	12.8	SCR	1.37 ± 1.59		6.04 ± 5.08	29.12 ± 11.63	4.60 ± 1.63	9.48 ± 2.08	3.42 ± 0.73
Navistar	2011	7.6	Adv EGR			9.44 ± 9.77				
Navistar	2011	12.4	Adv EGR			11.11 ± 1.98	16.04 ± 9.86	2.30 ± 0.87	7.12 ± 2.55	3.10 ± 1.18
GM	2009	8.1	Propane			60.82 ± 10.49	16.19 ± 9.86	1.25 ± 0.87	2.47 ± 2.08	0.80 ± 0.73
Navistar	2009	12.3	Adv EGR			5.00 ± 4.08		1.47 ± 1.08	2.22 ± 2.08	0.81 ± 0.73
DDC	2008	14.0	DOC/DPF							
DDC	2008	14.0	DOC/DPF							

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-5 Port vehicle UDDS cycle averaged concentrations

Test Article Engine				Average Toxic Concentration ppbv						
Make	MY	Disp. L	ATS Type	1,3-Butadiene	n-butane	benzene	toluene	ethyl benzene	m,p-xylene	o-xylene
Cummins	2010	8.3	SCR							
Cummins	2011	11.9	SCR							
Volvo/Mack	2011	12.8	SCR							
Navistar	2011	7.6	Adv EGR			9.64 ± 5.95				
Navistar	2011	12.4	Adv EGR			2.61 ± 1.98		2.64 ± 2.27	8.11 ± 8.32	3.03 ± 2.70
GM	2009	8.1	Propane	22.76 ± 1.23	17.44 ± 5.52	39.79 ± 1.98				
Navistar	2009	12.3	Adv EGR		6.32 ± 5.52	2.70 ± 1.98		1.06 ± 0.87	2.33 ± 2.08	0.92 ± 0.73
DDC	2008	14.0	DOC/DPF							
DDC	2008	14.0	DOC/DPF							

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-6 Port vehicle cold start UDDS cycle averaged concentrations

Test Article Engine				Average Toxic Concentration ppbv						
Make	MY	Disp. L	ATS Type	1,3-Butadiene	n-butane	benzene	toluene	ethyl benzene	m,p-xylene	o-xylene
Cummins	2010	8.3	SCR		9.11 ±	8.10 ±	12.40 ±	1.60 ±	3.28 ±	1.32 ±
Cummins	2011	11.9	SCR			2.94 ±		1.30 ±	3.30 ±	1.18 ±
Volvo/Mack	2011	12.8	SCR							
Navistar	2011	7.6	Adv EGR							
Navistar	2011	12.4	Adv EGR			5.22 ±		5.45 ±	14.39 ±	5.61 ±
GM	2009	8.1	Propane							
Navistar	2009	12.3	Adv EGR	2.02 ±	5.70 ±			1.55 ±	4.16 ±	1.47 ±
DDC	2008	14.0	DOC/DPF							
DDC	2008	14.0	DOC/DPF							

[1] All reported concentrations are greater than the average background concentration plus 1 stdev
[2] cold start emissions are based on single measurement and thus will have higher uncertainty.

Table 8-7 Bus vehicle cycle averaged concentrations

	Test Article Engine				Average Toxic Concentration ppbv						
Cycle	cycle	MY	Disp. L	ATS Type	1,3-Butadiene	n-butane	benzene	toluene	ethyl benzene	m,p-xylene	o-xylene
CS-CBD	Cum_6.7	2008	6.7	TWC		6.84 ± 6.84	2.79 ± 2.79		1.49 ± 1.49	2.14 ± 2.14	1.15 ± 1.15
CBDx2	Cum_6.7	2008	6.7	TWC					1.98 ± 2.89	5.04 ± 7.70	1.80 ± 2.70
CS-CBD	GM8.1	2007	8.1	DOC/DPF							
CBDx2	GM8.1	2007	8.1	DOC/DPF							

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-8 Refuse vehicle cycle averaged concentrations

	Test Article				Average Toxic Concentration ppbv						
Cycle	Make	Engine MY	Disp. L	ATS Type	1,3-Butadiene	n-butane	benzene	toluene	ethyl benzene	m,p-xylene	o-xylene
RTC	Cum_8.3	2012	8.3	SCR							
RTC	Cum_9.3	2011	9.3	SCR							
RTC	Nav_7.6	2011	7.6	Adv EGR			7.77 ± 2.99		1.02 ± 0.87	2.41 ± 2.08	1.14 ± 0.73
RTC	Nav_7.6	2008	7.6	Adv EGR							
UDDS	Cum_8.3	2012	8.3	SCR							
UDDS-CS	Cum_8.3	2012	8.3	SCR							
UDDS	Cum_9.3	2011	9.3	SCR							
UDDS-CS	Cum_9.3	2011	9.3	SCR							
UDDS	Nav_7.6	2011	7.6	Adv EGR			2.59 ± 1.98				
UDDS-CS	Nav_7.6	2011	7.6	Adv EGR							
UDDS	Nav_7.6	2008	7.6	Adv EGR							
UDDS-CS	Nav_7.6	2008	7.6	Adv EGR							

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

8.4.2 Discussion of Carbonyls & Ketones

As discussed previously many of the measurements for the non-regulated emissions were very low and sometimes negative. This section describes the carbonyl concentration in relationship to measurement detection limits to help understand the limitations in making un-regulated emission measurements.

Table 8-9 through Table 8-17 show the average and single standard deviation for the carbonyl emissions concentrations as measured for the back ground and vehicle samples. Table 8-9 shows the toxic average concentrations for the ambient samples. The ambient concentration for the toxics ranged from 0.15 µg/l for acetone to, 0.1 µg/l for acetal, and 0.02µg/l for Formaldehyde to below detection for most of the remaining carbonyls. The ambient concentrations have a single standard deviation approximately equal to their average measurement suggesting the analysis method and measurements are near the detection limits of the method.

To investigate the emissions from carbonyls from the vehicle samples, a lower threshold for the toxic results to be equal to the average concentration plus one standard deviation was used. The sample concentrations in Table 8-10 through Table 8-16 are presented with this threshold in mind. If the cycle average measured concentration is less than the established threshold, then no value is displayed and if it is larger than the threshold than a value will be displayed. Cells with no color represent measurements less than two times the threshold, color green represents less than five times, orange less than ten times, and red more than ten times the threshold value. Table 8-17 shows the percentage of samples for all vehicles for each species that were above the defined threshold.

As it can be seen in the tables, most of the data points are below the threshold. Amongst the reported results a majority of them were less than five times and the rest less than two times the threshold value. Therefore they represent measurement at or near measurement method detection limits. The following can be summarized about the results:

- Formaldehyde was the most significantly observed carbonyl from all the vehicles in all the categories and all the test cycles. It amounted to more than half (78%) of defined threshold of the average ambient background concentration pulse one standard deviation. Acetaldehyde was next, amounting to 27% of the above threshold limit of the respective acetaldehyde concentration. The detailed table below provides a clear understanding of the distribution of the above threshold values for all the thirteen carbonyls.
- More toxics emissions appear to be present for the port vehicles compared to the bus and refuse vehicles.
- The propane powered GM port vehicle showed the highest formaldehyde emissions for all the port cycles and the UDDS cycle. The regional cycle showed >50 times threshold values of formaldehyde, and the port cycles showed higher formaldehyde than the UDDS cycle for this vehicle.
- Advanced EGR vehicles had more above threshold emissions in comparison with the SCR technology vehicles. Although, the above threshold emissions from the EGR were less than five times the average threshold concentrations.

Table 8-9 Ambient background measured concentration and detection limits

Average background concentration (µg/l)												
Formal	Acetal	Acetone	Acrolein	Propional	Crotonal	Methac	MEK	Butyral	Benzal	Valeral	Tolual	Hexanal
0.023±0.006	0.11±0.013	0.152±0.104	0±0	0±0	0.028±0.019	0±0.002	0±0	0±0	0.004±0.009	0±0	0±0	0±0.003

Table 8-10 Port vehicle Near dock (PDT1) cycle averaged concentrations

Category	Engine M	Disp. L	ATS Type	Formal	Acetal	Acetone	Acrolein	Propional	Crotonal	Methac	MEK	Butyral	Benzal	Valeral	Tolual	Hexanal
C8.3	2010	8.3	SCR	0.09 ± 0.05												
C11.9	2011	11.9	SCR	0.03 ± 0.03												
V12.8	2011	12.8	SCR	0.17 ± 0.03	0.05 ± 0.02					0.01 ± 0.03						
N7.6	2011	7.6	Adv EGR	0.11 ± 0.03	0.04 ± 0.02									0.01 ± 0.01		
N12.4	2011	12.4	Adv EGR	0.08 ± 0.03	0.03 ± 0.02					0.01 ± 0.01		0.01 ± 0				
GM8.1	2009	8.11	Propane	1.18 ± 0.04	0.22 ± 0.02											
N12.3	2009	12.3	Adv EGR	0.13 ± 0.04												
D14a	2008	14	DOC/DPF	0.11 ± 0.03												
D14b	2008	14	DOC/DPF	0.13 ± 0.03						0.01 ± 0.01		0.01 ± 0				

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-11 Port vehicle Local (PDT2) cycle averaged concentrations

Category	Engine M	Disp. L	ATS Type	Formal	Acetal	Acetone	Acrolein	Propional	Crotonal	Methac	MEK	Butyral	Benzal	Valeral	Tolual	Hexanal
C8.3	2010	8.3	SCR	0.07 ± 0.03										0.00 ± 0.00		
C11.9	2011	11.9	SCR													
V12.8	2011	12.8	SCR	0.13 ± 0.03										0.00 ± 0.00		
N7.6	2011	7.6	Adv EGR	0.10 ± 0.03												
N12.4	2011	12.4	Adv EGR	0.09 ± 0.03						0.00 ± 0.01						
GM8.1	2009	8.11	Propane	1.50 ± 0.10	0.29 ± 0.05		0.03 ± 0.00									
N12.3	2009	12.3	Adv EGR	0.16 ± 0.03						0.00 ± 0.01			0.02 ± 0.01			
D14a	2008	14	DOC/DPF	0.07 ± 0.03												
D14b	2008	14	DOC/DPF	0.08 ± ###									0.02 ± ###			

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-12 Port vehicle Regional (PDT3) cycle averaged concentrations

Category	Engine M	Disp. L	ATS Type	Formal	Acetal	Acetone	Acrolein	Propional	Crotonal	Methac	MEK	Butyral	Benzal	Valeral	Tolual	Hexanal
C8.3	2010	8.3	SCR	0.06 ± 0.03								0.00 ± 0		0 ± 0.00		
C11.9	2011	11.9	SCR	0.03 ± 0.03												
V12.8	2011	12.8	SCR	0.08 ± 0.04										0.01 ± 0.01		
N7.6	2011	7.6	Adv EGR	0.10 ± 0.03									0.02 ± 0.0129			
N12.4	2011	12.4	Adv EGR	0.07 ± 0.03												
GM8.1	2009	8.11	Propane	1.51 ± 0.03	0.26 ± 0.02		0.03 ± 0.00					0.06 ± 0				
N12.3	2009	12.3	Adv EGR				0.01 ± 0.02									
D14a	2008	14	DOC/DPF													
D14b	2008	14	DOC/DPF													

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-13 Port vehicle UDDS cycle averaged concentrations

Category	Engine M	Disp. L	ATS Type	Formal	Acetal	Acetone	Acrolein	Propional	Crotonal	Methac	MEK	Butyral	Benzal	Valeral	Tolual	Hexanal
C8.3	2010	8.3	SCR	0.06 ± 0.03												
C11.9	2011	11.9	SCR	0.04 ± 0.03		1.16 ± 1.54										
V12.8	2011	12.8	SCR	0.04 ± 0.03												
N7.6	2011	7.6	Adv EGR	0.14 ± 0.03	0.03 ± 0.02											
N12.4	2011	12.4	Adv EGR	0.13 ± 0.03												
GM8.1	2009	8.11	Propane	1.59 ± 0.03	0.33 ± 0.02											
N12.3	2009	12.3	Adv EGR	0.24 ± 0.06	0.03 ± 0.02											
D14a	2008	14	DOC/DPF	0.09 ± 0.03												
D14b	2008	14	DOC/DPF	0.09 ± 0.03												

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-14 Port vehicle cold start UDDS cycle averaged concentrations

Category	Engine M	Disp. L	ATS Type	Formal	Acetal	Acetone	Acrolein	Propional	Crotonal	Methac	MEK	Butyral	Benzal	Valeral	Tolual	Hexanal
C8.3	2010	8.3	SCR	0.13 ± 0.13	0.03 ± 0.03											
C11.9	2011	11.9	SCR													
V12.8	2011	12.8	SCR	0.15 ± 0.15	0.02 ± 0.02											
N7.6	2011	7.6	Adv EGR													
N12.4	2011	12.4	Adv EGR													
GM8.1	2009	8.11	Propane													
N12.3	2009	12.3	Adv EGR	0.28 ± 0.28												
D14a	2008	14	DOC/DPF													
D14b	2008	14	DOC/DPF													

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-15 Bus vehicle cycle averaged concentrations

Category	Engine M	Disp. L	Cycle	Formal	Acetal	Acetone	Acrolein	Propional	Crotonal	Methac	MEK	Butyral	Benzal	Valeral	Tolual	Hexanal
C6.7	2008	8.1	CS-CBD	0.04 ± 0.04	0.03 ± 0.03											
C6.7	2008	8.1	CBDx2	0.03 ± 0.03	0.03 ± 0.02	0.74 ± 0.52										
GM8.1	2007		CS-CBD	0.21 ± 0.21	0.03 ± 0.03	1.16 ± 1.16										
GM8.1	2007		CBDx2	0.23 ± 0.27	0.03 ± 0.04											

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-16 Refuse vehicle cycle averaged concentrations

Category	Engine M	Disp. L	Cycle	Formal	Acetal	Acetone	Acrolein	Propional	Crotonal	Methac	MEK	Butyral	Benzal	Valeral	Tolual	Hexanal
C8.3r	2012	8.3	REF	0.08 ± 0.03						0.00 ± 0.00		0.00 ± 0				
C9.3	2011	9.3	REF	0.04 ± 0.03												
N7.6	2011	7.6	REF	0.07 ± 0							0 ± #	0.00 ± 0				
N7.6	2008	7.6	REF	0.17 ± 0												
C8.3r	2012	8.3	UDDS	0.05 ± 0						0.00 ± 0.00		0.00 ± 0				
C8.3r	2012	8.3	UDDS-CS	0.11 ± 0.1	0.03 ± 0					0.01 ± 0.01						
C9.3	2011	9.3	UDDS	0.04 ± 0												
C9.3	2011	9.3	UDDS-CS													
N7.6	2011	7.6	UDDS	0.14 ± 0	0.04 ± 0											
N7.6	2011	7.6	UDDS-CS													
N7.6	2008	7.6	UDDS	0.22 ± 0												
N7.6	2008	7.6	UDDS-CS													

[1] All reported concentrations are greater than the average background concentration plus 1 stdev

Table 8-17 Ambient background measured concentration and detection limits

	Formal	Acetal	Acetone	Acrolein	Propiona	Crotonal	Methac	MEK	Butyral	Benzal	Valeral	Tolual	Hexanal
Above	78%	27%	7%	0%	0%	0%	12%	0%	0%	5%	0%	0%	0%
Below	22%	73%	93%	100%	100%	100%	88%	100%	100%	95%	100%	100%	100%
2-5X	39%	0%	5%	0%	0%	0%	7%	0%	0%	0%	0%	0%	0%
5-10X	8%	2%	0%	0%	0%	0%	2%	0%	0%	0%	0%	0%	0%
>10X	7%	2%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%

8.4.3 Discussion of N_2O limits

This section describes the nitrogen dioxide (N_2O) concentrations in relationship to measured detection limits to help understand the limitations in making un-regulated emission measurements. The first part of the analysis describes the comparison between the MEL's laboratory NDIR CO_2 measurement. The reason for CO_2 comparison is to provide the reader confidence the samples were aligned properly and the bags were sampled properly and agree well with the MEL. After the CO_2 comparison the N_2O analysis is presented. Then a final description of the fuel specific, brake specific, and mile specific N_2O emissions are presented to show typical contributions of the measured values.

CO_2 NDIR and FTIR analysis and comparison

Figure 8-12 and Figure 8-13 below show the vehicle CO_2 and ambient CO_2 measurement comparisons between the FTIR and MEL laboratory NDIR instruments. The FTIR system is a bag measurement that was transported from UCR to an outside laboratory for N_2O Analysis. Additionally these systems report several other species which include CO_2. Since CO2 is a large signal by the FTIR they should roughly agree with the MEL laboratory NDIR measurement. Figure 8-12 shows that the source comparison varied from 0.2 % to 1% for the selected tests. The average ratio of FTIR/NDIR CO_2 averaged 0.84 with a 95% confidence standard deviation of 0.24. The 95% confidence suggests the measurement ranges from just over 1 to 1 to about 50% of the signal. For the ambient FTIR CO_2 data in Figure 8-13, the measurement uncertainty is around 0.05 %. Four points were driving the large 95% confidence value of 0.24. These occurred at low concentrations where the 0.05% FTIR uncertainty could explain the bias. As such, it appears all the provided N_2O data is reasonably and represents good bag samples and should be accurate for vehicle comparisons.

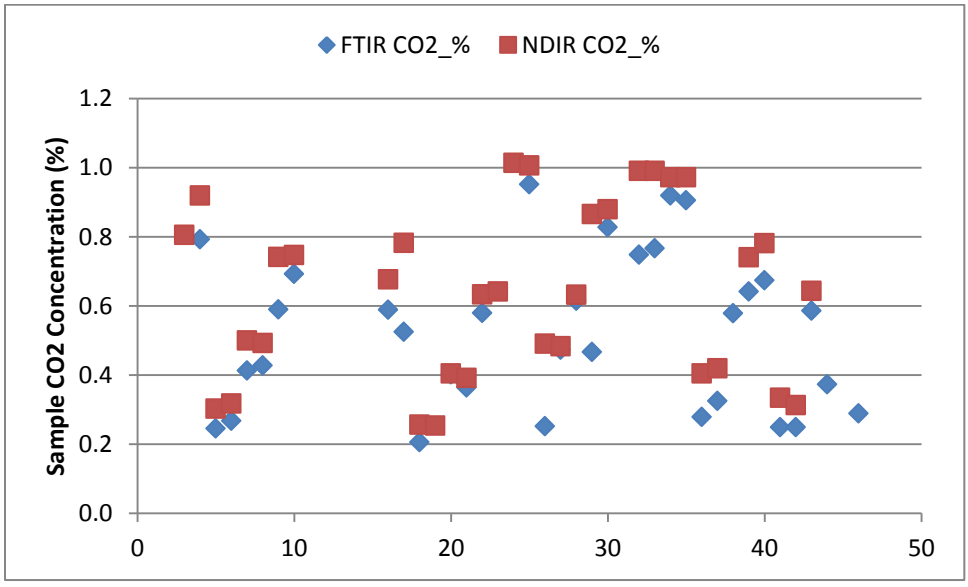


Figure 8-12 FTIR compared to laboratory CO_2 measurement for selected vehicle sources

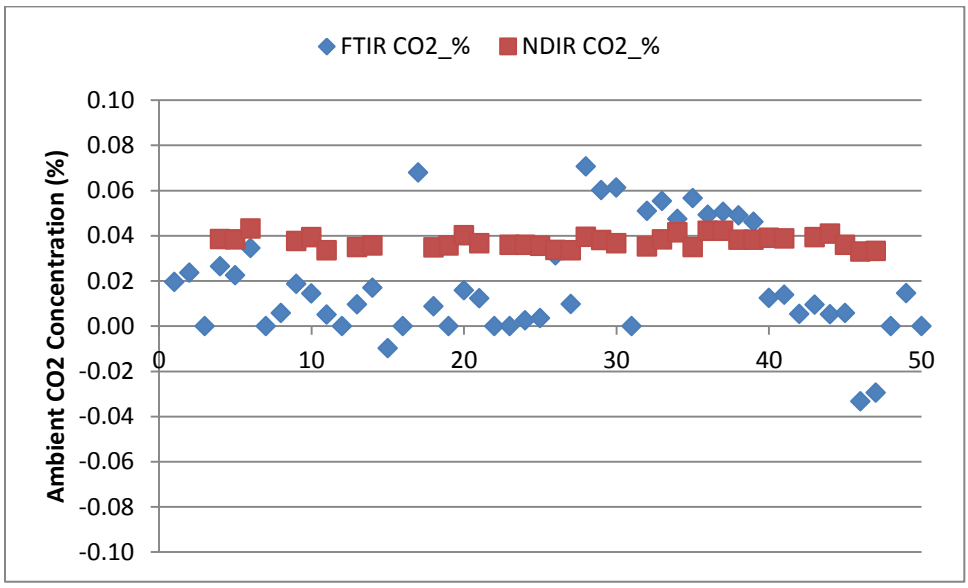


Figure 8-13 FTIR compared to laboratory CO_2 measurement for selected ambient bags

N_20 analysis and detections limits

Table 8-18 and Table 8-24 show the average N_2O concentrations for ambient as well as for sampled vehicles. The average measured background concentration for N_2O was 0.138 ppm with a single standard deviation of 0.264 ppm. The ambient N_2O concentrations have a single standard deviation approximately equal to twice their average measurement suggesting the analysis method and measurements are near the detection limits of the method or there is a large variability in the ambient N_2O concentrations.

Others show that the ambient concentrations for N_2O are around 0.314 – 0.320 ppm [14,15,16] with a steady increase of about 0.01ppm/decade. Their data suggest the true ambient average is probably closer to 0.325 ppm instead of the average of 0.138 ppm. Also this suggests the measurement method is not sensitive enough to quantify the presence of N_20 neither in the ambient nor the ability to measure source N_2O emissions near the ambient levels.

To examine the N_2O being emitted from vehicle samples, a lower threshold in the results was established and set equal to the average concentration plus one standard deviation (0.402 ppm). This threshold is only slightly higher than the average N_2O ambient concentration predicted by several studies[13,14,15].

The sample concentrations in Table 8-20 through Table 8-24 are presented with this threshold in mind. If the measured average cycle concentration is less than the established threshold no value is displayed and if it is larger than the threshold than a value will be displayed. Not all vehicles were sampled for N_2O analysis due to limited laboratory accessibility. The greyed cells represent test runs that were not analyzed for N_2O analysis.

---

[14] M. Gomes da silva∗, A. mikl´os, A. falkenroth, P. Hess, (2006) Photoacoustic measurement of N2O concentrations in ambient air with a pulsed optical parametric oscillator, Appl. Phys. B 82, 329–336 (2006)

[15] IPCC 2001 Climate Change 2001: The Scientific Basis, Chapter 4 - Atmospheric Chemistry and Greenhouse Gases, Final Report.

[16] European Union (2009) Assessment of N2O concentrations, http://www.eea.europa.eu/data-and-maps/figures/atmospheric-concentration-of-n2o-ppb

N_2O emission factors (g/mi)

The previous tables showed the N_2O concentrations relative to the ambient measured concentration and standard deviation of those values. Additionally the tables showed the calculated N_2O emission rates on a grams per mile basis. The N_2O emissions rates were calculated by correcting for ambient concentrations as shown by equation 2 below:

Equation 2

$$EF = * \frac{V_{mix} * \rho_{N20} * \left(C_i - C_{bk} * \left(1 - \frac{1}{DF} \right) \right)}{Miles}$$

Where:

EF	is the emission factor in g/mi
V_{mix}	volume through the CVS in m^3
ρ_{N20}	density of N_20 from ideal gas law at 1 atm and 20C and molar mass N_2O
C_i	sample concentration
C_{bk}	background concentration
DF	dilution ratio from CVS sampling system
Miles	distance traveled in miles

The N_2O measurements were very close to the ambient concentrations were negative numbers were reported. The reason for negative numbers is based on the correction of the ambient measured concentration exceeding the sample measurement. It is expected that many of the measurements are near the detection limits of the measuring method.

The general observations of the N_2O emissions from the vehicles tested can be summarized as:

- N_2O Analysis was done offsite when facilities were available. As such, not all vehicles or cycles were tested for N_2O. Only selected vehicles were tested for N_2O analysis.
- During the refuse and school bus testing there were no facilities for N_2O analysis thus they were not performed.
- More than half (64%) of the measured toxic emissions were below the defined threshold (0.4 ppm), the average ambient background concentration plus one standard deviation.
- Only the SCR equipped vehicles showed signs of N_2O emissions not the non-SCR equipped vehicles.
- The cold start UDDS did not show higher integrated N_2O emissions compared with hot start UDDS tests (with or with/out SCR). It is not clear from the testing if higher N20 emissions were created for a short duration at the cold start of the cold test cycles. Additional real time N_2O data would be necessary to evaluate the first 100 seconds of the cold start UDDS N_2O emissions.
- Of the values greater than the threshold, the average vehicle sample concentration was 1.06 ppm (only 2.6 times the threshold) and the single standard deviation was 0.44 ppm.
- The N_2O emission rate in mg/mi for port vehicles with higher than threshold concentrations ranged from 1.5 mg/mi to 17 mg/mi where the highest concentrations was

- N_2O emissions appear to be below or near detection limit for diesel and propane vehicles appear operated on the UDDS and port related test cycles.

Table 8-18 Ambient background measured concentration (ppm) and detection limits

Samples	Average	Stdev	95% Conf	Threshold
51	0.138	0.264	0.529	0.402

Table 8-19 Ambient and threshold related emission factors.

	Average Ambient [1]		Average Treashold [2]	
Calc	mg/mi	mg/gfuel	mg/mi	mg/gfuel
ave	1.87	5.59	3.64	10.89
stdev	1.00	3.02	1.92	5.79

[1] data is not corrected as per equation 1

[2] data is corrected for as per equation 1

Table 8-20 Port vehicle Near dock (PDT_1) cycle averaged concentrations

Make	Engine MY	Disp. L	ATS Type	Cycle Name	N20 Concentration ppm	N20 mg/mi ave	N20 mg/mi stdev
Cummins	2010	8.3	SCR	PDT_1	0.58 ± 0.40	4.44	5.28
Cummins	2011	11.9	SCR	PDT_1	0.76 ± 0.40	6.67	8.52
Volvo/Ma	2011	12.8	SCR	PDT_1		-0.20	0.32
Navistar	2011	7.6	Adv EGR	PDT_1			
Navistar	2011	12.4	Adv EGR	PDT_1		-2.03	2.34
GM	2009	8.11	Propane	PDT_1			
Navistar	2009	12.3	Adv EGR	PDT_1		0.69	1.05
DDC	2008	14	DOC/DPF	PDT_1			
DDC	2008	14	DOC/DPF	PDT_1			

[1] concentration values shown represent measurements above ambient concentration plus one standard deviation.

Table 8-21 Port vehicle Near dock (PDT_2) cycle averaged concentrations

Make	Engine MY	Disp. L	ATS Type	Cycle Name	N20 Concentration ppm	N20 mg/mi ave	N20 mg/mi stdev
Cummins	2010	8.3	SCR	PDT_2			
Cummins	2011	11.9	SCR	PDT_2	0.98 ± 0.40	17.94	
Volvo/Ma	2011	12.8	SCR	PDT_2			
Navistar	2011	7.6	Adv EGR	PDT_2			
Navistar	2011	12.4	Adv EGR	PDT_2		0.53	0.75
GM	2009	8.11	Propane	PDT_2			
Navistar	2009	12.3	Adv EGR	PDT_2		0.91	1.04
DDC	2008	14	DOC/DPF	PDT_2			
DDC	2008	14	DOC/DPF	PDT_2			

[1] concentration values shown represent measurements above ambient concentration plus one standard deviation.

Table 8-22 Port vehicle Near dock (PDT_3) cycle averaged concentrations

Make	Engine MY	Disp. L	ATS Type	Cycle Name	N20 Concentration ppm	N20 mg/mi ave	N20 mg/mi stdev
Cummins	2010	8.3	SCR	PDT_3			
Cummins	2011	11.9	SCR	PDT_3	1.71 ± 0.62	5.10	6.01
Volvo/Ma	2011	12.8	SCR	PDT_3		1.55	
Navistar	2011	7.6	Adv EGR	PDT_3			
Navistar	2011	12.4	Adv EGR	PDT_3		-0.74	0.81
GM	2009	8.11	Propane	PDT_3			
Navistar	2009	12.3	Adv EGR	PDT_3		0.33	0.39
DDC	2008	14	DOC/DPF	PDT_3			
DDC	2008	14	DOC/DPF	PDT_3			

[1] concentration values shown represent measurements above ambient concentration plus one standard deviation.

Table 8-23 Port vehicle Near dock (UDDS) cycle averaged concentrations

Make	Engine MY	Disp. L	ATS Type	Cycle Name	N20 Concentration ppm	N20 mg/mi ave	N20 mg/mi stdev
Cummins	2010	8.3	SCR	UDDS	0.72 ± 0.40	2.07	2.33
Cummins	2011	11.9	SCR	UDDS	1.50 ± 0.40	10.66	
Volvo/Ma	2011	12.8	SCR	UDDS		1.33	
Navistar	2011	7.6	Adv EGR	UDDS			
Navistar	2011	12.4	Adv EGR	UDDS		-1.10	
GM	2009	8.11	Propane	UDDS			
Navistar	2009	12.3	Adv EGR	UDDS		0.61	
DDC	2008	14	DOC/DPF	UDDS			
DDC	2008	14	DOC/DPF	UDDS			

[1] concentration values shown represent measurements above ambient concentration plus one standard deviation.

Table 8-24 Port vehicle Near dock (UDDS-CS) cycle averaged concentrations

Make	Engine MY	Disp. L	ATS Type	Cycle Name	N20 Concentration ppm	N20 mg/mi ave	N20 mg/mi stdev
Cummins	2010	8.3	SCR	UDDS-CS	1.51 ± 0.40	4.80	
Cummins	2011	11.9	SCR	UDDS-CS	0.75 ± 0.40	4.90	
Volvo/Ma	2011	12.8	SCR	UDDS-CS		2.49	
Navistar	2011	7.6	Adv EGR	UDDS-CS			
Navistar	2011	12.4	Adv EGR	UDDS-CS		-0.87	
GM	2009	8.11	Propane	UDDS-CS			
Navistar	2009	12.3	Adv EGR	UDDS-CS		0.36	
DDC	2008	14	DOC/DPF	UDDS-CS			
DDC	2008	14	DOC/DPF	UDDS-CS			

[1] concentration values shown represent measurements above ambient concentration plus one standard deviation.

8.4.4 Discussion of NH_3 limits

The measurement of NH_3 for a properly operating SCR equipped diesel vehicle can be close to that of a non-SCR equipped diesel vehicle. Thus, it was necessary to describe the measurement system to prevent misinterpreting the meaning of emissions at or below the detection limits of

the TDL measurement method. This analysis follows what was performed for the other non-regulated emissions.

This section describes the limits of detection for UCR's NH_3 measurement via a tunable diode laser (TDL) instrument. Past experience shows that NH_3 measurements are difficult and can be influenced with ambient NH_3 concentrations, water, and other sampling issues. The TDL has been configured to show low water interference (less than 1 ppm) and is calibrated on a per-test basis using a span of 40ppm and a zero of 5 ppm dry NH_3 concentrations. Zero ppm zero is not used since the correlation to the reference cell at zero is below 40% and is thus a non-representative value. A zero of 5 ppm was utilized as the zero point where a correlation coefficient of more than 80% was achieved. The upper span point was 100 ppm were the correlation coefficient was greater than 99%.

During the course of this measurement program several ambient tunnel background checks were performed to determine an appropriate measurement detection limit defined as the lower detection limit (LDL) for NH_3 system. Table 8-25 shows the average and standard deviation of 25 ambient samples collected with the TDL instrument over the two year testing program. The sum of the average bias and the standard deviation is used as the LDL. The LDL for the measurement program and typical usage is approximatly1 ppm. This agrees with our expectation that the TLD measurement is good to about 1 or 2 ppm.

Table 8-25 Measured ambient concentration during NH_3 back ground checks

Descritiption	Value	Units
N	25	#
ave	0.23	ppm
stdev	0.81	ppm
ave+stdev	**1.04**	ppm

[1] LDL defined as ave+1stdev or 1 ppm for the NH3 measurement

Table 8-26 shows of the 54 diesel tests conducted, only 2 were 5 times the LDL (i.e. greater than 5ppm), and 26 tests were above 2 time the LDL (2 ppm). Of the 2 tests above 5*LDL, both were for a cold start SCR equipped diesel vehicle. For the 26 tests above 2* LDL these were both SCR and non-SCR equipped vehicles. It is not expected that a non-SCR equipped vehicle had more NH_3 emissions than an SCR equipped vehicle.

Table 8-26 Diesel vehicles NH_3 concentration in relationship to NH_3 ambient background

All Tests	> 1* LDL	> 2*LDL	> 5*LDL	> 10 * LDL
54	43	26	2	0

Five of seven tests for the propane vehicle also had NH_3 greater than 5 ppm and 2 were over 50 ppm suggesting very high relative NH_3 emissions for the propane vehicles.

8.4.5 Discussion of EC/OC limits

This section describes the element and organic carbon (EC and OC) mass accumulation measurement and detection limits in relationship to measured detection limits to help understand the limitations in making un-regulated emission measurements.

This section describes the elemental and organic carbon PM emission results in relationship to measured detection limits to help understand the limitations in making un-regulated PM emission measurements. Table 8-28 through

show the average elemental and organic carbon filter loadings for all the vehicles tested. Table 8-27 shows the ambient tunnel blank results from the AQMD test program. The average measured background concentration for elemental and organic carbon was 0.5 µg/filter and 10 µg/filter respectively with a standard deviation near the measured levels, see Table 8-27. These tunnel blanks agree with previous research performed with the MEL.

The filter weight gain for EC and OC are not completely dependent on the sample volume through the filter, but are more subjective to handling and traveling to from the laboratory. Higher volumes through the filter do not necessarily produce larger tunnel blanks as has been demonstrated during other research projects. This suggests the EC/OC measurements (similar also for total PM) are a function of the mass on each filter tested. Thus, the detection limits were considered on a µg/filter basis and not a µg/liter. Since the standard deviation was near the average value, the estimated detection limits were set at 1 µg/filter and 20 µg/filter for EC and OC respectively.

To examine the elemental and organic carbon being emitted from vehicle samples, a lower threshold in the results was established and set equal to the average concentration plus one standard deviation. Table 8-27 shows the lower threshold for EC and OC at 1 µg/filer and 10 µg/filter respectively.

The filter weights in Table 8-28 through Table 8-34 are presented with in relationship to the threshold. If the measured average cycle filter weight is less than the established threshold no value is displayed and if it is larger than the threshold than a value is displayed. Cells with no color are less than two times the threshold, green is less five times, orange less than ten times, and red is more than ten times.

The general observations of the carbon and elemental emissions from the vehicles tested can be summarized as:

- More than half (69% for both) of the measured EC and OC emissions were below a defined threshold of the average ambient background concentration plus one standard deviation.
- Of the EC values over the threshold, 17% of those were less than two times the threshold. About 50% were between two and five times, 14% between five and ten times and only 17% were above ten times the threshold for EC.
- Of the OC values over the threshold, 65% of those were less than two times the threshold. About 30% were between two and five times, 0% between five and ten times and only 5% were above ten times the threshold for EC.

Table 8-27 Ambient background filter mass and estimated detection limits

Average Tunnel Blank		Estimated Detection Limits	
EC	OC	EC	OC
ug/filter	ug/filter	ug/filter	ug/filter
0.5 ± 0.5	10.1 ± 9.9	1	20

Table 8-28 Port vehicle Near dock (PDT_1) cycle averaged filter mass

Test Article Engines				Average Elemental and Organic Carbon Concentrations µg/filter					
Make	MY	Disp	ATS	EC			OC		
C8.3	2010	8.3	SCR						
C11.9	2011	11.9	SCR				10.1	±	10.0
V12.8	2011	12.8	SCR						
N7.6	2011	7.6	Adv EGR						
N12.4	2011	12.4	Adv EGR						
GM8.1	2009	8.11	Propane	14.3	±	12.4	47.7	±	41.5
N12.3	2009	12.3	Adv EGR	1.4	±	1.0	12.2	±	10.0
D14a	2008	14	DOC/DPF						
D14b	2008	14	DOC/DPF						

Table 8-29 Port vehicle Near dock (PDT_2) cycle averaged filter mass

Test Article Engines				Average Elemental and Organic Carbon Concentrations µg/filter					
Make	MY	Disp	ATS	EC			OC		
C8.3	2010	8.3	SCR				10.0	±	10.0
C11.9	2011	11.9	SCR						
V12.8	2011	12.8	SCR				12.0	±	10.0
N7.6	2011	7.6	Adv EGR				10.3	±	10.0
N12.4	2011	12.4	Adv EGR						
GM8.1	2009	8.11	Propane	39.2	±	35.6	57.2	±	51.5
N12.3	2009	12.3	Adv EGR				10.4	±	10.0
D14a	2008	14	DOC/DPF	2.0	±	2.8	16.0	±	15.7
D14b	2008	14	DOC/DPF				14.1	±	12.4

Table 8-30 Port vehicle Near dock (PDT_3) cycle averaged filter mass

Test Article Engines				Average Elemental and Organic Carbon Concentrations µg/filter					
Make	MY	Disp	ATS	EC			OC		
C8.3	2010	8.3	SCR	4.9	±	1.7	19.0	±	10.0
C11.9	2011	11.9	SCR						
V12.8	2011	12.8	SCR	6.6	±	4.6	26.9	±	10.0
N7.6	2011	7.6	Adv EGR				24.3	±	10.0
N12.4	2011	12.4	Adv EGR						
GM8.1	2009	8.11	Propane	92.4	±	81.0	58.8	±	51.0
N12.3	2009	12.3	Adv EGR				10.9	±	10.0
D14a	2008	14	DOC/DPF	8.8	±	2.3	26.4	±	10.0
D14b	2008	14	DOC/DPF				20.4	±	10.0

Table 8-31 Port vehicle Near dock (UDDS) cycle averaged filter mass

Test Article Engines				Average Elemental and Organic Carbon Concentrations µg/filter					
Make	MY	Disp	ATS	EC			OC		
C8.3	2010	8.3	SCR	1.6	±	1.0	16.7	±	10.0
C11.9	2011	11.9	SCR				10.7	±	10.0
V12.8	2011	12.8	SCR				10.8	±	10.0
N7.6	2011	7.6	Adv EGR				13.0	±	11.3
N12.4	2011	12.4	Adv EGR				11.9	±	10.8
GM8.1	2009	8.11	Propane	92.4	±	81.0	58.8	±	51.0
N12.3	2009	12.3	Adv EGR				10.9	±	10.0
D14a	2008	14	DOC/DPF	8.8	±	2.3	26.4	±	10.0
D14b	2008	14	DOC/DPF				20.4	±	10.0

Table 8-32 Port vehicle Near dock (UDDS-CS) cycle averaged filter mass

Test Article Engines				Average Elemental and Organic Carbon Concentrations µg/filter					
Make	MY	Disp	ATS	EC			OC		
C8.3	2010	8.3	SCR	2.2	±	2.2	18.8	±	18.8
C11.9	2011	11.9	SCR	1.9	±	1.9			
V12.8	2011	12.8	SCR	3.1	±	3.1	24.6	±	24.6
N7.6	2011	7.6	Adv EGR				29.2	±	29.2
N12.4	2011	12.4	Adv EGR				10.4	±	10.4
GM8.1	2009	8.11	Propane	63.1	±	63.1	835.8	±	835.8
N12.3	2009	12.3	Adv EGR	2.5	±	2.5	10.5	±	10.5
D14a	2008	14	DOC/DPF	2.2	±	2.2	27.0	±	27.0
D14b	2008	14	DOC/DPF				15.84	±	15.8

Table 8-33 Bus vehicle cycle averaged filter mass

Test Article Engines				Average Elemental and Organic Carbon Concentrations µg/filter			
Make	MY	Disp	ATS	EC	OC		
GM8.1	2008	8.1	LPG		31.166	±	10
GM8.1	2008	8.1	LPG				
C6.7	2007	6.7	DOC/DPF		13.449	±	10
C6.7	2007	6.7	DOC/DPF				

Table 8-34 Refuse vehicle cycle averaged filter mass

Test Article Engines				Average Elemental and Organic Carbon Concentrations µg/filter			
Make	MY	Disp	ATS	EC	OC		
	2012	8.3	SCR		17.018	±	18
	2011	9.3	SCR		16.159	±	10
	2011	7.6					
	2008	7.6			22.011	±	10

9 Summary

The SC AQMD path to cleaner air depends on achieving the strictest NOx standards for heavy-duty vehicles. Recently, the District saw data that indicated the in-use emissions exceeded the certification values. The University of California, Riverside (UCR) was contracted to test 16 heavy-duty vehicles, mainly diesel fueled engines, used for goods movement, refuse hauling and transit applications. The testing protocol involved measuring the emissions identified in the RFP while the vehicles operated following driving cycles that better represented the in-use conditions as well as certification conditions. The testing measured: 1) regulated emissions; 2) unregulated emissions such as ammonia and formaldehyde; 3) greenhouse gas levels of CO_2 and N_2O and 4) ultrafine PM emissions. A number of vehicles and engines were tested based on the population, emission standards and technology.

The emission results for PM and NO_x are summarized below:

- PM emissions from the diesel test vehicles were below 0.01 grams per brake horsepower-hour (g/bhp-h) measured over port drayage, CBD, and UDDS drive cycles. Cold start PM emissions were relatively high for two diesel vehicles; one was a port SCR equipped vehicle and the other was a refuse SCR equipped vehicle. The port vehicle was 17 times higher (22.9 mg/mi vs 1.33 mg/mi) and the refuse vehicle was 8 times higher (18.4 mg/mi vs 2.75 mg/mi). In both cases the high cold start emission factors were below the certification standard. PM emissions were well below the certification for all diesel tests, thus suggesting DPF-based solutions are robust and reliable in meeting targeted standards. In addition, PM emissions from a liquefied petroleum gas (LPG) test vehicle was approximately 0.14 g/bhp-hr measured over the UDDS cycle, which is above the certification standard.
- NO_x results covered a wide range of emission factors, where the emissions depended on the certification standard, vehicle application, driving cycle, and manufacturer. For example, NO_x emissions were lowest for goods movement vehicles powered by diesel engines equipped with SCR technology; however, increases from 0.112 g/mi (0.028 g/bhp-h) during high speed cruise operation to 5.36 g/mi (1.34 g/bhp-h) for low speed transient operation were measured. Unique to the high NO_x emissions was a condition in which the temperature of the SCR was less than 250°C. Advanced EGR 2010 certified engines showed higher NO_x emissions compared to SCR equipped engines, and pre-2010 certified engines were higher than the 2010 certified engines.
- The NO_x impact of SCR equipped diesel engines depends on the vehicles' duty cycles and manufacturers' implementation for low temperature SCR performance. For the near dock port cycle, the SCR was below 250°C approximately 80% of the time, 65% of the time for the local port cycle, and approximately 45% of the time for the regional port cycle. The percentage of time below 250°C varied significantly between manufacturers, from 8% to 30% for the near dock cycle, and from 41% to 64% for the regional cycle. The difference in time below 250°C suggests some manufacturers have better strategies for maintaining high exhaust temperatures than others.
- The SCR equipped engines were within their certification standards and were typically below 0.2 g/bhp-h. Only during low SCR temperature were the emissions found to be higher than the certification standard. In-use compliance testing does not enforce the

emissions standards when the SCR is below 250 °C, thus the SCR equipped vehicles were typically compliant based on the results presented in this report.

- Cold start NO_x emissions can be as high as 2.3 g/bhp-hr compared to an equivalent warm test of 0.006 g/bhp-h. Although cold start emissions do not contribute to the inventory, it is important to consider the extreme nature of cold start emissions if vehicles are allowed to cool frequently. The NO_x emissions accumulated in 1 mile after a cold start were equivalent to emissions accumulated during 32 miles of running hot.
- The 2010 certified diesel engines with advanced cooled EGR and no SCR were tested. These vehicles operated utilizing a lug curve with peak torque starting as low as 1000 revolutions per minute (RPM), where the driver was instructed to operate the vehicle down to 900 RPM before shifting. The truck behavior was unusual, and both UCR and WVU trained drivers commented on the strange operation. Additionally, the certified emissions had a family emission limit (FEL) of 0.5 g/bhp-hr for 2010 MY, but the measured NO_x emissions were around 1 g/bhp-hr (0.25 g/mi) for the UDDS cycle, which represents a certification-like cycle. Even the port cycles showed brake specific emissions higher than 1 g/bhp-hr and as high as 2 g/bhp-hr for the near dock cycle.
- Pre-2010 certified diesel engines exhibited regulated emissions that were very close to the standard and were found to be repeatable for randomly selected models tested. This suggests that pre-2010 emissions inventories may be more reliable than SCR-equipped diesel engines due to SCR performance variability.
- Most NO_x emissions from SCR equipped diesel refuse vehicles were produced during the compaction portion of the in-use test cycle. The high NO_x emissions corresponded with a low SCR exhaust temperature, where the emissions increased from 0.27 g/bhp-hr NO_x for the transient and curbside cycles to 3.8 g/bhp-hr NO_x for the compaction cycle.
- The percentage of NO_x as NO_2 ranged from 10% to near 90%, with the highest levels of NO_2 emissions from non-SCR-equipped diesel vehicles. NO_2 was highest for the pre-2010 certified engines (averaging 1.15 ± 0.48 g/mi for the UDDS cycle). In general NO_2 ratios were similar for all tests at around 45%±8%, except for the SCR equipped diesel vehicles, which showed high variability with a NO_2 ratio of 47%±36%.

The emission results for ammonia, hydrocarbons, toxics, and fine particles are summarized below:

- Ammonia (NH_3) emissions from the vehicles tested ranged from about 0.01 to 0.1 g/mi. The diesel vehicles' NH_3 emissions averaged 0.04±0.03 g/mi (0.01±0.01 g/hp-h), where the port vehicle emissions were similar (0.03±0.02 g/mi), but the propane school bus had relatively higher NH_3 emissions (0.48±0.04 g/mi) over the CBD test cycle. All the diesel vehicles showed cycle averaged raw NH_3 emission concentrations less than 10ppm. Of the 54 diesel tests conducted, only 2 vehicles had NH_3 emissions over 5 parts per million (ppm). Half of the tests were below 2 ppm. Five of seven propane vehicle tests had NH_3 emissions greater than 5 ppm and two were over 50 ppm, suggesting that relatively higher NH_3 emissions exist for the propane vehicles compared to the diesel vehicles.
- The emission factors for total hydrocarbon (THC), methane (CH_4), non-methane hydrocarbon (NMHC) and toxics were very low for all diesel vehicles tested. This agrees

with other research from the Advanced Collaborative Emissions Study (ACES) project that showed a 98% reduction from diesel engines with catalytic exhaust systems. THC, NMHC, and CH_4 emissions were at or below 0.09 g/mi, 0.06 g/mi, and 0.04 g/mi, respectively, for all vehicles (except the LPG vehicle) for both the UDDS and port regional cycles. Slightly higher THC, CH_4, and NMHC emissions were found for the lower power near dock port cycle (0.36 g/mi, 0.10 g/mi, and 0.29 g/mi, respectively). Toxic emissions were low and near the detection limits of the method where 75% of the measured carcinogenetic species (benzene, toluene, ethylbenzene, and xylenes - BTEX) were below the average ambient background concentration pulse one standard deviation (< 10 mg/mi and typically < 2 mg/mi background corrected). Carbonyl emissions were also low relative to the measurement method, where more than 75% of the measured species were below the same threshold except for formaldehyde. Formaldehyde showed a relatively higher emission concentration, with 75% of the measurements above the threshold. Even though the formaldehyde samples were relatively high, their absolute contribution were below 72 mg/mi, with an average of 18±19 mg/mi. Acetaldehyde was the next largest carbonyl with maximum emissions of 18 mg/mi and an average of 1.5±4 mg/mi. The rest of the carbonyls were below 2 mg/mi. Cold start UDDS emissions were similar to the hot start UDDS emissions for THC, CH_4, NMHC, and toxics (note the UDDS was performed as a 2xUDDS cycle, which may have minimized the cold start effect for the HCs and toxics).

- The LPG goods movement vehicle showed higher THC, NMHC, CH_4, and toxic emissions than the diesel vehicles tested. THC, NMHC and CH_4 were 22.4 g/mi, 1.43 g/mi, and 21.4 g/mi respectively for the UDDS hot cycle. BTEX and formaldehyde samples were more than 10 times the average ambient background concentration plus one standard deviation. The propane vehicle averaged 6.5±9.3 mg/mi, 9.7±12 mg/mi, and 22.4±19 mg/mi for 1,3-butadiene, n-butane, and benzene respectively for the BTEX sample. The Carbonyls were high for formaldehyde and acetaldehyde (241±253 mg/mi and 42±48 mg/mi respectively) with the remaining aldehydes below 2 mg/mi. These results should be confirmed with additional testing on LPG port vehicles.

- Real-time PM measurements suggest the reported reference PM emission rate may be lower due to low filter weights for DPF equipped vehicles. The PM mass of the gravimetric method averaged 0.78±1.57 mg/bhp-hr for selected diesel vehicles. The average PM mass from the real-time measurement method averaged 0.05±0.09 mg/bhp-hr for the same vehicles. The average filter weight for these selected vehicles ranged from 10-20 µg, where UCR's CVS tunnel blank averages were 5µg with a 5µg single standard deviation. Thus, there is speculation that some of the uncertainty may be artifacts on the filter. As such, real-time PM measurements are useful for identifying low level PM mass in addition to real-time analysis.

- Elemental carbon (EC) and organic carbon (OC) PM was very low for all the vehicles tested and was typically below 0.2 mg/mi and 2.2 mg/mi respectively. More than half (69%) of the measured EC and OC emissions were below the average ambient background concentration plus one standard deviation. The propane vehicles had the highest organic PM contribution (>10 mg/mi for the near dock port cycle).

- Fine-particle emissions were typically higher during the first 200 seconds of the cold start UDDS cycle compared to the hot stabilized UDDS cycle ($5x10^5$ #/cc vs $1x10^3$ #/cc,

respectively). The fine particle emissions appear to be higher for the regional port cycle compared to the near dock, local, and UDDS cycles ($8x10^4$ #/cc vs $1x10^3$ #/cc, respectively). The higher concentration of the regional port cycle may be a result of higher ATS temperatures and possible passive regenerations.

The results for greenhouse gas emissions and fuel economy are summarized below:

- The greenhouse gases (GHG) and fuel economy are characterized by CO_2 emissions for the diesel vehicle, but with the LPG truck, methane emissions represented approximately 8% of the GHG. The diesel fuel economy averaged 3.5 mi/gal (Port 1, 2 and UDDS) to 5.06 mi/gal (Port 3) for the port vehicles, 7.0 mi/gal for the school buses, and 4.2 mi/gal (UDDS) to 2.0 mi/gal (RTC) for the refuse haulers. The regional cycle (Port 3) showed 20% higher fuel economy than the more transient Port 1, 2, and UDDS cycles. The fuel economy from the refuse trash cycle (with integrated compaction phase) was about 50% lower than the transient UDDS cycle. The propane port vehicle showed 19% lower fuel economy than the diesel vehicles (3.3 mi/gal).
- The project measured N_2O greenhouse gases on selected tests. For those vehicles measured more than half (64%) of the N_2O emissions were below 0.4 ppm, which is the average ambient background concentration plus one standard deviation. The emission factors averaged 3.6±1.9 mg/mi with a maximum of 18 mg/mi (Cum_11.9 near dock port cycle).

The results for cross laboratory check are summarized below:

- The work comparison averaged around 3% negative bias (-3%), where UCR's laboratory was slightly lower than WVU's, with a spread of -9% to +4% on average. Both WVU and UCR show very low test-to-test variability, with a coefficient of variation (COV) less than 2% for all tests.
- The $bsCO_2$ was close and averaged around 5% positive bias, where UCR's laboratory was slightly higher than WVU's with a spread of 0% to 10% overall. Both WVU and UCR show very low test-to-test variability, with a COV less than 3% for all tests.
- The $bsNO_x$ correlation was also good, but the comparison varied for the SCR equipped vehicles due to the low emission levels and the variable conditions of the SCR. For the non-SCR equipped vehicles, the deviation averaged about 3% positive bias, where UCR's laboratory was slightly higher than WVU's, with an average of -2% to 8%. The NO_x correlation was poor for the cold start SCR equipped vehicles and for two refuse haulers due to variability in the aftertreatment systems.

In summary, the data from this study suggests that 2010 compliant SCR-equipped HDD vehicles are exhibiting high in-use NO_x emissions that can be as high as 2 g/hp-h under low load conditions represented by short trips or frequent stops. The cause of the high NO_x emissions appears to be low load exhaust temperatures and, thus, low SCR aftertreatment temperatures. For SCR-equipped diesel engines, some accounting of vehicle duty cycle and SCR exhaust temperature is needed to properly characterize NO_x inventories. Additionally, there were differences in SCR performance that varied between manufacturers, suggesting future performance will continue to vary. The ratio of NO_2 in the NO_x has been demonstrated to be about 45% for all diesel vehicles tested, where there is more variability with the SCR equipped diesels. Both NO_x emission factors and NO_2 ratios suggest NO_x emissions are more variable for SCR equipped diesels compared to non-SCR equipped diesel vehicles. This also suggests activity

studies are needed to assess the impact of SCR performance on NO_x inventories. Other results showed the diesel PM, CO, THC, and selected toxics were all very low, well below certification limits, and near the limits of the measurement method for all the tests performed. The low PM, CO, THC, and selected toxics for all the diesel vehicles tested suggest these emissions are well controlled. Looking ahead, the overall results suggest NO_x emissions are still a concern for selected activities, and SCR performance needs to be investigated during wide in-use, on-road operation to characterize its impact on local inventories.

Attachment A. Test Cycles

Central Business District (CBD)

The Central Business District (CBD) Cycle is a chassis dynamometer testing procedure for heavy-duty vehicles (*SAE J1376*). The CBD cycle represents a "sawtooth" driving pattern, which includes 14 repetitions of a basic cycle composed of idle, acceleration, cruise, and deceleration modes. The following are characteristic parameters of the cycle:

- Duration: 560 s
- Average speed: 20.23 km/h
- Maximum speed: 32.18 km/h (20 mph)
- Driving distance: 3.22 km
- Average acceleration: 0.89 m/s^2
- Maximum acceleration: 1.79 m/s^2

Vehicle speed over the duration of the CBD cycle is shown in Figure A-1.

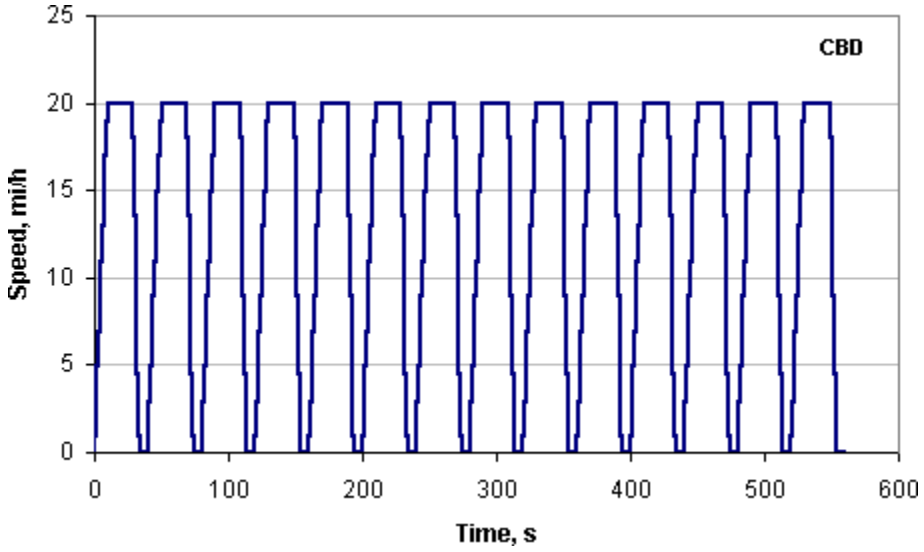


Figure A-1. CBD Driving Cycle

The standard CBD test cycle will be used for bus testing where three cycles will be combined for a triple CBD for a total sample time of 30 minutes. Performing the CBD cycle three times in one test allows for additional sample volumes to be collected for all batched type analysis (filters, DNPH, BETEX and N_2O). Preconditioning is defined as performing a previous triple CBD and a 20 minute soak to improve repeatability between hot repeats. Emissions analyses for gaseous emissions will also be collected over the triple CBD cycles. This cycle is shown in Figure A-2. The triple CBD cycle will be repeated in triplicate for repeatability metrics as described earlier.

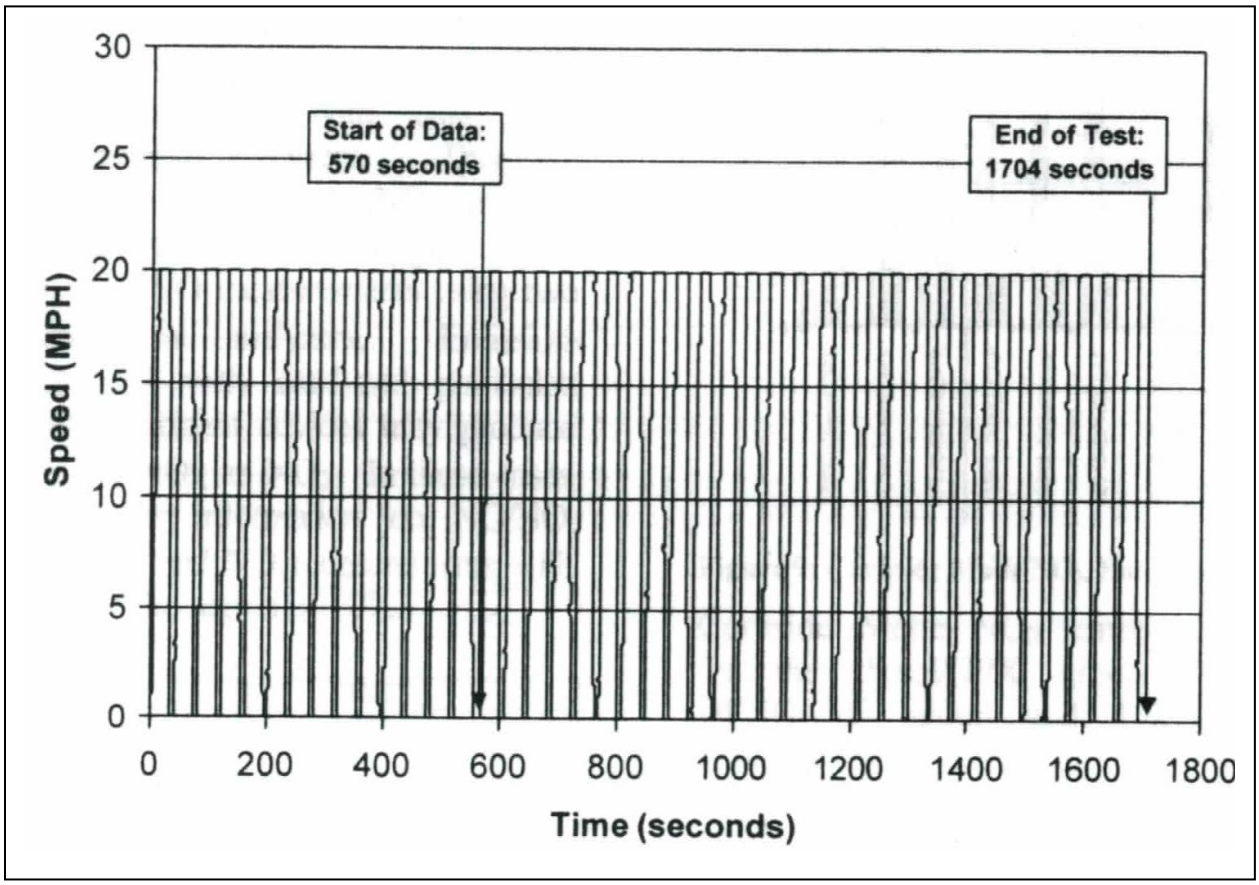


Figure A-2. Triple CBD Cycle

AQMD refuse truck cycle

The waste haulers cycle will be tested using the AQMD refuse truck cycle (AQMD-RTC). This cycle was developed by West Virginia University to simulate waste hauler operation and is a modification of the William H. Martin Refuse Truck Cycle. The original William H. Martin (WHM) refuse truck cycle was created from data logged from sanitation trucks operating in Pennsylvania. The modified cycle consists of a transport segment (phase 1), a curbside pickup segment (phase 2), and a compaction segment (phase 3), see Figure A-2. The modified cycle will be used for this study since this represents the operation of refuse haulers in the SC AQMD district.

The transient phase starts runs for 538 seconds, the curbside phase runs fro 1591 seconds where it starts at 539 and ends at 2130 seconds. The final phase is a compaction cycle that runs from 2201 to 2961 and is 760 seconds long.

The compaction load is simulated by applying a predetermined torque to the drive axel while maintaining a fixed speed of 30 mph. Previous studies by WVU have used an engine load varying between 20 hp to 80 hp for the compaction load, as shown in the right hand side of Figure A-2. To perform the compaction cycle the vehicle is accelerated up to 30 mph where no emissions are collected. Once steady state load conditions are achieved the emissions collection starts and then the varying load is applied. The emissions collection stops before the vehicle is decelerated back to zero speed.

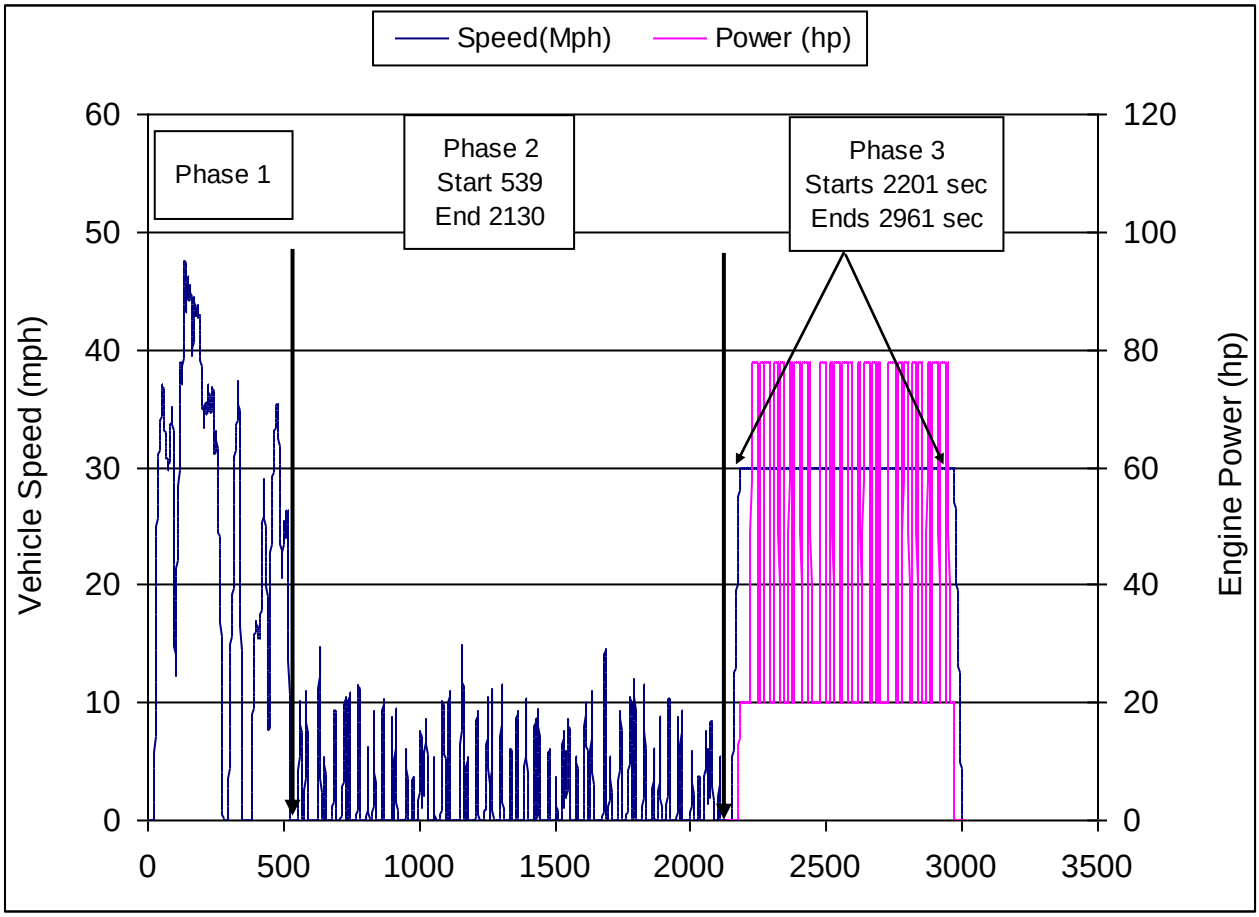


Figure A-3. AQMD Refuse Truck Cycle (AQMD-RTC)

Drayage Truck Port (DTP) cycle

The port cycle was developed by TIAX, the Port of Long Beach and the Port of Los Angeles. Over 1,000 Class 8 drayage trucks at these ports were data logged for trips over a four-week period in 2010. Five modes were identified on the basis of several driving behaviors average speed, maximum speed, energy per mile, distance, and number of stops. These behaviors are associated with different driving conditions such as queuing or on-dock movement, near-dock, local or regional movement, and highway movements. The data were compiled and analyzed to generate a best fit trip. The best-fit trip data was then additionally filtered (eliminating accelerations over 6 mph/s) to allow operation on a chassis dynamometer. The final driving schedule is called the drayage port tuck (DPT) cycle and is represented by 3 modes where each mode has three phases to best represent near dock, local, and regional driving as shown in Table A-5 and Figure A-3. Figure A-4 shows the preconditioning cycles that will be performed for the first test of the day. This will be accomplished after warming up the vehicle and chassis dyno.

Table A-1. Drayage Truck Port cycle by mode and phases

Description	Distance mi	Ave Speed mph	Max Speed mph	Phase 1	Phase 2	Phase 3
Near-dock	5.61	6.6	40.6	Creep	Low Speed Transient	Short High Speed Transient
Local	8.71	9.3	46.4	Creep	Low Speed Transient	Long High Speed Transient
Regional	27.3	23.2	59.3	Creep	Low Speed Transient	High Speed Cruise

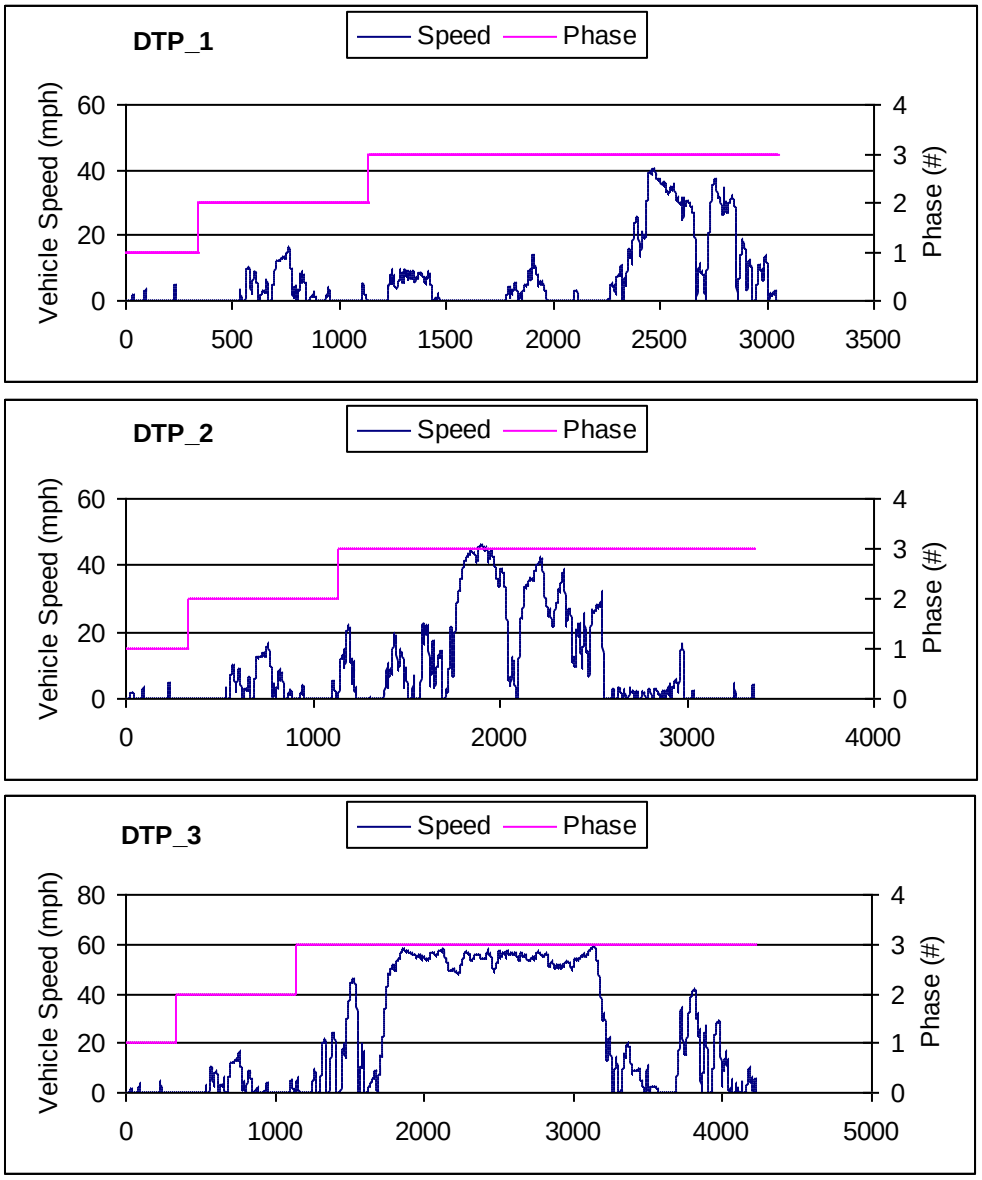


Figure A-4 Drayage truck port cycle near dock (DTP_1), local (DTP_2), and regional (DTP_3)

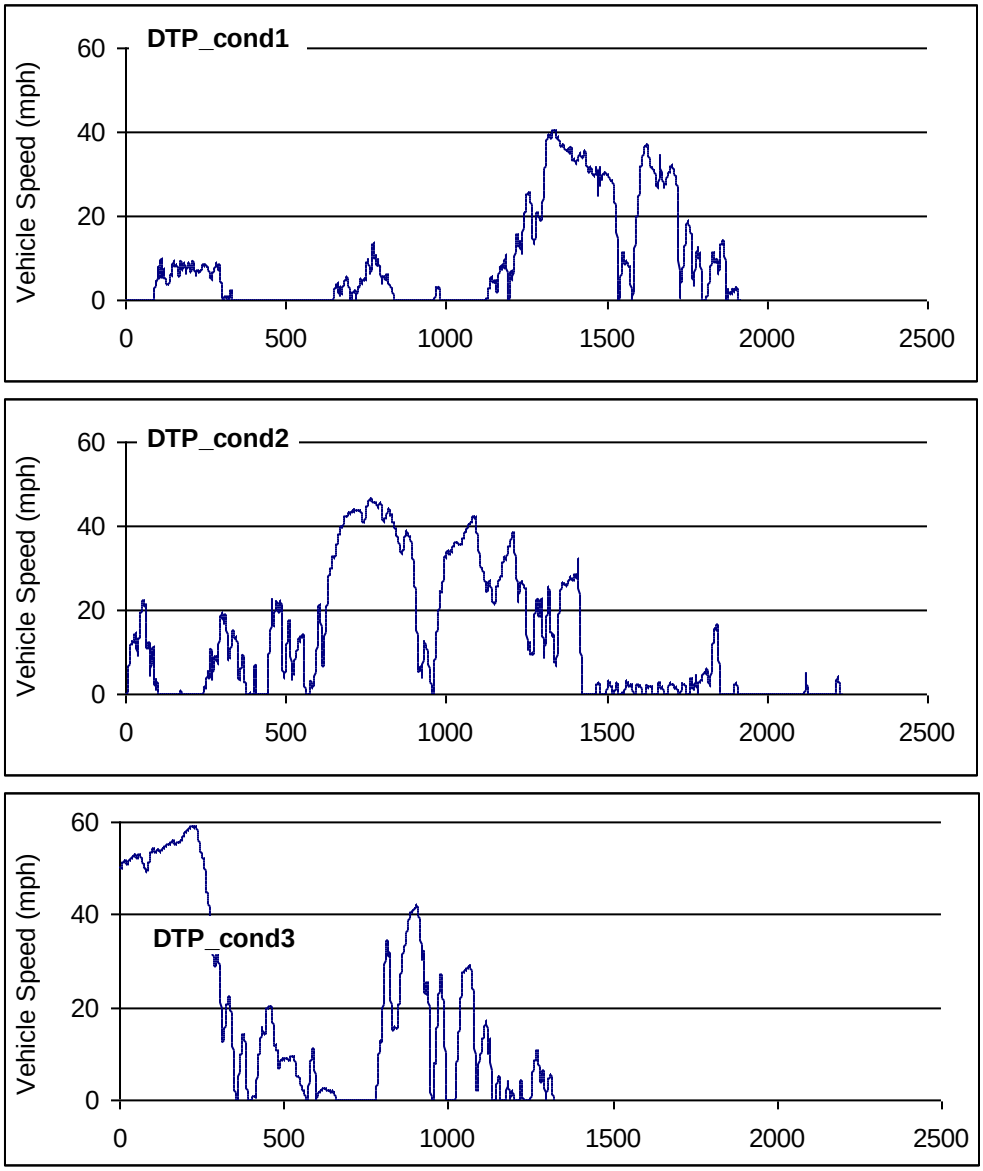


Figure A-5 Drayage truck port cycle conditioning segments consisting of phase 3 parts

UDDS Description

The Federal heavy-duty vehicle Urban Dynamometer Driving Schedule (UDDS) is a cycle commonly used to collect emissions data on engines already in heavy, heavy-duty diesel (HHD) trucks. This cycle covers a distance of 5.55 miles with an average speed of 18.8 mph, sample time of 1061 seconds, and maximum speed of 58 mph. This cycle will be performed as a double UDDS (2xUDDS) to collect sufficient sample for the batched media (exg. PM, BTEX, and DNPH) where the total sample time will be 2122 seconds. The 1x speed/time trace for the UDDS is provided below in Figures A-5.

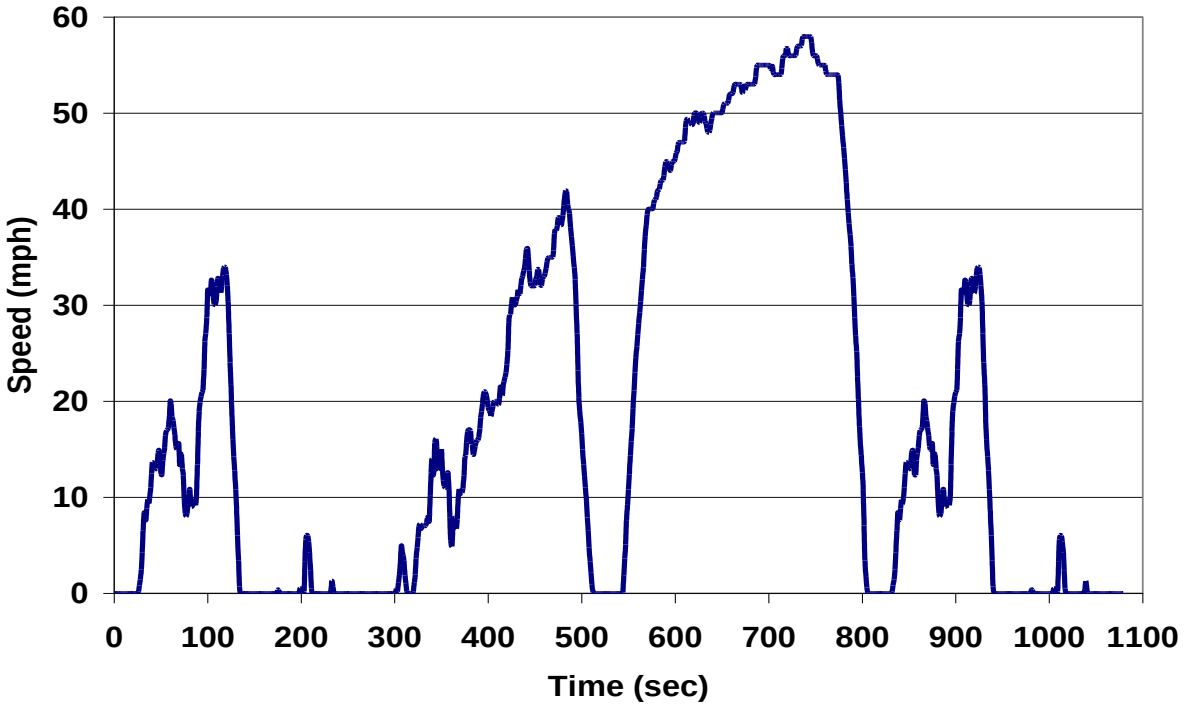


Figure A-5. Speed/Time Trace for a 1xUDDS cycle for the chassis dynamometer.

Attachment B: Brake Specific Emissions

This attachment includes the brake specific emission for all the vehicles tested. They are organized by emissions species.

NO_x Emissions Goods Movement

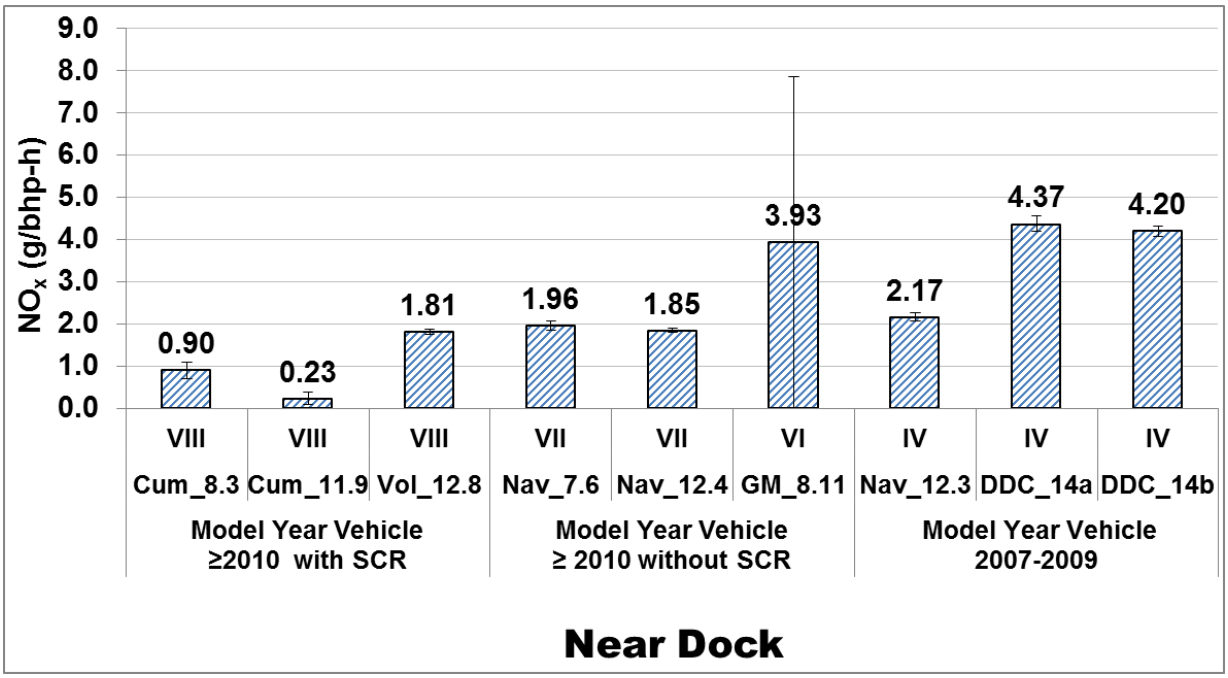


Figure B-1 Goods movement vehicle brake specific emissions

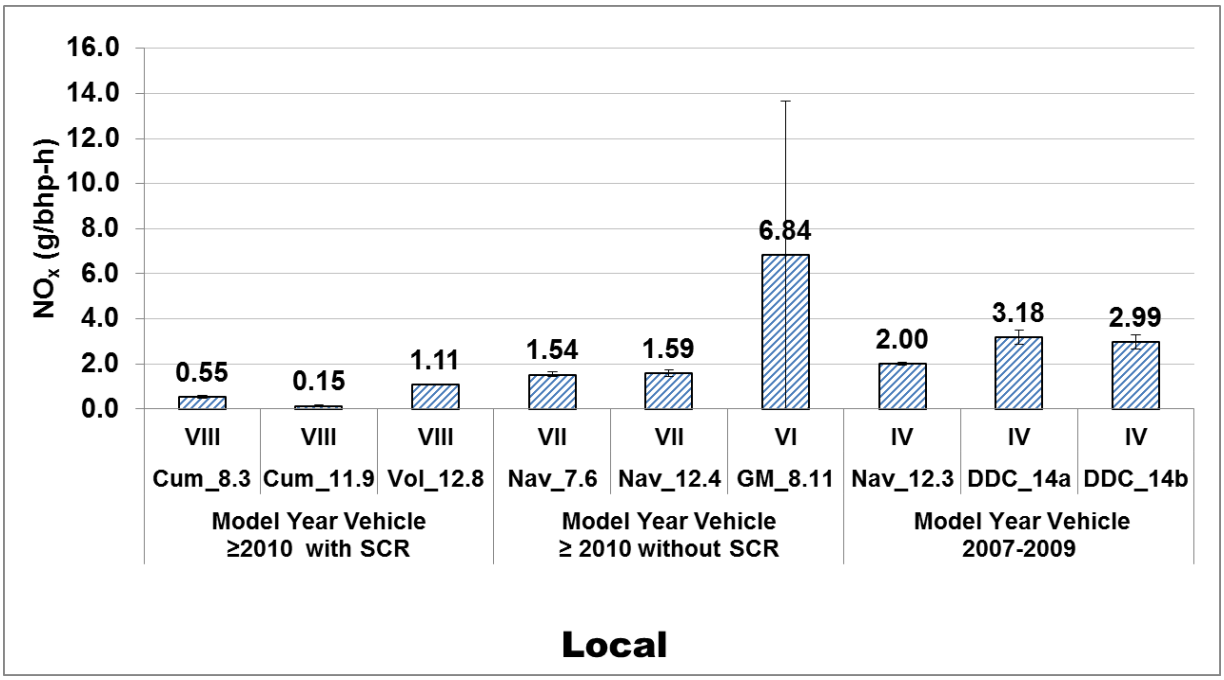


Figure B-2 Goods movement vehicle brake specific emissions

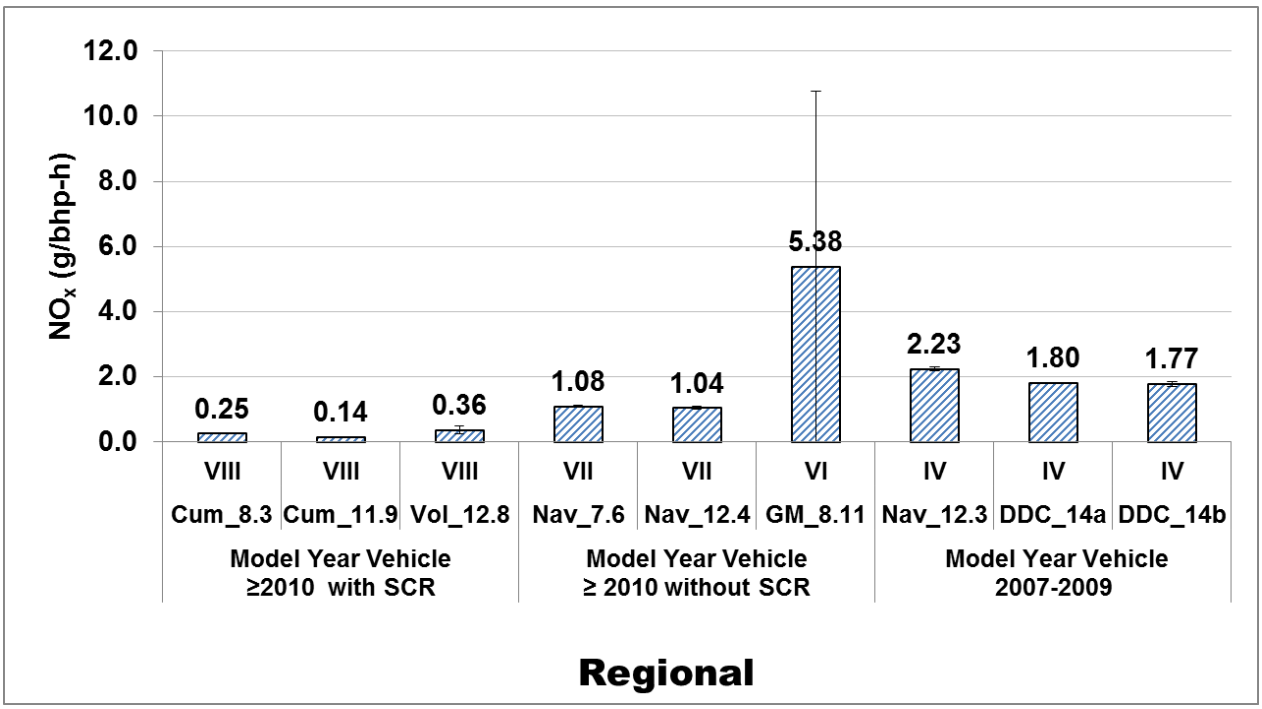


Figure B-3 Goods movement vehicle brake specific emissions

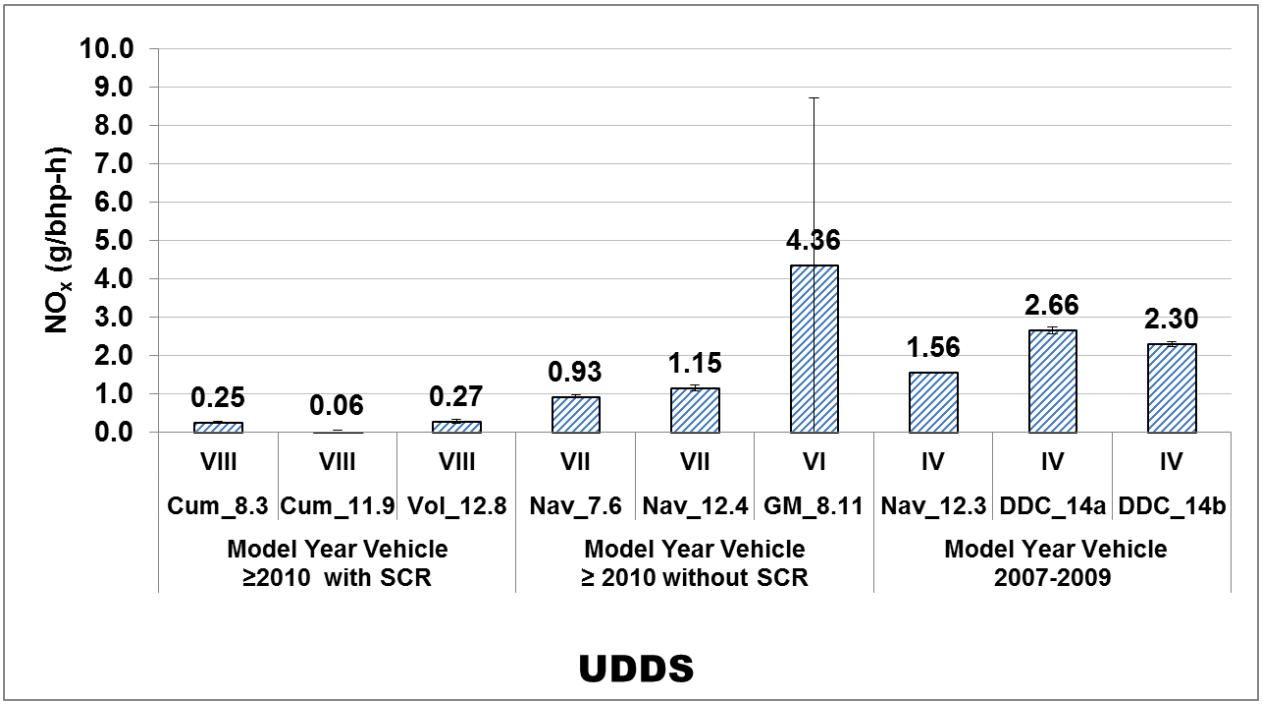


Figure B-4 Goods movement vehicle brake specific emissions

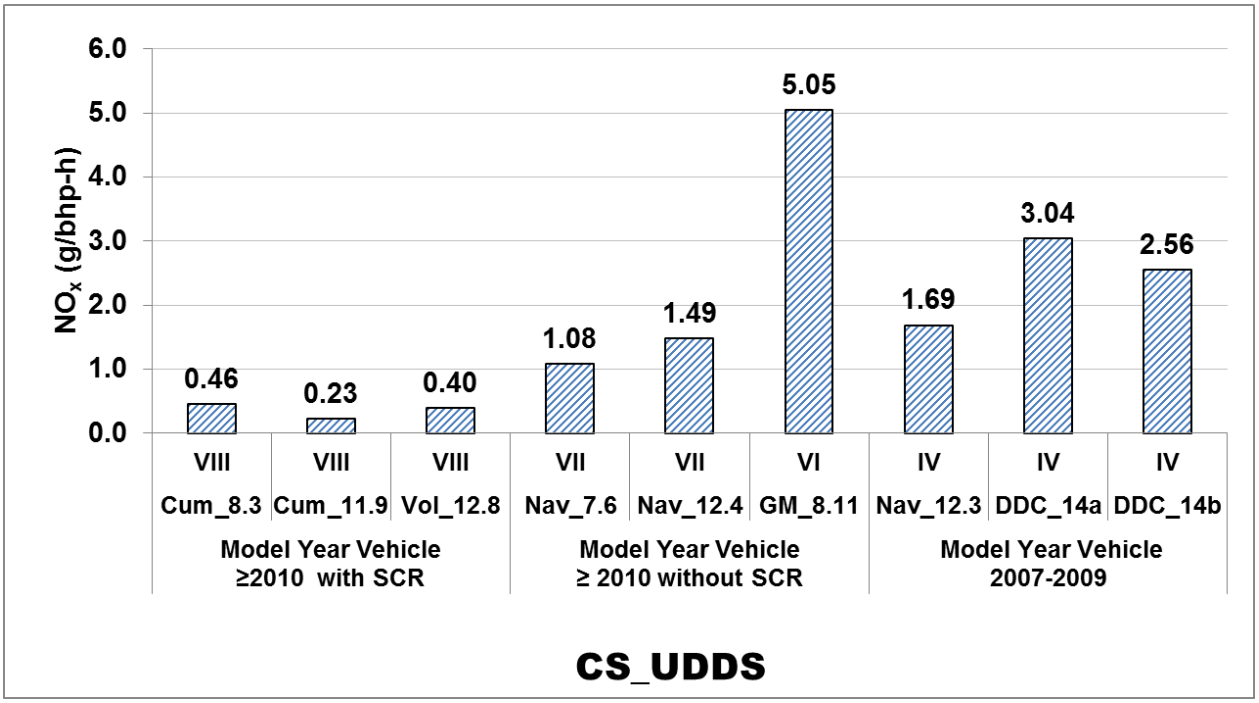


Figure B-5 Goods movement vehicle brake specific emissions

PM Emissions Goods Movement

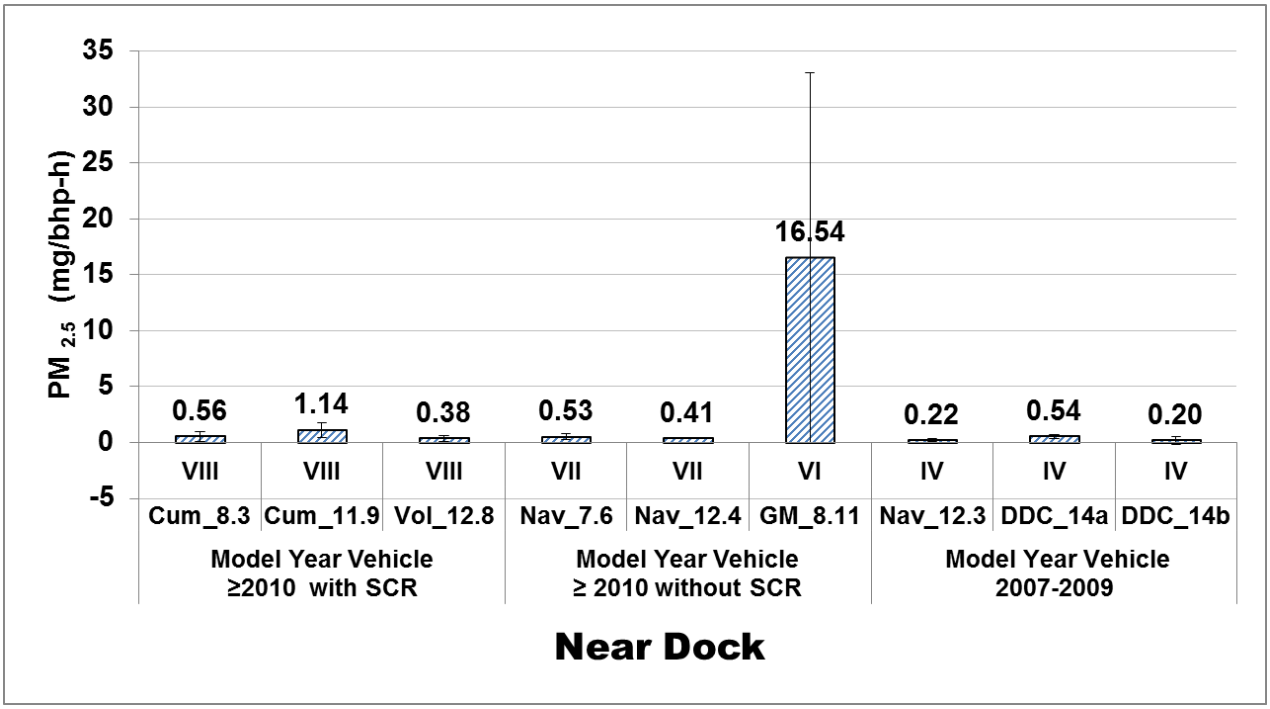
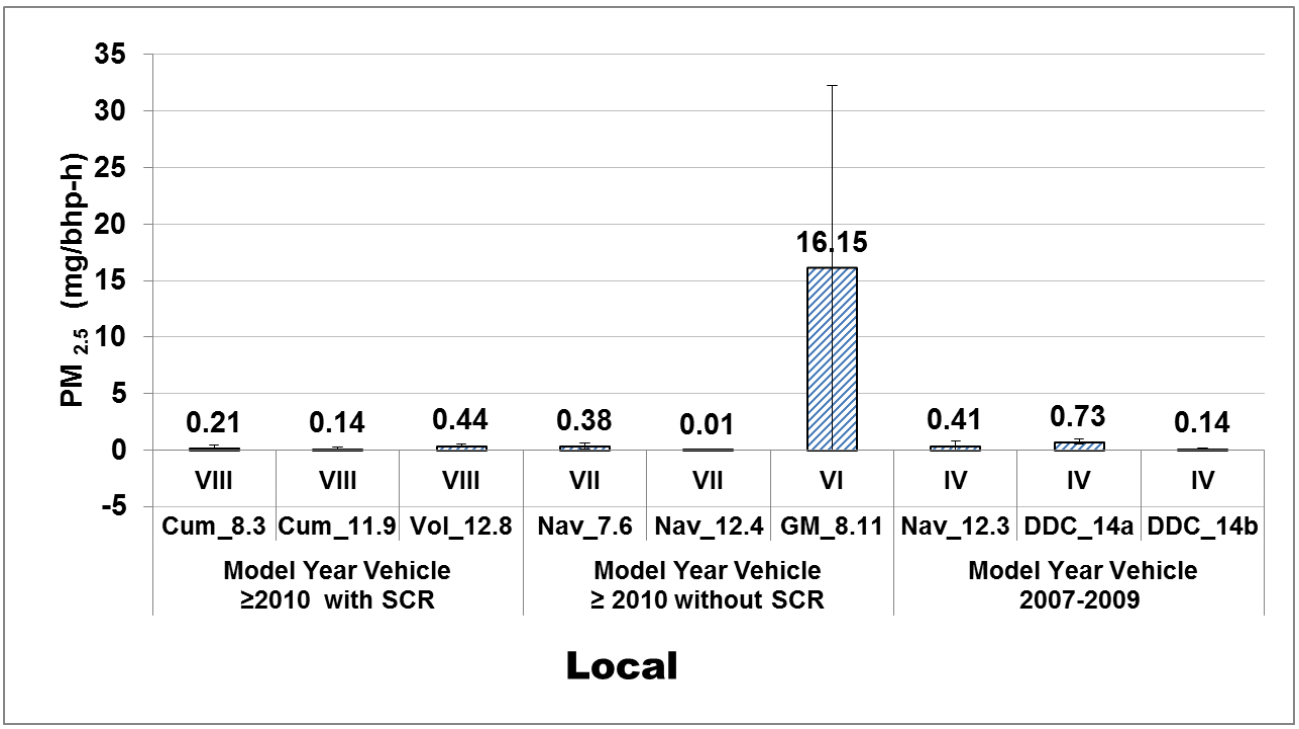
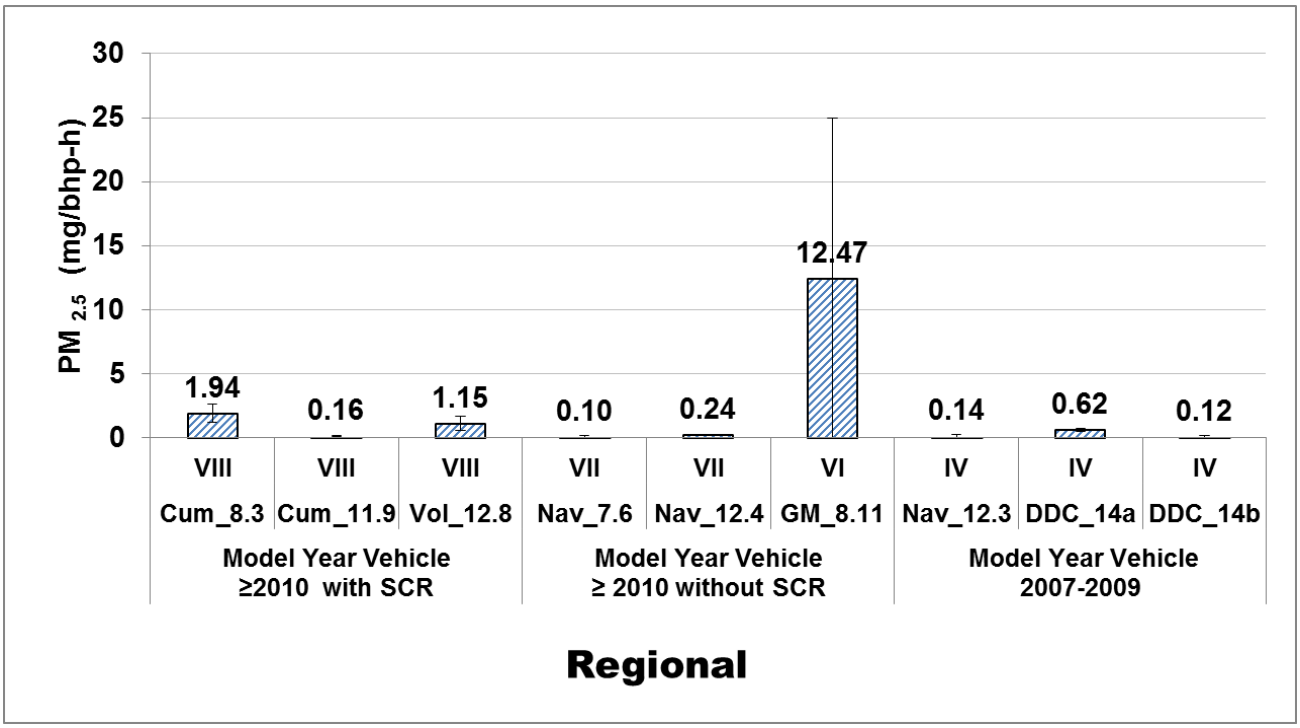
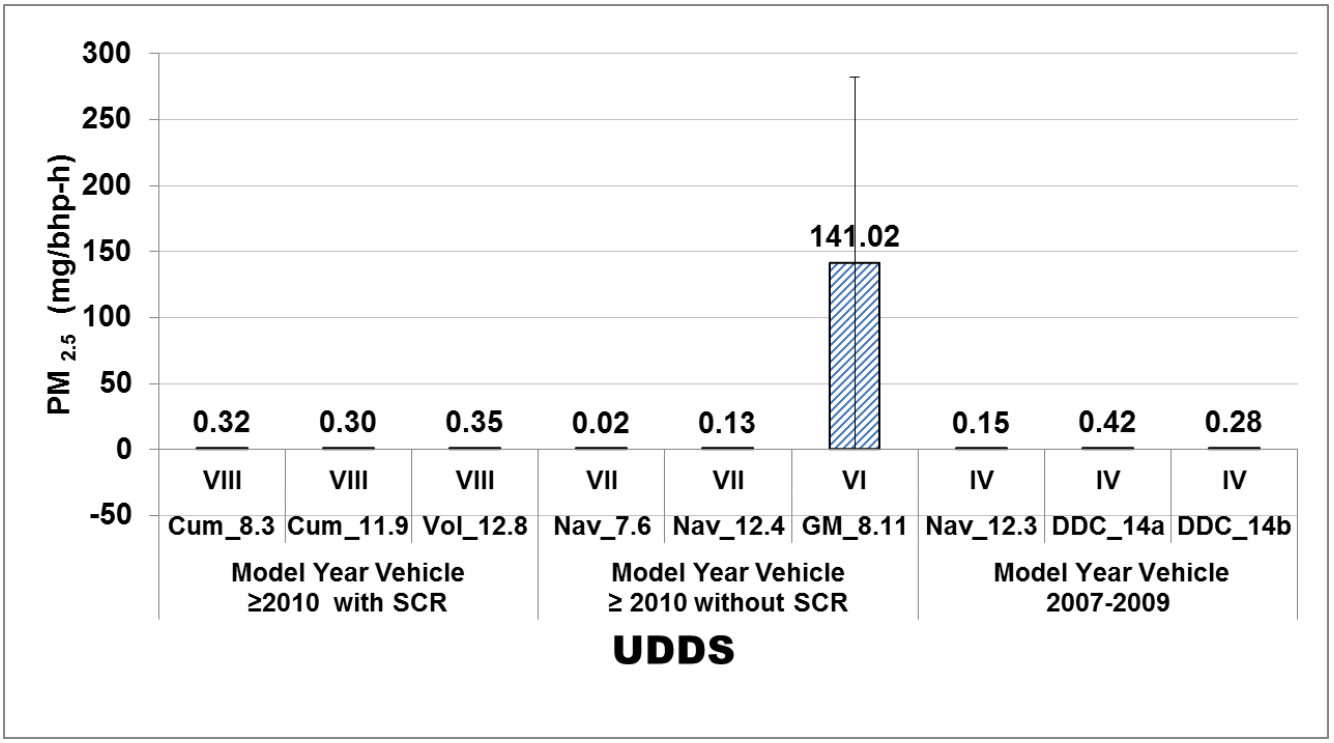
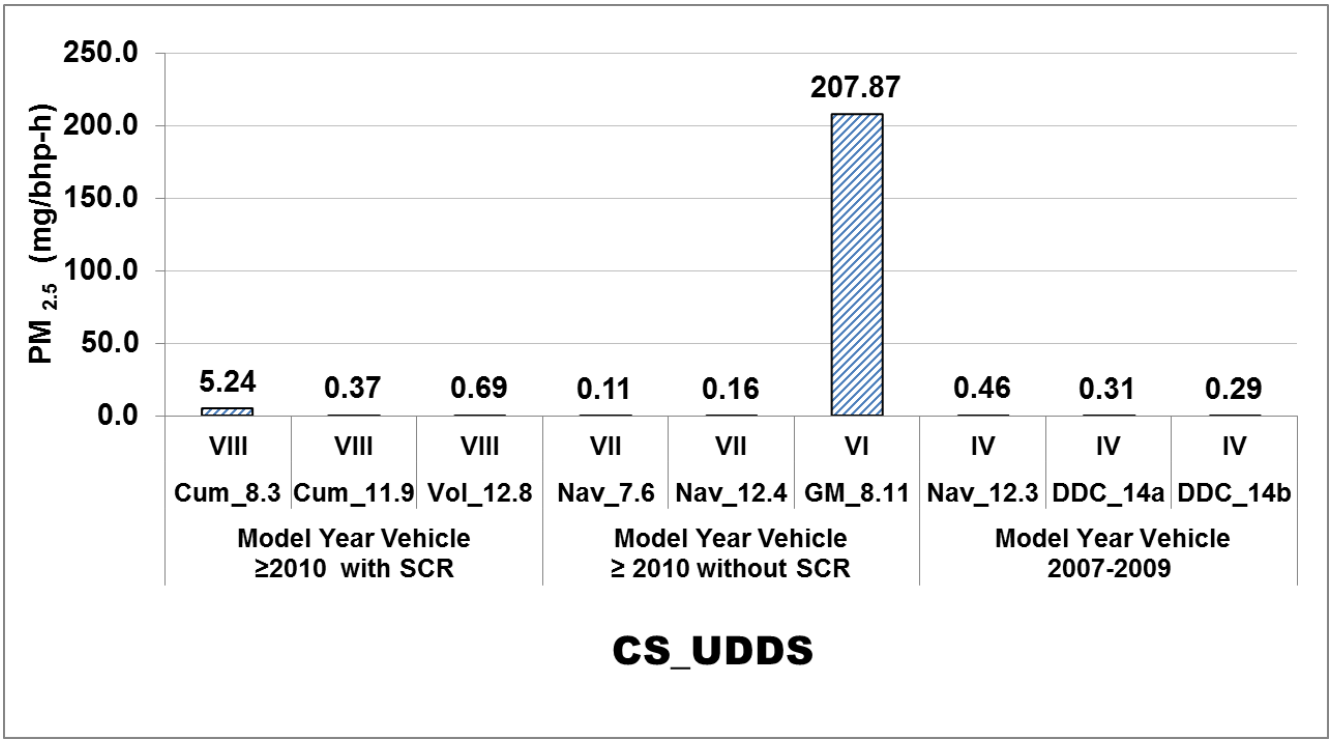


Figure B-6 Goods movement vehicle brake specific emissions

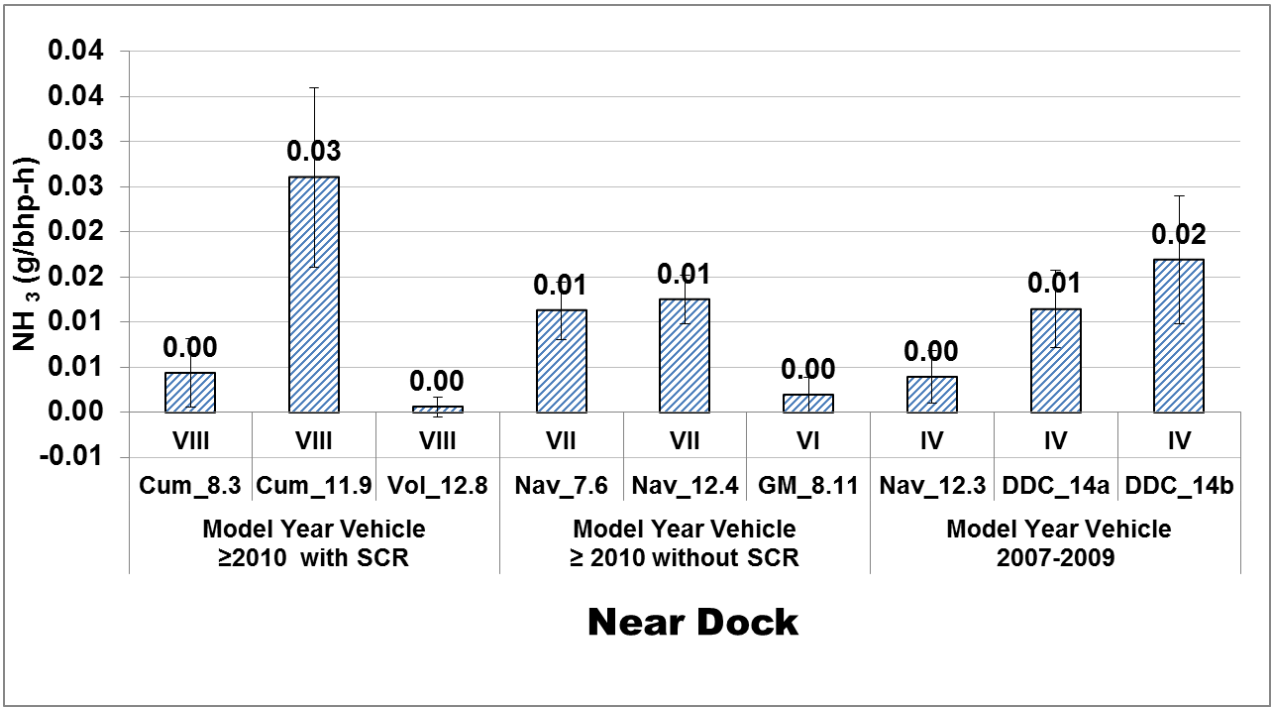

35
30
25
20
15
10
5
0
-5
PM 2.5 (mg/bhp-h)
0.21
0.14
0.44
0.38
0.01
16.15
0.41
0.73
0.14
VIII
VIII
VIII
VII
VII
VI
IV
IV
IV
Cum_8.3
Cum_11.9
Vol_12.8
Nav_7.6
Nav_12.4
GM_8.11
Nav_12.3
DDC_14a
DDC_14b
Model Year Vehicle ≥2010 with SCR
Model Year Vehicle ≥ 2010 without SCR
Model Year Vehicle 2007-2009
Local

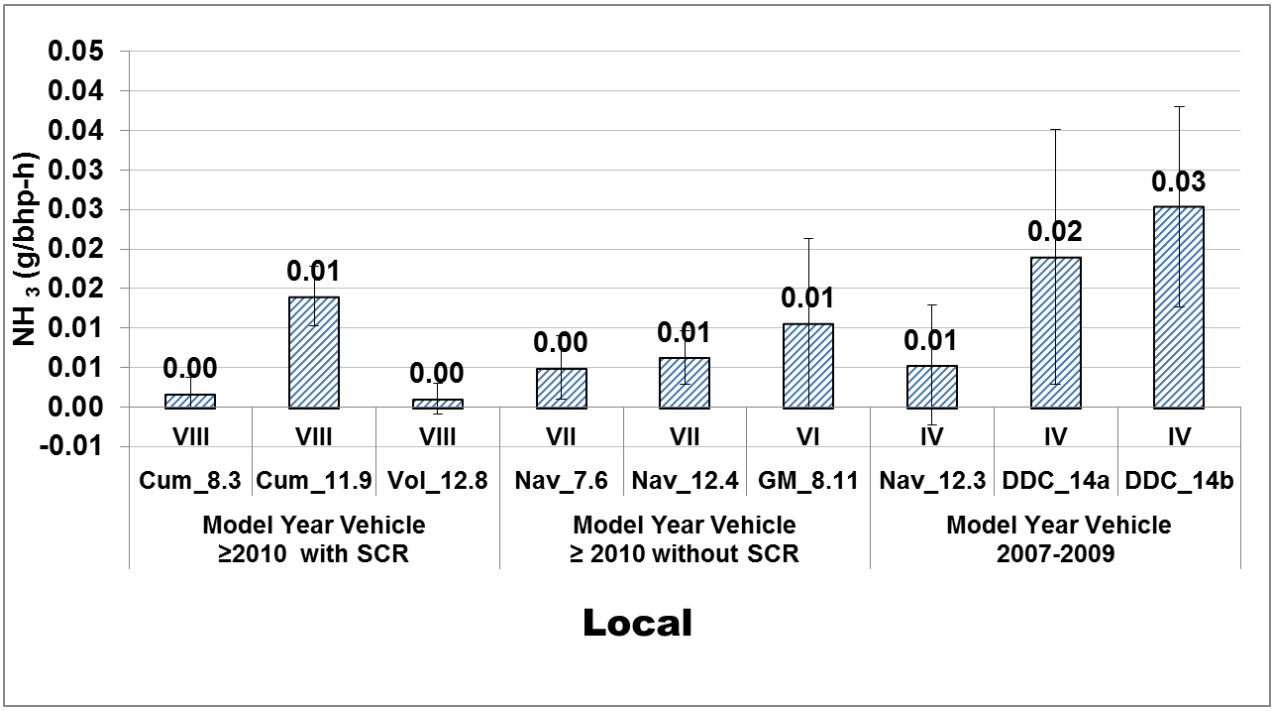

30
25
20
15
10
5
0
PM 2.5 (mg/bhp-h)
1.94
0.16
1.15
0.10
0.24
12.47
0.14
0.62
0.12
VIII
VIII
VIII
VII
VII
VI
IV
IV
IV
Cum_8.3
Cum_11.9
Vol_12.8
Nav_7.6
Nav_12.4
GM_8.11
Nav_12.3
DDC_14a
DDC_14b
Model Year Vehicle ≥2010 with SCR
Model Year Vehicle ≥ 2010 without SCR
Model Year Vehicle 2007-2009
Regional

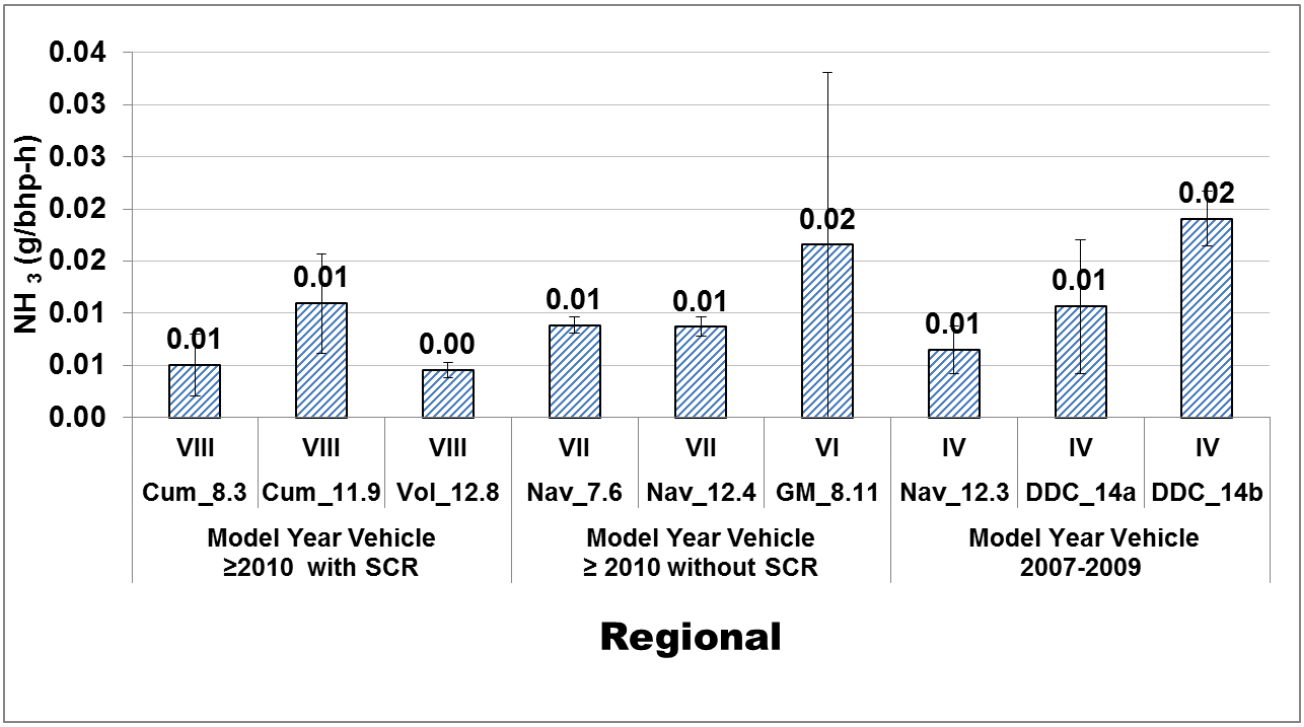

300
250
200
150
100
50
0
-50
PM 2.5 (mg/bhp-h)
0.32
0.30
0.35
0.02
0.13
141.02
0.15
0.42
0.28
VIII
VIII
VIII
VII
VII
VI
IV
IV
IV
Cum_8.3
Cum_11.9
Vol_12.8
Nav_7.6
Nav_12.4
GM_8.11
Nav_12.3
DDC_14a
DDC_14b
Model Year Vehicle ≥2010 with SCR
Model Year Vehicle ≥ 2010 without SCR
Model Year Vehicle 2007-2009
UDDS

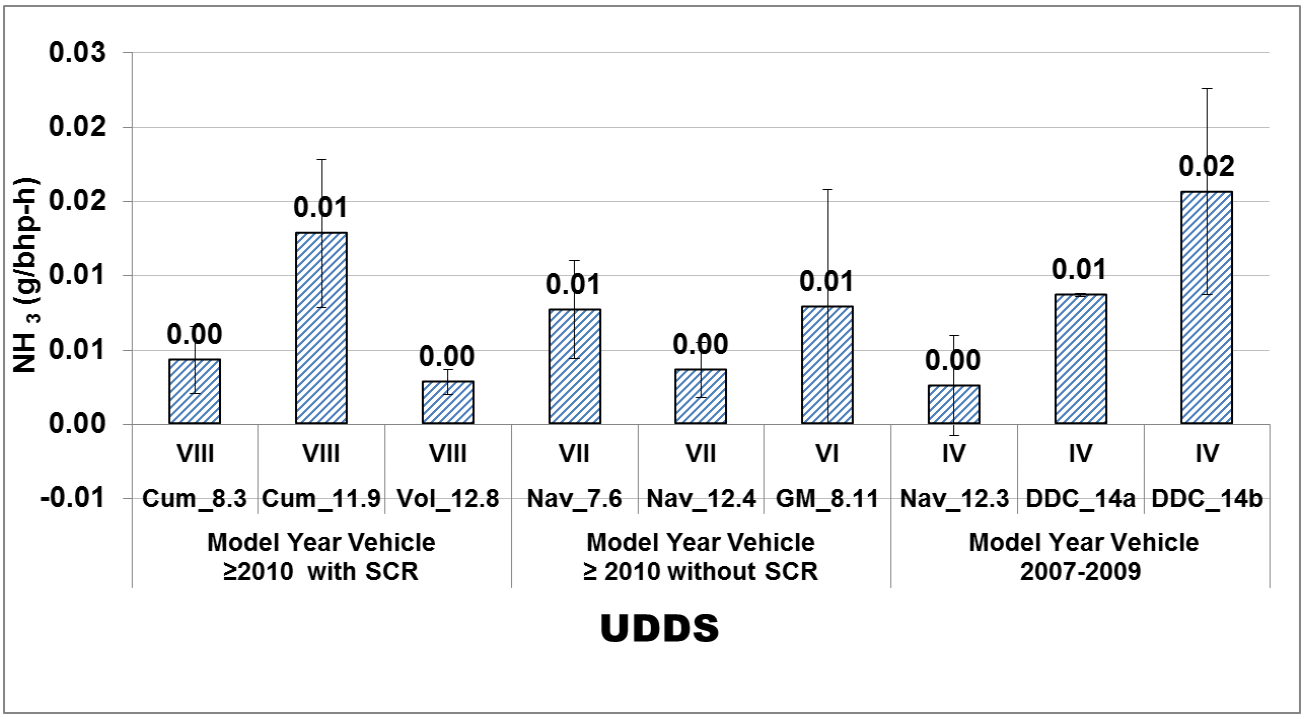

250.0
200.0
150.0
100.0
50.0
0.0
PM 2.5 (mg/bhp-h)
5.24
0.37
0.69
0.11
0.16
207.87
0.46
0.31
0.29
VIII
VIII
VIII
VII
VII
VI
IV
IV
IV
Cum_8.3
Cum_11.9
Vol_12.8
Nav_7.6
Nav_12.4
GM_8.11
Nav_12.3
DDC_14a
DDC_14b
Model Year Vehicle ≥2010 with SCR
Model Year Vehicle ≥ 2010 without SCR
Model Year Vehicle 2007-2009
CS_UDDS

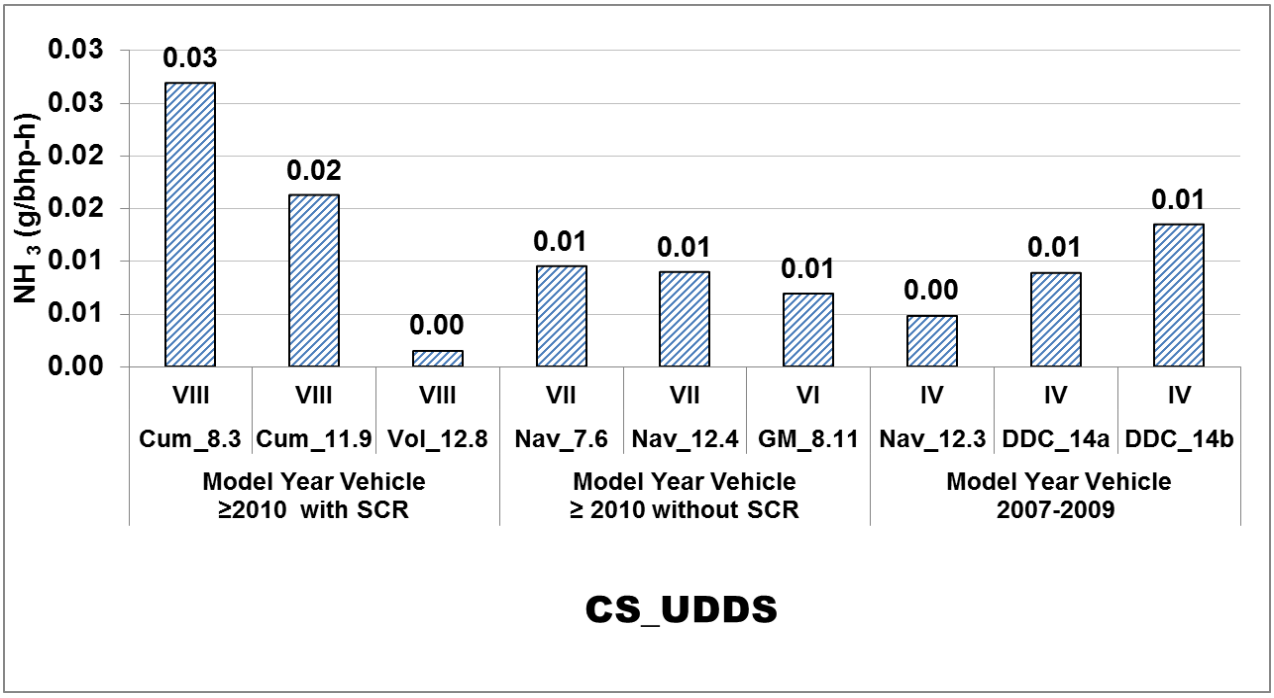
NH_3 Emissions Goods Movement






0.04
0.03
0.03
0.02
0.02
0.01
0.01
0.00
NH 3 (g/bhp-h)
0.01
0.01
0.00
0.01
0.01
0.02
0.01
0.01
0.02
VIII
VIII
VIII
VII
VII
VI
IV
IV
IV
Cum_8.3
Cum_11.9
Vol_12.8
Nav_7.6
Nav_12.4
GM_8.11
Nav_12.3
DDC_14a
DDC_14b
Model Year Vehicle ≥2010 with SCR
Model Year Vehicle ≥ 2010 without SCR
Model Year Vehicle 2007-2009
Regional


0.03
0.02
0.02
0.01
0.01
0.00
-0.01
NH 3 (g/bhp-h)
0.00
0.01
0.00
0.01
0.00
0.01
0.00
0.01
0.02
VIII
VIII
VIII
VII
VII
VI
IV
IV
IV
Cum_8.3
Cum_11.9
Vol_12.8
Nav_7.6
Nav_12.4
GM_8.11
Nav_12.3
DDC_14a
DDC_14b
Model Year Vehicle ≥2010 with SCR
Model Year Vehicle ≥ 2010 without SCR
Model Year Vehicle 2007-2009
UDDS


NH 3 (g/bhp-h)
0.03
0.03
0.02
0.02
0.01
0.01
0.00
0.03
0.02
0.00
0.01
0.01
0.01
0.00
0.01
0.01
VIII
VIII
VIII
VII
VII
VI
IV
IV
IV
Cum_8.3
Cum_11.9
Vol_12.8
Nav_7.6
Nav_12.4
GM_8.11
Nav_12.3
DDC_14a
DDC_14b
Model Year Vehicle ≥2010 with SCR
Model Year Vehicle ≥ 2010 without SCR
Model Year Vehicle 2007-2009
CS_UDDS

Attachment C: ECM Download and Inspection Summary

This appendix lists each of the vehicles tested and provides selected ECM down load information, fleet maintenance information, and other as-received information on the vehicle. The vehicles are listed in the order they were tested. All vehicles were inspected for MIL lamp issues and none were found. IN all but one case the vehicles appeared to have reasonable emissions results.
All vehicles inspected showed proper tire pressure, fluids, and operational capabilities to perform the desired cycles. No overheating or gross PM emitters were identified that would suggest eliminating the vehicle from the program.

The Navistar (12WZJ-B/2009) did show signs of drivability issues. The vehicle was not able to shift gears properly while performing the traces. This was a shared vehicle so WVU, UCR and AQMD were in discussion on how to proceed. WVU operated the vehicle on UCR's dyno and agreed there was a problem with shifting. The vehicle was driven on the roads with similar difficulties. UCR and WVU consulted with the leasing agency to find out the vehicle had low RPM torque and need to be shifted at lower RPM conditions. Once this was understood the vehicle was tested. The low RPM shifting is unusual and does not represent the normal operational modes as used by other diesel class 8 vehicles. Some issues in the emissions were discovered and are described in the results section.

The emission results for the propane goods movement vehicle suggest there may be an issue with the operation of the vehicle not identified by the fleet, MIL capabilities, or other indicators from the vehicle during preparations.

01_Vehicle Index C8.3 MY2010

- No faults from when the vehicle was received or as it left.
- Fleet maintains good records on vehicle maintenance.
- ECM down load is below

INSITE Lite 7.4.0.244			
Cummins Inc.			
Data Log Information			
	Start Date and Time:	22:33.8	
	Total Log Time:	20:59.1	
	Source Log Filename:	201111151016_ecm.csv	
	Destination Path	C:\Intelect\INSITE\Logs	
	Comments:		
Customer and Vehicle Information			
	Customer Name:	COCA-COLA	
	Vehicle Unit Number:	10B661690	
Engine Information			
	Model:		
	Serial Number:	0	
	ECM Part Number:	4993120	
ECM Status and Systems Analysis Download			
Date			15-Nov-11
Time			22:48.8
Accelerator Pedal Or Lever Position Sensor 2 Signal Voltage (V)			0.5
Accelerator Pedal Or Lever Position Sensor 2 Supply Voltage (V)			5
Accelerator Pedal or Lever Position Sensor Signal Voltage (V)			1.06
Accelerator Pedal or Lever Position Sensor Supply Voltage (V)			4.99
Aftertreatment Diesel Exhaust Fluid Dosing Unit State			Initializing
Aftertreatment Diesel Exhaust Fluid Dosing Valve Commanded Position			Closed
Aftertreatment Diesel Exhaust Fluid Line Heater 1 Status			Off
Aftertreatment Diesel Exhaust Fluid Line Heater 2 Status			Off
Aftertreatment Diesel Exhaust Fluid Line Heater 3 Status			Off
Aftertreatment Diesel Exhaust Fluid Line Heater 4 Status			Off
Aftertreatment Diesel Exhaust Fluid Line Pressure (psi)			0
Aftertreatment Diesel Exhaust Fluid Reverting Valve Position			Closed
Aftertreatment Diesel Exhaust Fluid Tank Heating Valve Position Commanded			Closed
Aftertreatment Diesel Oxidation Catalyst Intake Temperature (F)			223.7
Aftertreatment Diesel Oxidation Catalyst Intake Temperature Sensor Signal Voltage (V)			3.81
Aftertreatment Diesel Particulate Filter Differential Pressure (InHg)			0.1
Aftertreatment Diesel Particulate Filter Differential Pressure Sensor Signal Voltage (V)			0.75
Aftertreatment Diesel Particulate Filter Intake Temperature (F)			273.2
Aftertreatment Diesel Particulate Filter Intake Temperature Sensor Signal Voltage (V)			3.44
Aftertreatment Diesel Particulate Filter Lamp Status			Off

Aftertreatment Diesel Particulate Filter Operating State	SCR Catalyst
Aftertreatment Diesel Particulate Filter Outlet Pressure (psi)	0
Aftertreatment Diesel Particulate Filter Outlet Pressure Sensor Signal Voltage (V)	0.7
Aftertreatment Diesel Particulate Filter Outlet Temperature (F)	250.9
Aftertreatment Diesel Particulate Filter Outlet Temperature Sensor Signal Voltage (V)	3.63
Aftertreatment Diesel Particulate Filter Regeneration Start Switch Status	Off
Aftertreatment Diesel Particulate Filter Soot Load	Normal
Aftertreatment High Exhaust System Temperature Lamp Status	Off
Aftertreatment SCR Intake Temperature (F)	261
Aftertreatment SCR Intake Temperature Signal Voltage (V)	3.6
Aftertreatment SCR Outlet Temperature (F)	198
Aftertreatment SCR Outlet Temperature Signal Voltage (V)	4
Air Conditioning Pressure Switch	Off
Amber Warning Lamp Status	Off
Anti Theft Status	Unlocked
Barometric Air Pressure (InHg)	29
Barometric Air Pressure Sensor Signal Voltage (V)	3.69
Battery Voltage (V)	14.1
Brake Pedal Position Switch	Released
Calibration Software Phase	5030404
Catalyst Injector Tank Temperature (F)	61
Crankcase Pressure (inH2O)	2.5
Crankcase Pressure Sensor Signal Voltage (V)	1.75
Cruise Control Accelerate Switch	Off
Cruise Control Coast Switch	Off
Cruise Control On/Off Switch	Off
Cruise Control Set / Resume Switch	Neutral
Diagnostic Test Mode Switch	Off
Diesel Exhaust Fluid Low Level Lamp Status	Off
Diesel Exhaust Fluid Tank Level Readout (Percent)	95
ECM Time(Key On Time) (HH:MM:SS)	1085:42:10
EGR Cooler Efficiency (Percent)	100
EGR Differential Pressure (InHg)	0
EGR Differential Pressure Sensor Signal Voltage (V)	0.94
EGR Temperature (F)	155.8
EGR Temperature Sensor Signal Voltage (V)	4.81
EGR Valve Position Commanded (Percent)	0

EGR Valve Position Measured (Percent Open) (Percent)		0
EGR Valve Position Sensor Signal Voltage (V)		0
Electric Fuel Lift Pump Duty Cycle (Percent)		100
Electric Fuel Lift Pump Position		On
Engine Brake Output Circuit 1		Inactive
Engine Brake Output Circuit 2		Inactive
Engine Brake Output Circuit 3		Inactive
Engine Brake Output Circuit 4		Inactive
Engine Brake Selector Switch 1		Closed
Engine Brake Switch Level (Percent)		0
Engine Coolant Level		Normal
Engine Coolant Temperature (F)		185.4
Engine Coolant Temperature Sensor Signal Voltage (V)		0.81
Engine Distance (mi)		9959.6
Engine Hours (HH:MM:SS)		1021:56:47
Engine Oil Pressure (psi)		39
Engine Oil Pressure Sensor Signal Voltage (V)		2.38
Engine Oil Pressure Sensor Type		Analog
Engine Operating State		Low Speed G
Engine Protection Derate Suppress		Disable
Engine Protection Shutdown Override Switch		Off
Engine Speed (RPM)		795
Engine Speed Backup Sensor State		Valid Signal
Engine Speed Backup Synchronization State		Have Synchr
Engine Speed Main Sensor State		Valid Signal
Engine Speed Main Synchronization State		Have Synchr
Engine Speed Sensor Active		Main
Engine Speed Status		Good
Engine Warmup Protection Status		Inactive
Enhanced Exhaust Gas Pressure (InHg)		43.49
Exhaust Gas Pressure Sensor Signal Voltage (V)		1.19
Exhaust Volumetric Flowrate (ft3/s)		1.3
Fan Control Command (Percent)		100
Fan Control Multiplexed Request Level (Percent)		0
Fan Drive State		Off
Fast Idle Warmup Status		Inactive
Fuel Flow Rate Commanded (gph)		2.49
Fuel Pump Actuator Commanded Current (A)		1.38
Fuel Pump Actuator Duty Cycle (Percent)		39
Fuel Pump Actuator Measured Current (A)		1.44
Fuel Pump Actuator Position		Close

Fuel Rail Pressure Commanded (psi)		6526
Fuel Rail Pressure Measured (psi)		6307
Fuel Rail Pressure Sensor Signal Voltage (V)		1.1
Gear Down Protection State		Inactive
Idle Validation Switch		Idle
Instantaneous Fuel Economy (mpg)		0
Intake Air Heater 1		Off
Intake Air Heater 2		Off
Intake Manifold Air Temperature (F)		160.2
Intake Manifold Air Temperature Sensor Signal Voltage (V)		0.31
Intake Manifold Pressure (InHg)		2.7
Intake Manifold Pressure Sensor Signal Voltage (V)		1
J1939 Engine Source Address		0
J1939 Stop Broadcast Source Address One		0
J1939 Stop Broadcast Source Address Three		0
J1939 Stop Broadcast Source Address Two		0
Keyswitch		On
Keyswitch Off Counts		4784
Keyswitch On Counts		4785
Low Idle Adjustment Switch		Neutral
Parking Brake Switch State		Off
Percent Accelerator Pedal or Lever (Percent)		0
Percent Load (Percent)		0
Powertrain Protection Torque Limit (ft*lb)		4492
PTO Additional Switch		Off
PTO Decrement Switch		Off
PTO Increment Switch		Off
PTO On/Off Switch		Off
PTO Set / Resume Switch		Disable
PTO Status		Inactive
Red Stop Lamp Status		Off
Remote PTO Switch		Off
Sensor Supply 1 (V)		5

Sensor Supply 2 (V)		5
Sensor Supply 3 (V)		5
Sensor Supply 5 (V)		5
Sensor Supply 6 (V)		5
Transmission Gear Ratio		16
Transmission Status		Out of Gear
Trip Information Aftertreatment Diesel Exhaust Fluid Used (gal)		25.2
Trip Information Total Diesel Exhaust Fluid Used (gal)		36.6
Turbocharger Actuator Position Commanded (Percent Closed) (Percent)		89
Turbocharger Actuator Position Measured (Percent Closed) (Percent)		89
Turbocharger Actuator Position Sensor Signal Voltage (V)		0
Turbocharger Actuator Type		Electric
Turbocharger Compressor Intake Air Temperature (F)		115.6
Turbocharger Compressor Intake Air Temperature Sensor Signal Voltage (V)		0.69
Turbocharger Compressor Outlet Air Temperature (Calculated) (F)		134.6
Turbocharger Speed (RPM)		29633
Vehicle Speed (mph)		0
Wait To Start Lamp Status		Off
Water In Fuel Detected Total Accumulated Time (HH:MM:SS)		0:00:00
Water in Fuel Sensor Signal Voltage (V)		4.1
Water In Fuel State		No Water De

02_Vehicle Index Vol_12.8 MY 2011 Mack/MP8445C

- No faults from when the vehicle was received or as it left.
- Leased vehicle by WVU and thus all pre inspection of vehicle records were done by WVU.
- WVU has ECM down load in their records

03_Vehicle Index Navistar/12WZJ-B/2009

- No faults from when the vehicle was received or as it left.
- Leased vehicle by UCR and thus all pre inspection of vehicle records were done by leasing company and met DOT inspection requirements.

04_Vehicle Index GM/8.1/2008

- No faults from when the vehicle was received or as it left.
- Loaded vehicle by UCR and thus all pre inspection of vehicle records were done by fleet company and met DOT inspection requirements.

05_Vehicle Index Navistar/A475/2011

- No faults from when the vehicle was received or as it left.
- Leased vehicle by UCR and thus all pre inspection of vehicle records were done by leasing company and met DOT inspection requirements.

06_Vehicle Index Cummins/ISX11.9/2011

- No faults from when the vehicle was received or as it left.
- Leased vehicle by UCR and thus all pre inspection of vehicle records were done by leasing company and met DOT inspection requirements.

07_Vehicle Index Cummins/ISB 220/2007

- No faults from when the vehicle was received or as it left.

- Leased vehicle by UCR and thus all pre inspection of vehicle records were done by leasing company and met DOT inspection requirements.

08_Vehicle Index Bi-Phase/8.1l GM/2009

- No faults from when the vehicle was received or as it left.
- Loaned vehicle by UCR
- No ECM interface was possible thus, utilized fleet records and dash MIL lights for maintenance information.
- The vehicle did appear to run hot. No over temperature issues were identified. Fleet owner was asked about maintained records and no issues were identified.
- Vehicle had in excess of 1,000,000 miles. The engine was repowered to propane about 60,000 miles ago and sees 3,000 to 4,000 miles per year
- All fluids and tire pressures were suitable.

09_Vehicle Index Navistar/GDT260/2008

- No faults from when the vehicle was received or as it left.
- Fluids, tire pressure and other details all met UCR's inspection report logs
- Leased vehicle by UCR and thus all pre inspection of vehicle records were done by leasing company and met DOT inspection requirements.

10_Vehicle Index Navistar/A430/2011

- No faults from when the vehicle was received or as it left.
- Fluids, tire pressure and other details all met UCR's inspection report logs
- Leased vehicle by UCR and thus all pre inspection of vehicle records were done by leasing company and met DOT inspection requirements.

11_Vehicle Index DDC/60 14L/2008 SN = 06R1019569

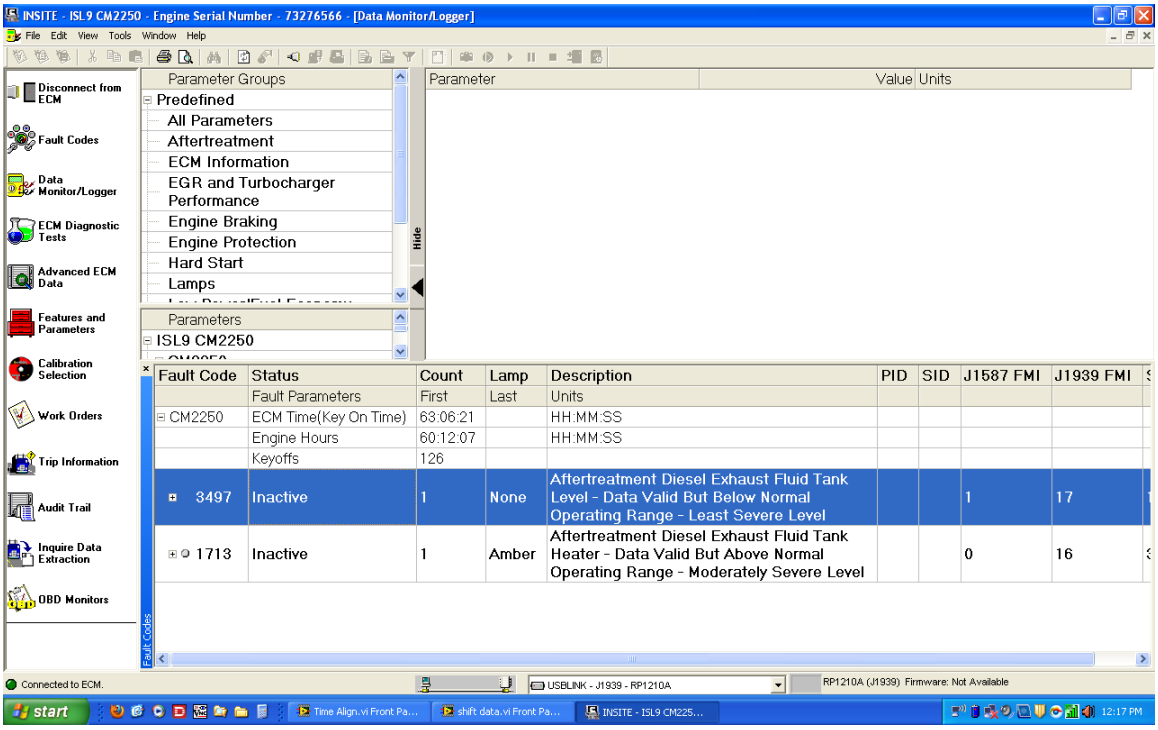
- No faults from when the vehicle was received or as it left.
- Loaned vehicle to UCR where fleet owner maintained good vehicle records

12_Vehicle Index DDC/60 14L/2008 SN = 06R1019704

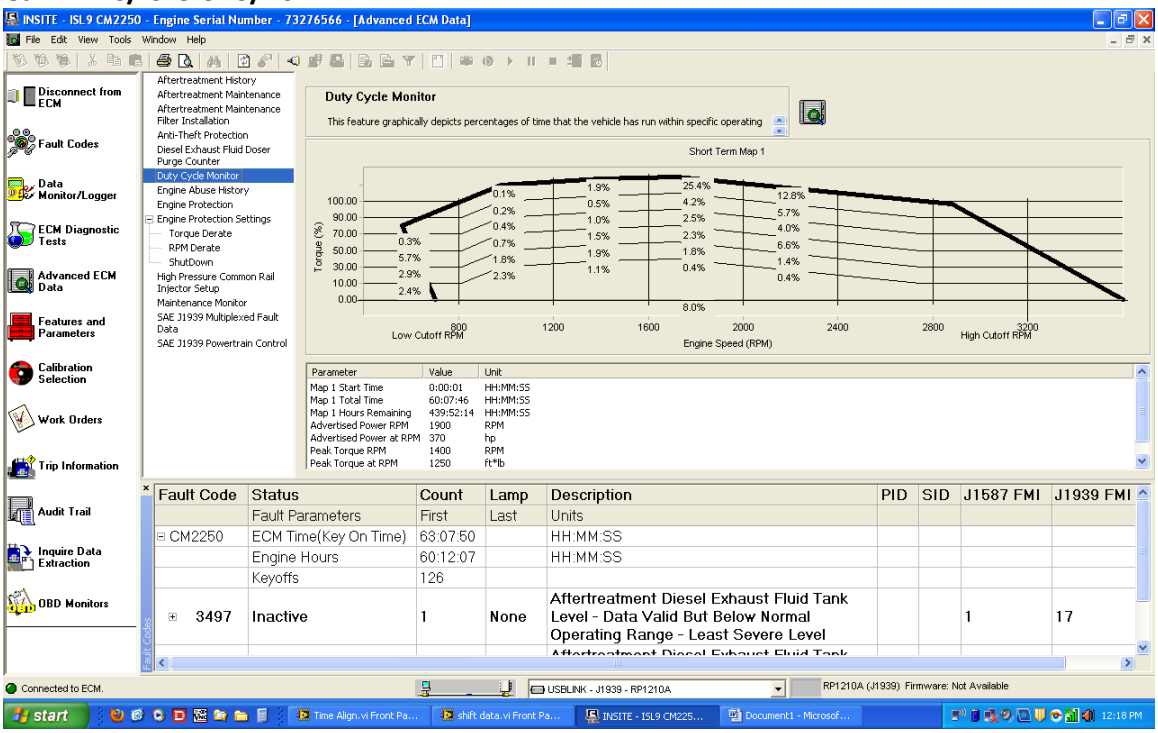
- No faults from when the vehicle was received or as it left.
- Loaned vehicle to UCR where fleet owner maintained good vehicle records

13 Refuse hauler Cummins/ISL9 370/2011

- No faults from when the vehicle was received or as it left.
- Leased vehicle by UCR and thus all pre inspection of vehicle records were done by leasing company and met DOT inspection requirements.
- ECM down load is below



Cummins/ISL9 370/2011



Cummins/ISL9 370/2011

14 Refuse hauler Navistar/A260/2011

- No faults from when the vehicle was received or as it left.
- Loaned vehicle to UCR

15 Refuse hauler Cummins/ISC 8.3/2012

- No faults from when the vehicle was received or as it left.
- Loaned vehicle to UCR

Attachment D: Vehicle Inspection Report

Veh. No.:_____ VIN:_____

ARRIVAL DATE:	ARRIVAL TIME:
AGENCY RELEASE SIGNATURE:	
DELIVERED BY:	

DEPARTURE DATE:	DEPARTURE TIME:
UCR ENGINEER RELEASE SIGNATURE:	
RETURNED TO:	

Retest? ☐Yes ☐No. If Yes, reason for retest:_____

Engine Compartment

			REMARKS
OIL LEVEL:	☐ FULL	☐ LOW	
COOLANT LEVEL:	☐ FULL	☐ LOW	
POWER STEERING FLUID:	☐ FULL	☐ LOW	
CONDITION OF BELTS:	☐ GOOD	☐ WORN	
CONDITION OF AIR FILTER:	☐ CLEAN	☐ DIRTY	
VISIBLE EXHAUST LEAKS:	☐ YES	☐ NO	
VISIBLE FLUID LEAKS:	☐ YES	☐ NO	
ENGINE APPEARANCE:	☐ CLEAN	☐ GREASY	

Equipment

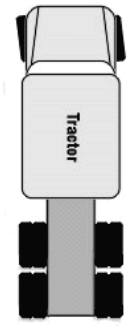
SERVICE BRAKES:	☐ GOOD	☐ POOR	☐ TOUCHY	
PARKING BRAKES:	☐ GOOD	☐ POOR		
POWER DIVIDER:	☐ GOOD	☐ DEFECTIVE	☐ NOT EQUIPPED	
TRANSMISSION:	☐ NORMAL	☐ SHIFTS HARD	☐ NOISY	
LUG NUT COVERS:	☐ YES	☐ NO	NUMBER MISSING:	
TIRE CONDITION:	FRONT		REAR	
	☐ GOOD	☐ WORN	☐ GOOD	☐ WORN
REMARKS:				

Vehicle Interior

UPHOLSTERY:	☐ CLEAN	☐ DIRTY	☐ STAINED	☐ DAMAGED	REMARKS:
CARPET:	☐ CLEAN	☐ DIRTY	☐ STAINED	☐ DAMAGED	REMARKS:
GENERAL APPEARANCE:	☐ CLEAN	☐ DIRTY			REMARKS:
GAUGES AND CONTROLS:	☐ OPERATE PROPERLY		☐ DEFECTIVE		REMARKS:

Vehicle Exterior (mark the location and describe any dents, scratches, damaged lights, mirrors etc. when the vehicle was received by UCR):

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____
7. _____
8. _____
9. _____



10. _____
11. _____
12. _____
13. _____
14. _____
15. _____
16. _____
17. _____
18. _____

Was this vehicle damaged while in UCR custody? ☐Yes ☐No. If Yes, explain:_____

_____

_____

General Remarks

_____

_____

_____

Vehicle Information Form

☐ Agency: _____

☐ Address: _____

☐ Contact Person: _____

☐ Phone Number/Email: _____

☐ Vehicle Manufacturer/ChassisType: _____

☐ Vehicle Occupancy Capacity: Seated _____ Standing _____

☐ Agency Vehicle #: _____ Licence Plate # : _____

☐ Vehicle Model Year: _____ VIN #:(17 DIGIT) _____

☐ GVWR Front: _____ Middle: _____ Rear: _____

☐ Curb Weight: Front: _____ Middle: _____ Rear: _____

☐ Vehicle Dimensions: Length: _____ Width: _____ Height: _____

☐ Mileage Odometer: _____ Hub Meter: _____

☐ Engine Manufacturer: _____ Model: _____ Year: _____

☐ Engine Serial#: _____ EPA Family Cert. #: _____

☐ Engine Displacement: _____ # of Cylinders: _____ Configuration: _____

☐ Max. Engine Power (hp) _____ hp @ _____ RPM

☐ Max. Engine Torque:(ft-lb.) _____ ft-lbs @ _____ RPM

☐ Idle Speed: _____ Governed Speed: _____ High Idle: _____

☐ Electronic Engine Control (☐Y/☐N) If Yes, Rebuild: _____

☐ Engine Rebuilt (☐Y/☐N) If Yes, Year of Rebuild: _____

☐ Primary Fuel Type: ☐D1 ☐D2 ☐CNG ☐LNG ☐ BD (%): Other (Specify): _____

☐ Number of Fuel Tanks: _____ Capacity: _____

☐ Oil Type: Weight _____ Brand _____

Aftertreatment Configuration:

- ☐ Oxidation Catalyst (☐Y/☐N) Manufacturer _____
- ☐ PM Trap (☐Y/☐N) Manufacturer _____
- ☐ SCR (☐Y/☐N) Manufacturer _____
- ☐ NOx Absorber (☐Y/☐N) Manufacturer _____
- ☐ NH3 Catalyst (☐Y/☐N) Manufacturer _____
- ☐ Other (☐Y/☐N) Manufacturer _____

☐ Total Number of Axles: _____ Number of Drive Axles: _____

☐ Transmission Type: Auto/Manual _____ Speeds: _____

☐ Transmission Manufacturer _____

☐ Hybrid Technology (☐Y/☐N) Comment: _____

☐ Tire Size: _____ Tire Manufacturer: _____ Type(☐Bias ☐Radial ☐Other)

☐ Tailpipe Size: _____ Location/Configuration: _____

Attachment E. Detailed Test Schedule

The test schedule will be dependent on vehicle availability and application. UCR expects that the refuse and goods movement vehicles will be needed for 4-5 days and the school bus for 3-4 days see Table E-1. Each vehicle will probably take a full week suggesting that the 6-8 N_2O samples approved by CARB will be available for each application.

Table E-1 Vehicle Usage Times

Application	Drive Cycles			
	Transit	Refuse truck	Goods movement	School bus
Preparation days	n/a	2	2	1
Test days	n/a	2	4	1
Total days	n/a	4	6	2

Tables 5 – 9, below show the expected test sequences, conditioning times, soak times, regeneration schedule, and N_2O grab samples. The tables also show that UCR proposes pulling the N_2O grab samples for only two hot cycles and one cold cycle. Each grab sample includes one diluted exhaust sample and one ambient sample. The six grab samples by UCR assume that WVU is not testing for that same week. If both UCR and WVU are testing during a particular week then UCR will only sample for one cold and one hot cycle. If UCR and WVU are both testing the samples to CARB will increase to eight which can also be managed according to ARB staff.

Table 5 Goods movment day 1 UDDS

event	time	N_2O	Cals
n/a	sec		
warm up	60		x,x
csUDDSx2	40	A, S	
soak	20		x
hotUDDSx2	40	A, S	
soak	20		x
hotUDDSx2	40		
soak	20		x
hotUDDSx2	40		
shutdown	30		x

Table 6 Goods movment day 2 Near Dock

event	time	N_2O	Cals
n/a	sec		
warm up	60		x
PortCond M1_Ph3	35		
soak	20		x
hotPort M1	60	A, S	
soak	20		x
hotPort M1	60		
soak	20		x
hotPort M1	60		
shutdown	30		x

Table 7 Goods movment day 3 Local

event	time	N_2O	Cals
n/a	sec		
warm up	60		x
PortCond M2_Ph3	40		
soak	20		x
hotPort M2	60	A, S	
soak	20		x
hotPort M2	60		
soak	20		x
hotPort M2	60		
shutdown	30		x

Note: **A, S** = **A**mbient, **S**ample

Table 8 Goods movment day 4 Regional

event	time	N_2O	Cals
n/a	sec		
warm up	60		x
PortCond M3_Ph3a,b	30		
soak	20		x
hotPort M3	75	A, S	
soak	20		x
hotPort M3	75		
soak	20		x
hotPort M3	75		
shutdown	30		x

Table 9 School bus day 1 CBD

event	time	N_2O	Cals
n/a	min		
warm up	60		x,x
csCBDx2	30	A, S	
soak	20		
hotCBDx2	30	A, S	
soak	20		x
hotCBDx2	30	A, S	
soak	20		
hotCBDx2	30		
shutdown	30		x

Table 10 Refuse hauler day 1

event	time	N_2O	Cals
n/a	sec		
warm up	60		x,x
csUDDSx2	40	A, S	
soak	20		
hotUDDSx2	40	A, S	
soak	20		x
hotUDDSx2	40	A, S	
soak	20		
hotUDDS2x	40		
shutdown	30		x

Table 11 Refuse hauler day 2

event	time	N_2O	Cals
n/a	sec		
warm up	60		x
csUDDSx2	40	A, S	
soak	20		
condAQMD ref	30		
soak	20		x
hotAQMD ref	30	A, S	
soak	20		
hotAQMD ref	30	A, S	
soak	20		x
hotAQMD ref	30		
shutdown	30		x

Attachment F: Quality Control Checks

This attachment discusses the data that was inspected for possible issues. Issues ranged from PM typo's, high standard deviations, and other checks. The purpose of this verification is to check the quality and consistency of the data. The final data set is validated by this procedure and includes all the regulated and not regulated emissions.. Below were investigated based on outlier stdev between replicate test.

Table A-1 Reregulated species evaluated for possible data issues

Test Article				Comment
1	Cummins/ISX11.9/2011	PDT_1	PM	High PM standard deviation could be due to measurement sensitivity. Possible high PM standard deviation on test, but filter weights were between 25 ug and 10 ug with a 5-10 ug uncertainty (tunnel blanks).
2	Navistar/12WZJ-B/2009	PDT_2	PM	High PM standard deviation could be due to measurement sensitivity. Possible high PM standard deviation on test, but filter weights were between 25 ug and 10 ug with a 5-10 ug uncertainty (tunnel blanks). PM spike observed by real time instruments. **DMM shows an outlier due to real** measurement on one test and not others.
3	Navistar/A430/2011	PDT_1	CO	High stdev on CO. The CO decreased from test 1 to test 3 (same for backup instrument). PM, although, showed a slight increase from test 1 to test 3, but filter weights were light 3 ug to 16 ug so it is hard to quantify.
c)	Bi-Phase/8.1l GM/2009	PDT_1	PM	Engine running very hot and may be creating PM. Variability high due to possible poor operation. No ECM codes or dash codes. Data represents in-use operation.
d)	Navistar/12WZJ-B/2009	PDT_1	CO2	Higher variability may be due to the Navistar drivability issue raised during testing. See Appendix xx for description of the drivability for the Navistar engine. **See presentation 2012.04.11 AQMD meeting 02d_CARB for details**. Put in the appendix.
e)	Bi-Phase/8.1l GM/2009	PDT_2	PM	Engine running very hot and may be creating PM. Variability high due to

				possible poor operation. No ECM codes or dash codes. Data represents in-use operation.
f)	Cummins/M2/2010	PDT_3	PM	PM filter weights over 100ug (easy to measure) and the PM increased from test 1 to test 3 (low to high). The measurable increasing trend suggests the PM for this test could be coming from the exhaust tubing surfaces and due to the high sustained loads of the Port3 cycle the PM attached to the surfaces could be released from the exhaust surfaces. This released PM then would enter into the CVS and be collected on the MEL gravimetric filter and TOX sampling probes. This would also happen in the environment and is real. The MEL was cleaned as described in the Experimental section and was not a contributing source of PM.
g)	Mack/MP8445C/2011	PDT_3	PM	The higher variability for this cycle/vehicle may be due to light filter weights. Light filter weights so variability would be higher (40 to 90 ug). PM was random and suggests from the engine and not the exhaust surfaces.
h)	Bi-Phase/8.1l GM/2009	PDT_3	NOx and PM	Engine running very hot and may be creating PM. Variability high due to possible poor operation. No ECM codes or dash codes. Data represents in-use operation. NOx looks real since both NOx and NO analyzer showed same response. Thus no changes were made.
i)	Navistar/A430/2011	UDDS	CO	High stdev on CO. The CO increased from test 1 to test 3 (same for backup instrument). PM also showed a slight increase from test 1 to test 3, but filter weights were light 3 ug to 16 ug so it is hard to quantify.
j)	Bi-Phase/8.1l GM/2009	UDDS	NOx and PM	Selected different UDDS cycle (did 5 only used 3). Others were aborted for MEL operational reasons.
k)	Cummins/ISB 220/2007	CS-CBD	PM	High PM standard deviation could be due to measurement sensitivity. Possible high PM standard deviation on test, but filter

l)	**Navistar/A260/2011**	UDDS	NOx	weights were between 60 ug (1) and 10 ug (2) with a 5-10 ug uncertainty (tunnel blanks). DMM showed same trend, but could decaying from first to last. PM could be hang-up from previous in-use operation. This is the test where two high frequency regenerations occurred. Two with regens and two without regens. Every other cycle they were occurring. The regens did not increase the PM mass, but did affect NOx and CO2. Analyze separately **UDDS** and **UDDS-regen.** Regens also occurred on the refuse truck cycle. Every test. Could not avoid them. Thus, in-use emissions should include regens for this vehicle.

EXHIBIT 7

OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

Air Toxics Hot Spots Program

Risk Assessment Guidelines

Guidance Manual for Preparation of Health Risk Assessments

February 2015

Air, Community, and Environmental Research Branch
Office of Environmental Health Hazard Assessment
California Environmental Protection Agency

Page Intentionally Left Blank

February 2015

Air Toxics Hot Spots Program
Risk Assessment Guidelines

The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments

Office of Environmental Health Hazard Assessment
California Environmental Protection Agency
George V. Alexeeff, Ph.D., Director

Project Leads: Daryn E. Dodge, Ph.D.[a] and Gregory Harris[b]

Reviewed by:

Melanie A. Marty, Ph.D.[a]

Assistant Deputy Director, Division of Scientific Affairs

David Siegel, Ph.D.[a]

Chief, Air, Community, and Environmental Research Branch

OEHHA acknowledges the following contributors:

Abdullah Mahmud, Ph.D[b]

Anthony Servin, P.E.[b]

Steven Yee[b]

Yan-Ping Zuo[b]

James F. Collins, Ph.D., D.A.B.T.[a]

Andrew G. Salmon, M.A., D.Phil.[a]

Aijun Albert Wang, Ph.D.[a]

[a] *Air, Community, and Environmental Research Branch*
Office of Environmental Health Hazard Assessment

[b] *California Air Resources Board*

Table of Contents

Table of Tables

Table of Figures

Preface

The draft of the *Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments* (Guidance Manual) is a description of the algorithms, recommended exposure variates, cancer and noncancer health values, and the air modeling protocols needed to perform a health risk assessment (HRA) under the Air Toxics Hot Spots Information and Assessment Act of 1987(Health and Safety Code Section 44300 et seq., see Appendix B). The Children's Environmental Health Protection Act of 1999 (Health and Safety Code Section 39606, also contained in Appendix B), which requires explicit consideration of infants and children in assessing risks from air toxics, necessitated revisions of the methods for both noncancer and cancer risk assessment, and of the exposure variates. This draft version of the Guidance Manual updates the previous version (OEHHA, 2003), and reflects advances in the field of risk assessment along with explicit consideration of infants and children.

The information presented in the draft manual is compiled from three technical support documents (TSDs) released by the Office of Environmental Health Hazard Assessment (OEHHA) for the Hot Spots Program. The three TSDs (which are also revised versions, replacing the original four Hot Spots TSDs adopted between 1999 and 2003) underwent public comment and peer review and were adopted for use in the Air Toxics Hot Spots program by the Director of OEHHA. The Technical Support Document for the Derivation of Noncancer Reference Exposure Levels (June, 2008) addressed the methodology for deriving acute, chronic and eight hour Reference Exposure Levels. The Technical Support Document for Cancer Potency Factors (May 2009) addresses the methodology for deriving cancer potency factors and adjusting cancer potency to account for the increased sensitivity of early-in-life exposure to carcinogens. The Technical Support Document for Exposure Assessment and Stochastic Analysis (June 2012) presents the exposure model for the Hot Spots program and reviews the available literature on exposure and relevant fate and transport variates. All three TSDs are available on OEHHA's web site at: http://www.oehha.ca.gov/air/hot_spots/index.html. Excerpts of these three TSDs are presented in this document. There is relatively little new information in the Guidance Manual since the adoption of the TSDs.

The draft Guidance Manual was released for public review. Public comments were received and changes were made in response to some comments. Responses were developed to all public comments. Both the Guidance Manual and OEHHA's response to comments were then reviewed by the State's Scientific Review Panel on Toxic Air Contaminants (SRP), who previously reviewed the three TSDs upon which this guidance is based. Following review by the SRP, OEHHA finalized this Guidance Manual. This Guidance Manual supersedes the risk assessment methods presented in the Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (OEHHA, 2003), which in turn replaced earlier guidance provided by the California Air Pollution Control Officer's Association (CAPCOA, 1993). This manual updates health effects values, exposure pathway variates (e.g., breathing rates), and

continues to use a tiered approach for performing HRAs based on current science and policy assessment. The Technical Support Document for Cancer Potency Factors (OEHHA, 2009) recommends a tenfold early-in-life potency factor adjustment for the third trimester and ages zero to less than two, and a threefold adjustment factor for ages two to less than sixteen. In addition, we recommend evaluating residency periods of nine, thirty and seventy years. This means that exposure variates are needed for the third trimester, ages zero to less than two, ages two to less than nine, ages two to less than 16, ages 16 to less than 30, and ages 16 to 70.

The tiered approach presented in this draft manual provides a risk assessor with flexibility and allows consideration of site-specific differences. Furthermore, risk assessors can tailor the level of effort and refinement of an HRA by using the point-estimate exposure variates or the stochastic treatment of distributions of exposure variates. The four-tiered approach to risk assessment primarily applies to residential cancer risk assessment. Compared to the OEHHA 2003 document, the exposure pathways in the Guidance Manual remain the same. The exposure and risk algorithms are similar, but they have been revised to accept new data or variables that are used in the tiered risk assessment approach.

The draft manual also contains example calculations and an outline for a modeling protocol and an HRA report. A software program, the Hot Spots Analysis and Reporting Program (HARP), has been developed by the Air Resources Board in consultation with OEHHA and Air Pollution Control/Air Quality Management District representatives. The HARP software, which is being updated with the new exposure variates and health values, is the recommended model for calculating and presenting HRA results for the Hot Spots Program. Information on obtaining the HARP software can be found on the ARB's web site at *www.arb.ca.gov* under the Hot Spots Program.

The intent of the Guidance Manual and the HARP software is to incorporate children's health concerns, update risk assessment practices, and to provide consistent risk assessment procedures. The use of consistent risk assessment methods and report presentation has many benefits, such as expediting the preparation and review of HRAs, minimizing revision and resubmission of HRAs, allowing a format for facility comparisons, and cost-effective implementation of HRAs and the Hot Spots Program. Risk assessments prepared with this Guidance Manual may be used for permitting new or modified stationary sources, or public notification, and risk reduction requirements of the Hot Spots Program. The use of uniform procedures allows comparison of risks from different facilities and enables identification of facilities that are problematic from a public health perspective. OEHHA reviews the HRAs to insure they are adequate for decision making, but does not play a role in permitting decisions that may result from the HRAs. OEHHA will provide advice to the Districts when requested on any of the risk assessment methods or health values they have used.

References

CAPCOA, 1993. CAPCOA Air Toxics Hot Spots Program Revised 1992 Risk Assessment Guidelines. California Air Pollution Control Officers Association, October 1993.

OEHHA, 2003. Air Toxics Hot Spots Risk Assessment Guidelines: The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments.

OEHHA, 2008. Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. Available online at: http://www.oehha.ca.gov

OEHHA, 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. May 2009. Available online at: http://www.oehha.ca.gov

OEHHA, 2012. Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis. Available online at http://www.oehha.ca.gov

1 - Introduction

1.1 Development of Guidelines

The Air Toxics Hot Spots Information and Assessment Act is designed to provide information to state and local agencies and to the general public on the extent of airborne emissions from stationary sources and the potential public health impacts of those emissions. The Hot Spots Act requires that the Office of Environmental Health Hazard Assessment (OEHHA) develop risk assessment guidelines for the Hot Spots program (Health and Safety Code (HSC) Section 44360(b)(2)) (see Appendix B for the text of the HSC). In addition, the Hot Spots Act specifically requires OEHHA to develop a "likelihood of risks" approach to health risk assessment. In response, OEHHA developed a tiered approach to risk assessment where a point estimate approach is first employed. If a more detailed analysis is needed, OEHHA has developed a stochastic, or probabilistic, approach using exposure factor distributions that can be applied in a stochastic estimate of the exposure. A detailed presentation of the tiered approach, risk assessment algorithms, selected exposure variates (e.g., breathing rate), and distributions with a literature review is presented in the *Air Toxics Hot Spots Program Risk Assessment Guidelines*; *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012). A summary of this information can be found in Chapter 5 of this document.

The Technical Support Document for the Derivation of Noncancer Reference Exposure Levels (OEHHA, 2008) addresses dose response relationships for noncancer health effects and the methodology for deriving acute, chronic and 8-hour Reference Exposure Levels (RELs). Currently there are 53 acute RELs, 82 chronic RELs, and 10 eight-hour RELs. Review and revision of RELs to take into account new information and sensitive subpopulations including infants and children is an ongoing process. All draft RELs for individual chemicals revised under the current noncancer methodology will undergo public comment and peer review, as mandated by the Hot Spots Act. The Technical Support Document for Cancer Potency Factors (OEHHA, 2009) addresses the methodology for deriving cancer potency factors and adjusting cancer potency to account for the increased sensitivity to early-in-life exposure to carcinogens. This document contains inhalation cancer potency factors and oral cancer potency factors for 142 toxicants and toxicant compound classes developed by OEHHA or developed by other authoritative bodies and endorsed by OEHHA. The OEHHA website (*www.oehha.ca.gov*) should be consulted for the most current adopted chronic, acute and 8-hour RELs and cancer potency factors. In addition, for a small subset of these substances that are subject to airborne deposition and hence human oral and dermal exposure, oral chronic RELs and oral cancer potency factors have been developed by OEHHA. A summary of cancer and noncancer health effects values can be found in Appendix L and Chapters 6 and 7 of the Guidance Manual. All three Technical Support Documents have undergone public and peer review and have been approved by the state's Scientific Review Panel on Toxic Air Contaminants and adopted by OEHHA. The Guidance Manual is undergoing the same public and peer review process.

The Guidance Manual contains a description of the algorithms, recommended exposure variates, and cancer and noncancer health values, and modeling protocols needed to perform a Hot Spots risk assessment under the Hot Spots Act (see Appendix B). The information for the Guidance Manual is taken from the three TSDs. The Guidance Manual supersedes the risk assessment methods presented in the Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (OEHHA, 2003).

The Guidance Manual is intended to address health risks from airborne contaminants released by stationary sources. Some of the methodology used is common to other regulatory risk assessment applications, particularly for California programs. However, if the reader needs to prepare a Health Risk Assessment (HRA) under another program, the HRA may need additional analyses. Therefore, appropriate California and federal agencies should be contacted. For example, if a facility must comply with HRA requirements under the Resource Conservation and Recovery Act (RCRA) or the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), the California Department of Toxic Substances Control (DTSC) must be contacted to determine if an HRA written to comply with AB 2588 will also satisfy RCRA/CERCLA requirements.

1.2 Use of the Guidance Manual

The intent in developing this Guidance Manual is to provide HRA procedures for use in the Air Toxics Hot Spots Program or for the permitting of existing, new, or modified stationary sources. The Air Resources Board (ARB) website (*www.arb.ca.gov*) provides more information on the Hot Spots Program and risk management guidelines, including recommendations for permitting existing, new, or modified stationary sources. The use of consistent risk assessment procedures and report presentation allows comparison of one facility to another, expedites the review of HRAs by reviewing agencies, and minimizes revision and resubmission of HRAs.

OEHHA recognizes that no one risk assessment procedure or set of exposure variates could perfectly address the many types of stationary facilities in diverse locations in California. Therefore a tiered risk assessment approach was developed to provide flexibility and allow consideration of site-specific differences. The tiered approach to risk assessment is discussed in detail in Chapter 8 of this Guidance.

These guidelines should be used in conjunction with the emission data collected and reported pursuant to requirements of the ARB's *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5*), and the *Emission Inventory Criteria and Guidelines Report for the Air Toxics "Hot Spots" Program* (EICG Report), which is incorporated by reference therein (see ARB's web site: http://www.arb.ca.gov/ab2588/2588guid.htm for the most current version, which was approved on August 27, 2007). This regulation outlines requirements for the collection of emission data, based on an inventory plan, which must be approved by the Air Pollution Control or Air Quality Management District (District). The emissions reported under this program are routine or predictable and include continuous

and intermittent releases and predictable process upsets or leaks. Emissions for unpredictable releases (e.g., accidental catastrophic releases) are not reported under this program.

For landfill sites, these guidelines should be applied to the results of the landfill testing required under Health and Safety Code Section 41805.5 as well as to any emissions reported under the emission inventory requirements of the Air Toxics Hot Spots Act (e.g., from flares or other on-site equipment). Districts should be consulted to determine the specific landfill testing data to be used.

1.3 Who is Required to Conduct a Risk Assessment

The Hot Spots Act requires that each local Air Pollution Control District or Air Quality Management District (hereinafter referred to as District) determine which facilities will prepare an HRA. As defined under the Hot Spots Act, an HRA includes a comprehensive analysis of the dispersion of hazardous substances in the environment, their potential for human exposure, and a quantitative assessment of both individual and population-wide health risks associated with those levels of exposure.

Districts are to determine which facilities will prepare an HRA based on a prioritization process outlined in the law. The process by which Districts identify priority facilities for risk assessment involves consideration of potency, toxicity, quantity of emissions, and proximity to sensitive receptors such as hospitals, daycare centers, schools, work-sites, and residences. The District may also consider other factors that may contribute to an increased potential for significant risk to human receptors. As part of this process Districts categorize facilities as high, intermediate, or low priority. The District prioritization process is described in the *CAPCOA Air Toxics Hot Spots Program Facility Prioritization Guidelines, July 1990* (CAPCOA, 1990), although some Districts may have adopted their own method for prioritizing facilities for the purposes of AB2588, permitting, etc. Consult the District for updates to the Prioritization Guidelines. See the Hot Spots Program on ARB's web site at *www.arb.ca.gov* for more information on facility prioritization procedures.

Facilities designated by a District as "high priority" are required to submit an HRA to the District within 150 days of designation. Districts may grant a 30-day extension. However, a District may require any facility to prepare and submit an HRA according to the District priorities established for purposes of the Hot Spots Act.

1.4 The Hot Spots Analysis and Reporting Program (HARP) Software

The ARB and the Districts have identified a critical need for software to assist with the programmatic aspects of the Hot Spots Program. HARP is computer software used by the ARB, OEHHA, Districts, and facility operators to promote statewide consistency, efficiency, and cost-effective implementation of HRAs and the Hot Spots Program. The HARP software package includes: 1) an Emissions Inventory Database Module, 2) an Air Dispersion Modeling Module, and 3) a Risk Analysis Module. The user-friendly Windows-based package provides for:

1. Electronic implementation of the risk assessment methods presented in the OEHHA guidelines (Guidance Manual);
2. Electronic data transfer from facilities and Districts;
3. The production of reports;
4. Facility prioritization;
5. Air dispersion modeling (AERMOD) of multiple emission releases or facilities for cumulative impact evaluations;
6. A summary report of acute, 8-hour, and chronic health hazard quotients or indices, and cancer risk at the point of maximum impact (PMI), maximally exposed individual resident (MEIR), maximally exposed individual worker (MEIW) and other receptors to be evaluated as needed;
7. Mapping displays of facility property boundaries, risk isopleths, and elevation contours;
8. The ability to display combined risk contours from multiple emission sources;
9. Output of data for use in other “off-the-shelf” Geographic Information Systems (GIS) programs for additional types of analysis; and
10. Census data for determining population-related health impacts showing the number of people exposed at various cancer risk levels and cancer burden.

1.5 Risk Assessment Review Process

The Hot Spots Act risk assessments are reviewed by the local District and by OEHHA. The Districts focus their review on the emissions data and the air dispersion modeling. OEHHA provides comments on the HRA’s general concordance with the Guidelines Manual and the completeness of the reported health risks. The District, taking into account the comments of OEHHA, approves the HRA or returns it to the facility for revision and resubmission. If the HRA is not revised and resubmitted by the facility within 60 days, the District may modify the HRA and approve it as modified. Based on the approved HRA, the District determines if there is a significant health risk associated with emissions from the facility. If the District determines that facility emissions pose a significant health risk, the facility operator provides notice to all exposed individuals regarding the results of the HRA and may be required to take steps to reduce emissions by implementing a risk reduction audit and plan. Notification is to be made according to

procedures specified by the District. Each District determines its own levels of significance for cancer and noncancer health effects for notification and risk reduction. See the Hot Spots Program on ARB's web site at *www.arb.ca.gov* for more information on significance levels selected by each District.

1.6 Uncertainty in Risk Assessment

OEHHA has striven to use the best science available in developing these risk assessment guidelines. However, there is a great deal of uncertainty associated with the process of risk assessment. The uncertainty arises from lack of data in many areas necessitating the use of assumptions. The assumptions used in these guidelines are designed to err on the side of health protection in order to avoid underestimation of risk to the public. Sources of uncertainty, which may overestimate or underestimate risk, include: 1) extrapolation of toxicity data in animals to humans, 2) uncertainty in the estimation of emissions, 3) uncertainty in the air dispersion models, and 4) uncertainty in the exposure estimates. In addition to uncertainty, there is a natural range or variability in measured parameters defining the exposure scenario. Scientific studies with representative sampling and large enough sample sizes can characterize this variability. In the specific context of a Hot Spots risk assessment, the source of variability with the greatest quantitative impact is variation among the human population in such properties as height, weight, food consumption, breathing rates, and susceptibility to chemical toxicants. OEHHA captures at least some of the variability in exposure by developing data driven distributions of intake rates, where feasible, in the TSD for Exposure Assessment (OEHHA, 2012).

Interactive effects of exposure to more than one carcinogen or toxicant are addressed in the risk assessment with default assumptions of additivity. Cancer risks from all carcinogens addressed in the HRA are added. Similarly, non-cancer hazard quotients for substances impacting the same target organ/system are added to determine the hazard index (HI). Although such effects of multiple chemicals are assumed to be additive by default, several examples of synergism (interactive effects greater than additive) are known. For substances that act synergistically, the HRA could underestimate the risks. Some substances may have antagonistic effects (lessen the toxic effects produced by another substance). For substances that act antagonistically, the HRA could overestimate the risks.

Other sources of uncertainty, which may underestimate or overestimate risk, can be found in exposure estimates where little or no data are available (e.g., soil half-life and dermal penetration of some substances from a soil matrix).

The differences among species and within human populations usually cannot be easily quantified and incorporated into risk assessments. Factors including metabolism, target site sensitivity, diet, immunological responses, and genetics may influence the response to toxicants. The human population is much more diverse both genetically and culturally (e.g., lifestyle, diet) than inbred experimental animals. The intraspecies variability among humans is expected to be much greater than in laboratory animals.

In most cases, cancer potency values have been estimated only for the single most affected tumor site. This represents a source of uncertainty in the cancer risk assessment. Adjustment for tumors at multiple sites induced by some carcinogens may result in a higher potency. Some recent assessments of carcinogens include such adjustments. Other uncertainties arise 1) in the assumptions underlying the dose-response model used, and 2) in extrapolating from large experimental doses, where other toxic effects may compromise the assessment of carcinogenic potential, to usually much smaller environmental doses.

When occupational epidemiological data are used to generate a carcinogenic potency or a health protective level for a non-carcinogen, less uncertainty is involved in the extrapolation from workplace exposures to environmental exposures. When using human data, no interspecies extrapolation is necessary eliminating a significant source of uncertainty. However, children are a subpopulation with hematological, nervous, endocrine, and immune systems that are still developing and may be more sensitive to the effects of toxicants. The worker population and risk estimates based on occupational epidemiological data are more uncertain for children than adults. Current risk assessment guidelines include procedures designed to address the possibly greater sensitivity of infants and children, but there are only a few compounds for which these effects have actually been measured experimentally. In most cases, the adjustment relies on default assumptions which may either underestimate or overestimate the true risks faced by infants and children exposed to toxic substances or carcinogens.

Risk estimates generated by an HRA should not be interpreted as the expected rates of disease in the exposed population but rather as estimates of potential for disease, based on current knowledge and a number of assumptions.

In the Hot Spots program, cancer risk is often expressed as the maximum number of new cases of cancer projected to occur in a population of one million people due to exposure to the cancer-causing substance over a 30-year residential period. However, there is uncertainty associated with the cancer risk estimate. An individual's risk of contracting cancer from exposure to facility emissions may be less or more than the risk calculated in the risk assessment. An individual's risk not only depends on the individual's exposure to a specific chemical but also on his or her genetic background, health, diet, lifestyle choices and other environmental and workplace exposures. OEHHA uses health-protective exposure assumptions to avoid underestimating risk. For example, the risk estimate for airborne exposure to chemical emissions uses the health-protective assumption that the individual has a high breathing rate and exposure began early in life when cancer risk is highest.

A Reference Exposure Level (REL) is the concentration level at or below which no adverse non-cancer health effects are anticipated for the specified exposure duration. RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of factors that account for uncertainties as well as individual differences in human susceptibility to chemical exposures. The factors used in the calculation of RELs are meant to err on the side of public health

protection in order to avoid underestimation of non-cancer hazards. Exceeding the REL does not automatically indicate an adverse health impact. However, increasing concentrations above the REL value increases the likelihood that the health effect will occur.

Risk assessments under the Hot Spots program are often used to compare one source with another and to prioritize concerns. Consistent approaches to risk assessment are necessary to fulfill this function.

1.7 Tiered Approach to Risk Assessment

OEHHA developed a tiered approach to accommodate consideration of site-specific data that may be more appropriate for a given facility than the default variate. The first tier is the simplest point estimate approach to estimating exposure to facility emissions. Tier 1 is the first step in conducting a comprehensive risk assessment using algorithms and point estimates of input values described in the *Technical Support Document for Exposure Assessment and Stochastic Analysis*. (OEHHA, 2012) Each facility conducts a Tier 1 risk assessment to promote consistency across the state in facility risk assessments and facilitate comparisons across facilities. To be health-protective, high-end estimates for the key intake exposure variates are used for the dominant exposure pathways.

Tier 2 allows use of site-specific point estimates of exposure variates as long as these estimates can be justified. For example, if there are data indicating that consumption of fish from an impacted body of water is lower than the OEHHA-recommended fish consumption rate, then the facility can use that data to generate a point estimate for sport-fish consumption from that body of water. The risk assessor must supply the data and methods used for the site-specific estimates, and the site-specific estimates must be reproducible and approved by both the District and OEHHA.

Tier 3 risk assessment involves stochastic analysis of exposure using data-based distributions for the key exposure variates compiled in the OEHHA (2012) *Technical Support Document*. Since a stochastic approach to risk assessment provides more information about the range of risk estimates based on the range of exposures, Tier 3 can serve as a useful supplement to the Tier 1 and 2 approaches. Variance propagation methods (e.g., Monte Carlo analysis) are used to derive a range of cancer risk estimates reflecting the known variability in the inputs. Finally, a Tier 4 approach would use distributions of exposure variates that may be more appropriate for a site, such as the distribution of fish consumption rates for a specific body of water impacted by a facility. As in a Tier 2 approach, the risk assessment must supply the data and methods used for the site-specific distributions for exposure variates, and the site-specific estimates must be justified to and reproducible by the Districts and OEHHA.

1.8 References

CAPCOA, 1990. *CAPCOA Air Toxics Hot Spots Program Facility Prioritization Guidelines.* California Air Pollution Control Officers Association, July 1990.

OEHHA, 2003. Air Toxics Hot Spots Risk Assessment Guidelines: The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments.

OEHHA, 2008. Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. Available online at: http://www.oehha.ca.gov

OEHHA, 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. May 2009. Available online at: http://www.oehha.ca.gov

OEHHA, 2012. Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis. Available online at http://www.oehha.ca.gov

2 - Overview of Health Risk Assessment

2.1 The Model for Risk Assessment

The standard approach currently used for health risk assessment (HRA) was originally proposed by the National Academy of Sciences in the 1983 book: *Risk Assessment in the Federal Government: Managing the Process* (NAS, 1983) and was updated in the Academy's 1994 book: *Science and Judgment in Risk Assessment* (NAS, 1994). In 2009 the National Academy published *Science and Decisions: Advancing Risk Assessment* (NAS, 2009), in which a number of recommendations are made on improving the risk assessment process and expanding it to include community concerns and cumulative risks. The four steps involved in the risk assessment process are 1) hazard identification, 2) exposure assessment, 3) dose-response assessment, and 4) risk characterization. These four steps are briefly discussed below.

2.2 Hazard Identification

For air toxics sources, hazard identification involves the pollutant(s) of concern emitted by a facility, and the types of adverse health effects associated with exposure to the chemical(s), including whether a pollutant is a potential human carcinogen or is associated with other types of adverse health effects. For the Air Toxics Hot Spots Program (Hot Spots), the emitted substances that are addressed in a risk assessment are found in the list of substances designated in the ARB's *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report* (EICG Report), which is incorporated by reference therein (ARB, 2007*)*. This list of substances is contained in Appendix A of this document and the EICG Report. The list of substances also identifies those substances that are considered human carcinogens or potential human carcinogens.

2.3 Exposure Assessment

The purpose of the exposure assessment is to estimate the extent of public exposure to emitted substances. For the Hot spots program, in practice this means estimating exposures for those emitted substances for which potential cancer risk or noncancer health hazards for acute, repeated 8-hour, and chronic exposures will be evaluated. This involves emission quantification, modeling of environmental transport, evaluation of environmental fate, identification of exposure routes, identification of exposed populations, and estimation of short-term (e.g., 1-hour maximum), 8-hour average, and long-term (annual) exposure levels. These activities are described in Chapters 4 and 5. Chapter 5 also discusses the tiered approach to risk assessment.

The ARB's Emission Inventory Criteria and Guidelines (EICG) Report provides assistance in determining those substances that must be evaluated in an HRA and the reporting requirements of facilities, while the Hot Spots Analysis and Reporting Program (HARP) software can be used to model ground level concentrations at specific off-site

locations resulting from facility emissions. The United States Environmental Protection Agency (U.S. EPA) has adopted the AERMOD air dispersion model into its list of regulatory approved models, in place of the previously used ISCST3 model. AERMOD is a steady-state plume model that incorporates air dispersion based on planetary boundary layer turbulence structure and scaling concepts, including treatment of both surface and elevated sources, and both simple and complex terrain (U.S. EPA, 2009). The Air Resources Board recommends AERMOD for Hot Spots risk assessments. The AERMOD air modeling software will be incorporated into the HARP software, which allows the user to input all dispersion parameters directly into the program to generate air dispersion data. Alternatively, the air dispersion data may be generated separately from HARP using other air dispersion models, and then imported into HARP to generate risk estimates. Data imported into HARP must already be in the format required by HARP. HARP has the flexibility to generate a summary of the risk data necessary for an HRA by either of the above approaches.

Most of the toxicants assessed under the Hot Spots program are volatile organic compounds that remain as gases when emitted into the air. These chemicals are not subject to appreciable deposition to soil, surface waters, or plants. Therefore, human exposure via ingestion or dermal exposure, at least at concentrations typically encountered in the ambient air, is not considered for volatile organic compounds in the Hot Spots risk assessments. While some models indicate potential for dermal exposure to certain volatile organic compounds, at this time, the Hot spots program does not consider this pathway. Significant exposure to volatile organic toxicants emitted into the air occurs through the inhalation pathway, and this pathway is the primary consideration in the Hot Spots risk assessments. A small subset of Hot Spots substances consists of semi-volatile organic and metal toxicants emitted partially or totally as particles subject to deposition. Ingestion and dermal pathways as well as the inhalation pathway must be evaluated for these chemicals. A few of these semi-volatile organic and metal toxicants must also include the breast milk ingestion pathway. Additional ingestion pathways may also need to be evaluated depending on the pathways of exposure for the specific receptor of interest. Table 5.1 in Chapter 5, Table 6.4 in Chapter 6, and Table 7.1 in Chapter 7 list the substances that must be evaluated for multipathway impacts. HARP is designed to assess potential health impacts posed by substances that must be analyzed by a multipathway approach.

2.4 Dose-Response Assessment

Dose-response assessment is the process of characterizing the relationship between exposure to an agent and incidence of an adverse health effect in exposed populations. In quantitative carcinogenic risk assessment, the dose-response relationship is expressed in terms of a potency slope that is used to calculate the probability or risk of cancer associated with an estimated exposure. Cancer potency factors are expressed as the 95^{th} percent upper confidence limit of the slope of the dose response curve estimated assuming continuous lifetime exposure to a substance. Typically, potency factors are expressed as units of inverse dose (e.g., $(mg/kg\ BW/day)^{-1}$) or inverse concentration (e.g., $(\mu g/m^3)^{-1}$). It is assumed in cancer risk assessments that risk is directly proportional to dose and that there is no threshold for carcinogenesis.

The Office of Environmental Health Hazard Assessment (OEHHA) has compiled cancer potency factors, which should be used in risk assessments for the Hot Spots program, in Table 7.1. Cancer potency factors listed in Table 7.1 were derived either by the U.S. EPA or by OEHHA, underwent public and peer-review, and were adopted for use in the program. Chapter 8 describes procedures for use of potency values in estimating excess cancer risk. For a detailed description of cancer potency factors, refer to the *Technical Support Document for Cancer Potency Factors* (OEHHA, 2009).

For noncarcinogenic effects, dose-response data developed from animal or human studies are used to develop acute, 8-hour, and chronic noncancer Reference Exposure Levels (RELs). The acute, 8-hour and chronic RELs are defined as the concentration at which no adverse noncancer health effects are anticipated even in sensitive members of the general population, with infrequent one hour exposures, repeated 8-hour exposures over a significant fraction of a lifetime, or continuous exposure over a significant fraction of a lifetime, respectively. The most sensitive health effect is chosen to develop the REL if the chemical affects multiple organ systems. Unlike cancer health effects, noncancer health effects are generally assumed to have thresholds for adverse effects. In other words, injury from a pollutant will not occur until exposure to that pollutant has reached or exceeded a certain concentration (i.e., threshold) and/or dose. The acute, 8-hour, and chronic RELs are air concentrations intended to be below the threshold for health effects for the general population.

The actual threshold for health effects in the general population is generally not known with any precision. Uncertainty factors are applied to the Lowest Observed Adverse Effects Level (LOAEL) or No Observed Adverse Effects Level (NOAEL) or Benchmark Concentration values from animal or human studies to help ensure that the chronic, 8-hour and acute REL values are below the threshold for human health for nearly all individuals. This guidance manual provides the acute, 8-hour, and chronic Reference Exposure Levels in Tables 6.1 through 6.3. Some substances that pose a chronic or repeated 8-hour inhalation hazard may also present a chronic hazard via non-inhalation routes of exposure (e.g., ingestion of contaminated water, foods, or soils, and dermal absorption). The oral RELs for these substances are presented in Table 6.4. The methodology and derivations for acute, 8-hour, and chronic, RELs are described in the *Technical Support Document for the Derivation of Noncancer Reference Exposure Levels* (OEHHA, 2008).

2.5 Risk Characterization

This is the final step of risk assessment. In this step, modeled concentrations and exposure information, which are determined through exposure assessment, are combined with potency factors and RELs that are developed through dose-response assessment. The use of cancer potency factors to assess total cancer risk and the use of the hazard index approach for evaluating the potential for noncarcinogenic health effects are described in Chapter 8. Example calculations for determining (inhalation) cancer risk and noncancer acute, 8-hour, and chronic hazard quotients and hazard indices are presented in Appendix I. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

Under the Hot Spots Act, health risk assessments are to quantify both individual and population-wide health impacts (Health and Safety Code, Section 44306) (Appendix B). The health risk assessments are facility specific and the calculated risk should be combined for all pollutants emitted by a single facility. For example, cancer risk from multiple carcinogens is considered additive. For exposures to multiple non-carcinogen pollutants, a hazard index approach is applied for air contaminants affecting the same organ system. All substances emitted by the facility that are on the Hot Spots Act list of substances must be identified in the HRA, including those on the list that do not have a potency value or REL.

For assessing risk, OEHHA has developed two methods for determining dose via inhalation, dermal absorption, and ingestion pathways. These two methods, the point estimate approach and the stochastic exposure assessment approach, are described below and in Chapters 5 and 8. Detailed presentations of these methods can be found in*: Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012*)*.

2.5.1 *Point Estimate Approach*

OEHHA provides information in this document on average and high-end values for key exposure pathways (e.g., breathing rate for the inhalation exposure pathway). The average and high-end of point estimates in this document are defined in terms of the probability distribution of values for that variate. The mean represents the average values for point estimates and the 95th percentiles represent the high-end point estimates from the distributions identified in OEHHA (2012). Thus, within the limitations of the data, average and high-end point estimates are supported by the distribution.

Tier 1 of the tiered approach to risk assessment, which is briefly discussed in Section 2.5.3 and presented in more detail in Chapter 8, utilizes a combination of the average and high-end point estimates to more realistically estimate exposure in multipathway risk assessments. This method uses high-end exposure estimates for the pathways that are the main drivers of exposure and the average point estimate for the other non-driving exposure pathways. This approach will lessen the issue of compounding high-end exposure estimates, while retaining a health-protective approach for the more important exposure pathways. It is unlikely that an individual receptor would be on the high-end of exposure for all exposure pathways. See Chapter 8 for detailed discussions of how this multipathway methodology is applied to cancer and noncancer calculations. The HARP software can perform this analysis (referred to as the derived approach in the HARP software).

In addition to using an estimate of average and high-end consumption rates, cancer risk evaluations at individual receptors are presented for 9, 30, and 70-year exposure durations. The 9 and 30-year durations correspond to the average and high-end of residency time recommended by U.S. EPA (1997). The California data presented in Appendix L of the Exposure TSD (OEHHA, 2012) are generally supportive of the nationwide data. The 9 and 70-year exposure durations present potential impacts over the range of residency periods, while the 30-year exposure duration is recommended

for use as the basis for estimating cancer risk at the MEIR in all HRAs. Population-wide impacts should use the 70-year exposure duration.

The parameters used for all exposure durations assume exposure begins in the last trimester of pregnancy and progresses through the exposure duration of interest (e.g., 9, 30, or 70 years). These assumptions are thus protective of children. Children have higher intake rates on a per kilogram body weight basis (e.g., they breathe, drink and eat more per kg body weight than adults) and thus receive a higher dose from contaminated media. See Chapter 5 for the point estimates that can be used to estimate impacts for children. Chapters 5 and 8 discuss how to calculate cancer risk based on various exposure durations and point estimates. Appendix I contains an example calculation and Chapter 9 clarifies how to present the findings in an HRA.

2.5.2 *Stochastic Exposure Assessment*

OEHHA was directed under the Air Toxics "Hot Spots" program (SB 1731, Calderon, stat. 1992; Health and Safety Code Section 44360(b)(2)) to develop a "likelihood of risk" approach to risk assessment. To satisfy this requirement, OEHHA developed a stochastic approach to risk assessment that utilizes distributions for exposure variates such as breathing rate and water consumption rate rather than a single point estimate. The variability in exposure can be propagated through the risk assessment model using the distributions as input and a Monte Carlo or similar method. The result of such an analysis is a range of risks that at least partially characterizes variability in exposure.

Distributions of key exposure variates that are presented in the *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012) were taken from the literature, if adequate, or developed from raw data of original studies. Intake variates such as vegetable consumption are relatively data rich; for these variates reasonable probability distributions can be constructed. However, the data necessary to characterize the variability in risk assessment variates are not always available. For example, for the fate and transport variates (e.g., fish bioaccumulation factors), there are only a few measurements for a given chemical available which precludes the adequate characterization of a probability distribution. We only developed distributions for those key exposure variates that were adequately characterized by data. Development of distributions is described in detail in the *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012).

2.5.3 *Tiered Approach to Risk Assessment*

OEHHA recommends using a tiered approach to risk assessment. Tier 1 is a standard point estimate approach using the recommended point estimates presented in this document. If site-specific information is available to modify some point estimates developed in the *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012) and is more appropriate to use than the recommended point estimates in this document, then Tier 2 allows use of that site-specific information. Site-specific information should be presented to the District before being used. The District may contact OEHHA for additional advice. Note that all non-default variates need to be adequately justified to OEHHA and the Districts to be used. In Tier 3, a stochastic approach to exposure assessment is used with the data distributions developed in the TSD (OEHHA, 2012) and presented in this document. Tier 4 is also a stochastic approach but allows for utilization of site-specific distributions, if they are justifiable (to OEHHA and the Districts) and more appropriate for the site under evaluation than those recommended in this document. Persons preparing an HRA that has a Tier 2 through Tier 4 evaluation must also include the results of a Tier 1 evaluation. Tier 1 evaluations are required for all HRAs prepared for the Hot Spots Program to promote consistency across the state for all facility risk assessments and allow comparisons across facilities. Chapter 8 provides a summary of the tiered approach and the TSD (OEHHA, 2012) discusses it in detail. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

2.6 References

ARB, 2007. *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report* (EICG Report).

NAS, 1983. National Academy of Sciences. *Risk Assessment in the Federal Government: Managing the Process*. National Research Council. National Academy Press, Washington D.C.

NAS, 1994. National Academy of Sciences. *Science and Judgment in Risk Assessment.* National Research Council. National Academy Press, Washington D.C.

NAS, 2009. National Academy of Sciences. *Science and Decisions: Advancing Risk Assessment*. National Academy Press, Washington DC.

OEHHA, 2008. Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. Available online at: http://www.oehha.ca.gov

OEHHA, 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. May 2009. Available online at: http://www.oehha.ca.gov

OEHHA, 2012. *Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis*. Available online at http://www.oehha.ca.gov

U.S. EPA (2009). AERMOD Implementation Guide. Last Revised: March 19, 2009.

U.S. EPA, 1997. *Exposure Factors Handbook, Volume I, General Factors*. EPA/600/P-95/002Fa.

AERMOD Implementation Workgroup, U. S. Environmental Protection Agency. Online at: http://www.epa.gov/ttn/scram/7thconf/aermod/aermod_implmtn_guide_19March2009.pdf

3 - Hazard Identification - Air Toxics Hot Spots Emissions

3.1 The Air Toxics Hot Spots List of Substances and Emissions Inventory

For air toxics sources, hazard identification involves identifying pollutants of concern and whether these pollutants are potential human carcinogens or associated with other types of adverse health effects. For the Air Toxics Hot Spots (Hot Spots) Program, the emitted substances that are addressed in a health risk assessment (HRA) are found in the list of hazardous substances designated in the Air Resources Board's (ARB's) *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report* (EICG Report), which is incorporated by reference therein (ARB, 2007). This list of substances is contained in both Appendix A of this document and the EICG Report. The list of substances also identifies those substances that are considered human carcinogens or potential human carcinogens.

The substances included on the Hot Spots Program list of substances are defined in the statute as those substances found on lists developed by the following sources:

- International Agency for Research on Cancer (IARC);
- U.S. Environmental Protection Agency (U.S. EPA);
- U.S. National Toxicology Program (NTP);
- ARB Toxic Air Contaminant Identification Program List;
- Hazard Evaluation System and Information Service (HESIS) (State of California);
- Proposition 65 (Safe Drinking Water and Toxic Enforcement Act of 1986) list of carcinogens and reproductive toxicants (State of California);
- Any additional substance recognized by the State Board as presenting a chronic or acute threat to public health when present in the ambient air.

All substances emitted by the facility that are on the Hot Spots Act list of substances must be identified in the HRA.

The ARB EICG Report (ARB, 2007) specifies that each facility subject to the Hot Spots Act must submit an Emission Inventory Report to the local air pollution control or air quality management district. This Emission Inventory Report must identify and account for all listed substances used, manufactured, formulated, or released by the facility. All routine, predictable releases must be reported. These inventory reports include the emission data necessary to estimate off-site levels of facility-released Hot Spots substances. These inventory reports will be discussed in further detail in Chapter 4. See Chapter 9 for an outline that specifies the content and recommended format for presenting the air dispersion modeling and HRA results. As presented in Appendix A, the EICG Report divides the list into three groups for reporting purposes. Potency or severity of toxic effects and potential for facility emission were considered in placing compounds into the three groups.

For the first group (listed in these guidelines in Appendix A-I), all emissions of these substances must be quantified in the HRA. For substances in the second group (listed in these guidelines in Appendix A-II), emissions are not quantified; however, facilities must report whether the substance is used, produced, or otherwise present on-site (i.e., these substances are simply listed in a table in the HRA). Lastly, substances in the third group (Appendix A-III) also only need to be reported in a table in the HRA if they are manufactured by the reporting facility.

Facilities that must comply with the Resource Conservation and Recovery Act and Comprehensive Environmental Response, Compensation and Liability Act (RCRA/CERCLA) requirements for risk assessment need to consult the California Department of Toxic Substances Control (DTSC) Remedial Project Manager to determine which substances must be evaluated in their risk assessment. Some RCRA/CERCLA facilities may emit substances which are not currently listed under the Hot Spots Program but which may require evaluation in a RCRA/CERCLA risk assessment.

3.2 References

ARB, 2007. *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report* (EICG Report).

4 - Air Dispersion Modeling

The information contained in this section is primarily an abbreviated version of the material found in Chapter 2 of the Air Toxics Hot Spots Risk Assessment Guidelines; Exposure Assessment and Stochastic Analysis Technical Support Document (OEHHA, 2012). Several references have been included in this section to indicate those areas that are covered in more detail in Chapter 2 of the Technical Support Document. However, some air dispersion concepts and procedures have been added to assist the reader in the health risk assessment (HRA) process. In particular, a brief summary of the Hot Spots Analysis and Reporting Program (HARP) software applicability to air dispersion analysis has been included. The HARP software has been developed by the Air Resources Board (ARB), in consultation with OEHHA and Air Pollution Control or Air Quality Management District (District) representatives. The HARP software is the recommended model for calculating and presenting HRA results for the Air Toxics Hot Spots Program (Hot Spots). Information on obtaining the HARP software can be found under the Hot Spots Program on the ARB's web site at *www.arb.ca.gov*. See Chapter 9 for an outline that specifies the content and recommended format for presenting the air dispersion modeling and HRA results.

The U.S. EPA has adopted the AERMOD air dispersion model into their list of regulatory approved models, in place of the previously used ISCST3 model. AERMOD is a steady-state plume model that incorporates air dispersion based on planetary boundary layer turbulence structure and scaling concepts, including treatment of both surface and elevated sources, and both simple and complex terrain (U.S. EPA, 2009). The Air Resources Board recommends AERMOD for Hot Spots risk assessments.

4.1 Air Dispersion Modeling in Exposure Assessment: Overview

Estimates of air concentrations of emitted toxicants in the surrounding community from a facility's air emissions are needed in order to determine cancer and noncancer risks. One approach to determining the concentration of air pollutants emitted from the facility is to do air monitoring in the surrounding community. However, there are a number of disadvantages to this approach. Ambient air monitoring is costly because good estimates of an annual average concentration typically require monitoring at least one day in six over a year. Because it is costly, monitoring is usually limited to a select number of pollutants, and a limited number of sites. There can be significant risks from some chemicals at or even below the monitoring detection limit, which can add considerable uncertainty to risk estimates if many of the measurements are below or near the detection limit. Monitoring measures not only facility emissions but also general ambient background as well. It can be difficult and expensive to distinguish between the two using monitoring, particularly if general ambient background levels are high relative to the contribution of facility emissions. These limitations often make it impractical to use monitoring in a program such as the Air Toxics Hot Spots program with hundreds of facilities.

Air dispersion models have several advantages over monitoring. Modeling can provide greater spatial detail and the costs are relatively cheap by comparison. For example, dispersion models can estimate the pollutant concentration in air at many receptor locations (hundreds to thousands) and for a multitude of averaging periods. Air dispersion models have been validated using air monitoring.

There are, however, uncertainties associated with the typical usage of air dispersion modeling. The use of meteorological data from the nearest airport may not ideally be the best representation of localized conditions. Gaussian plume air dispersion models ignore calm hours. This can bias model predictions towards underestimation. Some dispersion models offer limited chemical reactions within the algorithms; however, we generally assume the pollutant is inert for the near-field atmospheric travel time. This may bias estimated concentrations towards over-prediction for those pollutants that are highly reactive in the atmosphere. Air dispersion model results are only as good as the emissions estimates and emissions estimates can be uncertain. However, on the whole, the advantages of air dispersion modeling for a program like the Air Toxics Hot Spots far outweigh the disadvantages.

Professional judgment is required throughout the dispersion modeling process. The local air quality district has final authority on modeling protocols. The following guidance is intended to assist in the understanding of dispersion modeling for risk assessments.

Air dispersion modeling includes the following steps (see Figure 1):

1. Create an emission inventory of the toxic releases (Section 4.2)
2. Identify the source types (Section 4.3)
3. Identify the terrain type and land use (Section 4.4)
4. Determine the detail needed for the analysis: screening or refined (Section 4.5)
5. Identify the population exposure (Section 4.6)
6. Identify the receptor network (Section 4.7)
7. Obtain meteorological data (for refined air dispersion modeling only) (Section 4.8)
8. Select an air dispersion model (Section 4.9)
9. Prepare a modeling protocol and submit to the local Air District (hereafter referred to as “the District”) (Section 4.14)
10. Complete the air dispersion analysis
11. If necessary, redefine the receptor network and return to Step 10

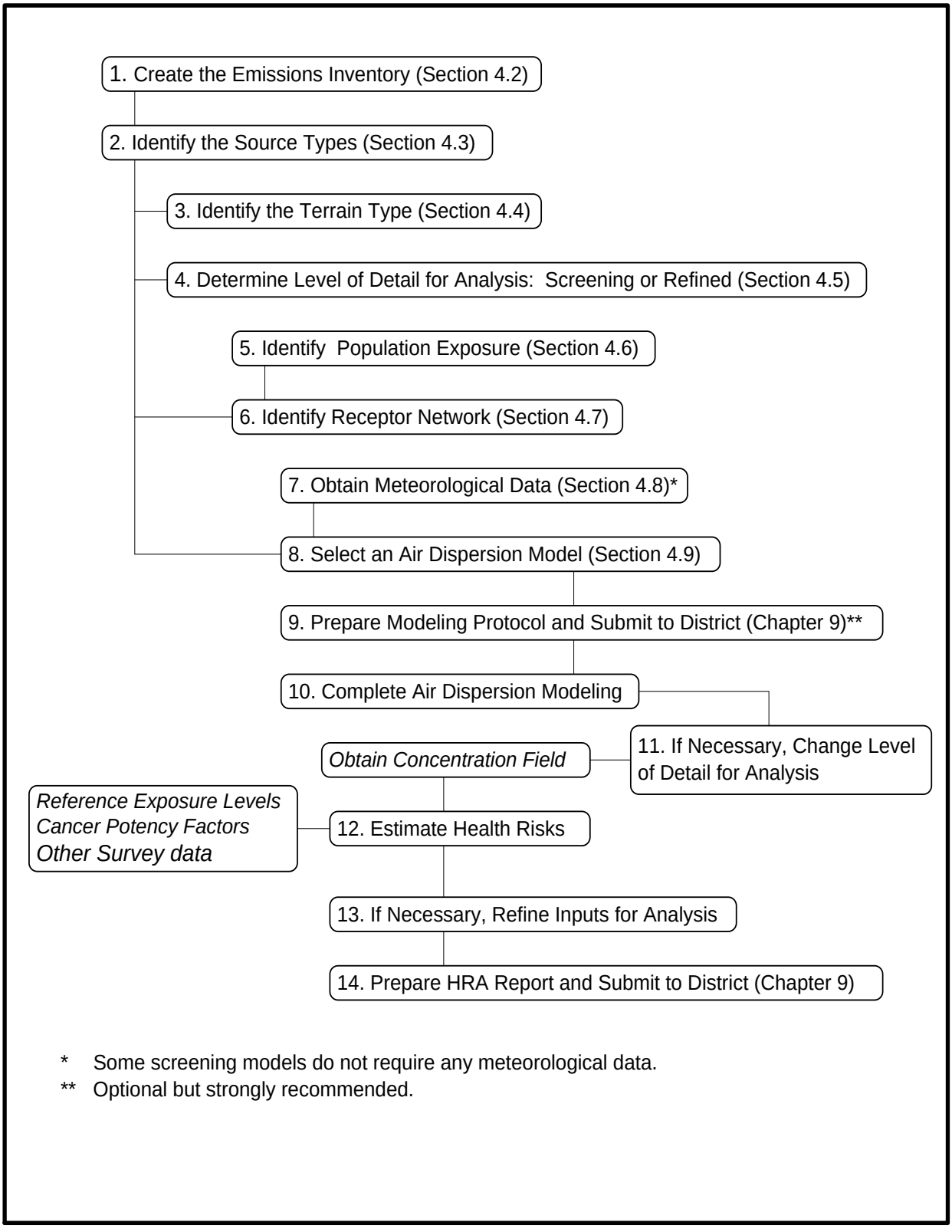
12. Complete the risk assessment

13. If necessary, refine the inputs and/or the model selection and return to Step 8

14. Present the HRA results (Chapter 9 provides an outline that specifies the content and recommended format of HRA results).

The output of the air dispersion modeling analysis includes a receptor field of ground level concentrations of the pollutant in ambient air. These concentrations can be used to estimate an inhaled or ingested dose for the estimation of multipathway cancer risk, or used to determine a hazard index for acute (inhalation), and chronic noncancer multipathway risks. It should be noted that in the Air Toxics "Hot Spots" program, facilities simulate the dispersion of the chemical emitted as an inert compound, and do not model any atmospheric transformations or dispersion of products from such reactions. The U.S. EPA Guideline on Air Quality Models (U.S. EPA, 2005) should be consulted when evaluating reactive pollutants for other regulatory purposes.

Figure 1 Overview of the Air Dispersion Modeling Process.



* Some screening models do not require any meteorological data.
** Optional but strongly recommended.

4.2 Emission Inventories

The Emission Inventory Reports (Inventory Reports) developed under the Hot Spots Program provide data to be used in the HRA and in the air dispersion modeling process. The Inventory Reports contain information regarding emission sources, emitted substances, emission rates, emission factors, process rates, and release parameters (area and volume sources may require additional release data beyond that generally available in Emissions Inventory reports). This information is developed according to the ARB's *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5)*, and the *Emission Inventory Criteria and Guidelines Report* (EICG Report), which is incorporated by reference therein (ARB, 2007*)*.

Updated emission data for process changes, emission factor changes, material/fuel changes, or shutdown must be approved by the District prior to the submittal of the health risk assessment (HRA). Ideally, the District review of updated emissions could be completed within the modeling protocol. In addition, it must be stated clearly in the risk assessment if the emission estimates are based on updated or revised emissions (e.g., emission reductions). This section summarizes the requirements that apply to the emission data which are used for Air Toxics "Hot Spots" Act risk assessments.

4.2.1 *Air Toxics Hot Spots Emissions*

As noted in Chapter 3, Hazard Identification, the HRA should identify all substances emitted by the facility, which are on the Hot Spots Act list of substances (see Appendix A of the Guidance Manual or the EICG Report). The EICG Report specifies that Inventory Reports must identify and account for all listed substances used, manufactured, formulated, or released by the facility. All routine, predictable releases must be reported. Under the regulations, the list is divided into three groups for reporting purposes. The first group (listed in Appendix A-I of the Inventory Guidelines Report) has all pollutants whose emissions must be quantified. The second group (listed in Appendix A-II of the Inventory Guidelines Report) includes substances where emissions do not need to be quantified; however, facilities must report whether the substance is used, produced, or otherwise present on-site. The third group (listed in Appendix A-III of the Emissions Inventory Guidelines Report) includes substances whose emissions need not be reported unless the substance is manufactured by the facility. Chemicals or substances in the second and third groups should be listed in a table in the risk assessment.

Facilities that must comply with the Resource Conservation and Recovery Act and Comprehensive Environmental Response, Compensation and Liability Act (RCRA/CERCLA) requirements for risk assessment need to consult the Department of Toxic Substances Control (DTSC) Remedial Project Manager to determine which substances must be evaluated in their risk assessment in addition to the list of "Hot Spots" chemicals. Some RCRA/CERCLA facilities may emit chemicals that are not currently listed under the "Hot Spots" Program. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.2.1.1 Emission Estimates Used in the Risk Assessment

The HRA must include emission estimates for all substances that are required to be quantified in the facility's emission inventory report. Specifically, HRAs should include both annual average emissions and maximum 1-hour emissions for each pollutant. Maximum 1-hour emissions are used for acute noncancer health impacts while annual emissions are used for chronic exposures (i.e., chronic and 8-hour noncancer health impacts or cancer risk assessment).

Emissions for each substance must be reported for individual emitting processes associated with unique devices within a facility. Total facility emissions for an individual air contaminant will be the sum of emissions, reported by process, for that facility. Information on daily and annual hours of operation, and relative monthly activity, must be reported for each emitting process. Devices and emitting processes must be clearly identified and described and must be consistent with those reported in the emissions inventory report.

The HRA should include tables that present the emission information (i.e., emission rates for each substance released from each process) in a clear and concise manner. The District may allow the facility operator to base the HRA on more current emission estimates than those presented in the previously submitted emission inventory report (i.e., actual enforceable emission reductions realized by the time the HRA is submitted to the District). If the District allows the use of more current emission estimates, the District must review and approve the new emissions estimates prior to use in the HRA. The HRA report must clearly state what emissions are being used and when any reductions became effective. Specifically, a table presenting emission estimates included in the previously submitted emission inventory report as well as those used for the HRA should be presented. The District should be consulted concerning the specific format for presenting the emission information. Chapter 9 provides an outline that specifies the content and recommended format of HRA results. A revised emission inventory report must be submitted to the District prior to submitting the HRA and forwarded by the District to the ARB, if revised emission data are used.

4.2.1.1.1 Molecular Weight Adjustments for the Emissions of Metal Compounds

For most of the Hot Spots toxic metals, the OEHHA cancer potency factors, acute and chronic RELs apply to the weight of the toxic metal atom contained in the overall compound. Some of the Hot Spots compounds contain various elements along with the toxic metal atom (e.g., "Nickel hydroxide", CAS number 12054-48-7, has a formula of H_2NiO_2). Therefore, an adjustment to the reported pounds of the overall compound is needed before applying the OEHHA cancer potency factor for "Nickel and compounds" to such a compound. This ensures that the cancer potency factor, acute or chronic REL is applied only to the fraction of the overall weight of the emissions that are associated with health effects of the metal. In other cases, the Hot Spots metals are already reported as the metal atom equivalent (e.g., CAS 7440-02-0, "Nickel"), and these cases do not use any further molecular weight adjustment. (Refer to Note [7] in Appendix A,

List of Substances in the EICG Report for further information on how the emissions of various Hot Spots metal compounds are reported.)

The appropriate molecular weight adjustment factors (MWAF) to be used along with the OEHHA cancer potency factors, acute and chronic RELs for Hot Spots metals can be found in the MWAF column[1] of the table containing OEHHA/ARB Approved Health Values for use in Hot Spots Facility Risk Assessments that is in Appendix L of this document.

As an example, the compound "Nickel hydroxide" has a molecular formula of H_2NiO_2. The atomic weight of each of the elements in this compound, and the fraction they represent of the total weight, are therefore as follows:

Element	Number of atoms	Atomic Weight	Fraction of Total Weight = MWAF
1 x Nickel (Ni)	1 x	58.70	58.70 / 92.714 = **0.6332** (MWAF for Nickel)
2 x Oxygen (O)	2 x	15.999	
2 x Hydrogen (H)	2 x	1.008	
Total Molecular Weight of H_2NiO_2:		92.714	

So, for example, assume that 100 pounds of "Nickel hydroxide" emissions are reported under CAS number 12054-48-7. To get the Nickel atom equivalent of these emissions, multiply by the listed MWAF (0.6332) for Nickel hydroxide:

- 100 pounds x 0.6332 = 63.32 pounds of Nickel atom equivalent.

This step should be completed prior to applying the OEHHA cancer potency factor for "Nickel and compounds" in a calculation for a prioritization score or risk assessment calculation. (Note - The HARP software automatically applies the appropriate MWAF for each Hot Spots chemical (by CAS number), so the emissions should not be manually adjusted when using HARP. Therefore, if using HARP, you would use 100 pounds for Nickel hydroxide and HARP will make the MWAF adjustment for you. If not using HARP, you would use 63.32 pounds.)

[1] The value listed in the MWAF column for Asbestos is not a molecular weight adjustment. This is a conversion factor for adjusting mass and fibers or structures. See Appendix C for more information on Asbestos reporting and risk assessment information or see the EICG report for reporting guidance.

4.2.1.2 Release Parameters

Emission release parameters (e.g., stack height and inside diameter, stack gas exit velocity, release temperature and emission source location in UTM coordinates) are needed as inputs to the air dispersion model. The Inventory Guidelines specify the release parameters that must be reported for each stack, vent, ducted building, exhaust site, or other site of exhaust release. Additional information may be required to characterize releases from non-stack (volume and area) sources; see U.S. EPA dispersion modeling guidelines or specific user's manuals. This information should also be included in the air dispersion section of the risk assessment. This information must be presented in tables included in the risk assessment. Note that some dimensional units needed for the dispersion model may require conversion from the units reported in the Inventory Report (e.g., Kelvin (K) vs. degrees Fahrenheit (°F)). Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.2.1.3 Operation Schedule

The HRA should include a discussion of the facility operation schedule and daily emission patterns. For AB2588 purposes, emissions should be reported based on routine and predictable operations. Weekly or seasonal emission patterns may vary and should be discussed. This is especially important in a refined HRA. Diurnal emission patterns should be simulated in the air dispersion model because of diurnal nature of meteorological observations. Diurnal evaluations are important to include since diurnal weather patterns and emission releases may cause significant differences in the concentration at a receptor of interest.

A table should be included listing the emission schedule on an hourly and yearly basis. In addition, the emission schedule and exposure schedule should corroborate any exposure adjustment factors used for approximating an inhaled dose. For more information about exposure adjustment factors, see Section 4.8.1. Alternatively, exposure adjustments can be made through refining the air dispersion analysis. See Section 4.11.1.2(h) for special case modeling or Appendix M. An alternative to including modeling that addresses diurnal influences would be to include a sensitivity study showing, and/or text explaining, the reason(s) why there are no significant differences due to diurnal influences on the emissions from the facility or at the receptor(s) of interest. For more guidance, you can contact the district or reviewing authority**.** Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.2.1.4 Emission Controls

The HRA should include a description of control equipment, the emitting processes it serves, and its efficiency in reducing emissions of substances on the Air Toxics “Hot Spots” list. The EICG Report requires that this information be included in the Inventory Reports, along with the emission data for each emitting process. If the control equipment did not operate full-time throughout the year, then the reported overall control efficiency must be adjusted to account for any predictable downtime of the

control equipment. Any entrainment of toxic substances to the atmosphere from control equipment should be accounted for; this includes fugitive releases during maintenance and cleaning of control devices (e.g., baghouses and cyclones). Contact the District for guidance with control equipment adjustments. Recommended default deposition rates that are used when calculating potential noninhalation health impacts are listed in Section 5.3.2. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.2.2 *Landfill Emissions*

Emission estimates for landfill sites should be based on testing required under Health and Safety Code, Section (HSC) 41805.5 (AB 3374, Calderon) and any supplemental AB 2588 source tests or emission estimates used to characterize air toxics emissions from landfill surfaces or through off-site migration. The District should be consulted to determine the specific Calderon data to be used in the HRA. The "Hot Spots" Program HRA for landfills should also include emissions of listed substances for all applicable power generation and maintenance equipment at the landfill site. Processes that need to be addressed include stationary internal combustion engines, flares, evaporation ponds, composting operations, boilers, and gasoline dispensing systems.

4.3 Source Characterization

Pollutants are released into the atmosphere in many different ways. The release conditions need to be properly identified and characterized to appropriately use the air dispersion models.

4.3.1 *Source Type*

Source types can be identified as point, line, area, or volume sources for input to the air dispersion model. Several air dispersion models have the capability to simulate more than one source type.

4.3.1.1 Point Sources

Point sources are probably the most common type of source and most air dispersion models have the capability to simulate them. Typical examples of point sources include exhaust stacks. Isolated vents from buildings are special examples of point sources.

4.3.1.2 Line Sources

The version 12345 or newer of the AERMOD can accommodate line sources. Line sources can be also treated as a special case of either an area or a volume source. Examples of line sources include: conveyor belts and rail lines, freeways, and busy roadways. Not all mobile sources may be subject to the Hot Spots program; however, non-motor vehicles that operate within a facility (e.g., ships, trains, and cranes, etc.) are subject to the Hot Spots program. For more information, see the ARB's Emission Inventory and Criteria Guidelines document or ARB's interpretation and guidance

memorandum to CAPCOA regarding mobile sources which are subject to the "Hot Spots" program. This memo can be found at http://www.arb.ca.gov/ab2588/motorv.pdf.

Mobile sources and rail lines are required to be evaluated under SB 352. SB 352 requires a risk assessment performed under the Hot Spots risk assessment guidance for proposed school sites within 500 feet of a busy roadway. Dedicated air dispersion models are available for motor vehicle emissions from roadways which are a special type of line source. These models (i.e., CALINE3, CAL3QHCR, and CALINE4) are designed to simulate the mechanical turbulence and thermal plume rise due to the motor vehicle activity on the roadway. However, these dedicated models use the Pasquill-Gifford dispersion stability classes for dispersion; the AERMOD dispersion model uses a more advanced continuous stability estimation method based on observations. The limitation with AERMOD is that the user needs to estimate initial mixing (Szo and Syo) for mechanical turbulence and thermal plume rise. Consult with the District prior to conducting roadway modeling to determine model use.

For practical information on how to simulate roadway emission dispersion using these models, see the California Air Pollution Control Officer's Association (CAPCOA) website at http://www.capcoa.org or the Sacramento Metropolitan AQMD (SMAQMD) website at http://www.airquality.org/ceqa/RoadwayProtocol.shtml. The SMAQMD has a document titled, "Recommended Protocol for Evaluating the Location of Sensitive Land Uses Adjacent to Major Roadways"(January, 2010). The ARB recommends this document for SB-352 risk assessments.

4.3.1.3 Area Sources

Emissions that are to be modeled as area sources are typical of fugitive sources characterized by non-buoyant emissions containing negligible vertical extent (e.g., no plume rise or emissions distributed over a large horizontal area).

Fugitive particulate (PM2.5, PM10, TSP) emission sources include areas of disturbed ground (e.g., open pits, parking lots) which may be present during operational phases of a facility's life. Also included are areas of exposed material (e.g., storage piles and slag dumps) and segments of material transport where potential fugitive emissions may occur (uncovered haul trucks or rail cars, emissions from unpaved roads). Fugitive emissions may also occur during stages of material handling where particulate material is exposed to the atmosphere (uncovered conveyors, hoppers, and crushers).

Other fugitive emissions emanating from many points of release may be modeled as area sources. Examples include fugitive emissions from valves, flanges, venting, and other connections that occur at ground level or at an elevated level or deck if on a building or structure. Modern dispersion models include an option for an initial vertical extent (Szo) where needed.

Modeling portable equipment as an area source is a case-by-case situation that should be discussed with the District or reviewing authority. Situations may exist where this type of operation is best represented as another type of release.

4.3.1.4 Volume Sources

Non-point sources with emissions containing an initial vertical extent should be modeled as volume sources. The initial vertical extent may be due to plume rise or a vertical distribution of numerous smaller sources over a given area. Examples of volume sources include buildings with natural fugitive or passive ventilation, and line sources such as conveyor belts and rail lines.

4.3.2 *Quantity of Sources*

The number of sources at a facility may influence the selection of the air dispersion model. Some dispersion models are capable of simulating only one source at a time, and are therefore referred to as single-source models (e.g., AERSCREEN).

In some cases, for screening purposes, single-source models may be used in situations involving more than one source using one of the following approaches:

- Combining all sources into one single “representative” source

In order to be able to combine all sources into one single source, the individual sources must have similar release parameters. For example, when modeling more than one stack as a single “representative” stack, the stack gas exit velocities and temperatures must be similar. In order to obtain a conservative estimate, the values leading to the higher concentration estimates should typically be used (e.g., the lowest stack gas exit velocity and temperature, the height of the shortest stack, and a receptor distance and spacing that will provide maximum concentrations, etc.).

- Running the model for each individual source and superimposing results

Superimposition of results of single sources of emissions is the actual approach followed by all the Gaussian models capable of simulating more than one source. Simulating sources in this manner may lead to conservative estimates if worst-case meteorological data are used or if the approach is used with a model that automatically selects worst-case meteorological conditions, especially wind direction. The approach will typically be more conservative the farther apart the sources are because each run would use a different worst-case wind direction.

Additional guidance regarding source merging is provided by the U.S. EPA (1995a). It should be noted that depending upon the population distribution, the total burden can actually increase when pollutants are more widely dispersed. If the total burden from the facility or zone of impact (see Section 4.6.1) could increase for the simplifying modeling assumptions described above, the District should be consulted.

4.4 Terrain Type

Two types of terrain characterizations are required to select the appropriate model. One classification is made according to land type and another one according to terrain topography.

4.4.1 *Terrain Type – Land Use*

Some air dispersion models (e.g., CALINE) use different dispersion coefficients (sigmas) depending on the land use over which the pollutants are being transported. The land use type is also used by some models to select appropriate wind profile exponents. Traditionally, the land type has been categorized into two broad divisions for the purposes of dispersion modeling: urban and rural. Accepted procedures for determining the appropriate category are those suggested by Irwin (1978): one based on land use classification and the other based on population.

The land use procedure is generally considered more definitive. Population density should be used with caution and should not be applied to highly industrialized areas where the population density may be low. For example, in low population density areas a rural classification would be indicated, but if the area is sufficiently industrialized the classification should already be "urban" and urban dispersion parameters should be used.

If the facility is located in an area where land use or terrain changes abruptly, for example, on the coast, the District should be consulted concerning the classification. If need be, the model should be run in both urban and rural modes and the District may require a classification that biases estimated concentrations towards over prediction. As an alternative, the District may require that receptors be grouped according to the terrain between source and receptor.

AERMOD is the U.S. EPA's preferred dispersion model for a wide range of applications in rural or urban conditions. The users should refer to section 5.0 of the AERMOD Implementation Guide to determine urban or rural conditions.

The Land Use and the Population Density Procedures discussed above are described as follows.

4.4.1.1 Land Use Procedure

(1) Classify the land use within the total area A, circumscribed by a 3 km radius circle centered at the source using the meteorological land use typing scheme proposed by Auer (1978) and shown in Table 4.1.

(2) If land use types I1, I2, C1, R2 and R3 account for 50 percent or more of the total area *A* described in (1), use urban dispersion coefficients. Otherwise, use appropriate rural dispersion coefficients.

4.4.1.2 Population Density Procedure

(1) Compute the average population density (*p*) per square kilometer with *A* as defined in the Land Use procedure described above. (Population estimates are also required to determine the exposed population; for more information see Section 4.6.3.)

(2) If *p* is greater than 750 people/km^2 use urban dispersion coefficients, otherwise, use appropriate rural dispersion coefficients.

Table 4.1 Identification and classification of land use types (Auer, 1978)

Used to define rural and urban dispersion coefficients in certain models.

Type	Use and Structures	Vegetation
I1	*Heavy Industrial* Major chemical, steel and fabrication industries; generally 3-5 story buildings, flat roofs	Grass and tree growth extremely rare; <5% vegetation
I2	*Light-moderate industrial* Rail yards, truck depots, warehouses, industrial parks, minor fabrications; generally 1-3 story buildings, flat roofs	Very limited grass, trees almost totally absent; <5% vegetation
C1	*Commercial* Office and apartment buildings, hotels; >10 story heights, flat roofs	Limited grass and trees; <15% vegetation
R1	*Common residential* Single family dwelling with normal easements; generally one story, pitched roof structures; frequent driveways	Abundant grass lawns and light-moderately wooded; >70% vegetation
R2	*Compact residential* Single, some multiple, family dwelling with close spacing; generally <2 story, pitched roof structures; garages (via alley), no driveways	Limited lawn sizes and shade trees; <30% vegetation
R3	*Compact residential* Old multi-family dwellings with close (<2 m) lateral separation; generally 2 story, flat roof structures; garages (via alley) and ashpits, no driveways	Limited lawn sizes, old established shade trees; <35% vegetation
R4	*Estate residential* Expansive family dwelling on multi-acre tracts	Abundant grass lawns and lightly wooded; >80% vegetation
A1	*Metropolitan natural* Major municipal, state, or federal parks, golf courses, cemeteries, campuses; occasional single story structures	Nearly total grass and lightly wooded; >95% vegetation
A2	Agricultural rural	Local crops (e.g., corn, soybean); >95% vegetation
A3	*Undeveloped* Uncultivated; wasteland	Mostly wild grasses and weeds, lightly wooded; >90% vegetation
A4	Undeveloped rural	Heavily wooded; >95% vegetation
A5	*Water surfaces* Rivers, lakes	

4.4.2 *Terrain Type - Topography*

Surface conditions and topographic features generate turbulence, modify vertical and horizontal winds, and change the temperature and humidity distributions in the boundary layer of the atmosphere. These in turn affect pollutant dispersion and models differ in their need to take these factors into account.

The classification according to terrain topography should ultimately be based on the topography at the receptor location with careful consideration of the topographical features between the receptor and the source. Differentiation of simple versus complex terrain is unnecessary with AERMOD. In complex terrain, AERMOD employs the well-known dividing-streamline concept in a simplified simulation of the effects of plume-terrain interactions. For other plume models, topography can be classified as follows:

4.4.2.1 Simple Terrain (also referred to as "Rolling Terrain")

Simple terrain is all terrain located below stack height including gradually rising terrain (i.e., rolling terrain). Note that *Flat Terrain* also falls in the category of simple terrain.

4.4.2.2 Intermediate Terrain

Intermediate terrain is terrain located above stack height and below plume height. The recommended procedure to estimate concentrations for receptors in intermediate terrain is to perform an hour-by-hour comparison of concentrations predicted by simple and complex terrain models. The higher of the two concentrations should be reported and used in the risk assessment.

4.4.2.3 Complex Terrain

Complex terrain is terrain located above plume height. Complex terrain models are necessarily more complicated than simple terrain models. There may be situations in which a facility is "overall" located in complex terrain but in which the nearby surroundings of the facility can be considered simple terrain. In such cases, receptors close to the facility in this area of simple terrain will "dominate" the risk analysis and there may be no need to use a complex terrain model. It is unnecessary to determine which terrain dominates the risk analysis for users of AERMOD.

4.5 Level of Detail: Screening vs. Refined Analysis

Air dispersion models can be classified according to the level of detail which is used in the assessment of the concentration estimates as "screening" or "refined". Refined air dispersion models use more robust algorithms capable of using representative meteorological data to predict more representative and usually less conservative estimates. Refined air dispersion models are, however, more resource intensive than their screening counterparts. It is advisable to first use a screening model to obtain conservative concentration estimates and calculate health risks. If the health risks are estimated to be above the threshold of concern, then use of a refined model to calculate

more representative concentration and health risk estimates would be warranted. There are situations when screening models represent the only viable alternative (e.g., when representative meteorological data are not available). The district or reviewing authority should be consulted to determine the appropriate method for determining the level of detail in the modeling analysis. The HARP software will incorporate the capability of using either representative meteorological data from AERMOD or the default meteorological conditions from the AERSCREEN model.

It is acceptable to use a refined air dispersion model in a "screening" mode for this program's health risk assessments. In this case, a refined air dispersion model is used:

- with worst-case meteorology instead of representative meteorology;

- with a conservative averaging period conversion factor to calculate longer term concentration estimates (see Section 4.10 for more discussion on screening air dispersion models and adjustments factors).

Note that use of worst case meteorology in a refined model is not the normal practice in New Source Review or Ambient Air Quality Standard evaluation modeling.

4.6 Population Exposure

The level of detail required for the analysis (e.g., screening or refined), and the procedures to be used in determining geographic resolution and exposed population require case-by-case analysis and professional judgment. The District should be consulted before beginning the population exposure estimates, and as results are generated, further consultation may be necessary. Some suggested approaches and methods for handling the breakdown of population and performance of a screening or detailed risk analysis are provided in this section.

In addition to estimating individual cancer risk at specific points such as the MEI (maximally exposed individual), OEHHA recommends determining the number of people who reside within the 1×10^{-6}, 1×10^{-5}, $1x\ 10^{-4}$, and higher cancer risk isopleths. For noncancer population evaluations, the number of people who reside within the 0.5, one, five, or higher hazard index isopleths should be reported. The HARP software can provide population exposure estimates as cancer burden or as the number of persons exposed to a selected (user identified) health risk/impact level. Information on obtaining the HARP software can be found under the Hot Spots Program on the ARB's web site at *www.arb.ca.gov*. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.6.1 *Zone(s) of Impact*

As part of the estimation of the population exposure for the cancer risk analysis, it is necessary to determine the geographic area affected by the facility's emissions. An initial approach to define a "zone of impact" surrounding the source is to generate an isopleth where the total excess lifetime cancer risk from inhalation exposure to all emitted carcinogens is greater than 10^{-6} (one in 1,000,000).

For noncarcinogens, a second, third, and fourth isopleth (to represent the chronic, 8-hour, and acute impacts) should be created to define the zone of impact for the hazard index from both inhalation and noninhalation pathways greater than or equal to 1.0. For clarity these isopleths may need to be presented on separate maps in the HRA.

Contact the District or reviewing authority to discuss inclusion of isopleth maps if all potential health risks fall within the facility boundary and no receptors have, or will ever, be present within the boundary (also see Section 4.7.1 for a discussion of on-site receptors).

The initial "zone of impact" can be determined as follows:

- Use a screening dispersion model (e.g., AERSCREEN) to obtain concentration estimates for each emitted pollutant at varying receptor distances from the source. Several screening models feature the generation of an automatic array of receptors which is particularly useful for determining the zone of impact. In order for the model to generate the array of receptors the user needs to provide some information normally consisting of starting distance, increment and number of intervals.

- Calculate total cancer risk and hazard index (HI) for each receptor location by using the methods provided in the risk characterization sections in Chapter 8 of the Air Toxics Hot Spots Risk Assessment Guidance Manual.

- Find the distance where the total inhalation cancer risk is equal to 10^{-6}; this may require redefining the receptor array in order to have two receptor locations that bound a total cancer risk of 10^{-6}. Next, find the distance where the chronic, 8-hour, and acute health hazard indices are declared significant by the District (e.g., acute, 8-hour, or chronic HI = 1.0).

Some Districts may prefer to use a cancer risk of 10^{-7} or an HI of 0.5 as the zone of impact. Therefore, the District should be consulted before modeling efforts are initiated. If the zone of impact is greater than 25 km from the facility at any point, then the District should be consulted. The District may specify limits on the area of the zone of impact. Ideally, these preferences would be presented in the modeling protocol (see Section 4.14).

Note that when depicting the risk assessment results, risk isopleths must present the total cancer and noncancer risk from both inhalation and noninhalation pathways. The zone of impact should be clearly shown on a map with geographic markers of adequate resolution (see Section 4.6.3.1). The text below discusses methodology for defining the zone of impact and has format recommendations. Chapter 9 provides an outline that specifies the content and recommended format of all HRA results.

The zone of impact can be defined once the exposure assessment (air dispersion modeling) process has determined the pollutant concentrations at each designated off-site receptor and a risk analysis (see Chapter 8) has been performed. For clarity, the cancer and noncancer zone(s) of impact should be presented on separate maps. A

map illustrating the carcinogenic zone of impact is required. The District may at its discretion ask for the map illustrating the potential carcinogenic zone of impact to identify the zone of impact for the minimum exposure pathways (inhalation, soil, dermal, and mother's milk) and the zone of impact for all applicable pathways of exposure (minimum pathways plus site/route dependent pathways). Two maps may be needed to accomplish this. The legend of these maps should state the level(s) used for the zone of impact and identify the exposure pathways that were included in the assessment.

The noncancer maps should also clearly identify the noncancer zones of impact. These include the acute (inhalation) zone of impact, 8-hour (inhalation) zone of impact and the chronic (including both inhalation, multipathway) zone of impact. The District may at its discretion require separate chronic inhalation and chronic multipathway zones of impact maps. For clarity, presentation of the two chronic zones of impact may also require two or more maps. The legend of these maps should state the level(s) used for the zone of impact and identify the exposure pathways (and target organs) that were included in the assessment. Further information regarding the methods for determination of hazard indices and cancer risk are discussed in Chapter 8 and Appendix I.

4.6.2 *Screening Population Estimates for Risk Assessments*

A screening risk assessment should include an estimate of the maximum exposed population. For screening risk assessments, a detailed description of the exposed population is not required. The impact area to be considered should be selected to be health protective (i.e., will not underestimate the number of exposed individuals). A health-protective assumption is to assume that all individuals within a large radius of the facility are exposed to the maximum concentration. If a facility must also comply with the RCRA/CERCLA risk assessment requirements, health effects to on-site workers may also need to be addressed. The DTSC's Remedial Project Manager should be consulted on this issue. The District should be consulted to determine the population estimate that should be used for screening purposes. Guidance for one screening method is presented here.

1. Use a screening dispersion model (e.g., AERSCREEN) to obtain concentration estimates for each emitted pollutant at varying receptor distances from the source. Several screening models feature the generation of an automatic array of receptors that is particularly useful for determining the zone of impact. In order for the model to generate the array of receptors, the user needs to provide some information normally consisting of starting distance, increment, and number of intervals.

2. Calculate the potential cancer risk and hazard index for each receptor location by using the methods provided in the risk characterization sections of this document (Chapter 8).

3. Find the distance where the potential cancer risk is equal to District specified levels (e.g., 10^{-6}); this may require redefining the receptor array in order to have

two receptor locations that bound a total cancer risk of 10^{-6}. This exercise should be repeated for the noncancer health impacts.

4. Calculate cancer burden by estimating the number of people in the grid and stipulate that all are exposed at the highest level.

4.6.3 *Refined Population Estimates for Risk Assessments*

The refined HRA requires a detailed analysis of the population exposed to emissions from the facility. Where possible, a detailed population exposure analysis provides estimates of the number of individuals in residences and offsite workplaces, as well as at sensitive receptor sites such as schools, daycare centers and hospitals. The District may require that locations with high densities of sensitive individuals be identified (e.g., schools, daycare centers, hospitals). These population analyses can include exposure estimates for workers and residents through the use of land use maps or other tools. The overall exposed residential and worker populations should be apportioned into smaller geographic subareas. The information needed for each subarea is:

1. The number of exposed persons, and
2. The receptor location at which the calculated ambient air concentration is assumed to be representative of the exposure to the entire population in the subarea.

A multi-tiered approach is suggested for the population analysis. Census tracts, which the facility could significantly impact, should be identified (see Section 4.6.3.1). A census tract should be divided into smaller subareas if it is close to the facility where ambient concentrations vary widely. The District may determine that census tracts provide sufficient resolution near the facility to adequately characterize population exposure or they may prefer the census information to be evaluated using smaller blocks. Further downwind where ambient concentrations are less variable, the census tract level may be acceptable to the District. The District may determine that the aggregation of census tracts (e.g., when the census tracts making up a city are combined) is appropriate for receptors that are considerable distances from the facility.

If a facility must also comply with the RCRA/CERCLA HRA requirements, health effects to on-site workers may also need to be addressed. The DTSC's Remedial Project Manager should be consulted on this issue. In some cases it may be appropriate to evaluate risks to on-site receptors. The district should be consulted about special cases for which evaluation of on-site receptors is appropriate, such as facilities frequented by the public or where people may reside (e.g., military facilities).

4.6.3.1 Census Tracts

For a refined risk assessment, the boundaries of census tracts can be used to define the geographic area to be included in the population exposure analysis. Digital maps showing the census tract boundaries in California can be obtained from "The Thomas

Guide"® on the World Wide Web. Statistics for each census tract can be obtained from the U.S. Census Bureau. The website address for the U.S. Census Bureau is http://www.census.gov. Numerous additional publicly accessible or commercially available sources of census data can be found on the World Wide Web. A specific example of a census tract is given in Appendix K. The HARP software includes U.S. census data and is a recommended tool for performing population exposure estimates.

The two basic steps in defining the area under analysis are:

(1) Identify the "zone of impact" (as defined previously in Section 4.6.1) on a map detailed enough to provide for resolution of the population to the subcensus tract level. (The U.S. Geological Survey (USGS) 7.5-minute series maps and the maps within the HARP software provide sufficient detail.) This is necessary to clearly identify the zone of impact, location of the facility, and sensitive receptors within the zone of impact. If significant development has occurred since the USGS survey, this should be indicated. A specific example of a 7.5-minute series map is given in Appendix K.

(2) Identify all census tracts within the zone of impact using a U.S. Bureau of Census or equivalent map (e.g., Thomas Brothers, HARP Software). If only a portion of the census tract lies within the zone of impact, then only the population that falls within the isopleth should be used in the population estimate or burden calculation. To determine this level of detail, local planning and zoning information may need to be collected. When this more detailed information is not available, then a less refined approach is to include the census data if the centroid of the census block falls within the isopleths of interest. The census tract boundaries should be transferred to a map, such as a USGS map (referred to hereafter as the "base map".)

An alternative approach for estimating population exposure in heavily populated urban areas is to apportion census tracts to a Cartesian grid cell coordinate system. This method allows a Cartesian coordinate receptor concentration field to be merged with the population grid cells. This process can be computerized and minimizes manual mapping of centroids and census tracts. The HARP software includes this function and will provide population estimates that are consistent with the methodology discussed here.

The District may determine that aggregation of census tracts (e.g., which census tracts making up a city can be combined) is appropriate for receptors that are located at considerable distances from the facility. If the District permits such an approach, it is suggested that the census tract used to represent the aggregate be selected in a manner to ensure that the approach is health protective. For example, the census tract included in the aggregate that is nearest (downwind) to the facility should be used to represent the aggregate.

4.6.3.1.1 Subcensus Tract

Within each census tract are smaller population units. These units [urban block groups (BG) and rural enumeration districts (ED)] contain about 1,100 persons. BGs are

further broken down into statistical units called blocks. Blocks are generally bounded by four streets and contain an average of 70 to 100 persons. However, this range in population is an average and population units may vary significantly. In some cases, the EDs are very large and identical to a census tract.

The area requiring detailed (subcensus tract) resolution of the exposed residential and worker population will need to be determined on a case-by-case basis through consultation with the District. The District may determine that census tracts provide sufficient resolution near the facility to adequately characterize population exposure.

Employment population data can be obtained at the census tract level from the U.S. Census Bureau or from local planning agencies. This degree of resolution will generally not be sufficient for most risk assessments. For the area requiring detailed analysis, zoning maps, general plans, and other planning documents should be consulted to identify subareas with worker populations.

The boundaries of each residential and employment population area should be transferred to the base map.

4.6.4 *Sensitive Receptor Locations*

Individuals who may be more sensitive to toxic exposures than the general population are distributed throughout the total population. Sensitive populations may include young children and chronically ill individuals. The District may require that locations with high densities of sensitive individuals be identified (e.g., schools, nursing homes, residential care facilities, daycare centers, and hospitals). The HRA should state what the District requirements are regarding identification of sensitive receptor locations.

Although protection of sensitive individuals is incorporated into OEHHA's risk assessment methodology in both cancer risk and noncancer risk assessment, the assessment of risk at the specific location of such sensitive individuals (e.g., schools, hospitals, or nursing homes) may be useful to assure the public that such individuals are being considered in the analysis. For some chemicals (e.g., mercury and manganese) children have been specifically identified as the sensitive subpopulation for noncancer health impacts, so it can be particularly appropriate to assess school sites.

4.7 Receptor Siting

4.7.1 *Receptor Points*

The modeling analysis should contain a network of receptor points with sufficient detail (in number and density) to permit the estimation of the maximum concentrations. Locations that must be identified include:

- The maximum estimated off-site impact or point of maximum impact (PMI),
- The maximum exposed individual at an existing residential receptor (MEIR),
- The maximum exposed individual at an existing occupational worker receptor (MEIW).

Note that some situations may also require that on-site receptor (worker or residential) locations be evaluated. The risk assessor can contact the District or reviewing authority for guidance if on-site exposure situations are present at the emitting facility. However, these on-site locations should be included in the HRA. Some examples where the health impacts of on-site receptors may be appropriate could be military base housing, prisons, universities, day care facilities, or locations where the public may have regular access for the appropriate exposure period (e.g., a lunch time café or museum for acute exposures). When a receptor lives and works on the facility, site, or property, then these receptors should be evaluated and reported under both residential and worker scenarios and the one that is most health protective should be used for risk management decisions. The cancer risk estimates for the onsite residents may use a 30-year exposure duration while the 25-year exposure duration is used for a worker. Under a Tier 2 analysis, alternate exposure durations may be evaluated and presented with all assumptions supported.

All of these locations (i.e., PMI, MEIR, and MEIW) must be identified for potential multipathway carcinogenic and noncarcinogenic effects. It is possible that the estimated PMI, MEIR, and MEIW risk for cancer, chronic noncancer, 8-hour, and acute noncarcinogenic risks occur at different locations or that some of these evaluations may not be necessary (e.g., the receptor does not exist). For example, some facilities will not have off-site workers in the vicinity of the facility and will not need to evaluate worker exposure, or the exposure situation may only require the evaluation of short-term carcinogenic or acute noncancer impacts (see Section 8.2.10 for a discussion of short-term projects). The approval to revise the exposure assessment for a receptor, or to omit the MEIW receptor, should be verified in writing with the District or reviewing authority and included in the HRA.

Other sensitive receptor locations may also be of interest and required to be included in the HRA. The District or reviewing authority should be consulted to determine which sensitive receptor locations must be included.

The results from a screening model (if available) can be used to identify the area(s) where the maximum concentrations are likely to occur. Receptor points should also be located at the population centroids (see Section 4.7.2) and sensitive receptor locations (see Section 4.6.4). The exact configuration of the receptor array used in an analysis will depend on the topography, population distribution patterns, and other site-specific factors. All receptor locations should be identified in the HRA using UTM (Universal Transverse Mercator) coordinates and receptor number. The receptor numbers in the summary tables should match receptor numbers in the computer output (e.g., HARP output files). In addition to actual UTM coordinates, the block/street locations (i.e., north side of 3,000 block of Smith Street) should be provided in the HRA for the PMI, MEIR, and MEIW for carcinogenic and noncarcinogenic health effects. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.7.1.1 Receptor Height

To evaluate localized impacts, receptor height should be taken into account at the point of maximum impact on a case-by-case basis. For example, receptor heights may have to be included to account for receptors significantly above ground level. Flagpole receptors at the height of the breathing zone of a person may need to be considered when the source receptor distance is less than a few hundred meters. Consideration must also be given to the noninhalation pathway analysis which requires modeling of chemical deposition onto soil or water at ground level. For the inhalation pathway, a health protective approach is to select a receptor height from 0 meters to 1.8 meters that will result in the highest predicted downwind concentration. Final approval of this part of the modeling protocol should be with the District or reviewing authority.

4.7.2 *Centroid Locations*

For each subarea analyzed, a centroid location (the location at which a calculated ambient concentration is assumed to represent the entire subarea) should be determined. When population is uniformly distributed within a population unit, a geographic centroid based on the shape of the population unit can be used. If only a portion of the census tract lies within the isopleth or area of interest, then only the population that falls within the isopleth should be used in the calculation for population exposure. To determine this level of detail, local planning and zoning information may need to be collected. Where populations are not uniformly distributed, a population-weighted centroid may be used. Another alternative uses the concentration at the point of maximum impact within that census tract as the concentration to which the entire population of that census tract is exposed. While this less refined approach is commonly accepted, Districts should be contacted to approve this method prior to its use in a risk assessment.

The centroids represent locations that should be included as receptor points in the dispersion modeling analysis. Annual average concentrations should be calculated at each centroid using the modeling procedures presented in this chapter.

For census tracts and BG/EDs, judgments can be made using census tracts maps and street maps to determine the centroid location. At the block level, a geographic centroid is sufficient.

4.7.3 *Spatial Averaging*

Since the inception of the “Hot Spots” and California’s Air Toxics Programs, HRA results for an individual receptor have typically been based on air dispersion modeling results at a single point or location. With a few exceptions, this method has been traditionally used for all types of receptors (e.g., PMI, MEIR, MEIW, pathway receptors, etc.). The assumptions used in risk assessment are designed to prevent underestimation of health impacts to the public resulting in a health protective approach. However, basing risk estimates on a single highest point (PMI, MEIR, or MEIW) does not take into account that a person does not remain at one location on their property, or in one location at the

workplace over an extended period of time. Therefore, the average air concentration over a small area is likely to be more representative than using the air concentration at a single point, particularly in those situations where concentrations fall off rapidly around that single point. The concept of averaging air concentrations over a small area is known as spatial averaging.

In order to understand how spatial averaging can impact air dispersion modeling results with various types of facilities, the ARB, in conjunction with the OEHHA, performed sensitivity analyses to evaluate the impacts of spatially averaging air dispersion modeling results (see Appendix C of the Air Toxics Hot Spots Program Risk Assessment Guidelines: Technical Support Document for Exposure Assessment and Stochastic Analysis (EASA)). Based on these sensitivity analyses, it is reasonable and appropriate to include spatial averaging techniques in air toxic risk assessments as supplemental information to Tier 1 information (i.e., modeling results that are based on the air concentration from a single point or location). While all risk assessments must include results based on Tier 1 methodology, the spatially averaged concentrations around the point of interest (e.g., PMI, MEIR, MEIW, multipathway exposure evaluations, etc.) could also be included as an option in risk assessments and acceptable for risk management decisions subject to approval by the District or reviewing agency. Spatial averaging is an option for the purpose of additional refinement to the risk assessment.

A few reasons that support the inclusion of spatially averaged modeled concentrations in risk assessment include the following:

- Averaging results over a small domain will give a more representative picture of individual exposure and risk than an estimate based on one single location within their property.
- Spatial averaging will allow air dispersion modeling and risk assessment results to be characterized as the estimated concentration and risk in a discrete area of interest, rather than an exact value for a single location.
- From a risk communication standpoint, the ARB and OEHHA feel it is more appropriate to present the modeling output and the calculated health impacts as the potential impacts within a small or discrete area, rather than an exact value at a specific point on a grid or map.
- Spatial averaging is the recommended procedure in ARB's Lead Risk Management Guidelines (2001) and has been used in several complex source HRAs [e.g., Roseville Railyard (2004), Ports of LA/LB (2006), Port of Oakland (2008)].
- Spatially averaging the deposition concentrations over pasture land, a garden, or a water body for multipathway exposure scenarios is a planned upgrade for the HARP Software. This will provide an option that will refine multipathway exposure assessments. Average deposition on these types of areas (e.g., a water body) is not necessarily well represented by the single highest point of deposition, or deposition at the geographic center of the water body. Likewise, since produce is grown over the entire surface of the garden and cows graze the

entire pasture, deposition is better estimated by evaluating the entire area rather than using a single point.

4.7.3.1 Spatial Averaging Methodology

The spatial averaging sensitivity study in Appendix C of the EASA is based on simulating emissions from point, volume, area, and line sources. Most source types (e.g., point) are simulated as a small, medium or large source. Line sources are only simulated as small and large. In addition, meteorological data collected at five different locations in California were used. Nested spatial average grids of various domains were used to study the differences on the spatial average concentration. In the case of the 20 meter by 20 meter spatial average nested grid, the spatial average concentration showed little change over the PMI for medium and large sources. In the case for small sources, the spatial average concentration is approximately 45% to 80% of the PMI concentration. Individual source type and meteorological conditions will cause variations in these results.

The results of the spatial averaging sensitivity study in Appendix C of the EASA shows that sources with low plume rise that result in a PMI, MEIW, or MEIR located at or near the property fence line are most sensitive to spatial averaging. Source types with high plume rise (e.g., tall stacks) show a PMI far downwind where the concentration gradient is more gradual and therefore spatial averaging has a lesser effect. While spatial averaging can be used regardless of source size or the location of the PMI, the following conditions generally apply when a source is a good candidate for spatial averaging:

- The MEIR, MEIW, or PMI is located at the fence line or close to the emission source.
- The concentration gradient is high near the PMI. This is more associated with low level plumes such as fugitive, volume, area, or short stacks.
- A long term average is being calculated to represent a multi-year risk analysis based on one to five years of meteorological data. Note that spatial averaging should **not** be used for short term (acute) calculations.

In general, the method for calculating the spatial average in air toxic risk assessments includes the following steps:

1. Locate the point(s) of interest and receptor(s) (i.e., PMI, MEIW, MEIR, and any additional receptor locations of interest or concern) with a grid resolution spacing of no greater than five meters. To achieve this, two or more modeling runs with successively finer nested grid resolutions may be needed to find the final location where the nested grid that will be used for spatial averaging will be placed.

2. Center the spatial average nested grid on the each receptor's location of interest determined in step 1. Limit the nested grid to no larger than 20 meters by 20 meters or 400 square meters. Note that if a portion of the centered and nested grid falls within the facility boundary and the receptor location of interest is outside of the boundary, then adjustments to the nested grid to obtain the spatially-averaged concentration for the offsite receptor are reasonable. This may be done by either repositioning the nested grid to cover 400 square meters of off-boundary area surrounding the receptor or center the nested grid and delete any on-site grid points so that only the offsite grid points surrounding the receptor are used in the spatially averaged concentration. The grid resolution spacing should be no greater than five meters. With a five meter grid resolution, the 20 meter by 20 meter domain will result in 25 receptors. The size, shape, and placement of the domain and the resolution of points are subject to approval by the District, ARB, or other reviewing authority. See the Sections 4.7.3.1.2 and 4.7.3.1.3 below for additional discussion on domain sizing and grid spacing at worksites, pastures, gardens, and water bodies.

3. Some configurations of source activity and meteorological conditions result in a predominant downwind plume center line that is significantly askew from one of the four ordinate directions. In this case, a tilted nested grid is necessary to coincide with the dominant plume centerline. Polar receptors are easier to implement than a tilted rectangular grid. The domain of the polar receptor field should be limited to a 15 meter radius. See Appendix C of the EASA for detailed instructions on tilted polar receptors.

4. Calculate the arithmetic mean of the long term period average concentration (e.g., annual average) of the nested grid of receptors to represent the spatial average. This average is used in the risk calculations.

5. Document and include all methods, assumptions, data, maps, and files used in the spatial averaging analysis and clearly present this information in the risk assessment following the requirements of the District or reviewing authority. Note that in the update to the HARP software, functionality will be included that will assist with spatial averaging and the methodology discussed.

The following sections discuss the use of spatial averaging for various receptor types and exposure pathways.

4.7.3.1.1 Residential Receptors

Follow the steps in Section 4.7.3 outlining the spatial averaging methodology. To remain health protective when evaluating a residential receptor, spatial averaging should not take place using large nested domains. The domain used for spatial averaging should be no larger than 20 meters by 20 meters with a maximum grid spacing resolution of equal to or less than five meters. This domain represents an area

that is approximately the size of a small urban lot. The size of the domain and resolution of points shall be subject to approval by the District, ARB, or other reviewing authority.

4.7.3.1.2 Worker Receptors

Offsite worker locations (e.g. MEIW) may also be a candidate for spatial averaging. However, workers can be at the same location during almost their entire daily work shift (e.g., desk/office workers). When this is the situation, then the traditional method of using a single location and corresponding modeled concentration is appropriate. If spatial averaging is used, care should be taken to determine the proper domain size and grid resolution. Follow the steps in Section 4.7.3 outlining the spatial averaging methodology. To be consistent with the residential receptor assumptions and remain health protective, a modeling domain size no larger than 20 meters by 20 meters is recommended with a grid spacing resolution of equal to or less than five meters. However, if workers routinely and continuously move throughout the worksite over a space greater than 20 meters by 20 meters, then a larger domain may be considered.

The HRA or modeling protocol shall support all assumptions used, including, but not limited to, documentation for all workers showing the area where each worker routinely performs their duties and the percentage of time spent in those areas. The final domain size should not be greater than the smallest area of worker movement. Other considerations for determining domain size and grid spacing resolution may include an evaluation of the concentration gradients across the worker area. The grid spacing used within the domain to find the concentration that will be used to calculate health impacts should be sufficient in number and detail to obtain a representative concentration across the area of interest. The size of the domain and resolution of points shall be subject to approval by the District, ARB, or other reviewing authority.

4.7.3.1.3 Pastures, Gardens, or Water Bodies

The simplified approach of using the concentration (deposition rate) at the centroid, a specific point of interest, or the PMI location for an area being evaluated for noninhalation exposures (e.g., a body of water used for fishing, a pasture used for grazing, area of a garden, etc.) is acceptable for use in HRA. However, evaluating deposition concentrations over pasture land, a garden, or a water body for multipathway exposure scenarios using spatial averaging could give more representative estimates of the overall deposition rate. Use of spatial averaging in this application is subject to approval by the District, ARB, or other reviewing authority.

If spatial averaging will be done, follow the steps in Section 4.7.3.1 outlining the spatial averaging methodology. When using spatial averaging over the deposition area, care should be taken to determine the proper domain size to make sure it includes all reasonable areas of potential deposition. The size and shape of the area of interest (e.g., pasture or water body) should be identified and used for the modeling domain. The grid spacing or resolution used within the domain should be sufficient in detail to obtain a representative deposition concentration across the area of interest. One way

to determine the grid resolution is to include an evaluation of the concentration gradients across the deposition area. The HRA or modeling protocol shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the pasture, garden, or water body, maps, representative coordinates, grid resolution, concentration gradients, etc.). The size of the domain and grid resolution is subject to approval by the reviewing authority.

In lieu of following the details in the paragraph above, the approach used for the other receptors (e.g., MEIR, MEIW) that uses a domain size not greater than 20 meters by 20 meters, located on the PMI within the area of interest, with a maximum grid spacing resolution of five meters, can be used. This default refined approach would apply to deposition areas greater than 20 meters by 20 meters. For smaller deposition areas, the simplified approach of using the PMI for the area, the concentration at the centroid or a specific point of interest, or averaging over the actual smaller domain can be used. This again is subject to approval by the reviewing authority.

The HRA or modeling protocol shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the water body, pasture, or garden; all data; maps; representative coordinates, and etc.), and the details clarifying how and where the averaging was done (e.g., location and magnitude of concentration gradients, the grid spacing used).

4.8 Meteorological Data

Refined air dispersion models require hourly meteorological data. The first step in obtaining meteorological data should be to check with the District and the ARB for data availability. Other sources of data include the National Weather Service (NWS), National Climatic Data Center (NCDC), Asheville, North Carolina, ARB meteorological database (METDB), military stations and private networks. Meteorological data for a subset of NWS stations are available from the U.S. EPA Support Center for Regulatory Air Models (SCRAM). The SCRAM can be accessed at www.epa.gov/scram001/main.htm. All meteorological data sources should be approved by the District. Data not obtained directly from the District or the ARB should be checked for quality, representativeness, and completeness. It should be approved by the District before use. U.S. EPA provides guidance (U.S. EPA, 1995e) for these data. Meteorological data may need further processing. Data users can consult with the District or the ARB on how to process the raw meteorological data. The risk assessment should indicate if the District required the use of a specified meteorological data set. All memos indicating District approval of meteorological data should be attached in an appendix. If no representative meteorological data are available, screening procedures should be used as indicated in Section 4.10.

The analyst should acquire enough meteorological data to ensure that the worst-case meteorological conditions are represented in the model results. The US-EPA Guideline on Air Quality Models (U.S. EPA 2005) prefers that the latest five years of consecutive meteorological data be used to represent long term averages (i.e., cancer and chronic impacts). Previous OEHHA guidance allowed the use of the worst-case year to save

computer time. The processing speed of modern computers has increased to the point where processing five years of data over one year is no longer burdensome. However, the District may determine that one year of representative meteorological data is sufficient to adequately characterize the facility's impact. This may especially be the case when five years of quality consecutive data are not available.

To determine long term average concentrations the data can be averaged. For calculation of the one-hour maximum concentrations needed to evaluate acute effects, the worst-case year should be used in conjunction with the maximum hourly emission rate. For example, the long term average concentration and one-hour maximum concentration at a single receptor for five years of meteorological data are calculated below:

Year	Annual Average ($\mu g/m^3$)	Maximum One-Hour ($\mu g/m^3$)
1	7	100
2	5	80
3	9	90
4	8	110
5	6	90
5-year average	7	

In the above example, the long-term average concentration over five years is 7 $\mu g/m^3$. Therefore, 7 $\mu g/m^3$ should be used to evaluate carcinogenic and chronic effects (i.e., annual average concentration). The one-hour maximum concentration is the highest one-hour concentration in the five-year period. Therefore, 110 $\mu g/m^3$ is the peak one-hour concentration that should be used to evaluate acute effects.

The higher hourly concentration usually occurs when meteorological dispersion conditions become worse, such as, calm or light wind, inversion, etc. Inversion usually happens in late afternoon through early morning. As the sun goes down, the atmospheric temperature near surface starts to fall, usually faster than the temperature in the upper atmosphere causing a temperature inversion layer to form and extend downward. This inversion layer usually sustains throughout the night, and remains until early morning. Because of the inversion (cold air sitting on warm air at the top of the inversion layer), pollutant vertical mixing is very low in the morning.

When predicted concentrations are high and the mixing height is very low for the corresponding averaging period, the modeling results deserve additional consideration. For receptors in the near field, it is within the model formulation to accept a very low mixing height for short durations. However, it would be unlikely that the very low mixing height would persist long enough for the pollutants to travel into the far field. In the

event that the analyst identifies any of these time periods, they should be discussed with the District on a case-by-case basis.

4.8.1 *Meteorological Data Formats*

Most short-term dispersion models require input of hourly meteorological data in a format which depends on the model. U.S. EPA provides software for processing meteorological data for use in U.S. EPA recommended dispersion models. U.S. EPA recommended meteorological processors include the Meteorological Processor for Regulatory Models (MPRM), PCRAMMET, and AERMET. Use of these processors will ensure that the meteorological data used in an U.S. EPA recommended dispersion model will be processed in a manner consistent with the requirements of the model.

Meteorological data for a subset of NWS stations are available on the World Wide Web at the U.S. EPA SCRAM address, http://www.epa.gov/scram001.

4.8.2 *Treatment of Calms*

Calms are hours when the wind speed is below the starting threshold of the anemometer. Gaussian plume models require a wind speed and direction to estimate plume dispersion in the downwind direction.

U.S. EPA's policy is to disregard calms until such time as an appropriate analytical approach is available. The recommended U.S. EPA models contain a routine that eliminates the effect of the calms by nullifying concentrations during calm hours and recalculating short-term and annual average concentrations. Certain models lacking this built-in feature can have their output processed by U.S. EPA's CALMPRO program (U.S. EPA, 1984a) to achieve the same effect. Because the adjustments to the concentrations for calms are made by either the models or the postprocessor, actual measured on-site wind speeds should always be input to the preprocessor. These actual wind speeds should then be adjusted as appropriate under the current U.S. EPA guidance by the preprocessor.

Following the U.S. EPA methodology, measured on-site wind speeds of less than 1.0 m/s, but above the instrument threshold, should be set equal to 1.0 m/s by the preprocessor when used as input to Gaussian models. Calms are identified in the preprocessed data file by a wind speed of 1.0 m/s and a wind direction equal to the previous hour. For input to AERMOD, no adjustment should be made to the site specific wind data. AERMOD can produce model estimates for conditions when the wind speed may be less than 1 m/s but still greater than the instrument threshold. Some air districts provide pre-processed meteorological data for use in their district that treats calms differently. Local air districts should be consulted for available meteorological data. In addition, to reduce the number of calms and missing winds in the surface data, EPA has developed a pre-processor – AERMINUTE – to process 1-minute ASOS wind data for generating hourly average wind speed and directions for input to AERMET in Stage 2. The details can be found in the EPA's AERMINUTE User's Instructions at:

http://www.epa.gov/ttn/scram/models/aermod/aerminute_userguide_v11059_draft.pdf

If the fraction of calm hours is excessive, then an alternative approach may need to be considered to characterize dispersion. The Calpuff model modeling system can simulate calm winds as well as complex wind flow and therefore is a viable alternative. The local air district should be consulted for alternative approaches.

4.8.3 *Treatment of Missing Data*

Missing data refer to those hours for which no meteorological data are available from the primary on-site source for the variable in question. When missing values arise, they should be handled in one of the following ways listed below, in the following order of preference:

(1) If there are other on-site data, such as measurements at another height, they may be used when the primary data are missing. If the height differences are significant, corrections based on established vertical profiles should be made. Site-specific vertical profiles based on historical on-site data may also be appropriate to use if their determination is approved by the reviewing authority. If there is question as to the representativeness of the other on-site data, they should not be used.

(2) If there are only one or two missing hours, then linear interpolation of missing data may be acceptable, however, caution should be used when the missing hour(s) occur(s) during day/night transition periods.

(3) If representative off-site data exist, they may be used. In many cases this approach may be acceptable for cloud cover, ceiling height, mixing height, and temperature. This approach will rarely be acceptable for wind speed and direction. The representativeness of off-site data should be discussed and agreed upon in advance with the reviewing authority.

(4) An imputation methodology may be acceptable, provided it is well-documented, sufficiently justified, and properly applied.

(5) Failing any of the above, the data field should be coded as missing using missing data codes appropriate to the applicable meteorological pre-processor.

Appropriate model options for treating missing data, if available in the model, should be employed. Substitutions for missing data should only be made in order to complete the data set for modeling applications, and should not be used to attain the “regulatory completeness” requirement of 90%. That is, the meteorological data base must be 90% complete on a monthly basis (before substitution) in order to be acceptable for use in air dispersion modeling. The use of any data substitution technique should be thoroughly documented to provide the District or reviewing authority with all the information necessary to determine its approvability.

If the recommended methods for addressing missing meteorological data cannot be achieved as described, then alternative approaches should be discussed and developed in conjunction with the District or reviewing authority.

4.8.4 *Representativeness of Meteorological Data*

The atmospheric dispersion characteristics at an emission source need to be evaluated to determine if the collected meteorological data can be used to adequately represent atmospheric dispersion for the project.

Such determinations are required when the available meteorological data are acquired at a location other than that of the proposed source. In some instances, even though meteorological data are acquired at the location of the pollutant source, they still may not correctly characterize the important atmospheric dispersion conditions.

Considerations of representativeness are always made in atmospheric dispersion modeling whether the data base is "on-site" or "off-site." These considerations call for the judgment of a meteorologist or an equivalent professional with expertise in atmospheric dispersion modeling. If in doubt, the District should be consulted.

4.8.4.1 Spatial Dependence

The location where the meteorological data are acquired should be compared to the source location for similarity of terrain features. For example, in complex terrain, the following considerations should be addressed in consultation with the District:

- Aspect ratio of terrain, i.e., ratio of:
    - Height of valley walls to width of valley;
    - Height of ridge to length of ridge; and
    - Height of isolated hill to width of hill at its base
- Slope of terrain
- Ratio of terrain height to stack/plume height
- Distance of source from terrain (i.e., how close to valley wall, ridge, isolated hill)
- Correlation of terrain feature to prevailing meteorological conditions

Likewise, if the source is located on a plateau or plain, the source of meteorological data used should be from a similar plateau or plain.

Judgments of representativeness should be made only when sites are climatologically similar. Sites in nearby, but different air sheds, often exhibit different weather patterns. For instance, meteorological data acquired along a shoreline are not normally representative of inland sites and vice versa.

Meteorological data collected need to be examined to determine if drainage, transition, and synoptic flow patterns are characteristics of the source, especially those critical to the regulatory application. Consideration of orientation, temperature, and ground cover should be included in the review.

An important aspect of space dependence is height above the ground. Where practical, meteorological data should be acquired at the release height, as well as above or below, depending on the buoyancy of the source's emissions. AERMOD at a minimum requires wind observations at a height above ground between seven times the local surface roughness height and 100 meters.

4.8.4.2 Temporal Dependence

To be representative, meteorological data must be of sufficient duration to define the range of sequential atmospheric conditions anticipated at a site. As a minimum, one full year of on-site meteorological data is necessary to prescribe this time series. Multiple years of data are used to describe variations in annual and short-term impacts. Consecutive years from the most recent, readily available 5-year period are preferred to represent these yearly variations.

4.8.4.3 Further Considerations

It may be necessary to recognize the non-homogeneity of meteorological variables in the air mass in which pollutants disperse. This non-homogeneity may be essential in correctly describing the dispersion phenomena. Therefore, measurements of meteorological variables at multiple locations and heights may be required to correctly represent these meteorological fields. Such measurements are generally required in complex terrain or near large land-water body interfaces.

It is important to recognize that, although certain meteorological variables may be considered unrepresentative of another site (for instance, wind direction or wind speed), other variables may be representative (such as temperature, dew point, cloud cover). Exclusion of one variable does not necessarily exclude all. For instance, one can argue that weather observations made at different locations are likely to be similar if the observers at each location are within sight of one another - a stronger argument can be made for some types of observations (e.g., cloud cover) than others. Although by no means a sufficient condition, the fact that two observers can “see” one another supports a conclusion that they would observe similar weather conditions.

Other factors affecting representativeness include change in surface roughness, topography and atmospheric stability. Currently there are no established analytical or statistical techniques to determine representativeness of meteorological data. The establishment and maintenance of an on-site data collection program generally fulfills the requirement for “representative” data. If in doubt, the District should be consulted.

4.8.5 *Alternative Meteorological Data Sources*

It is necessary, in the consideration of most air pollution problems, to obtain data on site-specific atmospheric dispersion. Frequently, an on-site measurement program must be initiated. As discussed in Section 4.8.3, representative off-site data may be used to substitute for missing periods of on-site data. There are also situations where current or past meteorological records from a National Weather Service station may suffice. These considerations call for the judgment of a meteorologist or an equivalent professional with expertise in atmospheric dispersion modeling. More information on Weather Stations including: National Weather Service (NWS), military observations, supplementary airways reporting stations, upper air and private networks, is provided in "On-Site Meteorological Program Guidance for Regulatory Modeling Applications" (U.S. EPA, 1995e).

4.8.5.1 Recommendations

On-site meteorological data should be processed to provide input data in a format consistent with the particular models being used. The input format for U.S. EPA short-term regulatory models is defined in U.S. EPA's MPRM. The input format for AERMOD is defined in the AERMET meteorological pre-processor. Processors are available on the SCRAM web site. The actual wind speeds should be coded on the original input data set. Wind speeds less than 1.0 m/s but above the instrument threshold should be set equal to 1.0 m/s by the preprocessor when used as input to Gaussian models. Wind speeds below the instrument threshold of the cup or vane, whichever is greater, should be considered calm, and are identified in the preprocessed data file by a wind speed of 1.0 m/s and a wind direction equal to the previous hour. For input to AERMOD, no adjustment should be made to the site specific wind data. AERMOD can produce model estimates for conditions when the wind speed may be less than 1 m/s but still greater than the instrument threshold.

If data are missing from the primary source, they should be handled as follows, in order of preference: (1) substitution of other representative on-site data; (2) linear interpolation of one or two missing hours; (3) substitution of representative off-site data; (4) use of a well-documented and justified imputation methodology; or (5) coding as a missing data field, according to the discussions in Section 4.8.3. The use of any data substitution technique should be thoroughly documented to provide the District or reviewing authority with all the information necessary to determine its approvability.

If the data processing recommendations in this section cannot be achieved, then alternative approaches should be discussed and developed in conjunction with the District or reviewing authority.

4.8.6 *Quality Assurance and Control*

The purpose of quality assurance and maintenance is the generation of a representative amount (90% of hourly values for a year on a monthly basis) of valid data. For more information on data validation consult reference U.S. EPA (1995e). Maintenance may

be considered the physical activity necessary to keep the measurement system operating as it should. Quality assurance is the management effort to achieve the goal of valid data through plans of action and documentation of compliance with the plans.

Quality assurance (QA) will be most effective when following a QA Plan which has been signed-off by appropriate project or organizational authority. The QA Plan should contain the following information (paraphrased and particularized to meteorology from Lockhart):

1. Project description - how meteorology data are to be used
2. Project organization - how data validity is supported
3. QA objective - how QA will document validity claims
4. Calibration method and frequency - for data
5. Data flow - from samples to archived valid values
6. Validation and reporting methods - for data
7. Audits - performance and system
8. Preventive maintenance
9. Procedures to implement QA objectives - details
10. Management support - corrective action and reports

It is important for the person providing the quality assurance (QA) function to be independent of the organization responsible for the collection of the data and the maintenance of the measurement systems. Ideally, the QA auditor works for a separate company.

4.9 Model Selection

There are several air dispersion models that can be used to estimate pollutant concentrations and new ones are likely to be developed. U.S. EPA added AERMOD, which incorporates the PRIME downwash algorithm, to the list of preferred models in 2005 as a replacement to ISCST3. CalPuff was added in 2003. The latest version of the U.S. EPA recommended models can be found at the SCRAM Bulletin board located at http://www.epa.gov/scram001. However, any model, whether a U.S. EPA guideline model or otherwise, must be approved for use by the local air district. Recommended models and guidelines for using alternative models are presented in this section. All air dispersion models used to estimate pollutant concentrations for risk assessment analyses must be in the public domain. Classification according to terrain, source type and level of analysis is necessary before selecting a model (see Section 4.4). The selection of averaging times in the modeling analysis is based on the health effects of concern. Annual average concentrations are required for an analysis of carcinogenic or other chronic effects. One-hour maximum concentrations are required for analysis of acute effects.

4.9.1 *Recommended Models*

Recommended air dispersion models to estimate concentrations for risk assessment analyses are generally referenced in US EPA's Guideline on Air Quality Models

available at http://www.epa.gov/scram001. Currently AERMOD is recommended for most refined risk assessments in flat or complex terrain and in rural or urban environments[1]. In addition, CalPuff is available where spatial wind fields are highly variable or transport distances are large (e.g., 50 km). AERSCREEN is a screening model based on AERMOD. AERSCREEN can be used when representative meteorological data are unavailable. CTSCREEN is available for screening risk assessments in complex terrain. The most current version of the models should be used for risk assessment analysis. Some facilities may also require models capable of special circumstances such as dispersion near coastal areas. For more information on modeling special cases see Sections 4.12 and 4.13.

Most air dispersion models contain provisions that allow the user to select among alternative algorithms to calculate pollutant concentrations. Only some of these algorithms are approved for regulatory application such as the preparation of health risk assessments. The sections in this guideline that provide a description of each recommended model contain information on the specific switches and/or algorithms that must be selected for regulatory application.

To further facilitate the model selection, the District should be consulted for additional recommendations on the appropriate model(s) or a protocol submitted for District review and approval (see Section 4.14.1).

4.9.2 *Alternative Models*

Alternative models are acceptable if applicability is demonstrated or if they produce results identical or superior to those obtained using one of the preferred models referenced in Section 4.9.1. For more information on the applicability of alternative models refer to the following documents:

- U.S. EPA (2005). "Guideline on Air Quality Models" Section 3.2.2
- U.S. EPA (1992). "Protocol for Determining the Best Performing Model"
- U.S. EPA (1985a). "Interim Procedures for Evaluating Air Quality Models – Experience with Implementation"
- U.S. EPA (1984b). "Interim Procedures for Evaluating Air Quality Models (Revised)"

4.10 Screening Air Dispersion Models

A screening model may be used to provide a maximum concentration that is biased toward overestimation of public exposure. Use of screening models in place of refined modeling procedures is optional unless the District specifically requires the use of a refined model. Screening models are normally used when no representative meteorological data are available and may be used as a preliminary estimate to determine if a more detailed assessment is warranted.

[1] AERMOD was promulgated by U.S. EPA as a replacement to ISCST3 on November 9, 2006.

Some screening models provide only 1-hour average concentration estimates. Other averaging periods can be estimated based on the maximum 1-hour average concentration in consultation and approval of the responsible air district. Because of variations in local meteorology, the exact factor selected may vary from one district to another. Table 4.2 provides guidance on the range and typical values applied. The conversion factors are designed to bias predicted longer term averaging periods towards overestimation.

Table 4.2 Recommended Factors to Convert Maximum 1-hour Avg. Concentrations to Other Averaging Periods (U.S. EPA, 2011, 1995a; ARB, 1994).

Averaging Time	Range	Typical SCREEN3 Recommended	AERSCREEN Recommended
3 hours	0.8 - 1.0	0.9	1.0
8 hours	0.5 - 0.9	0.7	0.9
24 hours	0.2 - 0.6	0.4	0.6
30 days	0.2 - 0.3	0.3	
Annual	0.06 - 0.1	0.08	0.1

AERSCREEN automatically provides the converted concentration for longer than 1-hour averaging periods. For area sources, the AERSCREEN 3, 8, and 24-hour average concentration are equal to the 1-hour concentration. No annual average concentration is calculated. SCREEN3 values are shown for comparison purposes.

4.10.1 *AERSCREEN*

The AERSCREEN (U.S. EPA, 2011) model is now available and should be used in lieu of SCREEN3 with approval of the local District. AERSCREEN is a screening level air quality model based on AERMOD. AERSCREEN does not require the gathering of hourly meteorological data. Rather, AERSCREEN requires the use of the MAKEMET program which generates a site specific matrix of meteorological conditions for input to the AERMOD model. MAKEMET generates a matrix of meteorological conditions based on local surface characteristics, ambient temperatures, minimum wind speed, and anemometer height.

AERSCREEN is currently limited to modeling a single point, capped stack, horizontal stack, rectangular area, circular area, flare, or volume source. More than one source may be modeled by consolidating the emissions into one emission source.

4.10.2 *Valley Screening*

The Valley model is designed to simulate a specific worst-case condition in complex terrain, namely that of a plume impaction on terrain under stable atmospheric conditions. The algorithms of the VALLEY model are included in other models such as SCREEN3 and their use is recommended in place of the VALLEY model. The usefulness of the VALLEY model and its algorithms is limited to pollutants for which only long-term average concentrations are required. For more information on the Valley model consult the user's guide (Burt, 1977).

4.10.2.1 Regulatory Options

Regulatory application of the Valley model requires the setting of the following values during a model run:

- Class F Stability (rural) and Class E Stability (urban)
- Wind Speed = 2.5 m/s
- 6 hours of occurrence of a single wind direction (not exceeding a 22.5 deg sector)
- 2.6 stable plume rise factor

4.10.3 *CTSCREEN*

The CTSCREEN model (Perry et al., 1990) is the screening mode of the Complex Terrain Dispersion Model (CTDMPLUS). CTSCREEN can be used to model single point sources only. It may be used in a screening mode for multiple sources on a case by case basis in consultation with the District. CTSCREEN is designed to provide conservative, yet theoretically sounder, worst-case 1-hour concentration estimates for receptors located on terrain above stack height. Internally-coded time-scaling factors are applied to obtain other averages (see Table 4.3). These factors were developed by comparing the results of simulations between CTSCREEN and CTDMPLUS for a variety of scenarios and provide conservative estimates (Perry et al., 1990). CTSCREEN produces identical results as CTDMPLUS if the same meteorology is used in both models. CTSCREEN accounts for the three-dimensional nature of the plume and terrain interaction and requires detailed terrain data representative of the modeling domain. A summary of the input parameters required to run CTSCREEN is given in Table 4.4. The input parameters are provided in three separate text files. The terrain topography file (TERRAIN) and the receptor information file (RECEPTOR) may be generated with a preprocessor that is included in the CTSCREEN package. In order to generate the terrain topography file the analyst must have digitized contour information.

Table 4.3 Time-scaling factors internally coded in CTSCREEN

Averaging Period	Scaling Factor
3 hours	0.7
24 hour	0.15
Annual	0.03

Table 4.4 Input Parameters Required to Run CTSCREEN

Parameter	File
Miscellaneous program switches	CTDM.IN
Site Latitude and Longitude (degrees)	CTDM.IN
Site TIME ZONE	CTDM.IN
Meteorology Tower Coordinates (user units)	CTDM.IN
Source Coordinates: x and y (user units)	CTDM.IN
Source Base Elevation (user units)	CTDM.IN
Stack Height (m)	CTDM.IN
Stack Diameter (m)	CTDM.IN
Stack Gas Temperature (K)	CTDM.IN
Stack Gas Exit Velocity (m/s)	CTDM.IN
Emission Rate (g/s)	CTDM.IN
Surface Roughness for each Hill (m)	CTDM.IN
Meteorology: Wind Direction (optional)	CTDM.IN
Terrain Topography	TERRAIN
Receptor Information (coordinates and associated hill number)	RECEPTOR

4.11 Refined Air Dispersion Models

Refined air dispersion models are designed to provide more representative concentration estimates than screening models. In general, the algorithms of refined models are more robust and have the capability to account for site-specific meteorological conditions. For more information regarding general aspects of model selection see Section 4.9.

4.11.1 *AERMOD*

For a wide variety of applications in all types of terrain, the recommended model is AERMOD. AERMOD is a steady-state plume dispersion model for assessment of pollutant concentrations from a variety of sources. AERMOD simulates transport and dispersion from multiple point, area, or volume sources based on an up-to-date characterization of the atmospheric boundary layer. Sources may be located in rural or urban areas and receptors may be located in simple or complex terrain. AERMOD accounts for building wake effects (i.e., plume downwash) based on the PRIME building downwash algorithms. The model employs hourly sequential preprocessed meteorological data to estimate concentrations for averaging times from one hour to one year (also multiple years). AERMOD is designed to operate in concert with two pre-processor codes: AERMET processes meteorological data for input to AERMOD, and AERMAP processes terrain elevation data and generates receptor information for input to AERMOD. Guidance on input requirements may be found in the AERMOD Users Guide.

4.11.1.1 Regulatory Options

U.S. EPA regulatory application of AERMOD requires the selection of specific switches (i.e., algorithms) during a model run. All the regulatory options can be set by selecting the DFAULT keyword. The U.S. EPA regulatory options, automatically selected when the DFAULT keyword is used, are:

- Stack-tip downwash
- Incorporates the effects of elevated terrain
- Includes calms and missing data processing routines
- Does not allow for exponential decay for applications other than a 4-hour half life for SO_2

Additional information on these options is available in the AERMOD User's Guide.

4.11.1.2 Special Cases

a. Building Downwash:
AERMOD automatically determines if the plume is affected by the wake region of buildings when their dimensions are given. The specification of building dimensions does not necessarily mean that there will be downwash. See

Section 4.13.1 for guidance on how to determine when downwash is likely to occur.

b. Area Sources:
The area source algorithm in AERMOD estimates source emission strength by integrating an area upwind of the receptor location. Receptors may be placed within the area itself, downwind of the area or adjacent to the area. However, since the vertical distribution parameter (σ_z) goes to zero as the downwind distance goes to zero, the plume function solution is infinite for a downwind receptor distance of zero. In order to avoid such singularity in the plume function solution, the AERMOD model arbitrarily sets the plume function to zero when the receptor distance is less than one meter. As a result, the area source algorithm will not provide reliable solutions for receptors located within or adjacent to very small areas, with dimensions on the order of a few meters across. In these cases, the receptor should be placed at least one meter outside of the area.

c. Volume Sources:
The volume source algorithms in AERMOD require an estimate of the initial distribution of the emission source. The initial distribution of emissions for a volume source is in the horizontal and vertical directions. When modeling volume source emissions, one needs to provide initial horizontal (σ_{y0}) and vertical (σ_{z0}) dimensions as accurate as possible so that pollutant buoyancy and dispersion are also calculated accurately. US EPA's AERMOD User Guide provides suggested procedures to estimate these initial dimensions based on source type (Table 3-1) (U.S. EPA, 2004a).

d. Line Sources:
Examples of line sources include conveyor belts or roads. Depending on the source, these can be modeled three ways; as a line source, as a series of volume sources, or as an elongated area source. Where the emission source is neutrally buoyant, such as a conveyor belt, AERMOD can be used according to the user guide. In the event that the line source is a roadway, then additional considerations are required.

At the present time, CALINE (CALINE3, CAL3QHCR, and CALINE4) is the only model dedicated to modeling the enhanced mechanical and thermal turbulence created by motor vehicles traveling on a roadway. Of these, CAL3QHCR is the only model that accepts hourly meteorological data and can estimate annual average concentrations. However, CALINE uses the Pasquill-Gifford stability categories which are used in the ISCST model. AERMOD is now the preferred plume model over ISCST3 with continuous plume dispersion calculations based on observations but AERMOD does not include the enhanced roadway turbulence. Therefore, in the case where roadway emissions dominate the risk assessment, it may be most important to simulate the enhanced thermal and mechanical turbulence from motor vehicles with the CAL3QHCR model.

In the case where roadway emissions are a subset of all emissions for the risk assessment, including roadway emissions along with facility emissions, it may be best to use AERMOD for all emissions, roadway and facility, in order to maintain continuity with one dispersion model for the risk assessment. If AERMOD is used, it is important to consider that a major freeway may act similar to a large building which can cause some mixing and therefore initial vertical dispersion. This dispersion could be estimated with sensitivity studies based on wind speed, wind angle, roadway orientation, roadway width, and etc. This could be a complex estimation and needs very adept modeling skills. Roadway modeling should be evaluated on a case-by-case basis in consultation with the District or the reviewing authority.

Line sources inputs include a composite fleetwide emission factor, roadway geometry, hourly vehicle activity (i.e., diurnal vehicle per hour pattern), hourly meteorological data, and receptor placement. For practical information on how to simulate roadway emissions using these models, see CAPCOA's website at http://www.capcoa.org or the Sacramento Metropolitan AQMD (SMAQMD) website at http://www.airquality.org/ceqa/RoadwayProtocol.shtml. The SMAQMD has a document titled, "Recommended Protocol for Evaluating the Location of Sensitive Land Uses Adjacent to Major Roadways"(January, 2010).

e. Complex Terrain:
AERMOD uses the Dividing Streamline (Hc) concept for complex terrain. Above Hc, the plume is assumed to be "terrain following" in the convective boundary layer. Below Hc, the plume is assumed to be "terrain impacting" in the stable boundary layer. AERMOD computes the concentration at any receptor as a weighted function between the two plume states (U.S. EPA, 2004b).

f. Deposition:
AERMOD contains algorithms to model settling and deposition and requires additional information to do so including particle size distribution. For more information consult the AERMOD User's Guide (U.S. EPA, 2004a).

g. Diurnal Considerations:
Systematic diurnal changes in atmospheric conditions are expected along the coast (or any large body of water) or in substantially hilly terrain. The wind speed and direction are highly dependent on time of day as the sun rises and begins to heat the Earth. The sun heats the surface of the land faster than the water surface. Therefore the air above the land warms up sooner than over water. This creates a buoyant effect of warm air rising over land and the cool air from over water moves in to fill the void. Near large bodies of water (e.g., the ocean) this is known as a sea breeze. In complex terrain this is known as upslope flow as the hot air follows the terrain upwards. When the sun sets and the surface of the land begins to cool, the air above also cools and creates a draining effect. Near the water this is the land breeze; in complex terrain this is known as downslope or drainage flow. In addition, for the sea breeze, the atmospheric

conditions change rapidly from neutral or stable conditions over water to unstable conditions over land.

Near the large bodies of water the sea breeze is typical in the afternoon and the land breeze is typical for the early morning before sunrise. In complex terrain upslope flow is typical in the afternoon, while drainage flow is typical at night. Diurnal profiles need to be evaluated in conjunction with the facility emissions since sources can have varied emission profiles (e.g., some sources are continuously emitting while others are intermittent). These intermittent emission profiles may be influenced by diurnal patterns; therefore, they need to be evaluated to properly estimate potential exposures. For these reasons, it is especially important to simulate facility emissions with a hourly diurnal pattern reflective of source activity so that the risk assessment is representative of daily conditions.

h. 8-hour Modeling for the Offsite Worker's Exposure and Residential Exposure: If the ground level air concentrations from a facility operating 5 days a week, 8 hours per day have been estimated by a 24 hour per day annual average, an adjustment factor can be applied to estimate the air concentration that an offsite worker with the same schedule would be exposed to. The 24-hour annual average concentration is multiplied times 4.2.

If the meteorology during the time that the facility is emitting is used, hourly model simulations need to be post-processed to cull out the data needed for the offsite worker exposure. See Appendix M for information on how to calculate the refined offsite worker concentrations using the hourly raw results from the AERMOD air dispersion model. For more discussion on worker exposure, see Section 4.8.1.

Eight-hour exposure modeling can be used to evaluate the potential for health impacts (including effects of repeated exposures) in children and teachers exposed during school hours. Although not required in the HRA, 8-hour exposure modeling could also be performed at the discretion of the District to a residential scenario (i.e., the MEIR) where a facility operates only a portion of the day and exposure to residences are not adequately reflected by averaging concentrations over a 24 hour day.

4.11.1.3 HARP Dispersion Analysis

It is highly recommended that air dispersion analysis be performed using the HARP software. HARP can perform refined dispersion analysis by utilizing the U.S. EPA standard program AERMOD. In the future, the updated version of HARP will link the AERMOD outputs with risk assessment modules.

4.11.2 *CTDMPLUS*

CTDMPLUS is a Gaussian air quality model for use in all stability conditions in complex terrain. In comparison with other models, CTDMPLUS requires considerably more

detailed meteorological data and terrain information that must be supplied using specifically designed preprocessors. CTDMPLUS was designed to handle up to 40 point sources.

4.12 Modeling to Obtain Concentrations used for Various Health Impacts

The following section outlines how emissions and air dispersion modeling results are used or adjusted for a receptor that is exposed to either a non-continuous or continuously emitting source.

4.12.1 *Emission Rates for Cancer, Chronic, and Acute Health Impacts*

As discussed in Section 4.2.1.1, the HRA should include both annual average emissions and maximum 1-hour emissions for each pollutant emitted by the facility. Maximum 1-hour emissions are used for acute noncancer health impacts while annual emissions are used for chronic exposures (i.e., chronic and 8-hour noncancer health impacts or cancer risk assessment). When applying the emission rates in the air dispersion analysis, it is important not to artificially inflate or deplete the reported emission inventory.

For annual average emissions, the emissions are spread evenly over the entire year for continuous emitting sources. However, for sources where the emission patterns vary (i.e., non-continuous emitting sources), the emission rate should also account for the facility's emission schedule. If appropriate, the variable emissions rate option (e.g., hour-of-day) should be used in the air dispersion analysis. For more information consult the AERMOD User's Guide (U.S. EPA, 2004a). Also, when calculating emission rates for acute health impacts, it is important the emission rates never exceed the reported maximum 1-hour emissions.

4.12.2 *Modeling and Adjustments for Inhalation Cancer Risk at a Worksite*

Modeled long-term averages are typically used for cancer risk assessments for residents and workers. In an inhalation cancer risk assessment for an offsite worker, the long-term average should represent what the worker breathes during their work shift. However, the long-term averages calculated from AERMOD typically represent exposures for receptors that were present 24 hours a day and seven days per week (i.e., the schedule of a residential receptor). To estimate the offsite worker's concentration, there are two approaches. The more refined, complex, and time consuming approach is to post-process the hourly raw dispersion model output and examine the hourly concentrations that fall within the offsite worker's shift. See Appendix M for information on how to simulate the long-term concentration for the offsite worker that can be used to estimate inhalation cancer risk.

In lieu of post-processing the hourly dispersion model output, the more typical approach is to obtain the long-term average concentration as you would for modeling a residential receptor and approximate the worker's inhalation exposure using an adjustment factor. The actual adjustment factor that is used to adjust the concentration may differ from the example below based on the specifics of the source and worker receptor

(e.g., work-shift overlap). Once the worker's inhalation concentration is determined, the inhalation dose is calculated using additional exposure frequency and duration adjustments. See Chapter 5 for more information on the inhalation dose equation.

4.12.2.1 Non-Continuous Sources

When modeling a non-continuously emitting source (e.g., operating for eight hours per day and five days per week), the modeled long-term average concentrations are based on 24 hours a day and seven days per week for the period of the meteorological data set. Even though the emitting source is modeled using a non-continuous emissions schedule, the long-term concentration is still based on 24 hours a day and seven days per week. Thus, this concentration includes the zero hours when the source was not operating. For the offsite worker inhalation risk, we want to determine the long-term concentration the worker is breathing during their work shift. Therefore, the long-term concentration needs to be adjusted so it is based only on the hours when the worker is present. For example, assuming the emitting source and worker's schedules are the same, the adjustment factor is 4.2 = (24 hours per day/8 hours per shift)x(7 days in a week/5 days in a work week). In this example, the long term residential exposure is adjusted upward to represent the exposure to a worker. Additional concentration adjustments may be appropriate depending on the work shift overlap. These adjustments are discussed below.

The calculation of the adjustment factor from a non-continuous emitting source is summarized in the following steps.

a. Obtain the long-term concentrations from air dispersion modeling as is typical for residential receptors (all hours of a year for the entire period of the meteorological data set).

b. Determine the coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule.

c. Calculate the worker adjustment factor (WAF) using Equation 4.1. When assessing inhalation cancer health impacts, a discount factor (*DF*) may also be applied if the offsite worker's schedule partially overlaps with the source's emission schedule. The discount factor is based on the number of coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule (see Equation 4.2). The DF is always less than or equal to one.

Please note that worker adjustment factor does not apply if the source's emission schedule and the offsite worker's schedule do not overlap. Since the worker is not present during the time that the source is emitting, the worker is not exposed to the source's emission (i.e., the DF in Equation 4.2 becomes 0).

$$WAF = \frac{H_{residentid}}{H_{source}} \times \frac{D_{residentid}}{D_{source}} \times DF \qquad \text{Eq. 4.1}$$

Where:
WAF = the worker adjustment factor
$H_{residential}$= the number of hours per day the long-term residential concentration is based on (always 24 hours)
H_{source} = the number of hours the source operates per day
$D_{residential}$ = the number of days per week the long-term residential concentration is based on (always 7 days)
D_{source}= the number of days the source operates per week
DF = a discount factor for when the offsite worker's schedule partially overlaps the source's emission schedule. Use 1 if the offsite worker's schedule occurs within the source's emission schedule. If the offsite worker's schedule partially overlaps with the source's emission schedule, then calculate the discount factor using Equation 4.2 below.

$$DF = \frac{H_{coincident}}{H_{worker}} \times \frac{D_{coincident}}{D_{worker}} \qquad \text{Eq. 4.2}$$

Where:
DF = the discount factor for assessing cancer impacts
$H_{coincident}$ = the number of hours per day the offsite worker's schedule and the source's emission schedule overlap
$D_{coincident}$= the number of days per week the offsite worker's schedule and the source's emission schedule overlap
H_{worker} = the number of hours the offsite worker works per day
D_{worker}= the number of days the offsite worker works per week

d. The final step is to estimate the offsite worker's inhalation concentration by multiplying the worker adjustment factor with the long-term residential concentration. The worker's concentration is then plugged into the dose equation and risk calculation.

The HARP software has the ability to calculate worker impacts using an approximation factor and, in the future, it will have the ability to post-process refined worker concentrations using the hourly raw results from an air dispersion analysis.

4.12.2.2 Continuous Sources

If the source is continuously emitting, then the worker is assumed to breathe the long-term annual average concentration during their work shift. Equation 4.1 becomes one and no concentration adjustments are necessary in this situation when estimating the inhalation cancer risk. Note however, if an assessor does not wish to apply the assumption the worker breathes the long-term annual average concentration during the work shift, then a refined concentration can be post-processed as described in Appendix M. All alternative assumptions should be approved by the reviewing authority and supported in the presentation of results.

4.12.3 *Modeling and Adjustments for Noncancer 8-Hour RELs*

For 8-hour noncancer health impacts, we evaluate if the receptor (e.g., worker or resident) is exposed to an 8 hour average concentration, occurring daily, that exceeds the 8-hour REL. The 8 hour RELs were derived primarily for the offsite worker scenario. Although not required in an HRA, residential receptors can be evaluated with an 8-hour

REL at the discretion of the District or Reviewing authority. For ease, we use a worker receptor in this discussion and in the discussion below for a non-continuously emitting source. The daily average concentration is intended to represent the long-term average concentration the worker is breathing during the work shift. In general, there are two approaches for estimating the concentration used for the 8-hour hazard index. The more refined, complex, and time consuming approach is to post-process the hourly dispersion model output and use only the hourly concentrations that are coincident with the offsite worker hours to obtain the long-term concentration. See Appendix M for information on how to simulate the daily average concentration through air dispersion modeling.

Before proceeding through a refined analysis described in Appendix M, the assessor may wish to approximate the long-term concentration, as described below, and calculate the 8-hour hazard index. In lieu of post-processing the hourly dispersion model output described in Appendix M, the more typical approach is to obtain the long-term average concentration as you would for modeling a residential receptor and approximate the worker's inhalation concentration using an adjustment factor. The method for applying the adjustment factor is described in the section below.

The results from the 8-hour hazard index calculations should not be combined with the chronic or acute hazard indices. Each of the potential noncancer health impacts should be reported independently. See Chapter 8 for more discussion on calculating health impacts.

4.12.3.1 Non-Continuous Sources

When modeling a non-continuously emitting source (e.g., operating for eight hours per day and five days per week), the modeled long-term average concentrations are based on 24 hours a day and seven days per week for the period of the meteorological data set. Even though the emitting source is modeled using a non-continuous emissions schedule, the long-term concentration is still based on 24 hours a day and seven days per week. Thus, this concentration includes the zero hours when the source was not operating. For the offsite worker 8-hour hazard index, we want to determine the long-term average daily concentration the worker may be breathing during their work shift. This is similar to the cancer approximation adjustment method with one difference; there is no adjustment for partial overlap between the worker's schedule and the source's emission schedule. The reason for this difference in methodology is because the 8-hour REL health factors are designed for repeated 8-hour exposures and cannot readily be adjusted to other durations of exposure. The 8-hour RELs should be used for typical daily work shifts of 8-9 hours. For further questions, assessors should contact OEHHA, the District, or reviewing authority to determine if the 8-hour RELs should be used in your HRA. Any discussions or directions to exclude the 8-hour REL evaluation should be documented in the HRA.

When calculating the long-term average daily concentration for the 8-hour REL comparison, the long-term residential concentration needs to be adjusted so it is based only on the operating hours of the emitting source with the assumption the offsite

worker's shift falls within the emitting source's schedule. For example, assuming the emitting source operates 8 hours per day, 5 days per week and the offsite worker's schedules falls anywhere within this period of emissions, then the adjustment factor is 4.2 = (24 hours per day/8 hours of emissions per day)x(7 days in a week/5 days of emissions per week). In this example, the long term residential exposure is adjusted upward to represent the 8-hour exposure to a worker. No adjustments are applied for partial work shift overlap with the emitting source. If the source emits at night, then see Appendix N for additional recommendations.

Using the approximation factor is a screening method. If the 8-hour hazard index is above a threshold of concern with this method, the district or assessor should contact OEHHA for further guidance regarding the substance of concern. If necessary, further evaluation can be performed using the refined daily average modeling methodology discussed in Appendix M.

The calculation of the adjustment factor from a non-continuous emitting source is summarized in the following steps.

b. Obtain the long-term concentrations from air dispersion modeling as is typical for residential receptors (all hours of a year for the entire period of the meteorological data set).

c. Calculate the worker adjustment factor (WAF) using Equation 4.3. The source's emission schedule is assumed to overlap offsite worker's schedule. Note that the worker adjustment factor and the 8-hour inhalation REL do not apply if the source's emission schedule and the offsite worker's schedule do not overlap at some point.

$$WAF = \frac{H_{residentid}}{H_{source}} \times \frac{D_{residentid}}{D_{source}} \qquad \text{Eq. 4.3}$$

Where:

WAF = the worker adjustment factor
$H_{residential}$= the number of hours per day the long-term residential concentration is based on (always 24 hours)
H_{source} = the number of hours the source operates per day
$D_{residential}$ = the number of days per week the long-term residential concentration is based on (always 7 days).
D_{source}= the number of days the source operates per week.

d. The final step is to estimate the offsite worker's daily average inhalation concentration by multiplying the WAF with the long-term residential concentration. The worker's concentration is then used to calculate the 8-hour hazard index. This method using the approximation factor is a screening method. If the 8-hour hazard index is above a threshold of concern, the district or assessor should contact OEHHA for further guidance regarding the substance of concern.

In the future, the HARP software will have the ability to use 8-hour RELs, calculate worker impacts using an approximation factor, and to post-process worker concentrations using the hourly raw results from an air dispersion analysis.

4.12.3.2 Continuous Sources

If the source is continuously emitting, then the worker is assumed to breathe the long-term annual average concentration during their work shift and no concentration adjustments are made when estimating 8-hour health impacts. Note however, if an assessor does not wish to assume the worker breathes the long-term annual average concentration during the work shift, then a refined concentration can be post-processed as described in Appendix M. All alternative assumptions should be approved by the reviewing authority and supported in the presentation of results.

Note that 8-hour RELs are not typically used for continuously emitting sources for residential receptors. In this situation it is only necessary to estimate a chronic Hazard Index using the annual average concentrations and chronic RELs. However, there may be situations where the District may wish to assess an 8-hour Hazard Index, for example, where there are significant differences in modeled concentration of emissions during the day due to diurnal wind patterns.

4.12.4 *Modeling and Adjustment Factors for Noncancer Chronic RELs*

Potential chronic noncancer health impacts use the long-term annual average concentration regardless of the emitting facility's schedule. No adjustment factors should be used to adjust this concentration. Chronic RELs are used to assess not only residential health impacts, but in many cases worker health impacts as well. There are currently only a limited number of substances with an 8-hour inhalation REL, and a facility may emit only, or mostly, substances that currently have just a chronic REL. Until there are 8-hour RELs for all the Hot Spots substances emitted from a specified facility, we recommend determining the chronic HI for the MEIW to adequately protect the offsite worker.

The results from the chronic hazard index calculations are not combined with the 8-hour or acute hazard indices. All potential noncancer results should be reported independently. See Chapter 8 for more discussion on calculating health impacts.

4.12.5 *Modeling and Adjustments for Oral Cancer Potencies and Oral RELs*

When estimating the cancer risk or noncancer health impacts from noninhalation pathways, no adjustment is made to the long-term annual average concentration regardless of the emitting facility's schedule. Since the media (e.g., soil) at the receptor location where deposition takes place for noninhalation pathways is continuously present, the concentrations used for all noninhalation pathways are not adjusted (up or down) by an adjustment factor. However, some adjustments are made to the concentration once the pollutants reach the media, for example, pollutants undergo decay in soils. In addition, when the dose for each pathway is calculated, exposure adjustments may also be made. See Chapter 5 of this document and the Technical

Support Document for Exposure Assessment and Stochastic Analysis (OEHHA, 2012) to get more information on these types of adjustments. Oral cancer potencies and oral RELs are used to assess both residential or worker health impacts.

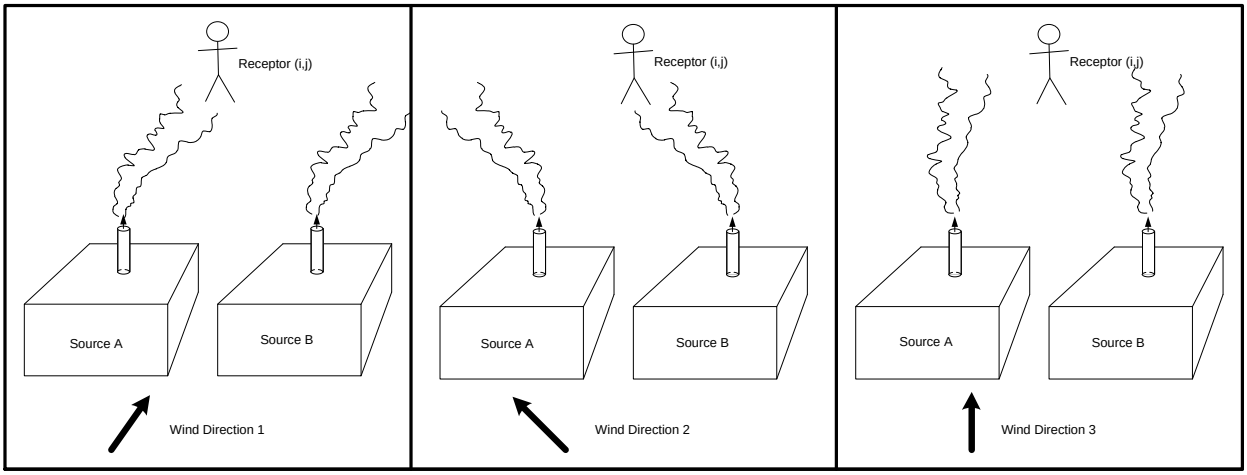
4.12.6 *Modeling One-Hour Concentrations using Simple and Refined Acute Calculations*

Modeled one-hour concentrations are needed for the acute health hazard index calculations. HARP has two methods to calculate this concentration: Simple and Refined. As an aid to understanding the differences between Simple and Refined, Figure 2 shows three possible conditions showing how wind direction may vary and impact a downwind receptor (i,j) differently from just two sources (A and B).

For the Simple calculation, HARP stores only the maximum one-hour concentration at each receptor (i,j) from each source (A and B) as the dispersion model marches down each hour of the simulation (e.g., one to five years of hourly data). At the end of the simulation period, HARP reports back only the maximum impacts at each receptor from each source regardless of which hour of the simulation period this occurred. For example, the Simple Maximum Acute Impacts would be the summation of Source A impacts from Wind Direction 1 and Source B impacts from Wind Direction 2 as shown in Figure 2.

For the Refined simulation, HARP stores each hourly concentration at each receptor (i,j) from each source. At the end of the simulation period, HARP evaluates the coincident impact at each receptor from all sources for each hour of the simulation period. In this case the maximum impacts will be identified by a particular hour of the period with associated wind speed, direction, and atmospheric conditions. For example, the Refined Maximum Acute impact from Sources A and B on receptor (i,j) could be from any wind direction (1,2, or 3) as shown in Figure 2. Since HARP stores all simulations for all sources – at all receptors – for all hours to calculate the refined impacts, there is great potential to fill large amounts of disk storage space. The Refined simulation provides a more representative picture of the maximum acute hazard index from a facility. The Simple calculation will provide an upper bound to the acute hazard index.

Figure 2 Acute Scenarios



4.13 Modeling Special Cases; Specialized Models

Special situations arise in modeling some sources that require considerable professional judgment; a few are outlined below. It is recommended that the reader consider retaining professional consultation services if the procedures are unfamiliar. The following sections, taken mostly from the document "On-Site Meteorological Program Guidance for Regulatory Modeling Applications" (U.S. EPA, 1995e), provide general information on data formats and representativeness. Some Districts may have slightly different recommendations from those given here.

4.13.1 *Building Downwash*

The entrainment of a plume in the wake of a building can result in the "downwash" of the plume to the ground. This effect can increase the maximum ground-level concentration downwind of the source. Therefore, stack sources must be evaluated to determine whether building downwash is a factor in the calculation of maximum ground-level concentrations.

The PRIME algorithm, included with AERMOD, has several advances in modeling building downwash effects including enhanced dispersion in the wake, reduced plume rise due to streamline deflection and increased turbulence, and continuous treatment of the near and far wakes (Schulman, 2000).

Complicated situations involving more than one building may necessitate the use of the Building Profile Input Program (BPIP) which can be used to generate the building dimension section of the input file of the ISC models (U.S. EPA, 1993). The BPIP program calculates each building's direction-specific projected width. The Building Profile Input Program for PRIME (BPIPPRM) is the same as BPIP but includes an algorithm for calculating downwash values for input into the PRIME algorithm which is contained in such models as AERMOD. The input structure of BPIPPRM is the same as that of BPIP.

4.13.2 *Deposition*

There are two types of deposition: wet deposition and dry deposition. Wet deposition is the incorporation of gases and particles into rain-, fog- or cloud water followed by a precipitation event and also rain scavenging of particles during a precipitation event. Wet deposition of gases is therefore more important for water soluble chemicals; particles (and hence particle-phase chemicals) are efficiently removed by precipitation events (Bidleman, 1988). Dry deposition refers to the removal of gases and particles from the atmosphere.

In the Air Toxics "Hot Spots" program, deposition is quantified for particle-bound pollutants and not gases. Wet deposition of water-soluble gas phase chemicals is thus not considered. When calculating pollutant mass deposited to surfaces without including depletion of pollutant mass from the plume, airborne concentrations remaining in the plume and deposition to surfaces can be overestimated, thereby resulting in overestimates of both the inhalation and multi-pathway risk estimates. However, neglecting deposition in the air dispersion model, while accounting for it in the multipathway health risk assessment, is a conservative, health protective approach (CAPCOA, 1987; Croes, 1988). Misapplication of plume depletion can also lead to possible underestimates of multi-pathway risk and for that reason no depletion is the default assumption. If plume depletion is incorporated, then some consideration for possible resuspension is warranted. An alternative modeling methodology accounting for plume depletion can be discussed with the Air District and used in an approved modeling protocol.

Although not generally used, several air dispersion models can provide downwind concentration estimates that take into account the upwind deposition of pollutants to surfaces and the consequential reduction of mass remaining in the plume. Air dispersion models having deposition and plume depletion algorithms require particle distribution data that are not always readily available. These variables include particle size, mass fraction, and density for input to AERMOD. In addition, the meteorological fields need to include additional parameters including relative humidity, precipitation, cloud cover, and surface pressure. Consequently, depletion of pollutant mass from the plume often is not taken into account.

In conclusion, multipathway risk assessment analyses normally incorporate deposition to surfaces in a screening mode, specifically by assigning a default deposition velocity of 2 cm/s for controlled sources and 5 cm/s for uncontrolled sources in lieu of actual measured size distributions (ARB, 1989). For particles (and particle-phase chemicals), the deposition velocity depends on particle size and is minimal for particles of diameter approximately 0.1-1 micrometer; smaller and larger particles are removed more rapidly.

4.13.3 *Short Duration Emissions*

Short-duration emissions (i.e., much less than an hour) require special consideration. In general, "puff models" provide a better characterization of the dispersion of pollutants having short-duration emissions. Continuous Gaussian plume models have traditionally

been used for averaging periods as short as about 10 minutes and are not recommended for modeling sources having shorter continuous emission duration.

4.13.4 *Fumigation*

Fumigation occurs when a plume that was originally emitted into a stable layer in the atmosphere is mixed rapidly to ground-level when unstable air below the plume reaches plume level. Fumigation can cause very high ground-level concentrations. Typical situations in which fumigation occurs are:

- Breaking up of a nocturnal radiation inversion by solar warming of the ground surface (rising warm unstable air); note that the break-up of a nocturnal radiation inversion is a short-lived event and should be modeled accordingly.

- Shoreline fumigation caused by advection of pollutants from a stable marine environment to an unstable inland environment

- Advection of pollutants from a stable rural environment to a turbulent urban environment

SCREEN3 incorporates concentrations due to inversion break-up and shoreline fumigation and is limited to maximum hourly evaluations. The Offshore and Coastal Dispersion Model incorporates overwater plume transport and dispersion as well as changes that occur as the plume crosses the shoreline – hourly meteorological data are needed from both offshore and onshore locations.

4.13.5 *Raincap on Stack*

The presence of a raincap or any obstacle at the top of the stack hinders the momentum of the exiting gas. The extent of the effect is a function of the distance from the stack exit to the obstruction and of the dimensions and shape of the obstruction.

On the conservative side, the stack could be modeled as having a non-zero, but negligible exiting velocity, effectively eliminating any momentum rise. Such an approach would result in final plume heights closer to the ground and therefore higher concentrations nearby. There are situations where such a procedure might lower the actual population-dose and a comparison with and without reduced exit velocity should be examined.

Plume buoyancy is not strongly reduced by the occurrence of a raincap. Therefore, if the plume rise is dominated by buoyancy, it is not necessary to adjust the stack conditions. (The air dispersion models determine plume rise by either buoyancy or momentum, whichever is greater.)

The stack conditions should be modified when the plume rise is dominated by momentum and in the presence of a raincap or a horizontal stack. Sensitivity studies with the SCREEN3 model, on a case-by-case basis, can be used to determine whether

plume rise is dominated by buoyancy or momentum. The District should be consulted before applying these procedures.

- Set exit velocity to 0.001 m/sec
- Turn stack tip downwash off
- Reduce stack height by 3 times the stack diameter

Stack tip downwash is a function of stack diameter, exit velocity, and wind speed. The maximum stack tip downwash is limited to three times the stack diameter in the AERMOD air dispersion model. In the event of a horizontal stack, stack tip downwash should be turned off and no stack height adjustments should be made. Note: This approach may not be valid for large (several meter) diameter stacks.

An alternative, more refined, approach could be considered for stack gas temperatures which are slightly above ambient (e.g., ten to twenty degrees Fahrenheit above ambient). In this approach, the buoyancy and the volume of the plume remain constant and the momentum is minimized.

- Turn stack tip downwash off
- Reduce stack height by 3 times the stack diameter ($3D_o$)
- Set the stack diameter (D_b) to a large value (e.g., 10 meters)
- Set the stack velocity to $V_b = V_o (D_o/D_b)^2$

Where V_o and D_o are the original stack velocity and diameter and V_b and D_b are the alternative stack velocity and diameter for constant buoyancy. This approach is advantageous when $D_b >> D_o$ and $V_b << V_o$ and should only be used with District approval.

In the presence of building downwash and in the event that PRIME downwash is being utilized in AERMOD, an alternative approach is recommended. PRIME algorithms use the stack diameter to define initial plume radius and to solve conservation laws. The user should input the actual stack diameter and exit temperature but set the exit velocity to a nominally low value (e.g., 0.001 m/s). Also since PRIME does not explicitly consider stack-tip downwash, no adjustments to stack height should be made.

Currently U.S. EPA is BETA testing options for capped and horizontal releases in AERMOD. It is expected that these options will replace the above guidance when BETA testing is complete.

4.13.6 *Landfill Sites*

Landfills should be modeled as area sources. The possibility of non-uniform emission rates throughout the landfill area should be investigated. A potential cause of non-uniform emission rates would be the existence of cracks or fissures in the landfill cap (where emissions may be much larger). If non-uniform emissions exist, the landfill should be modeled with several smaller areas assigning an appropriate emission factor to each one of them, especially if there are nearby receptors (distances on the same order as the dimensions of the landfill).

4.14 Specialized Models

Some models have been developed for application to very specific conditions. Examples include models capable of simulating sources where both land and water surfaces affect the dispersion of pollutants and models designed to simulate emissions from specific industries.

4.14.1 *Buoyant Line and Point Source Dispersion Model (BLP)*

BLP is a Gaussian plume dispersion model designed for the unique modeling problems associated with aluminum reduction plants, and other industrial sources where plume rise and downwash effects from stationary line sources are important.

4.14.1.1 Regulatory Application

Regulatory application of BLP model requires the selection of the following options:

- rural (IRU=l) mixing height option;
- default (no selection) for all of the following: plume rise wind shear (LSHEAR), transitional point source plume rise (LTRANS), vertical potential temperature gradient (DTHTA), vertical wind speed power law profile exponents (PEXP), maximum variation in number of stability classes per hour (IDELS), pollutant decay (DECFAC), the constant in Briggs' stable plume rise equation (CONST2), constant in Briggs' neutral plume rise equation (CONST3), convergence criterion for the line source calculations (CRIT), and maximum iterations allowed for line source calculations (MAXIT); and
- terrain option (TERAN) set equal to 0.0, 0.0, 0.0, 0.0, 0.0, 0.0.

For more information on the BLP model consult the user's guide (Schulman and Scire, 1980).

4.14.2 *Offshore and Coastal Dispersion Model (OCD)*

OCD (DiCristofaro and Hanna, 1989) is a straight-line Gaussian model developed to determine the impact of offshore emissions from point, area or line sources on the air quality of coastal regions. OCD incorporates "over-water" plume transport and dispersion as well as changes that occur as the plume crosses the shoreline. Hourly meteorological data are needed from both offshore and onshore locations. Additional data needed for OCD are water surface temperature, over-water air temperature, mixing height, and relative humidity.

Some of the key features include platform building downwash, partial plume penetration into elevated inversions, direct use of turbulence intensities for plume dispersion, interaction with the overland internal boundary layer, and continuous shoreline fumigation.

4.14.2.1 Regulatory Application

OCD has been recommended for use by the Minerals Management Service for emissions located on the Outer Continental Shelf (50 FR 12248; 28 March 1985). OCD is applicable for over-water sources where onshore receptors are below the lowest source height. Where onshore receptors are above the lowest source height, offshore plume transport and dispersion may be modeled on a case-by-case basis in consultation with the District.

4.14.3 *Shoreline Dispersion Model (SDM)*

SDM (PEI, 1988) is a hybrid multipoint Gaussian dispersion model that calculates source impact for those hours during the year when fumigation events are expected using a special fumigation algorithm and the MPTER regulatory model for the remaining hours.

SDM may be used on a case-by-case basis for the following applications:

- tall stationary point sources located at a shoreline of any large body of water;
- rural or urban areas;
- flat terrain;
- transport distances less than 50 km;
- 1-hour to 1-year averaging times.

4.15 Interaction with the District

The risk assessor must contact the District to determine if there are any specific requirements. Examples of such requirements may include, but are not limited to: specific receptor location guidance, specific usage of meteorological data, and specific report format (input and output). See Chapter 9 for more information on the format and content of modeling protocols and HRAs.

4.15.1 *Submittal of Modeling Protocol*

It is strongly recommended that a modeling protocol be submitted to the District for review and approval prior to extensive analysis with an air dispersion model. The modeling protocol is a plan of the steps to be taken during the air dispersion modeling process. Following is an example of the format that may be followed in the preparation of the modeling protocol. **Consult with the District to confirm format and content requirements or to determine the availability of District modeling guidelines before submitting the protocol.**

Outline for a Modeling Protocol

I. Introduction

Include the facility name, address, and a brief overview describing the facility's operations.

- Provide a description of the terrain and topography surrounding the facility and potential receptors.

- Indicate the format in which data will be provided. Ideally, the report and summary of data will be on paper and all data and model input and output files will be provided electronically (e.g., compact disk or CD).

- Identify the guidelines used to prepare the protocol (e.g., District Guidelines).

II. Emissions

For each pollutant and process whose emissions are required to be quantified in the HRA, list the annual average emissions (pounds/year and grams/second) and the maximum one-hour emissions (pounds/hour and grams/second)[2]. Maximum 1-hour emissions are used for acute noncancer health impacts while annual emissions are used for chronic exposures (i.e., chronic and 8-hour noncancer health impacts or cancer risk assessment).

- Identify the reference and method(s) used to determine emissions (e.g., source tests, emission factors, etc.). Clearly indicate any emission data that are not reflected in the previously submitted emission inventory report. In this event, a revised emission inventory report will need to be submitted to the District.

- Identify if this will be a multipathway assessment based on emitted substances.

III. Models / Modeling Assumptions

Specify the model and modeling assumptions

- Identify the model(s) to be used, including the version number.

- Identify the model options that will be used in the analysis.

---

[2] Except radionuclides, for which annual and hourly emissions are reported in Curies/year and millicuries/hour, respectively.

- Identify the modeling domain(s) and the spacing of receptor grid(s). Grid spacing should be sufficient in number and detail to capture the concentration at all of the receptors of interest.

- Indicate complex terrain options that may be used, if applicable.

- Identify the source type(s) that will be used to represent the facility's operations (e.g., point, area, or volume sources, flare options or other).

- Indicate the preliminary source characteristics (e.g., stack height, gas temperature, exit velocity, dimensions of volume source, etc.).

- Identify and support the use of urban or rural dispersion coefficients for those models that require dispersion coefficients. For other models, identify and support the parameters required to characterize the atmospheric dispersion due to land characteristics (e.g., surface roughness, Monin-Obukhov length).

IV. Meteorological Data

Specify the type, source, and year(s) of hourly meteorological data (e.g., hourly surface data, upper air mixing height information).

- State how the data are representative for the facility site.

- Describe QA/QC procedures.

- Identify any gaps in the data; if gaps exist, describe how the data gaps are filled.

V. Deposition

- Specify the method to calculate deposition (if applicable).

VI. Receptors

Specify the type and location of receptors. Include all relevant information describing how the individual and population-related receptors will be evaluated.

- Identify and describe the location(s) of known or anticipated potential sensitive receptors, the point of maximum impact (PMI), and the maximum exposed individual residential (MEIR) and worker (MEIW) receptors. Identify any special considerations or grids that will be used to model these receptors. This information should correspond with information provided in Section III (e.g., fine receptor spacing of 20 meters at the fence line and centered on the maximum impacts; coarse receptor spacing of 100 meters out to 2,000 meters; extra coarse spacing of 1,000 meters out to 20,000 meters).

- Identify if spatial averaging will be used. Include necessary background information on each receptor including how the domain and spacing will be determined for each receptor or exposure pathway.

- Describe how the cancer burden or population impact estimates are calculated. Clarify the same information for the presentation of noncancer population impacts (e.g., centroids of the census tracts in the area within the zone of impact).

- Specify that actual UTM coordinates and the block/street locations (i.e., north side of 3,000 block of Smith Street), where possible, will be provided for specified receptor locations.

- Identify and support the use of any exposure adjustments (e.g., time a location, diurnal).

- Include the list of anticipated exposure pathways that will be included and indicate which substance will be evaluated in the multipathway assessment. Identify if sensitive receptors are present and which receptors will be evaluated in the HRA.

VII. Maps

Identify how the information will be graphically presented.

- Indicate which cancer risk isopleths will be plotted for the cancer zone of impact (e.g., 10^{-7}, 10^{-6} see Section 4.6.1).

- Indicate the hazard quotients or hazard indices to be plotted for the noncancer acute, 8 hour, and chronic zones of impact (e.g., 0.5, 1.0, etc.).

4.16 Health Risk Assessment Report

This section describes the information related to the air dispersion modeling process that needs to be reported in the risk assessment. This section is also presented in Chapter 9, Summary of the Requirements for a Modeling Protocol and a Health Risk Assessment Report, in Section 9.2. The District may have specific requirements regarding format and content (see Section 4.15). Sample calculations should be provided at each step to indicate how reported emissions data were used. Reviewing agencies must receive input, output, and supporting files of various model analyses on computer-readable media (e.g., CD).

4.16.1 *Information on the Facility and its Surroundings*

Report the following information regarding the facility and its surroundings:

- Facility Name

- Location (UTM coordinates and street address)
- Land use type (see Section 2.4)
- Local topography
- Facility plot plan identifying:
  - source locations
  - property line
  - horizontal scale
  - building heights
  - emission sources

4.16.2 *Source and Emission Inventory Information*[3]

4.16.2.1 Release Parameters

Report the following information for each release location in table format:

- Release location identification number
- Release name
- Release type (e.g., point, volume, area, line, pit, etc.)
- Source identification number(s) used by the facility that emit out of this release location
- Release location using UTM coordinates
- Release parameters by release type (e.g., shown for point source):
  - Stack height (m), stack diameter (building dimensions for downwash), exhaust gas exit velocity (m/s), exhaust gas volumetric flow rate (ACFM), exhaust gas exit temperature (K), etc.

4.16.2.2 Source Description and Operating Schedule

The description and operating schedule for each source should be reported in table form including the following information:

- Source identification number used by the facility
- Source name
- Number of operating hours per day and per year (e.g., 0800-1700, 2700 hr/yr)
- Number of operating days per week (e.g., Mon-Sat)
- Number of operating days or weeks per year (e.g., 52 wk/yr excluding major holidays)
- Release point identification number(s) for where source emissions are released

[3] Health and Safety Code section 44346 authorizes facility operators to designate certain "Hot Spots" information as trade secret. Section 44361(a) requires districts to make health risk assessments available for public review upon request. Section 44346 specifies procedures to be followed upon receipt of a request for the release of trade secret information. See also the Inventory Guidelines Report regarding the designation of trade secret information in the Inventory Reports.

- Fraction of source emissions emitted at each release point by release point ID number

4.16.2.3 Emission Control Equipment and Efficiency

Report emission control equipment and efficiency by source and by substance

4.16.2.4 Emissions Data Grouped By Source

Report emission rates for each toxic substance, grouped by source (i.e., emitting device or process identified in Inventory Report), in table form including the following information:

- Source name
- Source identification number
- Substance name and CAS number (from Inventory Guidelines)
- Annual average emissions for each substance (lb/yr)
- Hourly maximum emissions for each substance (lb/hr)

4.16.2.5 Emissions Data Grouped by Substance

Report facility total emission rate by substance for all emitted substances listed in the Air Toxics “Hot Spots” Program including the following information:

- Substance name and CAS number (from Inventory Guidelines)
- Annual average emissions for each substance (lb/yr)
- Hourly maximum emissions for each substance (lb/hr)

4.16.2.6 Emission Estimation Methods

Report the methods used in obtaining the emissions data indicating whether emissions were measured or estimated. Clearly indicate any emission data that are not reflected in the previously submitted emission inventory report and submit a revised emission inventory report to the district. A reader should be able to reproduce the risk assessment without the need for clarification.

4.16.2.7 List of Substances

Include tables listing all "Hot Spots" Program substances which are emitted, plus any other substances required by the District. Indicate substances to be evaluated for cancer risks and noncancer health impacts.

4.16.3 *Exposed Population and Receptor Location*

Report the following information regarding exposed population and receptor locations. See Chapter 9 and specific sections within this chapter for more detailed information.

- Description of zone of impact including map showing the location of the facility, boundaries of zone of impact, census tracts, emission sources, sites of maximum exposure, and the location of all appropriate receptors. This should be a true map (one that shows roads, structures, etc.), drawn to scale, and not just a schematic drawing. USGS 7.5 minute maps or GIS based maps are usually the most appropriate choices. (If significant development has occurred since the user's survey, this should be indicated.)
- Separate maps for the cancer risk zone of impact and the hazard index (noncancer) zone of impact(s). The cancer zone of impact should include isopleths down to at least the 1/1,000,000 risk level. Because some districts use a level below 1/1,000,000 to define the zone of impact, the District should be consulted. Three separate maps (to represent both chronic, 8-hour, and acute HI) should be created to define the zone of impact for the hazard index from both inhalation and noninhalation pathways greater than or equal to 0.5. The point of maximum impact (PMI), maximum exposed individual at a residential receptor (MEIR), the maximum exposed individual worker (MEIW), and any other locations of interest for both cancer and noncancer risks should be located on the maps.
- Tables identifying population units and sensitive receptors (UTM coordinates, receptor IDs, and street addresses of specified receptors).
- Heights or elevations of the receptor points.
- For each receptor type (e.g., PMI, MEIR, MEIW, and any other location(s) of interest) that will utilize spatial averaging, the domain size and grid resolution must be clearly identified. If another domain or grid resolution other than 20 meters by 20 meters with 5-meter grid spacing will be used for a receptor, then care should be taken to determine the proper domain size and grid resolution that should be used. For a worker, the HRA shall support all assumptions used, including, but not limited to, documentation for all workers showing the area where each worker routinely performs their duties. The final domain size should not be greater than the smallest area of worker movement. Other considerations for determining domain size and grid spacing resolution may include an evaluation of the concentration gradients across the worker area. The grid spacing used within the domain should be sufficient in number and detail to obtain a representative concentration across the area of interest. When spatial averaging over the deposition area of a pasture, garden, or water body, care should be taken to determine the proper domain size to make sure it includes all reasonable areas of potential deposition. The size and shape of the pasture, garden, or water body of interest should be identified and used for the modeling domain. The grid spacing or resolution used within the domain should be sufficient in detail to obtain a representative deposition concentration across the area of interest. One way to determine the grid resolution is to include an evaluation of the concentration gradients across the deposition area. The HRA shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the pasture or water body, maps,

representative coordinates, grid resolution, concentration gradients, etc.). The use or spatial averaging is subject to approval by the reviewing authority. This includes the size of the domain and grid resolution that is used for spatial averaging of a worksite or multipathway deposition area.

4.16.4 *Meteorological Data*

If meteorological data were not obtained directly from the District, then the report must clearly indicate the data source and time period used. Meteorological data not obtained from the District must be submitted in electronic form along with justification for their use including information regarding representativeness and quality assurance.

The risk assessment should indicate if the District required the use of a specified meteorological data set. All memos indicating the District's approval of meteorological data should be attached in an appendix.

4.16.5 *Model Selection and Modeling Rationale*

The report should include an explanation of the model chosen to perform the analysis and any other decisions made during the modeling process. The report should clearly indicate the name of the models that were used, the level of detail (screening or refined analysis) and the rationale behind the selection.

Also report the following information for each air dispersion model used:

- version number
- selected options and parameters in table form
- Identify the modeling domain(s) and the spacing of receptor grid(s). Grid spacing should be sufficient in number and detail to capture the concentration at all receptors of interest.

4.16.6 *Air Dispersion Modeling Results*

- Maximum hourly and annual average concentrations of chemicals at appropriate receptors such as the residential and worker MEI receptors
- Annual average and maximum one-hour (and 30-day average for lead only) concentrations of chemicals at appropriate receptors listed and referenced to computer printouts of model outputs
- Model printouts (numbered), annual concentrations, maximum hourly concentrations
- Disk with input/output files for air dispersion program (e.g., the AERMOD input file containing the regulatory options and emission parameters, receptor locations, meteorology, etc.)
- Include tables that summarize the annual average concentrations that are calculated for all the substances at each site. The use of tables that present the relative contribution of each emission point to the receptor concentration is recommended. (These tables should have clear reference to the computer

model which generated the data. It should be made clear to any reader how data from the computer output were transferred to these tables.) [As an alternative, the above two tables could contain just the values for sites of maximum impact (i.e., PMI, MEIR and MEIW), and sensitive receptors, if required. All the values would be found in the Appendices.]

4.17 References

Auer Jr., A.H. (1978). Correlation of land use and cover with meteorological anomalies. Journal of Applied Meteorology, 17(5):636-643.

ARB (1994). ARB memorandum dated 4/11/94 from A. Ranzieri to J. Brooks on the subject, "One-hour to Thirty-day Average Screening Factor."

ARB (1989). "Screening Deposition Velocities," Internal memorandum from A. Ranzieri to G. Shiroma dated 8/17/89.

Bidleman, T.F. (1988). Atmospheric processes. Environmental Science & Technology, 22(4):361-367.

Bjorklund, J.R. and J.F. Bowers (1982). User's Instructions for the SHORTZ and LONGZ Computer Programs, Volumes I and II. EPA-903/9-82-004A and B. U.S. Environmental Protection Agency. Philadelphia, PA.

Burt, E.W. (1977). Valley Model User's Guide. EPA-450/2-77-018. U.S. Environmental Protection Agency, Research Triangle Park, NC.

CAPCOA (1987). "Deposition Rate Calculations for Air Toxics Source Assessments," in Air Toxics Assessment Manual, Appendix C.7.

Catalano, J.A., D.B. Turner and H. Novak (1987). User's Guide for RAM - Second Edition. U.S. Environmental Protection Agency. Research Triangle Park, NC. (Distributed as part of UNAMAP Version 6 Documentation)

Chico, T. and J. A. Catalano (1986). Addendum to the User's Guide for MPTER. U. S. Environmental Protection Agency. Research Triangle Park, NC.

Croes, B. (1988). "Deposition Rate Calculations for Air Toxic Risk Assessments in California," Proceedings of the 81st Annual Meeting of the Air Pollution Control Association, Dallas, TX, June 20-24, 1988.

DiCristofaro, D. C. and S. R. Hanna (1989). OCD: The Offshore and Coastal Dispersion Model, Version 4. Volume I: User's Guide, and Volume II: Appendices. Sigma Research Corporation, Westford, MA. (NTIS Nos. PB 93-144384 and PB 93-144392)

Irwin, J.S. (1978). Proposed Criteria for Selection of Urban Versus Rural Dispersion Coefficients. (Draft Staff Report). Meteorology and Assessment Division. U.S. Environmental Protection Agency, Research Triangle Park, NC. (Docket No. A-80-46, II-B-8).

OEHHA (2012). Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis. Available online at http://www.oehha.ca.gov

PEI Associates (1988). User's Guide to SDM - A Shoreline Dispersion Model. U.S. EPA Publication No. EPA-450/4-88-017. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Perry, S.G., D.J. Burns, A.J. Cimorelli (1990). User's Guide to CTDMPLUS: Volume 2. The Screening Mode (CTSCREEN). EPA-600/8-90-087. Office of Research and Development, U.S. Environmental Protection Agency, Research Triangle Park, NC.

Pierce, T.E., D.B. Turner, J.A. Catalano, and F.V. Hale (1982). PTPLU - A Single Source Gaussian Dispersion Algorithm User's Guide. EPA-600-/8-82-014. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Pierce, T.E. (1986). Addendum to PTPLU - A Single Source Gaussian Dispersion Algorithm. EPA/600/8-86-042. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Schulman, L.L., and J.S. Scire (l980). Buoyant Line and Point Source (BLP) Dispersion Model User's Guide. Document P-7304B. Environmental Research and Technology, Inc., Concord, MA. (NTIS No. PB 8l-l64642)

Schulman, L.L., Strimaitis, D. G., and Scire, J. S. (2000). Development and evaluation of the PRIME plume rise and building downwash model. Journal of the Air and Waste Management Association, 50:378-390,.

Tikvart, J. (1993). "Proposal for Calculating Plume Rise for Stacks with Horizontal Releases or Rain Caps for Cookson Pigment, Newark, New Jersey," Internal memorandum from J. Tikvart to K. Eng dated 7/9/93.

Turner, D. and J.H. Novak (1978). User's Guide for RAM. Vol. 1. Algorithm Description and Use, Vol. II. Data Preparation and Listings. EPA-600/8-78-016a and b. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1984a). Calms Processor (CALMPRO) User's Guide. EPA-901/9-84-001. U.S. Environmental Protection Agency, Region I, Boston, MA.

U.S. EPA (1984b). Interim Procedures for Evaluating Air Quality Models (Revised). EPA-450/4-84-023. U.S. Environmental Protection Agency, Research Triangle Park, NC. (NTIS No. PB 85-106060)

U.S. EPA (1985a). Interim Procedures for Evaluating Air Quality Models: Experience with Implementation. U.S. EPA Publication No. EPA-450/4-85-006. U.S. Environmental Protection Agency, Research Triangle Park, NC. (NTIS No. PB 85-242477)

U.S. EPA (1985b). Guideline for Determination of Good Engineering Practice Stack Height (Technical Support Document for the Stack Height Regulations) - Revised EPA-450/4-80-023R, U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1992). Protocol for Determining the Best Performing Model. U.S. EPA Publication No. EPA-454/R-92-025. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1993). User's Guide to the Building Profile Input Program (BPIP). Revised February, 1995. EPA-454/R-93-038. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1995a). Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised. EPA-450/R-92-019. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1995b). User's Guide for the Industrial Source Complex (ISC) Dispersion Models. Volume I: User Instructions. EPA-454/B-95-003a. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1995c). User's Guide for the Industrial Source Complex (ISC) Dispersion Models. Volume II: User Instructions. EPA-454/B-95-003a. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (1995d). SCREEN3 Model User's Guide. EPA-454/B-95-004. U.S. Environmental Protection Agency. Research Triangle Park, NC.

U.S. EPA (1995e). On-Site Meteorological Program Guidance For Regulatory Modeling Applications. EPA-450/4-87-013. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2004a). User's Guide for the AMS/EPA Regulatory Model - AERMOD. EPA-454/B-03-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2004b). AERMOD: Description of Model Formulation. EPA-454/R-03-004. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2005). Federal Register / Volume 70, Number 216 / November 9, 2005 / Rules and Regulations, 40 CFR Part 51 Appendix W, Revision to the Guideline on Air Quality Models, U.S. Environmental Protection Agency.

U.S. EPA (2009). AERMOD Implementation Guide. Last Revised: March 19, 2009. AERMOD Implementation Workgroup, U. S. Environmental Protection Agency. Online at: http://www.epa.gov/ttn/scram/7thconf/aermod/aermod_implmtn_guide_19March2009.pdf

U.S. EPA (2011). AERSCREEN User's Guide. EPA-454/B-11-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

5 - Exposure Assessment
Estimation of Concentration and Dose

5.1 Introduction

This chapter provides a summary of how toxicant ground level air concentrations estimated from air dispersion modeling or monitoring results are used to determine dose at receptors of interest. This chapter includes all the algorithms and data (e.g., point estimates, distributions, and transfer factors) that are needed to determine the substance-specific concentration in exposure media and the dose at a receptor of interest. The determination of exposure concentration and dose precedes the calculations of potential health impacts. See Chapter 8 and Appendix I for information on calculating potential health impacts.

At a minimum, three receptors are evaluated in Hot Spots health risk assessments (HRA) (see Section 4.7); these are:

- the Point of Maximum Impact (PMI),
- the Maximally Exposed Individual Resident (MEIR), and
- the Maximally Exposed Individual Worker (MEIW).

The PMI is defined as the receptor point(s) with the highest acute, 8-hour, chronic, or cancer health impact outside the facility boundary. The facility boundary is defined as the property line. Often the fence is on the property line. The MEIR is typically defined as the existing off-site residence(s) (i.e., house, apartment or other dwelling) with the highest acute, chronic, or cancer health impact. Calculating an 8-hour hazard index is not required for the MEIR, but can be performed at the discretion of the District. The MEIW is typically defined as the existing offsite workplace with the highest acute, 8-hour, chronic, or cancer health impact.

In addition, it may be necessary to determine risks at sensitive receptors (e.g., schools, day care centers, elder care centers, and hospitals). The District or reviewing authority should be consulted in order to determine the appropriate sensitive receptors for evaluation. Some situations may require that on-site receptor (worker or residential) locations be evaluated. Some examples where the health impacts of on-site receptors may be appropriate could be military base housing, prisons, universities, or locations where the public may have regular access for the appropriate exposure period (e.g., a lunch time café or museum for acute exposures). The risk assessor should contact the Air Pollution Control or Air Quality Management District (the District) for guidance about any on-site exposure situations at the emitting facility. These on-site locations should be included in the health risk assessment (HRA). If the facility emits multiple substances from two or more stacks, the acute, 8-hour, chronic, and cancer health impacts at the PMI may be located at different physical locations. The MEIR or MEIW cancer, acute, 8-hour, and chronic receptors may also be at different locations.

The process for determining dose at the receptor location, and ultimately potential health impacts, will likely include air dispersion modeling, and, with less frequency, air monitoring data. Air dispersion modeling combines the facility emissions and release parameters and uses default or site-specific meteorological conditions to estimate downwind, ground-level concentrations at various (user-defined) receptor locations. Air dispersion modeling is described in Chapter 4 and is presented in detail in the *Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis (OEHHA, 2012a*).

In summary, the process of using air dispersion modeling results as the basis of an HRA follows these four steps:

- Air dispersion modeling is used to estimate annual average and maximum one-hour ground level concentrations (GLC). The air dispersion modeling results are expressed as an air concentration or in terms of (Chi over Q) for each receptor point. (Chi over Q) is the modeled downwind air concentration (Chi) based on an emission rate of one gram per second (Q). (Chi over Q) is expressed in units of micrograms per cubic meter per gram per second, or ($\mu g/m^3$)/(g/s). (Chi over Q) is sometimes written as (χ/Q) and is sometimes referred to as the dilution factor.

- When multiple substances are evaluated, the χ/Q is normally utilized since it is based on an emission rate of one gram per second. The χ/Q at the receptor point of interest is multiplied by the substance-specific emission rate (in g/s) to yield the substance-specific ground-level concentration (GLC) in units of $\mu g/m^3$. The following equations illustrate this point.

$$\mathbf{GLC} = \left(\frac{\chi}{\mathbf{Q}}\right) \mathbf{x} \left(\mathbf{Q}_{\mathbf{substance}}\right)$$

$$\frac{\chi}{\mathbf{Q}} = \mathbf{(Chi\ \ over\ \ Q)\ in} \left(\frac{\frac{\mu \mathbf{g}}{\mathbf{m}^3}}{\frac{\mathbf{g}}{\mathbf{s}}} \right), \mathbf{\ from\ model\ results\ with\ unit\ emission\ rate}$$

$$\mathbf{Q}_{\mathbf{substance}} = \mathbf{\ substance\ specific\ emission\ rate} \left(\frac{\mathbf{g}}{\mathbf{s}}\right)$$

- The applicable exposure pathways (e.g., inhalation, soil contact, fish consumption) are identified for the emitted substances, and the receptor locations are identified. This determines which exposure algorithms in this chapter are ultimately used to estimate dose. After the exposure pathways are identified, the fate and transport algorithms described in this chapter are used to estimate concentrations in the applicable exposure media (e.g., soil or water) and the exposure algorithms are used to determine the substance-specific dose.

- The dose is used with cancer and noncancer health values to calculate the potential health impacts for the receptor (Chapter 8). An example calculation

using the high-end point-estimates for the inhalation (breathing) exposure pathway can be found in Appendix I. Appendix I and Chapters 5 (this Section) and 8 also contain information on how the annual average and maximum one-hour ground level concentrations are used for chronic, 8-hour, and acute health risk calculations.

The algorithms in this chapter are also used to calculate media concentrations and dose in the rare instance, for the Hot Spots program, when monitoring equipment was used rather than air dispersion modeling to obtain a receptor's substance-specific GLC. One situation that is specific to monitored data is the treatment of results below the sampling method level of detection (LOD). In short, it is standard risk assessment practice when monitoring results are reported both above and below the LOD to use one-half of the LOD for those sample concentrations reported below the LOD. If all testing or monitoring results fall below the LOD, then assessors should contact the District for appropriate procedures. For more information about reporting emissions under the Hot Spots Program, see the ARB's *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5),* and the *Emission Inventory Criteria and Guidelines Report* (EICG Report), which is incorporated by reference therein (ARB, 2007*)*.

The recommended model for calculating and presenting HRA results for the Hot Spots Program is the HARP software, available from the Air Resources Board (ARB). More information on HARP and directions for downloading the software can be found on the ARB's web site at http://www.arb.ca.gov/toxics/harp/downloads.htm.

5.2 Criteria for Exposure Pathway Evaluation

In order to determine total dose to the receptor the applicable pathways of exposure need to be identified. The inhalation pathway must be evaluated for all Hot Spots substances emitted by the facility. A small subset of Hot Spots substances is subject to deposition onto soil, plants, and water bodies. These substances need to be evaluated by the appropriate noninhalation pathways, as well as by the inhalation pathway, and the results must be presented in all HRAs. These substances include semi-volatile organic chemicals and heavy metals. Such substances are referred to as multipathway substances. Two steps are necessary to determine if a substance should be evaluated for multipathway impacts:

1. Determine whether the substance or its group (e.g., dioxins, PAHs) is listed in Table 5.1.
2. Determine if the substance has an oral reference exposure level (REL) listed in Table 6.4, or if it has an oral cancer slope factor listed in Table 7.1. Two other references for checking the presence of oral health factors are OEHHA's website (OEHHA, 2012b) and the *Consolidated Table of OEHHA/ARB Approved Risk Assessment Health Values* on the Air Resources Board website (ARB, 2012). Oral or noninhalation exposure pathways include the ingestion of soil, angler-caught fish, drinking water from surface water sources, mother's milk,

homegrown produce, beef, pork, chicken, eggs and cow's milk. The dermal pathway is also evaluated via contact with contaminated soil.

For all multipathway substances, the minimum exposure pathways that must be evaluated at every residential site (in addition to inhalation) are soil ingestion and dermal exposure. If dioxins, furans, PCBs, PAHs or lead are emitted, then the breast-milk consumption pathway also becomes mandatory. The other exposure pathways (e.g., the ingestion of homegrown produce or angler-caught fish) are evaluated on a site-by-site basis. If the resident can be exposed through an impacted exposure pathway, then it must be included in the HRA. However, if there are no vegetable gardens or fruit trees within the zone of impact for a facility, for example, then the produce pathways need not be evaluated. Note that on-site residential receptors are potentially subject to inhalation and noninhalation exposure pathways. Table 8.2 identifies the residential and worker receptor exposure pathways that are mandatory and those that are dependent on the site-specific decisions. While residents can be exposed though several exposure pathways, worker receptors are only evaluated for inhalation, soil ingestion, and dermal exposure using point estimates.

Table 5.1 shows the multipathway substances that, based on available scientific data, can be considered for each noninhalation exposure pathway. The exposure pathways that are evaluated for a substance depend on two factors: 1) whether the substance is considered a multipathway substance for the Hot Spots Program (Table 5.1), and 2) what the site-specific conditions are. A multipathway substance may be excluded from a particular exposure pathway because its physical-chemical properties can preclude significant exposure via the pathway. For example, some water-soluble substances do not appreciably bioaccumulate in fish; therefore, the fish pathway is not appropriate. In addition, if a particular exposure pathway is not impacted by the facility or is not present at the receptor site, then the pathway is not evaluated. For example, if a fishable water body is not impacted by the facility, or the water source is impacted but no receptor uses it for fishing, then the angler-caught fish pathway is not evaluated.

Table 5.1 Specific Pathways to be Analyzed for Each Multipathway Substance

Substance	Soil Ingestion	Dermal	Meat, Milk & Egg Ingestion	Fish Ingestion	Exposed Vegetable Ingestion	Leafy Vegetable Ingestion	Protected Vegetable Ingestion	Root Vegetable Ingestion	Water Ingestion	Breast Milk Ingestion
Inorganic chemicals										
Arsenic & compounds	X	X	X	X	X	X	X	X	X	
Beryllium & compounds	X	X	X	X	X	X	X	X	X	
Cadmium & compounds	X	X	X	X	X	X	X	X	X	
Chromium VI & compounds	X	X	X[a]	X	X	X	X	X	X	
Fluorides (soluble compounds)	X	X	X		X	X	X	X	X	
Lead & compounds	X	X	X	X	X	X	X	X	X	X
Mercury & compounds	X	X	X	X	X	X	X	X	X	
Nickel & compounds	X	X	X	X	X	X	X	X	X	
Selenium & compounds	X	X	X	X	X	X	X	X	X	
Organic chemicals										
Creosotes	X	X	X	X	X	X			X	X
Diethylhexylphthalate	X	X	X	X	X	X			X	
Hexachlorobenzene	X	X	X	X	X	X			X	
Hexachlorocyclohexanes	X	X	X	X	X	X			X	
4,4'-Methylene dianiline	X	X			X	X			X	
Pentachlorophenol[b]										
PCBs	X	X	X	X	X	X			X	X
Polychlorinated dibenzo-p-dioxins and dibenzofurans	X	X	X	X	X	X			X	X
PAHs	X	X	X	X	X	X			X	X

[a] Cow's milk only; no multipathway analysis for meat and egg ingestion

[b] To be evaluated by pathway in future amendments to the Hot Spots Program

5.3 Estimation of Concentrations in Air, Soil, and Water

Once emissions exit the source, the substances emitted will be dispersed in the air. The substances in the exhaust gas with high vapor pressures will remain largely in the vapor phase, and substances with lower vapor pressures will tend to adsorb to fly ash or other particulate matter. The emission plume may contain both vapor phase substances and particulates. A semivolatile organic toxicant can partition into both vapor and particulate phases. Particulates will deposit on vegetation, on soil, and in water at a rate that is dependent on the particle size. Use the 0.02 m/s deposition rate for emission sources that have verifiable particulate matter control devices or for emission sources that may be uncontrolled but only emit particulate matter that is less than 2.5 microns (e.g., internal combustion engines). The following algorithms are used to estimate concentrations in environmental media including air, soil, water, vegetation, and animal products.

5.3.1 *Air*

The ground level concentration (GLC, or C_{air} as shown in EQ 5.3.1) of a substance in air is a function of the facility emission rate and the dilution factor (χ/Q) at the points under evaluation.

A. Equation 5.3.1: $$C_{air} = Q_{substance} \times \chi/Q$$

1. C_{air} = Ground level concentration ($\mu g/m^3$)
2. $Q_{substance}$ = Substance emission rate (g/sec)
3. χ/Q = Dilution factor provided by dispersion modeling ($\mu g/m^3$/g/sec)

a. Recommended values for EQ 5.3.1:

1. $Q_{substance}$ = Facility-specific, substance emission rate
2. χ/Q = For point of interest, site specific, from dispersion modeling

b. Assumptions for EQ 5.3.1:

1. No plume depletion
2. Emission rate is constant, i.e., assumes steady state

5.3.2 *Soil*

The average concentration of the substance in soil (C_s) is a function of the deposition, accumulation period, chemical specific soil half-life, mixing depth, and soil bulk density. For simplicity and health protection, the Tier 1 default assumes 70-year soil deposition for the accumulation period at end of 70-year facility lifetime. The risk assessor may also choose a supplemental Tier 2 approach, subject to District approval or reviewing authority approval, in which the assessor applies a soil accumulation period based on the facility's start date of operation (e.g., historical date when emissions began), or the current exposure conditions, and the expected duration of operation.

A. Equation 5.3.2 A: $$C_s = Dep \times X / (K_s \times SD \times BD \times T_t)$$

1. C_s = Average soil concentration over the evaluation period ($\mu g/kg$)
2. Dep = Deposition on the affected soil area per day ($\mu g/m^2$-d)
3. X = Integral function for soil accumulation (d), see **EQ 5.3.2 C** below
4. K_s = Soil elimination constant (d^{-1})
5. SD = Soil mixing depth (m)
6. BD = Soil bulk density (kg/m^3)
7. T_t = Soil exposure duration or soil accumulation period (d)

a: Recommended default values for EQ 5.3.2 A:

1. Dep = Calculated in EQ 5.3.2 B
2. X = Calculated in EQ 5.3.2 C
3. K_s = Calculated in EQ 5.3.2 D
4. SD = 0.01 (m) for playground setting (soil ingestion and dermal pathways) and 0.15 (m) for agricultural setting (produce and meat pathways)
5. BD = 1,333 (kg/m^3)
6. T_t = 25,550 (d) = 70 years

b: Assumptions for EQ 5.3.2 A:

1. Substances are uniformly mixed in soil.
2. Substances are not leached or washed away, except where evidence exists to the contrary.
3. It is assumed that toxicants accumulate in the soil for 70 years from deposition over the 70 year lifespan of the facility. Use 70-year soil accumulation (T_t) for Tier 1 estimation of 9-, 30- and 70-year residential exposure, and 25-year off-site worker exposure.
4. For a receptor ingesting mother's milk, the mother is exposed from birth to 25 years of age; the infant is then born and receives mother's milk for one year. Default assumes 70-year soil accumulation for mother's milk pathway. See Table 5.1 for information on which substances or groups of substances must be evaluated by the mother's milk pathway.

B. Equation 5.3.2 B: $$\mathbf{Dep = C_{air} \times Dep\text{-}rate \times 86{,}400}$$

1. C_{air} = Ground level concentration ($\mu g/m^3$)
2. Dep-rate = Vertical rate of deposition (m/sec)
3. 86,400 = Seconds per day conversion factor (sec/d)

a: Recommended default values for EQ 5.3.2 B:

1. C_{air} = Calculated above in EQ 5.3.1 A
2. Dep-rate = Use 0.02 meters/second for controlled sources, or 0.05 meters/second for uncontrolled sources.

b: Assumptions for EQ 5.3.2 B:

1. Deposition rate remains constant. A deposition rate must be used when determining potential noninhalation health impacts. In the absence of facility specific information on the size of the emitted particles, the default values for deposition rate should be used. Currently, the default value of 0.02 meters per second is used for emission sources that have verifiable particulate matter control devices or for emission sources that may be uncontrolled but only emit particulate matter that is less than 2.5 microns

(e.g., internal combustion engines). The 0.05 meters per second default value is used for risk assessment if the emissions are uncontrolled. If other deposition rate factors are used, sufficient support documentation must be included with the HRA.

C. Equation 5.3.2 C: $$X = [\{e^{-Ks * Tf} - e^{-Ks * To}\} / K_s] + T_t$$

1. e = 2.718
2. K_s = Soil elimination constant
3. T_f = End of soil accumulation evaluation period (d)
4. T_o = Beginning of soil accumulation evaluation period (d)
5. T_t = Total days of soil exposure (soil accumulation period) T_f-T_o (d)

a: Recommended default values for EQ 5.3.2 C:

1: K_s = Calculated in EQ 5.3.2 D
2: T_f = 25,550 (d) = 70 years. Total soil exposure time at end of facility operation
3: T_o = 0 (d) The initial time (start period) of soil exposure to all receptors that are impacted by the soil pathway.

Note: Under a Tier 2 scenario, the risk assessor may also adjust T_f and T_t, subject to District approval, to replicate current soil accumulation and expected accumulation at the end of facility operation.

D. Equation 5.3.2 D: $$K_s = 0.693 / t_{1/2}$$

1. 0.693 = Natural log of 2
2. $t_{1/2}$ = Chemical specific soil half-life (d)

a: Recommended default values for EQ 5.3.2 D:

1. $t_{1/2}$ = Chemical-specific. See Table 5.2

5.3.3 *Water*

The water pathway is evaluated if a standing water body (e.g., pond or lake) is impacted by facility emissions and is used as a source for drinking water by food-producing animals or humans, or is a source of angler-caught fish. The average concentration of the substance in water (C_w) is a function of direct deposition and material carried in by surface run-off. However, only the contribution from direct deposition will be considered at this time.

A. Equation 5.3.3 A: $$C_w = C_{depw}$$

1. C_w = Average concentration in water (μg/kg)
2. C_{depw} = Contribution due to direct deposition (μg/kg)

B. Equation 5.3.3 B: $C_{depw} = Dep \times SA \times 365 / (WV \times VC)$

1. Dep = Deposition on water body per day ($\mu g/m^2/d$)
2. SA = Water surface area (m^2)
3. 365 = Days per year (d/yr)
4. WV = Water volume (kg)
5. VC = Number of volume changes per year

a: Recommended default values for EQ 5.3.3 B:

1. Dep = Calculated above in EQ 5.3.2 B
2. SA = Site specific water surface area (m^2)
3. WV = Site specific water volume in (kg) (1L = 1 kg)
4. VC = Site specific number of volume changes per year (SA, WV, and VC values can be obtained from the appropriate Department of Water Resources (DWR) Regional office)

b: Assumptions for EQ 5.3.3 B:

1. With the exception of dilution via number of volume changes per year, all material deposited into the water remains suspended or dissolved in the water column and is available for bioaccumulation in fish.

5.3.4 *Estimation of Concentrations in Vegetation, Animal Products, and Mother's Milk*

Estimates of the concentration of the substance in vegetation, animal products and mother's milk require the use of the results of the air, water, and soil environmental fate evaluation. Plants, animals and nursing mothers will be exposed to the substances at the concentrations previously calculated in Section 5.31 to 5.33 above.

5.3.4.1 Vegetation

The average concentration of a substance in and on vegetation (C_v) is a function of direct deposition of the substance onto the vegetation and of root translocation or uptake from soil contaminated by the substance. We currently recommend root translocation only for the inorganic compounds.

A. Equation 5.3.4.1 A: $C_v = C_{depv} + C_{trans}$

1. C_v = Average concentration in and on specific types of vegetation ($\mu g/kg$)
2. C_{depv} = Concentration due to direct deposition ($\mu g/kg$)
3. C_{trans} = Concentration in vegetation due to root translocation or uptake ($\mu g/kg$) – see EQ 5.3.4.1 C below

B. Equation 5.3.4.1 B: $C_{depv} = [Dep \times IF / (k \times Y)] \times (1 - e^{-kT})$

1. Dep = Deposition on affected vegetation per day ($\mu g/m^2/d$)
2. IF = Interception fraction
3. k = Weathering constant (d^{-1})
4. Y = Yield (kg/m^2)
5. e = Base of natural logarithm (2.718)
6. T = Growth period (d)

a: Recommended default values for EQ 5.3.4.1 B:

1. Dep = Calculated above in EQ 5.3.2 B
2. IF = Crop specific:
    - a: Root crops = 0.0
    - b: Leafy crops = 0.2
    - c: Protected crops = 0.0
    - d: Exposed crops = 0.1
    - e: Pasture = 0.7
3. k = 0.1 (d^{-1})
4. Y = 2 (kg/m^2) for root, leafy, protected, exposed and pasture [CA Department of Food and Agriculture dot maps]
5. T = 45 (d) for leafy crops
   T = 90 (d) for exposed crops

b: Crop-type definitions for EQ 5.3.4.1 B:

1. **Leafy** crop category consists of broad-leafed vegetables in which the leaf is the edible part. Examples include spinach, lettuce, cabbage, and kale.
2. **Root** crop category includes vegetables in which the edible portion is underground. Examples are potato, radish, and carrot.
3. **Exposed** produce category consists of crops with a small surface area subject to air deposition. Examples include strawberries, tomato, cucumber, zucchini, green bean and bell pepper.
4. **Protected** produce category consists of crops in which the edible part is not exposed to air deposition (e.g., the exposed skin of the crop is removed and not eaten). Examples are corn, pea, pumpkin and oranges.

Tables H-9 through H-15 in Appendix H provide more examples of various leafy, root, exposed and protected crop types.

c: Assumptions for EQ 5.3.4.1 B:

1. No deposition on root or protected crops
2. No uptake and translocation of deposited chemicals onto crops

C. Equation 5.3.4.1 C: (for inorganic compounds)

$$C_{trans} = C_s \times UF_2$$

1. C_s = Average soil concentration (μg/kg)
2. UF_2 = Uptake factor based on soil concentration

a: Recommended default values for EQ 5.3.4.1 C:

1. C_s = Calculated above in EQ 5.3.2 A
2. UF_2 = See Table 5.2

D. Equation 5.3.4.1 D: (for organic compounds)

$$UF_2 = [(0.03 \times K_{ow}^{0.77}) + 0.82] / [(K_{oc})(F_{oc})]$$

1. 0.03 = Empirical constant
2. K_{ow} = Octanol:water partition factor
3. 0.77 = Empirical constant
4. 0.82 = Empirical constant
5. K_{oc} = Organic carbon partition coefficient
6. F_{oc} = Fraction organic carbon in soil

a: Recommended default values for EQ 5.3.4.1 D:

1. K_{ow} = Chemical specific, see Table 5.2
2. K_{oc} = Chemical specific, see Table 5.2
3. F_{oc} = 0.1

b: Assumptions for EQ 5.3.4.1 D:

1. OEHHA currently has no recommended root uptake factors for organic compounds listed in Table 5.2. Evidence suggests this route is insignificant compared to airborne deposition. Nevertheless, if it becomes necessary in specific cases to assess root uptake for an organic compound, Equation 5.3.4.1 D would be the algorithm OEHHA recommends using to assess root uptake.

5.3.4.2 Animal Products

The average concentration of the substance in animal products (C_{fa}) depends on which routes of exposure exist for the animals. Animal exposure routes include inhalation, soil ingestion, ingestion of contaminated feed and pasture, and ingestion of contaminated water.

A. Equation 5.3.4.2:

$$C_{fa} = (\text{Inhalation} + \text{Water ingestion} + \text{Feed ingestion} + \text{Pasture/Grazing ingestion} + \text{Soil ingestion}) * Tco$$

1. C_{fa} = Average concentration in farm animals and their products (μg/kg)
2. Inhalation, water ingestion, etc. = Dose through inhalation, water ingestion, etc. (μg/d)
3. Tco = Chemical-specific transfer coefficient of contaminant from diet to animal product (d/kg)

a: Recommended default values for EQ 5.3.4.2:

1. Tco = See Tables 5.3a and 5.3b

b: Assumptions for EQ 5.3.4.2:

1. The Tco for a given chemical is the same for all exposure routes

5.3.4.2.1 Inhalation

A. Equation 5.3.4.2.1: $\text{Inhalation} = BR_a \times C_{air}$

1. Inhalation = Dose through inhalation (μg/d)
2. BR_a = Breathing rate for animal (m^3/d)
3. C_{air} = Ground-level concentration (μg/m^3)

a: Recommended default values for EQ 5.3.4.2.1:

1. BR_a = See Table 5.4
2. C_{air} = Calculated above in EQ 5.3.1 A

b: Assumptions for EQ 5.3.4.2.1:

1. All material inhaled is 100% absorbed

5.3.4.2.2 Water Ingestion

The water ingestion pathway is applied if there are surface water sources of drinking water, such as springs, ponds or lakes, which are exposed to airborne deposition of facility emissions. Due to the site-specific nature for this exposure pathway, OEHHA recommends that the risk assessor conduct a survey at the site to estimate the fraction of contaminated drinking water ingested by the animals, if such sources exist.

A. Equation 5.3.4.2.2: **Water ingestion = $WI_a \times FSW \times C_w$**

1. Water ingestion = Dose through water ingestion (μg/d)
2. WI_a = Water ingestion for animal (kg/d)
3. FSW = Fraction of water ingested from a contaminated body of water (site-specific)
4. C_w = Average concentration in water (μg/kg)
   For water 1 kg = 1 L

a: Recommended default values for EQ 5.3.4.2.2:

1. WI_a = See Table 5.4
2. FSW = Site specific fraction, need to survey water ingestion practices in affected area
3. C_w = Calculated above in EQ 5.3.3 A

5.3.4.2.3 Feed Ingestion

The fraction of feed intake by cattle, pigs and poultry that is contaminated by facility emissions can vary considerably depending on the manner in which the animals are raised. Due to the site-specific nature for this exposure pathway, OEHHA recommends that the risk assessor conduct a survey at the site to estimate the fraction of contaminated feed eaten by the animals. For a Tier 1 assessment, default values are provided by OEHHA (see Table 5.4 and Table 5.4 footnotes) for estimation of exposure to the animals.

Agricultural mixing depth should be used for calculating soil concentration for feed and pasture contamination.

5.3.4.2.3.1 Feed Ingestion

A. Equation 5.3.4.2.3.1: **Feed ingestion = $(1.0 - FG) \times FI \times L \times C_v$**

1. Feed ingestion = Dose through the ingestion of feed (μg/d) that is harvested after it is impacted by source emissions
2. FG = Fraction of diet provided by grazing (site-specific)
3. FI = Feed ingestion rate (kg/d)
4. L = Fraction of locally grown (source impacted) feed that is not pasture (site-specific)
5. C_v = Concentration in feed (μg/kg)

a: Recommended default values EQ 5.3.4.2.3.1:

1. FG = Default values in Table 5.4 footnote b, although a site-specific survey for the fraction of diet provided by grazing is recommended
2. FI = See Table 5.4
3. L = Default values in Table 5.4 footnote b, although a site-specific survey for fraction of locally grown (source impacted) feed that is not pasture is recommended
4. C_v = As calculated above in EQ 5.3.4.1 A

b: Assumptions for EQ 5.3.4.2.3.1:

1. Feed (FI) transported from an off-site location (i.e., not grown locally) is not contaminated by facility emissions.

5.3.4.2.3.2 Pasture/Grazing ingestion

A. Equation 5.3.4.2.3.2: **Pasture/Grazing ingestion = FG × C_v × FI**

1. Pasture/Grazing ingestion = Dose through pasture/grazing (μg/d)
2. FG = Fraction of diet provided by grazing (site-specific)
3. C_v = Concentration in pasture/grazing material (μg/kg)
4. FI = Feed ingestion rate (kg/d)

a: Recommended default values EQ 5.3.4.2.3.2:

1. FG = Default values in Table 5.4 for fraction of diet provided by grazing, although a site-specific survey is recommended
2. C_v = As calculated above in EQ 5.3.4.1 A
3. FI = See Table 5.4

5.3.4.2.4 Soil ingestion

The feeds provided to dairy and beef cattle may contain small quantities of soil. A larger fraction of soil by weight of food is taken up during grazing. Rooting behavior by pigs with access to soil will result in soil ingestion. Likewise, poultry with free access to soil or pasture will also ingest soil. Defaults for soil ingestion are shown in Table 5.4.

A. Equation 5.3.4.2.4 A: **Soil ingestion = SI_a × C_s**

1. Soil ingestion = Dose through soil ingestion (μg/d)
2. SI_a = Soil ingestion rate for animal (kg/d)
3. C_s = Average soil concentration (μg/kg)

a: Recommended default values for EQ 5.3.4.2.4 A:

1. SI_a = Calculated below
2. C_s = Calculated above in EQ 5.3.2 A

B. Equation 5.3.4.2.4 B: $SI_a = [(1 - FG) \times FS_f \times FI] + [FG \times FS_p \times FI]$

1. FG = Fraction of diet provided by grazing
2. FS_f = Soil ingested as a fraction of feed ingested
3. FI = Feed ingestion rate (kg/d)
4. FS_p = Soil ingested as a fraction of pasture ingested

a: Recommended default values for EQ 5.3.4.2.4 B:

1. FG = Site specific fraction of diet provided by grazing
2. FS_f = See Table 5.4
3. FI = See Table 5.4
4. FS_p = See Table 5.4

b: Assumptions for EQ 5.3.4.2.4 B:

1. The transfer coefficient is the same for all exposure routes.
2. Soil ingested in feed (FS_f) transported from an off-site location (i.e., not grown locally) is assumed not to be contaminated by facility emissions.

5.3.4.3 Bioaccumulation in Angler-Caught Fish

The average concentration in fish (C_f) is based on the concentration in water and a chemical-specific bioaccumulation factor.

A. Equation 5.3.4.3: $Ct = Cw \times BAF$

1. Ct = Concentration in wet weight tissue (muscle) of fish (µg/kg)
2. Cw = Concentration in water (µg/kg)
3. BAF = Fish bioaccumulation factor (unitless)

a: Recommended default values for Equation 5.3.4.3:

1. Cw = As calculated above in Equation 5.3.3 A
2. BAF = Chemical-specific; see Table 5.2

b: Assumptions for Equation 5.3.4.3:

1. For conversion of a chemical concentration in a volume of water shown as µg/L, 1 L water = 1 kg water; thus, for concentration of chemical in water, µg/L = µg/kg.
2. For organic chemicals, BAFs lipid-normalized to adult rainbow trout with 4% lipid content in muscle tissue
3. For organic chemicals, BAFs based on the freely dissolved fraction in water under conditions of average particulate organic carbon and dissolved organic carbon in U.S. lakes and other water bodies
4. For inorganic compounds, BAFs based on wet weight muscle tissue concentration and on the total water concentration of the inorganic compound in water.
5. Contaminant concentrations are uniform in water based on dispersion

5.3.4.4 Bioaccumulation in Mother's Milk

The average concentration of a chemical in mother's milk (C_m) is a function of the mother's exposure through all exposure routes (i.e., inhalation, ingestion via food, drinking water, and soil, and dermal absorption via skin contact with soil contaminated with the chemical), the contaminant half-life in the mother's body, and transfer of absorbed chemical to mother's milk. The contaminant half-life in the body and transfer to mother's milk is incorporated in biotransfer coefficients (Tco) in Equation 5.3.4.4. See the TSD (OEHHA, 2012a), Appendix J for details on development of biotransfer factors. The substances assessed by the mother's milk pathway are shown in Table 5.1.

A. Equation 5.3.4.4: $$C_m = [(D_{inder} \times Tco_{m\_inder}) + (D_{ing} \times Tco_{m\_ing})] \times BW$$

1. C_m = Concentration in mother's milk (mg/kg-milk)
2. D_{inder} = The sum of DOSEair + DOSEdermal through inhalation and dermal absorption (mg/kg-BW-day)
3. D_{ing} = The sum of DOSEfood + DOSEsoil + DOSEwater through ingestion (mg/kg-BW-day)
4. Tco_{m_inder} = Biotransfer coefficient from inhalation and dermal absorption to mother's milk (d/kg-milk)
5. Tco_{m_ing} = Biotransfer coefficient from ingestion to mother's milk (d/kg-milk)
6. BW = Body weight of mother (Kg)

a: Recommended cancer risk default values for EQ 5.3.4.4:

1. D_{ing} = As calculated through ingestion of soil in EQ 5.4.3.1.1 + home-grown produce in EQ 5.4.3.2.1 + home-raised animal products in EQ 5.4.3.2.2 + drinking water in EQ 5.4.3.3.1 + angler-caught fish in EQ 5.4.3.4.1
2. D_{inder} = As calculated through inhalation in EQ 5.4.1.1 + dermal exposure in EQ 5.4.2.1
3. Tco_{m_inder} = See Table 5.5
4. Tco_{m_ing} = See Table 5.5

b: Recommended noncancer risk default values for EQ 5.3.4.4:

1. D_{ing} = As calculated through ingestion of soil in EQ 5.4.3.1.2 + home-grown produce and home-raised animal products in EQ 5.4.3.2.3 + drinking water in EQ 5.4.3.3.2 + angler-caught fish in EQ 5.4.3.4.2
2. D_{inder} = As calculated through inhalation in EQ 5.4.1.1 + dermal exposure in EQ 5.4.2.2
3. Tco_{m_inder} = See Table 5.5
4. Tco_{m_ing} = See Table 5.5

c: Assumptions for EQ 5.3.4.4:

1. Default age of mother at birth is 25 years of age, then nurses the infant for 1 year; Use 16<30 year old high-end (95^{th} percentile) daily breathing rate and intake rates for D_{ing} and D_{inder} for estimating dose to mother.
2. For inhalation dose to mother's milk, it is recommended that the EF variate in EQ 5.4.1.1 is left out for calculation of inhalation dose in the mother's milk pathway.
3. Biotransfer coefficient, Tco_{m_inder}, the same for both inhalation and dermal pathways based on lack of first-pass metabolism through the liver for both of these pathways.
4. Biotransfer coefficient, Tco_{m_ing}, the same for all ingestion pathways based on first-pass metabolism through the liver.
5. For chemicals in Table 5.5 lacking either an oral or inhalation Tco, use the oral Tco for the absent inhalation Tco (i.e., for PCDDs and PCDFs and dioxin-like PCBs), or the inhalation Tco for the absent oral Tco (i.e., for lead) in Equation 5.3.4.4.
6. The concentration in the mother's milk is determined using the derived approach to risk assessment. This method allows use of the high-end dose point estimate for driving exposure pathways and the average dose point estimates for other exposure pathways. See Sections 8.2.6 (cancer) and 8.3.3 (noncancer) for the description of the methodology on how to implement the derived methodology.

Table 5.2a Substance-Specific Default Values for Organic Multipathway Substances

				Root Uptake Factors					
Multipathway Substance	**Log K_{oc}**	**Log K_{ow}**	**Fish BAF**	**Root**	**Leafy**	**Exposed**	**Protected**	**GRAF[2]**	**Soil HalfLife (days)**
Creosotes	NA	NA	$8 \times 10^{+2}$	NA	NA	NA	NA	1.0	$4.3 \times 10^{+2}$
Diethylhexyl-phthalate	5.34[1]	7.63[1]	$4 \times 10^{+1}$	NA	NA	NA	NA	1.0	$1.5 \times 10^{+1}$
Dioxins and Furans	NA	NA	$3 \times 10^{+5}$	NA	NA	NA	NA	0.43	$7.0 \times 10^{+3}$
Hexachlorobenzene	NA	NA	$8 \times 10^{+4}$	NA	NA	NA	NA	1.0	$1.0 \times 10^{+8}$
Hexachlorocyclo-hexanes	NA	NA	$3 \times 10^{+3}$	NA	NA	NA	NA	1.0	$9.4 \times 10^{+1}$
4,4'-Methylene dianiline	2.24[3]	1.59[4]	NA	NA	NA	NA	NA	1.0	$4.6 \times 10^{+2}$
Pentachlorophenol[5]									
Polycyclic Aromatic Hydrocarbons (PAHs)	NA	NA	$8 \times 10^{+2}$	NA	NA	NA	NA	1.0	$4.3 \times 10^{+2}$
Polychlorinated Biphenyls	NA	NA	$2 \times 10^{+6}$	NA	NA	NA	NA	1.0	$3.2 \times 10^{+3}$

(1) Averaged log Kow and Koc values determined by most reliable methods (Staples et al., 1997)

(2) GRAF (Gastrointestinal Relative Absorption Factor). The guidelines allow for adjusting for bioavailability where the evidence warrants. For example, there are good data which indicate that dioxin is not as available to an organism when bound to soil or fly ash matrices relative to when it is in solution or in food. Therefore, a bioavailability factor is incorporated into the model to account for this difference. When information becomes available for other chemicals of concern, this type of bioavailability will be incorporated into the model.

(3) Measured by Hansch et al. (1985)

(4) Estimated according to methodology of Lyman et al. (1990)

(5) To be evaluated for specific default values in future amendments to the Hot Spots Program.

NA - Data Not Available or Not Applicable

Table 5.2b Substance-Specific Default Values for Inorganic Multipathway Substances

				Root Uptake Factors					
Multipathway Substance	**Log K_{oc}**	**Log K_{ow}**	**Fish BAF**	**Root**	**Leafy**	**Exposed**	**Protected**	**GRAF[1]**	**Soil HalfLife (days)**
Arsenic & Inorganic Compounds	NA	NA	$2 \times 10^{+1}$	8×10^{-3}	1×10^{-2}	2×10^{-2}	7×10^{-2}	1.0	$1.0 \times 10^{+8}$
Beryllium & Compounds	NA	NA	$4 \times 10^{+1}$	5×10^{-3}	2×10^{-4}	8×10^{-3}	3×10^{-4}	1.0	$1.0 \times 10^{+8}$
Cadmium & Compounds	NA	NA	$4 \times 10^{+1}$	8×10^{-2}	1×10^{-1}	2×10^{-2}	1×10^{-2}	1.0	$1.0 \times 10^{+8}$
Chromium VI & Compounds	NA	NA	$2 \times 10^{+1}$	$3 \times 10^{+0}$	3×10^{-1}	2×10^{-2}	7×10^{-2}	1.0	$1.0 \times 10^{+8}$
Fluorides (soluble compounds)	NA	NA	NA	9×10^{-3}	4×10^{-2}	4×10^{-3}	4×10^{-3}	1.0	$1.0 \times 10^{+8}$
Lead & Compounds	NA	NA	$2 \times 10^{+1}$	4×10^{-3}	8×10^{-3}	7×10^{-3}	3×10^{-3}	1.0	$1.0 \times 10^{+8}$
Mercury & Inorganic Compounds[2]	NA	NA	$8 \times 10^{+1}$	2×10^{-2}	2×10^{-2}	9×10^{-3}	1×10^{-2}	1.0	$1.0 \times 10^{+8}$
Nickel and compounds	NA	NA	$2 \times 10^{+1}$	6×10^{-3}	1×10^{-2}	3×10^{-3}	3×10^{-2}	1.0	$1.0 \times 10^{+8}$
Selenium & compounds	NA	NA	$1 \times 10^{+3}$	7×10^{-2}	6×10^{-2}	4×10^{-2}	3×10^{-1}	1.0	$1.0 \times 10^{+8}$

(1) GRAF (Gastrointestinal Relative Absorption Factor). The guidelines allow for adjusting for bioavailability where the evidence warrants. For example, there are good data which indicate that dioxin is not as available to an organism when bound to soil or fly ash matrices relative to when it is in solution or in food. Therefore, a bioavailability factor is incorporated into the model to account for this difference. When information becomes available for other chemicals of concern, this type of bioavailability will be incorporated into the model.

(2) Methyl mercury (MeHg) is not represented in the category “mercury & inorganic compounds”. The BAF for methyl mercury is orders of magnitude higher than for inorganic mercury. Assessment of MeHg for the fish pathway is not directly applicable to the Hot Spots program, as no facilities are known to emit MeHg directly into the air (OEHHA, 2012; OEHHA, 2006), but it may be formed by action of microbes in sediment. Assessing the methylation of mercury deposited into a water body is difficult, and is also very water body-specific. At this time OEHHA cannot address this issue in the Hot Spots program, but will consider addressing this problem in future amendments of the Guidance.

NA - Data Not Available or Not Applicable.

Table 5.3a Animal Transfer Coefficients for Persistent Organic Chemicals

Organic Chemical	Tco (d/kg)[a]				
	Cow's Milk	Chicken Egg	Chicken Meat	Cattle Meat	Pig Meat
Diethylhexylphthalate	9×10^{-5}	0.04	0.002	6×10^{-4}	5×10^{-4}
Hexachlorobenzene	0.02	20	10	0.2	0.08
Hexachlorocyclohexanes	0.01	7	5	0.2	0.09
PAHs	0.01	0.003	0.003	0.07	0.06
Polychlorinated biphenyls					
Congener 77	0.001	6	4	0.07	0.4
81	0.004	10	7	0.2	0.4
105	0.01	10	7	0.6	0.7
114	0.02	10	7	0.9	0.7
118	0.03	10	7	1	0.7
123	0.004	10	7	0.2	0.7
126	0.04	10	7	2	0.7
156	0.02	10	8	0.9	2
157	0.01	10	8	0.5	2
167	0.02	10	8	1	2
169	0.04	10	8	2	2
189	0.005	10	8	0.2	1
Unspeciated (PCB 126)[b]	0.04	10	7	2	0.7
PCDD/Fs					
Congener 2,3,7,8-TCDD	0.02	10	9	0.7	0.1
1,2,3,7,8-PeCDD	0.01	10	9	0.3	0.09
1,2,3,4,7,8-HxCDD	0.009	10	6	0.3	0.2
1,2,3,6,7,8-HxCDD	0.01	10	6	0.4	0.1
1,2,3,7,8,9-HxCDD	0.007	7	3	0.06	0.02
1,2,3,4,6,7,8-HpCDD	0.001	5	2	0.05	0.2
OCDD	0.0006	3	1	0.02	0.1
2,3,7,8-TCDF	0.004	10	6	0.1	0.02
1,2,3,7,8-PeCDF	0.004	30	10	0.1	0.01
2,3,4,7,8-PeCDF	0.02	10	8	0.7	0.09
1,2,3,4,7,8-HxCDF	0.009	10	5	0.3	0.1
1,2,3,6,7,8-HxCDF	0.009	10	6	0.3	0.09
2,3,4,6,7,8-HxCDF	0.008	5	3	0.3	0.06
1,2,3,7,8,9-HxCDF	0.009	3	3	0.3	0.03
1,2,3,4,6,7,8-HpCDF	0.002	3	1	0.07	0.06
1,2,3,4,7,8,9-HpCDF	0.003	3	1	0.1	0.02
OCDF	0.002	1	0.6	0.02	0.03
Unspeciated (2,3,7,8-TCDD)[b]	0.02	10	9	0.7	0.1

[a] All Tco values were rounded to the nearest whole number.

[b] For unspeciated mixtures, use PCB 126 Tcos to represent the class of PCBs, and 2378-TCDD Tcos to represent the class of PCDDs/Fs.

Table 5.3b Animal Transfer Coefficients for Inorganic Chemicals

Inorganic Metals and Chemicals	Tco (d/kg)[a]				
	Cow's Milk	Chicken Egg	Chicken Meat	Cattle Meat	Pig Meat
Arsenic	5×10^{-5}	0.07	0.03	2×10^{-3}	0.01[b]
Beryllium	9×10^{-7}	0.09	0.2	3×10^{-4}	0.001
Cadmium	5×10^{-6}	0.01	0.5	2×10^{-4}	0.005
Chromium (VI)	9×10^{-6}	NA[c]	NA	NA	NA
Fluoride	3×10^{-4}	0.008	0.03	8×10^{-4}	0.004[b]
Lead	6×10^{-5}	0.04	0.4	3×10^{-4}	0.001[b]
Mercury	7×10^{-5}	0.8	0.1	4×10^{-4}	0.002[b]
Nickel	3×10^{-5}	0.02	0.02	3×10^{-4}	0.001
Selenium	0.009	3	0.9	0.04	0.5

[a] All Tco values were rounded to the nearest whole number.

[b] The meat Tco was estimated using the metabolic weight adjustment ratio of 4.8 from cattle to pig

[c] NA – no data available or was not applicable

Table 5.4 Point Estimates for Animal Pathway

Parameter	Beef Cattle	Lactating Dairy Cattle	Pigs	Meat Poultry	Egg-laying Poultry
BW (body weight in kg)	533	575	55	1.7	1.6
BR_a (inhalation rate in m^3/d)	107	115	7	0.4	0.4
WI_a (water consumption in kg/d)	45	110	6.6	0.16	0.23
FI (Food Intake in kg/d) DMI[a] and/or pasture grazing[b]	9	22	2.4	0.13	0.12
FS_f (soil fraction of feed)	0.01	0.01	NA	NA	NA
FS_p (soil fraction of pasture)	0.05	0.05	0.04	0.02	0.02

[a] Dry matter intake

[b] For beef and dairy cattle, pasture grazing is assumed to be leafy vegetation (grasses, including greenchop) and accounts for half of the cattle's diet (FG=0.5 in Section 5.3.4.2.3). The default assumes on-site pasture grazing contaminated by facility emissions. Fraction of feed or dry matter intake (e.g., hay, grain) grown on-site is assumed to be contaminated by facility emissions and fraction of feed that is grown off-site is not assumed to be contaminated. A default may be used that assumes all feed is grown off-site (L=0 in Section 5.3.4.2.3), but a survey is recommended to verify the fractions of feed grown on-site and off-site.

For pigs with access to soil, but usually confined to a pen, default assumes no pasture grazing (FG=0 in Section 5.3.4.2.3). For feed, estimated intake consists of equal portions of all plant types including exposed, leafy, protected and root in which 10% (L=0.1 in Section 5.3.4.2.3) of the diet is homegrown and contaminated by facility emissions. The fraction of feed that was transported from an off-site location is assumed not to be contaminated by facility emissions.

For poultry including egg-laying and broiler chickens that have access to soil, default assumes no pasture grazing (FG=0 in Section 5.3.4.2.3). Estimated feed intake is composed of equal proportions of all plant types with 5% (L=0.05 in Section 5.3.4.2.3) homegrown and contaminated by facility emissions. The fraction of feed grown off-site and transported to the receptor was not contaminated by facility emissions.

NA - Not applicable. Assume FS_f is equal to zero.

Table 5.5 Mother's Milk Transfer Coefficients (Tco_m)[a]

Chemical/chem. group	Tco_m (day/kg-milk)
PCDDs - oral[b]	3.7
PCDFs - oral[b]	1.8
Dioxin-like PCBs - oral[b]	1.7
PAHs – inhalation[c]	1.55
PAHs – oral	0.401
Lead - inhalation[d]	0.064

[a] These compound classes represent the chemicals of greatest concern for the mother's milk pathway under the Hot Spots program. It is expected that additional transfer coefficients will be developed for other multipathway chemicals in the Hot Spots Program as data becomes available and is reviewed.

[b] Use the oral Tco_m for the inhalation and dermal pathways. The PCDD, PCDF and dioxin-like PCB Tcos were derived using a Random-effects model from individual Tco_m estimates for 7 PCDDs, 9 PCDFs and 12 dioxin-like PCBs (See OEHHA, 2012, Appendix J).

[c] Use the inhalation Tco_m for the dermal pathway

[d] Use the inhalation Tco_m for the ingestion and dermal pathways

5.4 Estimation of Dose

Once the concentrations of substances are estimated in air, soil, water, plants, and animal products, they are used to evaluate estimated exposure to people. Exposure is evaluated by calculating the daily dose in milligrams per kilogram body weight per day (mg/kg/d). The following algorithms calculate this dose for exposure through inhalation, dermal absorption, and ingestion pathways. All chemicals must be assessed for exposure through inhalation. If there are emissions of one or more of the subset of semi- or non-volatile multipathway substances, the soil ingestion pathway and the dermal soil exposure pathway are also assessed. The mother's milk pathway may also be a mandatory pathway depending on the multipathway substance released (See Table 5.1). The other exposure pathways may also need to be assessed if a survey of the exposure site shows they are present (e.g., ingestion of water, home-grown crops, home-raised animal products, and angler-caught fish).

This section contains average and high-end point estimates and data distributions for adults and children for many exposure pathways. The point-estimates and data distributions for children fall within the 3rd trimester, 0<2, 2<9, and 2<16 year age groupings. The point-estimates and data distributions for adults fall within the 16<30 and 16-70 year age groupings. When evaluating 9-, 30-, and 70-year exposure durations for cancer risk assessment, assessors will use distributions starting at the third trimester.

Workers are assessed for cancer risk as adults using 8-hour breathing rate point estimates (See Table 5.8). Point estimates for workers are listed under "offsite worker." OEHHA has not developed stochastic distributions for worker exposure. Therefore, there is no Tier 3 stochastic approach for offsite worker cancer risk assessment.

5.4.1 *Estimation of Exposure through Inhalation*

The dose through the inhalation route is estimated for cancer risk assessment and noncancer hazard assessment. Both residential and offsite worker exposures are considered. Since residential exposure includes near-continuous long-term exposure at a residence and workers are exposed only during working hours (i.e., 8 hours/day), different breathing rate distributions are used.

5.4.1.1 Residential Inhalation Dose for Cancer Risk Assessment

Exposure through inhalation is a function of the breathing rate, the exposure frequency, and the concentration of a substance in the air. For residential exposure, the breathing rates are determined for specific age groups, so inhalation dose (Dose-air) is calculated for each of these age groups, 3rd trimester, 0<2, 2<9, 2<16, 16<30 and 16-70 years. OEHHA used the mother's breathing rates to estimate dose for the 3rd trimester fetus assuming the dose to the fetus during the 3rd trimester is the same as the mother's dose. These age-specific groupings are needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8). A Tier 1 evaluation uses the high-end point estimate (i.e., the 95th percentiles) breathing rates for the inhalation

pathway in order to avoid underestimating cancer risk to the public, including children. A possible exception for using high-end breathing rates are when there is exposure to multipathway substances and two of the non-inhalation pathways drive the risk, rather than the inhalation pathway (see Chapter 8).

A. Equation 5.4.1.1: $$\text{Dose-air} = C_{air} \times \{BR/BW\} \times A \times EF \times 10^{-6}$$

1. Dose-air = Dose through inhalation (mg/kg/d)
2. C_{air} = Concentration in air ($\mu g/m^3$)
3. {BR/BW} = Daily Breathing rate normalized to body weight (L/kg body weight - day)
4. A = Inhalation absorption factor (unitless)
5. EF = Exposure frequency (unitless), days/365 days
6. 10^{-6} = Micrograms to milligrams conversion, liters to cubic meters conversion

a: Recommended default values for EQ 5.4.1.1:

1. {BR/BW} = Daily breathing rates by age groupings, see As supplemental information, the assessor may wish to evaluate the inhalation dose by using the mean point estimates in Table 5.6 to provide a range of breathing rates for cancer risk assessment to the risk manager.
2. Table (point estimates) and Table 5.7 (parametric model distributions for Tier III stochastic risk assessment). For Tier 1 residential estimates, use 95th percentile breathing rates in Table 5.6.
3. A = 1
4. EF = 0.96 (350 days/365 days in a year for a resident)

b: Assumption for EQ 5.4.1.1:

1. The fraction of chemical absorbed (A) is the same fraction absorbed in the study on which the cancer potency or Reference Exposure Level is based.

As supplemental information, the assessor may wish to evaluate the inhalation dose by using the mean point estimates in Table 5.6 to provide a range of breathing rates for cancer risk assessment to the risk manager.

Table 5.6 Point Estimates of Residential Daily Breathing Rates for 3rd trimester, 0<2, 2<9, 2<16, 16<30 and 16-70 years (L/kg BW-day)

	3rd Trimester[a]	0<2 years	2<9 years	2<16 years	16<30 years	16<70 years
	L/kg-day					
Mean	225	658	535	452	210	185
95th Percentile	361	1090	861	745	335	290

[a] 3rd trimester **breathing rates** based on breathing rates of pregnant women using the assumption that the dose to the fetus during the 3rd trimester is the same as that to the mother.

Table 5.7 Daily Breathing Rate Distributions by Age Group for Residential Stochastic Analysis (L/kg BW-day)

	3rd Trimester	0<2 years	2<9 years	2<16 years	16<30 years	16-70 years
Distribution	Max extreme	Max extreme	Max extreme	Log-normal	Logistic	Logistic
Minimum	78	196	156	57	40	13
Maximum	491	2,584	1,713	1,692	635	860
Scale	59.31	568.09	125.59		40.92	36.19
Likeliest	191.50	152.12	462.61			
Location				-144.06		
Mean	225	658	535	452	210	185
Std Dev	72	217	168	172	75	67
Skewness	0.83	2.01	1.64	1.11	0.83	1.32
Kurtosis	3.68	10.61	7.88	6.02	5.17	10.83
Percentiles						
5%	127	416	328	216	96	86
10%	142	454	367	259	118	104
25%	179	525	427	331	161	141
50%	212	618	504	432	207	181
75%	260	723	602	545	252	222
80%	273	758	631	572	261	233
90%	333	934	732	659	307	262
95%	361	1090	861	745	335	290
99%	412	1430	1140	996	432	361

5.4.1.2 Offsite Worker (MEIW) Inhalation Dose for Cancer Risk Assessment

For worker exposure, the default assumes working age begins at 16 years, and that exposures to facility emissions occur during the work shift, typically up to 8 hours per day during work days. Breathing rates that occur over an 8-hour period vary depending on the intensity of the activity (See Table 5.8), and are used to estimate the inhalation dose. The 8-hour breathing rates may also be useful for cancer risk assessment of children and teachers exposed at schools during school hours.

Another risk management consideration for the offsite worker scenario for cancer assessment of a Hot Spots facility is whether there are women of child-bearing age at the MEIW location and whether the MEIW has a daycare center. Since the third trimester is only a short segment of the 25 year exposure duration used for the MEIW, the resulting risk estimate would not differ significantly. An exception to this assumption is high exposure to carcinogens over a short period, as might occur during short-term projects (see Section 8.2.10). In this case, risk assessment during the third trimester may be warranted. However, if there is onsite daycare at the MEIW, then the risks to the children will be underestimated using the offsite adult worker scenario due to increased exposure (per kg body weight) and increased sensitivity to carcinogen exposure (see Section 8.2.1). In this case, the Districts may wish to include a calculation of inhalation dose for the children in the onsite daycare, assuming they could be there from 0 to age 6 years.

Exposed workers may be engaged in activities ranging from desk work, which would reflect breathing rates of sedentary/passive or light activities, to farm worker activities, which would reflect breathing rates of moderate intensity (See Table 5.9). OEHHA recommends default (Tier 1) point estimate 8-hour breathing rates in L/kg-8-hrs based on the mean and 95th percentile of moderate intensity activities, 170 and 230 L/kg-8-hrs, respectively, for adults 16-70 years old.

Many facilities operate non-continuously, as in only 8-10 hours per day, but the air dispersion modeling is performed as if the emissions were uniformly emitted over 24 hours a day, 7 days per week. The air dispersion computer model used, including AERMOD and other models, typically calculate an annual average air concentration based on actual operating conditions but also include the hours of nonoperation in the average concentration.

Therefore, there are two components that determine the worker exposure to facility emissions:

1) What is the estimated concentration the worker is exposed to (i.e., breathes), during the work shift, and

2) What is the amount of time the offsite worker's schedule overlaps with the facility's emission schedule?

There are two approaches to estimating the modeled concentration the worker is breathing during the work shift. The first approach uses a worker adjustment factor (i.e.,

the WAF) to approximate what the worker is breathing based on the modeling run used for residential receptors. The second approach uses a special modeling run with the hourly raw results from an air dispersion analysis and is described in Appendix M.

The first and more basic approach is to obtain the long term average concentration as you would for modeling a residential receptor, then adjusting this exposure concentration using the calculated WAF (EQ 5.4.1.2 B) to estimate the concentration the offsite worker is exposed to during the work shift (shown as (C_{air} × WAF) in EQ 5.4.1.2 A). This method is characteristic of a default approach used in a Tier 1 assessment. Once the exposure concentration is determined, the worker's inhalation dose (Dose-air) can be calculated as shown in EQ 5.4.1.2 A.

The second approach for determining the air concentration the worker is exposed to uses a refined modeling run where the hourly raw dispersion model output are post processed to examine the hourly concentrations that fall within the offsite worker's shift. This method provides a more representative estimate of the air concentration, but is more complex, and time consuming than the first method. See Appendix M for information on how to simulate the long term concentration for the offsite worker that can be used to estimate inhalation cancer risk.

The HARP software has the ability to calculate worker impacts using an approximation factor and, in the future, it will have the ability to post process refined worker concentrations using the hourly raw results from an air dispersion analysis.

If the off-site worker's shift does not completely overlap the emission schedule of the facility, then a Discount Factor (DF) may be applied to the WAF. Calculation of the DF is shown in EQ 5.4.1.2 C. The default assumption is that the offsite worker's shift falls completely within the emission schedule of the facility, in which case DF=1. Use of a DF less than 1 requires a survey at the MIEW to verify that some portion of the off-site worker shift is not subject to the facility emissions.

A. Equation 5.4.1.2 A: $\textbf{Dose-air} = (C_{air} \times WAF) \times \{BR/BW\} \times A \times EF \times 10^{-6}$

1. Dose-air = Dose through inhalation (mg/kg/d)
2. C_{air} = Annual average concentration in air ($\mu g/m^3$)
3. WAF = Worker air concentration adjustment factor (unitless)
4. {BR/BW} = Eight-hour breathing rate normalized to body weight (L/kg body weight - day)
5. A = Inhalation absorption factor (unitless)
6. EF = Exposure frequency (unitless), days/365 days)
7. 10^{-6} = Micrograms to milligrams conversion, Liters to cubic meters conversion

a: Recommended default values for EQ 5.4.1.2 A:

1. WAF = See EQ. 5.4.1.2 B for formula to calculate WAF, or App. M for refined post-processing modeling to calculate WAF.
2. {BR/BW} = For workers, use age16-70 year, 95th percentile, moderate intensity 8-hour point estimate breathing rates (see Table 5.8). No worker breathing rate distributions exist for stochastic risk assessment.
3. A = 1
4. EF = 0.68 (250 days / 365 days). Equivalent to working 5 days/week, 50 weeks/year.

b: Assumption for EQ 5.4.1.2 A:

1. The fraction of chemical absorbed (A) through the lungs is the same fraction absorbed in the study on which the cancer potency factor is based.
2. The source emits during the daylight hours. Calculate WAF (EQ 5.4.1.2 B) if a special post-processing modeling run described in App. M was not completed. For nighttime emissions and exposure scenarios, see Appendix N.

B. Equation 5.4.1.2 B: $$WAF = (H_{res} / H_{source}) \times (D_{res} / D_{source}) \times DF$$

1. WAF = Worker adjustment factor (unitless)
2. H_{res} = Number of hours per day the annual average residential air concentration is based on (always 24 hours)
3. H_{source} = Number of hours the source operates per day
4. D_{res} = Number of days per week the annual average residential air concentration is based on (always 7 days)
5. D_{source} = Number of days the emitting source operates per week
6. DF = Discount factor, for when the offsite worker's schedule partially overlaps the source's emission schedule

b: Recommended default values for EQ 5.4.1.2 B:

1. DF = 1 for offsite worker's schedule occurring within the source's emission schedule. A site-specific survey may be used to adjust the DF using EQ 5.4.1.2 C.

C. Equation 5.4.1.2 C: $$DF = (H_{coincident} / H_{worker}) \times (D_{coincident} / D_{worker})$$

1. $H_{coincident}$ = Number of hours per day the offsite worker's schedule and the source's emission schedule coincide
2. H_{worker} = Number of hours the offsite worker works per day
3. $D_{coincident}$ = Number of days per week the offsite worker's schedule and the source's emission schedule coincide
4. D_{worker} = Number of days the offsite worker works per week

Tier 2 adjustments for EQ 5.4.1.2 A-C may be used for:

1. Eight-hour breathing rate. Point estimates in Table 5.8 for lower breathing rates of sedentary/passive and light intensity work activities may be substituted in site-specific Tier 2 scenarios. Table 5.9 can be used to estimate breathing rate intensities for various job activities. Use of different breathing rates requires a survey of the exposed workplace and approval by Air District, ARB and OEHHA.
2. Discount Factor (DF) in EQ 5.4.1.2 C. If a site-specific survey of the offsite worker schedule only partially overlaps with the source's emission schedule, then a DF less than 1 may be calculated. Use of a DF less than 1 requires a survey of the exposed workplace and approval by the Air District or ARB.

The 8-hour breathing rates are based on minute ventilation rates derived by U.S. EPA (2009). U.S. EPA employed a metabolic equivalent (METS) approach for estimating breathing rates. This method determines daily time-weighted averages of energy expenditure (expressed as multipliers of the basal metabolic rate) across different levels of physical activity. The 8-hour breathing rates shown in Table 5.8 are divided into three categories:

Sedentary & Passive Activities (METS $\leq$ 1.5)

Light Intensity Activities (1.5 < METs $\leq$ 3.0)

Moderate Intensity Activities (3.0 < METs $\leq$ 6.0)

For example, a METS = 1 is roughly equivalent to energy expenditure during sleep and is close to the basal metabolic rate. A METS activity that is two to three times greater (METS = 2 to 3) is characteristic of light intensity activities, such as administrative office work or sales work as shown in Table 5.9.

Under a Tier 1 scenario, the risk assessor may simply use the 95th percentile breathing rate for moderate intensity activities of 230 L/kg-8 hrs in Eq. 5.4.1.2 A to calculate the daily dose via the inhalation route to the worker. In an example of a Tier 2 scenario, the risk assessor surveys the workplace and determines that the worker(s) at the MEIW receptor are primarily sitting at a desk performing administrative-type work on a computer. Referring to Table 5.9, this activity corresponds most closely to "administrative office work" with a mean activity level of 1.7 and a SD = 0.3. This level of activity is considered "light intensity activity" (i.e., 1.5 < METs $\leq$ 3.0). With the prior approval of the Air District or ARB, the risk assessor may then use the 95th percentile breathing rate of 100 L/kg-8 hr for light intensity activities in Equation 5.4.1.2 A.

Table 5.8. Eight-Hour Breathing Rate (L/kg per 8 Hrs) Point Estimates for Males and Females Combined[a,b]

	0<2 years	2<9 years	2<16 years	16<30 years	16-70 years
	Sedentary & Passive Activities (METS $\leq$ 1.5)				
Mean	200	100	80	30	30
95th Percentile	250	140	120	40	40
	Light Intensity Activities (1.5 < METs $\leq$ 3.0)				
Mean	490	250	200	80	80
95th Percentile	600	340	270	100	100
	Moderate Intensity Activities (3.0 < METs $\leq$ 6.0)				
Mean	890	470	380	170	170
95th Percentile	1200	640	520	240	230

[a] For pregnant women, OEHHA recommends using the mean and 95th percentile 8-hour breathing rates based on moderate intensity activity of 16<30 year-olds for 3rd trimester.
[b.] Breathing rates in the table may be used for worker, school, or residential exposures

Table 5.9. METS Distributions for Workplace and Home Activities

Activity Description	Mean	Median	SD	Min	Max
Workplace Activities					
Administrative office work	1.7	1.7	0.3	1.4	2.7
Sales work	2.9	2.7	1.0	1.2	5.6
Professional	2.9	2.7	1.0	1.2	5.6
Precision/production/craft/repair	3.3	3.3	0.4	2.5	4.5
Technicians	3.3	3.3	0.4	2.5	4.5
Private household work	3.6	3.5	0.8	2.5	6.0
Service	5.2	5.3	1.4	1.6	8.4
Machinists	5.3	5.3	0.7	4.0	6.5
Farming activities	7.5	7.0	3.0	3.6	17.0
Work breaks	1.8	1.8	0.4	1.0	2.5
Household/Neighborhood Activities					
Sleep or nap	0.9	0.9	0.1	0.8	1.1
Watch TV	1.0	1.0	-	1.0	1.0
General reading	1.3	1.3	0.2	1.0	1.6
Eat	1.8	1.8	0.1	1.5	2.0
Do homework	1.8	1.8	-	1.8	1.8
General personal needs and care	2.0	2.0	0.6	1.0	3.0
Indoor chores	3.4	3.0	1.4	2.0	5.0
Care of plants	3.5	3.5	0.9	2.0	5.0
Clean house	4.1	3.5	1.9	2.2	5.0
Home repairs	4.7	4.5	0.7	4.0	6.0
General household chores	4.7	4.6	1.3	1.5	8.0
Outdoor chores	5.0	5.0	1.0	2.0	7.0
Walk/bike/jog (not in transit) age 20	5.8	5.5	1.8	1.8	11.3
Walk/bike/jog (not in transit) age 30	5.7	5.7	1.2	2.1	9.3
Walk/bike/jog (not in transit) age 40	4.7	4.7	1.8	2.3	7.1

Table 5.10 lists some WAFs for a few typical scenarios. For example, if the source is continuously emitting, then the offsite worker is assumed to breathe the long-term annual average concentration during their work shift. The WAF then becomes one and no concentration adjustments are necessary in this situation when estimating the inhalation cancer risk. If the source is non-continuously emitting for 8 hours/day, 5 days/week and the offsite worker's shift completely overlaps the emitting facility's operating schedule, then the WAF would be 4.2:

$$(24 \text{ hrs/day} / 8 \text{ hrs/day}) \times (7 \text{ days/week} / 5 \text{ days/week}) = 4.2$$

If the offsite worker's 8 hour/day shift only overlaps the emitting facility's operation schedule for 4 hrs/day, then the WAF is 2.1 because the DF = 0.5 will reduce the WAF by half: DF = (4 hrs/day / 8 hrs/day) x (5 days/week / 5 days/week) = 0.5

Table 5.10: Example Worker Adjustment Factors (WAF) to Convert a Long-Term Daily Average Emission Concentration to an Off-Site Worker Receptor Exposure

Off-Site Workers' Shift Overlap with Facility's Emission Schedule[a]	Facility Operating Schedule	Adjustment Factor
8 hrs/day, 5 days/week	Continuous (24 hrs/7 days/week)	1.0
8 hrs/day, 5 days/week[b]	Non-continuous (8 hrs/5 days/week)	4.2
4 hrs/day, 5 days/week	Non-continuous (8 hrs/5 days/week)	2.1

[a] Worker works 8 hours per day, 5 days per week
[b] Workers' work hours completely overlap the facilities operating hours

5.4.1.3 Inhalation Dose for Children at Schools and Daycare Facilities for Cancer Risk Assessment

The 8-hour breathing rates and inhalation dose equations (EQ 5.4.1.2 A-C) may also be used to estimate risk to children when exposures occur while at school or at day care facilities. Breathing rate point estimates to use in Table 5.8 depend on the ages of the children at the exposed schools and day cares. As a Tier 1 default, moderate intensity breathing rates are recommended. Equations 5.4.1.2 A-C is used in the same way to estimate dose in children as it is for workers.

5.4.1.4 Non-Cancer Inhalation Exposure for Workers and Residents

For typical daily work shifts of 8-9 hours, acute, 8-hour and chronic Reference Exposure Levels (RELs) described in Chapter 8 are used in health risk assessments to characterize the noncancer risks using the Hazard Index approach described in Chapter 8 and in OEHHA (2008). Uncertainty factors are already incorporated into the RELs used to assess noncancer risk, as explained in Chapter 8, so all that is needed to evaluate the noncancer hazard is the air concentration that the worker is exposed to. The modeled maximum 1-hour air concentration is determined for acute hazard assessment and the annual average air concentration is determined for chronic hazard assessment. The modeled average air concentration during a work shift is determined for 8-hour hazard assessment using the adjusted annual average air concentration described below.

The 8-hour RELs are primarily designed to address offsite worker inhalation exposure at the MEIW because they better characterize the daily intermittent exposures of workers than the chronic RELs do. They are used in estimating the 8 hour Hazard Index for offsite workers. The 8-hour RELs should be used for typical daily work shifts of 8-9 hours. For further questions, assessors should contact OEHHA, the District, or reviewing authority to determine if the 8-hour RELs should be used in your HRA. Any discussions or directions to exclude the 8-hour REL evaluation should be documented in the HRA.

Note, however, there are only a handful of 8-hour RELs currently adopted for use in the Hot Spots program. Therefore, we also recommend performing chronic noncancer exposure assessment for the offsite worker (MEIW) based on the annual average air concentration at the MEIW. Evaluation of the chronic Hazard Index should help protect workers who routinely work longer than 8 hour shifts. Exposure to multipathway substances also requires noncancer hazard assessment for the dermal and oral soil exposure pathways for offsite workers. Because there are few 8-hour RELs currently available, hazard assessment for the noninhalation pathways for multipathway substances is only applied when estimating the chronic Hazard Index.

In addition, the Districts may wish to determine if there is an onsite daycare at the MEIW and include a calculation of the chronic and 8-hour inhalation dose for children, although onsite hazard assessment is not a requirement for a Hot Spots risk assessment.

As explained in Section 5.4.1.2 for cancer risk, the modeled annual average air concentration is adjusted to the air concentration that the worker is actually exposed to if the facility operates non-continuously. The typical method for this adjustment is by calculating the Worker Adjustment Factor (WAF) shown in EQ 5.4.1.4 B and multiplying this value by the annual average air concentration (C_{air}, in $\mu g/m^3$) in EQ 5.4.1.4 A.

Unlike cancer risk assessment, no discount factor (DF) is applied in noncancer assessment for partial overlap between the worker's schedule and the source's emission schedule. Adjustments for worker vacations, work shifts for shortened weeks (e.g., 1 - 4 days), and worker time away on weekends are also not appropriate.

An alternative refined post-processing method, described in Appendix M, may be used to estimate the air concentration the worker is exposed to during their work schedule. OEHHA may be consulted about the particular chemical involved if it is important to make a more refined analysis.

The equation to adjust the annual average air concentration to a worker 8-hour exposure concentration (i.e., the adjusted annual average ground level concentration) is expressed as:

A. Equation 5.4.1.4 A: $$\text{Adjusted } C_{air} \ (\mu g/m^3) = C_{air} \times WAF$$

Where WAF is determined as:

B. Equation 5.4.1.4 B: $$WAF = (H_{res} / H_{source}) \times (D_{res} / D_{source})$$

a: Assumptions for EQ 5.4.1.4 B:

1. No adjustment of the WAF allowed for partial overlap of the worker's schedule and the source's emission schedule.

Alternatives for calculating off-site worker Adjusted C_{air} in EQ 5.4.1.4 A-B:

1. Rather than calculate the WAF for a non-continuous emitting facility, a post-processing of the hourly raw dispersion model output and examination of the hourly concentrations that fall within the offsite worker's shift can be conducted to estimate the air concentration the worker is exposed to. This method is a more refined, complex, and time consuming approach, but should result in a more representative exposure concentration. See Appendix M for information on how to simulate the exposure concentration for the off-site worker.
2. For continuously-emitting facilities (i.e., 24 hrs/day, 7 days/week), if an assessor does not wish to assume the worker breathes the long-term annual average concentration during the work shift, then a refined concentration can also be post-processed as described in Appendix M. All alternative assumptions should be approved by the reviewing authority and supported in the presentation of results.

For residential exposure to non-continuously operating facilities, the modeled maximum 1-hour and chronic air concentrations at the MEIR are determined for noncancer hazard assessment. Hazard assessment for repeated 8-hour exposure at the MEIR is not required. Chronic exposure assessment based on the annual average air concentration should adequately protect individuals, in part because residents are considered to be present at the MEIR at or near 24 hrs per day. Many facilities operate for periods longer than 8 hours per day and the hazards are better characterized based on chronic exposure. Nevertheless, differences between 8-hour and chronic exposures (i.e., higher daily 8-hour exposures vs. lower longer daily exposure 24 hrs/day) may result in different toxicological responses including potentially greater toxicological responses with either 8-hour or chronic exposure. There may also be cases such as special meteorological situations (e.g., significant diurnal-nocturnal meteorological differences) where the 8-hour REL will be more protective than the chronic REL. Thus, the air districts may also elect to have an 8-hour hazard assessment performed at the MEIR, using daily 8 hour exposures and the 8 hr RELs.

Eight-hour exposure assessment is not recommended for continuously emitting sources for residential receptors. In this situation it is only necessary to estimate chronic exposure based on the annual average concentration. However, there may be situations where the air district may wish to assess an 8-hour residential exposure to continuously operating facilities, for example, where there are significant differences in modeled concentration of emissions during the day due to diurnal wind patterns.

For estimating the air concentration from non-continuously operating facilities, EQ 5.4.1.4.A is also used to adjust the annual average concentration to what the residents are exposed to. This is the air concentration that the 8-hour REL will be compared to as discussed in Chapter 8. The alternative refined post-processing method described in Appendix M may also be used to estimate residential exposure.

In summary, the requirements for noncancer hazard assessment using the Hazard Index approach at the MEIW and MEIR are as follows.

For offsite worker exposure:

- Acute hazard assessment based on the maximum 1-hour air concentrations and 1-hour RELs
- Eight-hour hazard assessment based on daily average 8-hour exposure (estimated using adjusted annual average air concentration in EQ 5.4.1.4 A and B or by post-processing method in App. M) for those substances with 8-hour RELs
- Chronic hazard assessment based on annual average exposure and chronic RELs, and oral chronic RELs for noninhalation routes of multipathway substances

For residential exposure:

- Acute hazard assessment based on the maximum 1-hour air concentration and 1-hour RELs
- Eight-hour hazard assessment based on daily average 8-hour exposure not required, but can be performed at the discretion of the air districts for exposure to non-continuously operating facilities based on the adjusted annual average air concentration (EQ 5.4.1.4 A and B or method in App. M). Eight-hour assessments not recommended for exposure to continuously operating facilities
- Chronic hazard assessment based on annual average exposure and chronic RELs, and oral chronic RELs for noninhalation routes of multipathway substances

5.4.1.5 Exposure Frequency and Age Groupings for Noncancer Hazard Assessment

For cancer risk, the basic assumption is that risk is associated with cumulative dose of carcinogen. Thus, the dose used to estimate cancer risk can be adjusted for exposure frequency, as well as time spent within the MEIR or MEIW location. Chronic RELs are not necessarily related to cumulative dose. Thus, adjusting the estimated dose used to calculate hazard index for exposure frequency or time away from the MEIR or MEIW is not appropriate.

The average daily dose for chronic noncancer assessment is based on exposure beginning at birth to 70 years of age, necessitating calculation of a time-weighted average for age 0-2, 2-16 and 16-70 years. Since we are not applying Age Sensitivity Factors for assessing non-cancer hazard, the 3rd trimester is not explicitly called out for determining dose, as it is for cancer risk assessment. Rather adult exposure is considered, which would include pregnant women in any trimester. Both inhalation and oral RELs incorporate safety factors to protect sensitive human populations.

5.4.2 *Estimation of Exposure through Dermal Absorption*

Exposure through dermal absorption (dose-dermal) is a function of the soil or dust loading of the exposed skin surface, the amount of skin surface area exposed, and the concentration and availability of the substance. In the previous edition of OEHHA's

exposure guidelines document (OEHHA, 2000), we recommended using specified average and high-end point estimate values for four of the variates (body weight, exposed surface area of skin, soil load on skin and frequency of exposure) in the stochastic analysis for dermal dose. This equation required multiplying values together, which could lead to overly conservative exposure estimates when high-end values were used. By combining information from the four variates into one composite distribution, over-conservatism may be avoided.

To this end, OEHHA created a new variate, "annual dermal load", or ADL, which is a composite of the body surface area (BSA) per kg body weight, exposure frequency, and soil adherence variates. Point estimates from the composite "annual dermal load" can be used for point estimate assessments while parameters and information on the type of distribution (e.g., lognormal) can be used for Tier III stochastic risk assessments. For details on the development of the ADL, refer to the Technical Support Document for Exposure and Stochastic Analysis (OEHHA, 2012).

5.4.2.1 Dermal Dose for Cancer Risk Assessment

The dose through residential dermal exposure to contaminated soil varies by age and is calculated for each age group (e.g., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 and 16-70 yrs). These age-specific groupings are needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8). This pathway is also assessed for exposure to offsite workers; a separate ADL for offsite workers is presented in Table 5.11. Children at a MEIW daycare, if present, may also be assessed for exposure if the District deems it advisable.

A. Equation 5.4.2.1: $$\mathbf{Dose_{dermal} = ADL \times Cs \times ABS \times 10^{-9} / 365}$$

1. $Dose_{dermal}$ = Exposure dose through dermal absorption (mg/kg-d)
2. ADL = Annual dermal load (mg soil/kg BW-yr)
3. C_s = Average soil concentration (μg/kg)
4. ABS = Fraction absorbed across skin (unitless)
5. 10^{-9} = Conversion factor for chemical & soil (μg to mg, mg to kg)
6. 1/365 = Conversion factor for ADL from yrs to days

a: Recommended default values for EQ 5.4.2.1:

1. ADL = See Table 5.11 (point estimates) & Table 5.12 a-d (distributions)
2. C_s = Calculated above in EQ 5.3.2 A
3. ABS = See Table 5.13

b: Assumption for EQ 5.4.2.1:

1. The ADL for the third trimester of the fetus is based on the ADL of the mother; when normalized to body weight, we assume that exposure to the

mother and the fetus will be the same. The mother's exposure is based on that of adults 16-30 years of age in Table 5.11 and 5.12d.

2. Exposure frequency (EF) for vacation time spent away from exposure does not appear as a variate in EQ 5.4.2.1, as it is incorporated in the ADL and includes a 2-week vacation per year away from dermal soil exposure for both residents and offsite workers.

Climate will strongly influence people's choice of clothing. Due to California's varied climatic regions and existing data on clothing choices at different temperatures, three levels of climatic conditions, warm, mixed, and cold, are used to describe California's climate regions:

1. A warm climate is characteristic of Southern California areas such as Los Angeles, which can have warm to hot temperatures throughout the year.
2. A "mixed" climate is one that has warm-to-hot temperatures during much of the year (daily highs over 80 degrees are common), roughly from April to October, and cold temperatures (lows near or below freezing) during the remainder of the year. The mountains and central valley are examples of a mixed climate.
3. A cold climate is representative of San Francisco, Eureka, and other northern coastal communities, which have cool temperatures (daily highs of less than 65 degrees) for the majority of the year and can receive a considerable amount of fog and rainfall.

OEHHA recommends consulting the local air district for assistance on selecting the most appropriate climate.

Table 5.11 Recommended Annual Dermal Load Point Estimates (in mg/kg-yr) for Dermal Exposure

	3rd Trimester[a]	Children 0<2 yrs	Children 2<9 yrs	Children 2<16 yrs	Adults[b]	Offsite Worker[c]
Warm climate						
Mean	1.2×10^3	3.6×10^3	7.5×10^3	6.4×10^3	1.2×10^3	2.6×10^3
95th percentile	2.6×10^3	4.3×10^3	9.1×10^3	8.5×10^3	2.6×10^3	5.0×10^3
Mixed climate						
Mean	1.1×10^3	2.2×10^3	6.6×10^3	5.7×10^3	1.1×10^3	2.6×10^3
95th percentile	2.4×10^3	2.9×10^3	8.7×10^3	8.1×10^3	2.4×10^3	5.0×10^3
Cold climate						
Mean	0.7×10^3	1.2×10^3	3.1×10^3	2.8×10^3	0.7×10^3	2.6×10^3
95th percentile	2.1×10^3	1.9×10^3	5.2×10^3	5.1×10^3	2.1×10^3	5.0×10^3

[a] The ADL for the 3rd trimester of the fetus is based on the ADL of the mother; when normalized to body weight, we assume that exposure to the mother and the fetus will be the same

[b] Residential adult ADLs are for both 16<30 and 16-70 year age groups

[c] Assumes exposure only to face, hands and forearms regardless of climate region

Tables 5.12a - d Annual Dermal Load Distributions by Age Group and Climate for Stochastic Analysis

Table 5.12a Annual Dermal Load (mg/kg-yr) Distributions for the 0<2 Year Age Group

Climate Type	Warm climate	Mixed climate	Cold climate
Distribution	Student's t	Logistic	Triangular
Minimum			0.2×10^3
Likeliest			0.7×10^3
Maximum			2.6×10^3
Scale	0.41	0.28	
Deg. freedom	3		
Midpoint	3.6×10^3		
Mean	3.6×10^3	2.2×10^3	1.2×10^3
50th percentile	3.6×10^3	2.2×10^3	0.9×10^3
90th percentile	4.1×10^3	2.8×10^3	1.9×10^3
95th percentile	4.3×10^3	2.9×10^3	1.9×10^3
99th percentile	4.7×10^3	3.1×10^3	2.1×10^3

Table 5.12b Annual Dermal Load (mg/kg-yr) Distributions for the 2<9 Year Age Group

Climate Type	Warm climate	Mixed climate	Cold climate
Distribution	Min extreme	Min extreme	Triangular
Minimum			0.4×10^3
Likeliest	8.0×10^3	7.3×10^3	1.9×10^3
Maximum			6.9×10^3
Scale	0.1	1.3	
Mean	7.5×10^3	6.6×10^3	3.1×10^3
50th percentile	7.7×10^3	6.5×10^3	2.3×10^3
90th percentile	8.7×10^3	8.4×10^3	5.1×10^3
95th percentile	9.1×10^3	8.7×10^3	5.2×10^3
99th percentile	9.7×10^3	9.4×10^3	5.7×10^3

Table 5.12c Annual Dermal Load (mg/kg-yr) Distributions for the 2<16 Year Age Group

Climate Type	Warm climate	Mixed climate	Cold climate
Distribution	Min extreme	Logistic	Triangular
Minimum			0.3×10^3
Likeliest	7.2×10^3		1.6×10^3
Maximum			6.9×10^3
Scale	1.29	0.91	
Mean	6.4×10^3	5.7×10^3	2.8×10^3
50th percentile	6.6×10^3	5.7×10^3	2.2×10^3
90th percentile	8.1×10^3	7.7×10^3	4.8×10^3
95th percentile	8.5×10^3	8.1×10^3	5.1×10^3
99th percentile	9.3×10^3	8.9×10^3	5.6×10^3

Table 5.12d Annual Dermal Load (mg/kg-yr) Distributions for Residential Adults (Age 16-30 and 16-70 Years)[a] and Offsite Workers

Receptor	Residential Adult			Offsite Worker
Climate Type	Warm	Mixed	Cold	All Climates[b]
Distribution	Beta	Beta	Gamma	Lognormal
Minimum	0.2×10^3	0.02×10^3		
Maximum	3.3×10^3	0.3×10^3		
Scale			0.07	
Mean	1.2×10^3	1.1×10^3	0.7×10^3	2.6×10^3
50th percentile	1.2×10^3	1.0×10^3	0.5×10^3	2.3×10^3
90th percentile	2.4×10^3	2.1×10^3	1.6×10^3	4.5×10^3
95th percentile	2.6×10^3	2.4×10^3	2.1×10^3	5.0×10^3
99th percentile	2.9×10^3	2.6×10^3	2.3×10^3	6.4×10^3

[a] The ADL distribution for the 3rd trimester is based on the ADL distribution of the mother; we assume the same ADL distribution for residential adult (the mother) and the fetus

[b] Face, hands and forearms are exposed only, regardless of climate

Table 5.13 Dermal Absorption Fraction Factors (ABS) as Percent from Soil for Semi-Volatile and Solid Chemicals under the OEHHA "Hot Spots" Program

Chemical	ABS
Inorganic chemicals	
Arsenic	6
Beryllium	3
Cadmium	0.2
Chromium (VI)	2
Fluorides (soluble compounds)	3
Lead	3
Mercury	4
Nickel	2
Selenium	3
Organic chemicals	
Creosotes	13
Diethylhexylphthalate	9
Hexachlorobenzene	4
Hexachlorocyclohexanes	3
4,4'methylene dianiline	10
Pentachlorophenol	[a]
Polychlorinated biphenyls	14
Polychlorinated dibenzo-p-dioxins and dibenzofurans	3
Polycyclic aromatic hydrocarbons	13

[a] To be determined in future amendments to the Hot Spots Program

Skin permeability is related to the solubility or strength of binding of the chemical in the delivery matrix (soil or other particles) versus the receptor matrix, the skin's stratum corneum. Fractional dermal absorption point estimate values were derived by OEHHA from available literature sources for the semi-volatile and nonvolatile chemicals in the "Hot Spots" program. The rationale for the chemical-specific dermal absorption fraction values, and the use of default values in cases where sufficient data are lacking, can be found in Appendix F of the Technical Support Document for Exposure and Stochastic Analysis (OEHHA, 2012).

5.4.2.2 Chronic Noncancer Dermal Dose

Dermal exposure, and thus annual dermal load (ADL), varies by age group. Therefore, a time-weighted average ADL for age 0-70 years (0-2, 2-16, and 16-70 years) is estimated for chronic residential exposure using ADL values in Table 5.12. This exposure pathway is also assessed for offsite workers using the offsite worker ADL values in Table 5.12d. Children at a MEIW daycare, if present, may also be assessed for exposure if the District deems it advisable. The contribution to the dermal dose is determined for each age group in EQ 5.4.2.2:

A. Equation 5.4.2.2: $\mathbf{Dose_{dermal} = ADL \times Cs \times ABS \times 10^{-9} \times ED/AT \times (1/350)}$

1. $Dose_{dermal}$ = Exposure dose through dermal absorption (mg/kg/d)
2. ADL = Annual dermal load (mg/kg-yr), age-specific
3. Cs = Average soil concentration (μg/kg)
4. ABS = Fraction absorbed across skin (unitless)
5. 10^{-9} = Conversion factor for chemical & soil (μg to mg, mg to kg)
6. 1/350 = Conversion factor for ADL from yrs to days (Note: this conversion is needed to remove EF, expressed as 350 days/365 days, from the ADLs in Table 5.12a-d)
7. ED = Exposure duration for specified age groups: 2 yrs for 0<2, 14 yrs for 2<16, 54 yrs for 16-70 for residential exposure,
8. AT = Averaging time for residential exposure – 70 yrs

a: Recommended default values for EQ 5.4.2.2:

1. ADL = See Table 5.11 for point estimates by age group, climate region and receptor type (resident or worker)
2. Cs = Calculated above in EQ 5.3.2 A
3. ABS = See Table 5.13

b: Recommended off-site worker default modifications to EQ 5.4.2.2:

1. Chronic dermal dose to the off-site worker assumes only adult exposure and is incorporated into the off-site worker ADL in Table 5.12d.
2. A time-weighted average estimate of dose is not necessary and the ED and AT variates are left out of EQ 5.4.2.2 for dermal dose to the worker.

c: Recommended nursing mother default modifications to EQ 5.4.2.2:

1. For dermal dose to mother's milk, use the ADL for age 16-30 years in Table 5.12d.
2. The ED and AT variates in EQ 5.4.2.2 are left out for dermal dose in the mother's milk pathway.

d: Assumptions for EQ 5.4.2.2:

1. For cancer risk assessment, Exposure Frequency (EF) for vacation time away from exposure is incorporated into the ADLs shown in Tables 5.11 and 5.12 using the basic assumption that cancer risk is associated with cumulative dose of carcinogen. The dose used to estimate cancer risk can be adjusted for EF, and for time spent within the MEIR or MEIW location. Chronic RELs are not necessarily related to cumulative dose. Thus, adjusting the estimated dose for EF at the MEIR or MEIW is not appropriate, and the unadjusted daily rate is used in EQ 5.4.2.2.
2. For worker exposure, the annual average concentration should not be adjusted to account for worker and facility emission schedules, as done for

inhalation cancer risk assessment. The pollutant will be deposited and accumulate in the soil in the absence or presence of the worker; therefore, the total deposition and soil concentration will be dependent on the annual average air concentration.

For residential chronic exposure, the dermal dose contribution for each age group is summed together to obtain the time-weighted average daily dermal dose for chronic hazard assessment:

(ADL age 0<2 × Cs × ABS × 10^{-9} × 2 / 70 × (1/350)) +

(ADL age 2<16 × Cs × ABS × 10^{-9} × 14 / 70 × (1/350)) +

(ADL age 16-70 × Cs × ABS × 10^{-9} × 54 / 70 × (1/350)) = Chronic $Dose_{dermal}$

5.4.3 *Estimation of Exposure through Ingestion*

Exposure through ingestion is a function of the concentration of the substance in the ingested soil, water, and food, the gastrointestinal absorption of the substance, and the amount ingested.

5.4.3.1 Exposure through Ingestion of Soil

There are no distributions for soil ingestion currently recommended. Tier III stochastic risk assessments should include a high-end point estimate of soil ingestion, soil loading, exposure frequency and soil area.

5.4.3.1.1 Soil Ingestion Dose for Cancer Risk

The exposure dose through residential soil ingestion varies by age and is calculated for each age group ((e.g., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 and 16-70 yrs). These age-specific groupings are needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8). This pathway is also assessed for exposure to off-site workers. Children at a MEIW daycare, if present, may also be assessed for exposure if the District deems it advisable. The dose from inadvertent soil ingestion can be estimated by the point estimate approach using the following general equation:

A. Equation 5.4.3.1.1: $DOSE_{soil} = C_{soil} \times GRAF \times SIR \times 10^{-9} \times EF$

1. $DOSE_{soil}$ = Dose from soil ingestion (mg/kg BW-day)
2. 10^{-9} = Conversion factor (μg to mg, mg to kg)
3. C_{soil} = Concentration of contaminant in soil (μg/kg)
4. GRAF = Gastrointestinal relative absorption fraction, chemical-specific (unitless)
5. SIR = Soil ingestion rate (mg/kg BW-day)
6. EF = Exposure frequency (unitless), (days/365 days)

a: Recommended default values for EQ 5.4.3.1.1:

1. C_{soil} = Calculated above in EQ 5.3.2 A
2. GRAF = See Table 5.2
3. SIR = See Table 5.14
4. EF = 350 d/year resident, 250 d/year worker

In this approach, it is assumed that the soil ingested contains a representative concentration of the contaminant(s) and the concentration is constant over the exposure period.

The term **GRAF**, or gastrointestinal relative absorption factor, is defined as the fraction of contaminant absorbed by the GI tract relative to the fraction of contaminant absorbed from the matrix (feed, water, other) used in the study(ies) that is the basis of either the cancer potency factor (CPF) or the Reference Exposure Level (REL). If no data are available to distinguish absorption in the toxicity study from absorption from the environmental matrix in question (i.e., soil), then GRAF = 1. The GRAF allows for adjustment for absorption from a soil matrix if it is known to be different from absorption across the GI tract in the study used to calculate the CPF or REL. In most instances, the GRAF will be 1.

Table 5.14 Recommended Soil Ingestion Rate (SIR) Estimates for Adults and Children (mg/kg-day)*

Age Groups (years)	Mean (mg/kg-day)	95th % (mg/kg-day)
3rd Trimester[a]	0.7	3
0<2	20	40
2<9	5	20
2<16	3	10
16<30	0.7	3
16 to 70	0.6	3
PICA adult	NR	-

[a] Assumed to be the mother's soil ingestion rate (adult age 16 <30)
* Soil includes outdoor settled dust
NR = No recommendation

5.4.3.1.2 Chronic Noncancer Dose for Soil Ingestion

The soil ingestion rate varies by age. A time-weighted average approach is used to combine soil intake rates of the age groupings (i.e., 0<2 yrs, 2<16 yrs, and 16-70 yrs) to determine the residential soil ingestion dose for chronic noncancer hazard assessment. This pathway is also assessed for exposure to offsite workers using the adult intake values for age 16-70 years in Table 5.14. Children at a MEIW daycare, if present, may also be assessed for exposure if the District deems it advisable. The contribution to the soil ingestion dose by each age group is determined in EQ 5.4.3.1.2:

A. Equation 5.4.3.1.2: $$DOSE_{soil} = C_{soil} \times GRAF \times SIR \times 10^{-9} \times ED/AT$$

1. $DOSE_{soil}$ = Dose from soil ingestion (mg/kg BW-day)
2. 10^{-9} = Conversion factor (μg to mg, mg to kg)
3. C_{soil} = Concentration of contaminant in soil (μg/kg)
4. GRAF = Gastrointestinal relative absorption fraction, unitless; chemical-specific
5. SIR = Soil ingestion rate (mg/kg BW-day)
6. ED = Exposure duration for a specified age group: 2 yrs for 0<2, 14 yrs for 2<16, 54 yrs for 16-70
7. AT = Averaging time for lifetime exposure – 70 yrs

a: Recommended default values for EQ 5.4.3.1.2:

1. C_{soil} = Calculated above in EQ 5.3.2 A
2. GRAF = See Table 5.2
3. SIR = See Table 5.14; use 16-70 age group SIR for workers

b: Recommended off-site worker default modifications to EQ 5.4.3.1.2:

1. A time-weighted average estimate of dose is not necessary and the ED and AT variates are left out of EQ 5.4.3.1.2 for oral soil dose to the worker.

c: Recommended nursing mother default modifications to EQ 5.4.3.1.2:

1. For mother's ingested soil dose to milk, use the SIR for age 16-30 years in Table 5.14.
2. The ED and AT variates in EQ 5.4.3.1.2 are left out for soil ingestion dose in the mother's milk pathway.

d: Assumptions for EQ 5.4.3.1.2:

1. For worker exposure, the annual average concentration should not be adjusted to account for overlap of worker and facility emission schedules. The pollutant will be deposited and accumulate in the soil in the absence or presence of the worker; therefore, the total deposition and soil concentration will be dependent on the annual average air concentration.

For residential exposure, the soil ingestion dose contribution for each age group is summed together to obtain the time-weighted average daily soil intake dose for chronic hazard assessment:

(SIR for age 0<2 yrs × C_{soil} × GRAF × 10^{-9} × 2 / 70) +

(SIR for age 2<16 yrs × C_{soil} × GRAF × 10^{-9} × 14 / 70) +

(SIR for age 16-70 yrs × C_{soil} × GRAF × 10^{-9} × 54 / 70) = Chronic $Dose_{soil}$

5.4.3.2 Exposure through Ingestion of Food

The exposure through food ingestion can be through ingestion of home-grown plant products (categorized as leafy, protected, exposed and root produce), home-raised animals (categorized as meat, cow's milk and eggs), angler-caught fish and mother's milk. When a specific food pathway is a dominant pathway (e.g., homegrown produce), and multiple pathways such as home raised meat, milk, and eggs categories all need to be assessed, the 95th percentile default consumption rate for the driving exposure pathway is used, while the mean consumption values for the remaining exposure pathways (i.e., food categories) are used. See Section 8.2.6 for a complete discussion of the methodology on how to implement the derived methodology.

5.4.3.2.1 Dose for Cancer Risk from Home-Grown Produce

Exposure through ingesting home-grown produce (DOSEp) is a function of the type of crop (i.e., exposed, leafy, protected, root), gastrointestinal relative absorption factor, bioavailability and the fraction of plant ingested that is homegrown. The calculation is done for each type of crop, then summed to get total dose for this pathway. The

exposure dose through ingestion of home-grown produce varies by age and is calculated for each age group (e.g., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 and 16-70 yrs). These age-specific groupings are needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8).

A. Equation 5.4.3.2.1: $$DOSEp = C_v \times IP \times GRAF \times L \times EF \times 10^{-6}$$

1. DOSEp = Exposure dose through ingestion of home-grown produce (mg/kg/d)
2. C_v = Concentration in specific type of crop, i.e., exposed, leafy, protected, root (μg/kg)
3. IP = Consumption of specific type of crop (g/kg BW*day)
4. GRAF = Gastrointestinal relative absorption factor (unitless)
5. L = Fraction of plant type consumed that is home-grown or locally grown (unitless)
6. EF = Exposure frequency (unitless, days/365 days)
7. 10^{-6} = Conversion factors (μg/kg to mg/g)

a: Recommended default values for Equation 5.4.3.2.1:

1. C_v = Calculated above in EQ 5.3.4.1 A
2. IP = See Table 5.15 (point estimates) and 5.16a-e (distributions)
3. GRAF = See Table 5.2
4. L = Site-specific survey is recommended. Otherwise, see Table 5.17 for Tier I default values
5. EF = 0.96 (350 d/365 d in a yr)

Once the dose for each type of crop that applies is calculated (See Section 5.3.4.1 for definition of crops types), the doses are summed to get the total dose for the home-grown produce pathway:

Total DOSEp = DOSEp (leafy) + DOSEp (root) + DOSEp (exposed) + DOSEp (protected)

The total home-grown produce dose will need to be calculated for each age group that applies.

5.4.3.2.2 Dose for cancer risk from home-raised meat, eggs, and cow's milk

Exposure through ingesting home-raised or farm animal products ($DOSE_{fa}$) is a function of the type of food (meat, eggs and cow's milk), gastrointestinal relative absorption factor, bioavailability and the fraction of food ingested that is home-raised. The only meat sources considered here are beef, pork and poultry. Unlike the home-grown produce pathway, the dose is calculated and presented separately for each type of home-raised food. The age-specific groupings to determine dose (3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 yrs or 16-70 yrs) is needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8).

A. Equation 5.4.3.2.2: $DOSE_{fa} = C_{fa} \times I_{fa} \times GRAF \times L \times EF \times 10^{-6}$

1. $DOSE_{fa}$ = Exposure dose through ingestion of home-raised animal product (mg/kg/d)
2. C_{fa} = Concentration in animal product, e.g., beef, pork, poultry, dairy, eggs (μg/kg)
3. I_{fa} = Consumption of animal product (g/kg BW-day)
4. GRAF = Gastrointestinal relative absorption factor (unitless)
5. L = Fraction of animal product consumed that is home-raised or locally produced (unitless)
6. EF = Exposure frequency (unitless, days/365 days)
7. 10^{-6} = Conversion factors (μg/kg to mg/g)

a: Recommended default values for EQ 5.4.3.2.2:

1. C_{fa} = Calculated above in EQ 5.3.4.2 A
2. I_{fa} = See Table 5.15 (point estimates) and Table 5.16a-e (distributions)
3. GRAF = See Table 5.2
4. L = Site-specific survey is recommended. Otherwise, see Table 5.17 for Tier I default values
5. EF = 0.96 (350 days / 365 days in a year)

5.4.3.2.3 Chronic Noncancer Dose for Ingestion of Food

For oral noncancer hazard assessment, a time-weighted average approach is used to combine food ingestion rates for the age groups (i.e., 0<2, 2<16 and 16-70 yrs) to estimate the chronic dose for residential exposure. The equation used to estimate dose through home-grown produce and home-raised meat/eggs/cow's milk is similar and is shown below in one equation. Similar to the cancer risk dose calculation, home-grown produce is presented as a total dose for all types of crops (See Section 5.4.3.2.1) and home-raised animal product dose is presented separately for each type of animal product that applies (See Section 5.4.3.2.2).

The contribution to the food intake dose is determined for each age group in EQ 5.4.3.2.3:

A. Equation 5.4.3.2.3: $\mathbf{DOSE_{food} = C_{food} \times I_{food} \times GRAF \times L \times 10^{-6} \times ED/AT}$

1. $DOSE_{food}$ = Exposure dose through ingestion of home-grown produce or home-raised animal product (mg/kg/d)
2. C_{food} = Concentration (μg/kg) in produce (e.g., exposed, leafy, protected, root) or animal product (e.g., beef, pork, poultry, dairy, eggs)
3. I_{food} = Consumption of produce or animal product (g/kg BW-day)
4. GRAF = Gastrointestinal relative absorption factor (unitless)
5. L = Fraction of produce or animal product consumed that is home-grown (unitless)
6. 10^{-6} = Conversion factors (μg/kg to mg/g)
7. ED = Exposure duration for a specified age group (2 yrs for 0<2, 14 yrs for 2<16, 54 yrs for 16-70
8. AT = Averaging time for lifetime exposure: 70 yrs

a: Recommended default values for EQ 5.4.3.2.3:

1. C_{food} = Calculated above in EQ 5.3.4.1 A (for home-grown produce) or EQ 5.3.4.2 A (for home-raised animal products)
2. I_{food} = Age-specific, see Table 5.15 for point estimates
3. GRAF = See Table 5.2
4. L = Site-specific survey is recommended. Otherwise, see Table 5.17 for Tier I default values

b: Recommended nursing mother default modifications to EQ 5.4.3.2.3:

1. For the mother's dose to milk through ingested food, use the food intake rates for age 16-30 years in Table 5.15 and 5.16d.
2. The ED and AT variates in EQ 5.4.3.2.3 are left out for ingested food dose in the mother's milk pathway.

Following calculation of the intake dose contributions for each age group, the intake rates for home-grown produce and the intake rates for home-raised animal products are summed separately to obtain the residential time-weighted average intake dose for chronic residential exposure to home-grown produce and to home-raised animal products:

(I_{food} for age 0<2 yrs × C_{food} × GRAF × L × 10^{-6} × 2 / 70) +

(I_{food} for age 2<16 yrs × C_{food} × GRAF × L × 10^{-6} × 14 / 70) +

(I_{food} for age 16-70 yrs × C_{food} × GRAF × L × 10^{-6} × 54 / 70) = Chronic $Dose_{food}$

Table 5.15 Recommended Average and High End Point Estimate Values for Home Produced Food Consumption (g/kg-day)

Food Category	Third Trimester		Ages 0<2		Ages 2<9	
Produce	Avg.	High End	Avg.	High End	Avg.	High End
Exposed	1.9	5.9	11.7	30.2	7.4	21.7
Leafy	0.9	3.2	3.8	10.8	2.5	7.9
Protected	1.7	5.8	5.9	17.5	4.7	13.3
Root	1.7	4.6	5.7	15.3	3.9	10.8
Meat						
Beef	2.0	4.8	3.9	11.3	3.5	8.6
Poultry	0.9	2.9	2.9	10.5	2.2	7.8
Pork	1.8	4.7	4.5	11.4	3.7	9.0
Milk	5.4	15.9	50.9	116	23.3	61.4
Eggs	1.6	4.2	6.1	15.0	3.9	9.4
	Ages 2>16		Ages 16<30		Ages 16-70	
Produce	Avg.	High End	Avg.	High End	Avg.	High End
Exposed	1.9	5.9	1.9	5.9	1.8	5.6
Leafy	0.9	3.2	0.9	3.2	1.1	3.4
Protected	1.7	5.8	1.7	5.8	1.6	5.2
Root	1.7	4.6	1.7	4.6	1.5	4.2
Meat						
Beef	2.0	4.8	2.0	4.8	1.7	4.4
Poultry	0.9	2.9	0.9	2.9	0.9	2.8
Pork	1.8	4.7	1.8	4.7	1.5	3.8
Milk	5.4	15.9	5.4	15.9	4.3	13.2
Eggs	1.6	4.2	1.6	4.2	1.3	3.4

[a] Food consumption values for 3rd trimester calculated by assuming that the fetus receives the same amount of contaminated food on a per kg BW basis as the mother (adult age 16 to less than 30).

Table 5.16a - e Parametric Models of Per Capita Food Consumption by Age Group for Stochastic Analysis

Table 5.16a Per Capita Food Consumption (g/kg-day) for Ages 0<2

Food Category	Distrib. Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape	Like-liest
Produce								
Exposed	Gamma	60			0.01	6.56	0.830	
Leafy	Gamma	167			0.01	3.30	1.161	
Protected	LogN	67	6.03	7.31				
Root	Gamma	83			0.06	4.44	1.28	
Meat								
Beef	LogN	16	1.97	1.73				
Poultry	LogN	58	4.5	4.08				
Pork	LogN	230	3.00	4.46				
Dairy	Max Ext.	169				27.82		33.79
Eggs	LogN	172	6.11	4.21				

Table 5.16b Per Capita Food Consumption (g/kg-day) for Ages 2<9

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape	Rate
Produce								
Exposed	Exponential	206						0.14
Leafy	LogN	127	2.64	3.89				
Protected	Weibull	68			0.02	4.76	1.063	
Root	LogN	60	3.95	3.85				
Meat								
Beef	LogN	35	3.55	2.79				
Poultry	LogN	17	3.71	2.67				
Pork	LogN	66	2.25	2.84				
Milk	LogN	12	23.4	20.78				
Eggs	LogN	38	3.93	3.00				

Table 5.16c Per Capita Food Consumption (g/kg-day) for Ages 2<16

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	Gamma	60			0.01	6.54	0.8325
Leafy	LogN	68	1.83	2.91			
Protected	Gamma	47			0.00	3.69	0.9729
Root	LogN	51	3.10	3.44			
Meat							
Beef	LogN	10	2.96	2.49			
Poultry	LogN	27	2.98	2.52			
Pork	LogN	48	1.84	2.79			
Milk	LogN	35	16.8	19.2			
Eggs	LogN	71	3.16	2.95			

Table 5.16d Per Capita Food Consumption (g/kg-day) for Ages 16-30[a]

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	Gamma	70			0.01	2.05	0.9220
Leafy	Weibull	191			0.00	0.88	0.8732
Protected	LogN	93	1.81	3.31			
Root	LogN	43	1.69	1.69			
Meat							
Beef	LogN	26	1.98	1.54			
Poultry	LogN	26	1.80	1.42			
Pork	LogN	242	1.01	1.74			
Milk	Gamma	22			0.02	5.66	0.9421
Eggs	LogN	29	1.55	1.36			

[a] These distributions are also recommended for the third trimester. Food consumption values for 3rd trimester are calculated by assuming that the fetus receives the same amount of contaminated food on a per kg BW basis as the mother (adult age 16<30).

Table 5.16e Per Capita Food Consumption (g/kg-day) for Ages 16-70

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	Gamma	148			0.01	2.07	0.8628
Leafy	Gamma	83			0.00	1.15	0.9713
Protected	Gamma	78			0.01	1.90	0.8325
Root	Gamma	14			0.00	1.28	1.166
Meat							
Beef	LogN	20	1.75	1.40			
Poultry	LogN	18	1.53	1.18			
Pork	LogN	190	0.97	1.59			
Milk	Gamma	20			0.00	4.50	0.9627
Eggs	LogN	30	1.3	1.01			

Table 5.17 Default Values for L in EQs 5.4.3.2.1., 5.4.3.2.2 and 5.4.3.2.3: Fraction of Food Intake that is Home-Produced

Food Type	Households that Garden[a]	Households that Farm[a]
Avg. Total Veg & Fruits	0.137	0.235
	Households that Garden/Hunt[b]	**Households that Farm[b]**
Beef	0.485	0.478
Pork	0.242	0.239
Poultry	0.156	0.151
Eggs	0.146	0.214
Total Dairy (Cow's milk)	0.207	0.254

[a] As a default for home-produced leafy, exposed, protected and root produce, OEHHA recommends 0.137 as the fraction of produce that is home-grown. The households that grow their own vegetables and fruits are the population of concern. In rural situations where the receptor is engaged in farming, OEHHA recommends 0.235 as the default value for fraction of leafy, exposed, protected and root produce that is home-grown.

[b] OEHHA recommends the fraction home-raised under "Households that raise animals/hunt" (for beef, pork, poultry (chicken), eggs and dairy (cow's milk), with the exception of rural household receptors engaged in farming. OEHHA recommends that the fractions listed under "Households that farm" be used for the rural household receptors.

5.4.3.3 Exposure through Ingestion of Water

Intake of drinking water varies by age on a ml per kg body weight per day basis resulting in differences in exposure dose by age. The age-specific groupings to determine dose are needed in order to properly use the age sensitivity factors for

cancer risk assessment (see Chapter 8) and to calculate a time-weighted average dose for chronic noncancer assessment.

5.4.3.3.1 Dose for Cancer Risk through Ingestion of Water

$DOSE_{water}$ is calculated for each age group (i.e., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 yrs and 16-70 yrs), then incorporated into EQ 8.2.5 in Chapter 8 to determine cancer risk through exposure in drinking water.

A. Equation 5.4.3.3.1: $$\mathbf{DOSE_{water} = C_w \times WIR \times ABS_{wa} \times Fdw \times EF \times 10^{-6}}$$

1. $DOSE_{water}$ = Exposure dose through ingestion of water (mg/kg BW/d)
2. C_w = Water concentration (μg/L)
3. WIR = Water ingestion rate (ml/kg BW-day)
4. ABS_{wa} = Gastrointestinal relative absorption factor (unitless)
5. Fdw = Fraction of drinking water from contaminated source
6. EF = Exposure frequency (unitless, days/365 days)
7. 10^{-6} = Conversion factors (mg/μg)(L/ml)

a: Recommended default values for EQ 5.4.3.3.1:

1. C_w = Calculated above 5.3.3 A
2. WIR = See 5.18 (point estimates) and Table 5.19 (distributions)
3. ABS_{wa} = Default set to 1
4. Fdw = Default set to 1, although a site-specific survey is recommended for this variate
5. EF = 0.96 (350 days/365 days in a year)

5.4.3.3.2 Chronic Noncancer Dose through Ingestion of Water

Because water intake varies by age group, a time-weighted average intake approach is used to determine the daily water ingestion dose for chronic residential exposure. The contribution to the water ingestion dose is determined for each age group (i.e., 0<2, 2<16 and 16-70 yrs) in EQ 5.4.3.3.2.

A. Equation 5.4.3.3.2:

$$\mathbf{DOSE_{water} = C_w \times WIR \times ABS_{wa} \times Fdw \times 10^{-6} \times ED/AT}$$

1. $DOSE_{water}$ = Exposure dose through ingestion of water (mg/kg BW/d)
2. C_w = Water concentration (μg/L)
3. WIR = Water ingestion rate (ml/kg BW-day)
4. ABS_{wa} = Gastrointestinal absorption factor
5. Fdw = Fraction of drinking water from contaminated source (site-specific)
6. 10^{-6} = Conversion factors (mg/μg)(L/ml)
7. ED = Exposure duration for a specified age group: 2 yrs for 0<2, 14 yrs for 2<16, 54 yrs for 16-70
8. AT = Averaging time for residential exposure: 70 yrs

a: Recommended default values for EQ 5.4.3.3.2:

1. C_w = Calculated above in 5.3.3 A
2. WIR = See 5.18 (point estimates)
3. ABS_{wa} = Default set to 1
4. Fdw = Default set to 1, although a site-specific survey is recommended for this variate

b: Recommended nursing mother default modifications to EQ 5.4.3.3.2:

1. For the dose to mother's milk through water ingestion, use the WIR for age 16-30 years in Table 5.18.
2. The ED and AT variates in EQ 5.4.3.3.2 are left out for ingested water dose in the mother's milk pathway.

The water intake dose contribution for each age group is summed together to obtain the time-weighted average daily residential water ingestion dose:

(WIR for age 0<2 yrs × C_w × ABS_{wa} × Fdw × 10^{-6} × 2 / 70) +

(WIR for age 2<16 yrs × C_w × ABS_{wa} × Fdw × 10^{-6} × 14 / 70) +

(WIR for age 16-70 yrs × C_w × ABS_{wa} × Fdw × 10^{-6} × 54 / 70) = Chronic $Dose_{water}$

Table 5.18 Recommended Point Estimate Tap Water Intake Rates (ml/kg-day)

Point Estimates				
Using Mean Values	**For the Age Period**	**9-year scenario**	**30-year scenario**	**70-year scenario**
	3rd trimester	18	18	18
	0<2 years	113	113	113
	2<9 years	26	-	-
	2<16 years	-	24	24
	16-30 years	-	18	-
	16-70 years	-	-	18
Using 95th-percentile values	**For the Age Period**	**9-year scenario**	**30-year scenario**	**70-year scenario**
	3rd trimester	47	47	47
	0<2 years	196	196	196
	2<9 years	66	-	-
	2<16 years	-	61	61
	16-30 years	-	47	-
	16-70 years	-	-	45

Table 5.19 Recommended Distributions of Tap Water Intake Rates (ml/kg-day) for Stochastic Risk Assessment

	9-year scenario	**30-year scenario**	**70-year scenario**
0<2 years	**Max Extreme** Likeliest = 93 Scale = 35	**Max Extreme** Likeliest = 93 Scale = 35	**Max Extreme** Likeliest = 93 Scale = 35
2<9 years	**Weibull** Location = 0.02 Scale = 29 Shape = 1.3		
2<16 years		**Gamma** Location = 0.19 Scale = 15.0 Shape = 1.6	**Gamma** Location = 0.19 Scale = 15.0 Shape = 1.6
16-30 years		**Gamma** location=0.49 scale=13.6 shape=1.26	
16-70 years			**Beta** min=0.17 max=178 alpha=1.5 beta= 12.9

5.4.3.4 Exposure through Ingestion of Angler-caught Fish

Exposure through ingestion of angler-caught fish ($DOSE_{fish}$) is a function of the fraction of fish ingested that is caught in the exposed water body, which differs for each age grouping, and the gastrointestinal absorption factor. Ingestion of angler-caught fish on a mg/kg body weight per day basis varies by age resulting in differences in exposure dose by age. The age-specific groupings to determine dose is needed primarily to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8) and to calculate a time-weighted average dose for chronic noncancer assessment.

5.4.3.4.1 Cancer Risk Dose via Ingestion of Angler-Caught Fish

$DOSE_{fish}$ is calculated for each age group separately (i.e., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 yrs and 16-70 yrs), then incorporated into EQ 8.2.5 in Chapter 8 to determine cancer risk through exposure to angler-caught fish.

A. Equation 5.4.3.4.1: $$DOSE_{fish} = C_t \times I_{fish} \times Gf \times L \times EF \times 10^{-6}$$

1. $DOSE_{fish}$ = Dose via ingestion of angler-caught fish (mg/kg BW-day)
2. C_t = Concentration in fish muscle tissue (μg/kg)
3. I_{fish} = Angler-caught fish ingestion rate (g/kg BW per day)
4. Gf = Gastrointestinal absorption factor (unitless)
5. L = Fraction of fish caught at exposed site (unitless)
6. EF = Exposure frequency (days/365 days)
7. 10^{-6} = Conversion factor (mg/μg, kg/g)

a: Recommended default values for Equation 5.4.3.4.1:

1. C_t = Calculated above in Equation 5.3.4.7
2. I_{fish} = See Table 5.20 (point estimates) and Table 5.21 (distributions)
3. Gf = Default set to 1
4. L = Default set to 1 for fraction of fish caught locally, although a site-specific survey is recommended for this variate
5. EF = 0.96 (350 days/365 days in a yr)

5.4.3.4.2 Chronic Noncancer Dose via Ingestion of Angler-Caught Fish

Angler-caught fish consumption varies by age group. A time-weighted average intake for residential consumption over 70 years is used to determine dose for average and high-end exposure. The contribution to the angler-caught fish consumption dose is determined for each age group in EQ 5.4.3.4.2:

A. Equation 5.4.3.4.2: $\mathbf{DOSE_{fish} = C_t \times I_{fish} \times Gf \times L \times 10^{-6} \times ED/AT}$

1. $DOSE_{fish}$ = Dose via ingestion of angler-caught fish (mg/kg BW-day)
2. C_t = Concentration in fish muscle tissue (μg/kg)
3. I_{fish} = Angler-caught fish ingestion rate (g/kg BW per day)
4. Gf = Gastrointestinal absorption factor (unitless)
5. L = Fraction of fish caught at exposed site (unitless)
6. 10^{-6} = Conversion factor (mg/μg, kg/g)
7. ED = Exposure duration for a specified age group: 2 yrs for 0<2, 14 yrs for 2<16 and 54 yrs for 16-70
8. AT = Averaging time for chronic exposure – 70 yrs

a: Recommended default values for Equation 5.4.3.4.2:

1. C_t = Calculated above in Equation 5.3.4.7
2. I_{fish} = See Table 5.20 (point estimates)
3. Gf = Default set to 1
4. L = Default set to 1 for fraction of fish caught locally, although a site-specific survey is recommended for this variate

b: Recommended nursing mother default modifications to EQ 5.4.3.4.2:

1. For the dose to mother's milk through fish consumption, use the I_{fish} for age 16-30 years in Table 5.20.
2. The ED and AT variates in EQ 5.4.3.4.2 are left out for the dose via fish consumption in the mother's milk pathway.

Following calculation of the angler-caught fish consumption dose contribution for each age group, 0<2 yr, 2<16 yr and 16-70 yr fish consumption doses are summed together to obtain the residential chronic dose:

(I_{fish} for age 0<2 yrs × C_t × Gf × L × 10^{-6} × 2 / 70) +

(I_{fish} for age 2<16 yrs × C_t × Gf × L × 10^{-6} × 14 / 70) +

(I_{fish} for age 16-70 yrs × C_t × Gf × L × 10^{-6} × 54 / 70) = Chronic $Dose_{fish}$

Table 5.20 Point Estimate Values for Angler-Caught Fish Consumption (g/kg-day) by Age Group

	Third Trimester	0 <2 Years	2<9 Years	2<16 Years	16<30 Years	16-70 Years
Mean	0.38	0.18	0.36	0.36	0.38	0.36
95th Percentile	1.22	0.58	1.16	1.16	1.22	1.16

Table 5.21 Empirical Distribution for Angler-Caught Fish Consumption (g/kg-day)

Mean	Percentile									
	10th	20th	30th	40th	50th	60th	70th	80th	90th	95th
Third trimester, 2<9, 2<16, 16<30 and 16-70-year age groups										
0.36	0.06	0.09	0.12	0.16	0.21	0.27	0.36	0.50	0.79	1.16
0<2-year age group										
0.18	0.03	0.05	0.06	0.08	0.11	0.14	0.18	0.25	0.40	0.58

5.4.3.5 Mother's Milk

Exposure through mother's milk ingestion (Dose-Im) is a function of the average concentration of the substance in mother's milk and the amount of mother's milk ingested. The minimum pathways that the nursing mother is exposed to include inhalation, soil ingestion, and dermal, since the chemicals evaluated by the mother's milk pathway are multipathway chemicals. Other pathways may be appropriate depending on site conditions (e.g., the presence of vegetable gardens or home grown chickens). The compounds currently considered for the mother's milk pathway are:

1. Dioxins and Furans (PCDDS and PCDFs)
2. Polychlorinated biphenyls (PCBs)
3. Polycyclic Aromatic Hydrocarbons (PAHs), including creosotes
4. Lead

These compound classes represent the chemicals of greatest concern for the mother's milk pathway under the Hot Spots program, and for which data are available to estimate transfer coefficients. It is expected that additional transfer coefficients will be developed for other multipathway chemicals in the Hot Spots Program as data becomes available and is reviewed. The nursing mother in the mother's milk pathway is not herself subject to the mother's milk pathway. The summed average daily dose (mg/kg BW-day) from all pathways is calculated for the nursing mother using the equations that follow.

5.4.3.5.1 Cancer Risk Dose to Infant via Mother's Milk

A. Equation 5.4.3.5.1: $$\textbf{Dose-Im} = C_m \times BMI_{bw} \times EF \times 10^{-3}$$

1. Dose-Im = Dose to infant through ingestion of mother's milk (mg/kg BW per day)
2. C_m = Concentration of contaminant in mother's milk (mg/kg milk)
3. BMI_{bw} = Daily breast-milk ingestion rate (g/kg BW-day)
4. EF = Frequency of exposure (days / 365 days)
5. 10^{-3} = Conversion factor (kg to g)

a: Recommended default values for EQ 5.4.3.5.1:

1. C_m = See EQ 5.3.4.8
2. BMI_{bw} = See Table 5.22 for point estimates. For distribution (parametric model) for Tier 3 stochastic risk assessments see Table 5.23.
3. EF = 1 (all 365 days of the first year of birth)

b: Assumptions for EQ 5.4.3.5.1:

1. For the MEIR, mother is exposed from birth up to 25 years of age when the infant is born. The exposed infant is then fully breastfed only during the first year of life.
2. For cancer risk assessment, exposure of breast-feeding infants to contaminants in breast milk applies only to the first year of the 0<2 yr age group for calculation of risk to this group, which then can be summed with the risk calculated for the other age groups (See Chapter 8).

5.4.3.5.2 Chronic Noncancer Dose to Infant via Mother's Milk

For oral noncancer hazard assessment, exposure of the infant through mother's milk ingestion occurs during the first year of life. After one year of age, the mother's milk pathway is not a factor for noncancer assessment.

A. Equation 5.4.3.5.2: $\text{Dose-Im} = C_m \times BMI_{bw} \times 10^{-3}$

1. Dose-Im = Dose to infant through ingestion of mother's milk (mg/kg BW/d)
2. C_m = Concentration of contaminant in mother's milk (mg/kg milk)
3. BMI_{bw} = Daily breast-milk ingestion rate (g/kg BW-day)
4. 10^{-3} = Conversion factor (kg to g)

a: Recommended default values for EQ 5.4.3.5.2:

1. C_m = See EQ 5.3.4.8
2. BMI_{bw} = See Table 5.22 for point estimates

Table 5.22 Default Point Estimates for Breast Milk Intake (BMI_{bw}) for Breastfed Infants

Infant Group	Intake (g/kg-day)
Fully breastfed over the first year (i.e., fed in accordance with AAP recommendations)	
Mean	101
95th percentile	139

Table 5.23 Recommended Distribution of Breast Milk Intake Rates Among Breastfed Infants for Stochastic Assessment* (Averaged Over an Individual's First Year of Life)

	Mean (SD)	Percentile							
		5	10	25	50	75	90	95	99
Intake (g/kg-day)	101 (23)	62	71	85	101	116	130	139	154

* For stochastic analysis, the mother's milk data are normally distributed.

5.5 References

ARB 2007. *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5),* and the *Emission Inventory Criteria and Guidelines Report* (EICG Report).

ARB, 2012. Consolidated Table of OEHHA / ARB Approved Risk Assessment Health Values. Air Resouces Board, California Environmental Protection Agency. Online at: http://www.arb.ca.gov/toxics/healthval/healthval.htm

Hausch C, Leo AJ, Medchem Project Issue No. 26, Claremont CA: Pomona College (1985)

Lyman WJ, Reehl WF, Rosenblatt DH et al., 1990. Handbook of Chemical Property Estimation Methods. American Chemical Society, Wash. DC

OEHHA, 2000. *Air Toxics Hot Spots Program Risk Asessment Guidelines: Part IV Technical Support Document for Exposure Assessment and Stochastic Analysis.* September 2000.

OEHHA, 2012a. *Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis*. Available online at http://www.oehha.ca.gov

OEHHA, 2012b. Hot Spots Unit Risk and Cancer Potency Values. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Online at: http://oehha.ca.gov/air/hot_spots/pdf/CPFs042909.pdf

Staples CA, Peterson DR, Parkerton TF, Adams WJ. 1997. The environmental fate of phthalate esters: A literature review. Chemosphere 35(4):667-749.

U.S. EPA. (2009). *Metabolically derived human ventilation rates: A revised approach based upon oxygen consumption rates*. U.S. Environmental Protection Agency, National Center for Environmental Assessment, Washington, DC; EPA/600/R-06/129F

6 - Dose-Response Assessment for Noncarcinogenic Endpoints

6.1 Derivation of Toxicity Criteria for Noncancer Health Effects

Dose-response assessment describes the quantitative relationship between the amount of exposure to a substance (the dose) and the incidence or occurrence of an adverse health impact (the response). Dose-response information for noncancer health effects is used to determine Reference Exposure Levels (RELs). Inhalation RELs are air concentrations or doses at or below which adverse noncancer health effects are not expected even in sensitive members of the general population under specified exposure scenarios. The acute RELs are for infrequent 1 hour exposures that occur no more than once every two weeks in a given year, although this time frame of exposure does not necessarily apply to chemicals that can bioaccumulate (e.g., dioxins and furans, PCBs, and various metals). The chronic RELs are for 24 hour per day exposures for at least a significant fraction of a lifetime, defined as about 8 years (≥12 percent of a 70-year lifespan). The 8-hour RELs are for repeated 8-hour exposures for a significant fraction of a lifetime such as the exposures that offsite workers might typically receive. Eight-hour RELs are only available for 10 chemicals at present, but OEHHA will develop 8-hour RELs as we re-evaluate our existing RELs to ensure they are protective of children's health, and as we develop RELs for new chemicals. There are oral chronic RELs for some chemicals in the Hot Spots program that are semivolatile or nonvolatile and thus subject to deposition and oral ingestion or dermal exposure. The methodology for developing RELs is similar to that used by U.S. EPA in developing the inhalation Reference Concentrations (RfCs) and oral Reference Doses (RfDs).

Review and revision of RELs to take into account new information and sensitive subpopulations including infants and children is an ongoing process. All draft RELs for individual chemicals revised under the current noncancer methodology will undergo public comment and peer review, as mandated by the Hot Spots Act. .

The first step in determining an acute, 8-hour, or chronic REL is to determine a point of departure. The point of departure is preferably determined by the benchmark concentration procedure applied to human or animal studies, but if this method of calculation cannot be used with a particular data set, a no observed adverse effect level (NOAEL) or lowest observed adverse effect level (LOAEL) may be used as the point of departure. The benchmark concentration method (also referred to as the benchmark dose method for oral exposures) is a preferred method to estimate a point of departure because it takes all of the available dose-response data into account to statistically estimate, typically, a 5 percent response rate.

Dosimetric or toxicokinetic adjustments are often made to the point of departure to adjust for differences in dosimetry or kinetics across species or among humans. Time adjustments are generally applied to adjust experimental exposure to the exposure of

interest for the REL (e.g., 1 hour for acute, continuous for chronic). A modified Haber's equation is used where needed to adjust studies with different exposure times to the one-hour period needed for acute RELs. A simple Haber's law (C x T) adjustment for exposure period duration is used for most 8-hour and chronic RELs.

The time and dosimetry adjusted point of departure is divided by uncertainty factors that reflect the limitations in the current toxicology of the chemical. For example, an interspecies uncertainty factor is applied to account for the differences between humans and animals when an animal study is used. An intraspecies uncertainty factor is usually included to account for differences in susceptibility among the human population. In addition, where benchmark dose modeling is not suitable and a NOAEL is not available, a LOAEL to NOAEL uncertainty factor may be applied when the LOAEL serves as the point of departure. If a chronic study is not available to serve as a basis for a chronic REL, then a subchronic uncertainty factor (for chronic and 8-hour RELs only) may also be applied. Finally, if there are data deficiencies, for example, lack of a developmental toxicity study for a chemical, then a database deficiency factor may be applied. The individual uncertainty factors, which range from 2 to 10 depending on the limitations in the data, are multiplied together for a total uncertainty factor. The point of departure is then divided by the total UF to obtain the REL.

The most sensitive toxicological end point is selected as the basis for the REL when there are multiple adverse health effects. The selection of the most sensitive endpoint as the basis for a REL helps ensure that the REL is protective for all health effects. The use of uncertainty factors helps ensure that the REL is protective for nearly all individuals, including sensitive subpopulations, within the limitations of current scientific knowledge. For detailed information on the methodology and derivations for RELs, including guidance on selection of uncertainty factors, see the Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document for the Derivation of Noncancer Reference Exposure Levels (OEHHA, 2008).

It should be emphasized that exceeding the acute or chronic REL does not necessarily indicate that an adverse health impact will occur. The REL is not the threshold where population health effects would first be seen. However, levels of exposure above the REL have an increasing but undefined probability of resulting in an adverse health impact, particularly in sensitive individuals (e.g., depending on the toxicant, the very young, the elderly, pregnant women, and those with acute or chronic illnesses). The significance of exceeding the REL is dependent on the seriousness of the health endpoint, the strength and interpretation of the health studies, the magnitude of combined safety factors, and other considerations. In addition, there is a possibility that a REL may not be protective of certain small, unusually sensitive human subpopulations. Such subpopulations can be difficult to identify and study because of their small numbers, lack of knowledge about toxic mechanisms, and other factors. It may be useful to consult OEHHA staff when a REL is exceeded (hazard quotient or hazard index is greater than 1.0). Chapter 8 discusses the methods used for determining potential noncancer health impacts and Appendix I presents example calculations used to determine a hazard quotient (HQ) and hazard indices (HI).

Tables 6.1 through 6.3 list the currently adopted acute, 8-hour, and chronic inhalation RELs. Some substances that pose a long-term inhalation hazard may also present a chronic hazard via non-inhalation (oral, dermal) routes of exposure. The oral RELs for these substances are presented in Table 6.3. Appendix L provides a consolidated listing of all the acute, 8-hour, and chronic RELs with the respective target organs that are approved for use by OEHHA and ARB for the Hot Spots Program. Periodically, new or updated RELs are adopted by OEHHA and these guidelines will be updated to reflect those changes. See OEHHA's web site at *www.oehha.ca.gov* (look under "Air", then select "Hot Spots Guidelines") to determine if any new or updated RELs have been adopted since the last guideline update.

6.2 Acute Reference Exposure Levels

OEHHA developed acute RELs for assessing potential noncancer health impacts for short-term, one-hour peak exposures to facility emissions (OEHHA, 2008; http://www.oehha.ca.gov/air/allrels.html). By definition, an acute REL is an exposure that is not likely to cause adverse health effects in a human population, including sensitive subgroups, exposed to that concentration (in units of micrograms per cubic meter or $\mu g/m^3$) for the specified exposure duration on an intermittent basis.

The target organ systems and the acute RELs for each substance are presented in Table 6.1. Many acute RELs are based on mild adverse effects, such as mild irritation of the eyes, nose, or throat, or may result in other mild adverse physiological changes. For most individuals, it is expected that the mild irritation and other adverse physiological changes will not persist after exposure ceases. For RELs that have been recently developed or revised, the notation "sensory irritation" has been added in parenthesis in Table 6.1 for those chemicals that have an acute REL based on sensory irritation of the respiratory system (i.e., nose , throat) and/or eyes.

Other acute RELs are based on reproductive/developmental endpoints, such as teratogenicity or fetotoxicity, which are considered severe adverse effects. The inhalation pathway is the only pathway to assess for acute exposure. Other non-inhalation pathways of exposure are evaluated for worker and residential scenarios where the exposures are chronic or repeated daily in nature. The oral RELs are used to evaluate the non-inhalation pathways of exposure. Noninhalation (oral) RELs are discussed in Section 6.5. Chapter 8 discusses the methods used for determining noncancer acute health impacts. Appendix I presents an example calculation used to determine an HQ and HI.

Table 6.1 Acute Inhalation Reference Exposure Levels (RELs) and Acute Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Acute Inhalation REL ($\mu g/m^3$)	Acute Hazard Index Target Organ Systems(s)
Acetaldehyde	75-07-0	$4.7 \times 10^{+2}$	Eyes; Respiratory System (sensory irritation)
Acrolein	107-02-8	$2.5 \times 10^{+0}$	Eyes; Respiratory System (sensory irritation)
Acrylic Acid	79-10-7	$6.0 \times 10^{+3}$	Eyes; Respiratory System
Ammonia	7664-41-7	$3.2 \times 10^{+3}$	Eyes; Respiratory System
Arsenic and Inorganic Arsenic Compounds (including arsine)	7440-38-2	2.0×10^{-1}	Development; Cardiovascular System; Nervous System
Benzene	71-43-2	$2.7 \times 10^{+1}$	Reproductive/Developmental; Immune System; Hematologic System
Benzyl Chloride	100-44-7	$2.4 \times 10^{+2}$	Eyes; Respiratory System
1,3-Butadiene	106-99-0	$6.6 \times 10^{+2}$	Development
Caprolactam	105-60-2	$5.0 \times 10^{+1}$	Eyes (sensory irritation)
Carbon Disulfide	75-15-0	$6.2 \times 10^{+3}$	Nervous System; Reproductive/Developmental
Carbon Monoxide [a]	630-08-0	$2.3 \times 10^{+4}$	Cardiovascular System
Carbon Tetrachloride	56-23-5	$1.9 \times 10^{+3}$	Alimentary System (Liver); Nervous System Reproductive/Developmental
Chlorine	7782-50-5	$2.1 \times 10^{+2}$	Eyes; Respiratory System
Chloroform	67-66-3	$1.5 \times 10^{+2}$	Nervous System; Respiratory System; Reproductive/Developmental
Chloropicrin	76-06-2	$2.9 \times 10^{+1}$	Eyes; Respiratory System
Copper and Compounds	7440-50-8	$1.0 \times 10^{+2}$	Respiratory System
1,4-Dioxane	123-91-1	$3.0 \times 10^{+3}$	Eyes; Respiratory System
Epichlorohydrin	106-89-8	$1.3 \times 10^{+3}$	Eyes; Respiratory System
Ethylene Glycol Monobutyl Ether	111-76-2	$1.4 \times 10^{+4}$	Eyes; Respiratory System
Ethylene Glycol Monoethyl Ether	110-80-5	$3.7 \times 10^{+2}$	Reproductive/Developmental
Ethylene Glycol Monoethyl Ether Acetate	111-15-9	$1.4 \times 10^{+2}$	Nervous System; Reproductive/Developmental
Ethylene Glycol Monomethyl Ether	109-86-4	$9.3 \times 10^{+1}$	Reproductive/Developmental
Formaldehyde	50-00-0	$5.5 \times 10^{+1}$	Eyes (sensory irritation)
Hydrogen Chloride	7647-01-0	$2.1 \times 10^{+3}$	Eyes; Respiratory System
Hydrogen Cyanide	74-90-8	$3.4 \times 10^{+2}$	Nervous System
Hydrogen Fluoride	7664-39-3	$2.4 \times 10^{+2}$	Eyes; Respiratory System
Hydrogen Selenide	7783-07-5	$5.0 \times 10^{+0}$	Eyes; Respiratory System
Hydrogen Sulfide [a]	7783-06-4	$4.2 \times 10^{+1}$	Nervous System
Isopropanol	67-63-0	$3.2 \times 10^{+3}$	Eyes; Respiratory System
Mercury and Inorganic Mercury Compounds	7439-97-6	6.0×10^{-1}	Nervous System; Development
Methanol	67-56-1	$2.8 \times 10^{+4}$	Nervous System
Methyl Bromide	74-83-9	$3.9 \times 10^{+3}$	Nervous System; Respiratory System; Reproductive/Developmental

Substance	Chemical Abstract Service Number (CAS)	Acute Inhalation REL ($\mu g/m^3$)	Acute Hazard Index Target Organ Systems(s)
Methyl Chloroform	71-55-6	$6.8 \times 10^{+4}$	Nervous System
Methyl Ethyl Ketone	78-93-3	$1.3 \times 10^{+4}$	Eyes; Respiratory System
Methylene Chloride	75-09-2	$1.4 \times 10^{+4}$	Nervous System; Cardiovascular System
Nickel and Nickel Compounds	7440-02-0	2.0×10^{-1}	Immune System
Nitric Acid	7697-37-2	$8.6 \times 10^{+1}$	Respiratory System
Nitrogen Dioxide [a]	10102-44-0	$4.7 \times 10^{+2}$	Respiratory System
Ozone [a]	10028-15-6	$1.8 \times 10^{+2}$	Eyes; Respiratory System
Perchloroethylene (Tetrachloroethylene)	127-18-4	$2.0 \times 10^{+4}$	Eyes; Nervous System; Respiratory System
Phenol	108-95-2	$5.8 \times 10^{+3}$	Eyes; Respiratory System
Phosgene	75-44-5	$4.0 \times 10^{+0}$	Respiratory System
Propylene Oxide	75-56-9	$3.1 \times 10^{+3}$	Eyes; Respiratory System; Reproductive/Developmental
Sodium Hydroxide	1310-73-2	$8.0 \times 10^{+0}$	Eyes; Skin; Respiratory System
Styrene	100-42-5	$2.1 \times 10^{+4}$	Eyes; Respiratory System; Reproductive/Developmental
Sulfates [a]	N/A	$1.2 \times 10^{+2}$	Respiratory System
Sulfur Dioxide [a]	7446-09-5	$6.6 \times 10^{+2}$	Respiratory System
Sulfuric Acid and Oleum	7664-93-9 8014-95-7	$1.2 \times 10^{+2}$	Respiratory System
Tetrachloroethylene (Perchloroethylene)	127-18-4	$2.0 \times 10^{+4}$	Eyes; Nervous System; Respiratory System
Toluene	108-88-3	$3.7 \times 10^{+4}$	Nervous System; Respiratory System; Eyes; Reproductive/Developmental
Triethylamine	121-44-8	$2.8 \times 10^{+3}$	Nervous System; Eyes
Vanadium Pentoxide	1314-62-1	$3.0 \times 10^{+1}$	Eyes; Respiratory System
Vinyl Chloride	75-01-4	$1.8 \times 10^{+5}$	Nervous System; Eyes; Respiratory System
Xylenes (m,o,p-isomers)	1330-20-7	$2.2 \times 10^{+4}$	Eyes; Respiratory System; Nervous System

[a] California Ambient Air Quality Standard

6.3 8-hour Reference Exposure Levels

OEHHA has developed 8-hour RELs for assessing potential noncancer health impacts for exposures to the general public that occur on a recurrent basis, but only during a portion of each day (OEHHA, 2008; http://www.oehha.ca.gov/air/allrels.html). Eight-hour RELs are compared to air concentrations that represent an average (daily) 8-hour exposure. They were designed to address off-site worker exposure at the MEIW, but may also be used at the Districts' discretion to characterize 8-hour residential noncancer exposures, particularly for non-continuous facility operations where exposure is based on air concentrations during facility operation (i.e., the zero emission hours are not included) rather than averaged over 24-hours/day, 7 days/week as assessed for chronic exposure. The 8-hour RELs can also be used to assess exposure of students and teachers while at school (OEHHA, 2008). These RELs were developed because of concerns that applying the chronic REL in some scenarios was

overly conservative. By definition, an 8-hour REL is an exposure that is not likely to cause adverse health effects in a human population, including sensitive subgroups, exposed to that concentration (in units of micrograms per cubic meter or $\mu g/m^3$) for an 8-hour exposure duration on a regular (including daily) basis.

The RELs, target organ systems, and the averaging time for substances that can present a potential hazard from inhalation for 8 hours on a daily basis are presented in Table 6.2. Chapter 8 discusses the methods used for determining noncancer 8-hour health impacts. Appendix I presents an example calculation used to determine an HQ and HI.

Any substances in Table 6.2 with Development or Reproductive System as a target organ system are represented in HARP and in the Appendix L REL tables under the single endpoint "Reproductive/Development".

Table 6.2 Eight-Hour Inhalation Reference Exposure Levels (RELs) and 8-Hour Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Acetaldehyde	75-07-0	$3.0 \times 10^{+2}$	Respiratory System
Acrolein	107-02-8	7.0×10^{-1}	Respiratory System
Arsenic & Inorganic Arsenic Compounds	7440-38-2	1.5×10^{-2}	Cardiovascular System; Development; Nervous System; Respiratory System; Skin
Benzene	71-43-2	$3.0 \times 10^{+0}$	Hematologic System
1,3-Butadiene	106-99-0	$9.0 \times 10^{+0}$	Reproductive System
Caprolactam	105-60-2	$7.0 \times 10^{+0}$	Respiratory System
Formaldehyde	50-0-0	$9.0 \times 10^{+0}$	Respiratory System
Manganese & Manganese Compounds	7439-96-5	1.7×10^{-1}	Nervous System
Mercury & Inorganic Mercury Compounds	7439--97-6	6.0×10^{-2}	Nervous System; Development; Kidney
Nickel & Nickel Compounds	7440-02-0	6.0×10^{-2}	Respiratory System; Immune System

6.4 Chronic Reference Exposure Levels

OEHHA has developed chronic RELs for assessing noncancer health impacts from long-term exposure. (OEHHA, 2008; see also http://www.oehha.ca.gov/air/allrels.html) A chronic REL is a concentration level (expressed in units of micrograms per cubic meter ($\mu g/m^3$) for inhalation exposure and in a dose expressed in units of milligrams per kilogram-day (mg/kg-day) for oral exposures) at or below which no adverse health effects are anticipated following long-term exposure. Long-term exposure for these purposes has been defined by U.S. EPA as at least 12% of a lifetime, or about eight years for humans. Table 6.3 lists the chronic noncancer RELs that should be used in

the assessment of chronic health effects from inhalation exposure. Appendix L provides a consolidated listing of all the acute, 8-hour and chronic RELs and target organs that are approved for use by OEHHA and ARB for the Hot Spots Program. Periodically, new or updated RELs are adopted by OEHHA. See OEHHA's web site http://www.oehha.ca.gov/air/allrels.html to determine if any new or updated RELs have been adopted since the last guideline update.

The organ system(s) associated with each chronic REL are also presented in Table 6.3. Any substances in Table 6.3 with Development or Reproductive System as a target organ system are represented in HARP and in the Appendix L REL tables under the single endpoint "Reproductive/Development". Chapter 8 discusses the methods used for determining potential noncancer health impacts and Appendix I presents example calculations used to determine a HQ and HI.

Table 6.3 Chronic Inhalation Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Acetaldehyde [a]	75-07-0	$1.4 \times 10^{+2}$	Respiratory System
Acrolein	107-02-8	3.5×10^{-1}	Respiratory System
Acrylonitrile	107-13-1	$5.0 \times 10^{+0}$	Respiratory System
Ammonia	7664-41-7	$2.0 \times 10^{+2}$	Respiratory System
Arsenic & Inorganic Arsenic Compounds	7440-38-2	1.5×10^{-2}	Cardiovascular System; Development; Nervous System; Respiratory System; Skin
Benzene	71-43-2	$3.0 \times 10^{+0}$	Hematologic System
Beryllium and Beryllium Compounds	7440-41-7	7.0×10^{-3}	Immune System; Respiratory System
1,3-Butadiene	106-99-0	$2.0 \times 10^{+0}$	Reproductive System
Cadmium and Cadmium Compounds	7440-43-9	2.0×10^{-2}	Kidney; Respiratory System
Caprolactam	105-60-2	$2.2 \times 10^{+0}$	Respiratory System
Carbon Disulfide	75-15-0	$8.0 \times 10^{+2}$	Nervous System; Reproductive System
Carbon Tetrachloride	56-23-5	$4.0 \times 10^{+1}$	Alimentary System (Liver); Development; Nervous System
Chlorine	7782-50-5	2.0×10^{-1}	Respiratory System
Chlorine Dioxide	10049-04-4	6.0×10^{-1}	Respiratory System
Chlorinated Dibenzo-*p*-dioxins [b]			
2,3,7,8-Tetrachlorodibenzo-*p*-dioxin [b]	1746-01-6	4.0×10^{-5}	Alimentary System (Liver); Development; Endocrine System; Hematologic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzo-*p*-dioxin [b]	40321-76-4	4.0×10^{-5}	
1,2,3,4,7,8-Hexachlorodibenzo-*p*-dioxin [b]	39227-28-6	4.0×10^{-4}	
1,2,3,6,7,8-Hexachlorodibenzo-*p*-dioxin [b]	57653-85-7	4.0×10^{-4}	
1,2,3,7,8,9-Hexachlorodibenzo-*p*-dioxin [b]	19408-74-3	4.0×10^{-4}	
1,2,3,4,6,7,8-Heptachlorodibenzo-*p*-dioxin [b]	35822-46-9	4.0×10^{-3}	
1,2,3,4,6,7,8,9-Octachlorodibenzo-*p*-dioxin [b]	3268-87-9	1.3×10^{-1}	

Table 6.3 Chronic Inhalation Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Chlorinated Dibenzofurans [b]			
2,3,7,8-Tetrachlorodibenzofuran [b]	5120-73-19	4.0×10^{-4}	Alimentary System (Liver); Development; Endocrine System; Hematologic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzofuran [b]	57117-41-6	1.3×10^{-3}	
2,3,4,7,8-Pentachlorodibenzofuran [b]	57117-31-4	1.3×10^{-4}	
1,2,3,4,7,8-Hexachlorodibenzofuran [b]	70648-26-9	4.0×10^{-4}	
1,2,3,6,7,8-Hexachlorodibenzofuran [b]	57117-44-9	4.0×10^{-4}	
1,2,3,7,8,9-Hexachlorodibenzofuran [b]	72918-21-9	4.0×10^{-4}	
2,3,4,6,7,8-Hexachlorodibenzofuran [b]	60851-34-5	4.0×10^{-4}	
1,2,3,4,6,7,8-Heptachlorodibenzofuran [b]	67562-39-4	4.0×10^{-3}	
1,2,3,4,7,8,9-Heptachlorodibenzofuran [b]	55673-89-7	4.0×10^{-3}	
1,2,3,4,6,7,8,9-Octachlorodibenzofuran [b]	39001-02-0	1.3×10^{-1}	
Chlorobenzene	108-90-7	$1.0 \times 10^{+3}$	Alimentary System (Liver); Kidney; Reproductive System
Chloroform	67-66-3	$3.0 \times 10^{+2}$	Alimentary System (Liver); Development; Kidney
Chloropicrin	76-06-2	4.0×10^{-1}	Respiratory System
Chromium VI & Soluble Chromium VI Compounds (except chromic trioxide)	18540-29-9	2.0×10^{-1}	Respiratory System
Chromic Trioxide (as chromic acid mist)	1333-82-0	2.0×10^{-3}	Respiratory System
Cresol Mixtures	1319-77-3	$6.0 \times 10^{+2}$	Nervous System
1,4-Dichlorobenzene	106-46-7	$8.0 \times 10^{+2}$	Alimentary System (Liver); Kidney; Nervous System; Respiratory System
1,1-Dichloroethylene (Vinylidene Chloride)	75-35-4	$7.0 \times 10^{+1}$	Alimentary System (Liver)
Diesel Exhaust [a]	N/A	$5.0 \times 10^{+0}$	Respiratory System
Diethanolamine	111-42-2	$3.0 \times 10^{+0}$	Hematologic System; Respiratory System
N,N-Dimethylformamide	68-12-2	$8.0 \times 10^{+1}$	Alimentary System (Liver); Respiratory System
1,4-Dioxane	123-91-1	$3.0 \times 10^{+3}$	Alimentary System (Liver); Cardiovascular System; Kidney
Epichlorohydrin	106-89-8	$3.0 \times 10^{+0}$	Eyes; Respiratory System
1,2-Epoxybutane	106-88-7	$2.0 \times 10^{+1}$	Cardiovascular System; Respiratory System
Ethylbenzene	100-41-4	$2.0 \times 10^{+3}$	Alimentary System (Liver); Kidney; Development; Endocrine System
Ethyl Chloride	75-00-3	$3.0 \times 10^{+4}$	Alimentary System (Liver); Development
Ethylene Dibromide	106-93-4	8.0×10^{-1}	Reproductive System
Ethylene Dichloride	107-06-2	$4.0 \times 10^{+2}$	Alimentary System (Liver)
Ethylene Glycol	107-21-1	$4.0 \times 10^{+2}$	Development; Kidney; Respiratory System
Ethylene Glycol Monoethyl Ether	110-80-5	$7.0 \times 10^{+1}$	Hematologic System; Reproductive System
Ethylene Glycol Monoethyl Ether Acetate	111-15-9	$3.0 \times 10^{+2}$	Development

Table 6.3 Chronic Inhalation Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Ethylene Glycol Monomethyl Ether	109-86-4	$6.0 \times 10^{+1}$	Reproductive System
Ethylene Glycol Monomethyl Ether Acetate	110-49-6	$9.0 \times 10^{+1}$	Reproductive System
Ethylene Oxide	75-21-8	$3.0 \times 10^{+1}$	Nervous System
Fluorides (except hydrogen fluoride)	N/A	$1.3 \times 10^{+1}$	Bone and Teeth; Respiratory System
Formaldehyde	50-00-0	$9.0 \times 10^{+0}$	Respiratory System
Glutaraldehyde	111-30-8	8.0×10^{-2}	Respiratory System
Hexane (n-)	110-54-3	$7.0 \times 10^{+3}$	Nervous System
Hydrazine	302-01-2	2.0×10^{-1}	Alimentary System (Liver); Endocrine System
Hydrogen Chloride	7647-01-0	$9.0 \times 10^{+0}$	Respiratory System
Hydrogen Cyanide	74-90-8	$9.0 \times 10^{+0}$	Cardiovascular System; Endocrine System; Nervous System
Hydrogen Fluoride	7664-39-3	$1.4 \times 10^{+1}$	Bone and Teeth; Respiratory System
Hydrogen Sulfide	7783-06-4	$1.0 \times 10^{+1}$	Respiratory System
Isophorone	78-59-1	$2.0 \times 10^{+3}$	Alimentary System (Liver); Development
Isopropanol	67-63-0	$7.0 \times 10^{+3}$	Development; Kidney
Maleic Anhydride	108-31-6	7.0×10^{-1}	Respiratory System
Manganese & Manganese Compounds	7439-96-5	9.0×10^{-2}	Nervous System
Mercury & Inorganic Mercury Compounds	7439-97-6	3.0×10^{-2}	Nervous System; Development; Kidney
Methanol	67-56-1	$4.0 \times 10^{+3}$	Development
Methyl Bromide	74-83-9	$5.0 \times 10^{+0}$	Development; Nervous System; Respiratory System
Methyl Chloroform	71-55-6	$1.0 \times 10^{+3}$	Nervous System
Methyl Isocyanate	624-83-9	$1.0 \times 10^{+0}$	Reproductive System; Respiratory System
Methyl tertiary-Butyl Ether	1634-04-4	$8.0 \times 10^{+3}$	Alimentary System (Liver); Eyes; Kidney
Methylene Chloride	75-09-2	$4.0 \times 10^{+2}$	Cardiovascular System; Nervous System
4,4'-Methylene Dianiline (& its dichloride)	101-77-9	$2.0 \times 10^{+1}$	Alimentary System (Liver); Eyes
Methylene Diphenyl Isocyanate	101-68-8	7.0×10^{-1}	Respiratory System
Naphthalene	91-20-3	$9.0 \times 10^{+0}$	Respiratory System
Nickel & Nickel Compounds (except nickel oxide)	7440-02-0	1.4×10^{-2}	Hematologic System; Respiratory System
Nickel Oxide	1313-99-1	2.0×10^{-2}	Respiratory System
Perchloroethylene (Tetrachloroethylene)[a]	127-18-4	$3.5 \times 10^{+1}$	Alimentary System (Liver); Kidney
Phenol	108-95-2	$2.0 \times 10^{+2}$	Alimentary System (Liver); Cardiovascular System; Kidney; Nervous System
Phosphine	7803-51-2	8.0×10^{-1}	Alimentary System (Liver); Hematologic System; Kidney; Nervous System; Respiratory System

Table 6.3 Chronic Inhalation Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Phosphoric Acid	7664-38-2	$7.0 \times 10^{+0}$	Respiratory System
Phthalic Anhydride	85-44-9	$2.0 \times 10^{+1}$	Respiratory System
Polychlorinated biphenyls (PCBs)[b]			
3,3',4,4'-Tetrachlorobiphenyl (77) [b]	35298-13-3	4.0×10^{-1}	Alimentary System (Liver); Developmental; Endocrine System; Hematologic System; Reproductive System; Respiratory System
3,4,4',5-Tetrachlorobiphenyl (81) [b]	70362-50-4	1.3×10^{-1}	
2,3,3',4,4'- Pentachlorobiphenyl (105) [b]	32598-14-4	$1.3 \times 10^{+0}$	
2,3,4,4'5- Pentachlorobiphenyl (114) [b]	74472-37-0	$1.3 \times 10^{+0}$	
2,3'4,4',5- Pentachlorobiphenyl (118) [b]	31508-00-6	$1.3 \times 10^{+0}$	
2',3,4,4',5- Pentachlorobiphenyl (123) [b]	65510-44-3	$1.3 \times 10^{+0}$	
3,3',4,4',5- Pentachlorobiphenyl (126) [b]	57465-28-8	4.0×10^{-4}	
2,3,3',4,4',5-Hexachlorobiphenyl (156) [b]	38380-08-4	$1.3 \times 10^{+0}$	
2,3,3',4,4',5'-Hexachlorobiphenyl (157) [b]	69782-90-7	$1.3 \times 10^{+0}$	
2,3',4,4',5,5'-Hexachlorobiphenyl (167) [b]	52663-72-6	$1.3 \times 10^{+0}$	
3,3',4,4'5,5'- Hexachlorobiphenyl (169) [b]	32774-16-6	1.3×10^{-3}	
2,3,3'4,4',5,5'-Heptachlorobiphenyl (189) [b]	39635-31-9	$1.3 \times 10^{+0}$	
Propylene	115-07-1	$3.0 \times 10^{+3}$	Respiratory System
Propylene Glycol Monomethyl Ether	107-98-2	$7.0 \times 10^{+3}$	Alimentary System (Liver)
Propylene Oxide	75-56-9	$3.0 \times 10^{+1}$	Respiratory System
Selenium and Selenium compounds (other than Hydrogen Selenide)	7782-49-2	$2.0 \times 10^{+1}$	Alimentary System (Liver); Cardiovascular System; Nervous System
Silica (crystalline, respirable)	N/A	$3.0 \times 10^{+0}$	Respiratory System
Styrene	100-42-5	$9.0 \times 10^{+2}$	Nervous System
Sulfuric Acid	7664-93-9	$1.0 \times 10^{+0}$	Respiratory System
Toluene	108-88-3	$3.0 \times 10^{+2}$	Development; Nervous System; Respiratory System
2,4-Toluene Diisocyanate	584-84-9	7.0×10^{-2}	Respiratory System
2,6-Toluene Diisocyanate	91-08-7	7.0×10^{-2}	Respiratory System
Trichloroethylene [a]	79-01-6	$6.0 \times 10^{+2}$	Eyes; Nervous System
Triethylamine	121-44-8	$2.0 \times 10^{+2}$	Eyes
Vinyl Acetate	108-05-4	$2.0 \times 10^{+2}$	Respiratory System
Xylenes (m, o, p-isomers)	1330-20-7	$7.0 \times 10^{+2}$	Nervous System; Respiratory System; Eyes

[a] These peer-reviewed values were developed under the Toxic Air Contaminant (TAC) Program mandated by AB1807 (California Health and Safety Code Sec. 39650 *et seq*.).

[b] The OEHHA has adopted the World Health Organization Toxicity Equivalency Factor (TEF) scheme for evaluating the cancer risk and noncancer hazard due to exposure to samples containing mixtures of polychlorinated dibenzo-*p*-dioxins (PCDD) (also referred to as chlorinated dioxins and dibenzofurans), polychlorinated dibenzofurans (PCDF) and polychlorinated biphenyls (PCBs). The TEF values are revised from time to time to reflect new data and increased scientific knowledge. Currently OEHHA recommends use of the 2005 revision to the WHO TEF values (WHO_{05}-TEF). See Appendix E for more information about the scheme and for the methodology for calculating 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) equivalents for PCDD and PCDFs. For

convenience, OEHHA has calculated chronic REL values for speciated PCDDs, PCDFs and PCBs based on the WHO_{05} TEF values and the chronic REL for 2,3,7,8-TCDD using the procedure discussed in Appendix E. The chronic REL values can be used to calculate a hazard index when the mixtures are speciated from individual congener ground level concentrations. In those cases where speciation of dioxins and furans has not been performed, then 2,3,7,8-TCDD serves as the surrogate for dioxin and furan emissions.

N/A Not Applicable

6.5 Chronic Oral (Noninhalation) Reference Exposure Levels

As specified throughout the guidelines, estimates of long-term exposure resulting from facility air emissions of specific compounds must be analyzed for both inhalation and noninhalation (multipathway) pathways of exposure for humans. Facilities often emit substances under high temperature and pressure in the presence of particulate matter. While some of these substances are expected to remain in the vapor phase, other substances such as metals and semi-volatile organics can be either emitted as particles, form particles after emission from the facility, or adhere to existing particles. Some substances will partition between vapor and particulate phases. Substances in the particulate phase can be removed from the atmosphere by settling and, thus, potentially present a significant hazard via noninhalation pathways.

Particulate-associated chemicals can be deposited directly onto soil, onto the leaves or fruits of crops, or onto surface waters. Exposure via the oral route is the predominant noninhalation pathway, resulting in the noninhalation RELs being referred to as 'oral RELs' in this document. The oral RELs are used for both ingestion and dermal exposures, and are applied using the chronic non-inhalation exposures in the residential scenario and the worker scenarios. The oral RELs are expressed as doses in milligrams of substance (consumed and dermally absorbed) per kilogram body weight per day (mg/kg-day).

Table 6.4 lists the chronic noncancer RELs to be used in the assessment of chronic health effects from noninhalation pathways of exposure. Any substances in Table 6.4 with Development or Reproductive System as a target organ system are represented in HARP and in the Appendix L REL tables under the single endpoint "Reproductive/Development". Appendix L provides a consolidated listing of all chronic RELs and target organs that are approved for use by OEHHA and ARB for the Hot Spots Program. Periodically, new or updated RELs are adopted by OEHHA and these guidelines will be updated to reflect those changes. See OEHHA's web page at http://www.oehha.ca.gov/air/allrels.html to determine if any new or updated RELs have been adopted since the last guideline update. Chapter 8 discusses the methods used for determining potential noncancer health impacts and Appendix I presents example calculations used to determine a HQ and HI.

Table 6.4 Chronic Noninhalation 'Oral' Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service No. (CAS)	Chronic Oral REL (mg/kg-day)	Chronic Oral Hazard Index Target Organ System(s)
Arsenic & Inorganic Arsenic Compounds	7440-38-2	3.5×10^{-6}	Development; Nervous System; Respiratory System; Cardiovascular System; Skin
Beryllium and Beryllium Compounds	7440-41-7	2.0×10^{-3}	Alimentary System (Gastrointestinal Tract)
Cadmium and Cadmium Compounds	7440-43-9	5.0×10^{-4}	Kidney
Chlorinated Dibenzo-*p*-dioxins [a]			
2,3,7,8-Tetrachlorodibenzo-*p*-dioxin [a]	1746-01-6	1.0×10^{-8}	Alimentary System (Liver); Developmental; Endocrine System; Hematologic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzo-*p*-dioxin [a]	40321-76-4	1.0×10^{-8}	
1,2,3,4,7,8-Hexachlorodibenzo-*p*-dioxin [a]	39227-28-6	1.0×10^{-7}	
1,2,3,6,7,8-Hexachlorodibenzo-*p*-dioxin [a]	57653-85-7	1.0×10^{-7}	
1,2,3,7,8,9-Hexachlorodibenzo-*p*-dioxin [a]	19408-74-3	1.0×10^{-7}	
1,2,3,4,6,7,8-Heptachlorodibenzo-*p*-dioxin [a]	35822-46-9	1.0×10^{-6}	
1,2,3,4,6,7,8,9-Octachlorodibenzo-*p*-dioxin [a]	3268-87-9	3.3×10^{-5}	
Chlorinated Dibenzofurans [a]			
2,3,7,8-Tetrachlorodibenzofuran [a]	5120-73-19	1.0×10^{-7}	Alimentary System (Liver); Development; Endocrine System; Hematologic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzofuran [a]	57117-41-6	3.3×10^{-7}	
2,3,4,7,8-Pentachlorodibenzofuran [a]	57117-31-4	3.3×10^{-8}	
1,2,3,4,7,8-Hexachlorodibenzofuran [a]	70648-26-9	1.0×10^{-7}	
1,2,3,6,7,8-Hexachlorodibenzofuran [a]	57117-44-9	1.0×10^{-7}	
1,2,3,7,8,9-Hexachlorodibenzofuran [a]	72918-21-9	1.0×10^{-7}	
2,3,4,6,7,8-Hexachlorodibenzofuran [a]	60851-34-5	1.0×10^{-7}	
1,2,3,4,6,7,8-Heptachlorodibenzofuran [a]	67562-39-4	1.0×10^{-6}	
1,2,3,4,7,8,9-Heptachlorodibenzofuran [a]	55673-89-7	1.0×10^{-6}	
1,2,3,4,6,7,8,9-Octachlorodibenzofuran [a]	39001-02-0	3.3×10^{-5}	
Chromium VI & Soluble Chromium VI Compounds (including chromic trioxide)	18540-29-9	2.0×10^{-2}	Hematologic System
Fluorides (including hydrogen fluoride)	7664-39-3	4.0×10^{-2}	Bone and Teeth
Mercury & Mercury Inorganic Compounds	7439-97-6	1.6×10^{-4}	Kidney; Nervous System; Development
Nickel & Nickel Compounds (including nickel oxide)	7440-02-0	1.1×10^{-2}	Development
Polychlorinated biphenyls (PCBs) (speciated)[a]			
3,3',4,4'-Tetrachlorobiphenyl (77)[a]	35298-13-3	1.0×10^{-4}	Alimentary System (Liver); Developmental; Endocrine System; Hematologic System; Reproductive System; Respiratory System
3,4,4',5-Tetrachlorobiphenyl (81)[a]	70362-50-4	3.3×10^{-5}	
2,3,3',4,4'- Pentachlorobiphenyl (105)[a]	32598-14-4	3.3×10^{-4}	
2,3,4,4'5- Pentachlorobiphenyl (114)[a]	74472-37-0	3.3×10^{-4}	
2,3'4,4',5- Pentachlorobiphenyl (118)[a]	31508-00-6	3.3×10^{-4}	
2',3,4,4',5- Pentachlorobiphenyl (123)[a]	65510-44-3	3.3×10^{-4}	
3,3',4,4',5- Pentachlorobiphenyl (126)[a]	57465-28-8	1.0×10^{-7}	
2,3,3',4,4',5-Hexachlorobiphenyl (156)[a]	38380-08-4	3.3×10^{-4}	
2,3,3',4,4',5'-Hexachlorobiphenyl (157)[a]	69782-90-7	3.3×10^{-4}	
2,3',4,4',5,5'-Hexachlorobiphenyl (167)[a]	52663-72-6	3.3×10^{-4}	
3,3',4,4'5,5'- Hexachlorobiphenyl (169)[a]	32774-16-6	3.3×10^{-7}	
2,3,3'4,4',5,5'- Heptachlorobiphenyl (189)[a]	39635-31-9	3.3×10^{-4}	

Table 6.4 Chronic Noninhalation 'Oral' Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service No. (CAS)	Chronic Oral REL (mg/kg-day)	Chronic Oral Hazard Index Target Organ System(s)
Selenium and Selenium Compounds (other than hydrogen selenide)	7782-49-2	5.0×10^{-3}	Alimentary System (Liver); Cardiovascular System; Nervous System

a The OEHHA has adopted the World Health Organization Toxicity Equivalency Factor (TEF) scheme for evaluating the cancer risk and noncancer risk due to exposure to samples containing mixtures of polychlorinated dibenzo-*p*-dioxins (PCDD) (also referred to as chlorinated dioxins and dibenzofurans), polychlorinated dibenzofurans (PCDF), and polychlorinated biphenyls (PCBs). The TEF values are revised from time to time to reflect new data and increased scientific knowledge. Currently OEHHA recommends use of the 2005 revision to the WHO TEF values (WHO_{05}-TEF). See Appendix E for more information about the scheme and for the methodology for calculating 2,3,7,8-equivalents for PCDD and PCDFs. For convenience, OEHHA has calculated chronic 'oral' REL values for speciated PCDDs, PCDFs, and PCBs based on the WHO_{05} TEF values and the chronic 'oral' REL for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin using the procedure discussed in Appendix E. The chronic 'oral' REL values can be used to calculate a hazard index when the mixtures are speciated from individual congener ground level concentrations. In those cases where speciation of dioxins and furans has not been performed, then 2,3,7,8-TCDD serves as the surrogate for dioxin and furan emissions.

6.6 References

OEHHA, 2008. Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. Available online at: http://www.oehha.ca.gov

Page Intentionally Left Blank

7 - Dose-Response Assessment for Carcinogens

7.1 Introduction

Dose-response assessment characterizes the quantitative relationship between the amount of exposure to a substance (the dose) and the incidence or occurrence of injury (the response). The process often involves establishing a toxicity value or criterion to use in assessing potential health risk. The toxicity criterion, or health guidance value, for carcinogens is the cancer potency slope (potency factor), which describes the potential risk of developing cancer per unit of average daily dose over a 70-year lifetime. Cancer inhalation and oral potency factors have been derived by the Office of Environmental Health Hazard Assessment (OEHHA) or by the United States Environmental Protection Agency (U.S. EPA) and approved by the State's Scientific Review Panel on Toxic Air Contaminants. They are available for many of the substances listed in Appendix A (List of Substances) as carcinogens. Table 7.1 and Appendix L list the inhalation and oral cancer potency factors that should be used in multipathway health risk assessments (HRAs) for the Hot Spots Program.

The details on the methodology of dose-response assessment for carcinogens and the approved cancer potency factors are provided in the Air Toxics Hot Spots Risk Assessment Guidelines. Part II. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. May, 2009. (OEHHA, 2009; see http://www.oehha.ca.gov/air/hot_spots/tsd052909.html).

7.2 Carcinogenic Potency

Cancer potency factors used for both the inhalation and oral routes in the Hot Spots program are generally the 95% upper confidence limits (UCL) on the modeled dose-response slope at the low dose range. The cancer slope factor assumes continuous lifetime exposure to a substance, and is expressed in units of inverse dose [i.e., $(mg/kg/day)^{-1}$]. Another common potency expression is in units of inverse concentration [$(\mu g/m^3)^{-1}$)] when the slope is based on exposure concentration rather than dose; this is termed the unit risk factor. To accommodate the use of age-specific exposure variates, the Hot Spots program has translated the unit risk factors based on concentration to units of inverse dose. This allows calculation of risk for age groupings, as exposure varies with age. It also allows for application of Age Sensitivity Factors for early life exposures.

It is assumed in cancer risk assessments that risk is directly proportional to dose and that, for most carcinogens, there is no threshold for carcinogenesis. The derivation of inhalation and oral cancer potency factors takes into account information on pharmacokinetics, when available, and on the mechanism of carcinogenic action.

Table 7.1 and Appendix L list inhalation and oral cancer potency factors that should be used in risk assessments for the Hot Spots Program. Chapter 8 describes procedures for use of potency factors in estimating potential cancer risk.

7.2.1 *Inhalation Cancer Potency Factors*

The risk assessment methodology and algorithms presented in Chapter 8 express the inhalation cancer slope factors in units of inverse dose (i.e., $(mg/kg/day)^{-1}$). Breathing rates, expressed in units of liters per kilogram of body weight-day (L/kg-day), are multiplied with the air concentrations, coupled with the appropriate unit conversion factor, to estimate dose in mg/kg-day. This allows estimation of average and high-end cancer risk point estimates. Estimation of a distribution of cancer risk based on variability in breathing rate can be obtained by Monte Carlo methods using the distributions of breathing rates in L/kg-day, which can then be converted to a dose distribution in mg/kg BW based on the intake rate. Unit risk factors [in the units of inverse concentration (i.e., $(\mu g/m^3)^{-1}$], which were used in previous guidelines for the Hot Spots program, are still listed in the TSD (OEHHA, 2009) and may prove useful in other risk assessment applications.

The average daily inhalation dose (mg/kg-day) multiplied by the cancer potency factor $(mg/kg\text{-}day)^{-1}$ will give the inhalation cancer risk (unitless), which is an expression of the chemical's cancer risk during a 70-year lifespan of exposure. For example, an inhalation cancer risk of 5×10^{-6} is the same as stating that an individual has an estimated probability of developing cancer from their exposure of 5 chances per million people exposed. A more complete description of how potential cancer risk is calculated from the exposure dose and cancer potency factors is provided in Chapter 8. Appendix I presents an example calculation for determining cancer risk.

A list of current inhalation potency factors is provided in Table 7.1. Periodically, new or revised cancer potency factors will be peer reviewed by the State's Scientific Review Panel on Toxic Air Contaminants (SRP) and adopted by the Director of OEHHA. For new or updated numbers, consult the OEHHA web site at (http://www.oehha.ca.gov/air/hot_spots/tsd052909.html) to determine if any new or updated cancer potency factors have been adopted since this guideline update. New cancer potency factors that have been approved by the SRP and adopted by the Director of OEHHA should be incorporated into Hot Spots risk assessment for facilities that emit those chemicals.

7.2.2 *Oral Cancer Potency Factors*

Under the Hot Spots Program, a few substances are evaluated for exposure and risk from non-inhalation pathways – these are referred to as multipathway substances. Multipathway substances have the potential to impact a receptor through inhalation and noninhalation (oral and dermal) exposure routes. These substances include heavy metals and semi-volatile organic substances such as dioxins, furans, and polycyclic aromatic hydrocarbons (PAHs). These substances commonly exist in the particle

phase or partially in the particle phase when emitted into the air. They can therefore be deposited onto soil, vegetation, and water. Noninhalation exposure pathways considered under the Hot Spots Program include the ingestion of soil, homegrown produce, meat, milk, surface water, breast milk, and fish as well as dermal exposure to contaminants deposited in the soil. See Table 5.1 for a list of the multipathway substances.

Table 7.1 and Appendix L list oral cancer potency factors in units of $(mg/kg\text{-}day)^{-1}$ that should be used for assessing the potential cancer risk for these substances through noninhalation exposure pathways. The cancer risk from these individual pathways is calculated by multiplying the dose (mg/kg-day) times the oral cancer potency factor $(mg/kg\text{-}day)^{-1}$ to yield the potential cancer risk (unitless) from non-inhalation exposures. Chapter 5 provides all of the algorithms to calculate exposure dose through all of the individual exposure pathways. Appendix I provides a sample calculation for dose and cancer risk using the inhalation exposure pathway.

Three carcinogens (cadmium, beryllium, and nickel), although subject to deposition, are only treated as carcinogenic by the inhalation route and not by the oral route. Therefore, there are no oral cancer potency factors for these substances. However, the oral doses of these substances need to be estimated because of their noncancer toxicity. See Chapters 6 and 8, and Appendices I and L for dose-response factors, and calculations to address these substances.

Table 7.1 Inhalation and Oral Cancer Potency Factors

Substance	Chemical Abstract Service Number (CAS)	Inhalation Potency Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
Acetaldehyde	75-07-0	1.0×10^{-2}	
Acetamide	60-35-5	7.0×10^{-2}	
Acrylamide	79-06-1	$4.5 \times 10^{+0}$	
Acrylonitrile	107-13-1	$1.0 \times 10^{+0}$	
Allyl chloride	107-05-1	2.1×10^{-2}	
2-Aminoanthraquinone	117-79-3	3.3×10^{-2}	
Aniline	62-53-3	5.7×10^{-3}	
Arsenic (inorganic)	7440-38-2	$1.2 \times 10^{+1}$	$1.5 \times 10^{+0}$
Asbestos [#]	1332-21-4	$2.2 \times 10^{+2}$ [#]	
Benz[*a*]anthracene [BaP]	56-55-3	3.9×10^{-1}	$1.2 \times 10^{+0}$
Benzene	71-43-2	1.0×10^{-1}	
Benzidine	92-87-5	$5.0 \times 10^{+2}$	
Benzo[*a*]pyrene	50-32-8	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
Benzo[*b*]fluoranthrene [BaP]	205-99-2	3.9×10^{-1}	$1.2 \times 10^{+0}$
Benzo[*j*]fluoranthrene [BaP]	205-82-3	3.9×10^{-1}	$1.2 \times 10^{+0}$
Benzo[*k*]fluoranthrene [BaP]	207-08-9	3.9×10^{-1}	$1.2 \times 10^{+0}$
Benzyl chloride	100-44-7	1.7×10^{-1}	
Beryllium	7440-41-7	$8.4 \times 10^{+0}$	
Bis(2-chloroethyl) ether	111-44-4	$2.5 \times 10^{+0}$	
Bis(chloromethyl)ether	542-88-1	$4.6 \times 10^{+1}$	
1,3-Butadiene	106-99-0	6.0×10^{-1}	
Cadmium (and compounds)	7440-43-9	$1.5 \times 10^{+1}$	
Carbon tetrachloride	56-23-5	1.5×10^{-1}	
Chlorinated Dibenzo-p-dioxins [A]			
2,3,7,8-Tetrachlorodibenzo-*p*-dioxin	1746-01-6	$1.3 \times 10^{+5}$	$1.3 \times 10^{+5}$
1,2,3,7,8-Pentachlorodibenzo-*p*-dioxin	40321-76-4	$1.3 \times 10^{+5}$	$1.3 \times 10^{+5}$
1,2,3,4,7,8-Hexachlorodibenzo-*p*-dioxin	39227-28-6	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,6,7,8-Hexachlorodibenzo-*p*-dioxin	57653-85-7	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,7,8,9-Hexachlorodibenzo-*p*-dioxin	19408-74-3	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,4,6,7,8-Heptachlorodibenzo-*p*-dioxin	35822-46-9	$1.3 \times 10^{+3}$	$1.3 \times 10^{+3}$
1,2,3,4,,6,7,8,9-Octachlorodibenzo-*p*-dioxin	3268-87-9	$3.9 \times 10^{+1}$	$3.9 \times 10^{+1}$
Chlorinated Dibenzofurans [A]			
2,3,7,8-Tetrachlorodibenzofuran	5120-73-19	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,7,8-Pentachlorodibenzofuran	57117-41-6	$3.9 \times 10^{+3}$	$3.9 \times 10^{+3}$
2,3,4,7,8-Pentachlorodibenzofuran	57117-31-4	$3.9 \times 10^{+4}$	$3.9 \times 10^{+4}$
1,2,3,4,7,8-Hexachlorodibenzofuran	70648-26-9	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,6,7,8-Hexachlorodibenzofuran	57117-44-9	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,7,8,9-Hexachlorodibenzofuran	72918-21-9	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
2,3,4,6,7,8-Hexachlorodibenzofuran	60851-34-5	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$

Table 7.1 Inhalation and Oral Cancer Potency Factors

Substance	Chemical Abstract Service Number (CAS)	Inhalation Potency Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
1,2,3,4,6,7,8-Heptachlorodibenzofuran	67562-39-4	$1.3 \times 10^{+3}$	$1.3 \times 10^{+3}$
1,2,3,4,7,8,9-Heptachlorodibenzofuran	55673-89-7	$1.3 \times 10^{+3}$	$1.3 \times 10^{+3}$
1,2,3,4,,6,7,8,9-Octachlorodibenzofuran	39001-02-0	$3.9 \times 10^{+1}$	$3.9 \times 10^{+1}$
Chlorinated paraffins	108171-26-2	8.9×10^{-2}	
Chloroform	67-66-3	1.9×10^{-2}	
4-Chloro-*o*-phenylenediamine	95-83-0	1.6×10^{-2}	
p-Chloro-*o*-toluidine	95-69-2	2.7×10^{-1}	
Chromium (hexavalent)	18540-29-9	$5.1 \times 10^{+2}$	5×10^{-1}
Chrysene [BaP]	218-01-9	3.9×10^{-2}	1.2×10^{-1}
Creosote	8001-58-9	*	
p-Cresidine	120-71-8	1.5×10^{-1}	
Cupferron	135-20-6	2.2×10^{-1}	
2,4-Diaminoanisole	615-05-4	2.3×10^{-2}	
2,4-Diaminotoluene	95-80-7	$4.0 \times 10^{+0}$	
Dibenz[*a,h*]acridine [BaP]	226-36-8	3.9×10^{-1}	$1.2 \times 10^{+0}$
Dibenz[*a,j*]acridine [BaP]	224-42-0	3.9×10^{-1}	$1.2 \times 10^{+0}$
Dibenz[*a,h*]anthracene [BaP]	53-70-3	$4.1 \times 10^{+0}$	$4.1 \times 10^{+0}$
Dibenzo[*a,e*]pyrene [BaP]	192-65-4	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
Dibenzo[*a,h*]pyrene [BaP]	189-64-0	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
Dibenzo[*a,l*]pyrene [BaP]	189-55-9	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
Dibenzo[*a,l*]pyrene [BaP]	191-30-0	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
7H-Dibenzo[*c,g*]carbazole [BaP]	194-59-2	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
1,2-Dibromo-3-chloropropane	96-12-8	$7.0 \times 10^{+0}$	
1,4-Dichlorobenzene	106-46-7	4.0×10^{-2}	
3,3'-Dichlorobenzidine	91-94-1	$1.2 \times 10^{+0}$	
1,1-Dichloroethane	75-34-3	5.7×10^{-3}	
Diesel exhaust [B]	NA	$1.1 \times 10^{+0}$	
Diethylhexylphthalate	117-81-7	8.4×10^{-3}	8.4×10^{-3}
p-Dimethylaminoazobenzene	60-11-7	$4.6 \times 10^{+0}$	
7,12-Dimethylbenz[*a*]anthracene [BaP]	57-97-6	$2.5 \times 10^{+2}$	$2.5 \times 10^{+2}$
1,6-Dinitropyrene [BaP]	42397-64-8	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
1,8-Dinitropyrene [BaP]	42397-65-9	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
2,4-Dinitrotoluene	121-14-2	3.1×10^{-1}	
1,4-Dioxane	123-91-1	2.7×10^{-2}	
Epichlorohydrin	106-89-8	8.0×10^{-2}	
Ethyl benzene	100-41-4	8.7×10^{-3}	1.1×10^{-2}
Ethylene dibromide	106-93-4	2.5×10^{-1}	
Ethylene dichloride	107-06-2	7.2×10^{-2}	
Ethylene oxide	75-21-8	3.1×10^{-1}	

Table 7.1 Inhalation and Oral Cancer Potency Factors

Substance	Chemical Abstract Service Number (CAS)	Inhalation Potency Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
Ethylene thiourea	96-45-7	4.5×10^{-2}	
Formaldehyde	50-00-0	2.1×10^{-2}	
Hexachlorobenzene	118-74-1	$1.8 \times 10^{+0}$	
Hexachlorocyclohexanes (technical grade)	608-73-1	$4.0 \times 10^{+0}$	$4.0 \times 10^{+0}$
Hydrazine	302-01-2	$1.7 \times 10^{+1}$	$3.0 \times 10^{+0}$
Indeno[1,2,3-*cd*]pyrene [BaP]	193-39-5	3.9×10^{-1}	$1.2 \times 10^{+0}$
Lead and lead compounds	7439-92-1	4.2×10^{-2}	8.5×10^{-3}
Lindane	58-89-9	$1.1 \times 10^{+0}$	$1.1 \times 10^{+0}$
Methyl tertiary-butyl ether	1634-04-4	1.8×10^{-3}	
3-Methylcholanthrene [BaP]	56-49-5	$2.2 \times 10^{+1}$	$2.2 \times 10^{+1}$
5-Methylchrysene [BaP]	3697-24-3	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
4, 4'-Methylene bis(2-chloroaniline) (MOCA)	101-14-4	$1.5 \times 10^{+0}$	
Methylene chloride	75-09-2	3.5×10^{-3}	
4,4'-Methylenedianiline	101-77-9	$1.6 \times 10^{+0}$	$1.6 \times 10^{+0}$
Michler's ketone	90-94-8	8.6×10^{-1}	
Naphthalene	91-20-3	1.2×10^{-1}	
Nickel (and compounds)	7440-02-0	9.1×10^{-1}	
5-Nitroacenaphthene [BaP]	602-87-9	1.3×10^{-1}	1.3×10^{-1}
6-Nitrochrysene [BaP]	7496-02-8	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
2-Nitrofluorene [BaP]	607-57-8	3.9×10^{-2}	1.2×10^{-1}
1-Nitropyrene [BaP]	5522-43-0	3.9×10^{-1}	$1.2 \times 10^{+0}$
4-Nitropyrene [BaP]	57835-92-4	3.9×10^{-1}	$1.2 \times 10^{+0}$
N-Nitroso-n-butylamine	924-16-3	$1.1 \times 10^{+1}$	
N-Nitroso-N-methylethylamine	10595-95-6	$2.2 \times 10^{+1}$	
N-Nitrosodi-*n*-propylamine	621-64-7	$7.0 \times 10^{+0}$	
N-Nitrosodiethylamine	55-18-5	$3.6 \times 10^{+1}$	
N-Nitrosodimethylamine	62-75-9	$1.6 \times 10^{+1}$	
N-Nitrosodiphenylamine	86-30-6	9.0×10^{-3}	
p-Nitrosodiphenylamine	156-10-5	2.2×10^{-2}	
N-Nitrosomorpholine	59-89-2	$6.7 \times 10^{+0}$	
N-Nitrosopiperidine	100-75-4	$9.4 \times 10^{+0}$	
N-Nitrosopyrrolidine	930-55-2	$2.1 \times 10^{+0}$	
Pentachlorophenol	87-86-5	1.8×10^{-2}	
Perchloroethylene	127-18-4	2.1×10^{-2}	5.1×10^{-2}
Polychlorinated biphenyls (PCBs) (unspeciated mixture)	1336-36-3		
(high risk) [P1]		$2.0 \times 10^{+0}$	$2.0 \times 10^{+0}$
(low risk) [P2]		4.0×10^{-1}	4.0×10^{-1}
(lowest risk) [P3]		7.0×10^{-2}	7.0×10^{-2}

Table 7.1 Inhalation and Oral Cancer Potency Factors

Substance	Chemical Abstract Service Number (CAS)	Inhalation Potency Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
Polychlorinated biphenyls[P4] (PCBs) (speciated)			
3,3',4,4'-Tetrachlorobiphenyl (77)	35298-13-3	$1.3 \times 10^{+1}$	$1.3 \times 10^{+1}$
3,4,4',5-Tetrachlorobiphenyl (81)	70362-50-4	$3.9 \times 10^{+1}$	$3.9 \times 10^{+1}$
2,3,3',4,4'- Pentachlorobiphenyl (105)	32598-14-4	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2,3,4,4'5- Pentachlorobiphenyl (114)	74472-37-0	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2,3'4,4',5- Pentachlorobiphenyl (118)	31508-00-6	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2',3,4,4',5- Pentachlorobiphenyl (123)	65510-44-3	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
3,3',4,4',5- Pentachlorobiphenyl (126)	57465-28-8	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
2,3,3',4,4',5-Hexachlorobiphenyl (156)	38380-08-4	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2,3,3',4,4',5'-Hexachlorobiphenyl (157)	69782-90-7	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2,3',4,4',5,5'-Hexachlorobiphenyl (167)	52663-72-6	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
3,3',4,4'5,5'- Hexachlorobiphenyl (169)	32774-16-6	$3.9 \times 10^{+3}$	$3.9 \times 10^{+3}$
2,3,3'4,4',5,5'- Heptachlorobiphenyl (189)	39635-31-9	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
Potassium bromate	7758-01-2	4.9×10^{-1}	
1,3-Propane sultone	1120-71-4	$2.4 \times 10^{+0}$	
Propylene oxide	75-56-9	1.3×10^{-2}	2.4×10^{-1}
1,1,2,2-Tetrachloroethane	79-34-5	2.0×10^{-1}	
Thioacetamide	62-55-5	$6.1 \times 10^{+0}$	
2,4-Toluene diisocyanate	584-84-9	3.9×10^{-2}	
2,6-Toluene diisocyanate	91-08-7	3.9×10^{-2}	
1,1,2-Trichloroethane (vinyl trichloride)	79-00-5	5.7×10^{-2}	
Trichloroethylene	79-01-6	7.0×10^{-3}	1.5×10^{-2}
2,4,6-Trichlorophenol	88-06-2	7.0×10^{-2}	
Urethane	51-79-6	$1.0 \times 10^{+0}$	
Vinyl chloride	75-01-4	2.7×10^{-1}	

Notes for Table 7.1

Asbestos: [100 PCM fibers/m^3]$^{-1}$ A unit risk factor of 2.7 x 10^{-6} ($\mu g/m^3$)$^{-1}$ and an inhalation cancer potency factor of 2.2 x 10^{+2} (mg/kg BW*day)$^{-1}$ are available (see Appendix C for explanation).

BaP PAHs and PAH Derivatives: Many have potency equivalency factors relative to benzo[*a*]pyrene (see Appendix G). For multipathway chemicals, including PAHs, the oral slope factor is considered the same as the inhalation potency factor unless otherwise noted in the Table.

A Polychlorinated Dibenzo-*p*-dioxins, Polychlorinated Dibenzofurans and speciated poly chlorinated biphenyls: (see Appendix E). For convenience, OEHHA has calculated cancer potency factors for speciated polychlorinated dibenzo-*p*-dioxin, polychlorinated dibenzofuran and polychlorinated biphenyl congeners using the procedure in Appendix E.

B Diesel Exhaust is listed as a Toxic Air Contaminant by the Air Resources Board as "Particulate Matter from Diesel-Fueled Engines". (See Appendix D)

***** Creosote: Can be calculated using Potency Equivalency Factors contained in the benzo[*a*]pyrene Toxic Air Contaminant document and in Appendix G of these guidelines.

P1 Polychlorinated Biphenyls (PCBs): High Risk is for use in cases where congeners with more than four chlorines do not comprise less (are greater) than one-half percent of total PCBs. The high risk number is the default for unspeciated PCB mixtures.

P2 The low risk number is generally not applicable to the Hot Spots program. The Hot Spots program addresses PCBs emitted by stationary facilities. It cannot be assumed that such emissions would occur by simple evaporation. There is a dermal absorption factor applied in evaluation of the dermal pathway for PCBs so the medium risk would not apply to dermal exposure (OEHHA, 2009). The water pathway does not include an assumption that PCB isomers are water soluble, so the medium number would not apply to the water pathway.

P3 Polychlorinated Biphenyls (PCBs): Lowest Risk is for use in cases where congeners with more than four chlorines comprise less than one-half percent of total PCBs. In order for the low number to be used, scientific justification needs to be presented.

P4 Number in parentheses is the IUPAC #, the PCB nomenclature is IUPAC. For multipathway chemicals, including PCBs, the oral slope factor is considered the same as the inhalation potency factor unless otherwise noted in the Table.

7.3 References

OEHHA, 2009. Air Toxics Hot Spots Risk Assessment Guidelines. Part II. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. May, 2009. Available online at: http://www.oehha.ca.gov/air/hot_spots/tsd052909.html

8 - Risk Characterization for Carcinogens and Noncarcinogens and the Requirements for Hot Spots Risk Assessments

8.1 Introduction

Risk characterization is the final step of the health risk assessment (HRA). In this step, information developed through the exposure assessment is combined with information from the dose-response assessment to characterize risks to the general public from emissions. In the Hot Spots program, OEHHA conducts the dose-response assessment during the development of cancer potency factors and Reference Exposure Levels. These are used in conjunction with the exposure estimates to estimate cancer risk and evaluate hazard from noncancer toxicity of emitted chemicals. Under the Air Toxics Hot Spots (Hot Spots) Act, risk characterizations should present both individual and population-wide health risks (Health and Safety Code Section (HSC) 44306). Persons preparing HRAs for the Hot Spots Program should consult the local Air Pollution Control or Air Quality Management District (District) to determine if the District has special guidelines to assist with HRA format or other requirements of the Hot Spots Program.

OEHHA is recommending that a 30-year exposure duration be used as the basis for estimating cancer risk at the maximum exposed individual resident (MEIR) in the Hot Spots Program. This exposure duration represents the time of residency for 90 to 95% of Californians at a single location and should provide adequate public health protection against individual risk. We also recommend including the 9 and 70-year cancer risk at the MEIR as supplemental information. Note that a 70-year exposure duration is required to estimate cancer burden or provide an estimate of population-wide risk.

This chapter provides guidance on how to evaluate the risk characterization component of risk assessments required by the Hot Spots Program. A general summary of the risk characterization components includes the following items and information.

- The locations of the point of maximum impact (PMI), the MEIR, and the maximum exposed individual worker (MEIW) are to be identified. The PMI, MEIW, and MEIR for cancer risk and for noncancer hazard indices (averaging times for acute 1-hour, repeated 8-hour, and chronic hazard indices) may not be the same location; all should be identified.

- The location of any specified sensitive receptors (e.g., schools, hospitals, daycare, or eldercare facilities - contact the District or reviewing authority for more information) should be identified

- Estimates of population-wide cancer risk and noncancer hazard

This information must be clearly presented in cross-referenced text, tables, figures, and maps. Chapter 9 provides an outline that specifies the content and recommended format of HRA results. The HARP software is the recommended model for calculating HRA results for the Hot Spots Program. Information on obtaining the HARP software can be found under the Air Toxics Program on the ARB's web site at *www.arb.ca.gov*.

8.1.1 *Tiered Approach to Risk Assessment*

The tiered approach for risk assessment that is presented in detail in the TSD (OEHHA, 2012) and summarized here should be reviewed prior to conducting the health risk assessment. The tiered approach to risk assessment and the health impacts evaluation described here are included in the HARP software.

The tiered approach provides a risk assessor with flexibility and allows consideration of site-specific differences (Table 8.1). The four-tiered approach to risk assessment is intended to primarily apply to residential cancer risk assessment, both for inhalation and noninhalation pathways. Risk assessors can tailor the level of effort and refinement of an HRA by using either the point estimate exposure assumptions as the basis of the exposure and risk assessment, or both the point estimate and a stochastic treatment of exposure factor distributions.

Table 8.1 The Tiered Approach to Risk Assessment

Tier	Description	When Applied
Tier 1	Utilizes OEHHA default point estimates of exposure variates	All risk assessments must include a Tier 1 assessment
Tier 2	Utilizes site-specific point estimates for exposure variates (justified, and approved by OEHHA)	A Tier 2 approach may be presented in addition to Tier 1
Tier 3	Utilizes OEHHA distributions of exposure variates	A Tier 3 approach may be presented in addition to Tier 1
Tier 4	Utilizes site-specific distributions of exposure variates (justified, and approved by OEHHA)	A Tier 4 approach may be presented in addition to Tier 1

Tier 1 is a standard point estimate approach that uses the recommended exposure variate (e.g., breathing or water ingestion rate) point estimates presented in this document. Derivations of these values are described in detail in OEHHA (2012). The results of the Tier 1 evaluations are required to be presented in the risk characterization section for all HRAs prepared for the Hot Spots Program. Thus, persons preparing an HRA using Tier 2 through Tier 4 evaluations must also include the risk characterization results of a Tier 1 evaluation in the HRA.

As discussed in OEHHA (2012), if the risk characterization results from a Tier 1 assessment are above a regulatory level of concern, the risk assessor may want to proceed with more site-specific analysis as described in Tier 2, or use a more resource-intensive stochastic modeling effort described in Tier 3 and Tier 4 (for cancer risk). While further evaluation may provide more information to the risk manager on which to base decisions, the Tier 1 evaluation is useful in comparing risks among a large number of facilities and must be included in all HRAs.

Tier 2 analysis allows the use of available and justifiable site-specific exposure variates (e.g., fish consumption), when presenting the potential health impacts. The site-specific information applied in a Tier 2 assessment must be adequately justified and approved by OEHHA and the District. In Tier 3, a stochastic approach to exposure assessment is taken using the distributions for the exposure pathways presented in the TSD (OEHHA, 2012) and in Chapter 5 of this Guidance Manual. The exposure distributions apply only to a residential receptor and are used only for the determination of cancer risk. OEHHA has not developed exposure intake distributions for workers to use in the offsite worker exposure scenario. Tier 4 is also a stochastic approach for the residential exposure scenario but allows for utilization of site-specific exposure variate distributions if they are justifiable and more appropriate for the site under evaluation than those derived in OEHHA (2012). Alternative site-specific distributions must be approved by OEHHA and the District. For an off-site worker cancer risk evaluation, Tiers 3 and 4 do not apply. Tier 3 and Tier 4 analyses show what a distribution of potential cancer risk may be to an individual or population based on a distribution of exposure inputs (e.g., water ingestion rate) rather than specific point estimates of exposure.

Table 8.2 summarizes OEHHA's recommendations for use of the four Tiers in cancer and noncancer risk assessment.

Table 8.2 Tiers for Residential and Offsite Worker Cancer and Noncancer Hot Spots Risk Assessments

Tier	Cancer		Non Cancer Chronic and 8-Hour	
	Inhalation	Noninhalation	Inhalation	Noninhalation
Tier-1	X	X	X	X
Tier-2	X	X		X[b]
Tier-3	X[a]	X[a]		
Tier-4	X[a]	X[a]		

[a] Applies to residential exposure scenario only
[b] Applies to chronic noncancer exposure only

OEHHA has not developed a stochastic approach (Tier 3 or 4) for estimating noncancer health impacts using acute, 8-hour, and chronic Reference Exposure Levels (RELs). Tier 1 is the only option for determining noncancer health impacts from inhalation exposure since calculating the hazard quotient involves dividing the ground level air concentrations for the specified exposure duration by the appropriate RELs. However, chronic noninhalation noncancer risks involve a calculation of dose from oral or dermal pathways to which site-specific evaluations could be considered under a Tier 2 approach.

Small foot-print facilities – Tier 2 or Tier 4

Some facilities subject to the Air Toxics Hot Spots Act (e.g., some in the industry-wide categories such as gas stations or dry cleaners) have very small zones of impact. In some of these instances, there will be very few receptors within the zone of impact. It isn't possible to develop special recommendations for exposure variates for all possible exposure scenarios. Alternative breathing rates (point estimates or distributions) may be used as part of Tier 2 or Tier 4 risk assessments with appropriate supporting justification in the case of a very small zone of impact. OEHHA is willing to work with risk managers at ARB and the Districts on this issue.

8.2 Risk Characterization for Carcinogens

Cancer risk is calculated by multiplying the daily inhalation or oral dose (calculated in Chapter 5), by a cancer potency factor, the age sensitivity factor, the frequency of time spent at home (for residents only), and the exposure duration divided by averaging time, to yield the excess cancer risk (see section 8.2.4). As described below, the excess cancer risk is calculated separately for each age grouping and then summed to yield cancer risk at the receptor location. A brief description of the age sensitivity factors, exposure duration, and frequency of time spent at home are included in Sections 8.2.1 to 8.2.3 below. These factors are discussed in detail in OEHHA (2009) and OEHHA (2012).

8.2.1 *Adjustment for Early Life Stage Exposures to Carcinogens*

Studies have shown that young animals are more sensitive than adult animals to exposure to many carcinogens (OEHHA, 2009). Therefore, OEHHA developed age sensitivity factors (ASFs) to take into account the increased sensitivity to carcinogens during early-in-life exposure (Table 8.3). These factors were developed and described in detail in OEHHA (2009). In the absence of chemical-specific data, OEHHA recommends a default ASF of 10 for the third trimester to age 2 years, and an ASF of 3 for ages 2 through 15 years to account for potential increased sensitivity to carcinogens during childhood.

Table 8.3 Age Sensitivity Factors by Age Group for Cancer Risk Assessment

Age Group	Age Sensitivity Factor (unitless)
3rd Trimester	10
0<2 years	10
2<9 years	3
2<16 years	3
16<30 years	1
16-70 years	1

For specific carcinogens where data indicate enhanced sensitivity during life stages other than the immediate postnatal and juvenile periods, or for which data demonstrate ASFs different from the default ASFs, the chemical-specific data should be used in order to adequately protect public health.

The risk assessments generated under the Air Toxics Hot Spots Act are reviewed by OEHHA. If a risk assessor had data indicating there are no windows of susceptibility early in life or that a different ASF should be used for a specific carcinogen and wanted to use these data, OEHHA would review the material as part of the review of the risk assessment.

8.2.2 *Fraction of Time Spent at Home for Cancer Risk Assessment*

OEHHA and ARB evaluated information from activity patterns databases to estimate the fraction of time at home (FAH) during the day (OEHHA, 2012). This information can be used to adjust exposure duration and cancer risk from a specific facility's emissions, based on the assumption that exposure to the facility's emissions are not occurring away from home. From the third trimester to age <2 years, 85% of time is spent at home (Table 8.4). From age 2 through <16 years, 72% of time is spent at home. From age 16 years and greater, 73% of time is spent at home. Facilities with any school within the 1×10^{-6} (or greater) isopleth should use FAH = 1 for the child age groups (3rd Trimester, 0<2 years, and 2<16 years). See Appendix I for an example calculation using the FAH.

Table 8.4 Recommendations for Fraction of Time at Home (FAH) for Evaluating Residential Cancer Risk

Age Range	Fraction of Time at Residence
3rd Trimester, and 0<2 years	0.85[1]
2<16 years[2]	0.72[1]
16-70 years[3]	0.73

[1] Use FAH = 1 if a school is within the 1×10^{-6} (or greater) cancer risk isopleth

[2] Also use FAH = 0.72 for 2<9 yr age group.
[3] Also use FAH = 0.73 for 16<30 yr age group.

The FAH is calculated based on a diary of trips taken over a 24-hour period on the survey day. Ninety-five percent of the diary days were on weekdays. Participants can select "vacation" as one of their trips. However, vacation time represented only a fraction (0.68%) of the over 175,000 trips recorded in the survey. Because much of these vacation trips were presumed to be within-day trips and were only a small fraction of total trips, there is likely little overlap with the Exposure Frequency (EF) variate used in the dose equations in Chapter 5.

8.2.3 *Exposure Duration for Estimating Cancer Risk to Residents and Off-Site Workers*

OEHHA recommends that an exposure duration (residency time) of 30 years be used to estimate individual cancer risk for the maximally exposed individual resident (MEIR) (Table 8.5). OEHHA also recommends that the 30-year exposure duration be used as the basis for public notification and risk reduction audits and plans. The Districts, however, may opt to use the 70 year cancer risk for notification and risk reduction audits and plans.

Note that the 30-year exposure duration starts in the third trimester to accommodate the increased susceptibility of exposures in early life (OEHHA, 2009), and would apply to both the point estimate and stochastic approaches.

Table 8.5 Summary of Recommendations for Exposure Duration for Individual Cancer Risk at the MEIR and MEIW

Receptor	***Recommendation***
Resident (MEIR)	30 years
Resident (supplemental Information)	9 years for central tendency; 70 years for maximum (lifetime)
Worker (MEIW)	25 years

Exposure durations of 9-years and 70-years are also recommended to be evaluated for the MEIR to show the range of cancer risk based on residency periods. If a facility is notifying the public regarding cancer risk, the 9- and 70-year cancer risk estimates are useful for people who have resided in their current residence for periods shorter and longer than 30 years.

The 9-, 30-, and 70-year exposures are chosen to coincide with U.S. EPA's estimates of the average (9 years), high-end estimates (30-years) of residence time, and a lifetime residency (70 years). These estimates are also consistent with what is known about residence time in California. Together, the 9-, 30-, and 70-year cancer risk calculations provide a useful presentation of cancer risk and the relationship to duration of residency and, thus, exposure to a facility's emissions.

For the maximally exposed individual worker (MEIW), OEHHA recommends using an exposure duration of 25 years to estimate individual cancer risk for the off-site worker scenario (Table 8.5). This duration represents approximately the 95th percentile of job tenure with the same employer in the U.S.

8.2.4 *Calculating Residential and Offsite Worker Inhalation Cancer Risk*

Residential Receptors

For residential inhalation exposure, cancer risk must be separately calculated for specified age groups (Eq. 8.2.4A, see Section 8.2.1), because of age differences in sensitivity to carcinogens and age differences in intake rates (per kg body weight). Separate risk estimates for these age groups provide a health-protective estimate of cancer risk by accounting for greater susceptibility in early life, including both age-related sensitivity and amount of exposure. The following equation illustrates the formula for calculating residential inhalation cancer risk. See Appendix I for a detailed example calculation.

A. Equation 8.2.4 A: $$RISK_{inh\text{-}res} = DOSE_{air} \times CPF \times ASF \times ED/AT \times FAH$$

7. $RISK_{inh\text{-}res}$ = Residential inhalation cancer risk
8. $DOSE_{air}$ = Daily inhalation dose (mg/kg-day)
9. CPF = Inhalation cancer potency factor ($mg/kg\text{-}day^{-1}$)
10. ASF = Age sensitivity factor for a specified age group (unitless)
11. ED = Exposure duration (in years) for a specified age group
12. AT = Averaging time for lifetime cancer risk (years)
13. FAH = Fraction of time spent at home (unitless)

a: Recommended default values for EQ 8.2.4 A:

5. $DOSE_{air}$ = Calculated for each age group from Eq. 5.4.1
6. CPF = Substance-specific (see Table 7.1)
7. ASF = See Section 8.2.1
8. ED = 0.25 years for 3rd trimester, 2 years for 0<2, 7 years for 2<9, 14 years for 2<16, 14 years for 16<30, 54 years for 16-70
9. AT = 70 years*
10. FAH = See Table 8.4

*Although AT actually sums to 70.25 years when the 3rd trimester (0.25 years) is included, OEHHA recommends rounding AT = 70 years (and rounding residential exposure durations at 9- and 30-years rather than 9.25- and 30.25-years) to simplify the calculation without causing a significant adjustment. Note that the dose for the 3rd trimester is based on the breathing rate of pregnant women using the assumption that the dose to the fetus during the 3rd trimester is the same as that to the mother.

Cancer risks calculated above for individual age groups are summed to estimate cancer risk for 9-, 30- and 70-year exposures as shown below. Note that this example includes the Fraction of Time Spent at Home (FAH) for each age grouping.

Calculation of Inhalation Cancer Risk from the Third Trimester to Age Nine:

$$RISK_{inh\text{-}res} = (DOSE_{air}\ third\ trimester \times CPF \times 10 \times 0.25/70\ years \times FAH_{3rd\ tri\ <2}) + (DOSE_{air}\ age\ 0<2 \times CPF \times 10 \times 2/70 \times FAH_{3rd\ tri\ <2}) + (DOSE_{air}\ age\ 2<9 \times CPF \times 3 \times 7/70\ years \times FAH_{2<9})$$

Calculation of Inhalation Cancer Risk from Third Trimester to Age 30:

$$RISK_{inh\text{-}res} = (DOSE_{air}\ third\ trimester \times CPF \times 10 \times 0.25/70\ years \times FAH_{3rd\ tri\ <2}) + (DOSE_{air}\ age\ 0<2 \times CPF \times 10 \times 2/70 \times FAH_{3rd\ tri\ <2}) + (DOSE_{air}\ age\ 2<16 \times CPF \times 3 \times 14/70 \times FAH_{2<16}) + (DOSE_{air}\ age\ 16<30 \times CPF \times 1 \times 14/70\ years \times FAH_{16\text{-}30})$$

Calculation of Inhalation Cancer Risk from Third Trimester to Age 70:

$$RISK_{inh\text{-}res} = (DOSE_{air}\ third\ trimester \times CPF \times 10 \times 0.25/70\ years \times FAH_{3rd\ tri\ <2}) + (DOSE_{air}\ age\ 0<2 \times CPF \times 10 \times 2/70 \times FAH_{3rd\ tri\ <2}) + (DOSE_{air}\ age\ 2<16 \times CPF \times 3 \times 14/70 \times FAH_{2<16}) + (DOSE_{air}\ age\ 16<70 \times CPF \times 1 \times 54/70\ years \times FAH_{16\text{-}70})$$

Expressing cancer risk in "chances per million" is useful as a risk communication tool for the public, but cancer risk can also be expressed in other ways, such as "chances per 100,000" (cancer risk × 10^5) or "chances per 10 million" (cancer risk × 10^7). To convert the resulting cancer risk estimate to chances of developing cancer per million individuals exposed, multiply the cancer risk by 10^6:

$$Cancer\ risk \times 10^6 = chances\ per\ million$$

For exposure to multiple carcinogenic substances, Table 8.7 and Table I.5 in Appendix I are examples of how cancer risks of individual substances are summed to determine the total cancer risk.

Worker Receptors

For assessment of off-site worker cancer risk at the MEIW, the default assumes working age begins at 16 years. Note that the residential FAH factor in Eq. 8.2.4.A above does not apply for workers. The daily inhalation dose ($DOSE_{air}$) (as calculated in Chapter 5, EQ 5.4.1.2) is based on the adjusted 8-hour concentration at the MEIW (for non-continuous sources) and amount of time the offsite worker's schedule overlaps with the facility's emission schedule. The duration of exposure at the MEIW receptor is 25 years, as discussed in the TSD (OEHHA, 2012).

B. Equation 8.2.4 B: $RISK_{inh\text{-}work} = DOSE_{air} \times CPF \times ASF \times ED/AT$

1. $RISK_{inh\text{-}work}$ = Worker inhalation cancer risk

a: Recommended default values for EQ 8.2.4 B:

1. $DOSE_{air}$ = Calculated for workers in Eq. 5.4.1.2
2. CPF = Substance specific (see Table 7.1)
3. ASF = 1 for working age 16-70 yrs (See Section 8.2.1)
4. ED = 25 years
5. AT = 70 yrs for lifetime cancer risk

Work Locations with Daycare Facilities:

An additional risk management consideration for offsite worker cancer risk assessment of a Hot Spots facility is whether there are women of child bearing age at the MEIW location and whether the MEIW has a daycare center. In the case of women of child-bearing age at the MEIW, the Districts may wish to treat the off-site MEIW in the same way as the residential scenario to account for the higher susceptibility during the third trimester of pregnancy (i.e., use of an ASF=10 for third trimester exposure). If there is onsite daycare at the MEIW, then the risks to the children will be underestimated using the offsite adult worker scenario. In this case, the Districts may wish to include a cancer risk assessment for the children in the onsite daycare, assuming they could be there from 0 to age 6 years (ED = 6 years) and using the appropriate exposure factors to calculate $DOSE_{air}$, fraction of time at worksite (e.g., hrs at daycare per 24 hrs), and ASFs in EQ 8.2.4 B to account for the higher susceptibility of infants and children to carcinogens.

Children at a MEIW daycare may also be assessed for noninhalation exposures. Typically, soil ingestion and dermal exposure will be the most common noninhalation pathways. However, all pathways that are present at the daycare should be included. See section 8.2.6 for more discussion of multipathway risk assessment methods.

8.2.5 *Calculation of Noninhalation Cancer Risk*

A small subset of Hot Spots substances is subject to deposition onto the soil, plants, and water bodies (see Table 5.1). These substances need to be evaluated by the appropriate noninhalation pathways, as well as by the inhalation pathway, and the risk characterization results must be presented in all HRAs. These substances include semi-volatile organic chemicals and heavy metals.

For all multipathway substances, the exposure pathways that must be evaluated at every residential and worker site (in addition to inhalation) are soil ingestion and dermal exposure. If PAHs (and creosotes), lead, dioxins, furans, or PCBs are emitted, then the breast-milk consumption pathway becomes mandatory for residential receptors. OEHHA has developed transfer coefficients for these chemicals from the mother to breast milk (see OEHHA, 2012 for details). The other exposure pathways (e.g.,

ingestion of homegrown produce or fish) are only evaluated for residential receptors if the facility impacts that exposure medium and the receptor under evaluation can be exposed to that medium or pathway. For example, if the facility does not impact a fishable body of water within the isopleth of the facility, or the impacted water body does not sustain fish that are consumed by fishers, then the fish pathway will not be considered for that facility or receptor.

Table 8.6 identifies the residential receptor exposure pathways that are mandatory and those that are dependent on the available routes of exposure. Table 8.6 also identifies the three exposure pathways that are relevant for a worker receptor. The cancer risk estimates should be presented in the risk characterization section of the risk assessment for all the appropriate pathways.

Table 8.6 Mandatory and Site/Route Dependent Exposure Pathways

Mandatory Exposure Pathways	Site/Route Dependent Exposure Pathways
• Inhalation[w] • Soil Ingestion[w] • Dermal Exposure to Contaminated Soil[w] • Breast Milk Consumption[*]	• Homegrown Produce Ingestion • Angler-Caught Fish Ingestion • Drinking Water Ingestion • Home-Raised Animal Product Ingestion (Dairy (Cow's) Milk, Meat (Beef, Pork, Chicken) and Egg).

(w) Identifies the appropriate exposure pathways that should be evaluated for a worker. These pathways are inhalation, dermal exposure, and the soil ingestion pathway.

(*) If PAHs (including creosotes), lead, dioxins, furans, or PCBs are emitted, then the breast-milk consumption pathway becomes mandatory.

The noninhalation residential cancer risk is calculated using the same steps as inhalation cancer risk described in Section 8.2.4. A dose (see Chapters 4 and 5) from the pathway under evaluation (e.g., soil ingestion) is multiplied by the substance-specific oral slope factor, expressed in units of inverse dose (i.e., $(mg/kg/day)^{-1}$) (Table 7.1), the appropriate age sensitivity factor (ASF), and exposure duration divided by averaging time to yield the cancer risk for a specified age grouping. Cancer risk for each age group is summed as appropriate for the exposure duration. The FAH factor is relevant only to the inhalation pathway and is not appropriate to use in the noninhalation pathways.

Equation 8.2.5 illustrates the formula for calculating noninhalation cancer risk. Details (data, algorithms, and guidance) for each exposure pathway are presented in Chapter 5 and in OEHHA (2012).

A. Equation 8.2.5: $RISK_{noninh} = DOSE_{noninh} \times CPF_{oral} \times ASF \times ED/AT$

1. $RISK_{noninh}$ = Noninhalation pathway cancer risk
2. $DOSE_{noninh}$ = Daily dose (mg/kg-day) for a specified non-inhalation pathway for each age group
3. CPF_{oral} = Oral cancer potency (slope) factor (mg/kg-day^{-1})
4. ASF = Age sensitivity factor for a specified age group (unitless)
5. ED = Exposure duration (in years) for a specified age group
6. AT = Averaging time for lifetime cancer risk

a: Recommended default values for EQ 8.2.5:

1. $DOSE_{noninh}$ = Calculated in Chapter 5 dose algorithms for each age group and for each noninhalation route in Table 8.6 the receptor is exposed to
2. CPF_{oral} = Substance-specific (see Table 7.1)
3. ASF = See Section 8.2.1
4. ED = Residents: 0.25 years for 3rd trimester, 2 years for 0<2, 7 years for 2<9, 14 years for 2<16, 14 years for 16<30, 54 years for 16-70
   = Offsite worker: 25 yrs
5. AT = 70 years

Estimating cancer risk for 9-, 30- and 70-years by summing the individual age-group cancer risks is the same as that shown for the inhalation route in Section 8.2.4. The exception is that the FAH variate is only appropriate for the residential inhalation pathway and is not a factor for oral and dermal exposure pathways.

Calculation of Noninhalation Cancer Risk from Third Trimester to Age 30:

$RISK_{noninh\text{-}res}$ = ($DOSE_{noninh}$ third trimester × CPF × 10 × 0.25/70 years) + ($DOSE_{noninh}$ age 0<2 × CPF × 10 × 2/70) + ($DOSE_{noninh}$ age 2<16 × CPF × 3 × 14/70) + ($DOSE_{noninh}$ age 16<30 × CPF × 1 × 14/70 years)

To convert this estimated probability of risk to chances per million of developing cancer, multiply the estimated cancer risk for each noninhalation exposure route by 10^6. This result is useful communication tool to compare risks for each pathway of exposure.

$$\text{Cancer risk} \times 10^6 = \text{cancer risk expressed as chances per million}$$

For assessment of the offsite worker the typical noninhalation pathways that apply for worker cancer risk are the dermal exposure pathway and the soil ingestion pathway.

Children at a MEIW daycare may also be assessed for noninhalation exposures. Typically, soil ingestion and dermal exposure will be the most common noninhalation pathways. However, all pathways that are present at the daycare should be included.

8.2.6 *Multipathway Cancer Risk Methodology*

Under a Tier 1 assessment, it is necessary to calculate the total cancer risk from both inhalation and noninhalation exposures if multipathway substances are emitted from the facility. The calculation of cancer risk that includes exposure to a multipathway substance or substances has three steps:

1) Calculate cancer risk for the inhalation pathway (EQ 8.2.4 A for residents, EQ 8.2.4 B for off-site workers) for all substances, and the noninhalation pathways that apply (EQ 8.2.5) for all multipathway substances, using high-end point estimates of intake rates.
2) For each multipathway substance, identify the two exposure pathways with the highest risk. These are the dominant pathways that are to be assessed using high-end point estimates of intake rates for the total cancer risk. For all other pathways, the average point estimate of intake rates may be used to calculate the pathway cancer risk (See OEHHA (2012) for more information).
3) To calculate total cancer risk, all inhalation and noninhalation pathways are summed together for all substances.

The final cancer risk calculation using a combination of high-end and average exposure parameters is referred to as the derived risk in the HARP software. This is described in Chapter 1, Section 1.4.1 of OEHHA (2012). The inhalation route is almost always one of the two dominant pathways in a multipathway cancer risk assessment. Therefore, in most cases only one noninhalation pathway would be calculated using a high-end dose point estimate. For all other pathways, the average point estimate may be used to calculate the pathway cancer risk.

For example, if dermal exposure and soil ingestion risks are calculated, then the cancer risks from these pathways would be summed along with the inhalation cancer risks to give the total cancer risk for the single multipathway substance:

Cancer Risk (inhalation) + Cancer Risk (dermal) + Cancer Risk (soil) = Total Risk

The mother's milk pathway also becomes a mandatory pathway to assess risk in nursing infants if the mother is exposed to specific substances (see Table 5.1).

Many facilities will emit multiple carcinogenic substances. If multiple substances are emitted, the substance-specific cancer risks for all exposure pathways are summed to give the (total) multipathway cancer risk at the receptor location. The HARP software will display not only the multipathway risk for each carcinogenic substance, but also show a breakdown of the cancer risk from each exposure pathway. Table 8.7 shows the results of a multipathway risk assessment for a hypothetical facility. While not presented in the following table, it is critical to identify the driving exposure pathways and the driving substances in a multipathway cancer risk assessment when summarizing and presenting the HRA results. See Chapter 9 for more information.

Table 8.7 Multipathway Assessment of a Hypothetical Facility 30-Year Cancer Risk

Substance	Cancer Risk[a]	Cancer risk[b] (chances per million)
Arsenic	1.1×10^{-5} (i) 3×10^{-7} (ni)	11 (i) 0.3 (ni)
Benzene	2.92×10^{-4} (i)	292 (i)
2,3,7,8-TCDD (dioxin)	1.06×10^{-4} (i) 5.7×10^{-5} (ni)	106 (i) 57 (ni)
1,3-Butadiene	6.0×10^{-6} (i)	6 (i)
Total Facility Cancer Risk	**4.723×10^{-4}**	**472**

[a] As calculated in EQ 8.2.4 A or EQ 8.2.5
[b] Calculated as: cancer risk × 10^6 = chances per million
i = inhalation pathway contribution
ni = noninhalation pathway contribution

Cancer risk in Table 8.7 for the multipathway substances, arsenic and 2,3,7,8-TCDD, is arranged by the inhalation pathway risk and the sum of all noninhalation pathway risks. The total facility multipathway cancer risk is the sum of all inhalation and noninhalation pathways.

Cancer risks from different substances are treated additively in risk assessment generally, and in the Hot Spots Program in part because many carcinogens act through the common mechanism of DNA damage. The additive assumption is reasonable from a public health point of view. Other possible interactions of multiple carcinogens include synergism (effects are greater than additive) or antagonism (effects are less than additive). The type of interaction is both chemical and dose dependent and in most cases the data are not available to adequately characterize these interactions.

8.2.7 *Multipathway Cancer Risk for Infant Exposure to Mother's Milk*

The mother' milk pathway becomes mandatory if the nursing mother is exposed to one or more of the following multipathway substances: dioxins and furans, PCBs, PAHs including creosotes, and lead. The default assumption inherent in the intake rate is that the infant's only source of food is breast for the first year (e.g., is fully breastfed, see OEHHA, 2012, for details), which is one-half of the 0<2 year age group used in the Hot Spots program. Thus, the cancer risk by the mother's milk pathway will need to be calculated with a modified cancer risk equation using a different exposure duration:

A. Equation 8.2.7: $\mathbf{RISK_{mm} =}$ Dose-Im $\mathbf{\times CPF_{oral} \times ASF \times ED/AT}$

1. $RISK_{mm}$ = Infant cancer risk via mother's milk pathway
2. Dose-Im = Daily dose (mg/kg-day) to infant from mother's milk
3. CPF_{oral} = Oral cancer slope factor (mg/kg-day^{-1})
4. ASF = Age sensitivity factor for infant (unitless)
5. ED = Exposure duration (in years) for infant
6. AT = Averaging time for lifetime cancer risk

a: Recommended default values for EQ 8.2.7:

6. Dose-Im = Calculated from EQ 5.4.3.5.2, dose to infant via mother's milk
7. CPF_{oral} = Substance-specific (see Table 7.1)
8. ASF = 10 (See Section 8.2.1)
9. ED = 1 yr (1st yr of 0<2 yr age group)
10. AT = 70 years

Once the cancer risk is determined for the mother's milk pathway for each applicable substance, the pathway risk is summed with other pathway risks.

For Tier 1, the derived approach for cancer risk assessment should be used if the mother's milk pathway applies. As outlined in Section 8.2.6, the two dominant pathways will be calculated using high-end point estimates of intake rates; all additional pathways may be calculated using average point estimates of intake rates. There will be four mandatory pathways to assess (inhalation, mother's milk, soil ingestion and dermal exposure) for cancer risk when exposure to dioxins/furans, PCBs, PAHs including creosotes, and/or lead occurs. Therefore, if the infant is exposed to no other additional site-specific noninhalation pathway(s), only the two dominant pathways among the four will be assessed for cancer risk using high-end point estimates of intake rates; and the others would be assessed using the average point estimate of intake rate.

In short, multipathway cancer risk for a substance is estimated by summing the potential inhalation and noninhalation cancer risks for the receptor location of interest. See the discussion of Tier 1 in Section 8.2.6 or the TSD for more information on the method used to determine the multipathway cancer risk.

8.2.8 *Cancer Risk Characterization for Stochastic Risk Assessment*

Risk characterization for a stochastic risk assessment is similar to that described for the point-estimate approach. However, the stochastic risk assessment produces a distribution of risk that accounts for some of the natural variability in exposure-related factors, such as breathing rates or water intake. The cancer risk distribution for inhalation cancer risk, for example, is generated by multiplying randomly selected values from the breathing rate distribution by the ground level air concentration, and the cancer potency factor. A variation of the Monte Carlo method called Latin hypercube sampling is the method by which the values from the breathing rate distribution are

selected. If noninhalation pathways need to be evaluated, the same process is followed for each pathway and the risk is summed to give an overall inhalation and noninhalation cancer risk distribution. Further, the specification of Age Sensitivity Factors and the need to separately calculate risks require that a Monte Carlo sampling be conducted for each age group and the cancer risk distributions are then summed across age groups.

The HARP software will perform an HRA using a Monte Carlo analysis with either OEHHA-provided or user-provided data distributions and will include the statistics for the distributions. In risk assessments that have chosen to use the distribution of exposure variates, the cancer risk distribution for a 30-year residential exposure duration (MEIR) should be presented in the risk characterization section We also recommend including the 9 and 70-year cancer risk at the MEIR as supplemental information. Note that a 70-year exposure duration is required to estimate cancer burden or provide an estimate of population-wide risk. A stochastic approach has not been developed for acute, 8-hour, and chronic noncancer health impacts or worker (MEIW) exposures.

8.2.9 *Use of Individual Cancer Risk and Population-wide Cancer Risk*

Cancer risk for an individual receptor and a representation of population-wide cancer risk are both important components of a risk assessment. The individual receptor approach reflects the exposures that may occur to an individual receptor over a period of time at a specific location. The individual cancer risk approach has some inherent limitations in terms of illustrating and potentially protecting population-based public health. For example, a facility with a small emissions footprint may impact a few individuals with a high individual potential cancer risk; whereas, a facility with a larger emission footprint may have a lower potential cancer risk for an individual receptor but expose many more people to those levels. Since this larger emitting facility can impact many more people, the population-wide health impacts are magnified due to the larger number of people exposed to the facility's emissions. This potential for higher population impacts is not captured by the individual receptor risk methodology. Therefore, the individual and population-wide heath impacts should be presented for all facilities to provide a more complete illustration of the facility's health impacts.

8.2.9.1 Population Risk

For facilities with large emission footprints (e.g., refineries, ports, or rail yards, etc.), population-based health impacts are critical to provide a better illustration of the potential impacts of emissions since large numbers of people may be exposed to the emissions. The individual cancer risk approach has some inherent limitations in terms of protecting public health. A small facility with a single stack can impact a few individuals with an individual cancer risk that is unacceptable, whereas a large facility may have an individual cancer risk that is below the acceptable limit for individual risk but exposes many more people. Thus, the population-wide impacts are larger for the large facility. Population-wide risk is independent of individual risk, and assumes that a population (not necessarily the same individuals) will live in the impacted zone over a

70-year period. Thus, a 70-year exposure duration is required for estimates of population-wide risks.

To evaluate population risk, one method that regulatory agencies have used is the cancer burden method to account for the number of excess cancer cases that could occur in a population.

Cancer Burden

The cancer burden can be calculated by multiplying the cancer risk at a census block centroid by the number of people who live in the census block, and adding up the estimated number of potential cancer cases across the zone of impact. The result of this calculation is a single number that is intended to estimate of the number of potential cancer cases within the population that was exposed to the emissions for a lifetime (70 years).

The cancer burden is calculated on the basis of lifetime (70-year) risks (whereas individual cancer risk at the MEIR is based on 30-year residential exposure). Cancer burden is independent of how many people move in or out of the vicinity of an individual facility. For example, if 10,000 people are exposed to a carcinogen at a concentration with a 1×10^{-5} cancer risk for a lifetime the cancer burden is 0.1, and if 100,000 people are exposed to a 1×10^{-5} risk the cancer burden is 1.

Estimate of Population Wide Risk

An estimate of the number of people exposed at various cancer risk levels can provide perspective on the magnitude of the potential public health threat posed by a facility. This approach is intended as a replacement for or addition to the cancer burden calculation used by some Districts in the past. The new approach provides a much easier way for the general public to interpret results when compared to cancer burden estimates. A facility in a sparsely populated area can have a public health impact different from the same facility in a highly populated area; however, under the cancer burden method, those differences may not be seen. Some suggested approaches and methods for performance of a screening or refined population exposure analyses are provided in Section 4.6.

The District or reviewing authority should be consulted before beginning the population exposure estimates and, as results are generated, further consultation may be necessary. Note that a 70-year exposure duration is required to estimate cancer burden or provide an estimate of population-wide risk.

The zone of impact for estimating the number of persons exposed to a cancer risk from facility emissions should be set at a minimum of a 10^{-6} cancer risk level (see Section 4.6.1). Some Districts may prefer to use a cancer risk of 10^{-7} to define the carcinogenic zone of impact. The total number of persons exposed to a series of potential risk levels can be presented to aid risk managers in understanding the magnitude of the potential public health impacts.

The HARP software can provide population-level risk estimates as cancer burden or as the number of persons exposed to a selected (user-identified) cancer risk level at block level centroids.

8.2.9.2 Population Estimates for Noncancer Health Impacts

A noncancer chronic, 8-hour, and acute population estimate of the number of people exposed to acute, 8-hour, and chronic HQs or HIs exceeding 0.5 or 1.0, in increments of 1.0, should also be presented. For example, a facility with a maximum chronic HI of 4.0 would present the number of people exposed to a chronic HI of 0.5, 1.0, 2.0, 3.0, and 4.0. The isopleths used in this determination should be drawn using the smallest feasible grid size. The same methods that are described in Chapter 4 and Section 8.2.9 (for the population exposure estimate for cancer risk) should be used in the chronic, 8-hour and acute population estimates. Population estimates for acute, 8-hour, and chronic health impacts should be presented separately.

8.2.9.3 Factors That Can Impact Population Risk – Cumulative Impacts

Although the Hot Spots program is designed to address the impacts of single facilities and not aggregate or cumulative impacts, there are a number of known factors that influence the susceptibility of the exposed population and thus may influence population risk. Socioeconomic status influences access to health care, nutrition, and outcome after cancer diagnosis. Community unemployment can affect exposure and residency time near a facility. Factors that affect the vulnerability of the population are discussed in the report *Cumulative Impacts: Building a Scientific Foundation* (OEHHA, 2010). Information on many of these factors is relatively easy to obtain at the census tract level. The OEHHA recommends that these types of factors be considered by the risk manager, along with the quantitative measures of population risk. OEHHA is in the process of developing guidance on quantification of the impact of these factors.

8.2.10 *Cancer Risk Evaluation of Short Term Projects*

The local air pollution control districts sometimes use the risk assessment guidelines for the Hot Spots program in permitting decisions for short-term projects such as construction or waste site remediation. Frequently, the issue of how to address cancer risks from short-term projects arises.

Cancer potency factors are based on animal lifetime studies or worker studies where there is long-term exposure to the carcinogenic agent. There is considerable uncertainty in trying to evaluate the cancer risk from projects that will only last a small fraction of a lifetime. There are some studies indicating that dose rate changes the potency of a given dose of a carcinogenic chemical. In others words, a dose delivered over a short time period may have a different potency than the same dose delivered over a lifetime.

The OEHHA's evaluation of the impact of early-in-life exposure has reduced some of the uncertainty in evaluating the cancer risk to the general population for shorter-term exposures, as it helps account for susceptibility to carcinogens by age at exposure (OEHHA, 2009).

Due to the uncertainty in assessing cancer risk from very short-term exposures, we do not recommend assessing cancer risk for projects lasting less than two months at the MEIR. We recommend that exposure from projects longer than 2 months but less than 6 months be assumed to last 6 months (e.g., a 2-month project would be evaluated as if it lasted 6 months). Exposure from projects lasting more than 6 months should be evaluated for the duration of the project. In all cases, for assessing risk to residential receptors, the exposure should be assumed to start in the third trimester to allow for the use of the ASFs (OEHHA, 2009). Thus, for example, if the District is evaluating a proposed 5-year mitigation project at a hazardous waste site, the cancer risks for the residents would be calculated based on exposures starting in the third trimester through the first five years of life.

For the MEIW, we recommend using the same minimum exposure requirements used for the residential receptor (i.e., no evaluation for projects less than 2 months; projects longer than 2 months but less than 6 months are assumed to last 6 months; projects longer than 6 months would be evaluated for the duration of the project). Although the off-site worker scenario assumes that the workers are 16 years of age or older with an Age-Sensitivity Factor of 1, another risk management consideration for short-term project cancer assessment is whether there are women of child bearing age at the worksite and whether the MEIW receptor has a daycare center. In this case, the Districts may wish to treat the off-site MEIW in the same way as the residential scenario to account for the higher susceptibility during the third trimester of pregnancy, and for higher susceptibility of infants and children.

Finally, the risk manager may want to consider a lower cancer risk threshold for risk management for very short-term projects. Typical District guidelines for evaluating risk management of Hot Spots facilities range around a cancer risk of 1 per 100,000 exposed persons as a trigger for risk management. Permitting thresholds also vary for each District. There is valid scientific concern that the rate of exposure may influence the risk – in other words, a higher exposure to a carcinogen over a short period of time may be a greater risk than the same total exposure spread over a much longer time period. In addition, it is inappropriate from a public health perspective to allow a lifetime acceptable risk to accrue in a short period of time (e.g., a very high exposure to a carcinogen over a short period of time resulting in a 1×10^{-5} cancer risk). Thus, consideration should be given for very short term projects to using a lower cancer risk trigger for permitting decisions.

8.3 Noncancer Acute, 8-Hour, and Chronic Inhalation Health Impacts – the Hazard Index Approach

All substances in the Hot Spots Program that have noncancer health impacts at a receptor must be evaluated through the inhalation pathway. Estimates of noncancer inhalation health impacts are determined by dividing an airborne concentration at the receptor by the appropriate Reference Exposure Level (REL). This is termed the Hazard Index Approach. A REL is used as an indicator of potential noncancer health impacts and is defined as the concentration at which no adverse noncancer health effects are anticipated. When a health impact calculation is performed for a single substance, then it is called the hazard quotient (HQ). Each REL for a substance will have one or more target organ systems (e.g., respiratory system, nervous system, etc.) where the substance can have a noncancer health impact. Thus, all HQs have specified target organ systems associated with them. The sum of the Hazard Quotients of all chemicals emitted that impact the same target organ is termed the Hazard Index. Inhalation RELs for noncancer health impacts have been developed for acute, 8-hour, and chronic exposures to a number of Hot Spots substances. Acute RELs are designed to protect against the maximum 1-hour ground level concentration at the receptor. Eight-hour RELs are designed to protect people with daily 8-hour schedules, such as offsite workers, in an impacted zone. The 8-hour RELs should be used for typical daily work shifts of 8-9 hours. For further questions, assessors should contact OEHHA, the District, or reviewing authority to determine if the 8-hour RELs should be used in your HRA. Any discussions or directions to exclude the 8-hour REL evaluation should be documented in the HRA. Chronic RELs protect against long-term exposure to the annual average air concentration spread over 24 hours/day, 7 days/week.

OEHHA has added 8-hour RELs to the set of noncancer RELs that were previously comprised of acute and chronic RELs (OEHHA, 2008). Specifically, 8-hour RELs are air concentrations at or below which health impacts would not be expected even for sensitive subpopulations in the general population with repeated daily 8-hour exposures over a significant fraction of a lifetime. The 8-hour RELs can be used to evaluate the potential for health impacts (including effects of repeated exposures) in offsite workers, and to children and teachers exposed during school hours. Although not required in the HRA, they could also be applied by the Districts to a residential scenario where a facility operates only a portion of the day and exposure to residences is not adequately reflected by averaging concentrations over a 24 hour day. The number of chemicals with 8-hour RELs will increase as OEHHA re-evaluates RELs for chemicals under SB-25 to ensure that they are protective of children's health.

Acute, 8-hour, and chronic RELs are needed because the dose metrics and even the health impact endpoints may be different with the different exposure durations of acute, daily 8-hour, and chronic exposures. Also, although chronic REL values are lower or set the same as 8-hour RELs, there are some cases such as special meteorological situations (e.g., significant diurnal-nocturnal meteorological differences) or intermittent exposures where the 8-hour REL may be more protective than the chronic REL.

Chapter 4 describes air dispersion modeling and both Chapter 6 and Appendix L list the needed dose-response information to evaluate non-cancer hazards. Appendix I presents sample calculations for determining acute HQs and HIs, 8-hour HQs and HIs, and chronic multipathway HQs and HIs. Chapter 9 provides an outline of information required for risk characterization. The HARP software will calculate the HQ and HI for Hot Spots risk assessments.

8.3.1 *Calculation of Noncancer Inhalation Hazard Quotient and Hazard Index*

To calculate the acute HQ, the maximum 1-hour ground level concentration (in $\mu g/m^3$) of a substance at a receptor is divided by the acute 1-hour REL (in $\mu g/m^3$) for the substance:

$$\text{Acute Hazard Quotient} = \frac{\text{1-Hour Max Concentration } (\mu g/m^3)}{\text{Acute REL } (\mu g/m^3)}$$

To calculate the chronic HQ, the annual average ground level concentration of a substance is divided by the chronic REL for the substance:

$$\text{Chronic Hazard Quotient} = \frac{\text{Annual Average Concentration } (\mu g/m^3)}{\text{Chronic REL } (\mu g/m^3)}$$

To calculate the 8-hour HQ, the adjusted annual average ground level concentration of a substance (represented as "Adjusted C_{air}" in EQ 5.4.1.4 A) is divided by the 8-hour REL for the substance:

$$\text{8-hour Hazard Quotient} = \frac{\text{Adjusted Annual Average Concentration } (\mu g/m^3)}{\text{8-hour REL } (\mu g/m^3)}$$

The daily 8-hour average ground level concentrations used for calculating the 8-hour HQs are derived as described in Chapter 4.

An HQ of 1.0 or less indicates that adverse health effects are not expected to result from exposure to emissions of that substance. As the HQ increases above one, the probability of human health effects increases by an undefined amount. However, it should be noted that a HQ above one is not necessarily indicative of health impacts due to the application of uncertainty factors in deriving the RELs.

If a receptor is exposed to multiple substances that target the same organ system, then the HQs for the individual substances are summed to obtain a Hazard Index (HI) for that target organ.

Table 8.8 is an example of an HRA spreadsheet showing acute inhalation HQs arranged by target organ system for several substances. The bottom row shows the summed HQs by target organ system to derive the HIs.

Table 8.8 Individual Hazard Quotients and Total Hazard Index for Acute Inhalation Exposure

Substance	Reproductive/ Developmental	Nervous System	Cardiovascular System	Respiratory System	Eye
Ammonia				0.6	0.6
Arsenic	0.2	0.2	0.2		
Benzene	0.02				
Chlorine				0.7	0.7
Total Hazard Index	**0.22**	**0.2**	**0.2**	**1.3**	**1.3**

A more detailed example of calculating HQs and HIs and of determining noncancer health impacts is shown in Appendix I.

Hazard quotients or HIs for different target organs are not summed together (e.g., do not add the impacts for the eye to the cardiovascular system). Chapter 6 and Appendix L have lists of the organ systems affected by each substance. Unlike the cancer risk algorithms, no exposure duration adjustment (e.g., 9 yrs / 70 yrs) should be made for noncancer assessments.

There are limitations to this method of assessing cumulative noncancer health impacts. The impact on organ systems may not be additive if health effects occur by different mechanisms. However, the impact on organ systems could also be synergistic. An analysis by a trained health professional familiar with the substance's toxicological literature is usually needed to determine the public health significance of an HQ or HI above one. It is recommended that the Air District contact OEHHA if this situation presents itself. For assessing the noncancer health impacts of lead, different procedures are used; please see Appendix F.

8.3.2 *Calculating Noninhalation (oral) Noncancer Hazard Quotient and Hazard Index*

Similar to the situation with multipathway carcinogenic substances, multipathway substances that present a noncancer hazard are assessed by noninhalation routes of exposure (see Table 8.6). Noninhalation routes of exposure are assessed only for chronic exposure. There are no oral acute RELs since it is generally anticipated that health effects from a single exposure via the oral route at typical environmental levels resulting from deposition of facility emissions would be insignificant relative to the inhalation route. The multipathway substances with noninhalation RELs, called chronic oral RELs, are shown in Table 6.4. Similar to inhalation exposure, the hazard quotient

for a noninhalation pathway is obtained by dividing the dose in milligrams per kilogram-day (mg/kg-day) by the oral REL also expressed in units of mg/kg-day:

$$\text{Chronic Non-inhalation HQ} = \frac{\text{Chronic Noninhalation Dose (mg/kg-day)}}{\text{Chronic Oral REL (mg/kg-day)}}$$

The calculated chronic oral HQs are combined with the chronic inhalation HQs for determining the chronic HIs for each affected target organ (see Section 8.3.4). The point estimates and algorithms for calculating the oral dose for all applicable exposure pathways and receptors (e.g., workers or residents) are explained in Chapter 5.

The chronic oral dose calculated in mg/kg-day is based on a time-weighted average 70-year residential exposure combining the 0<2, 2<16 and 16-70 year age groups. Unlike the assessment of cancer risk, no exposure duration adjustment should be made when estimating HQs. In other words, the variates ED and AT in the cancer risk EQ 8.2.5 in Section 8.2.5 are not used for estimating the noncancer HQs. See Appendix I for an example calculation.

8.3.3 *Multipathway Noncancer Risk Methodology*

To determine multipathway chronic noncancer health impacts, it is necessary to calculate the total hazard index from both inhalation and noninhalation exposures. The calculation of HIs has several steps:

1) First, the inhalation HQ is calculated for each substance emitted (Section 8.3.1).
2) Second, if the substance has an oral REL, then the non-inhalation HQ is calculated as shown above using high-end point-estimates for intake rates for each noninhalation pathway that applies.
3) Third, if there are more than two noninhalation pathways to consider for a multipathway substance, then the oral HQ is calculated using high-end point estimates in the dose equation for the two dominant pathways. For any additional noninhalation pathways, the HQs are calculated using average point estimates in the dose equation. This step applies only to residential receptors.
4) Fourth, all noninhalation pathway HQs for a multipathway substance are then summed together by target organ to obtain the total noninhalation HQ for a multipathway substance.
5) The final step is to sum the inhalation and noninhalation HQs together by target organ to determine the HIs. This step is displayed in Table 8.9. If there is only one substance, then the multipathway HQ is the same as the HI.

Table 8.9 Substance-Specific Chronic Inhalation and Noninhalation Hazard Quotients and the Hazard Index by Target Organ System

Substance	Respiratory System	Hematologic System	Alimentary System	Endocrine System	Development	Reproductive System	Nervous System	Cardiovascular System	Skin
Ammonia	0.8								
Arsenic					0.04(i) 0.1(ni)		0.04(i) 0.1(ni)	0.04(i) 0.1(ni)	0.04(i) 0.1(ni)
Benzene		0.08			0.08		0.08		
2,3,7,8-TCDD (dioxin)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)			
Nickel	0.4(i)	0.4(i)	0.1(ni)						
Hazard Index	**1.50**	**0.78**	**0.40**	**0.3**	**0.52**	**0.30**	**0.22**	**0.14**	**0.14**

i = inhalation pathway contribution

ni = noninhalation pathway contribution

Table 8.9 shows the calculated chronic HIs by combining the chronic inhalation HQs and chronic oral HQs. The HQs or HIs for different target organs are not added together (e.g., do not add the impacts for the respiratory system to the nervous system). The noninhalation pathways for TCDD and arsenic in Table 8.9 have all the noninhalation pathways that apply incorporated into their HQ values. For example, the noninhalation value for arsenic (HQs = 0.1) includes at least the soil ingestion and dermal soil pathways in the HQs because these are the mandatory noninhalation pathways to take into account with exposure to a multipathway substance. For TCDD, the mother's milk pathway is an additional mandatory noninhalation pathway to take into account (See Table 5.1). If there are exposures to any of the site-specific pathways, then these would be included too. A more detailed example calculation of HIs is shown in Appendix I.

When exposure to more than two noninhalation pathways occur, using the high-end point estimates of intake rates for only the two dominant noninhalation pathways will lessen the issue of compounding high-end exposure estimates, while retaining a health-protective approach for the more important exposure pathways. It is unlikely that an individual receptor would be on the high-end of exposure for all the non-inhalation intake parameters (exposure pathways).

8.3.4 *Summary - Acute, 8-Hour and Chronic Hazard Index Calculation at the MEIR and MEIW*

Eight-hour RELs were developed principally for exposure of individuals during 8-hour work schedules. The 8-hour RELs should be used for typical daily work shifts of 8-9 hours. For further questions, assessors should contact OEHHA, the District, or reviewing authority to determine if the 8-hour RELs should be used in your HRA. Any discussions or directions to exclude the 8-hour REL evaluation should be documented in the HRA. There are currently only a limited number of substances with an 8-hour inhalation REL. Over time as the science supporting REL values for individual substances is reviewed and the RELs are revised by OEHHA, more 8-hour RELs will be developed.

Therefore, for the MEIR, we recommend:

- Estimating the acute Hazard Index based on the maximum 1-hour air concentration and 1-hour RELs
- Estimating the chronic Hazard Index based on the annual average air concentration and the chronic RELs, and the oral RELs for multipathway substances

An 8-hour hazard index based on the daily average 8-hour exposure is not required for the MEIR, but can be performed at the discretion of the District for exposure to non-continuously operating facilities using the adjusted annual average air concentration (See EQ 5.4.1.4 A and B or method in App. M). Eight-hour hazard assessments are not recommended for exposure to continuously operating facilities.

For the MEIW, we recommend:

- Estimating the acute Hazard Index based on the maximum 1-hour air concentration and 1-hour RELs
- Estimating the 8-hour Hazard Index based on daily average 8-hour exposure for those chemicals with 8-hour RELs
- Estimating the chronic Hazard Index based on the annual average air concentration and chronic RELs, and oral RELs for multipathway substances

Until there are 8-hour RELs for many of the Hot Spots substances that have a chronic REL value, we recommend determining the chronic HI for the MEIW to adequately protect the offsite worker.

8.3.5 *Evaluation of Background Criteria Pollutants*

The District should be contacted to determine if the contribution of background criteria pollutants to respiratory health effects is required to be included in an HRA for the Hot Spots Program. If inclusion is required, the methods for calculating the health impact from acute and chronic exposure (respiratory endpoint) is the standard HI approach (see Sections 8.3.1 and 8.3.4). There are currently no 8-hour RELs for criteria

pollutants, so 8-hour health impacts from criteria pollutants are not assessed in HRAs. The background criteria pollutant contribution should be calculated if the HI from the facility's emissions exceeds 0.5 in either the acute or chronic assessment for the respiratory endpoint.

The most recent criteria pollutant concentration data should be obtained from the ARB's ambient air monitoring network and can be found in the *California Almanac of Emissions and Air Quality* on their web site at www.arb.ca.gov. For determining the criteria pollutant contribution in HI calculations, the annual average concentration data should be taken from a monitoring site near the facility. If background contributions are unavailable, the District may direct the risk assessor to make an alternative assumption. The criteria pollutants that should be included in acute and chronic assessments for the respiratory endpoint are ozone, nitrogen dioxide, sulfur dioxide, sulfates, and hydrogen sulfide.

8.4 Uses of Exposure Duration Adjustments for Onsite Receptors

Onsite workers are protected by CAL OSHA and typically are not evaluated under the Hot Spots program. Exceptions may include a worker who also lives on the facility property such as at prisons, military bases, and universities that have worker housing within the facility. Another scenario where the District may require assessment of on-site worker exposure and risk is when a facility (e.g., airport) has multiple businesses owned by different entities within the facility/property (e.g., rental car agencies, restaurants, etc.). In these situations the evaluation of onsite cancer risks, and/or acute, 8-hour, and chronic noncancer hazard indices is appropriate under the Hot Spots program. If the onsite receptor under evaluation can be exposed through a noninhalation exposure pathway, then that exposure pathway must also be included. When a receptor lives and works on the facility, site, or property, then these receptors should be evaluated and reported under both residential and worker scenarios and the one that is most health-protective should be used for risk management decisions.

The cancer risk estimates for the on-site residents may use a 30-year exposure duration while the 25-year exposure duration is used for a worker. Under a Tier 2 analysis, alternate exposure durations may be evaluated and presented with all assumptions supported. See section 8.2.10 for more discussion of short-term exposures.

Other situations that may require on-site receptor assessment include the presence of locations where the public may have regular access for the appropriate exposure period (e.g., a lunchtime café, store, or museum for acute exposures). The District or reviewing authority should be consulted on the appropriate evaluations for the risk for all onsite receptors.

8.5 References

ERG, 2008. Summary Report of the Peer Review Meeting: EPA's Draft Framework for Determining a Mutagenic Mode of Action for Carcinogenicity. Final Report. Submitted to Risk Assessment Forum, Office of the Science Advisor, U.S. Environmental Protection Agency, Washington, DC., by Eastern Research Group. May 23, 2008.

OEHHA, 2010. *Cumulative Impacts: Building a Scientific Foundation*. Available online at: http://www.oehha.ca.gov

OEHHA, 2008. *Air Toxics Hot Spots Program Risk Assessment Guidelines*. Technical Support Document for Deriving Noncancer Refernce Exposure Levels. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Available online at: http://www.oehha.ca.gov

OEHHA, 2009. *Air Toxics Hot Spots Program Risk Assessment Guidelines. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures*. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. May 2009. Available online at: http://www.oehha.ca.gov

OEHHA, 2012. *Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis*. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Available online at http://www.oehha.ca.gov

U.S. EPA, 2005a. Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens EPA/630/R-03/003F March 2005.

U.S.EPA, 2005b. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum, Washington, DC. EPA/630/P-03/001F.

9 - Summary of the Requirements for a Modeling Protocol and a Health Risk Assessment Report

The AB 2588 program is a community right-to-know act. Although risk assessment is a technical field, AB 2588 risk assessments need to be clear and understandable to the educated lay person. An Executive Summary that explains the process and the results of the risk assessment in lay terms is necessary. Clear risk communication is imperative in situations where the facility is required to notify the surrounding community. In addition, the risk assessment is by law reviewed by the local Air Pollution Control or Air Quality Management District (District) and OEHHA in order to ensure that AB 2588 risk assessment procedures have been followed. This chapter clarifies the type of information that is needed for District and OEHHA review of modeling protocols and health risk assessments (HRAs).

The material presented here is intended to promote transparent, consistent presentation and efficient review of the modeling protocol and the health risk assessment report (products). We recommend that persons preparing these products consult with the local District to determine if the District has modeling or HRA guidelines that supersede these products. If the District does not have guidelines for these products, then we recommend Section 9.1 be used for modeling protocols and Section 9.2 be used for the presentation of HRAs. Persons preparing modeling protocols and HRAs should specify the guidelines that were used to prepare their products.

9.1 Submittal of a Modeling Protocol

It is strongly recommended that a modeling protocol be submitted to the District for review and approval prior to extensive analysis with an air dispersion model. The modeling protocol is a plan of the steps to be taken during the air dispersion modeling and risk assessment process. We encourage people who are preparing protocols to take advantage of the protocol step and fully discuss anticipated methodologies for any portion of your project that may need special consideration. Below, we have provided an example of the format that may be followed in the preparation of the modeling protocol. **Consult with the District to confirm format and content requirements or to determine the availability of District modeling guidelines before submitting the protocol.**

9.1.1 *Outline for a Modeling Protocol*

I. Introduction

Include the facility name, address, and a brief overview describing the facility's operations.

- Provide a description of the terrain and topography surrounding the facility and potential receptors.

- Indicate the format in which data will be provided. Ideally, the report and summary of data will be on paper and all data and model input and output files will be provided electronically (e.g., compact disk or CD).

- Identify the guidelines used to prepare the protocol (e.g., District Guidelines).

II. Emissions

For each pollutant and process whose emissions are required to be quantified in the HRA, list the annual average emissions (pounds/year and grams/second) and the maximum one-hour emissions (pounds/hour and grams/second)[1]. Maximum 1-hour emissions are used for acute noncancer health impacts while annual emissions are used for chronic exposures (i.e., chronic and 8-hour noncancer health impacts or cancer risk assessment).

- Identify the reference and method(s) used to determine emissions (e.g., source tests, emission factors, etc.). Clearly indicate any emission data that are not reflected in the previously submitted emission inventory report. In this event, a revised emission inventory report will need to be submitted to the District.

- Identify if this will be a multipathway assessment based on emitted substances.

[1] Except radionuclides, for which annual and hourly emissions are reported in Curies/year and millicuries/hour, respectively.

III. Models / Modeling Assumptions

Specify the model and modeling assumptions

- Identify the model(s) to be used, including the version number.
- Identify the model options that will be used in the analysis.
- Identify the modeling domain(s) and the spacing of receptor grid(s). Grid spacing should be sufficient in number and detail to capture the concentration at all of the receptors of interest.
- Indicate complex terrain options that may be used, if applicable.
- Identify the source type(s) that will be used to represent the facility's operations (e.g., point, area, or volume sources, flare options or other).
- Indicate the preliminary source characteristics (e.g., stack height, gas temperature, exit velocity, dimensions of volume source, etc.).
- Identify and support the use of urban or rural dispersion coefficients for those models that require dispersion coefficients. For other models, identify and support the parameters required to characterize the atmospheric dispersion due to land characteristics (e.g., surface roughness, Monin-Obukhov length).

IV. Meteorological Data

Specify the type, source, and year(s) of hourly meteorological data (e.g., hourly surface data, upper air mixing height information).

- State how the data are representative for the facility site.
- Describe QA/QC procedures.
- Identify any gaps in the data; if gaps exist, describe how the data gaps are filled.

V. Deposition

- Specify the method to calculate deposition (if applicable).

VI. Receptors

Specify the type and location of receptors. Include all relevant information describing how the individual and population-related receptors will be evaluated.

- Identify and describe the location(s) of known or anticipated potential sensitive receptors, the point of maximum impact (PMI), the maximum exposed individual residential (MEIR), and worker (MEIW) receptors. Identify any special considerations or grids that will be used to model these receptors. This information should correspond with information provided in Section III (e.g., fine receptor spacing of 20 meters at the fence line and centered on the maximum impacts; coarse receptor spacing of 100 meters out to 2,000 meters; extra coarse spacing of 1,000 meters out to 20,000 meters).

- Identify if spatial averaging will be used. Include necessary background information on each receptor including how the domain and spacing will be determined for each receptor or exposure pathway.

- Describe how the cancer burden or population impact estimates are calculated. Clarify the same information for the presentation of noncancer population impacts (e.g., centroids of the census tracts in the area within the zone of impact).

- Specify that actual UTM coordinates and the block/street locations (i.e., north side of 3,000 block of Smith Street), where possible, will be provided for specified receptor locations.

- Identify and support the use of any exposure adjustments (e.g., time at location, diurnal).

- Include the list of anticipated exposure pathways that will be included and indicate which substance will be evaluated in the multipathway assessment. Identify if sensitive receptors are present and which receptors will be evaluated in the HRA.

VII. Maps

Identify how the information will be graphically presented.

- Indicate which cancer risk isopleths will be plotted for the cancer zone of impact (e.g., 10^{-7}, 10^{-6} see Section 4.6.1).

- Indicate the hazard quotients or hazard indices to be plotted for the noncancer acute, 8 hour, and chronic zones of impact (e.g., 0.5, 1.0, etc.).

9.2 Health Risk Assessment Report

The purpose of this section is to provide an outline to assist with the preparation and review of HRAs. This outline specifies the key components that should be included in HRAs. All information used for the report must be presented in the HRA. Ideally, the HRA report and a summary of data used in the HRA will be on paper and all data and model input and output files will be provided electronically (e.g., CD). Persons preparing HRAs for the Hot Spots Program should consult the District to determine if HRA guidelines or special formats are to be followed when preparing and presenting the HRA's results.

If District guidelines or formats do not exist that supersede this outline, then the HRA should follow the format presented here. If the HRA is prepared for other programs, the reviewing authority should be consulted for clarification of format and content. We recommend that those persons preparing HRAs specify the guidelines that were used to prepare their product. **The HRA may be considered deficient by the reviewing authority if components that are listed here are not included**.

9.2.1 *Outline for the Health Risk Assessment Report*

I. Table of Contents

- Section headings with page numbers indicated.
- Tables of tables and Table of figures with page numbers indicated.
- Appendices with page numbers indicated.

II. Executive Summary

Overview of all relevant information regarding the project or facility.

- Facility identifier number (consult the District).

- Description of facility operations and a list identifying emitted substances including table of maximum 1-hour emissions, and annual average emissions.

- Provide a brief description of acute, 8-hour, chronic, and cancer health impacts of the emitted substances, based on OEHHA's descriptions in the appropriate Technical Support Documents.

- Text presenting overview of dispersion modeling and exposure assessment.

- Text describing estimated cancer risk for carcinogens, noncancer Hazard Quotients and Hazard Indices and a table showing target organ systems by substance for noncancer impacts.

- Summarize the individual and population-wide health impacts including the driving substance(s) and the driving exposure pathways:

    - Location (block/street location; e.g., north side of 3,000 block of Smith Street) and description of the off-site point of maximum impact (PMI), maximum exposed individual resident (MEIR), and maximum exposed individual worker (MEIW).

    - Location (block/street location; e.g., north side of 3,000 block of Smith Street) and description of any on-site receptors that were evaluated at the facility (consult District or agency).

    - Location (block/street location; e.g., north side of 3,000 block of Smith Street) and description of any sensitive receptors that are required by the district or reviewing authorities (consult District or agency).

      NOTE: When presenting information described in the following bullets, cancer risk should be presented separately for a residential 30-year, Tier–1 analysis. Results of other exposure assumptions (e.g., 9 or 70-year) or other tier evaluations should also be presented, and must be clearly labeled. For the Hot Spots Program, while the 30-year exposure duration is recommended as the basis for public notification and risk reduction audits and plans, the District has discretion to use the 70 year exposure scenario for its decisions. In addition, the 70 year cancer risk must be calculated to estimate population-wide impacts.

    - Text presenting an overview of the total cancer risk (including multipathway substances, if present) at the PMI, MEIR, MEIW, and sensitive receptors. Provide a table of cancer risk by substance for the MEIR and MEIW (if applicable). Include a statement indicating which of the substances appear to contribute most to (drive) the potential health impacts. In addition, identify the exposure pathways evaluated in the HRA.

    - Provide a map of the facility and surroundings and identify the location of the MEIR, MEIW, PMI, and other locations or receptors of interest.

    - Provide a map of 30-year and 70-year cancer risk zone of impact(s), if applicable.

    - Text presenting an overview of the acute and chronic noncancer hazard quotients and the (total) hazard indices for the PMI, MEIR, MEIW, and sensitive receptors. Additionally, include 8-hour hazard quotients and hazard indices for the MEIW. Include separate statements (for acute, 8-hour, and chronic exposures) indicating which

of the substances appear to drive the potential health impacts. In addition, clearly identify the primary target organ(s) that are impacted from acute, 8-hour, and chronic exposures.

- Identify any sensitive subpopulations (e.g., child daycare facilities, schools, nursing homes) of concern.

- Table and text presenting an overview of estimates of population exposure (e.g., cancer burden or population estimates from HARP) (consult District or agency) (see Section 8.4).

- Version of the Risk Assessment Guidelines and computer program(s) used to prepare the risk assessment (e.g., HARP).

III. Risk Assessment Procedures

A. Hazard identification

- Table and text identifying all substances emitted from the facility, plus any other substances required by the District or reviewing authority. Include the CAS number of the substance and the physical form of the substance if possible. [The Hot Spots substances are listed in Appendix A, and also in the ARB's Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report (EICG Report), which is incorporated by reference therein (ARB, 1997)].

- Table and text identifying all substances that are evaluated for cancer risk and/or noncancer acute, 8-hour, and chronic health impacts. In addition, identify any multipathway substances that present a cancer risk or chronic noncancer hazard via noninhalation routes of exposure.

- Describe the types and amounts of continuous or intermittent predictable emissions from the facility that occurred during the reporting year. As required by statute, releases from a facility include spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping (fugitive), leaching, dumping, or disposing of a substance into ambient air. Include the substance(s) released and a description of the processes that resulted in long-term and continuous releases.

B. Exposure Assessment

This section describes the information related to the air dispersion modeling process that needs to be reported in the risk assessment; the information is also presented in Chapter 4 (see Section 4.15). The District may have specific requirements regarding format and content (see Section 4.14). Sample calculations should be provided at each step to indicate how reported emissions

data were used. Reviewing agencies must receive input, output, and supporting files of various model analyses on computer-readable media (e.g., CD).

1. Information on the Facility and its Surroundings

Report the following information regarding the facility and its surroundings:

- Facility Name
- Location (UTM coordinates and street address)
- Land use type (see Section 2.4)
- Local topography
- Facility plot plan identifying:
  - source locations
  - property line
  - horizontal scale
  - building heights
  - emission sources

2. Source and Emission Inventory Information

a. Release Parameters

Report the following information for each release location in table format:

- Release location identification number
- Release name
- Release type (e.g., point, volume, area, line, pit, etc.)
- Source identification number(s) used by the facility for sources that emit out of this release location
- Release location using UTM coordinates
- Release parameters by release type (e.g., shown for point source):
- Stack height (m), stack diameter (building dimensions for downwash, exhaust gas exit velocity (m/s), exhaust gas volumetric flow rate (ACFM), exhaust gas exit temperature (K), etc.

b. Source Description and Operating Schedule

The description and operating schedule for each source should be reported in table form including the following information:

- Source identification number used by the facility
- Source name
- Number of operating hours per day and per year (e.g., 0800-1700, 2700 hr/yr)
- Number of operating days per week (e.g., Mon-Sat)

- Number of operating days or weeks per year (e.g., 52 wk/yr excluding major holidays)
- Release point identification number(s) for where source emissions are released
- Fraction of source emissions emitted at each release point by release point ID number

c. *Emission Control Equipment and Efficiency*

- Report emission control equipment and efficiency by source and by substance

d. *Emissions Data Grouped By Source*

Report emission rates for each toxic substance, grouped by source (i.e., emitting device or process identified in Inventory Report), in table form including the following information:

- Source name
- Source identification number
- Substance name and CAS number (from Inventory Guidelines)
- Annual average emissions for each substance (lb/yr)
- Hourly maximum emissions for each substance (lb/hr)

e. *Emissions Data Grouped by Substance*

Report facility total emission rate by substance for all emitted substances listed in the Air Toxics “Hot Spots” Program including the following information:

- Substance name and CAS number (from Inventory Guidelines)
- Annual average emissions for each substance (lb/yr)
- Hourly maximum emissions for each substance (lb/hr)

f. *Emission Estimation Methods*

Report the methods used in obtaining the emissions data indicating whether emissions were measured or estimated. Clearly indicate any emission data that are not reflected in the previously submitted emission inventory report and submit a revised emission inventory report to the district. A reader should be able to reproduce the risk assessment without the need for clarification.

g. List of Substances

Include tables listing all "Hot Spots" Program substances which are emitted, plus any other substances required by the District. Indicate substances to be evaluated for cancer risks and noncancer effects.

h. Exposed Population and Receptor Location

Report the following information regarding exposed population and receptor locations:

- Description of zone of impact including map showing the location of the facility, boundaries of zone of impact, census tracts, emission sources, sites of maximum exposure, and the location of all appropriate receptors. This should be a true map (one that shows roads, structures, etc.), drawn to scale, and not just a schematic drawing. USGS 7.5 minute maps or GIS based maps are usually the most appropriate choices. (If significant development has occurred since the user's survey, this should be indicated.)

- Separate maps for the cancer risk zone of impact and the hazard index (noncancer) zone of impact(s). The cancer zone of impact should include isopleths down to at least the 1/1,000,000 risk level. Because some districts use a level below 1/1,000,000 to define the zone of impact, the District should be consulted. For the noncancer zone of impact, three separate isopleths (to represent chronic, 8-hour, and acute HI) should be created to define the zone of impact for the hazard index from both inhalation and noninhalation pathways greater than or equal to 0.5. The point of maximum impact (PMI), maximum exposed individual at a residential receptor (MEIR), and maximum exposed individual worker (MEIW) for both cancer and noncancer risks should be located on the maps.

- Tables identifying population units and sensitive receptors (UTM coordinates, receptor IDs or index from the modeling, and street addresses of specified receptors)

- Heights or elevations of the receptor points.

- **Spatial averaging**: For each receptor type (e.g., PMI, MEIR, and MEIW, or other location of interest) that will utilize spatial averaging, the domain size and grid resolution must be clearly identified. If another domain or grid resolution other than 20 meters by 20 meters with 5-meter grid spacing will be used for a receptor, then care should be taken to determine the proper domain size and grid resolution that should be used. For a worker, the HRA shall support all assumptions used, including, but not limited to, documentation for all workers

showing the area where each worker routinely performs their duties. The final domain size should not be greater than the smallest area of worker movement. Other considerations for determining domain size and grid spacing resolution may include an evaluation of the concentration gradients across the worker area. The grid spacing used within the domain should be sufficient in number and detail to obtain a representative concentration across the area of interest. When spatial averaging over the deposition area of a pasture, garden, or water body, care should be taken to determine the proper domain size to make sure it includes all reasonable areas of potential deposition. The size and shape of the pasture, garden, or water body of interest should be identified and used for the modeling domain. The grid spacing or resolution used within the domain should be sufficient in detail to obtain a representative deposition concentration across the area of interest. One way to determine the grid resolution is to include an evaluation of the concentration gradients across the deposition area. The HRA shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the pasture or water body, maps, representative coordinates, grid resolution, concentration gradients, etc.). The use or spatial averaging is subject to approval by the reviewing authority. This includes the size of the domain and grid resolution that is used for spatial averaging of a worksite or multipathway deposition area.

3. Meteorological Data

If meteorological data were not obtained directly from the District, then the report must clearly indicate the data source and time period used. Meteorological data not obtained from the District must be submitted in electronic form along with justification for their use including information regarding representativeness and quality assurance.

The risk assessment should indicate if the District required the use of a specified meteorological data set. All memos indicating the District's approval of meteorological data should be attached in an appendix.

4. Model Selection and Modeling Rationale

The report should include an explanation of the model chosen to perform the analysis and any other decisions made during the modeling process. The report should clearly indicate the name of the models that were used, the level of detail (screening or refined analysis) and the rationale behind the selection.

Also report the following information for each air dispersion model used:

- Version number
- Selected options and parameters in table form

- Identify the modeling domain(s) and the spacing of receptor grid(s). Grid spacing should be sufficient in number and detail to capture the concentration at all receptors of interest.

5. Air Dispersion Modeling Results

The report should include tables, text, and appendices that clearly present all of the following information

- Maximum hourly and annual average concentrations of chemicals at appropriate receptors such as the residential and worker MEI receptors

- Annual average and maximum one-hour (and 30-day average for lead only) concentrations of chemicals at appropriate receptors listed and referenced to computer printouts of model outputs

- Model printouts (numbered), annual concentrations, maximum hourly concentrations

- Disk with input/output files for air dispersion program (e.g., the AERMOD input file containing the regulatory options and emission parameters, receptor locations, meteorology, etc.)

- Include tables that summarize the annual average concentrations that are calculated for all the substances at each site. The use of tables that present the relative contribution of each emission point to the receptor concentration is recommended. (These tables should have clear reference to the computer model which generated the data. It should be made clear to any reader how data from the computer output were transferred to these tables.) [As an alternative, the above two tables could contain just the values for sites of maximum impact (i.e., PMI, MEIR and MEIW), and sensitive receptors, if required. All the values would be found in the Appendices.]

C. ***Health Values Used in Dose-Response and Dose Estimates***

- Provide tables of the acute, 8-hour and chronic inhalation RELs, chronic oral RELs (if applicable), and cancer potency factors for each substance that is quantified in the HRA.

- Identify the guidelines (title and date) that were used to obtain these factors, or indicate whether newly approved values obtained from the OEHHA website were used.

- Provide a table of target organ systems for each noncancer substance, including acute (1 hour), 8-hour, and chronic inhalation, and chronic oral (if applicable).

- Include tables of the estimated dose for each substance by each exposure pathway at the PMI, MEIR, MEIW, and at any sensitive receptor locations (required by the District).

D. ***Risk Characterization***

The Hot Spots Analysis and Reporting Program (HARP) will generate the risk characterization data needed for the outline below. Any data needed to support the risk characterization findings should be clearly presented and referenced in the text and appendices. A listing of HARP output files that meet these HRA requirements is provided in this outline under the section entitled "Appendices". All HARP files should be included in the HRA. Ideally, the HRA report and a summary of data used in the HRA will be on paper and all data and model input and output files will be provided electronically (e.g., CD). Information on obtaining copies of HARP is available on the California Air Resources Board's Internet web site under the Air Toxics Program at *www.arb.ca.gov.*

NOTE: The cancer risk for the PMI, MEIR, and sensitive receptors of interest must be presented in the HRA's text, tables, and maps. OEHHA recommends that cancer risk for a 30-year exposure duration be presented for the MEIR, and that cancer risk for 9-year and 70-year exposure durations for the MEIR be presented to provide the risk managers with supplemental information. Note that the assessment of population impacts must be based on a 70-year exposure duration; thus all risk assessments need to estimate cancer risk for a 70-year exposure duration in order to report the number of individuals residing in the risk isopleths, or to calculate cancer burden if the District so requires. In addition, some Districts may opt to make risk management decisions based on a 70-year exposure duration. The MEIW location should use a 25-year exposure period.

All HRAs must include the results of a Tier-1 exposure assessment (see Chapter 2 and 8, or the 2012 TSD). If the reviewing authority specifies that additional exposure periods should be presented, or if persons preparing the HRA would like to present additional information (i.e., exposure duration adjustments or the inclusions of risk characterizations using Tier-2 through Tier-4 exposure data), then this information should be presented in separate, clearly titled, sections, tables, and text.

The following information should be presented in this section of the HRA. If not fully presented here, then by topic, clearly identify the section(s) and pages within the HRA where this information is presented.

- Description of receptors to be quantified.

- Table and text providing the location [UTM coordinates, receptor ID number or index from the modeling, and the block/street address

(e.g., north side of 3,000 block of Smith Street)] and description of the PMI, MEIR, and MEIW for both cancer and noncancer risks.

- Separate tables and text providing description of the PMI and MEIR for 30-year cancer risk, and 9- or 70-year cancer risk.

- Tables and text describing MEIW 25-year cancer risk.

- Table and text providing the location [UTM coordinates, receptor ID number or index from the modeling, and the block/street address (e.g., north side of 3,000 block of Smith Street)] and description of any sensitive receptor that is of interest to the District or reviewing authorities (consult District or agency).

- Provide any exposure information that is used for risk characterization (e.g., concentrations at receptors, emissions information, census information, figures, zone of impact maps, etc.). If multipathway substances are emitted, identify the site/route dependent exposure pathways (e.g., water ingestion) for the receptor(s), where appropriate (e.g., MEIR).

- Provide a summary of the site-specific inputs used for each exposure pathway (e.g., water or grazing intake assumptions). This information may be presented in an appendix with the information clearly presented and cross-referenced to the text. In addition, provide reference to the appendix (section and page number) that contains the modeling (i.e., HARP/dispersion modeling) files that show the same information.

- If any exposure parameters were used other than those provided in the Air Toxics Risk Assessment Guidelines: Technical Support Document for Exposure Assessment and Stochastic Analysis (2012), they must be presented in detail. The derivation and data used must be presented so that it is clear to the reviewer. The justification for using site-specific exposure parameters must be clearly presented.

- Table and text presenting the potential multipathway cancer risk by substance, by pathway, and total, at the PMI, MEIR, MEIW, and sensitive receptor locations (required by the District).

- Table and text presenting the acute (inhalation only) and chronic noncancer (inhalation and oral) hazard quotients (by substance, exposure pathways, and target organs) and the (total) hazard indices by substance and target organs for the PMI, MEIR, MEIW, and sensitive receptors. For 8-hour exposure at the MEIW (inhalation only), table and text presenting hazard quotients (by substance, exposure pathways, and target organs) and the (total) hazard indices by substance and target organs. Note:

Chronic noncancer results should be shown with inhalation and oral contributions (shown separately) and for the combined (multipathway) impact.

- Identify any sensitive subpopulations (e.g., child daycare facilities, schools, nursing homes) of concern.

- Table and text presenting estimates of population exposure (e.g., population exposure estimates or cancer burden from HARP) (consult District or agency). Tables should indicate the number of persons exposed to a (total) cancer risk greater than 10^{-7}, 10^{-6}, 10^{-5}, 10^{-4}, etc., and total hazard quotient or hazard index greater than 0.5, 1.0, 2.0, and 3.0, etc. Provide a table that shows excess cancer burden for each population unit and the total excess cancer burden, if cancer burden calculation is required.

- Provide maps that illustrate the HRA results for the three sub-bullet points below. These maps should be an actual street map of the area impacted by the facility with elevation contours and actual UTM coordinates, and the facility boundaries clearly labeled. In some cases the elevation contours will make the map too crowded and should therefore not appear. This should be a true map (one that shows roads, structures, etc.), drawn to scale, and not just a schematic drawing. USGS 7.5-minute maps are usually the most appropriate choice (see Section 4.6).

    - The facility (emission points and boundaries), the locations of the PMI, MEIR, MEIW, and sensitive receptors.

    - Maps of the cancer zone of impacts (e.g., 10^{-6} or 10^{-7} levels - consult District or Agency). The map should clearly identify the zone of impact for the inhalation pathway, the minimum exposure pathways (soil ingestion, dermal exposure, and breast-milk consumption) if multipathway substances are emitted, and the zone of impact for all the applicable exposure pathways (minimum exposure pathways plus any additional site/route specific pathways) for multipathway analyses. Two maps may be needed to accomplish this. The legend of these maps should state the level(s) used for the zone of impact and identify the exposure pathways that were included in the assessment.

    - Maps of the noncancer hazard index (HI) zone of impacts (e.g., 0.5 or 1.0 - consult District or Agency). The noncancer maps should clearly identify the noncancer zones of impact. These include the acute (inhalation), 8-hour (inhalation), chronic (inhalation), and chronic (multipathway) zones of impact. For clarity, presentation of the noncancer zones of impact may require two or more maps. The

legend of these maps should state the level(s) used for the zone of impact and identify the exposure pathways.

- The risk assessor may want to include a discussion of the strengths and weaknesses of the risk analyses and associated uncertainty directly related to the facility HRA.

- If appropriate, comment on the possible alternatives for control or remedial measures. How do the risks compare?

- If possible, identify any community concerns that influence public perception of risk.

- Sample calculations may be needed for all analyses in the HRA if proprietary software other than HARP was used. The District should be consulted. These calculations should be clearly presented and referenced to the findings they are supporting in the HRA text.

- Version of the Risk Assessment Guidelines and computer program used to prepare the risk assessment.

- If software other than HARP is used for the health assessment modeling, all supporting material must be included with the HRA (e.g., all algorithms and parameters used in a clear, easy to review format).

E. ***References***

Include any references used for the HRA in this section.

F. ***Appendices***

The appendices should contain all data, sample calculations, assumptions, and all modeling and risk assessment files that are needed to reproduce the HRA results. Ideally, a summary of data used in the HRA will be on paper and all data and model input and output files will be provided electronically (e.g., CD), unless otherwise specified by the district or reviewing authority. All appendices and the information they contain should be referenced, clearly titled, and paginated.

Potential Appendix Topics (if not presented elsewhere in the HRA report):

- List of all receptors locations (UTM coordinates, receptor ID number or index from the modeling, and the block/street address (e.g., north side of 3,000 block of Smith Street)) for the PMI, MEIR, MEIW, and sensitive receptors.
- List of all emitted substances.
- All emissions files.

- List of dose-response factors (Reference Exposure Levels and cancer potency factors).
- All air dispersion modeling input and output files. Detailed discussions of meteorological data, regulatory options, emission parameters, receptor locations, etc.
- Census data.
- Maps.
- Identify the site/route dependent exposure pathways for the receptor(s), where appropriate (e.g., MEIR). Provide a summary of the site-specific inputs used for each pathway (e.g., water or grazing intake assumptions) and the data to support them.
- All calculations used to determine emissions, concentrations, and potential health impacts at the PMI, MEIR, MEIW, and sensitive receptors.
- All HRA model input and output (HARP) files for receptors of concern.
- (Total) cancer and noncancer impacts by receptor, substance, and exposure pathway (by endpoint for noncancer) at all receptors.
- Presentation of alternate risk assessment methods (e.g., alternate exposure durations, or Tier-2 to Tier-4 evaluations with supporting information).

Page Intentionally Left Blank

List of Abbreviations

A - Area
AB2588 - Air Toxics “Hot Spots” Information and Assessment Act, 1987
ACFM - Actual Cubic Feet per Minute
ADL - Annual Dermal Load
AQMD - Air Quality Management District (District)
ARB - Air Resources Board
ASF - Age Sensitivity Factor
AT - Average Time for Lifetime Cancer Risk
BAF - Bioaccumulation Factor
BG - Urban Block Groups
BLP - Buoyant Line and Point Source Dispersion Model
BMI - Breast Milk Intake
BPIP - Building Profile Input Program
BPIPPRM - Building Profile Input Program for PRIME
BSA - Body Surface Area
BW - Bodyweight
C_{air} - annual average air concentration
CALMPRO - Calms processor program
CAPCOA - California Air Pollution Control Officer’s Association
CAS - Chemical Abstracts Service
CERCLA - Comprehensive Environmental Response, Compensation and Liability Act
C_f - Average concentration of a substance in fish
C_m - Average concentration of a substance in mother’s milk (mislabeled on 114 as Cf)
C_{fa} - Average concentration of a substance in animal products
CONST2 - Constant in the Briggs’ stable plume rise equation using BLP
CONST3 - Constant in the Briggs’ neutral plume rise equation using BLP
CPF - Cancer Potency Factor
CRIT - Convergence criterion for the line source calculations using BLP
Cs - Concentration of Substance in the Soil
CTDMPLUS - Complex Terrain Dispersion Model
CTSCREEN - Complex Terrain Screening Model
Cv - Average concentration of a substance in and on vegetation
Cw - Concentration of a Substance in the Water
DECFAC - Pollutant decay factor for use with BLP
DF - Discount Factor
$DOSE_{air}$ - Daily inhaled dose
$DOSE_{fa}$ - Exposure through ingesting home-raised or farm animal products
$DOSE_{fish}$ - Exposure through ingestion of angler-caught fish
Dose-Im - Exposure through mother’s milk ingestion
$DOSE_p$ - Exposure through ingesting home-grown produce
$DOSE_{water}$ - Exposure through ingesting water

DTHTA - Vertical potential temperature gradient
DTSC - Department of Toxic Substance Control
EASA - Exposure Assessment and Stochastic Analysis
ED - Rural Enumeration Districts or Exposure Duration (in years)
EF - Exposure Frequency
EICG - Emission Inventory Criteria and Guidelines
EPA - Environmental Protection Agency
EQ - Equation
F - Fahrenheit
FAH - Fraction of Time at Home
FG - Fraction of diet provided by grazing
GIS - Geographic Information Systems
GLC - Ground-Level Concentrations
GRAF - Gastrointestinal Relative Absorption Factor
HARP - Hot Spots Analysis and Reporting Program
HESIS - Hazard Evaluation System and Information Service
HI - Hazard Index
HQ - Hazard Quotient
HRA - Health Risk Assessment
HSC - Health and Safety Code
IARC - International Agency for Research on Cancer
IDELS - Maximum variation in number of stability classes per hour (BLP option)
ISCST3 - Industrial Source Complex Short Term
IUPAC - International Union of Pure and Applied Chemistry
K - Kelvin
L - Fraction of locally-grown (source-impacted) feed that is not pasture (site-specific)
LOAEL - Lowest Observed Adverse Effects Level
LOD - Level of Detection
LSHEAR - Plume rise wind shear (BLP option)
LTRANS - Transitional point source plume rise (BLP option)
MAXIT - Maximum iterations allowed for line source calculations (BLP option)
MEIR - Maximally Exposed Individual Resident
MEIW - Maximally Exposed Individual Worker
METDB - Meteorological Database
METS - Metabolic Equivalents
MPRM - Meteorological Processor for Regulatory Models
MWAF - Molecular Weight Adjustment Factor
NAS - National Academy of Sciences
NCDC - National Climatic Data Center
NOAEL-No Observed Adverse Effects Level
NTP - National Toxicology Program
NWS - National Weather Station
OCD - Offshore and Coastal Dispersion Model
OEHHA - Office of Environmental Health Hazard Assessment
p - Population density
PAH - Polycyclic Aromatic Hydrocarbons

PCB - Polychlorinated Biphenyl
PCDD - Polychlorinated dibenzo-p-dioxins
PCDF - Polychlorinated dibenzofurans
PEXP - Vertical wind speed power law profile exponents
PM2.5 - Particulate Matter less than 2.5 microns in diameter
PM10 - Particulate Matter less than 10 microns in diameter
PMI - Point of Maximum Impact
QA - Quality Assurance
QC - Quality Control
RCRA - Resource Conservation and Recovery Act
REL - Reference Exposure Level
RfC - Reference Concentration
RfD - Reference Dose
SCRAM - Support Center for Regulatory Air Models
SDM - Shoreline Dispersion Model
SIR - Soil Ingestion Rate
SMAQMD - Sacramento Metropolitan Air Quality Management District
SRP - Scientific Review Panel
TAC - Toxic Air Contaminant
Tco – Biotransfer coefficient
TEF - Toxic Equivalency Factor
TERAN – Terrain option in BLP
TSD - Technical Support Document
TSP - Total Suspended Particulates
UCL - Upper Confidence Limits
USGS - U.S. Geological Survey
UTM - Universal Transvers Mercator
WAF - Worker Adjustment Factor
WHO - World Health Organization

Page Intentionally Left Blank

Index of Selected Terms and Acronyms

OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

Air Toxics Hot Spots Program

Appendices

Guidance Manual for Preparation of Health Risk Assessments

Air, Community, and Environmental Research Branch

Office of Environmental Health Hazard Assessment

California Environmental Protection Agency

Page intentionally left blank

Table of Contents

Page intentionally left blank

Appendix A:

Air Toxics Hot Spots Program

List of Substances*

*The List of Substances presented in Appendix A is periodically updated by the California Air Resources Board (ARB). The most recent update at the time of preparation of this document was August 27, 2007. Future updates may be obtained from the ARB web site (http://www.arb.ca.gov/ab2588/2588guid.htm).

Appendix A-I

Substances For Which

Emissions Must Be Quantified

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
75070	Acetaldehyde		c	20	1	2	3	4				
60355	Acetamide		c	2	1	2	3	4				
75058	Acetonitrile	06/91		200	1	2						
98862	Acetophenone	06/91		100	1	2						
53963	2-Acetylaminofluorene [PAH-Derivative, POM]		c	100	1	2		4	5			
107028	Acrolein			0.05	1	2						
79061	Acrylamide		c	0.01	1	2	3	4				
79107	Acrylic acid	06/91		5	1	2						
107131	Acrylonitrile		c	0.1	1	2	3	4	5			
107051	Allyl chloride		c	5	1	2		4				
7429905	Aluminum	06/91		100	1							
1344281	Aluminum oxide (fibrous forms)	06/91		100							7	
117793	2-Aminoanthraquinone [PAH-Derivative, POM]		c	5	1	2		4	5			
92671	4-Aminobiphenyl [POM]		c	100	1	2	3	4	5			
61825	Amitrole		c	0.1			3	4	5			
7664417	Ammonia			200	1	2						
6484522	Ammonium nitrate	06/91		100	1							
7783202	Ammonium sulfate	06/91		100	1							
62533	Aniline	09/90	c	5	1	2		4				
90040	o-Anisidine		c	100	1	2	3	4	5			
-	Anthracene [PAH, POM], (see PAH)											
7440360	Antimony	06/91		1							7	
*	Antimony compounds including but not limited to:	06/91		1	1	2						[7]
1309644	Antimony trioxide	09/90	c	1	1	2	3	4				[7]
7440382	Arsenic		c	0.01	1	2	3	4	5			
1016	Arsenic compounds (inorganic) including but not limited to:		c	0.01	1	2	3	4	5			[7]
7784421	Arsine			0.01	1	2					7	[7]
1017	Arsenic compounds (other than inorganic)	06/91		0.1	1							[7]
-	Asbestos (see Mineral fibers)											
7440393	Barium	06/91		1							7	
*	Barium Compounds	06/91		1	1							[7]

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
-	Benz[a]anthracene [PAH, POM], (see PAH)											
71432	Benzene		c	2	1	2	3	4	5			
92875	Benzidine (and its salts) [POM]		c	0.0001	1	2	3	4	5			
1020	Benzidine-based dyes [POM] including but not limited to:		c	0.0001	1	2	3					
1937377	Direct Black 38 [PAH-Derivative, POM]		c	0.0001	1	2		4	5			
2602462	Direct Blue 6 [PAH-Derivative, POM]		c	0.0001	1	2		4	5			
16071866	Direct Brown 95 (technical grade) [POM]	09/89	c	0.0001	1	2		4				
-	Benzo[a]pyrene [PAH, POM], (see PAH)											
-	Benzo[b]fluoranthene [PAH, POM], (see PAH)											
271896	Benzofuran	06/91	c	100				4				
98077	Benzoic trichloride {Benzotrichloride}		c	10	1	2		4	5			
-	Benzo[j]fluoranthene [PAH, POM] (see PAH)											
-	Benzo[k]fluoranthene [PAH, POM] (see PAH)											
98884	Benzoyl chloride	06/91		100	1							
94360	Benzoyl peroxide	06/91		100							7	
100447	Benzyl chloride		c	1	1	2		4				
7440417	Beryllium		c	0.001	1	2	3	4	5			
*	Beryllium compounds	09/89	c	0.001	1	2	3	4	5			[7]
92524	Biphenyl [POM]	06/91		0.5	1	2						
111444	Bis(2-chloroethyl) ether {DCEE}	09/89	c	0.05	1	2		4				
542881	Bis(chloromethyl) ether		c	0.001	1	2	3	4	5			
103231	Bis(2-ethylhexyl) adipate	06/91		100	1							
7726956	Bromine			0.5		2						
*	Bromine compounds (inorganic) including but not limited to:			100	1	2						[7]
7789302	Bromine pentafluoride	11/06		100							7	
10035106	Hydrogen bromide	11/06		20							7	
7758012	Potassium bromate			0.1	1		3	4				[7]
75252	Bromoform	06/91		100	1	2		4				
106990	1,3-Butadiene		c	0.1	1	2	3	4	5			
540885	t-Butyl acetate	11/06		200							7	

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
141322	Butyl acrylate	06/91		100	1							
71363	n-Butyl alcohol	06/91		100	1							
78922	sec-Butyl alcohol	06/91		100	1							
75650	tert-Butyl alcohol	06/91		100	1							
85687	Butyl benzyl phthalate	06/91		100	1							
7440439	Cadmium		c	0.01	1	2	3	4	5			
*	Cadmium compounds		c	0.01	1	2	3	4	5			[7]
156627	Calcium cyanamide	06/91		100	1	2						
105602	Caprolactam	06/91		100	1	2						
2425061	Captafol	09/89	c	100				4				
133062	Captan	09/90	c	100	1	2		4				
63252	Carbaryl [PAH-Derivative, POM]	06/91		100	1	2						
1050	Carbon black extracts		c	2	1		3	4				
75150	Carbon disulfide	09/89		200	1	2		4				
56235	Carbon tetrachloride		c	1	1	2	3	4	5			
463581	Carbonyl sulfide	06/91		100	1	2						
1055	Carrageenan (degraded)		c	100			3	4				
120809	Catechol	06/91		100	1	2						
133904	Chloramben	06/91		100	1	2						
57749	Chlordane	09/89	c	10	1	2		4				
108171262	Chlorinated paraffins (average chain length, C12; approximately 60% Chlorine by weight)	09/89	c	2			3	4	5			
7782505	Chlorine			0.5	1	2						
10049044	Chlorine dioxide	06/91		1	1							
79118	Chloroacetic acid	06/91		100	1	2						
532274	2-Chloroacetophenone	06/91		0.1	1	2						
106478	p-Chloroaniline	07/96	c	100				4			7	
1058	Chlorobenzenes including but not limited to:	06/91		100	1							
108907	Chlorobenzene			200	1	2						
25321226	Dichlorobenzenes (mixed isomers) including:	06/91		100	1						7	
95501	1,2-Dichlorobenzene	06/91		200	1						7	
541731	1,3-Dichlorobenzene	06/91		100	1						7	

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
106467	p-Dichlorobenzene {1,4-Dichlorobenzene}		c	5	1	2	3		5			
120821	1,2,4-Trichlorobenzene	06/91		200	1	2						
510156	Chlorobenzilate [POM] {Ethyl-4,4'-dichlorobenzilate}	09/90	c	100	1	2		4				
67663	Chloroform		c	10	1	2	3	4	5			
107302	Chloromethyl methyl ether (technical grade)		c	100	1	2		4	5			
1060	Chlorophenols including but not limited to:		c	100	1		3					
95578	2-Chlorophenol	11/06		10	1		3					
120832	2,4-Dichlorophenol	06/91	c	100	1						7	
87865	Pentachlorophenol	09/90	c	10	1	2		4				
25167833	Tetrachlorophenols including but not limited to:	11/06		10							7	
58902	2,3,4,6-Tetrachlorophenol	07/96	c	100	1						7	
95954	2,4,5-Trichlorophenol	06/91	c	100	1	2						
88062	2,4,6-Trichlorophenol		c	2	1	2		4				
95830	4-Chloro-o-phenylenediamine		c	10			3	4	5			
76062	Chloropicrin			2							7	
126998	Chloroprene			5	1	2						
95692	p-Chloro-o-toluidine		c	0.5			3	4				
7440473	Chromium	06/91		0.001							7	
*	Chromium compounds (other than hexavalent)	06/91		0.001	1	2						[7]
18540299	Chromium, hexavalent (and compounds) including but not limited to:		c	0.0001	1	2	3	4	5			[7]
10294403	Barium chromate	06/91	c	0.001	1	2			5			[7]
13765190	Calcium chromate	06/91	c	0.001	1	2			5			[7]
1333820	Chromium trioxide	06/91	c	0.0001	1	2			5			[7]
7758976	Lead chromate	06/91	c	0.001	1	2			5			[7]
10588019	Sodium dichromate	06/91	c	0.0001	1	2			5			[7]
7789062	Strontium chromate	06/91	c	0.001	1	2			5			[7]
-	Chrysene [PAH, POM], (see PAH)											
7440484	Cobalt	06/91		0.5							7	
*	Cobalt compounds	06/91		0.5	1	2						[7]
1066	Coke oven emissions		c	0.05	1	2	3	4	5			

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
7440508	Copper			0.1		2						
*	Copper compounds	09/89		0.1	1	2						[7]
1070	Creosotes		c	0.05	1		3	4				
120718	p-Cresidine		c	1			3	4	5			
1319773	Cresols (mixtures of) {Cresylic acid} including:			5	1	2						
108394	m-Cresol	06/91		5	1	2						
95487	o-Cresol	06/91		5	1	2						
106445	p-Cresol	06/91		5	1	2						
4170303	Crotonaldehyde	07/96	c	50							7	
98828	Cumene	06/91		200	1	2						
80159	Cumene hydroperoxide	06/91		100	1							
135206	Cupferron		c	0.5				4	5			
57125	Cyanide compounds (inorganic) including but not limited to:	06/91		0.05	1	2						[8]
74908	Hydrocyanic acid			10		2						
110827	Cyclohexane	06/91		200	1							
108930	Cyclohexanol	07/96		200							7	
66819	Cycloheximide			2						6		
	Decabromodiphenyl oxide [POM] (see Polybrominated diphenyl ethers)	06/91										
1075	Dialkylnitrosamines including but not limited to:			0.001	1							
924163	N-Nitrosodi-n-butylamine		c	0.0001	1		3	4	5			
1116547	N-Nitrosodiethanolamine		c	100	1		3	4	5			
55185	N-Nitrosodiethylamine		c	0.001	1		3	4	5			
62759	N-Nitrosodimethylamine		c	0.01	1	2	3	4	5			
621647	N-Nitrosodi-n-propylamine		c	0.01	1		3	4	5			
10595956	N-Nitrosomethylethylamine		c	0.001	1		3	4				
615054	2,4-Diaminoanisole		c	5			3	4				
1078	Diaminotoluenes (mixed isomers) including but not limited to:	09/90	c	100	1			4				
95807	2,4-Diaminotoluene {2,4-Toluene diamine}		c	0.05	1	2	3	4	5			
334883	Diazomethane	06/91	c	5	1	2						
226368	Dibenz[a,h]acridine [POM]		c	0.5	1	2	3	4	5			

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
224420	Dibenz[a,j]acridine [POM]		c	0.5	1	2	3	4	5			
-	Dibenz[a,h]anthracene [PAH, POM], (see PAH)											
194592	7H-Dibenzo[c,g]carbazole		c	0.05	1	2	3	4	5			
-	Dibenzo[a,e]pyrene [PAH, POM], (see PAH)											
-	Dibenzo[a,h]pyrene [PAH, POM], (see PAH)											
-	Dibenzo[a,i]pyrene [PAH, POM], (see PAH)											
-	Dibenzo[a,l]pyrene [PAH, POM], (see PAH)											
132649	Dibenzofuran [POM]	06/91		100	1	2						
-	Dibenzofurans (chlorinated) (see Polychlorinated dibenzofurans) [POM]											
96128	1,2-Dibromo-3-chloropropane {DBCP}		c	0.01	1	2	3	4	5			
96139	2,3-Dibromo-1-propanol	07/96	c	50				4				
84742	Dibutyl phthalate	06/91		100	1	2						
-	p-Dichlorobenzene (1,4-Dichlorobenzene) (see Chlorobenzenes)											
91941	3,3'-Dichlorobenzidine [POM]		c	0.1	1	2	3	4	5			
72559	Dichlorodiphenyldichloroethylene {DDE} [POM]	09/89	c	100	1	2		4				
75343	1,1-Dichloroethane {Ethylidene dichloride}	09/90	c	20	1	2		4				
94757	Dichlorophenoxyacetic acid, salts and esters {2,4-D}	06/91		100	1	2						
78875	1,2-Dichloropropane {Propylene dichloride}	09/90	c	20	1	2		4				
542756	1,3-Dichloropropene		c	10	1	2	3	4	5			
62737	Dichlorovos {DDVP}	09/89	c	0.5	1	2		4				
115322	Dicofol [POM]	06/91		100	1	2						
--	Diesel engine exhaust	09/90	c		1		3	4				[9]
9901	Diesel engine exhaust, particulate matter **{**Diesel PM**}**	09/90	c	0.1	1		3	4				[9]
9902	Diesel engine exhaust, total organic gas	09/90	c	10	1		3	4				[9]
#	Diesel fuel (marine)	06/91	c									
111422	Diethanolamine	06/91		20	1	2						
117817	Di(2-ethylhexyl) phthalate {DEHP}		c	20	1	2	3	4	5			
64675	Diethyl sulfate		c	100	1	2	3	4	5			
119904	3,3'-Dimethoxybenzidine [POM]		c	100	1	2	3	4	5			
60117	4-Dimethylaminoazobenzene [POM]		c	0.01	1	2	3	4	5			

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
121697	N,N-Dimethylaniline	06/91		200	1	2						
57976	7,12-Dimethylbenz[a]anthracene [PAH-Derivative, POM]	09/90	c	0.0001	1	2		4				
119937	3,3'-Dimethylbenzidine {o-Tolidine} [POM]		c	10	1	2	3	4	5			
79447	Dimethyl carbamoyl chloride		c	100	1	2	3	4	5			
68122	Dimethyl formamide	09/90	c	100	1	2	3					
57147	1,1-Dimethylhydrazine		c	0.1	1	2	3	4	5			
131113	Dimethyl phthalate	06/91		50	1	2						
77781	Dimethyl sulfate		c	0.01	1	2	3	4	5			
534521	4,6-Dinitro-o-cresol (and salts)	06/91		100	1	2						
51285	2,4-Dinitrophenol	06/91		100	1	2						
42397648	1,6-Dinitropyrene [PAH-Derivative, POM]	06/91	c	0.001	1	2	3	4				
42397659	1,8-Dinitropyrene [PAH-Derivative, POM]	06/91	c	0.05	1	2	3	4				
25321146	Dinitrotoluenes (mixed isomers) including but not limited to:	06/91		100							7	
121142	2,4-Dinitrotoluene	09/89	c	0.5	1	2		4				
606202	2,6-Dinitrotoluene	06/91		100							7	
123911	1,4-Dioxane		c	5	1	2	3	4	5			
-	Dioxins (Chlorinated dibenzodioxins) (see Polychlorinated dibenzo-p-dioxins) [POM]											
630933	Diphenylhydantoin [POM]		c	100	1	2		4				
122667	1,2-Diphenylhydrazine {Hydrazobenzene} [POM]		c	100	1	2		4	5			
1090	Environmental Tobacco Smoke		c	2	1		3	4				
106898	Epichlorohydrin		c	2	1	2	3	4	5			
106887	1,2-Epoxybutane	06/91		100	1	2						
1091	Epoxy resins	09/89		100						6		
140885	Ethyl acrylate		c	200	1	2	3	4	5			
100414	Ethyl benzene	06/91		200	1	2						
75003	Ethyl chloride {Chloroethane}			200	1	2		4				
-	Ethyl-4,4'-dichlorobenzilate (see Chlorobenzilate)											
74851	Ethylene	06/91		200							7	
106934	Ethylene dibromide {EDB, 1,2-Dibromoethane}		c	0.5	1		3	4	5	6		
107062	Ethylene dichloride {EDC, 1,2-Dichloroethane}		c	2	1	2	3	4	5			

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
107211	Ethylene glycol	06/91		200	1	2						
151564	Ethyleneimine {Aziridine}	06/91		100	1	2						
75218	Ethylene oxide		c	0.5	1	2	3	4	5	6		
96457	Ethylene thiourea		c	2	1	2	3	4	5			
1101	Fluorides and compounds including but not limited to:	09/89		100		2						
7664393	Hydrogen fluoride			50	1	2					7	
1103	Fluorocarbons (brominated)			200						6		[10]
1104	Fluorocarbons (chlorinated) including but not limited to:			200	1					6		[10]
76131	Chlorinated fluorocarbon {CFC-113} {1,1,2-Trichloro-1,2,2-trifluoroethane}			200	1	2				6		
75456	Chlorodifluoromethane {Freon 22}	07/96		200	1					6	7	
75718	Dichlorodifluoromethane {Freon 12}	11/06		200							7	
75434	Dichlorofluoromethane {Freon 21}	07/96		200	1					6	7	
75694	Trichlorofluoromethane {Freon 11}	07/96		200	1					6	7	
50000	Formaldehyde		c	5	1	2	3	4	5	6		
110009	Furan	07/96	c	5				4				
--	Gasoline engine exhaust including but not limited to:	09/89	c				3					[9]
--	Gasoline engine exhaust (condensates & extracts)	06/91	c					4				[9]
9910	Gasoline engine exhaust, particulate matter	09/90	c	100			3	4				[9]
9911	Gasoline engine exhaust, total organic gas	09/90	c	100			3	4				[9]
1110	Gasoline vapors		c	200	1	2	3	4				[11]
111308	Glutaraldehyde			0.1	1					6		
1115	Glycol ethers and their acetates including but not limited to:			100	1	2				6		
111466	Diethylene glycol	09/90		100	1					6		
111966	Diethylene glycol dimethyl ether	09/90		100	1	2				6		
112345	Diethylene glycol monobutyl ether	09/90		100	1	2				6		
111900	Diethylene glycol monoethyl ether	09/90		100	1	2				6		
111773	Diethylene glycol monomethyl ether	09/90		100	1	2				6		
25265718	Dipropylene glycol	09/90		100	1					6		

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
34590948	Dipropylene glycol monomethyl ether	09/90		100	1					6		
629141	Ethylene glycol diethyl ether	09/90		100	1	2				6		
110714	Ethylene glycol dimethyl ether	09/90		100	1	2				6		
111762	Ethylene glycol monobutyl ether	09/90		200	1	2				6		
110805	Ethylene glycol monoethyl ether	09/89		50	1	2				6		
111159	Ethylene glycol monoethyl ether acetate	09/90		100	1	2				6		
109864	Ethylene glycol monomethyl ether	09/89		10	1	2				6		
110496	Ethylene glycol monomethyl ether acetate	09/90		200	1	2				6		
2807309	Ethylene glycol monopropyl ether	09/90		100	1	2				6		
107982	Propylene glycol monomethyl ether	09/90		200	1					6		
108656	Propylene glycol monomethyl ether acetate	09/90		100	1					6		
112492	Triethylene glycol dimethyl ether	09/90		100	1	2				6		
76448	Heptachlor	09/89	c	100	1	2		4				
118741	Hexachlorobenzene		c	0.1	1	2	3		5			
87683	Hexachlorobutadiene	06/91		0.1	1	2						
608731	Hexachlorocyclohexanes (mixed or technical grade), including but not limited to:		c	0.05	1		3	4	5			
319846	alpha-Hexachlorocyclohexane	07/96	c	0.1	1		3	4	5		7	
319857	beta-Hexachlorocyclohexane	07/96	c	0.1	1		3	4	5		7	
58899	Lindane {gamma-Hexachlorocyclohexane}	09/90	c	0.1	1	2		4				
77474	Hexachlorocyclopentadiene			2	1	2						
67721	Hexachloroethane	09/90	c	200	1	2		4				
680319	Hexamethylphosphoramide		c	100	1	2	3	4	5			
110543	Hexane	06/91		200	1	2						
302012	Hydrazine		c	0.01	1	2	3	4	5			
7647010	Hydrochloric acid			20	1	2						
-	Hydrocyanic acid (see Cyanide compounds)											
7783064	Hydrogen sulfide			5	1	2						
123319	Hydroquinone	06/91		100	1	2						
-	Indeno[1,2,3-cd]pyrene [PAH, POM], (see PAH)											
13463406	Iron pentacarbonyl	07/96		5							7	
1125	Isocyanates including but not limited to:			0.05						6		

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
822060	Hexamethylene-1,6-diisocyanate	06/91		0.05	1	2						
101688	Methylene diphenyl diisocyanate {MDI} [POM]	06/91		0.1	1	2						
624839	Methyl isocyanate			1	1	2						
-	Toluene-2,4-diisocyanate (see Toluene diisocyanates)											
-	Toluene-2,6-diisocyanate (see Toluene diisocyanates)											
78591	Isophorone	06/91		200	1	2						
78795	Isoprene, except from vegetative emission sources	07/96	c	200			3					
67630	Isopropyl alcohol	06/91		200	1							
80057	4,4'-Isopropylidenediphenol [POM]	06/91		100	1	2						
7439921	Lead		c	0.5	1			4		6		
1128	Lead compounds (inorganic) including but not limited to:		c	0.5	1		3					[7]
301042	Lead acetate		c	1	1	2		4	5			[7] [12]
-	Lead chromate (see Chromium, hexalent)											
7446277	Lead phosphate		c	2	1			4	5			[7]
1335326	Lead subacetate	09/90	c	2	1	2		4				[7] [12]
1129	Lead compounds (other than inorganic)	06/91		5	1	2						[7]
108316	Maleic anhydride			0.5	1	2						
7439965	Manganese			0.1	1	2						
*	Manganese compounds	09/89		0.1	1	2						[7]
7439976	Mercury			1	1	2		4		6		
*	Mercury compounds including but not limited to:	09/89		1	1	2		4				[7]
7487947	Mercuric chloride			1		2						[7]
593748	Methyl mercury {Dimethylmercury}			1		2						[7]
67561	Methanol			200	1	2						
72435	Methoxychlor [POM]	06/91		100	1	2						
75558	2-Methylaziridine {1,2-Propyleneimine}		c	100	1	2	3	4				
74839	Methyl bromide {Bromomethane}			20	1	2				6		
74873	Methyl chloride {Chloromethane}	06/91		20	1	2						
71556	Methyl chloroform {1,1,1-Trichloroethane}			200	1	2				6		
56495	3-Methylcholanthrene [PAH-Derivative, POM]	09/90	c	0.001	1	2		4				

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
3697243	5-Methylchrysene [PAH-Derivative, POM]		c	0.05	1	2	3	4	5			
101144	4,4'-Methylene bis(2-chloroaniline) {MOCA} [POM]		c	0.1	1	2	3	4	5			
75092	Methylene chloride {Dichloromethane}		c	50	1	2	3	4	5	6		
101779	4,4'-Methylenedianiline (and its dichloride) [POM]		c	0.1	1	2	3	4	5			
78933	Methyl ethyl ketone {2-Butanone}	06/91		200	1	2						
60344	Methyl hydrazine	06/91		100	1	2						
74884	Methyl iodide {Iodomethane}		c	100	1	2		4	5			
108101	Methyl isobutyl ketone {Hexone}	06/91		20	1	2						
75865	2-Methyllactonitrile {Acetone cyanohydrin}	07/96		50							7	
80626	Methyl methacrylate			200	1	2				6		
109068	2-Methylpyridine	07/96		100							7	
1634044	Methyl tert-butyl ether	06/91		200	1	2						
90948	Michler's ketone [POM]		c	0.1	1	2		4	5			
1136	Mineral fibers (fine mineral fibers which are man-made, and are airborne particles of a respirable size greater than 5 microns in length, less than or equal to 3.5 microns in diameter, with a length to diameter ratio of 3:1) including but not limited to:	06/91	c	100	1	2					7	
1056	Ceramic fibers	09/89	c	100	1	2	3	4				
1111	Glasswool fibers	09/89	c	100	1	2	3	4				
1168	Rockwool	09/89	c	100	1	2	3					
1181	Slagwool	09/89	c	100	1	2	3					
1135	Mineral fibers (other than man-made) including but not limited to:			100		2					7	
1332214	Asbestos		c	0.0001	1	2	3	4	5			
12510428	Erionite		c	100		2	3	4				
1190	Talc containing asbestiform fibers		c	100		2	3	4				
1313275	Molybdenum trioxide	06/91		100	1							
-	Naphthalene [PAH, POM], (see PAH)											
7440020	Nickel		c	0.1	1	2	3	4	5			
*	Nickel compounds including but not limited to:		c	1	1	2	3	4	5			[7]
373024	Nickel acetate	06/91	c	0.1	1	2			5			[7]
3333673	Nickel carbonate	06/91	c	0.1	1	2			5			[7]

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
13463393	Nickel carbonyl		c	0.1	1	2		4	5			[7]
12054487	Nickel hydroxide	06/91	c	0.1	1	2			5			[7]
1271289	Nickelocene	06/91	c	0.1	1	2			5			[7]
1313991	Nickel oxide	06/91	c	0.1	1	2			5			[7]
12035722	Nickel subsulfide		c	0.1	1	2		4	5			[7]
1146	Nickel refinery dust from the pyrometallurgical process	09/89	c	0.1				4				
7697372	Nitric acid	06/91		50	1							
139139	Nitrilotriacetic acid		c	100	1			4	5			
602879	5-Nitroacenaphthene [PAH-Derivative, POM]	11/06	c	2	1	2	3	4				
98953	Nitrobenzene			0.5	1	2						
92933	4-Nitrobiphenyl [POM]	09/89	c	100	1	2		4				
7496028	6-Nitrochrysene [PAH-Derivative, POM]	06/91	c	0.001	1	2	3	4				
607578	2-Nitrofluorene [PAH-Derivative, POM]	06/91	c	5	1	2	3	4				
302705	Nitrogen mustard N-oxide		c	0.05			3	4				
100027	4-Nitrophenol	06/91		100	1	2						
79469	2-Nitropropane		c	0.01	1	2	3	4	5			
5522430	1-Nitropyrene [PAH-Derivative, POM]	06/91	c	0.5	1	2	3	4				
57835924	4-Nitropyrene [PAH-Derivative, POM]	11/06	c	1				4				
86306	N-Nitrosodiphenylamine	11/06	c	10	1	2	3	4				
156105	p-Nitrosodiphenylamine [POM]		c	5	1	2		4	5			
684935	N-Nitroso-N-methylurea		c	100	1	2		4	5			
59892	N-Nitrosomorpholine		c	0.01	1	2	3	4	5			
100754	N-Nitrosopiperidine		c	1			3	4	5			
930552	N-Nitrosopyrrolidine		c	0.05			3	4	5			
*	Oleum (see Sulfuric acid and oleum)											
--	PAHs (Polycyclic aromatic hydrocarbons) [POM] including but not limited to:				1	2						[13]
1151	PAHs, total, w/o individ. components reported [PAH, POM]			50	1	2						
1150	PAHs, total, with individ. components also reported [PAH, POM]			50	1	2						
83329	Acenaphthene [PAH, POM]	07/96		50	1							

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
208968	Acenaphthylene [PAH, POM]	07/96		50	1							
120127	Anthracene [PAH, POM]	06/91		50	1	2					7	
56553	Benz[a]anthracene [PAH, POM]		c	0.5	1	2	3	4	5			
50328	Benzo[a]pyrene [PAH, POM]		c	0.05	1	2	3	4	5			
205992	Benzo[b]fluoranthene		c	0.5	1	2	3	4	5			
192972	Benzo[e]pyrene [PAH, POM]	07/96		0.5	1							
191242	Benzo[g,h,i]perylene [PAH, POM]	07/96		0.5	1							
205823	Benzo[j]fluoranthene [PAH, POM]		c	0.5	1	2	3	4	5			
207089	Benzo[k]fluoranthene [PAH, POM]		c	0.5	1	2	3	4	5			
218019	Chrysene [PAH, POM]	09/90	c	5	1	2		4				
53703	Dibenz[a,h]anthracene [PAH, POM]		c	0.1	1	2	3	4	5			
192654	Dibenzo[a,e]pyrene [PAH, POM]		c	0.05	1	2	3	4	5			
189640	Dibenzo[a,h]pyrene [PAH, POM]		c	0.001	1	2	3	4	5			
189559	Dibenzo[a,i]pyrene [PAH, POM]		c	0.001	1	2	3	4	5			
191300	Dibenzo[a,l]pyrene [PAH, POM]		c	0.001	1	2	3	4	5			
206440	Fluoranthene [PAH, POM]	07/96	c	0.5	1							
86737	Fluorene [PAH, POM]	07/96	c	0.5	1							
193395	Indeno[1,2,3-cd]pyrene [PAH, POM]		c	0.5	1	2	3	4	5			
91576	2-Methyl naphthalene [PAH, POM]	07/96	c	50	1							
91203	Naphthalene [PAH, POM]		c	0.1	1	2						
198550	Perylene [PAH, POM]	07/96	c	0.5	1							
85018	Phenanthrene [PAH, POM]	07/96	c	0.5	1							
129000	Pyrene [PAH, POM]	07/96	c	0.5	1							
#	PAH-Derivatives (Polycyclic aromatic hydrocarbon derivatives) [POM] (including but not limited to those substances listed in Appendix A with the bracketed designation [PAH-Derivative, POM])	06/91										[14]
56382	Parathion	06/91		100	1	2						
1336363	PCBs (Polychlorinated biphenyls), total [POM] including but not limited to:		c	0.01	1	2	3	4	5	6		
32598133	3,3',4,4'-Tetrachlorobiphenyl (PCB 77)	11/06	c	0.01		2	3	4	5			
70362504	3,4,4',5-Tetrachlorobiphenyl (PCB 81)	11/06	c	0.01		2	3	4	5			

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
32598144	2,3,3',4,4'-Pentachlorobiphenyl (PCB 105)	11/06	c	0.01		2	3	4	5			
74472370	2,3,4,4',5-Pentachlorobiphenyl (PCB 114)	11/06	c	0.002		2	3	4	5			
31508006	2,3',4,4',5-Pentachlorobiphenyl (PCB 118)	11/06	c	0.01		2	3	4	5			
65510443	2,3',4,4',5'-Pentachlorobiphenyl (PCB 123)	11/06	c	0.01		2	3	4	5			
57465288	3,3',4,4',5-Pentachlorobiphenyl (PCB 126)	11/06	c	0.00001		2	3	4	5			
38380084	2,3,3',4,4',5-Hexachlorobiphenyl (PCB 156)	11/06	c	0.002		2	3	4	5			
69782907	2,3,3',4,4',5'-Hexachlorobiphenyl (PCB 157)	11/06	c	0.002		2	3	4	5			
52663726	2,3',4,4',5,5'-Hexachlorobiphenyl (PCB 167)	11/06	c	0.1		2	3	4	5			
32774166	3,3',4,4',5,5'-Hexachlorobiphenyl (PCB 169)	11/06	c	0.0001		2	3	4	5			
39635319	2,3,3',4,4',5,5'-Heptachlorobiphenyl (PCB 189)	11/06	c	0.01		2	3	4	5			
82688	Pentachloronitrobenzene {Quintobenzene}	06/91		100	1	2						
79210	Peracetic acid	06/91		100	1							
127184	Perchloroethylene {Tetrachloroethene}		c	5	1	2	3	4	5	6		
2795393	Perfluorooctanoic acid {PFOA} and its salts, esters, and sulfonates	11/06		10							7	
108952	Phenol			200	1	2						
106503	p-Phenylenediamine	06/91		100	1	2						
90437	2-Phenylphenol [POM]	06/91		100	1	2						
75445	Phosgene			2	1	2						
7723140	Phosphorus			0.1	1	2						
--	Phosphorus compounds:	09/89				2						
7803512	Phosphine			0.01	1	2					7	
7664382	Phosphoric acid	09/89		50	1	2						
10025873	Phosphorus oxychloride	09/89		0.1		2						
10026138	Phosphorus pentachloride	09/89		0.1		2						
1314563	Phosphorus pentoxide	09/89		0.1		2						
7719122	Phosphorus trichloride	09/89		0.1		2						
126738	Tributyl phosphate	09/89		100		2						
78400	Triethyl phosphine	09/89		100		2						
512561	Trimethyl phosphate	09/89		100		2						
78308	Triorthocresyl phosphate [POM]	09/89		0.5	1	2						
115866	Triphenyl phosphate [POM]	09/89		100	1	2						

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
101020	Triphenyl phosphite [POM]	09/89		100	1	2						
85449	Phthalic anhydride			0.01	1	2						
2222	Polybrominated diphenyl ethers {PBDEs}, including but not limited to:	11/06		1							7	
1163195	Decabromodiphenyl oxide [POM]	06/91		1	1	2						
--	Polychlorinated dibenzo-p-dioxins {PCDDs or Dioxins} [POM], including but not limited to:		c		1	2						
1086	Dioxins, total, w/o individ. isomers reported {PCDDs} [POM]		c	0.000001	1	2						
1085	Dioxins, total, with individ. isomers also reported {PCDDs} [POM]		c	0.000001	1	2						
1746016	2,3,7,8-Tetrachlorodibenzo-p-dioxin {TCDD} [POM]		c	0.000001	1	2	3	4	5			
40321764	1,2,3,7,8-Pentachlorodibenzo-p-dioxin [POM]		c	0.000001	1	2						
39227286	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin [POM]		c	0.000001	1	2		4				
57653857	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin [POM]		c	0.000001	1	2						
19408743	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin [POM]		c	0.000001	1	2						
35822469	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin [POM]		c	0.000001	1	2						
3268879	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1	2						
41903575	Total Tetrachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1	2						
36088229	Total Pentachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1	2						
34465468	Total Hexachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1	2						
37871004	Total Heptachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1	2						
--	Polychlorinated dibenzofurans {PCDFs or Dibenzofurans} [POM], including but not limited to:		c		1	2						
1080	Dibenzofurans (Polychlorinated dibenzofurans) {PCDFs} [POM]		c	0.000001	1	2						
51207319	2,3,7,8-Tetrachlorodibenzofuran [POM]		c	0.000001	1	2						
57117416	1,2,3,7,8-Pentachlorodibenzofuran [POM]		c	0.000001	1	2						
57117314	2,3,4,7,8-Pentachlorodibenzofuran [POM]		c	0.000001	1	2						
70648269	1,2,3,4,7,8-Hexachlorodibenzofuran [POM]		c	0.000001	1	2						
57117449	1,2,3,6,7,8-Hexachlorodibenzofuran [POM]		c	0.000001	1	2						

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
72918219	1,2,3,7,8,9-Hexachlorodibenzofuran [POM]		c	0.000001	1	2						
60851345	2,3,4,6,7,8-Hexachlorodibenzofuran [POM]		c	0.000001	1	2						
67562394	1,2,3,4,6,7,8-Heptachlorodibenzofuran [POM]		c	0.000001	1	2						
55673897	1,2,3,4,7,8,9-Heptachlorodibenzofuran [POM]		c	0.000001	1	2						
39001020	1,2,3,4,6,7,8,9-Octachlorodibenzofuran [POM]	07/96	c	0.000001	1	2						
55722275	Total Tetrachlorodibenzofuran [POM]	07/96	c	0.000001	1	2						
30402154	Total Pentachlorodibenzofuran [POM]	07/96	c	0.000001	1	2						
55684941	Total Hexachlorodibenzofuran [POM]	07/96	c	0.000001	1	2						
38998753	Total Heptachlorodibenzofuran [POM]	07/96	c	0.000001	1	2						
#	POM (Polycyclic organic matter) (including but not limited to those substances listed in Appendix A with the bracketed designation of [POM], [PAH, POM], or [PAH-Derivative, POM])	09/89			1	2						[15]
1120714	1,3-Propane sultone		c	0.05	1	2	3	4	5			
57578	beta-Propiolactone		c	10	1	2	3	4	5			
123386	Propionaldehyde	06/91		200	1	2						
114261	Propoxur {Baygon}	06/91		100	1	2						
115071	Propylene			200	1	2						
75569	Propylene oxide		c	10	1	2	3	4	5			
-	1,2-Propyleneimine (see 2-Methylaziridine)											
110861	Pyridine	06/91		100							7	
91225	Quinoline	06/91		100	1	2						
106514	Quinone	06/91		100	1	2						
1165	Radionuclides including but not limited to:		c	100	1	2		4				[16]
24267569	Iodine-131	09/89	c	100	1	2		4				
1166	Radon and its decay products	09/89	c	100	1			4				
50555	Reserpine [POM]		c	100	1	2		4	5			
#	Residual (heavy) fuel oils	06/91	c									
7782492	Selenium			0.5		2						
*	Selenium compounds including but not limited to:			0.5	1	2						[7]
7783075	Hydrogen selenide	11/06		0.1							7	
7446346	Selenium sulfide	09/90	c	0.1		2		4	5			[7]

Substances for Which Emissions Must Be Quantified

Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
1175	Silica, crystalline (respirable)			0.1	1		3	4				
7440224	Silver	06/91		2							7	
*	Silver compounds	06/91		2	1							[7]
1310732	Sodium hydroxide			2	1	2						
100425	Styrene		c	100	1	2	3			6		
96093	Styrene oxide		c	100	1	2	3	4				
*	Sulfuric acid and oleum											
8014957	Oleum	11/06		100							7	
7446719	Sulfur trioxide	11/06		100							7	
7664939	Sulfuric acid	06/91		2	1							
100210	Terephthalic acid	06/91		100	1							
79345	1,1,2,2-Tetrachloroethane	09/90	c	1	1	2		4				
-	Tetrachlorophenols (see Chlorophenols)											
7440280	Thallium	06/91		100							7	
*	Thallium compounds	06/91	c	100							7	[7]
62555	Thioacetamide		c	0.01			3	4	5			
62566	Thiourea		c	0.1	1		3	4	5			
7550450	Titanium tetrachloride	06/91		100	1	2						
108883	Toluene			200	1	2		4		6		
-	2,4-Toluenediamine (see 2,4-Diaminotoluene)											
26471625	Toluene diisocyanates including but not limited to:	06/91	c	0.1	1		3					
584849	Toluene-2,4-diisocyanate		c	0.1	1	2	3		5			
91087	Toluene-2,6-diisocyanate		c	0.1	1	2	3		5			
95534	o-Toluidine		c	10	1	2	3	4	5			
8001352	Toxaphene {Polychlorinated camphenes}		c	100	1	2	3	4	5			
-	1,1,1-Trchloroethane (see Methyl chloroform)											
79005	1,1,2-Trichloroethane {Vinyl trichloride}	06/91	c	1	1	2		4				
79016	Trichloroethylene		c	20	1	2		4				
-	2,4,6-Trichlorophenol (see Chlorophenols)											
96184	1,2,3-Trichloropropane	07/96	c	200			3	4			7	
121448	Triethylamine	06/91		20	1	2						
1582098	Trifluralin	06/91		100	1	2						

Substances for Which Emissions Must Be Quantified												
Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3]	**Carcinogen** [Note 4]	**Applicable Degree of Accuracy (lb/yr)** [Note 5]	**Source List(s)** [Note 6]							**Other Note(s)**
25551137	Trimethylbenzenes including but not limited to:	11/06		100	1							
95636	1,2,4-Trimethylbenzene	06/91		5	1							
540841	2,2,4-Trimethylpentane	06/91		100	1	2						
51796	Urethane {Ethyl carbamate}		c	0.1	1	2	3	4	5			
7440622	Vanadium (fume or dust)	06/91		10							7	[17]
1314621	Vanadium pentoxide	11/06		10		2						
108054	Vinyl acetate	06/91		200	1	2						
593602	Vinyl bromide		c	20	1	2	3	4				
75014	Vinyl chloride		c	0.5	1	2	3	4	5			
100403	4-Vinylcyclohexene	07/96	c	5			3					
75025	Vinyl fluoride	07/96	c	200			3					
75354	Vinylidene chloride			20	1	2						
1206	Wood preservatives (containing arsenic and chromate)	09/89		100						6		
1330207	Xylenes (mixed**)** including:			200	1	2				6		
108383	m-Xylene	06/91		200	1	2						
95476	o-Xylene	06/91		200	1	2						
106423	p-Xylene	06/91		200	1	2						
7440666	Zinc			2		2						
*	Zinc compounds including but not limited to:	09/89		2	1	2						[7]
1314132	Zinc oxide			2		2						[7]

Appendix A-II

Substances for Which Production, Use, or Other Presence Must Be Reported

Substances for Which Production, Use, or Other Presence Must be Reported

Emittent ID [Note 1]	Substance Name [Note 2]	Add Date [Note 3]	Carcinogen [Note 4]	Source List(s) [Note 6]							Other Note(s)
26148685	A-alpha-C {2-Amino-9H-pyrido[2,3-b]indole}	09/89	c			3	4				[18]
34256821	Acetochlor	09/89	c				4				
62476599	Acifluorfen [POM]	09/90	c	1	2		4				
3688537	AF-2		c			3	4				
1000	Aflatoxins		c			3	4	5			
15972608	Alachlor	09/89	c				4				
309002	Aldrin	09/89	c				4				
107186	Allyl alcohol	06/91								7	
60093	p-Aminoazobenzene {4-Aminoazobenzene} [POM]		c	1	2	3	4				
97563	o-Aminoazotoluene [POM]		c	1	2	3	4	5			
6109973	3-Amino-9-ethylcarbazole hydrochloride [POM]	09/89	c	1	2		4	5			
125848	Aminoglutethimide	09/90					4				
82280	1-Amino-2-methylanthraquinone [PAH-Derivative, POM]		c	1	2		4	5			
68006837	2-Amino-3-methyl-9H-pyrido(2,3-b) indole {MeA-alpha-C}	09/89	c			3	4				
712685	2-Amino-5-(5-nitro-2-furyl)-1,3,4-thiadiazole		c			3	4				
134292	o-Anisidine hydrochloride		c				4	5			
104949	p-Anisidine	06/91								7	
140578	Aramite		c			3	4				
492808	Auramine [POM]		c	1	2	3	4	5			
446866	Azathioprine		c	1	2	3	4	5			
103333	Azobenzene [POM]	09/90	c	1	2		4				
98873	Benzal chloride	06/91								7	
55210	Benzamide	06/91								7	
1694093	Benzyl violet 4B [POM]		c	1	2	3	4				
1025	Betel quid with tobacco		c			3	4				
494031	N-N-Bis(2-chloroethyl)-2-naphthylamine {Chlornaphazine} [PAH-Derivative, POM]		c	1	2	3	4	5			
108601	Bis(2-chloro-1-methylethyl) ether	06/91								7	
1030	Bitumens, extracts of steam-refined and air-refined bitumens		c			3	4				
1035	Bleomycins		c			3					
75274	Bromodichloromethane	09/90	c				4				

Substances for Which Production, Use, or Other Presence Must be Reported

Emittent ID [Note 1]	Substance Name [Note 2]	Add Date [Note 3]	Carcinogen [Note 4]	Source List(s) [Note 6]							Other Note(s)
1689845	Bromoxynil	06/91					4				
25013165	Butylated hydroxyanisole {BHA}		c			3	4				
123728	Butyraldehyde	06/91								7	
3068880	beta-Butyrolactone		c			3	4				
630080	Carbon monoxide	09/89					4				
143500	Chlordecone {Kepone}		c			3	4				
6164983	Chlordimeform	09/89	c				4				
115286	Chlorendic acid	09/89	c			3	4	5			
124481	Chlorodibromomethane	09/90	c				4				
563473	3-Chloro-2-methylpropene	09/89	c				4	5			
1065	Chlorophenoxy herbicides		c			3					
1897456	Chlorothalonil	09/89	c				4				
1059	p-Chloro-o-toluidine (strong acid salts)	06/91	c			3					
4680788	C. I. Acid Green 3 [POM] Note: "C.I." means "color index"	06/91		1	2					7	
569642	C. I. Basic Green 4 [POM]	06/91		1	2					7	
989388	C. I. Basic Red 1 [POM]	06/91		1	2					7	
569619	C. I. Basic Red 9 monohydrochloride [POM]	09/89	c	1	2		4	5			
2832408	C. I. Disperse Yellow 3 [POM]	06/91		1	2					7	
87296	Cinnamyl anthranilate [POM]	09/89	c	1	2		4	5			
6358538	Citrus Red No. 2 [POM]		c	1	2	3	4				
8007452	Coal tars	09/89	c			3	4	5			
21725462	Cyanazine	09/90					4				
14901087	Cycasin		c			3	4				
13121705	Cyhexatin	09/89					4	5			
3468631	D and C Orange No. 17 [PAH-Derivative, POM]	09/90	c	1	2		4				
81889	D and C Red No. 19 [POM]	09/90	c	1	2		4				
2092560	D and C Red No. 8 [PAH-Derivative, POM]	06/91	c	1	2		4				
5160021	D and C Red No. 9 [PAH-Derivative, POM]	09/90	c	1	2		4				
1596845	Daminozide	09/90	c				4				
50293	DDT {1,1,1-Trichloro-2,2-bis(p-chlorophenyl)ethane} [POM]		c	1	2	3	4	5			
613354	N,N'-Diacetylbenzidine [POM]		c	1	2	3	4				
2303164	Diallate	06/91								7	
39156417	2,4-Diaminoanisole sulfate		c				4	5			

Substances for Which Production, Use, or Other Presence Must be Reported

Emittent ID [Note 1]	Substance Name [Note 2]	Add Date [Note 3]	Carcinogen [Note 4]	Source List(s) [Note 6]							Other Note(s)
101804	4,4'-Diaminodiphenyl ether [POM]		c	1	2	3	4	5			
764410	1,4-Dichloro-2-butene	09/90	c				4				
28434868	3,3'-Dichloro-4,4'-diaminodiphenyl ether [POM]	09/89	c	1	2	3	4				
72548	Dichlorodiphenyldichloroethane {DDD} [POM]	09/89	c	1	2		4				
540590	1,2-Dichloroethylene	06/91								7	
78886	2,3-Dichloropropene	06/91								7	
60571	Dieldrin	09/89	c				4				
1464535	Diepoxybutane		c			3	4	5			
1615801	1,2-Diethylhydrazine		c			3	4				
84662	Diethyl phthalate	06/91								7	
101906	Diglycidyl resorcinol ether {DGRE}		c			3	4	5			
94586	Dihydrosafrole		c			3	4				
20325400	3,3'-Dimethoxybenzidine dihydrochloride [POM]	06/91	c	1	2		4				
55738540	trans-2-[(Dimethylamino)methylimino]-5-[2-(5-nitro-2-furyl)vinyl-1,3,4-oxadiazol		c			3	4				
540738	1,2-Dimethylhydrazine		c			3	4				
105679	2,4-Dimethylphenol {2,4-Xylenol}	06/91								7	
513371	Dimethylvinylchloride {DMVC}	09/89	c				4	5			
25154545	Dinitrobenzenes (mixtures of) including:	09/90					4			7	
99650	m-Dinitrobenzene	06/91								7	
528290	o-Dinitrobenzene	06/91								7	
100254	p-Dinitrobenzene	06/91								7	
39300453	Dinocap	09/90					4				
88857	Dinoseb	09/89					4				
117840	n-Dioctyl phthalate	06/91								7	
2475458	Disperse Blue 1 [PAH-Derivative, POM]	06/91	c	1	2	3	4				
541413	Ethyl chloroformate	06/91								7	
62500	Ethyl methanesulfonate		c			3	4				
2164172	Fluometuron	06/91								7	
133073	Folpet	09/89	c				4				
3570750	2-(2-Formylhydrazino)-4-(5-nitro-2-furyl)thiazole		c			3	4				
60568050	Furmecyclox	09/90	c				4				
67730114	Glu-P-1 {2-Amino-6-methyldipyrido[1,2-a:3',2'-d]imidazole}		c			3	4				

Substances for Which Production, Use, or Other Presence Must be Reported

Emittent ID [Note 1]	Substance Name [Note 2]	Add Date [Note 3]	Carcinogen [Note 4]	Source List(s) [Note 6]							Other Note(s)
67730103	Glu-P-2 {2-Aminodipyrido[1,2-a:3',2'-d]imidazole}		c			3	4				
765344	Glycidaldehyde		c			3	4				
556525	Glycidol	09/90	c				4				
16568028	Gyromitrin {Acetaldehyde methylformylhydrazone}		c				4				
2784943	HC Blue 1	09/89	c				4	5			
1024573	Heptachlor epoxide	09/89	c				4				
1335871	Hexachloronaphthalene [PAH-Derivative, POM]	06/91		1	2					7	
10034932	Hydrazine sulfate		c				4	5			
76180966	IQ {2-Amino-3-methylimidazo[4,5-f]quinoline}		c			3	4				
78842	Isobutyraldehyde	06/91								7	
120581	Isosafrole	09/90	c				4				
4759482	Isotretinoin						4				
77501634	Lactofen [POM]	09/89	c	1	2		4				
1131	Lubricant base oils and derived products, specifically vacuum distillates, acid treated oils, aromatic oils, mildly solvent-refined oils, mildly hydrotreated-oils and used engine oils.	09/89	c			3	4	5			
8018017	Mancozeb	09/90	c				4				
12427382	Maneb	09/90	c				4				
59052	Methotrexate	09/89					4				
96333	Methyl acrylate	06/91								7	
590965	Methylazoxymethanol	09/90	c				4				
592621	Methylazoxymethanol acetate	09/89	c			3	4				
101611	4,4'-Methylene bis (N,N-dimethyl) benzenamine [POM]		c	1	2		4	5			
838880	4,4'-Methylene bis(2-methylaniline) [POM]	09/89	c	1	2	3	4				
74953	Methylene bromide	06/91								7	
66273	Methyl methanesulfonate		c			3	4				
129157	2-Methyl-1-nitroanthraquinone (uncertain purity) [PAH-Derivative, POM]		c	1	2	3	4				
70257	N-Methyl-N'-nitro-N-nitrosoguanidine		c			3	4				
-	N-Methyl-N-nitrosourethane (see N-Nitroso-N-methylurethane)										
924425	N-Methyloacrylamide	09/90	c				4				
9006422	Metiram	09/90					4				
1140	Mineral oils (untreated and mildly treated oils; and those used in occupations such as mulespinning, metal machining, and jute		c			3	4	5			

Substances for Which Production, Use, or Other Presence Must be Reported

Emittent ID [Note 1]	Substance Name [Note 2]	Add Date [Note 3]	Carcinogen [Note 4]	Source List(s) [Note 6]							Other Note(s)
	processing).										
2385855	Mirex		c			3	4	5			
315220	Monocrotaline		c			3	4				
505602	Mustard gas {Sulfur mustard}		c			3	4	5			
134327	1-Naphthylamine [PAH-Derivative, POM]	09/90	c	1	2		4				
91598	2-Naphthylamine [PAH-Derivative, POM]		c	1	2	3	4	5			
54115	Nicotine	09/90					4				
1148	Nitrilotriacetic acid (salts) including but not limited to:	06/91	c			3					
18662538	Nitrilotriacetic acid, trisodium salt monohydrate	06/91	c				4				
99592	5-Nitro-o-anisidine		c				4	5			
1836755	Nitrofen (technical grade)		c			3	4	5			
51752	Nitrogen mustard {Mechlorethamine}	09/89	c			3	4	5			
55867	Nitrogen mustard hydrochloride	06/91	c				4	5			
55630	Nitroglycerin	06/91								7	
88755	2-Nitrophenol	06/91								7	
57835924	4-Nitropyrene [PAH-Derivative, POM]	09/89	c	1	2	3	4				
759739	N-Nitroso-N-ethylurea	09/89	c				4	5			
60153493	3-(N-Nitrosomethylamino)propionitrile	09/89	c			3	4				
64091914	4-(N-Nitrosomethylamino)-1-(3-pyridyl)-1-butanone {NNK}		c			3	4				
615532	N-Nitroso-N-methylurethane		c			3	4				
4549400	N-Nitrosomethylvinylamine		c			3	4	5			
16543558	N-Nitrosonornicotine		c			3	4	5			
13256229	N-Nitrososarcosine		c			3	4	5			
303479	Ochratoxin A [POM]	09/90	c	1	2		4				
2234131	Octachloronaphthalene [PAH-Derivative, POM]	06/91		1	2					7	
2646175	Oil Orange SS [PAH-Derivative, POM]		c	1	2	3	4				
20816120	Osmium tetroxide	06/91								7	
794934	Panfuran S {Dihydroxymethylfuratrizine}		c			3	4				
122601	Phenyl glycidyl ether	09/90	c			3	4				
57410	Phenytoin [POM]		c	1	2	3	4	5			
88891	Picric acid	06/91								7	
1155	Polybrominated biphenyls {PBBs{ [POM]		c	1	2	3	4	5			

Substances for Which Production, Use, or Other Presence Must be Reported

Emittent ID [Note 1]	Substance Name [Note 2]	Add Date [Note 3]	Carcinogen [Note 4]	Source List(s) [Note 6]							Other Note(s)
53973981	Polygeenan	09/89	c				4				
3761533	Ponceau MX [PAH-Derivative, POM]		c	1	2	3	4				
3564098	Ponceau 3R [PAH-Derivative, POM]		c	1	2	3	4				
36791045	Ribavirin	09/90					4				
94597	Safrole		c			3	4	5			
1180	Shale oils		c			3	4				
132274	Sodium o-phenylphenate [POM]		c	1	2	3	4				
128449	Sodium saccharin	09/89	c				4				
1185	Soots		c			3	4				
10048132	Sterigmatocystin [POM]		c	1	2	3	4				
95067	Sulfallate		c			3	4	5			
5216251	p-alpha,alpha,alpha-Tetrachlorotoluene	09/90	c				4				
961115	Tetrachlorvinphos	06/91								7	
509148	Tetranitromethane	09/90	c				4				
139651	4,4'-Thiodianiline [POM]		c	1	2	3	4				
1314201	Thorium dioxide		c				4	5			
1200	Tobacco products, smokeless		c			3	4				
1205	alpha-chlorinated Toluenes		c			3					
636215	o-Toluidine hydrochloride		c				4	5			
106490	p-Toluidine	09/90	c				4				
52686	Trichlorfon	06/91								7	
68768	Tris(aziridinyl)-p-benzoquinone {Triaziquone}	09/90	c				4				
52244	Tris(1-aziridinyl) phosphine sulfide {Thiotepa}		c			3	4	5			
126727	Tris(2,3-dibromopropyl)phosphate	09/89	c				4				
62450060	Trp-P-1 {3-Amino-1,4-dimethyl-5H-pyrido[4,3-b]indole}		c			3	4				
62450071	Trp-P-2 {3-Amino-1-methyl-5H-pyrido[4,3-b]indole}		c			3	4				
72571	Trypan blue [PAH-Derivative, POM]		c	1	2	3	4				
106876	4-Vinyl-1-cyclohexene diepoxide {Vinyl cyclohexene dioxide}	09/90	c				4				
81812	Warfarin [POM]			1	2		4				
87627	2,6-Xylidene	06/91					4				
12122677	Zineb	09/90	c				4				

Appendix A-III

Substances Which Need Not Be Reported Unless Manufactured By the Facility

Substances Which Need Not Be Reported Unless Manufactured By the Facility									
Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3])	**Carcinogen** [Note 4]	**Source Lists** [Note 6]					**Other Note(s)**
546883	Acetohydroxamic acid	09/90					4		
50760	Actinomycin D	09/90	c				4		
23214928	Adriamycin [PAH-Derivative, POM]		c	1	2	3	4	5	
28981977	Alprazolam [POM]	09/90		1	2		4		
39831555	Amikacin sulfate	09/90					4		
54626	Aminopterin						4		
1005	Analgesic mixtures containing phenacetin		c			3	4	5	
1010	Androgenic (anabolic) steroids including but not limited to:		c			3	4		
58184	Methyltestosterone	09/90					4		
434071	Oxymetholone		c				4	5	
58220	Testosterone and its esters including but not limited to:	09/89					4		
315377	Testosterone enanthate	09/90					4		
50782	Aspirin	06/91					4		
115026	Azaserine		c			3	4		
5411223	Benzphetamine hydrochloride [POM]	09/90		1	2		4		
154938	Bischloroethyl nitrosourea		c			3	4		
55981	1,4-Butanediol dimethanesulfonate {Busulfen/Myleran}		c			3	4	5	
41575944	Carboplatin	09/90					4		
474259	Chenodiol	09/90					4		
305033	Chlorambucil		c			3	4	5	
56757	Chloramphenicol		c			3	4		
1620219	Chlorcyclizine hydrochloride [POM]			1	2		4		
13010474	1-(2-Chloroethyl)-3-cyclohexyl-1-nitrosourea {CCNU}		c			3	4	5	
13909096	1-(2-Chloroethyl)-3-(4-methylcyclohexyl)-1-nitrosourea {Methyl CCNU}		c			3			
15663271	Cisplatin		c			3	4		
50419	Clomiphene citrate [POM]	09/90		1	2		4		
50180	Cyclophosphamide		c			3	4		
147944	Cytarabine	09/89					4		
4342034	Dacarbazine		c			3	4	5	
17230885	Danazol	09/90					4		
20830813	Daunomycin [PAH-Derivative, POM]		c	1	2	3	4		
23541506	Daunorubicin hydrochloride [PAH-Derivative, POM]	09/90		1	2		4		
84173	Dienestrol [POM]	09/90	c	1	2		4		

Substances Which Need Not Be Reported Unless Manufactured By the Facility									
Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3])	**Carcinogen** [Note 4]	**Source Lists** [Note 6]					**Other Note(s)**
564250	Doxycycline	09/90					4		
379793	Ergotamine tartrate [POM]	09/90		1	2		4		
1095	Estrogens, non-steroidal including but not limited to:		c			3		5	
56531	Diethylstilbestrol [POM]		c	1	2	3	4	5	
1100	Estrogens, steroidal including but not limited to:		c			3		5	
1068	Conjugated estrogens	09/90	c				4		
50282	Estradiol 17 beta		c				4	5	
53167	Estrone		c				4	5	
57636	Ethinyl estradiol		c				4	5	
72333	Mestranol		c			3	4	5	
33419420	Etoposide [POM]	09/90			2				
54350480	Etretinate						4		
51218	Fluorouracil	09/89					4		
76437	Fluoxymesterone	09/90					4		
13311847	Flutamide	09/90					4		
67458	Furazolidone	09/90	c				4		
126078	Griseofulvin		c			3	4		
23092173	Halazepam [POM]	09/90		1	2		4		
3778732	Ifosfamide	09/90					4		
9004664	Iron dextran complex		c			3	4	5	
303344	Lasiocarpine	09/89	c			3	4		
554132	Lithium carbonate	06/91					4		
919164	Lithium citrate	06/91					4		
846491	Lorazepam [POM]	09/90		1	2		4		
595335	Megestrol acetate	06/91					4		
148823	Melphalan		c			3	4	5	
9002680	Menotropins	09/90					4		
6112761	Mercaptopurine	09/90					4		
531760	Merphalan	09/89	c				4		
3963959	Methacycline hydrochloride	06/91					4		
60560	Methimazole	09/90					4		
15475566	Methotrexate sodium	09/90					4		
484208	5-Methoxypsoralen		c			3			

Substances Which Need Not Be Reported Unless Manufactured By the Facility									
Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3])	**Carcinogen** [Note 4]	**Source Lists** [Note 6]					**Other Note(s)**
56042	Methylthiouracil		c			3	4		
443481	Metronidazole		c			3	4	5	
59467968	Midazolam hydrochloride [POM]	09/90		1	2		4		
62015398	Misoprostol	09/90					4		
50077	Mitomycin C		c			3	4		
70476823	Mitoxantrone hydrochloride [PAH-Derivative, POM]	09/90		1	2		4		
139913	5-(Morpholinomethyl)-3-[(5-nitrofurfurylidene)amino]-2-oxazolidinone		c			3	4		
86220420	Nafarelin acetate [PAH-Derivative, POM]	09/90		1	2		4		
3771195	Nafenopin [POM]		c	1	2	3	4		
1405103	Neomycin sulfate	09/90					4		
56391572	Netilmicin sulfate	09/90					4		
61574	Niridazole		c			3	4		
67209	Nitrofurantoin	06/91	c				4		
59870	Nitrofurazone	09/90	c				4		
555840	1-[(5-Nitrofurfurylidene)amino]-2-imidazolidinone		c			3	4		
531828	N-[4-(5-Nitro-2-furyl)-2-thiazolyl]acetamide		c			3	4		
6533002	Norgestrel	09/90					4		
79572	Oxytetracycline	06/91					4		
115673	Paramethadione	09/90					4		
52675	Penicillamine	06/91					4		
57330	Pentobarbital sodium	09/90					4		
63989	Phenacemide	09/90					4		
62442	Phenacetin		c			3	4	5	
94780	Phenazopyridine hydrochloride		c			3	4	5	
3546109	Phenesterin	09/89	c				4	5	
50066	Phenobarbital		c			3	4		
59961	Phenoxybenzamine [POM]	09/89	c	1	2		4		
63923	Phenoxybenzimide hydrochloride [POM]	09/90	c	1	2	3	4	5	
54911	Pipobroman	09/90					4		
18378897	Plicamycin [PAH-Derivative, POM]	09/90		1	2		4		
366701	Procarbazine hydrochloride		c			3	4	5	
57830	Progesterone		c			3	4	5	
1160	Progestins including but not limited to:		c			3			

Substances Which Need Not Be Reported Unless Manufactured By the Facility									
Emittent ID [Note 1]	**Substance Name** [Note 2]	**Add Date** [Note 3])	**Carcinogen** [Note 4]	**Source Lists** [Note 6]					**Other Note(s)**
71589	Medroxyprogesterone acetate		c			3	4		
68224	Norethisterone		c				4	5	
51525	Propylthiouracil		c			3	4	5	
302794	all-trans-Retinoic acid	09/89					4		
1167	Retinol/retinyl esters	09/89	c				4		
81072	Saccharin		c			3	4	5	
3810740	Streptomycin sulfate	06/91					4		
18883664	Streptozotocin		c			3	4	5	
54965241	Tamoxifen citrate [POM]	09/90		1	2		4		
846504	Temazepam [POM]	09/90		1	2		4		
64755	Tetracycline hydrochloride	06/91					4		
50351	Thalidomide						4		
154427	Thioguanine	09/90					4		
49842071	Tobramycin sulfate	09/90					4		
299752	Treosulfan		c			3	4		
28911015	Triazolam [POM]	09/90		1	2		4		
13647353	Trilostane	09/90					4		
127480	Trimethadione	06/91					4		
66751	Uracil mustard		c			3	4		
26995915	Urofollitropin	09/90					4		
99661	Valproate						4		
143679	Vinblastine sulfate [POM]	09/90		1	2		4		
2068782	Vincristine sulfate [POM]	09/90		1	2		4		

NOTES TO APPENDIX A:

[1] Emittent ID (the emittent identification number) is the Chemical Abstract Service (CAS) number where available, or an ARB-assigned 4-digit emittent ID code.

A dash ("-") is shown for the Emittent ID for substances which are alphabetized under a group header or synonym elsewhere on the list. Refer to the cross reference indicated in parenthesis, "( )".

A double dash ("- -") is shown for the Emittent ID to indicate that the entry is a non-reportable group header for the substances immediately following it.

An asterisk ("*") is shown for the Emittent ID to indicate that the emissions of unspecified metal compounds shall be reported as the metal atom equivalent. See Note [7].

A pound sign ("#") is shown for the Emittent ID to indicate that the individual, component listed substances must be reported for this mixture or group.

[2] Individual substances listed under a group heading must be reported individually. Other, unspecified substances in the group must be summed and reported using the emittent ID of the group heading.

The square bracket designation, "[ ]", indicates that the substance is a component of the chemical group heading(s) within the brackets.

The braces designation, "{ }", indicates a synonym for the substance listed.

[3] The date the Board approved addition of the substance to the original list. The original list was approved by the Board in July 1988.

[4] The letter "c" indicates that for purposes of this section the substance shall be treated as a human carcinogen or potential human carcinogen.

[5] Applicable degree of accuracy (in lbs/year except where noted). Radionuclides must be reported in Curie units, and the accuracy must be considered accordingly. Refer to section VII.E. and Appendix B.

[6] Substances are required to be included on the Hot Spots list based on the following lists cited in Health & Safety Code section 44321:

1 = California Air Resources Board (44321(c));

2 = Environmental Protection Agency (44321(e));

3 = International Agency for Research on Cancer;

4 = Governor's List of Carcinogens and Reproductive Toxicants; (44321(a); Labor Code section 6382(b)(1)): (44321(b); HSC section 25249.8):

5 = National Toxicology Program (44321(a));

6 = Hazard Evaluation System and Information Service (44321(d));

7 = Added pursuant to HSC section 44321 (f).

[7] Emissions of unspecified metal compounds shall be reported as the amount of the metal atom equivalent, using the metal emittent identification number for the metal itself (or the emittent identification number indicated on the table, such as for reporting inorganic versus other-than-inorganic arsenic compounds).

For unspecified metal compounds which contain two or more listed metals (e.g., zinc chromate), each component metal shall be reported as the amount of the appropriate metal atom equivalent (i.e., the zinc portion of the weight as zinc equivalent and the chromate portion as hexavalent chromium equivalent.

For specific, individually listed metal compounds (e.g., Lead chromate), emissions shall be reported for the compound (as pounds of whole compound), using the emittent identification number for that compound.

[8] Compounds of the form "X-CN", where formal dissociation can occur. Report as the amount of Cyanide equivalent in the compound using an emittent identification code of 1073.

[9] Emissions of these mixtures shall be reported as emissions of total particulate matter and total organic gas, using the following emittent identification numbers:

9901 Diesel exhaust, particulate matter 9910 Gasoline exhaust, particulate matter

9902 Diesel exhaust, total organic gas

9911 Gasoline exhaust, total organic gas

Individually listed substances from gasoline exhaust must also be reported. Emissions of diesel engine exhaust particulate matter (diesel PM), shall be reported as diesel PM using emittent ID 9901.

[10] The emittent identification number 1105 has been discontinued for all facilities reporting for the first time and for all updates. Use the listed replacement emittent identification codes 1103 and 1104.

[11] Emissions of the individual, component listed substances must be reported in addition to the total gasoline vapors emissions.

[12] These lead compounds are listed here so that the inorganic lead fraction will be quantified and reported if these individual compounds cannot be quantified.

[13] PAH: (Polycyclic Aromatic Hydrocarbon) - An organic compound consisting of a fused ring structure containing at least two (2) benzene rings, and which may also contain additional fused rings not restricted exclusively to hexagonal rings.

The structure does not include any heteroatoms or substituent groups. The structure includes only carbon and hydrogen.

PAHs are a subgroup of POM and have a boiling point of greater than or equal to 100 C.

[14] PAH-DERIVATIVE: (Polycyclic Aromatic Hydrocarbon Derivative) - An organic compound consisting of a fused ring structure containing at least two (2) benzene rings, and which may also contain additional fused rings not restricted exclusively to hexagonal rings. The fused ring structure does not contain heteroatoms. The structure does contain one or more substituent groups.

PAH-Derivatives are a subgroup of POM and have a boiling point of greater than or equal to 100 C.

[15] POM: (Polycyclic Organic Matter) - Includes organic compounds with more than one benzene ring, and which have a boiling point of greater than or equal to 100 C.

[16] Radionuclides and other radioactive substances shall be reported in units of Curies per year (for annual average emissions) and in units of milliCuries per hour (for maximum hourly emissions).

[17] Emissions of Vanadium (fume or dust) shall be reported as the amount of the vanadium atom equivalent, using the identification number 7440622.

[18] The emittent identification number 1001 has been replaced with the CAS number 26148685.

NOTE: The notation “11/06” indicates most recently added substances.

Appendix B: Regulations and Legislation

B.1. Air Toxics Hot Spots Program Overview

(Air Resources Board, 2011: see http://www.arb.ca.gov/ab2588/overview.htm)

INTRODUCTION

The Air Toxics "Hot Spots" Information and Assessment Act (AB 2588, 1987, Connelly) was enacted in September 1987. Under this, stationary sources are required to report the types and quantities of certain substances their facilities routinely release into the air. Emissions of interest are those that result from the routine operation of a facility or that are predictable, including but not limited to continuous and intermittent releases and process upsets or leaks.

The goals of the Air Toxics "Hot Spots" Act are to collect emission data, to identify facilities having localized impacts, to ascertain health risks, and to notify nearby residents of significant risks. In September 1992, the "Hot Spots" Act was amended by Senate Bill (SB) 1731 (Calderon) to address the reduction of significant risks. The bill requires that owners of significant-risk facilities reduce their risks below the level of significance.

The Act requires that toxic air emissions from stationary sources (facilities) be quantified and compiled into an inventory according to criteria and guidelines developed by the ARB, that each facility be prioritized to determine whether a risk assessment must be conducted, that the risk assessments be conducted according to methods developed by the Office of Environmental Health Hazard Assessment (OEHHA), that the public be notified of significant risks posed by nearby facilities, and that emissions which result in a significant risk be reduced. Since the amendment of the statute in 1992 by enactment of SB 1731, facilities that pose a potentially significant health risks to the public are required to reduce their risks, thereby reducing the near-source exposure of Californians to toxic air pollutants. Owners of facilities found to pose significant risks by a district must prepare and implement risk reduction audit and plans within 6 months of the determination.

The Air Resources Board (ARB) is required to develop a program to make the emission data collected under the "Hot Spots" Program available to the public. If requested, districts must make health risk assessments available for public review. Districts must also publish annual reports which summarize the health risk assessment program, rank facilities according to the cancer risk posed, identify the facilities posing non-cancer health risks, and describe the status of the development of control measures.

The "Hot Spots" Program has complemented the ARB's existing air toxics identification and control programs. It has located sources of substances not previously under evaluation, and it has provided exposure information necessary to prioritize substances for control measures and develop regulatory action. Also, the preparation of the "Hot Spots" emission inventory made facility owners aware of their toxics problems. As a result, facilities have taken voluntary steps to reduce emissions of air toxics. Limited district and facility surveys have identified voluntary reductions of over 1.9 million pounds per year in the emission of air toxics from just 21 facilities in California. The benefits that come from this type of action are less risk to workers and to the public, reduced operation costs, demonstration of emission reduction options for other sources, and improved community relations.

The Act was further modified by AB 564, chaptered on September 19, 1996. The passage of AB 564 amended the Hot Spots statute in several ways, including adding provisions that: exempt specified low priority facilities from further compliance with the Hot Spots program; reinstate exempted facilities if specified criteria are met; specify an alternative evaluation process for facilities subject to district permit programs; and other changes to exempt specified facilities from further compliance with the Hot Spots Program.

B.2. Health and Safety Code Related to Air Toxics Hot Spots.

PART 6. AIR TOXICS "HOT SPOTS" INFORMATION AND ASSESSMENT
(Part 6 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384. Note: Sections 44380 and 44384 became operative Jan. 1, 1988.)

CHAPTER 1: LEGISLATIVE FINDINGS AND DEFINITIONS

44300. This part shall be known and may be cited as the Air Toxics "Hot Spots" Information and Assessment Act of 1987.

44301. The Legislature finds and declares all of the following:

(a) In the wake of recent publicity surrounding planned and unplanned releases of toxic chemicals into the atmosphere, the public has become increasingly concerned about toxics in the air.

(b) The Congressional Research Service of the Library of Congress has concluded that 75 percent of the United States population lives in proximity to at least one facility that manufactures chemicals. An incomplete 1985 survey of large chemical companies conducted by the Congressional Research Service documented that nearly every chemical plant studied routinely releases into the surrounding air significant levels of substances proven to be or potentially hazardous to public health.

(c) Generalized emissions inventories compiled by air pollution control districts and air quality management districts in California confirm the findings of the Congressional Research Service survey as well as reveal that many other facilities and businesses which do not actually manufacture chemicals do use hazardous substances in sufficient quantities to expose, or in a manner that exposes, surrounding populations to toxic air releases.

(d) These releases may create localized concentrations or air toxics "hot spots" where emissions from specific sources may expose individuals and population groups to elevated risks of adverse health effects, including, but not limited to, cancer and contribute to the cumulative health risks of emissions from other sources in the area. In some cases where large populations may not be significantly affected by adverse health risks, individuals may be exposed to significant risks.

(e) Little data is currently available to accurately assess the amounts, types, and health impacts of routine toxic chemical releases into the air. As a result, there exists significant uncertainty about the amounts of potentially hazardous air pollutants which are released, the location of those releases, and the concentrations to which the public is exposed.

(f) The State of California has begun to implement a long-term program to identify, assess, and control ambient levels of hazardous air pollutants, but additional legislation is needed to provide for the collection and evaluation of information concerning the amounts, exposures, and short- and long-term health effects of hazardous substances regularly released to the surrounding atmosphere from specific sources of hazardous releases.

(g) In order to more effectively implement control strategies for those materials posing an unacceptable risk to the public health, additional information on the sources of potentially hazardous air pollutants is necessary.
(h) It is in the public interest to ascertain and measure the amounts and types of hazardous releases and potentially hazardous releases from specific sources that may be exposing people to those releases, and to assess the health risks to those who are exposed.

44302. The definitions set forth in this chapter govern the construction of this part.

44303. "Air release" or "release" means any activity that may cause the issuance of air contaminants, including the actual or potential spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing of a substance into the ambient air and that results from the routine operation of a facility or that is predictable, including, but not limited to, continuous and intermittent releases and predictable process upsets or leaks.

44304. "Facility" means every structure, appurtenance, installation, and improvement on land which is associated with a source of air releases or potential air releases of a hazardous material.

44306. "Health risk assessment" means a detailed comprehensive analysis prepared pursuant to Section 44361 to evaluate and predict the dispersion of hazardous substances in the environment and the potential for exposure of human populations and to assess and quantify both the individual and populationwide health risks associated with those levels of exposure.

44307. "Operator" means the person who owns or operates a facility or part of a facility.

44308. "Plan" means the emissions inventory plan which meets the conditions specified in Section 44342.

44309. "Report" means the emissions inventory report specified in Section 44341.

CHAPTER 2: FACILITIES SUBJECT TO THIS PART

44320. This part applies to the following:
(a) Any facility which manufactures, formulates, uses, or releases any of the substances listed pursuant to Section 44321 or any other substance which reacts to form a substance listed in Section 44321 and which releases or has the potential to release total organic gases, particulates, or oxides of nitrogen or sulfur in the amounts specified in Section 44322.
(b) Except as provided in Section 44323, any facility which is listed in any current toxics use or toxics air emission survey, inventory, or report released or compiled by a district. A district may, with the concurrence of the state board, waive the application of this part pursuant to this subdivision for any facility which

the district determines will not release any substance listed pursuant to Section 44321 due to a shutdown or a process change.

44321. For the purposes of Section 44320, the state board shall compile and maintain a list of substances that contains, but is not limited to, all of the following:

(a) Substances identified by reference in paragraph (1) of subdivision (b) of Section 6382 of the Labor Code and substances placed on the list prepared by the National Toxicology Program and issued by the United States Secretary of Health and Human Services pursuant to paragraph (4) of subsection (b) of Section 241 of Title 42 of the United States Code. For the purposes of this subdivision, the state board may remove from the list any substance which meets both of the following criteria:
  (1) No evidence exists that it has been detected in air.
  (2) The substance is not manufactured or used in California, or, if manufactured or used in California, because of the physical or chemical characteristics of the substance or the manner in which it is manufactured or used, there is no possibility that it will become airborne.

(b) Carcinogens and reproductive toxins referenced in or compiled pursuant to Section 25249.8, except those which meet both of the criteria identified in subdivision (a).

(c) Substances designated by the state board as toxic air contaminants pursuant to subdivision (b) of Section 39657 and substances on the candidate list of potential toxic air contaminants and the list of designated toxic air contaminants prepared by the state board pursuant to Article 3 (commencing with Section 39660) of Chapter 3.5 of Part 2, including, but not limited to, all substances currently under review and scheduled or nominated for review and substances identified and listed for which health effects information is limited.

(d) Substances for which an information or hazard alert has been issued by the repository of current data established pursuant to Section 147.2 of the Labor Code.

(e) Substances reviewed, under review, or scheduled for review as air toxics or potential air toxics by the Office of Air Quality Planning and Standards of the Environmental Protection Agency, including substances evaluated in all of the following categories or their equivalent: preliminary health and source screening, detailed assessment, intent to list, decision not to regulate, listed, standard proposed, and standard promulgated.

(f) Any additional substances recognized by the state board as presenting a chronic or acute threat to public health when present in the ambient air, including, but not limited to, any neurotoxicants or chronic respiratory toxicants not included within subdivision (a), (b), (c), (d), or (e).

44322. This part applies to facilities specified in subdivision (a) of Section 44320 in accordance with the following schedule:

(a) For those facilities that release, or have the potential to release, 25 tons per year or greater of total organic gases, particulates, or oxides of nitrogen or sulfur, this part becomes effective on July 1, 1988.

(b) For those facilities that release, or have the potential to release, more than 10 but less than 25 tons per year of total organic gases, particulates, or oxides of nitrogen or sulfur, this part becomes effective July 1, 1989.
(c) For those facilities that release, or have the potential to release, less than 10 tons per year of total organic gases, particulates, or oxides of nitrogen or sulfur, the state board shall, on or before July 1, 1990, prepare and submit a report to the Legislature identifying the classes of those facilities to be included in this part and specifying a timetable for their inclusion.

44323. A district may prepare an industrywide emissions inventory and health risk assessment for facilities specified in subdivision (b) of Section 44320 and subdivisions (a) and (b) of Section 44322, and shall prepare an industrywide emissions inventory for the facilities specified in subdivision (c) of Section 44322, in compliance with this part for any class of facilities that the district finds and determines meets all of the following conditions:
(a) All facilities in the class fall within one four-digit Standard Industrial Classification Code.
(b) Individual compliance with this part would impose severe economic hardships on the majority of the facilities within the class.
(c) The majority of the class is composed of small businesses.
(d) Releases from individual facilities in the class can easily and generically be characterized and calculated.

44324. This part does not apply to any facility where economic poisons are employed in their pesticidal use, unless that facility was subject to district permit requirements on or before August 1, 1987. As used in this section, "pesticidal use" does not include the manufacture or formulation of pesticides.

44325. Any solid waste disposal facility in compliance with Section 41805.5 is in compliance with the emissions inventory requirements of this part.

CHAPTER 3: AIR TOXICS EMISSION INVENTORIES

44340.
(a) The operator of each facility subject to this part shall prepare and submit to the district a proposed comprehensive emissions inventory plan in accordance with the criteria and guidelines adopted by the state board pursuant to Section 44342.
(b) The proposed plan shall be submitted to the district on or before August 1, 1989, except that, for any facility to which subdivision (b) of Section 44322 applies, the proposed plan shall be submitted to the district on or before August 1, 1990. The district shall approve, modify, and approve as modified, or return for revision and resubmission, the plan within 120 days of receipt.
(c) The district shall not approve a plan unless all of the following conditions are met:
(1) The plan meets the requirements established by the state board pursuant to Section 44342.

(2) The plan is designed to produce, from the list compiled and maintained pursuant to Section 44321, a comprehensive characterization of the full range of hazardous materials that are released, or that may be released, to the surrounding air from the facility. Air release data shall be collected at, or calculated for, the primary locations of actual and potential release for each hazardous material. Data shall be collected or calculated for all continuous, intermittent, and predictable air releases.
(3) The measurement technologies and estimation methods proposed provide state-of-the-art effectiveness and are sufficient to produce a true representation of the types and quantities of air releases from the facility.
(4) Source testing or other measurement techniques are employed wherever necessary to verify emission estimates, as determined by the state board and to the extent technologically feasible. All testing devices shall be appropriately located, as determined by the state board.
(5) Data are collected or calculated for the relevant exposure rate or rates of each hazardous material according to its characteristic toxicity and for the emission rate necessary to ensure a characterization of risk associated with exposure to releases of the hazardous material that meets the requirements of Section 44361. The source of all emissions shall be displayed or described.

44341. Within 180 days after approval of a plan by the district, the operator shall implement the plan and prepare and submit a report to the district in accordance with the plan. The district shall transmit all monitoring data contained in the approved report to the state board.

44342. The state board shall, on or before May 1, 1989, in consultation with the districts, develop criteria and guidelines for site-specific air toxics emissions inventory plans which shall be designed to comply with the conditions specified in Section 44340 and which shall include at least all of the following:

(a) For each class of facility, a designation of the hazardous materials for which emissions are to be quantified and an identification of the likely source types within that class of facility. The hazardous materials for quantification shall be chosen from among, and may include all or part of, the list specified in Section 44321.
(b) Requirements for a facility diagram identifying each actual or potential discrete emission point and the general locations where fugitive emissions may occur. The facility diagram shall include any nonpermitted and nonprocess sources of emissions and shall provide the necessary data to identify emission characteristics. An existing facility diagram which meets the requirements of this section may be submitted.
(c) Requirements for source testing and measurement. The guidelines may specify appropriate uses of estimation techniques including, but not limited to, emissions factors, modeling, mass balance analysis, and projections, except that source testing shall be required wherever necessary to verify emission estimates to the extent technologically feasible. The guidelines shall specify conditions and

locations where source testing, fence-line monitoring, or other measurement techniques are to be required and the frequency of that testing and measurement.

(d) Appropriate testing methods, equipment, and procedures, including quality assurance criteria.

(e) Specifications for acceptable emissions factors, including, but not limited to, those which are acceptable for substantially similar facilities or equipment, and specification of procedures for other estimation techniques and for the appropriate use of available data.

(f) Specification of the reporting period required for each hazardous material for which emissions will be inventoried.

(g) Specifications for the collection of useful data to identify toxic air contaminants pursuant to Article 2 (commencing with Section 39660) of Chapter 3.5 of Part 2.

(h) Standardized format for preparation of reports and presentation of data.

(i) A program to coordinate and eliminate any possible overlap between the requirements of this chapter and the requirements of Section 313 of the Superfund Amendment and Reauthorization Act of 1986 (Public Law 99-499). The state board shall design the guidelines and criteria to ensure that, in collecting data to be used for emissions inventories, actual measurement is utilized whenever necessary to verify the accuracy of emission estimates, to the extent technologically feasible.

44343. The district shall review the reports submitted pursuant to Section 44341 and shall, within 90 days, review each report, obtain corrections and clarifications of the data, and notify the Office of Environmental Health Hazard Assessment, the Department of Industrial Relations, and the city or county health department of its findings and determinations as a result of its review of the report.

44344. Except as provided in Section 44391, emissions inventories developed pursuant to this chapter shall be updated every four years, in accordance with the procedures established by the state board. Those updates shall take into consideration improvements in measurement techniques and advancing knowledge concerning the types and toxicity of hazardous material released or potentially released.

44344.4.

(a) Except as provided in subdivision (d) and in Section 44344.7, a facility shall be exempt from further compliance with this part if the facility's prioritization scores for cancer and noncancer health effects are both equal to or less than one, based on the results of the most recent emissions inventory or emissions inventory update. An exempt facility shall no longer be required to pay any fee or submit any report to the district or the state board pursuant to this part.

(b) Except for facilities that are exempt from this part pursuant to subdivision (a), a facility for which the prioritization scores for cancer and noncancer health effects are both equal to or less than 10, based on the results of the most recent emissions inventory or emissions inventory update, shall not be required to pay any fee or submit any report to the district or the state board pursuant to this part,

except for the quadrennial emissions inventory update required pursuant to Section 44344. A district may, by regulation, establish a fee to be paid by a facility operator in connection with the operator's submission to the district of a quadrennial emissions inventory update pursuant to this subdivision. The fee shall not be greater than one hundred twenty-five dollars ($125). A district may increase the fee above that amount upon the adoption of written findings that the costs of processing the emission inventory update exceed one hundred twenty-five dollars ($125). However, the district shall not adopt a fee greater than that supported by the written findings.

(c) For the purposes of this part, "prioritization score" means a facility's numerical score for cancer health effects or noncancer health effects, as determined by the district pursuant to Section 44360 in a manner consistent with facility prioritization guidelines prepared by the California Air Pollution Control Officers Association and approved by the state board.

(d) Notwithstanding subdivision (a) and Section 44344.7, if a district has good cause to believe that a facility may pose a potential threat to public health and that the facility therefore does not qualify for an exemption claimed by the facility pursuant to subdivision (a), the district may require the facility to document the facility's emissions and health impacts, or the changes in emissions expected to occur as a result of a particular physical change, a change in activities or operations at the facility, or a change in other factors. The district may deny the exemption if the documentation does not support the claim for the exemption.

44344.5.

(a) The operator of any new facility that previously has not been subject to this part shall prepare and submit an emissions inventory plan and report.

(b) Notwithstanding subdivision (a), a new facility shall not be required to submit an emissions inventory plan and report if all of the following conditions are met:

(1) The facility is subject to a district permit program established pursuant to Section 42300.

(2) The district conducts an assessment of the potential emissions or their associated risks, whichever the district determines to be appropriate, attributable to the new facility and finds that the emissions will not result in a significant risk. A risk assessment conducted pursuant to this paragraph shall comply with paragraph (2) of subdivision (b) of Section 44360.

(3) The district issues a permit authorizing construction or operation of the new facility.

44344.6. A district shall redetermine a facility's prioritization score, or evaluate the prioritization score as calculated and submitted by the facility, within 90 days from the date of receipt of a quadrennial emissions inventory update pursuant to Section 44344 or subdivision (b) of Section 44344.4, within 90 days from the date of receipt of an emissions inventory update submitted pursuant to Section 44344.7, or within 90 days from the date of receiving notice that a facility has completed the implementation of a plan prepared pursuant to Section 44392.

44344.7.

(a) A facility exempted from this part pursuant to subdivision (a) of Section 44344.4 shall, upon receipt of a notice from the district, again be subject to this part and the operator shall submit an emissions inventory update for those sources and substances for which a physical change in the facility or a change in activities or operations has occurred, as follows:
  (1) The facility emits a substance newly listed pursuant to Section 44321.
  (2) A sensitive receptor has been established or constructed within 500 meters of the facility after the facility became exempt.
  (3) The facility emits a substance for which the potency factor has increased.

(b) The operator of a facility exempted from this part pursuant to subdivision (a) of Section 44344.4 shall submit an emissions inventory update for those sources and substances for which a particular physical change in the facility or a change in activities or operations occurs if, as a result of the particular change, either of the following has occurred:
  (1) The facility has begun emitting a listed substance not included in the previous emissions inventory.
  (2) The facility has increased its emissions of a listed substance to a level greater than the level previously reported for that substance, and the increase in emissions exceeds 100 percent of the previously reported level.

(c) Notwithstanding subdivision (b), a physical change or change in activities or operations at a facility shall not cause the facility to again be subject to this part if all of the following conditions are met:
  (1) The physical change or change in activities or operations is subject to a district permit program established pursuant to Section 42300.
  (2) The district conducts an assessment of the potential changes in emissions or their associated risks, whichever the district determines to be appropriate, attributable to the physical change or change in activities or operations and finds that the changes in emissions will not result in a significant risk. A risk assessment conducted pursuant to this paragraph shall comply with paragraph (2) of subdivision (b) of Section 44360.
  (3) The district issues a permit for the physical change or change in activities or operations.

44345.

(a) On or before July 1, 1989, the state board shall develop a program to compile and make available to other state and local public agencies and the public all data collected pursuant to this chapter.

(b) In addition, the state board, on or before March 1, 1990, shall compile, by district, emissions inventory data for mobile sources and area sources not subject to district permit requirements, and data on natural source emissions, and shall incorporate these data into data compiled and released pursuant to this chapter.

44346.

(a) If an operator believes that any information required in the facility diagram specified pursuant to subdivision (b) of Section 44342 involves the release of a trade secret, the operator shall nevertheless make the disclosure to the district, and shall notify the district in writing of that belief in the report.

(b) Subject to this section, the district shall protect from disclosure any trade secret designated as such by the operator, if that trade secret is not a public record.

(c) Upon receipt of a request for the release of information to the public which includes information which the operator has notified the district is a trade secret and which is not a public record, the following procedure applies:

(1) The district shall notify the operator of the request in writing by certified mail, return receipt requested.

(2) The district shall release the information to the public, but not earlier than 30 days after the date of mailing the notice of the request for information, unless, prior to the expiration of the 30-day period, the operator obtains an action in an appropriate court for a declaratory judgment that the information is subject to protection under this section or for a preliminary injunction prohibiting disclosure of the information to the public and promptly notifies the district of that action.

(d) This section does not permit an operator to refuse to disclose the information required pursuant to this part to the district.

(e) Any information determined by a court to be a trade secret, and not a public record pursuant to this section, shall not be disclosed to anyone except an officer or employee of the district, the state, or the United States, in connection with the official duties of that officer or employee under any law for the protection of health, or to contractors with the district or the state and its employees if, in the opinion of the district or the state, disclosure is necessary and required for the satisfactory performance of a contract, for performance of work, or to protect the health and safety of the employees of the contractor.

(f) Any officer or employee of the district or former officer or employee who, by virtue of that employment or official position, has possession of, or has access to, any trade secret subject to this section, and who, knowing that disclosure of the information to the general public is prohibited by this section, knowingly and willfully discloses the information in any manner to any person not entitled to receive it is guilty of a misdemeanor. Any contractor of the district and any employee of the contractor, who has been furnished information as authorized by this section, shall be considered an employee of the district for purposes of this section.

(g) Information certified by appropriate officials of the United States as necessary to be kept secret for national defense purposes shall be accorded the full protections against disclosure as specified by those officials or in accordance with the laws of the United States.

(h) As used in this section, "trade secret" and "public record" have the meanings and protections given to them by Section 6254.7 of the Government Code and Section 1060 of the Evidence Code. All information collected pursuant to this chapter, except for data used to calculate emissions data required in the facility

diagram, shall be considered "air pollution emission data," for the purposes of this section.

CHAPTER 4: RISK ASSESSMENT

44360.

(a) Within 90 days of completion of the review of all emissions inventory data for facilities specified in subdivision (a) of Section 44322, but not later than December 1, 1990, the district shall, based on examination of the emissions inventory data and in consultation with the state board and the State Department of Health Services, prioritize and then categorize those facilities for the purposes of health risk assessment. The district shall designate high, intermediate, and low priority categories and shall include each facility within the appropriate category based on its individual priority. In establishing priorities pursuant to this section, the district shall consider the potency, toxicity, quantity, and volume of hazardous materials released from the facility, the proximity of the facility to potential receptors, including, but not limited to, hospitals, schools, day care centers, worksites, and residences, and any other factors that the district finds and determines may indicate that the facility may pose a significant risk to receptors. The district shall hold a public hearing prior to the final establishment of priorities and categories pursuant to this section.

(b)

(1) Within 150 days of the designation of priorities and categories pursuant to subdivision (a), the operator of every facility that has been included within the highest priority category shall prepare and submit to the district a health risk assessment pursuant to Section 44361. The district may, at its discretion, grant a 30-day extension for submittal of the health risk assessment.

(2) Health risk assessments required by this chapter shall be prepared in accordance with guidelines established by the Office of Environmental Health Hazard Assessment. The office shall prepare draft guidelines which shall be circulated to the public and the regulated community and shall adopt risk assessment guidelines after consulting with the state board and the Risk Assessment Committee of the California Air Pollution Control Officers Association and after conducting at least two public workshops, one in the northern and one in the southern part of the state. The adoption of the guidelines is not subject to Chapter 3.5 (commencing with Section 11340) of Part 1 of Division 3 of Title 2 of the Government Code. The scientific review panel established pursuant to Section 39670 shall evaluate the guidelines adopted under this paragraph and shall recommend changes and additional criteria to reflect new scientific data or empirical studies.

(3) The guidelines established pursuant to paragraph (2) shall impose only those requirements on facilities subject to this subdivision that are necessary to ensure that a required risk assessment is accurate and complete and shall specify the type of site-specific factors that districts may take into account in determining when a single health risk assessment may be allowed under subdivision (d). The guidelines shall, in addition, allow the operator of a

facility, at the operator's option, and to the extent that valid and reliable data are available, to include for consideration by the district in the health risk assessment any or all of the following supplemental information:

(A) Information concerning the scientific basis for selecting risk parameter values that are different than those required by the guidelines and the likelihood distributions that result when alternative values are used.

(B) Data from dispersion models, microenvironment characteristics, and population distributions that may be used to estimate maximum actual exposure.

(C) Risk expressions that show the likelihood that any given risk estimate is the correct risk value.

(D) A description of the incremental reductions in risk that occur when exposure is reduced.

(4) To ensure consistency in the use of the supplemental information authorized by subparagraphs (A), (B), (C), and (D) of paragraph (3), the guidelines established pursuant to paragraph (2) shall include guidance for use by the districts in considering the supplemental information when it is included in the health risk assessment.

(c) Upon submission of emissions inventory data for facilities specified in subdivisions (b) and (c) of Section 44322, the district shall designate facilities for inclusion within the highest priority category, as appropriate, and any facility so designated shall be subject to subdivision (b). In addition, the district may require the operator of any facility to prepare and submit health risk assessments, in accordance with the priorities developed pursuant to subdivision (a).

(d) The district shall, except where site specific factors may affect the results, allow the use of a single health risk assessment for two or more substantially identical facilities operated by the same person.

(e) Nothing contained in this section, Section 44380.5, or Chapter 6 (commencing with Section 44390) shall be interpreted as requiring a facility operator to prepare a new or revised health risk assessment using the guidelines established pursuant to paragraph (2) of subdivision (a) of this section if the facility operator is required by the district to begin the preparation of a health risk assessment before those guidelines are established.

44361.

(a) Each health risk assessment shall be submitted to the district. The district shall make the health risk assessment available for public review, upon request. After preliminary review of the emissions impact and modeling data, the district shall submit the health risk assessment to the Office of Environmental Health Hazard Assessment for review and, within 180 days of receiving the health risk assessment, the State office shall submit to the district its comments on the data and findings relating to health effects. The district shall consult with the state board as necessary to adequately evaluate the emissions impact and modeling data contained within the risk assessment.

(b) For the purposes of complying with this section, the Office of Environmental Health Hazard Assessment may select a qualified independent contractor to review the data and findings relating to health effects. The office shall not select an independent contractor to review a specific health risk assessment who may have a conflict of interest with regard to the review of that health risk assessment. Any review by an independent contractor shall comply with the following requirements:
(1) Be performed in a manner consistent with guidelines provided by the office.
(2) Be reviewed by the office for accuracy and completeness.
(3) Be submitted by the office to the district in accordance with this section.

(c) The district shall reimburse the Office of Environmental Health Hazard Assessment or the qualified independent contractor designated by the office pursuant to subdivision (b), within 45 days of its request, for its actual costs incurred in reviewing a health risk assessment pursuant to this section.

(d) If a district requests the Office of Environmental Health Hazard Assessment to consult with the district concerning any requirement of this part, the district shall reimburse the office, within 45 days of its request, for the costs incurred in the consultation.

(e) Upon designation of the high priority facilities, as specified in subdivision (a) of Section 44360, the Office of Environmental Health Hazard Assessment shall evaluate the staffing requirements of this section and may submit recommendations to the Legislature, as appropriate, concerning the maximum number of health risk assessments to be reviewed each year pursuant to this section.

44362.

(a) Taking the comments of the Office of Environmental Health Hazard Assessment into account, the district shall approve or return for revision and resubmission and then approve, the health risk assessment within one year of receipt. If the health risk assessment has not been revised and resubmitted within 60 days of the district's request of the operator to do so, the district may modify the health risk assessment and approve it as modified.

(b) Upon approval of the health risk assessment, the operator of the facility shall provide notice to all exposed persons regarding the results of the health risk assessment prepared pursuant to Section 44361 if, in the judgment of the district, the health risk assessment indicates there is a significant health risk associated with emissions from the facility. If notice is required under this subdivision, the notice shall include only information concerning significant health risks attributable to the specific facility for which the notice is required. Any notice shall be made in accordance with procedures specified by the district.

44363.

(a) Commencing July 1, 1991, each district shall prepare and publish an annual report which does all of the following:
   (1) Describes the priorities and categories designated pursuant to Section 44360 and summarizes the results and progress of the health risk assessment program undertaken pursuant to this part.
   (2) Ranks and identifies facilities according to the degree of cancer risk posed both to individuals and to the exposed population.
   (3) Identifies facilities which expose individuals or populations to any noncancer health risks.
   (4) Describes the status of the development of control measures to reduce emissions of toxic air contaminants, if any.

(b) The district shall disseminate the annual report to county boards of supervisors, city councils, and local health officers and the district board shall hold one or more public hearings to present the report and discuss its content and significance.

44364. The state board shall utilize the reports and assessments developed pursuant to this part for the purposes of identifying, establishing priorities for, and controlling toxic air contaminants pursuant to Chapter 3.5 (commencing with Section 39650) of Part 2.

44365.

(a) If the state board finds and determines that a district's actions pursuant to this part do not meet the requirements of this part, the state board may exercise the authority of the district pursuant to this part to approve emissions inventory plans and require the preparation of health risk assessments.

(b) This part does not prevent any district from establishing more stringent criteria and requirements than are specified in this part for approval of emissions inventories and requiring the preparation and submission of health risk assessments. Nothing in this part limits the authority of a district under any other provision of law to assess and regulate releases of hazardous substances.

44366.

(a) In order to verify the accuracy of any information submitted by facilities pursuant to this part, a district or the state board may proceed in accordance with Section 41510.

CHAPTER 5: FEES AND REGULATIONS

44380.

(a) The state board shall adopt a regulation which does all of the following:
   (1) Sets forth the amount of revenue which the district must collect to recover the reasonable anticipated cost which will be incurred by the state board and the Office of Environmental Health Hazard Assessment to implement and administer this part.

(2) Requires each district to adopt a fee schedule which recovers the costs of the district and which assesses a fee upon the operator of every facility subject to this part, except as specified in subdivision (b) of Section 44344.4. A district may request the state board to adopt a fee schedule for the district if the district's program costs are approved by the district board and transmitted to the state board by April 1 of the year in which the request is made.

(3) Requires any district that has an approved toxics emissions inventory compiled pursuant to this part by August 1 of the preceding year to adopt a fee schedule, as described in paragraph (2), which imposes on facility operators fees which are, to the maximum extent practicable, proportionate to the extent of the releases identified in the toxics emissions inventory and the level of priority assigned to that source by the district pursuant to Section 44360.

(b) Commencing August 1, 1992, and annually thereafter, the state board shall review and may amend the fee regulation.

(c) The district shall notify each person who is subject to the fee of the obligation to pay the fee. If a person fails to pay the fee within 60 days after receipt of this notice, the district, unless otherwise provided by district rules, shall require the person to pay an additional administrative civil penalty. The district shall fix the penalty at not more than 100 percent of the assessed fee, but in an amount sufficient in its determination, to pay the district's additional expenses incurred by the person's noncompliance. If a person fails to pay the fee within 120 days after receipt of this notice, the district may initiate permit revocation proceedings. If any permit is revoked, it shall be reinstated only upon full payment of the overdue fee plus any late penalty, and a reinstatement fee to cover administrative costs of reinstating the permit.

(d) Each district shall collect the fees assessed pursuant to subdivision (a). After deducting the costs to the district to implement and administer this part, the district shall transmit the remainder to the Controller for deposit in the Air Toxics Inventory and Assessment Account, which is hereby created in the General Fund. The money in the account is available, upon appropriation by the Legislature, to the state board and the Office of Environmental Health Hazard Assessment for the purposes of administering this part.

(e) For the 1997-98 fiscal year, air toxics program revenues for the state board and the Office of Environmental Health Hazard Assessment shall not exceed two million dollars ($2,000,000), and for each fiscal year thereafter, shall not exceed one million three hundred fifty thousand dollars ($1,350,000). Funding for the Office of Environmental Health Hazard Assessment for conducting risk assessment reviews shall be on a fee-for-service basis.

44380.1. A facility shall be granted an exemption by a district from paying a fee in accordance with Section 44380 if all of the following criteria are met:

(a) The facility primarily handles, processes, stores, or distributes bulk agricultural commodities or handles, feeds, or rears livestock.

(b) The facility was required to comply with this part only as a result of its particulate matter emissions.

(c) The fee schedule adopted by the district or the state board for these types of facilities is not solely based on toxic emissions weighted for potency or toxicity.

44380.5. In addition to the fee assessed pursuant to Section 44380, a supplemental fee may be assessed by the district, the state board, or the Office of Environmental Health Hazard Assessment upon the operator of a facility that, at the operator's option, includes supplemental information authorized by paragraph (3) of subdivision (b) of Section 44360 in a health risk assessment, if the review of that supplemental information substantially increases the costs of reviewing the health risk assessment by the district, the state board, or the office. The supplemental fee shall be set by the state board in the regulation required by subdivision (a) of Section 44380 and shall be set in an amount sufficient to cover the direct costs to review the information supplied by an operator pursuant to paragraph (3) of subdivision (b) of Section 44360.

44381.

(a) Any person who fails to submit any information, reports, or statements required by this part, or who fails to comply with this part or with any permit, rule, regulation, or requirement issued or adopted pursuant to this part, is subject to a civil penalty of not less than five hundred dollars ($500) or more than ten thousand dollars ($10,000) for each day that the information, report, or statement is not submitted, or that the violation continues.
(b) Any person who knowingly submits any false statement or representation in any application, report, statement, or other document filed, maintained, or used for the purposes of compliance with this part is subject to a civil penalty of not less than one thousand dollars ($1,000) or more than twenty-five thousand dollars ($25,000) per day for each day that the information remains uncorrected.

44382. Every district shall, by regulation, adopt the requirements of this part as a condition of every permit issued pursuant to Chapter 4 (commencing with Section 42300) of Part 4 for all new and modified facilities.

44384. Except for Section 44380 and this section, all provisions of this part shall become operative on July 1, 1988.

CHAPTER 6: FACILITY RISK REDUCTION AUDIT AND PLAN

44390. For purposes of this chapter, the following definitions apply:

(a) "Airborne toxic risk reduction measure" or "ATRRM" means those in-plant changes in production processes or feedstocks that reduce or eliminate toxic air emissions subject to this part. ATRRM's may include:
    (1) Feedstock modification.
    (2) Product reformulations.
    (3) Production system modifications.
    (4) System enclosure, emissions control, capture, or conversion.
    (5) Operational standards and practices modification.

(b) Airborne toxic risk reduction measures do not include measures that will increase risk from exposure to the chemical in another media or that increase the risk to workers or consumers.
(c) "Airborne toxic risk reduction audit and plan" or "audit and plan" means the audit and plan specified in Section 44392.

44391.
(a) Whenever a health risk assessment approved pursuant to Chapter 4 (commencing with Section 44360) indicates, in the judgment of the district, that there is a significant risk associated with the emissions from a facility, the facility operator shall conduct an airborne toxic risk reduction audit and develop a plan to implement airborne toxic risk reduction measures that will result in the reduction of emissions from the facility to a level below the significant risk level within five years of the date the plan is submitted to the district. The facility operator shall implement measures set forth in the plan in accordance with this chapter.
(b) The period to implement the plan required by subdivision (a) may be shortened by the district if it finds that it is technically feasible and economically practicable to implement the plan to reduce emissions below the significant risk level more quickly or if it finds that the emissions from the facility pose an unreasonable health risk.
(c) A district may lengthen the period to implement the plan required by subdivision (a) by up to an additional five years if it finds that a period longer than five years will not result in an unreasonable risk to public health and that requiring implementation of the plan within five years places an unreasonable economic burden on the facility operator or is not technically feasible.
(d)
  (1) The state board and districts shall provide assistance to smaller businesses that have inadequate technical and financial resources for obtaining information, assessing risk reduction methods, and developing and applying risk reduction techniques.
  (2) Risk reduction audits and plans for any industry subject to this chapter which is comprised mainly of small businesses using substantially similar technology may be completed by a self-conducted audit and checklist developed by the state board. The state board, in coordination with the districts, shall provide a copy of the audit and checklist to small businesses within those industries to assist them to meet the requirements of this chapter.
(e) The audit and plan shall contain all the information required by Section 44392.
(f) The plan shall be submitted to the district, within six months of a district's determination of significant risk, for review of completeness. Operators of facilities that have been notified prior to January 1, 1993, that there is a significant risk associated with emissions from the facility shall submit the plan by July 1, 1993. The district's review of completeness shall include a substantive analysis of the emission reduction measures included in the plan, and the ability

of those measures to achieve emission reduction goals as quickly as feasible as provided in subdivisions (a) and (b).

(g) The district shall find the audit and plan to be satisfactory within three months if it meets the requirements of this chapter, including, but not limited to, subdivision (f). If the district determines that the audit and plan does not meet those requirements, the district shall remand the audit and plan to the facility specifying the deficiencies identified by the district. A facility operator shall submit a revised audit and plan addressing the deficiencies identified by the district within 90 days of receipt of a deficiency notice.

(h) Progress on the emission reductions achieved by the plan shall be reported to the district in emissions inventory updates. Emissions inventory updates shall be prepared as required by the audit and plan found to be satisfactory by the district pursuant to subdivision (g).

(i) If new information becomes available after the initial risk reduction audit and plan, on air toxics risks posed by a facility, or emission reduction technologies that may be used by a facility that would significantly impact risks to exposed persons, the district may require the plan to be updated and resubmitted to the district.

(j) This section does not authorize the emission of a toxic air contaminant in violation of an airborne toxic control measure adopted pursuant to Chapter 3.5 (commencing with Section 39650) or in violation of Section 41700.

44392. A facility operator subject to this chapter shall conduct an airborne toxic risk reduction audit and develop a plan which shall include at a minimum all of the following:

(a) The name and location of the facility.

(b) The SIC code for the facility.

(c) The chemical name and the generic classification of the chemical.

(d) An evaluation of the ATRRM's available to the operator.

(e) The specification of, and rationale for, the ATRRMs that will be implemented by the operator. The audit and plan shall document the rationale for rejecting ATRRMs that are identified as infeasible or too costly.

(f) A schedule for implementing the ATRRMs. The schedule shall meet the time requirements of subdivision (a) of Section 44391 or the time period for implementing the plan set by the district pursuant to subdivision (b) or (c) of Section 44391, whichever is applicable.

(g) The audit and plan shall be reviewed and certified as meeting this chapter by an engineer who is registered as a professional engineer pursuant to Section 6762 of the Business and Professions Code, by an individual who is responsible for the processes and operations of the site, or by an environmental assessor registered pursuant to Section 25570.3.

44393. The plan prepared pursuant to Section 44391 shall not be considered to be the equivalent of a pollution prevention program or a source reduction program, except insofar as the audit and plan elements are consistent with source reduction, as defined in Section 25244.14, or subsequent statutory definitions of pollution prevention.

44394. Any facility operator who does not submit a complete airborne toxic risk reduction audit and plan or fails to implement the measures set forth in the plan as set forth in this chapter is subject to the civil penalty specified in subdivision (a) of Section 44381, and any facility operator who, in connection with the audit or plan, knowingly submits any false statement or representation is subject to the civil penalty specified in subdivision (b) of Section 44381.

B.3. Toxic Air Contaminants Program Overview

(Air Resources Board, 2011: see http://www.arb.ca.gov/toxics/background.htm)

AB 1807 Program

In 1983, the California Legislature established a two-step process of risk identification and risk management to address the potential health effects from air toxic substances and protect the public health of Californians. During the first step (identification), the ARB and the Office of Environmental Health Hazard Assessment (OEHHA) determine if a substance should be formally identified as a toxic air contaminant (TAC) in California. During this process, the ARB and the OEHHA staff draft a report that serves as the basis for this determination. The ARB staff assesses the potential for human exposure to a substance and the OEHHA staff evaluates the health effects. A thorough public process assures accountability and public input. Public workshops are conducted to allow for direct exchanges of information with interested constituencies. The draft risk assessments themselves are published and widely distributed with a public notice requesting comment to further assure involvement. The final risk assessment (identification) report includes a record of the public comments and how they were addressed. After the ARB and the OEHHA staff hold several comment periods and workshops, the report is then submitted to an independent, nine member, Scientific Review Panel (SRP), who review the report for its scientific accuracy. If the SRP approves the report, they develop specific scientific findings which are officially submitted to the ARB. The ARB staff then prepares a hearing notice and draft regulation to formally identify the substance as a TAC. Based on the input from the public and the information gathered from the report, the Board will decide whether to identify a substance as a TAC. Any person may petition the Board to review a previous determination by providing new evidence.

In the second step (risk management), the ARB reviews the emission sources of an identified TAC to determine if any regulatory action is necessary to reduce the risk. The analysis includes a review of controls already in place, the available technologies and associated costs for reducing emissions, and the associated risk. Public outreach is an essential element in the development of a control plan and any control measure to ensure that the ARB efforts are cost-effective and appropriately balance public health protection and economic growth.

In 1993, the California Legislature amended the AB 1807 program for the identification and control of TACs (AB 2728). Specifically, AB 2728 required the ARB to identify the 189 federal hazardous air pollutants as TACs. For those substances that have not previously been identified under AB 1807 and identified under AB 2728, health effects values will need to be developed. This report will serve as a basis for that evaluation. For substances that were not identified as TACs and are on the TAC Identification List, this report will provide information to evaluate which substances may be entered into the air toxics identification process.

B.4. Senate Bill 352. Schoolsites: sources of pollution

CHAPTER 668
FILED WITH SECRETARY OF STATE OCTOBER 3, 2003
APPROVED BY GOVERNOR OCTOBER 2, 2003

THE PEOPLE OF THE STATE OF CALIFORNIA DO ENACT AS FOLLOWS:

SECTION 1.
The Legislature finds and declares all of the following:

(a) Many studies have shown significantly increased levels of pollutants, particularly diesel particulates, in close proximity to freeways and other major diesel sources. A recent study of Los Angeles area freeways measured diesel particulate levels up to 25 times higher near freeways than those levels elsewhere. Much of the pollution from freeways is associated with acute health effects, exacerbating asthma and negatively impacting the ability of children to learn.
(b) Cars and trucks release at least forty different toxic air contaminants, including, but not limited to, diesel particulate, benzene, formaldehyde, 1,3-butadiene and acetaldehyde. Levels of these pollutants are generally concentrated within 500 feet of freeways and very busy roadways.
(c) Current state law governing the siting of schools does not specify whether busy freeways should be included in environmental impact reports of nearby "facilities." Over 150 schools are already estimated to be within 500 feet of extremely high traffic roadways.
(d) A disproportionate number of economically disadvantaged pupils may be attending schools that are close to busy roads, putting them at an increased risk of developing bronchitis from elevated levels of several pollutants associated with traffic. Many studies have confirmed that increased wheezing and bronchitis occurs among children living in high traffic areas.
(e) It is therefore the intent of the Legislature to protect school children from the health risks posed by pollution from heavy freeway traffic and other nonstationary sources in the same way that they are protected from industrial pollution.

SECTION 2.

Section 17213 of the Education Code is amended to read:
17213. The governing board of a school district may not approve a project involving the acquisition of a schoolsite by a school district, unless all of the following occur:
(a) The school district, as the lead agency, as defined in Section 21067 of the Public Resources Code, determines that the property purchased or to be built upon is not any of the following:
(1) The site of a current or former hazardous waste disposal site or solid waste disposal site, unless if the site was a former solid waste disposal site, the governing board of the school district concludes that the wastes have been removed.

(2) A hazardous substance release site identified by the Department of Toxic Substances Control in a current list adopted pursuant to Section 25356 of the Health and Safety Code for removal or remedial action pursuant to Chapter 6.8 (commencing with Section 25300) of Division 20 of the Health and Safety Code.

(3) A site that contains one or more pipelines, situated underground or aboveground, that carries hazardous substances, acutely hazardous materials, or hazardous wastes, unless the pipeline is a natural gas line that is used only to supply natural gas to that school or neighborhood.

(b) The school district, as the lead agency, as defined in Section 21067 of the Public Resources Code, in preparing the environmental impact report or negative declaration has consulted with the administering agency in which the proposed schoolsite is located, pursuant to Section 2735.3 of Title 19 of the California Code of Regulations, and with any air pollution control district or air quality management district having jurisdiction in the area, to identify both permitted and nonpermitted facilities within that district's authority, including, but not limited to, freeways and other busy traffic corridors, large agricultural operations, and railyards, within one-fourth of a mile of the proposed schoolsite, that might reasonably be anticipated to emit hazardous air emissions, or to handle hazardous or acutely hazardous materials, substances, or waste. The school district, as the lead agency, shall include a list of the locations for which information is sought.

(c) The governing board of the school district makes one of the following written findings:

(1) Consultation identified none of the facilities or significant pollution sources specified in subdivision (b).

(2) The facilities or other pollution sources specified in subdivision (b) exist, but one of the following conditions applies:

(A) The health risks from the facilities or other pollution sources do not and will not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the school.

(B) The governing board finds that corrective measures required under an existing order by another governmental entity that has jurisdiction over the facilities or other pollution sources will, before the school is occupied, result in the mitigation of all chronic or accidental hazardous air emissions to levels that do not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school. If the governing board makes this finding, the governing board shall also make a subsequent finding, prior to the occupancy of the school, that the emissions have been mitigated to these levels.

(C) For a schoolsite with a boundary that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor, the governing board of the school district determines, through analysis pursuant to paragraph (2) of subdivision (b) of Section 44360 of the Health and Safety Code, based on appropriate air dispersion modeling, and after considering any potential mitigation measures, that the air quality at the proposed site

is such that neither short-term nor long-term exposure poses significant health risks to pupils.

(D) The governing board finds that neither of the conditions set forth in subparagraph (B) or (C) can be met, and the school district is unable to locate an alternative site that is suitable due to a severe shortage of sites that meet the requirements in subdivision (a) of Section 17213. If the governing board makes this finding, the governing board shall adopt a statement of Overriding Considerations pursuant to Section 15093 of Title 14 of the California Code of Regulations.

(d) As used in this section:

(1) "Hazardous air emissions" means emissions into the ambient air of air contaminants that have been identified as a toxic air contaminant by the State Air Resources Board or by the air pollution control officer for the jurisdiction in which the project is located. As determined by the air pollution control officer, hazardous air emissions also means emissions into the ambient air from any substance identified in subdivisions (a) to (f), inclusive, of Section 44321 of the Health and Safety Code.

(2) "Hazardous substance" means any substance defined in Section 25316 of the Health and Safety Code.

(3) "Acutely hazardous material" means any material defined pursuant to subdivision (a) of Section 25532 of the Health and Safety Code.

(4) "Hazardous waste" means any waste defined in Section 25117 of the Health and Safety Code.

(5) "Hazardous waste disposal site" means any site defined in Section 25114 of the Health and Safety Code.

(6) "Administering agency" means any agency designated pursuant to Section 25502 of the Health and Safety Code.

(7) "Handle" means handle as defined in Article 1 (commencing with Section 25500) of Chapter 6.95 of Division 20 of the Health and Safety Code.

(8) "Facilities" means any source with a potential to use, generate, emit or discharge hazardous air pollutants, including, but not limited to, pollutants that meet the definition of a hazardous substance, and whose process or operation is identified as an emission source pursuant to the most recent list of source categories published by the California Air Resources Board.

(9) "Freeway or other busy traffic corridors" means those roadways that, on an average day, have traffic in excess of 50,000 vehicles in a rural area as defined in Section 50101 of the Health and Safety Code, and 100,000 vehicles in an urban area, as defined in Section 50104.7 of the Health and Safety Code.

SECTION 3.

Section 21151.8 of the Public Resources Code is amended to read:

21151.8.

(a) An environmental impact report or negative declaration may not be approved for any project involving the purchase of a schoolsite or the construction of a new

elementary or secondary school by a school district unless all of the following occur:

(1) The environmental impact report or negative declaration includes information that is needed to determine if the property proposed to be purchased, or to be constructed upon, is any of the following:
   (A) The site of a current or former hazardous waste disposal site or solid waste disposal site and, if so, whether the wastes have been removed.
   (B) A hazardous substance release site identified by the Department of Toxic Substances Control in a current list adopted pursuant to Section 25356 of the Health and Safety Code for removal or remedial action pursuant to Chapter 6.8 (commencing with Section 25300) of Division 20 of the Health and Safety Code.
   (C) A site that contains one or more pipelines, situated underground or aboveground, that carries hazardous substances, acutely hazardous materials, or hazardous wastes, unless the pipeline is a natural gas line that is used only to supply natural gas to that school or neighborhood, or other nearby schools.
   (D) A site that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor.

(2) The school district, as the lead agency, in preparing the environmental impact report or negative declaration has notified in writing and consulted with the administering agency in which the proposed schoolsite is located, pursuant to Section 2735.3 of Title 19 of the California Code of Regulations, and with any air pollution control district or air quality management district having jurisdiction in the area, to identify both permitted and nonpermitted facilities within that district's authority, including, but not limited to, freeways and busy traffic corridors, large agricultural operations, and railyards, within one-fourth of a mile of the proposed schoolsite, that might reasonably be anticipated to emit hazardous emissions or handle hazardous or acutely hazardous materials, substances, or waste. The notification by the school district, as the lead agency, shall include a list of the locations for which information is sought.

(3) The governing board of the school district makes one of the following written findings:
   (A) Consultation identified no facilities of this type or other significant pollution sources specified in paragraph (2).
   (B) The facilities or other pollution sources specified in paragraph (2) exist, but one of the following conditions applies:
      (i) The health risks from the facilities or other pollution sources do not and will not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school.
      (ii) Corrective measures required under an existing order by another agency having jurisdiction over the facilities or other pollution sources will, before the school is occupied, result in the mitigation of all chronic or accidental hazardous air emissions to levels that do not constitute an actual or potential endangerment of public health to persons who

would attend or be employed at the proposed school. If the governing board makes a finding pursuant to this clause, it shall also make a subsequent finding, prior to occupancy of the school, that the emissions have been so mitigated.

(iii) For a schoolsite with a boundary that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor, the governing board of the school district determines, through analysis pursuant to paragraph (2) of subdivision (b) of Section 44360 of the Health and Safety Code, based on appropriate air dispersion modeling, and after considering any potential mitigation measures, that the air quality at the proposed site is such that neither short-term nor long-term exposure poses significant health risks to pupils.

(C) The facilities or other pollution sources specified in paragraph (2) exist, but conditions in clause (i), (ii) or (iii) of subparagraph (B) cannot be met, and the school district is unable to locate an alternative site that is suitable due to a severe shortage of sites that meet the requirements in subdivision (a) of Section 17213 of the Education Code. If the governing board makes this finding, the governing board shall adopt a statement of Overriding Considerations pursuant to Section 15093 of Title 14 of the California Code of Regulations.

(4) Each administering agency, air pollution control district, or air quality management district receiving written notification from a lead agency to identify facilities pursuant to paragraph (2) shall provide the requested information and provide a written response to the lead agency within 30 days of receiving the notification. The environmental impact report or negative declaration shall be conclusively presumed to comply with this section as to the area of responsibility of any agency that does not respond within 30 days.

(b) If a school district, as a lead agency, has carried out the consultation required by paragraph (2) of subdivision (a), the environmental impact report or the negative declaration shall be conclusively presumed to comply with this section, notwithstanding any failure of the consultation to identify an existing facility or other pollution source specified in paragraph (2) of subdivision (a).

(c) As used in this section and Section 21151.4, the following definitions shall apply:

(1) "Hazardous substance" means any substance defined in Section 25316 of the Health and Safety Code.

(2) "Acutely hazardous material" means any material defined pursuant to subdivision (a) of Section 25532 of the Health and Safety Code.

(3) "Hazardous waste" means any waste defined in Section 25117 of the Health and Safety Code.

(4) "Hazardous waste disposal site" means any site defined in Section 25114 of the Health and Safety Code.

(5) "Hazardous air emissions" means emissions into the ambient air of air contaminants that have been identified as a toxic air contaminant by the State Air Resources Board or by the air pollution control officer for the jurisdiction in which the project is located. As determined by the air pollution control officer, hazardous air emissions also means emissions into the ambient air from any

substances identified in subdivisions (a) to (f), inclusive, of Section 44321 of the Health and Safety Code.

(6) "Administering agency" means an agency designated pursuant to Section 25502 of the Health and Safety Code.

(7) "Handle" means handle as defined in Article 1 (commencing with Section 25500) of Chapter 6.95 of Division 20 of the Health and Safety Code.

(8) "Facilities" means any source with a potential to use, generate, emit or discharge hazardous air pollutants, including, but not limited to, pollutants that meet the definition of a hazardous substance, and whose process or operation is identified as an emission source pursuant to the most recent list of source categories published by the California Air Resources Board.

(9) "Freeway or other busy traffic corridors" means those roadways that, on an average day, have traffic in excess of 50,000 vehicles in a rural area, as defined in Section 50101 of the Health and Safety Code, and 100,000 vehicles in an urban area, as defined in Section 50104.7 of the Health and Safety Code.

B.5. Senate Bill 25, Children's Environmental Health Protection.

CHAPTER 731
FILED WITH SECRETARY OF STATE OCTOBER 10, 1999
APPROVED BY GOVERNOR OCTOBER 7, 1999

THE PEOPLE OF THE STATE OF CALIFORNIA DO ENACT AS FOLLOWS:

SECTION 1.
The Legislature finds and declares all of the following:

(a) Infants and children have a higher ventilation rate than adults relative to their body weight and lung surface area, resulting in a greater dose of pollution delivered to their lungs.
(b) Children have narrower airways than adults. Thus, irritation or inflammation caused by air pollution that would produce only a slight response in an adult can result in a potentially significant obstruction of the airway in a young child.
(c) Children spend significantly more time outdoors, especially in the summer, when ozone air pollution levels are typically highest. National statistics show that children spend an average of 50 percent more time outdoors than adults.
(d) Air pollution is known to exacerbate asthma and be a trigger for asthma attacks in infants and children, 500,000 of whom are afflicted with this chronic lung disease in California.
(e) Infant's and children's developing organs and tissues are more susceptible to damage from some environmental contaminants than are adult organs and tissues.
(f) It is the intent of the Legislature in enacting this act, to require that the state's air quality standards and airborne toxic control measures be reviewed to determine if they adequately protect the health of infants and children, and that these standards and measures be revised if they are determined to be inadequate.
(g) It is also the intent of the Legislature in enacting this act to require the State Air Resources Board and the Office of Environmental Health Hazard Assessment to consider the health impacts to all populations of children, including special subpopulations of infants and children that comprise a meaningful portion of the general population, such as children with asthma, cystic fibrosis, or other respiratory conditions or diseases, in setting or revising standards pursuant to this act.

SECTION 2.
Part 3 (commencing with Section 900) is added to Division 1 of the Health and Safety Code, to read:
PART 3. CHILDREN'S ENVIRONMENTAL HEALTH CENTER 900. There is hereby created the Children's Environmental Health Center within the Environmental Protection Agency. The primary purposes of the center shall include all of the following:

(a) To serve as the chief advisor to the Secretary for Environmental Protection and to the Governor on matters within the jurisdiction of the Environmental Protection

Agency relating to environmental health and environmental protection as each of those matters relates to children.

(b) To assist the boards, departments, and offices within the Environmental Protection Agency to assess the effectiveness of statutes, regulations, and programs designed to protect children from environmental hazards.

(c) To coordinate within the Environmental Protection Agency and with other state agencies, regulatory efforts, research and data collection, and other programs and services that impact the environmental health of children, and coordinate with appropriate federal agencies conducting related regulatory efforts and research and data collection.

(d) In consultation with the State Air Resources Board and the Office of Environmental Health Hazard Assessment, and notwithstanding Section 7550.5 of the Government Code, to report to the Legislature and the Governor no later than December 31, 2001, on the progress of the state board and the office toward implementing the act that added this part during the 1999-2000 Regular Session and to make recommendations for any statutory or regulatory changes that may be necessary to carry out the intent of that act to protect the public health, including infants and children, from air pollutants and toxic air contaminants.

SECTION 3.
Section 39606 of the Health and Safety Code is amended to read:
39606.

(a) The state board shall do both of the following:
  (1) Based upon similar meteorological and geographic conditions and consideration for political boundary lines whenever practicable,divide the state into air basins to fulfill the purposes of this division.
  (2) Adopt standards of ambient air quality for each air basin in consideration of the public health, safety, and welfare, including,but not limited to, health, illness, irritation to the senses, aesthetic value, interference with visibility, and effects on the economy. These standards may vary from one air basin to another. Standards relating to health effects shall be based upon the recommendations of the Office of Environmental Health Hazard Assessment.

(b) In its recommendations for submission to the state board pursuant to paragraph (2) of subdivision (a), the Office of Environmental Health Hazard Assessment, to the extent that information is available, shall assess the following:
  (1) Exposure patterns, including, but not limited to, patterns determined by relevant data supplied by the state board, among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.
  (2) Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.
  (3) The effects on infants and children of exposure to ambient air pollutants and other substances that have a common mechanism of toxicity.
  (4) The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

(c) In assessing the factors specified in subdivision (b), the office shall use current principles, practices, and methods used by public health professionals who are experienced practitioners in the field of human health effects assessment. The scientific basis or scientific portion of the method used by the office to assess the factors set forth in subdivision (b) shall be subject to peer review as described in Section 57004 or in a manner consistent with the peer review requirements of Section 57004. Any person may submit any information for consideration by the entity conducting the peer review, which may receive oral testimony.

(d)

(1) No later than December 31, 2000, the state board in consultation with the office, shall review all existing health-based ambient air quality standards to determine whether, based on public health, scientific literature, and exposure pattern data, the standards adequately protect the health of the public, including infants and children, with an adequate margin of safety. The state board shall publish a report summarizing these findings.

(2) The state board shall revise the highest priority ambient air quality standard determined to be inadequate to protect infants and children with an adequate margin of safety, based on its report, no later than December 31, 2002. Following the revision of the highest priority standard, the state board shall revise any additional standards determined to be inadequate to protect infants and children with an adequate margin of safety, at the rate of at least one per year. The standards shall be established at levels that adequately protect the health of the public, including infants and children, with an adequate margin of safety (e) Nothing in this section shall restrict the authority of the state board to consider additional information in establishing ambient air quality standards or to adopt an ambient air quality standard designed to protect vulnerable populations other than infants and children.

SECTION 4.

Section 39617.5 is added to the Health and Safety Code, to read:

39617.5.

(a) Not later than January 1, 2003, the state board shall do all of the following:

(1) Evaluate the adequacy of the current monitoring network for its ability to gather the data necessary to determine the exposure of infants and children to air pollutants including criteria air pollutants and toxic air contaminants.

(2) Identify areas where the exposure of infants and children to air pollutants is not adequately measured by the current monitoring network.

(3) Recommend changes to improve air pollution monitoring networks and data collection to more accurately reflect the exposure of infants and children to air pollutants.

(b) In carrying out this section, the state board, in cooperation with the districts, shall expand its existing monitoring program in six communities around the state in nonattainment areas, as selected by the state board, to include special monitoring of children's exposure to air pollutants and toxic contaminants. The expanded program shall include placing air pollution monitors near schools, day care centers, and outdoor recreational facilities that are in close proximity to, or

downwind from, major industrial sources of air pollutants and toxic air contaminants, including, freeways and major traffic areas. The purpose of the air pollution monitors shall be to conduct sampling of air pollution levels affecting children. Monitoring may include the use of fixed, mobile, and other monitoring devices, as appropriate.

(c) The expanded monitoring program shall include the following:
  (1) Monitoring during multiple seasons and at multiple locations within each community at schools, day care centers, recreational facilities, and other locations where children spend most of their time.
  (2) A combination of upgrading existing fixed monitoring sites, establishing new fixed monitoring sites, and conducting indoor and outdoor sampling and personal exposure measurements in each community to provide the most comprehensive data possible on the levels of children's exposure to air pollutants and toxic air contaminants.

(d) Data collected from expanded air quality monitoring activities conducted pursuant to this section may be used for any purpose authorized by law, including, but not limited to, determinations as to whether an area has attained or has not attained the state and national ambient air quality standards, if the monitoring devices from which the data was collected meet the monitoring requirements specified in Section 58.14 of Title 40 of the Code of Federal Regulations for special purpose monitors, all other monitoring requirements of Part 58 of Title 40 of the Code of Federal Regulations, and all applicable requirements specified in regulations adopted by the state board.

SECTION 5.
Section 39660 of the Health and Safety Code is amended to read:
39660.

(a) Upon the request of the state board, the office, in consultation with and with the participation of the state board, shall evaluate the health effects of and prepare recommendations regarding substances, other than pesticides in their pesticidal use, which may be or are emitted into the ambient air of California and that may be determined to be toxic air contaminants.

(b) In conducting this evaluation, the office shall consider all available scientific data, including, but not limited to, relevant data provided by the state board, the State Department of Health Services, the Occupational Safety and Health Division of the Department of Industrial Relations, the Department of Pesticide Regulation, international and federal health agencies, private industry, academic researchers, and public health and environmental organizations. The evaluation shall be performed using current principles, practices, and methods used by public health professionals who are experienced practitioners in the fields of epidemiology, human health effects assessment, risk assessment, and toxicity.

(c)
  (1) The evaluation shall assess the availability and quality of data on health effects, including potency, mode of action, and other relevant biological factors, of the substance, and shall, to the extent that information is available, assess all of the following:

(A) Exposure patterns among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.
(B) Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.
(C) The effects on infants and children of exposure to toxic air contaminants and other substances that have a common mechanism of toxicity.
(D) The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

(2) The evaluation shall also contain an estimate of the levels of exposure that may cause or contribute to adverse health effects. If it can be established that a threshold of adverse health effects exists, the estimate shall include both of the following factors:
(A) The exposure level below which no adverse health effects are anticipated.
(B) An ample margin of safety that accounts for the variable effects that heterogeneous human populations exposed to the substance under evaluation may experience, the uncertainties associated with the applicability of the data to human beings, and the completeness and quality of the information available on potential human exposure to the substance. In cases in which there is no threshold of significant adverse health effects, the office shall determine the range of risk to humans resulting from current or anticipated exposure to the substance.

(3) The scientific basis or scientific portion of the method used by the office to assess the factors set forth in this subdivision shall be reviewed in a manner consistent with this chapter by the Scientific Review Panel on Toxic Air Contaminants established pursuant to Article 5 (commencing with Section 39670). Any person may submit any information for consideration by the panel, which may receive oral testimony.

(d) The office shall submit its written evaluation and recommendations to the state board within 90 days after receiving the request of the state board pursuant to subdivision (a). The office may, however, petition the state board for an extension of the deadline, not to exceed 30 days, setting forth its statement of the reasons that prevent the office from completing its evaluation and recommendations within 90 days. Upon receipt of a request for extension of, or noncompliance with, the deadline contained in this section, the state board shall immediately transmit to the Assembly Committee on Rules and the Senate Committee on Rules, for transmittal to the appropriate standing, select, or joint committee of the Legislature, a statement of reasons for extension of the deadline, along with copies of the office's statement of reasons that prevent it from completing its evaluation and recommendations in a timely manner.

(e)

(1) The state board or a district may request, and any person shall provide, information on any substance that is or may be under evaluation and that is manufactured, distributed, emitted, or used by the person of whom the request is made, in order to carry out its responsibilities pursuant to this chapter. To the extent practical, the state board or a district may collect the

information in aggregate form or in any other manner designed to protect trade secrets.

(2) Any person providing information pursuant to this subdivision may, at the time of submission, identify a portion of the information submitted to the state board or a district as a trade secret and shall support the claim of a trade secret, upon the written request of the state board or district board. Subject to Section 1060 of the Evidence Code, information supplied that is a trade secret, as specified in Section 6254.7 of the Government Code, and that is so marked at the time of submission, shall not be released to any member of the public. This section does not prohibit the exchange of properly designated trade secrets between public agencies when those trade secrets are relevant and necessary to the exercise of their jurisdiction if the public agencies exchanging those trade secrets preserve the protections afforded that information by this paragraph.

(3) Any information not identified as a trade secret shall be available to the public unless exempted from disclosure by other provisions of law. The fact that information is claimed to be a trade secret is public information. Upon receipt of a request for the release of information that has been claimed to be a trade secret, the state board or district shall immediately notify the person who submitted the information, and shall determine whether or not the information claimed to be a trade secret is to be released to the public. The state board or district board, as the case may be, shall make its determination within 60 days after receiving the request for disclosure, but not before 30 days following the notification of the person who submitted the information. If the state board or district decides to make the information public, it shall provide the person who submitted the information 10 days' notice prior to public disclosure of the information.

(f) The office and the state board shall give priority to the evaluation and regulation of substances based on factors related to the risk of harm to public health, amount or potential amount of emissions, manner of, and exposure to, usage of the substance in California, persistence in the atmosphere, and ambient concentrations in the community. In determining the importance of these factors, the office and the state board shall consider all of the following information, to the extent that it is available:

(1) Research and monitoring data collected by the state board and the districts pursuant to Sections 39607, 39617.5, 39701, and 40715, and by the United States Environmental Protection Agency pursuant to paragraph (2) of subsection (k) of Section 112 of the federal act (42 U.S.C. Sec. 7412(k)(2)).

(2) Emissions inventory data reported for substances subject to Part 6 (commencing with Section 44300) and the risk assessments prepared for those substances.

(3) Toxic chemical release data reported to the state emergency response commission pursuant to Section 313 of the Emergency Planning and Community Right-To-Know Act of 1986 (42 U.S.C. Sec. 11023) and Section 6607 of the Pollution Prevention Act of 1990 (42 U.S.C. Sec. 13106).

(4) Information on estimated actual exposures to substances based on geographic and demographic data and on data derived from analytical methods that measure the dispersion and concentrations of substances in ambient air.

SECTION 6.
Article 4.5 (commencing with Section 39669.5) is added to Chapter 3.5 of Part 2 of Division 26 of the Health and Safety Code, to read:
Article 4.5. Special Provisions For Infants And Children
39669.5. The Legislature finds and declares that certain toxic air contaminants may pose risks that cause infants and children to be especially susceptible to illness and that certain actions are necessary to ensure their safety from toxic air contaminants.

(a) By July 1, 2001, the following shall occur
  (1) The office, in consultation with the state board, shall establish a list of up to five toxic air contaminants identified or designated by the state board pursuant to Section 39657 that may cause infants and children to be especially susceptible to illness. In developing the list, the office shall take into account public exposures to toxic air contaminants, whether by themselves or interacting with other toxic air contaminants or criteria pollutants, and the factors listed in subdivision (c) of Section 39660. The office shall submit a report containing the list and its reasons for including the toxic air contaminants on the list to the Scientific Review Panel on Toxic Air Contaminants established pursuant to Article 5 (commencing with Section 39670).
  (2) The scientific review panel, in a manner consistent with this chapter, shall review the list of toxic air contaminants submitted by the office pursuant to paragraph (1). As part of the review, any person may submit any information for consideration by the panel, which may receive oral testimony.

(b)
  (1) Within two years of the establishment of the list required pursuant to subdivision (a), the state board shall review and, as appropriate, revise any control measures adopted for the toxic air contaminants identified on the list, to reduce exposure to those toxic air contaminants pursuant to Article 4 (commencing with Section 39665), to protect public health, and particularly infants and children.
  (2) Within three years of the establishment of the list required pursuant to subdivision (a), for up to five of those toxic air contaminants for which no control measures have been previously adopted, the state board shall prepare a report on the need for regulations, following the procedure specified in Section 39665. The state board shall adopt within that same three-year timeframe, as appropriate, any new control measures to reduce exposure to those toxic air contaminants pursuant to Article 4 (commencing with Section 39665), to protect public health, particularly infants and children.

(c) Beginning July 1, 2004, the office shall annually evaluate at least 15 toxic air contaminants identified or designated by the state board pursuant to Section 39657, and provide threshold exposure levels and nonthreshold health values, as

appropriate, for those toxic air contaminants. The activities required pursuant to this subdivision shall continue until all toxic air contaminants are evaluated. The levels shall be established pursuant to the procedures adopted for health and risk assessments pursuant to paragraph (2) of subdivision (b) of Section 44360, and taking into account the factors listed in subdivision (c) of Section 39660. Based on this evaluation, and after review by the scientific review panel as prescribed in paragraph (2) of subdivision (a), the office shall update the list established pursuant to subdivision (a), by July 1, 2005, and each year thereafter. Within three years of the initial or subsequent listing update, for up to five of the toxic air contaminants contained on that list for which no control measures have been previously adopted, or for at least five of the toxic air contaminants if more than five toxic air contaminants have been identified, the state board shall prepare a report on the need for regulation, following the procedure specified in Section 39665. The state board shall adopt within that three-year timeframe, as appropriate, new control measures, pursuant to Article 4 (commencing with Section 39665), to reduce exposure to those toxic air contaminants, to protect public health, and particularly infants and children.

(d) Toxic air contaminants evaluated and listed pursuant to this section shall not include substances in those uses that are not subject to regulation by the state board pursuant to this chapter.

SECTION 7.
Section 40451 of the Health and Safety Code is amended to read:
40451.

(a) The south coast district shall use the Pollutant Standards Index developed by the Environmental Protection Agency and shall report and forecast pollutant levels daily for dissemination in the print and electronic media.

(b) Using existing communication facilities available to it, the south coast district shall notify all schools and, to the extent feasible and upon request, daycare centers in the South Coast Air Basin whenever any federal primary ambient air quality standard is predicted to be exceeded.

(c) Whenever it becomes available, the south coast district shall disseminate to schools, amateur adult and youth athletic organizations, and all public agencies operating parks and recreational facilities in the south coast district the latest scientific information and evidence regarding the need to restrict exercise and other outdoor activities during periods when federal primary air quality standards are exceeded.

(d) Once every two months and annually, the south coast district shall report on the number of days and locations that federal and state ambient air quality standards were exceeded and the number of days and locations of these occurrences.

SECTION 7.5.
Section 40451 of the Health and Safety Code is amended to read:
40451.

(a) The south coast district shall use the Pollutant Standards Index developed by the United States Environmental Protection Agency and shall report and forecast

pollutant levels daily for dissemination in the print and electronic media. Commencing July 1, 2001, the south coast district shall also include in its report and forecast levels of PM2.5 in excess of the 24-hour federal ambient air standard, as adopted in July 1997, or any standard adopted by the United States Environmental Protection Agency that succeeds that standard.

(b) Using existing communication facilities available to it, the south coast district shall notify all schools and, to the extent feasible and upon request, daycare centers in the South Coast Air Basin whenever any federal primary ambient air quality standard is predicted to be exceeded. Commencing July 1, 2001, using communication facilities available to it, the south coast district shall also notify all schools in the South Coast Air Basin when the ambient level of PM2.5 is predicted to exceed the 24-hour federal ambient air standard, as adopted in July 1997, or any standard adopted by the United States Environmental Protection Agency that succeeds that standard.

(c) Whenever it becomes available, the south coast district shall disseminate to schools, amateur adult and youth athletic organizations, and all public agencies operating parks and recreational facilities in the south coast district the latest scientific information and evidence regarding the need to restrict exercise and other outdoor activities during periods when federal primary air quality standards and the 24-hour federal ambient air standard for PM2.5, as adopted in July 1997, or any standards adopted by the United States Environmental Protection Agency that succeed those standards, are exceeded.

(d) Once every two months and annually, the south coast district shall report on the number of days and locations that federal and state ambient air quality standards were exceeded. Commencing July 1, 2001, the south coast district shall also include in that report the number of days and locations on and at which the 24-hour federal ambient air standard for PM2.5, as adopted in July 1997, or any standard adopted by the United States Environmental Protection Agency that succeeds that standard, is exceeded.

SECTION 8.

Section 7.5 of this bill incorporates amendments to Section 40451 of the Health and Safety Code proposed by both this bill and SB 1195. It shall only become operative if

(1) both bills are enacted and become effective on or before January 1, 2000,

(2) each bill amends Section 40451 of the Health and Safety Code, and

(3) this bill is enacted after SB 1195, in which case Section 7 of this bill shall not become operative.

SECTION 9.

Notwithstanding Section 17610 of the Government Code, if the Commission on State Mandates determines that this act contains costs mandated by the state, reimbursement to local agencies and school districts for those costs shall be made pursuant to Part 7 (commencing with Section 17500) of Division 4 of Title 2 of the Government Code. If the statewide cost of the claim for reimbursement does not exceed one million dollars ($1,000,000), reimbursement shall be made from the State Mandates Claims Fund.

Page intentionally left blank

Appendix C:

Asbestos Conversion Factors & Cancer Potency Factor

C-1. Overview

The purpose of this appendix is to provide information about the asbestos conversion factors and inhalation cancer potency factor. The table below summarizes the available conversion factors and inhalation cancer potency factor for asbestos. The subsequent sections of this appendix provide information on how the conversion factors and cancer potency factor were derived.

PCM Fibers to TEM Structure Conversion Factor	1 PCM Fiber = 320 TEM Structures
Mass to Fiber Conversion Factor	0.003 μg = 100 asbestos PCM Fibers
Asbestos Inhalation Cancer Potency Factor	$2.2 \times 10^{+2}$ $(mg/kg\text{-}day)^{-1}$

C-2. PCM Fiber to TEM Structure Conversion Factor

Two analytical methods have been used for the analysis of asbestos samples: phase contrast microscopy (PCM), the primary method used historically to analyze asbestos samples, and transmission electron microscopy (TEM), the current state-of-the-art method.

PCM analysis was developed earlier and has been preferred in the past over TEM because it could be done more quickly and was less expensive. However, one major limitation of PCM analysis, especially in outdoor environments, is that the analyst cannot distinguish asbestos from non-asbestos fibers, such as cellulose, talc, or gypsum. Also, PCM cannot detect fibers that have a diameter of about 0.3 microns or less, which could substantially underestimate the asbestos fiber concentrations. These limitations make PCM impractical for the analysis of ambient asbestos samples.

TEM is the preferred analytical method for outdoor asbestos samples because of its ability to detect small fibers (greater than or equal to 0.0002 microns in diameter) and to distinguish between asbestos fibers and non-asbestos fibers. The term “TEM structures” is often used to describe asbestos fibers detected by this method. TEM is the method recommended by the Office of Environmental Health Hazard Assessment (OEHHA). However, TEM measurements cannot be directly related to the cancer

potency factors because the studies upon which OEHHA's risk assessment was based used PCM analysis. Thus, the TEM measurements must be converted to PCM-equivalent units. The actual relationship between PCM and TEM measurements is quite variable: ARB (1990) found a range of 100 to 1000 for the ratio of TEM structures to PCM fibers for three occupational studies. For the purpose of the Air Toxics Hot Spots Program, asbestos should be converted using the geometric center of the range as defined by ARB (1990). To convert PCM fibers to TEM structures or vice versa use the following relationship:

1 PCM Fiber = 320 TEM structures

C-3. Mass to Fiber Conversion Factor

Asbestos is reported in units of pounds per year under the Air Toxics Hot Spots Program. To convert asbestos fibers to mass, the following relationship is used:

0.003 μg = 100 asbestos fibers PCM.

This conversion factor was derived from information published by the United States Environmental Protection Agency (U.S. EPA) (U.S. EPA, 1986). The number of asbestos PCM fibers associated with a given mass of asbestos can vary appreciably. In addition, U.S. EPA has stated that this conversion factor is the geometric mean of measured relationships between optical fiber counts and mass airborne chrysotile in several published studies, that the range of the conversion factor between the different studies is large (0.0005 - 0.015 μg asbestos/100 asbestos PCM fibers), and that the factor carries with it an appreciable uncertainty. Additionally, if the asbestos was analyzed using TEM, the TEM structures must be converted to PCM fibers first.

C-4. Asbestos Inhalation Cancer Potency Factor

The unit risk factor for asbestos fibers is 1.9×10^{-4} in units of $(100 \text{ PCM fibers/m}^3)^{-1}$. The unit risk factor is based on epidemiological studies in which PCM fiber measurements were used. This unit risk factor is listed in Chapter 7 and in the Asbestos Toxic Air Contaminant (TAC) identification document (CDHS, 1986) and in OEHHA, 2009. The asbestos cancer potency factor is for mesothelioma. Since the unit risk factor is in units of concentration or dose, complications arise when the emitted asbestos quantities are reported in mass units (pounds/year and maximum pounds/hour) for the Air Toxics Hot Spots Program.

For the purpose of an Air Toxics Hot Spots Risk Assessment, the cancer potency factor $(\text{mg/kg body weight})^{-1}$ may be calculated from the fiber cancer potency factor using the relationship of 0.003 μg = 100 fibers PCM, 70 kg body weight, 20 m^3 breathed per day, and a factor of 1000 to convert μg asbestos into mg:

$$1.9\times10^{-4}\left(100\,PCM\,fibers/m^3\right)^{-1}\times\frac{70kg}{20m^3}\times\frac{1000}{0.003\mu g/100fibers}=2.2\times10^{+2}\left(mg/kg\,bodyweight\right)^{-1}$$

In order to use this cancer potency factor under the Air Toxics Hot Spots Program, the measured asbestos concentration should be expressed as microgram per cubic meter. For example, if the measured asbestos concentrations are in units of TEM structures per cubic meter, the asbestos concentration should be first converted to PCM fibers per cubic meters and then into units of microgram per cubic meters using the conversion factors as shown in the sections above.

See Chapter 8 for more information on calculating cancer risk and Appendix I for an example of how cancer risk is calculated for the inhalation pathway. Note, while the example in Appendix I uses non-asbestos substances, it is still applicable since it illustrates the steps that are used for asbestos, including use of Age Sensitivity Factors.

C-5. References

ARB, 1990. Proposed Control Measure for Asbestos-Containing Serpentine Rock in Surfacing Applications, Technical Support Document, Air Resources Board, February 1990.

CDHS, (1986) California Department of Health Services (CDHS) 1986. Report to the Air Resources Board on Asbestos. Part B. Health Effects of Asbestos. Epidemiological Studies Section, Berkeley, CA.

OEHHA, 2009. The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II. Technical Support Document for Describing Available Cancer Potency Factors, Office of Environmental Health Hazard Assessment, May 2009. Available online at http://www.oehha.ca.gov

USEPA, 1986. Airborne Asbestos Health Assessment Update. EPA/600/8-84/003F, Office of Health and Environmental Assessment, Washington, DC.

Page intentionally left blank

Appendix D:

Risk Assessment Procedures to Evaluate Particulate Emissions from Diesel-Fueled Engines

D-1. Introduction

The objective of this appendix is to discuss procedures for estimating the cancer and noncancer health risk from exposure to particulate matter (PM) emissions from diesel-fueled engines (diesel exhaust). It will also clarify the requirements and recommendations for acute noncancer and multipathway cancer and chronic risk assessment for diesel PM. In addition to the notification and risk reduction requirements under the Hot Spots Program, this appendix should facilitate the use of the *Risk Reduction Plan to Reduce Particulate Matter Emissions from Diesel-Fueled Engines and Vehicles* (ARB, 2000) (Diesel Guidelines). The Diesel Guidelines were developed by the Air Resources Board (ARB) with assistance from the Office of Environmental Health Hazard Assessment (OEHHA) in October 2000. The Diesel Guidelines are intended to assist local Air Pollution Control and Air Quality Management Districts (Districts) and sources of diesel PM emissions in making consistent risk management decisions.

In advance of performing a health risk assessment (HRA), it is recommended that the District and the stationary source of diesel emissions reach a consensus on the HRA approach for estimating health impacts from diesel exhaust. See Chapter 9 for an outline of a modeling protocol.

D-2. Calculations/Risk Assessment Procedures

In August 1998, the ARB identified diesel exhaust as a toxic air contaminant (TAC) (ARB, 1998). In the identification report, OEHHA provided an inhalation noncancer chronic reference exposure level (REL) of 5 micrograms per cubic meter ($\mu g/m^3$) and a range of inhalation cancer potency factors of 1.3×10^{-4} to 2.4×10^{-3} $(\mu g/m^3)^{-1}$. The Scientific Review Panel on Toxic Air Contaminants recommended a "reasonable estimate" inhalation unit risk factor of 3.0×10^{-4} $(\mu g/m^3)^{-1}$. From the unit risk factor an inhalation cancer potency factor of 1.1 $(mg/kg\text{-}day)^{-1}$ may be calculated. These noncancer and cancer health factors were developed based on whole (gas and particulate matter) diesel exhaust. The surrogate for whole diesel exhaust is diesel PM. PM_{10} (particulate matter, ten microns or less in size) is the basis for the risk calculations.

D-2.1 Cancer

An inhalation cancer risk is required for every HRA (The methods for calculating inhalation cancer risk can be found in Chapters 5, 7, and 8.). When comparing whole diesel exhaust to speciated components of diesel exhaust (e.g., PAHs, metals), the cancer risk from inhalation exposure to whole diesel exhaust will outweigh the

multipathway cancer risk from the speciated components. For this reason, there will be few situations where an analysis of multipathway risk is necessary.

The District may elect to require a multipathway analysis if reliable data are available and the District decides that it is necessary. If the District elects to require a multipathway analysis, the components of the diesel exhaust will need to be speciated since there is no oral cancer potency factor for diesel PM. It is recommended that the District be consulted on the procedures for conducting a multipathway analysis for diesel exhaust. The District may wish to use speciation data from the ARB. If so, a resource for speciation data is available on the ARB's website at www.arb.ca.gov/emisinv/speciate/speciate.htm.

If a multipathway analysis is required, the speciated data should be compared with the substances in Table 5.1. Any substances in the speciation profile that are listed in Table 5.1 and have an oral cancer potency factor in Table 7.1 should be included in the multipathway analysis. Multipathway cancer risks are estimated following the procedures in Chapters 5 and 8 of this document. These procedures require summing the cancer risk from each carcinogen to estimate the total facility cancer risk.

D-2.2 Noncancer Chronic

To determine noncancer chronic inhalation health impacts from exposure to diesel exhaust use the methods described in Chapters 6 and 8.

The District may elect to require a multipathway analysis if reliable data are available and they feel it is necessary. If the District elects to require a multipathway analysis, the components of the diesel exhaust will need to be speciated since there is no oral reference exposure level for diesel PM. A resource for speciation data at the ARB is identified above. It is recommended that the District be consulted on the procedures for conducting a multipathway analysis. If a multipathway analysis is required, the speciated data should be compared with the substances in Table 5.1. Any substances in the speciation profile that are listed in Table 5.1 and have an oral chronic REL in Table 6.4 should be included in the multipathway analysis. Multipathway chronic risks are estimated following the procedures in Chapters 5 and 8 of this document.

Note that the effect estimate for cardiovascular mortality from exposure to ambient PM when applied to diesel PM results in many more cardiovascular deaths than lung cancer deaths.

D-2.3 Noncancer Acute

There may be certain unusual situations where an evaluation of the acute health effects may be warranted. One possible situation is when a nearby receptor is located above the emission release point (e.g. on a hillside or in a multistory apartment building). Since there is no acute REL for diesel exhaust, the components of the exhaust will need to be speciated to determine the potential acute health impacts. It is recommended that the District be consulted on the procedures for conducting an acute analysis. If an acute analysis is required, the speciated data should be compared with the substances in Table 6.1. Any substances in the speciation profile that are listed in Table 6.1 should be included in the acute analysis. A resource for speciation data at the ARB is identified above. Acute risks are estimated following the procedures in Chapters 6 and 8 of this document.

D-3. References

ARB 1998. Air Resources Board, “Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant, Appendix III, Part A, Exposure Assessment,” April 1998.

ARB 2000. Air Resources Board, “Risk Reduction Plan to Reduce Particulate Matter Emissions from Diesel-Fueled Engines and Vehicles,” October 2000.

Page intentionally left blank

Appendix E:

Toxicity Equivalency Factors for Polychlorinated Dibenzo-p-Dioxins, Dibenzofurans and Polychlorinated Biphenyls

Polychlorinated dibenzo-p-dioxins and dibenzofurans (dioxins and furans) and polychlorinated biphenyls (PCBs) vary considerably in their potency for causing both cancer and noncancer health impacts. A Toxics Equivalents Factors (TEF) scheme, based on both cancer and noncancer toxicity studies, has been developed to relate the potency of various dioxin and furan congeners and PCB congeners to the potency of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD). A detailed explanation of the World Health Organization's 2005 Toxicity Equivalents Factor (WHO_{05}-TEF) scheme (van den Berg et al., 2006), the latest scheme adopted by OEHHA, is available in OEHHA (2011). Where the dioxin and furan and PCB mixtures are not speciated, the cancer risk and noncancer hazard index are based on the most potent congeners. A facility may choose to speciate dioxin and furan and PCB emissions in order to obtain a more accurate picture of the risks.

There are two mathematically equivalent procedures for estimating the cancer risk or the non-cancer hazard quotient using the TEF scheme. In the first method, the concentration or dose of 2,3,7,8-TCDD equivalents is calculated based on the individual congener concentration or dose multiplied by the TEF for that congener. Cancer risk is estimated by multiplying the cancer slope for 2,3,7,8-TCDD by the "TCDD equivalents" concentration or dose. The noncancer hazard quotient would be calculated by dividing the "TCDD equivalents" concentration by the REL. In the second method, TEF-adjusted potency factors or RELs are used with individual congeners.

Using the first procedure, the concentration of each congener listed in Table E-1 is multiplied by the WHO_{05}-TEF for that congener to estimate the concentration of 2,3,7,8-TCDD "toxic equivalents" of the mixture. For example, for 1,2,3,4,7,8-hexachlorodibenzodioxin, the concentration ($\mu g/m^3$) may be multiplied by 0.1 to give the concentration equivalent to 2,3,7,8-TCDD. Congeners not listed in the table are assumed to have no dioxin-like toxicity. The 2,3,7,8-TCDD equivalent concentrations for each congener in the mixture are summed and treated as 2,3,7,8-TCDD for the purposes of calculating cancer and noncancer risks. Thus, to estimate cancer risk, the "toxic equivalents" concentration is multiplied by the breathing rate to give dose (see equation 5.4.1.1), and then multiplied by the cancer potency factor for 2,3,7,8-TCDD (Table 7.1) to give the cancer risk for the entire mixture.

To estimate the chronic non-cancer inhalation hazard index, the ground level concentration of the 2,3,7,8-TCDD equivalents of the mixture is divided by the chronic reference exposure level for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin to give an inhalation hazard index for the entire mixture. Similarly, the oral chronic hazard index of the mixture is calculated by estimating the 2,3,7,8-TCDD equivalents dose and dividing by the oral chronic REL for 2,3,7,8-TCDD. The inhalation and oral hazard indices are then summed to get a total chronic Hazard Index.

In order to determine the inhalation chronic hazard index by the second procedure, the ground level concentration of each dioxin and furan congener is divided by the chronic REL for each congener in Table 6.3 and the hazard quotients summed to give the inhalation chronic hazard index. The oral chronic hazard quotient is calculated by determining the oral dose of each congener and dividing by the individual chronic oral REL (Table 6.4) for each congener. The oral hazard quotients are then summed to give the oral chronic hazard index for dioxins and furans and PCBs. The oral hazard index is then added to the inhalation hazard index to give the total chronic hazard index for dioxins and furans and PCBs.

In those cases where speciation of dioxins and furans has not been performed, then 2,3,7,8-TCDD serves as the surrogate for dioxin and furan emissions. Given that 2,3,7,8-TCDD is the most potent congener in the class, the resulting risk estimate for an unspeciated mixture may be deemed significant enough to trigger health concerns. In this case, it would then be advisable to speciate the mixture and run a screening estimate using the speciated data.

As noted above, the TEF scheme includes TEFs for individual coplanar PCB congeners (see Table E-1) (OEHHA, 2011). These are the congeners that have dioxin-like biological effects. Where data are available on individual PCB congeners emitted by a facility, then the congener-specific TEFs are used. 2,3,7,8-TCDD also serves as the surrogate for the coplanar PCB congeners. To calculate the noncancer inhalation and oral RELs for individual PCB congeners shown in Tables 6.3 and 6.4, respectively, the inhalation and oral RELs for 2,3,7,8-TCDD were divided by the PCB congener TEFs in Table E-1. If only total PCB data are available, then the PCB slope factors for high, low and lowest risk provided in Table 7.1 can be used for cancer risk determination. The high risk potency factor is the default for unspeciated PCB mixtures.

As of February, 2015, there is no approved method that can be used to assess the noncancer hazard of an unspeciated PCB mixture. Persons preparing HRAs for the Hot Spots Program should consult with OEHHA and the local Air Pollution Control or Air Quality Management District if an assessment of the noncancer hazard for unspeciated PCB mixtures is needed.

TABLE E-1. WHO/05 TOXIC EQUIVALENCY FACTORS (TEFS)

Congener		TEF_{WHO-05}
PCDDs		
2,3,7,8-TCDD		1
1,2,3,7,8-PeCDD		1
1,2,3,4,7,8-HxCDD		0.1
1,2,3,6,7,8-HxCDD		0.1
1,2,3,7,8,9-HxCDD		0.1
1,2,3,4,6,7,8-HpCDD		0.01
1,2,3,4,6,7,8,9-OCDD		0.0003
PCDFs		
2,3,7,8-TCDF		0.1
1,2,3,7,8-PeCDF		0.03
2,3,4,7,8-PeCDF		0.3
1,2,3,4,7,8-HxCDF		0.1
1,2,3,6,7,8-HxCDF		0.1
1,2,3,7,8,9-HxCDF		0.1
2,3,4,6,7,8-HxCDF		0.1
1,2,3,4,6,7,8-HpCDF		0.01
1,2,3,4,7,8,9-HpCDF		0.01
1,2,3,4,6,7,8,9-OCDF		0.0003
PCBs (IUPAC #, Structure)		
77	3,3',4,4'-TCB	0.0001
81	3,4,4',5-TCB	0.0003
105	2,3,3',4,4'-PeCB	0.00003
114	2,3,4,4',5-PeCB	0.00003
118	2,3',4,4',5-PeCB	0.00003
123	2',3,4,4',5-PeCB	0.00003
126	3,3',4,4',5-PeCB	0.1
156	2,3,3',4,4',5-HxCB	0.00003
157	2,3,3',4,4',5'-HxCB	0.00003
167	2,3',4,4',5,5'-HxCB	0.00003
169	3,3',4,4',5,5'-HxCB	0.03
170	2,2',3,3',4,4',5-HpCB	-
180	2,2',3,4,4',5,5'-HpCB	-
189	2,3,3',4,4',5,5'-HpCB	0.00003

References

OEHHA, 2011. Technical Support Document for Cancer Potency Factors, Appendix C: Use of the Toxicity Equivalency Factor (TEFWHO 05) Scheme for Estimating Toxicity of Mixtures of Dioxin-Like Chemicals. Office of Environmental Health Hazard Assessment, Sacramento, CA. January, 2011. Available at http://www.oehha.ca.gov/air/hot_spots/pdf/AppCdioxinTEFs013111.pdf.

Van den Berg M, Birnbaum L, Denison M, De Vito M, Farland W, Feeley M, Fiedler H, Hakansson H, Hanberg A, Haws L, Rose M, Safe S, Schrenk D, Tohyama C, Tritscher A, Tuomisto J, Tysklind M, Walker N, Peterson RE. 2006. The 2005 World Health Organization reevaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. Toxicol Sci 93:223-241.

Appendix F:

Overview of the Lead Risk Assessment Procedures

F.1 Introduction

The objective of this appendix is to provide a method for estimating potential cancer and noncancer health effects due to airborne lead exposure. This appendix should facilitate the use of the *Risk Management Guidelines for New, Modified, and Existing Sources of Lead* (Lead RM Guidelines) (ARB, 2001) for analysis of lead exposure. The Lead RM Guidelines were developed by the Air Resources Board (ARB) with assistance from Office of Environmental Health Hazard Assessment (OEHHA) and Department of Health Services (DHS) in March 2001 to assist local air districts and sources of lead in making consistent risk management decisions for new, modified, and existing sources of lead.

In April 1997, the ARB identified inorganic lead as a toxic air contaminant (TAC) (ARB, 1997). Lead is unique among other TACs identified by ARB in several ways. First, infants and children are particularly susceptible to the health effects of lead, and the risk assessment is based on health effects in children. Second, the chronic noncancer effects are related to blood lead levels (BLLs) as opposed to ambient air concentrations. These BLLs reflect current and past exposure from a number of sources; air emissions may only be a small part of the total exposure. Third, based on recommendations of the OEHHA and the Scientific Review Panel on Toxic Air Contaminants (SRP), the ARB did not identify a threshold level for chronic noncancer health effects due to lead exposure. Threshold levels are levels below which no adverse health effects are expected to occur. Since acute, 8-hour or chronic Reference Exposure Levels (RELs) are based on threshold levels, none were developed for lead. Thus, a hazard index approach is not used for lead. Instead, air concentrations are compared to defined air lead levels associated with specified percentages of children with BLL $\geq$ 10 µg/dL. Acceptable risk is based on minimizing the number of children at or above a BLL of 10 µg/dL.

F.2 Methods for Estimation of Health Risk Effects

Methods for estimating site-specific noncancer and cancer potential health impacts from exposure to lead emissions are given in the Lead RM Guidelines. The noncancer health effects pose greater public health significance than the cancer health effects. Minimizing noncancer health effects of lead will therefore also minimize cancer health effects.

Chronic noncancer health risks are estimated based on neurodevelopmental health risks to children and would also be protective of adults. These health effects can be evaluated using a tiered approach based on blood lead level distribution in the population.

Potential multipathway cancer risks are estimated following the procedures in Chapters 5 and 8 of this document. These procedures require summing individual cancer risk from each carcinogen to estimate the total facility cancer risk.

In advance of performing a health risk assessment (HRA), it is recommended that the Air Pollution Control or Air Quality Management District (District) and the stationary source of lead air emissions reach a consensus on the HRA approach for estimating chronic noncancer and cancer health risks. See Chapter 9 for an outline of a modeling protocol.

F.2.1 Tiered Approach for Estimating Noncancer Risks due to Lead Exposure

The Lead Risk Management Guidelines provide three tiers of analysis to determine baseline BLL distributions for estimating risk. Although there is a simple risk management option provided in the Lead RM Guidelines, in a risk assessment for the Air Toxics Hot Spots program one of the following tiers must be used to report estimates of the percent of children estimated to be above 10 μg/dL blood lead. The tiered approach is based on an assessment of neurodevelopmental risk, with the BLL distribution in the population as the most significant factor. The BLL distribution consists of two components: 1) the baseline BLL distribution due to all sources of exposure; and 2) the exposure due to emissions from a facility.

Tier I is a default approach that requires minimal site-specific information on concentrations of lead in environmental media other than air. Tier I uses two default BLL distributions, one for a high exposure scenario and one for an average exposure scenario. The exposure scenario is determined using the median age of the homes in the census tract and the ratio of area income to the poverty level. The default baseline BLL distribution for each of the exposure scenarios is based on a review of neighborhood and community blood lead studies. The assessor determines the 30-day average lead concentration due to the facility averaged over the 1 square kilometer area centered on the Maximum Offsite Concentration (MOC). The percentage of children with BLLs greater than or equal to 10 micrograms per deciliter ($\geq$ 10 μg/dL) is determined using Table F-1 (also found on page 17 in the Lead RM Guidelines), the air lead concentration, and the determined exposure scenario. The 10 μg/dL threshold level has been identified by the Centers for Disease Control and Prevention (CDC) as a level where potential health effects may occur. The public health goal of management practices should be to implement procedures/practices to prevent BLLs at or above this level. The estimated percentage of children with BLLs $\geq$ 10 μg/dL is then used with risk management levels given in Chapter III, Section D of the Lead RM Guidelines to assist in making risk management decisions.

TABLE F-1 PERCENTAGE OF CHILDREN WITH BLOOD LEAD LEVELS ≥ 10 μG/DL FOR VARIOUS AIR LEAD CONCENTRATIONS AT TWO EXPOSURE SCENARIOS

Air Lead Concentration in the Maximum Exposure Area (30-day average) [μg/m^3]	Percent ≥ 10 μg/dL	
	High Exposure Scenario	Average Exposure Scenario
Baseline*	5.1	1.2
0.02	5.4	1.4
0.06	6.1	1.7
0.10	6.8	2.2
0.20	8.9	3.4
0.25	9.8	4.1
0.50	15.9	8.9
0.75	22.4	15.4
1.0	29.1	23.0
1.5	42.5	39.0

* The baseline represents BLLs due to lead in soil, dust, water, food, and background air lead concentrations.

Tier II requires the development of site-specific baseline BLL distributions within the impacted population using site-specific estimates of lead levels in environmental media, including soil, dust, water, and/or food, using the U.S. EPA Integrated Exposure Uptake Biokinetic (IEUBK) model. The IEUBK model calculates the probability of an individual exceeding a specific BLL based on site-specific information. The aggregate of the individual BLLs is used to estimate the neurodevelopmental risk in the maximum exposure area. A detailed discussion of this tier is beyond the scope of this overview; see Appendix D in the Lead RM Guidelines for a discussion of the IEUBK model and its use.

Tier III involves actual blood lead sampling of the population impacted by the facility to define the baseline BLLs. In Tier III, the facility is responsible for conducting BLL testing to establish a site-specific BLL distribution. The Lead RM Guidelines recommend the neurodevelopmental risk be calculated as the probability of children in an affected exposure area having a BLL ≥ 10 μg/dL using the results of the blood lead sampling. It is highly unlikely that this option would be used due to the cost incurred and the fact that the sampled population must consent to the sampling and an appropriate sampling strategy must be developed to adequately characterize the blood lead levels of the impacted population.

For further information on the tiered approach using the Tier I, Tier II, or Tier III, please see Chapter II of the *ARB Risk Management Guidelines for New, Modified, and Existing Sources of Lead* (ARB, 2001). This document can be downloaded from the ARB web site at http://www.arb.ca.gov/toxics/lead/lead.htm or can be requested by calling (916) 323-4327.

F.2.2 Methods for Estimating Potential Cancer Risks due to Lead

While lead has a unique noncancer assessment methodology, the determination of potential multipathway cancer risk is the same as other carcinogens. Chapters 5, 7, and 8, and Appendices I and L provide all the needed information for calculating potential cancer risk. The health risk assessment should report the multipathway cancer risks from lead emissions.

Chapter III in the Lead RM Guidelines provides methods for determining risk management of lead exposure, using the results from the cancer risk calculation, and the local District's defined significance levels.

F.3 References

ARB, 1997. Proposed Identification Inorganic Lead as a Toxic Air Contaminant, Parts A, B, C. California Air Resources Board. April, 1997.

ARB, 2001. ARB Risk Management Guidelines for New, Modified, and Existing Sources of Lead. California Air Resources Board. March 2001

Appendix G:

PAH Potency Factors and Selection of Potency Equivalency Factors (PEF) for PAHs based on Benzo(*a*)pyrene Potency

The Office of Environmental Health Hazard Assessment (OEHHA) has developed a Potency Equivalency Factor (PEF) procedure to assess the relative potencies of PAHs and PAH derivatives as a group. Benzo(a)pyrene (BaP) was chosen as the primary representative of the class of polycyclic aromatic hydrocarbons (PAHs) because of the large amount of toxicological data available on BaP (versus the relatively incomplete database for other PAHs), and because it serves as the referent PAH for the Potency Equivalency Factors. This procedure can address the impact of carcinogenic PAHs in ambient air since they are usually present together. This procedure was approved by the Scientific Review Panel (SRP) on Toxic Air Contaminants (TAC) as part of the Health Effects Assessment of Benzo(a)pyrene during the TAC identification process (OEHHA, 1993).

Due to the variety of data available on the carcinogenicity and mutagenicity of PAHs, an order of preference for the use of available data in assessing relative potency was developed. If a health effects evaluation and quantitative risk assessment leading to a cancer potency value had been conducted on a specific PAH, then those values were given the highest preference. Cancer potency values for PAHs developed by this process are shown in Table G-1.

TABLE G-1: POTENCIES OF PAHS AND DERIVATIVES[1]

Chemicals	Cancer potency factors $(mg/kg\text{-}day)^{-1}$	Unit risks $(\mu g/m^3)^{-1}$
Benzo[*a*]pyrene	11.5	1.1×10^{-3}
Dibenz[*a*,*h*]anthracene	4.1	1.2×10^{-4}
7,12-dimethylbenzanthracene	250	7.1×10^{-2}
3-methylcholanthrene	22	6.3×10^{-3}
Naphthalene[2]	0.12	3.4×10^{-5}
5-nitroacenaphthene	0.13	3.7×10^{-5}

[1] Source: OEHHA (1993); Collins *et al*. (1998); OEHHA (2009). It is assumed that unit risks for inhalation have the same relative activities as cancer potencies for oral intake.

If potency values have not been developed for specific compounds, a carcinogenic activity relative to BaP, rather than a true potency, can be developed. These relative activity values are referred to as Potency Equivalency Factors or PEFs. For air contaminants, the relative potency to BaP based on data from inhalation studies would be optimal. Otherwise, intrapulmonary or intratracheal administration studies would be most relevant, since such studies are in the target organ of interest. Next in order of

preference is information on activity by the oral route and skin painting. Intraperitoneal and subcutaneous administration rank at the bottom of the *in vivo* tests considered useful for PEF development because of their lack of relevance to environmental exposures. Next, in decreasing order of preference, are genotoxicity data, which exist for a large number of compounds. In many cases genotoxicity information is restricted to mutagenicity data. Finally, there are data on structure-activity relationships among PAH compounds. Structure-activity considerations may help identify a PAH as carcinogenic, but at this time have not been established as predictors of carcinogenic potency.

Using this order of preference, PEFs were derived for 21 PAHs and are presented in Table G-2 (OEHHA, 1993: Collins *et al*., 1998).

The cancer potency comparisons show that some PAHs are more potent than BaP, while other PAHs analyzed were less or much less potent. These comparisons indicated that considering all PAHs to be equivalent in potency to BaP would likely overestimate the cancer potency of a PAH mixture, but such an assumption would be health protective and likely to be helpful in a screening estimate of PAH risks (OEHHA, 1993). If one assumes that PAHs are as carcinogenic as they are genotoxic, then their hazard relative to BaP would be dependent on their concentration in the environment. In light of the limited information available on other PAHs, BaP remains an important representative or surrogate for this group of air pollutants.

Detailed descriptions on the criteria used for developing individual PEFs can be found in OEHHA (2009). OEHHA continues to review all recent literature pertaining to the carcinogenicity and mutagenicity of PAHs. New cancer potency values for PAHs may be developed if an adequate health effects evaluation and quantitative risk assessment can be performed. Also, some current PEFs may be modified based on new data. Any changes to the potency values and PEFs for PAHs will be reflected in the HARP program when they occur. It is incumbent on the risk assessor to access the most recent version of the HARP program to ensure that the most up-to-date PAH potency values are used.

TABLE G-2. OEHHA PEF WEIGHTING SCHEME FOR PAHS AND THEIR RESULTING CANCER POTENCY VALUES.[1]

PAH or derivative	PEF	Unit Risk $(\mu g/m^3)^{-1}$	Inhalation Slope Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
benzo[*a*]pyrene (index compound)	**1.0**	**1.1×10^{-3}**	**3.9**	**$1.2 \times 10^{+1}$**
benz[*a*]anthracene	0.1	1.1×10^{-4}	3.9×10^{-1}	1.2
benzo[b]fluoranthene	0.1	1.1×10^{-4}	3.9×10^{-1}	1.2
benzo[*j*]fluoranthene	0.1	1.1×10^{-4}	3.9×10^{-1}	1.2
benzo[k]fluoranthene	0.1	1.1×10^{-4}	3.9×10^{-1}	1.2
dibenz[*a*,*j*]acridine	0.1	1.1×10^{-4}	3.9×10^{-1}	1.2
dibenz[*a*,*h*]acridine	0.1	1.1×10^{-4}	3.9×10^{-1}	1.2
7H-dibenzo[c,*g*]carbazole	1.0	1.1×10^{-3}	3.9	$1.2 \times 10^{+1}$
dibenzo[*a*,*e*]pyrene	1.0	1.1×10^{-3}	3.9	$1.2 \times 10^{+1}$
dibenzo[*a*,*h*]pyrene	10	1.1×10^{-2}	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
dibenzo[*a*,*i*]pyrene	10	1.1×10^{-2}	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
dibenzo[*a*,*l*]pyrene	10	1.1×10^{-2}	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
indeno[1,2,3-*cd*]pyrene	0.1	1.1×10^{-4}	3.9×10^{-1}	1.2
5-methylchrysene	1.0	1.1×10^{-3}	3.9	$1.2 \times 10^{+1}$
1-nitropyrene	0.1	1.1×10^{-4}	3.9×10^{-1}	1.2
4-nitropyrene	0.1	1.1×10^{-4}	3.9×10^{-1}	1.2
1,6-dinitropyrene	10	1.1×10^{-2}	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
1,8-dinitropyrene	1.0	1.1×10^{-3}	3.9	$1.2 \times 10^{+1}$
6-nitrochrysene	10	1.1×10^{-2}	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
2-nitrofluorene	0.01	1.1×10^{-5}	3.9×10^{-2}	1.2×10^{-1}
chrysene	0.01	1.1×10^{-5}	3.9×10^{-2}	1.2×10^{-1}

1. Source: OEHHA (1993)

References

Collins, J.F., Brown, J.P., Alexeeff, G.V., and Salmon, A.G. 1998. Potency equivalency factors for some polycyclic aromatic hydrocarbons and polycyclic aromatic hydrocarbon derivatives. Regul. Toxicol. Pharmacol. 28:45-54.

OEHHA, 1993. Benzo[*a*]pyrene as a Toxic Air Contaminant. Part B. Health Effects of Benzo[*a*]pyrene. Air Toxicology and Epidemiology Section, Berkeley, CA.

OEHHA, 2009. The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II. Technical Support Document for Describing Available Cancer Potency Factors, Office of Environmental Health Hazard Assessment, May 2009.

Appendix H:

Recommendations for Estimating Concentrations of Longer Averaging Periods from the Maximum One-Hour Concentration for Screening Purposes

H.1 Introduction

The U.S. Environmental Protection Agency (U.S. EPA) AERSCREEN air dispersion model can be used to estimate the maximum one-hour concentration downwind from an emissions source. The AERSCREEN model results (or AERMOD with screening meteorological data) can also be used to estimate concentrations for longer averaging periods, such as the maximum annual average concentration. In addition, it is permissible to use the AERMOD air dispersion model in a screening mode with identical meteorological conditions as used in the AERSCREEN model to superimpose results from multiple sources.

This method to assess short-term and long-term impacts may be used as a first-level screening indicator to determine if a more refined analysis is necessary. In the event that representative meteorological data are not available, the screening assessment may be the only computer modeling method available to assess source impacts.

In California, this standard procedure will generally bias concentrations towards over-prediction in most cases when the source is a continuous release. However, in the case when a source is not continuous, these screening factors may not be biased towards over prediction. In this case, we recommend an alternative procedure for estimating screening value concentrations for longer averaging periods than one-hour for intermittent releases.

H.2 Current Procedures

The current screening factors used to estimate longer term averages (i.e., 3-hour, 8-hour, 24-hour, 30-day, and annual averages) from maximum one-hour concentrations in California are shown in Table H.1. The factors are U.S. EPA recommended values with the exception of the 30-day factor. The 30-day factor is an ARB recommended value (ARB, 1994). The maximum and minimum values are recommended limits to which one may diverge from the general case, (U.S. EPA, 1992). Diverging from the general case should only be done on a case by case basis with prior approval from the reviewing agency.

TABLE H.1 RECOMMENDED FACTORS TO CONVERT MAXIMUM 1-HOUR AVG. CONCENTRATIONS TO OTHER AVERAGING PERIODS (U.S. EPA, 2011, 1992; ARB, 1994).

Averaging Time	Range	Typical SCREEN3 Recommended	AERSCREEN Recommended
3 hours	0.8 - 1.0	0.9	1.0
8 hours	0.5 - 0.9	0.7	0.9
24 hours	0.2 - 0.6	0.4	0.6
30 days	0.2 - 0.3	0.3	
Annual	0.06 - 0.1	0.08	0.1

AERSCREEN automatically provides the converted concentration for longer than 1-hour averaging periods. For area sources, the AERSCREEN 3, 8, and 24-hour average concentrations are equal to the 1-hour concentration. No annual average concentration is calculated. SCREEN3 values are shown for comparison purposes.

H.3 Definitions

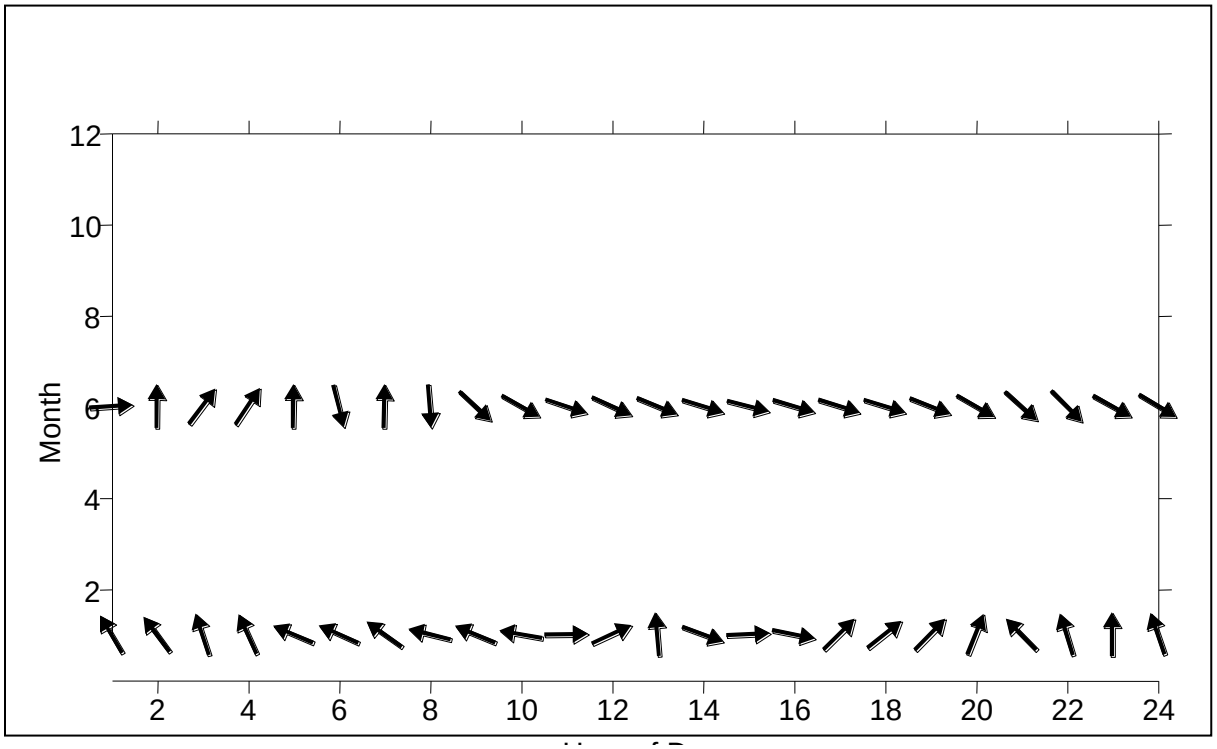
It is convenient to define the following terms relating to sources with respect to the duration of the release.

- Continuous Release – This is a release that is continuous over the duration of a year. An example of this type of release would be fugitive emissions from a 24-hour per day, 7-day per week operation or an operation that is nearly continuous.
- Intermittent Release – Many emissions fall under this category. These are emission types that are not continuous over the year. Any operation that has normal business hours (e.g., 8 am to 6 pm) would fall into this category.
- Systematic Release – These are intermittent releases that occur at a specific time of the day. As an example, these type of releases can occur when a process requires clean out at the end of the work day. Thereby releasing emissions only at the end of the workday systematically. Systematic releases are similar to intermittent releases with a shorter duration during the normal operating schedule.
- Random Release – These are intermittent releases that can occur any time during the operating schedule. An example of this type of release would be of the type that depends on batch processing. For example, a brake shop may emit pollutants only when the brakes are cleaned which happens randomly throughout the normal business hours.

H.4 Screening Factors

The U.S. EPA screening factors, as shown in Table H.1, compensate for the effects of varying conditions of wind speed, wind direction, ambient temperature, atmospheric stability, and mixing height over longer averaging periods, even though it is not explicitly indicated in the U.S. EPA Guidance (U.S. EPA, 1992). Figure H.1 shows the variability in wind direction over a 24-hour period. The data are averaged for two seven-day periods from data collected at Los Angeles International Airport (LAX). Figure H.1 was compiled for data collected in 1989 for January 1 to January 7 and June1 through June 7, 1989. The ordinate in Figure H.1 shows the months of the year. Only two months are plotted. The abscissa shows the hour of the day.

FIGURE H.1: HOURLY WIND DIRECTION - LOS ANGELES, JANUARY (BOTTOM – 1) AND JUNE (TOP - 6)



As seen in Figure H.1, the wind direction changes throughout all hours of the day. In addition, the wind direction for LAX, in the overnight and early morning hours, can vary from January to June. During the afternoon hours of 1400 – 1600, the wind direction is similar in both months of January and June.

The standard U.S. EPA screening factor to estimate the maximum 24-hour concentration from the maximum 1-hour concentration is 0.4, as seen in Table H.1. Figure H.2 shows that for 15 of 24 hours the wind blows from the west-northwest during June. A 24-hour screening factor could be 0.6 (0.6 ≈ 15hrs/24hrs) based on wind direction alone. This is consistent with the upper bound of the adjustment factors shown in Table H.1. Including the variability for wind speed, ambient temperature, and

atmospheric stability could further reduce the estimated scaling factor of 0.6 closer towards the U.S. EPA recommended value of 0.4.

H.5 Intermittent Release

Support for the U.S. EPA screening factor is demonstrated for a continuous release (i.e., 24 hours per day) in the description above. It is important to be cautious when applying the U.S. EPA screening factors to an intermittent source for the purposes of estimating an annual average concentration (e.g., a business that may only emit during normal operating hours of 8 am to 6 pm).

Intermittent emissions, such as those from burning barrels, testing a standby diesel generator, or any normal business hour operation (e.g., 8am to 6pm Monday through Friday), could have the effect of eliminating some of the annual variability of meteorological conditions. For example, emissions only during the daytime could eliminate the variability of a drainage flow pattern in mountainous terrain. Guidance for estimating long-term averages for a screening approach and intermittent emissions is not available.

For a source located in the LAX meteorological domain, an emission pattern confined to the hours of 1400 to 1600 would eliminate any variability associated with the wind direction. In this case, estimating a 24-hour average with the U.S. EPA scaling factor of 0.4 would be incorrect.

In the event the emissions are intermittent but randomly distributed throughout the day, the scaling factor of 0.4 may be appropriate because the natural diurnal variability of meteorological conditions are concurrent with emissions. An additional pro-rating of the concentration, when estimating a 24-hour concentration, would be required to discount due to the intermittent nature of the emissions.

We recommend the following steps to estimate a screening based estimate of annual average concentrations from intermittent emissions.

1. Estimate the maximum one-hour concentration ($\chi_{1\text{-hr}}$) based on the AERSCREEN model approach (or similar, e.g., AERMOD with screening meteorological data) for possible meteorological conditions consistent with the operating conditions and the actual hourly emission rate. It is acceptable to estimate downwind concentrations using all meteorological combinations available to AERSCREEN. However, it is possible to be selective for the choices of meteorological conditions and still be conservative. For example, daytime only emissions need not be evaluated for nighttime stable atmospheric conditions.

2. Estimate the concentration for the longest averaging period applicable based on the length of time of the systematic or randomly distributed emissions and the factors in Table H.1. For example, the longest averaging period concentration that may be estimated with the U.S. EPA scaling factors is an 8-hour concentration ($\chi_{8\text{-hr}}$) for emissions that are systematically released for 12 hours.

Scaling factors between 8-hours and 12-hours are not available. In the case of the 8-hour concentration, the U.S. EPA screening factor of 0.7 $\pm$ 0.2 to estimate the maximum 8-hour concentration is appropriate.

The U.S. EPA Screening Guidance allows for deviation from the suggested conversion factor on a case-by-case basis. We recommend the lower end of the range for the conversion factor (i.e., 0.5 for the 8-hour average) when estimating an annual average concentration. This is because variability associated with seasonal differences in wind speed, wind direction, and atmospheric stability would not be addressed otherwise. As seen in Figure H.1, there are seasonal differences in the wind direction.

For example, if X is the length of time of systematic or randomly distributed emissions, the following scalars can apply.

- $X \leq 2$ hrs; Scalar = 1.0 to estimate a 1-hour average
- 3 hrs $\leq X \leq$ 7 hrs; Scalar = 0.8 to estimate a 3-hour average
- 8 hrs $\leq X \leq$ 20 hrs; Scalar = 0.5 to estimate an 8-hour average (the selection of 20 hours is arbitrary)
- 21 hrs $\leq X \leq$ 24 hrs; this may be a continuous release, use standard screening procedures.

3. Estimate the annual average concentration (χ_{annual}) by assuming the longer averaging period estimated above is persistent for the entire year. In the above example the 8-hour concentration is assumed to be persistent for an entire year to estimate an annual average concentration (i.e., the annual average concentration is assumed to be equal to the 8-hour concentration).

   In addition, the annual average concentration should be pro-rated over the final averaging period based on the pro-rated emissions (i.e., the calculation should include the fact that for some hours over the year, the emission rate is zero).

   For example, if Y is the number of operating hours in the year (e.g., Y = X * 365), the following may apply.

   $(\chi_{annual}) = (\chi_{1\text{-}hr})$ (Scalar) (Y/8760hrs/yr)

4. The hourly emission rate should be calculated based on the assumed operating schedule in the steps above. An example for a facility operating Y hours per year follows.

   $(q_{hourly}) = (Q_{yearly})/(Y$ hrs/yr)

5. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

   $GLC = (\chi_{annual})\ (q_{hourly})$

   $= (\chi_{1\text{-}hr})(Scalar)\ (Yhrs/8760hrs)\ (Q_{yearly})/(Y\ hrs/yr)$

   $= (\chi_{1\text{-}hr})(Scalar)\ (Q_{yearly})/(8760\ hrs/yr)$

Practically speaking, the above five steps condense down to determining three values. The first value is the maximum 1-hour concentration. The second value is the Scalar (either 1.0, 0.8, or 0.5). And the third value is the hourly emission rate estimated by emissions uniformly distributed over the entire year (8760 hours). The operating hours per year drops out of the calculations for an annual average concentration provided the emissions are based on an annual inventory (See step 5).

In the event that the acute averaging period is required and the emissions are based on an annual inventory, then the annual operating hours are required.

Below are four examples using the steps as outlined above. In each case, the annual average concentration is the desired value for use in risk assessment calculations.

<u>Example 1 - Fugitive Gasoline Station Emissions</u>

Emissions are **continuous** for 24 hours per day and 365 days per year.

1. Estimate the maximum 1-hour concentration with the AERSCREEN model (or similar screening modeling approach), $\chi_{1\text{-}hr}$.

2. Estimate the annual average concentration, $\chi_{annual,}$ with the U.S. EPA screening factor of 0.08.

3. $(\chi_{annual}) = (\chi_{1\text{-}hr})(0.08)$

4. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 24 hours per day and 365 days per year (8760 hours per year).

5. $(q_{hourly}) = (Q_{yearly})/(8760\ hrs/yr)$

6. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

   $GLC = (\chi_{annual})\ (q_{hourly})$

   $GLC = (\chi_{1\text{-}hr})(0.08)\ (Q_{yearly})/(8760\ hrs/yr)$

Example 2 - Dry Cleaner Emissions

Emissions are **intermittent** over the year but **systematic** for 10 hours per day, 5 days per week and 50 weeks per year.

1. Estimate the maximum 1-hour concentration with the AERSCREEN model (or similar screening modeling approach), $\chi_{1\text{-hr}}$.

2. Estimate the maximum 8-hour average concentration, $\chi_{8\text{-hr,}}$ with the U.S. EPA screening factor of 0.7 $\pm$0.2 as the longest averaging period of continuous release. The averaging period would need to be less than 10 hours. Use the lower range of the screening factor, 0.5, because the annual average is the final product and variability due to seasonal differences are not accounted for otherwise.

   $(\chi_{8\text{-hr}}) = (\chi_{1\text{-hr}})(0.5)$

3. Assume the worst-case 8-hour concentration is persistent throughout the year and pro-rate the concentration based on emissions over the year. For this dry cleaner, there are 2500 hours of operating condition emissions. Therefore the annual average is calculated as follows.

   $(\chi_{annual}) = (\chi_{8\text{-hr}})$ (2500hrs/8760hrs)

   $= (\chi_{1\text{-hr}})(0.5)$ (2500hrs/8760hrs)

4. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 2500 hours per year.

   $(q_{hourly}) = (Q_{yearly})/(2500$ hrs/yr)

5. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

   $GLC = (\chi_{annual})\ (q_{hourly})$

   $= (\chi_{1\text{-hr}})(0.5)$ (2500hrs/8760hrs) $(Q_{yearly})/(2500$ hrs/yr)

   $= (\chi_{1\text{-hr}})(0.5)\ (Q_{yearly})/(8760$ hrs/yr)

Example 3 - Burning Barrel Emissions

Emissions are **intermittent** over the year and **random** during daylight hours for two hours per burn, two burns per week, and 52 weeks per year.

1. Estimate the maximum 1-hour concentration with the AERSCREEN model (or similar screening modeling approach), $\chi_{1\text{-hr}}$. Meteorological combinations may be restricted to daytime conditions for this screening analysis.

2. Estimate the maximum 8-hour average concentration, $\chi_{8\text{-hr}}$, with the U.S. EPA screening factor of 0.7 ±0.2 as the longest averaging period where the emissions have the potential to be randomly distributed. Depending on the day of the year and latitude of the emissions, the daylight hours can vary. For this example, we assume the daylight hours can be as short as 10 hours per day to as long as 14 hours per day. Since the emissions are randomly distributed throughout the daylight hours, the longest averaging period we can scale with U.S. EPA scaling factors is a 10 hour average. In this case, the averaging period becomes the 8-hour average and the scaling factor becomes 0.7±0.2. Again since this is for an annual average, we use the lower end of the range, 0.5.

   $(\chi_{8\text{-hr}}) = (\chi_{1\text{-hr}})(0.5)$

3. Assume the worst-case 8-hour concentration is persistent throughout the year and pro-rate the concentration based on the emissions over the year. For the burning barrels there are 208 hours of operating condition emissions (208 hrs = (2hrs/burn)(2burns/wk)(52wk/yr)). Therefore the annual average concentration is calculated as follows.

   $(\chi_{annual}) = (\chi_{8\text{-hr}})$ (208hrs/8760hrs)

   $= (\chi_{1\text{-hr}})(0.5)$ (208hrs/8760hrs)

4. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 208 hours per year.

   $(q_{hourly}) = (Q_{yearly})/(208 \text{ hrs/yr})$

5. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

   $GLC = (\chi_{annual})\ (q_{hourly})$

   $= (\chi_{1\text{-hr}})(0.5)$ (208hrs/8760hrs) $(Q_{yearly})/(208 \text{ hrs/yr})$

   $= (\chi_{1\text{-hr}})(0.5)\ (Q_{yearly})/(8760 \text{ hrs/yr})$

The above methods were used in a recent modeling evaluation for emissions from a burning barrel (example 3 above) (ARB, 2002). Table H.2, below, shows results from the modeling evaluation. Shown in Table H.2 are the maximum annual average concentrations based on the screening approach outlined above as well as a refined approach with site specific meteorological data from four locations, Alturas, Bishop, San Benito, and Escondido. As seen in Table H.2, the screening evaluation as described in the example overestimates the values calculated based on the refined analysis. This is the desired outcome of a screening approach.

TABLE H.2: MAXIMUM ANNUAL AVERAGE CONCENTRATION (χ/Q) ABOVE AMBIENT CONDITIONS - BURNING BARREL EMISSIONS

Met. City	**Alturas**	**Bishop**	**San Benito**	**Escondido**	Screening
D (m)	**($\mu g/m^3$)/(g/s)**	**($\mu g/m^3$)/(g/s)**	**($\mu g/m^3$)/(g/s)**	**($\mu g/m^3$)/(g/s)**	***($\mu g/m^3$)/(g/s)***
20	44	61	85	110	*590*
50	12	16	22	30	*230*
100	4	5	7	9	*85*

Notes: (a) Annual χ/q is based on 208 hours of emissions at 1 g/s.

(b) χ/q is the concentration in $\mu g/m^3$ based on an hourly emission rate of 1 g/s.

Example 4 - Standby Diesel Engine Testing

Emissions are **intermittent** over the year and **systematic** for two hours per week and 50 weeks per year. The engine testing is conducted at 2 pm on Fridays.

1. Estimate the maximum 1-hour concentration with the AERSCREEN model (or similar screening modeling approach), $\chi_{1\text{-hr}}$. Meteorological combinations may be restricted to daytime conditions in this screening analysis because the engine test is conducted at 2 pm.

2. In this case, the emission schedule is systematically fixed over a two hour period. Therefore, the longest averaging period which is applicable for the U.S. EPA screening factors is one-hour because a two-hour conversion factor is not available. Therefore, we assume the maximum 1-hour concentration is persistent for the entire year. We still prorate the concentration based on the emissions. There are 100 hours of engine testing per year. Therefore the annual average concentration becomes.

   $(\chi_{annual}) = (\chi_{1\text{-hr}})$ (100hrs/8760hrs)

3. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 100 hours per year.

   $(q_{hourly}) = (Q_{yearly})/(100$ hrs/yr)

4. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

$$GLC = (\chi_{annual})\ (q_{hourly})$$

$$= (\chi_{1\text{-}hr})\ (100hrs/8760hrs)\ (Q_{yearly})/(100\ hrs/yr)$$

$$= (\chi_{1\text{-}hr})\ (Q_{yearly})/(8760\ hrs/yr)$$

H.6 Implementation

The approach outlined above has been implemented in the Hot Spots Analysis and Reporting Program (HARP). The HARP software has been developed in consultation with OEHHA, Air Resources Board (ARB), and District representatives. The HARP software is the recommended model for calculating and presenting HRA results for the Hot Spots Program. Information on obtaining the HARP software can be found on the ARB's web site at www.arb.ca.gov. Note, since the HARP software is a tool that uses the methods specified in this document, the software will be available after these guidelines have undergone public and peer review, been endorsed by the state's Scientific Review Panel (SRP) on Toxic Air Contaminants, and adopted by OEHHA.

H.7 References

ARB (1994). ARB memorandum dated 4/11/94 from A. Ranzieri to J. Brooks on the subject, "One-hour to Thirty-day Average Screening Factor."

ARB (2002). Staff Report: Initial Statement of Reasons for the Proposed Airborne Toxic Control Measure to Reduce Emissions of Toxic Air Contaminants from Outdoor Residential Waste Burning, January 2002. California Air Resources Board.

U.S. EPA (1992). Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised, October 1992, EPA-454/R-92-019. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2011). AERSCREEN User's Guide. EPA-454/B-11-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Appendix I:

Calculation Examples for Estimating Potential Health Impacts

This appendix provides four example calculations to illustrate the procedures for estimating the potential health impacts from a facility. The examples are intended to assist the risk assessor in understanding the steps associated with conducting the final step of risk assessment, risk characterization. The four examples provided in this appendix evaluate the inhalation cancer risk, the noncancer acute hazard quotient (HQ) and hazard index (HI), the noncancer 8-hour HQ and HI, and the multipathway (inhalation and oral) noncancer chronic HQ and HI. Specific requirements for health risk assessment (HRA) under the Hot Spots Program are presented in Chapter 8. The HARP software will perform the calculations that are presented here and required in the guidelines. See the ARB's website at www.arb.ca.gov for more information on HARP.

I.1 Sample Calculation for Inhalation Cancer Health Risk Assessment

The following example illustrates the steps for calculating cancer risk at the maximum exposed individual resident (MEIR) using the high-end point-estimate for the inhalation exposure pathway. For each included substance, the steps involved in this sample calculation include:

- Determine the annual average concentration and look-up the inhalation cancer potency factor for each substance
- For each age range, calculate the inhalation dose
- For each age range, calculate the cancer risk using the OEHHA cancer potency factor and the appropriate Age Sensitivity Factor
- Sum the cancer risks from each age range for the exposure duration of interest, and express the risk in chances per million

In this example, the inhalation dose and risk are calculated for the third trimester, ages 0<2, 2<9, 2<16, 16<30, and 16-70 using the high-end daily breathing rate for each age range.

This example focuses on the 30-year cancer risk calculation and does not cover the steps for completing a noninhalation or multipathway HRA. Algorithms to estimate point-estimate and stochastic multipathway exposure can be found in Chapter 5. For simplicity, it is recommended that the risk assessor use HARP to conduct a multipathway risk assessment or stochastic risk assessment.

Step one - Determine the annual average concentration at the MEIR and look-up the inhalation cancer potency factor for each emitted substance.

The risk assessor would obtain the annual average concentrations from the air dispersion modeling results. This step has been completed for this example. Table I.1 presents the annual average concentrations at a hypothetical facility. In addition,

Table I.1 also presents inhalation cancer potency factors for each substance, which also can be found in Chapter 7 and Appendix L. Note that where no inhalation cancer potency has been developed for a substance, the tables in this example have been annotated with N/A since it will not be possible to conduct a quantitative risk assessment for these substances. As previously stated, this example does not take into account multipathway effects for the substances listed in Table I.1. It is recommended that the risk assessor use HARP for conducting such an analysis.

TABLE I.1 ANNUAL AVERAGE CONCENTRATIONS AT THE MEIR AND INHALATION CANCER POTENCY FACTORS

Substance	Annual Average Concentrations ($\mu g/m^3$)	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$
Ammonia	160	N/A
Arsenic	0.0015	12
Benzene	5	0.10
Chlorine	0.08	N/A
Chlorobenzene	20	N/A
2,3,7,8-TCDD (dioxin)	0.000004	130,000
Nickel	0.02	0.91

Step two - Determine the inhalation dose for each substance.

Once you have determined the annual average concentration for the emitted substance, the equation below is used to calculate the inhalation dose for each age range and each substance. This equation is listed in Section 5.4.1 of this document, and is also described in the *Air Toxics Hot Spots Risk Assessment Guidelines: Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012)

$$Dose-air = (Cair)\left(\frac{BR}{BW}\right)(A)(EF)\left(1\times10^{-6}\right)$$

Where:

Dose-air	=	Dose through inhalation (mg/kg/d)
Cair	=	Concentration in air ($\mu g/m^3$)
BR/BW	=	Daily breathing rate normalized to body weight (L/kg BW-day). See Table I.2 for the daily breathing rate for each age range.
A	=	Inhalation absorption factor
EF	=	Exposure frequency (unitless, days/365 days)
$1x10^{-6}$	=	Milligrams to micrograms conversion (10^{-3} mg/μg), cubic meters to liters conversion (10^{-3} m^3/l)

A summary of the exposure point-estimates and data distributions for use in risk assessment can be found in Chapter 5 of this document. For more detail on point-estimates and data distributions, see OEHHA (2012). The recommended default values presented in Table I.2 can be used when site-specific information is not available. Note that in this example the mean daily breathing rates listed in the table are for information purposes only. In some cases, the mean value can be used for a Tier-1 risk assessment or applied when multiple noninhalation routes of exposure dominate the risk (See Chapter 8.2.6).

TABLE I.2 RECOMMENDED DEFAULT VALUES FOR INHALATION DOSE EQUATION

Variable	Recommended Default Value		
EF	0.96 (350 days/365 days) Assumes 2-week vacation away from exposure		
Daily Breathing Rates (DBR) (L/kg BW-day) for 9, 30, and 70-year exposures in examples below	**Period**	**Mean**	**95th percentile**
	3rd trimester	225	**361**
	0<2 yrs	658	**1090**
	2<9 yrs	535	**861**
	2<16 yrs	452	**745**
	16<30 yrs	210	**335**
	16-70 yrs	185	**290**
	(For other DBRs see Chapter 5)		
A	1[a]		

[a] OEHHA's Hot Spots inhalation cancer potency factors for the Hot Spots program have already been adjusted where necessary to allow for the inhalation absorption factor, so a value of 1 is used in this equation.

The following equation shows the calculation for the inhalation dose of arsenic for the third trimester by using the annual average concentration for arsenic (Table I.1) and the recommended default values in Table I.2. Note that the high-end (95th percentile) daily breathing rates are used to estimate the third trimester inhalation dose in this example.

$$\text{Arsenic}(\text{dose}-\text{air})_{(\text{third trimester})} = \left(\frac{0.0015\mu g}{m^3}\right)\left(\frac{361 liters}{kg-day}\right)(1)(0.96)\left(\frac{1\text{x}10^{-3}\ mg}{1\mu g}\right)\left(\frac{1\text{x}10^{-3}\ m^3}{liters}\right)$$

$$\text{Arsenic}(\text{dose}-\text{air})_{(\text{third trimester})} = 5.2\times10^{-7} mg/kg-day$$

To estimate the 30-year arsenic inhalation dose, this calculation is repeated using 95th percentile breathing rates for 0<2 years, 2<16 years, and 16<30 years shown in Table I.2:

$$\text{Arsenic}(\text{dose}-\text{air})_{(0<2yrs)} = \left(\frac{0.0015\mu g}{m^3}\right)\left(\frac{1090 liters}{kg-day}\right)(1)(0.96)\left(\frac{1\text{x}10^{-3}\ mg}{1\mu g}\right)\left(\frac{1\text{x}10^{-3}\ m^3}{liters}\right)$$

Arsenic (dose-air) $_{(0<2\ \text{yrs})}$ = 1.6 × 10^{-6} mg/kg-day

$$\text{Arsenic}(\text{dose}-\text{air})_{(2<16yrs)} = \left(\frac{0.0015\mu g}{m^3}\right)\left(\frac{745 liters}{kg-day}\right)(1)(0.96)\left(\frac{1\text{x}10^{-3}\ mg}{1\mu g}\right)\left(\frac{1\text{x}10^{-3}\ m^3}{liters}\right)$$

Arsenic (dose-air) $_{(2<16\ \text{yrs})}$ = 1.1 × 10^{-6} mg/kg-day

$$\text{Arsenic}(\text{dose}-\text{air})_{(16<30yrs)} = \left(\frac{0.0015\mu g}{m^3}\right)\left(\frac{335 liters}{kg-day}\right)(1)(0.96)\left(\frac{1\text{x}10^{-3}\ mg}{1\mu g}\right)\left(\frac{1\text{x}10^{-3}\ m^3}{liters}\right)$$

Arsenic (dose-air) $_{(16<30\ \text{yrs})}$ = 4.8 × 10^{-7} mg/kg-day

To estimate 70-year exposure, the 95th percentile breathing rate for ages16-70 years in Table I.2 is used instead of the breathing rate for 16<30 years. The arsenic dose for the 16<70 year age bin is 4.2 × 10^{-7} mg/kg-day. Therefore, the age bins that are used for the 70-year scenario include the third trimester, ages 0<2 years, 2<16 years, and 16<70 years.

To estimate the 9-year arsenic inhalation dose, the 95th percentile breathing rates are used for the third trimester, ages 0<2 years and 2<9 years.

These calculations are repeated for each substance under evaluation using their respective annual average concentrations. For our hypothetical facility, we have calculated each inhalation dose for each substance by age bin. In reality, you only need to calculate the dose for the age bins that are required for the exposure duration of interest for your assessment (e.g., 30 years, etc.). However, Table I.3 shows the results from our analysis for all age bins so that potential risk for any exposure duration can be calculated.

TABLE I.3 CALCULATED INHALATION DOSES FOR SUBSTANCES

Substance		Calculated Dose (mg/kg-day)*				
	3rd Tri.	0<2 yrs	2<9 yrs	2<16 yrs	16<30 yrs	16-70 yrs
Ammonia	N/A	N/A	N/A	N/A	N/A	N/A
Arsenic	5.2×10^{-7}	1.6×10^{-6}	1.2×10^{-6}	1.1×10^{-6}	4.8×10^{-7}	4.2×10^{-7}
Benzene	1.7×10^{-3}	5.2×10^{-3}	4.1×10^{-3}	3.6×10^{-3}	1.6×10^{-3}	1.4×10^{-3}
Chlorine	N/A	N/A	N/A	N/A	N/A	N/A
Chlorobenzene	N/A	N/A	N/A	N/A	N/A	N/A
2,3,7,8-TCDD (dioxin)	1.4×10^{-9}	4.2×10^{-9}	3.3×10^{-9}	2.9×10^{-9}	1.3×10^{-9}	1.1×10^{-9}
Nickel	6.9×10^{-6}	2.1×10^{-5}	1.7×10^{-5}	1.4×10^{-5}	6.4×10^{-6}	5.6×10^{-6}

* The doses shown in this table are rounded and the rounded numbers are used for risk calculations in this example.

Step three – Determine inhalation cancer risk for the MEIR.

Once you have calculated the inhalation dose, then the cancer risk is calculated for each age bin and the risk estimates are summed for the exposure duration of interest. To complete this step, the dose for each age bin is multiplied by the inhalation cancer potency factor, the Age Sensitivity Factor (ASF), the exposure duration for the specified age bin over the averaging time (i.e., AT is always 70 years), and the fraction of time spent at home, to determine the cancer risk. Studies have shown that infants and children are more sensitive than adults to exposure to many carcinogens (OEHHA, 2009). Therefore, OEHHA applied ASFs to take into account the increased sensitivity to carcinogens during early-in-life exposure. OEHHA and ARB also evaluated information from activity patterns databases to estimate the percentage of the day that people are home (OEHHA, 2012). This information can be used to adjust exposure duration and risk from a specific facility's emissions, based on the assumption that exposure to the facility's emissions are not occurring while the person is away from home. The ASF and FAH variables are only used when estimating residential cancer risk (e.g., the MEIR)

The risk calculation is performed for each age bin and each substance. The total cancer risk for each substance is the sum of the cancer risks from each age bin for the exposure duration of interest.

$$\text{Cancer Risk} = \left(\text{Inhalation Dose}\,\frac{mg}{kg-day}\right)\left(\text{Cancer Potency}\,\frac{kg-day}{mg}\right)(ASF)(FAH)\left(\frac{EDyrs}{ATyrs}\right)$$

Where:

Cancer Risk	=	Unitless expression of risk (see below)
Inhalation Dose	=	In mg/kg-d
Cancer Potency	=	Chemical specific (in $(mg/kg\text{-}d)^{-1}$ or as kg-d/mg)
ASF	=	Age sensitivity factor (unitless)
FAH	=	Fraction of time spent at home (unitless)
ED	=	Exposure duration (years)
AT	=	Averaging time period over which exposure duration is averaged (always 70 years).

The age sensitivity factor, exposure duration, and fraction of time spent at home are shown for each age range in Table I.4. However, if it is determined there is a school located within the cancer risk isopleths of $1x10^{-6}$ (one chance per million) or greater for the duration of interest (e.g., 30-year analysis), then the fraction of time at the residence is assumed to be one (1) for ages 3rd trimester to less than 16. Thus, cancer risks and the associated isopleths must first be determined using one (1) as the fraction of time at the residence before the FAH values between ages 3rd trimester to less than 16 in Table I.4 can be utilized. See Chapter 8 for more information on calculating a zone of impact, isopleths, and population exposure. In this example, we assume there is no school located within the cancer risk isopleth of $1x10^{-6}$ or greater; therefore, the FAH factors in Table I.4 are used.

TABLE I.4 INPUTS FOR AGE SENSITIVITY FACTOR, EXPOSURE DURATION, AND THE FRACTION OF TIME SPENT AT HOME

	3rd Tri.	0<2 yrs	2<9 yrs	2<16 yrs	16<30 yrs	16-70 yrs
Age Sensitivity Factor	10	10	3	3	1	1
Exposure Duration (years)	0.25	2	7	14	14	54
Fraction of Time Spent at Home (FAH)	0.85*	0.85*	0.72*	0.72*	0.73	0.73

* FAH is 1 for ages 3rd trimester to less than 16 unless it is determined there is no school located within the cancer risk isopleth of $1x10^{-6}$ or greater.

The equation below shows the calculation of the cancer risk from arsenic for the third trimester:

$$\text{Arsenic Cancer Risk}_{\text{(third trimester)}} = \left(\frac{5.2\times10^{-7}mg}{kg-d}\right)\left(\frac{12kg-d}{mg}\right)(10)(0.85)\left(\frac{0.25yrs}{70yrs}\right)$$

$$\text{Arsenic Cancer Risk}_{\text{(third trimester)}} = 1.9 \times 10^{-7}$$

To estimate the 30-year cancer risk from arsenic, the cancer risk calculation is repeated for the age bins 0<2, 2<16, and 16<30 years using the appropriate age-related inputs for average daily inhalation dose, ASF, FAH and ED in Tables I.3 and I.4, respectively:

$$\text{Arsenic Cancer Risk}_{(0<2\,\text{yrs})} = \left(\frac{1.6\times10^{-6}\,mg}{kg-d}\right)\left(\frac{12\,kg-d}{mg}\right)(10)(0.85)\left(\frac{2\,yrs}{70\,yrs}\right)$$

Arsenic Cancer Risk $_{(0<2\ \text{yrs})}$ = 4.7×10^{-6}

$$\text{Arsenic Cancer Risk}_{(2<16\,\text{yrs})} = \left(\frac{1.1\times10^{-6}\,mg}{kg-d}\right)\left(\frac{12\,kg-d}{mg}\right)(3)(0.72)\left(\frac{14\,yrs}{70\,yrs}\right)$$

Arsenic Cancer Risk $_{(2<16\ \text{yrs})}$ = 5.7×10^{-6}

$$\text{Arsenic Cancer Risk}_{(16<30\,\text{yrs})} = \left(\frac{4.8\times10^{-7}\,mg}{kg-d}\right)\left(\frac{12\,kg-d}{mg}\right)(1)(0.73)\left(\frac{14\,yrs}{70\,yrs}\right)$$

Arsenic Cancer Risk $_{(16<30\ \text{yrs})}$ = 8.4×10^{-7}

Calculated arsenic cancer risks for each age range are then summed together as shown in the example below to estimate the total 30-year cancer risk from arsenic:

Total Arsenic Cancer Risk $_{(30\text{-year})}$ = Arsenic Cancer Risk $_{(\text{third trimester})}$ +

Arsenic Cancer Risk $_{(0<2\ \text{yrs})}$ +

Arsenic Cancer Risk $_{(2<16\ \text{yrs})}$ +

Arsenic Cancer Risk $_{(16<30\ \text{yrs})}$

$= (1.9 \times 10^{-7}) + (4.7 \times 10^{-6}) + (5.7 \times 10^{-6}) + (8.4 \times 10^{-7}) = 1.1 \times 10^{-5}$

To estimate the 70-year cancer risk from arsenic, the cancer risk calculation is repeated for the last trimester to birth, ages 0<2, 2<16, and 16<70 using the appropriate age-related inputs from Tables I.3 and I.4. The calculated arsenic cancer risks for each

age range from the third trimester to age 70 are then summed together to estimate the total 70-year cancer risk from arsenic.

To estimate the 9-year cancer risk from arsenic, the cancer risk calculation is repeated for the last trimester to birth, ages 0<2 and 2<9 using the appropriate age-related inputs from Tables I.3 and I.4. The calculated arsenic cancer risks for each age range from the third trimester to age 9 are then summed together to estimate the 9-year cancer risk from arsenic.

Step four – Express cancer risk in chances per million.

The final step converts the cancer risk in scientific notation to a whole number that expresses the cancer risk in "chances per million"; to complete this step, multiply the estimated cancer risk by a factor of $1x10^6$ (i.e., 1 million).

(Total Cancer Risk) ($1x10^6$) = Total Cancer Risk in chances per million

For a hypothetical facility, the equation below shows the calculation for the inhalation cancer risk of arsenic as a result of 30-year exposure to arsenic:

$$\left(1.1\times10^{-5}\right)\left(1\times10^{6}\right)=11\,\text{chances per million}$$

Use the substance-specific inhalation dose and inhalation cancer potency factor to determine the cancer risk for each substance by repeating these steps. Sum the individual substance cancer risks to give you the total facility (inhalation) cancer risk. Table I.5 shows the individual substance and total facility inhalation cancer risk. In this example, a hypothetical facility poses a (inhalation) cancer risk of 658 chances per million at the MEIR. Note, although not presented here, a facility emitting arsenic or dioxins should also evaluate cancer risk from noninhalation exposure pathways. *Note that although rounding was utilized for ease throughout this example, rounding should not take place until the final answer.*

TABLE I.5 HYPOTHETICAL FACILITY INHALATION 30-YEAR CANCER RISK

Substance	Cancer risk* (chances per million)
Ammonia	N/A*
Arsenic	11
Benzene	310
Chlorine	N/A**
Chlorobenzene	N/A**
2,3,7,8-TCDD (dioxin)	326
Nickel	11
Total Facility Inhalation Cancer Risk	**658**

* The calculated numbers in each step are rounded and the rounded numbers were used in succeeding calculation steps in this example.

** N/A: Inhalation cancer potency factor is not applicable.

While this example illustrates the steps used to calculate cancer risk using the inhalation dose algorithm, steps one through four can also be used to calculate noninhalation cancer risk and ultimately multipathway (inhalation and noninhalation pathway) cancer risk. To determine noninhalation cancer risk, an assessor should use the appropriate exposure pathway algorithm presented in Chapter 5. For example, equation 5.4.3.1.1 (Chapter 5) would be used to determine dose for the soil ingestion pathway. Once the assessor has determined the ingestion dose by age group, the cancer risk for that pathway is calculated using the substance-specific oral slope factor and the appropriate age-sensitivity factors. Oral slope factors can be found in Appendix L and Chapter 7. To calculate multipathway cancer risk, the cancer risks for all substances and exposure pathways are summed. See Chapter 8 for further discussion.

I.2 Sample Calculation of Noncancer Acute Hazard Indices

Risk characterization for noncancer health impacts are expressed as a hazard quotient (for individual substances) or a hazard index (for multiple substances). In addition, all hazard quotients (HQ) and hazard indices (HI) must be determined by target organ system. The example below illustrates the approach for calculating a noncancer acute HQ and HI at the MEIR. The steps involved in this sample calculation include: 1) determining the 1-hour maximum concentration, the acute reference exposure level (REL), and the target organ systems for each substance; 2) calculating the acute HQ for each substance and applying the calculated HQ to the specified target organs for each substance; and 3) calculating the acute HI by summing each HQ from each substance by target organ system. As discussed in Chapter 8, the following example is provided to assist the risk assessor in understanding how to calculate an acute HQ and HI. Using HARP, both the acute HQ and HI will automatically be calculated at each receptor. No exposure duration adjustment should be made for acute noncancer assessments. Specific requirements for risk assessment under the Hot Spots Program can be found in Chapters 8 and 9.

Step one - Determine the 1-hour maximum concentration at the MEIR, the acute reference exposure level, and the target organ systems for each emitted substance.

The risk assessor would obtain the 1-hour maximum concentrations from the air dispersion modeling results. This step has been completed for this example. Table I.6 presents the maximum 1-hour concentrations, target organ systems, and acute RELs for seven substances. Note that where an acute REL has not been developed for a substance, the tables in this example have been annotated with "N/A".

TABLE I.6 CONCENTRATIONS, ACUTE RELS, AND TARGET ORGAN SYSTEM(S) FOR SUBSTANCES AT THE MEIR

Substance	1-hour Maximum Concentration ($\mu g/m^3$)	Acute REL ($\mu g/m^3$)	Target Organ System(s)
Ammonia	1900	3200	Respiratory System; Eye
Arsenic	0.03	0.20	Reproductive/Development ; Cardiovascular System; Nervous System
Benzene	0.54	27	Reproductive/Development; Immune System; Hematologic System
Chlorine	140	210	Respiratory System; Eye
Chlorobenzene	60	N/A	N/A
2,3,7,8-TCDD (dioxin)	0.00001	N/A	N/A
Nickel	0.08	0.20	Immune System

In this example, chlorobenzene and 2,3,7,8-TCDD (dioxin) do not have acute REL values. The acute RELs and their corresponding target organ system(s) can be found in Table 6.1 (Chapter 6) and also in Appendix L.

Step two - Determine the hazard quotient for each substance.

The hazard quotients for each substance are calculated by taking the acute maximum 1-hour concentration and dividing by the substance-specific acute REL. The following equation shows how to calculate the hazard quotient for ammonia.

$$\text{Acute Hazard Quotient} = \frac{\left(\text{Maximum 1-hr Concentration}\right)}{\left(\text{Acute REL}\right)} \Rightarrow \text{Acute Hazard Quotient}_{(\text{ammonia})} = \frac{\left(1900\ \mu g/m^3\right)}{\left(3200\ \mu g/m^3\right)} = 0.6$$

Step three – Determine the acute HI for all emitted substances.

The acute HQ calculated above for a substance applies to all the target organs listed under that substance. The acute HI is calculated by summing each hazard quotient for each substance by target organ system. For example, add the HQs for all substances that impact the respiratory system, then repeat this step for the next target organ system (e.g., reproductive/development system). This step is repeated until all target organs (for the substances emitted) are individually totaled. See Table 6.1 for target organ system information. Note, do not add together the HQs or HIs for different target organ systems (e.g., do not add the impacts for the respiratory system to that for reproductive/development). Table I.7 shows individual hazard quotients for each substance and total hazard index.

TABLE I.7 INDIVIDUAL HAZARD QUOTIENTS AND TOTAL HAZARD INDEX FOR ACUTE EXPOSURE

Substance	Immune System	Reproductive/ Development	Hematologic System	Nervous System	Cardiovascular System	Respiratory System	Eye
Ammonia						0.6	0.6
Arsenic		0.2		0.2	0.2		
Benzene	0.02	0.02	0.02				
Chlorine						0.7	0.7
Chlorobenzene							
2,3,7,8-TCDD (dioxin)							
Nickel	0.4						
Total Acute Hazard Index*	**0.42**	**0.22**	**0.02**	**0.2**	**0.2**	**1.3**	**1.3**

* The total hazard index is the sum of the rounded individual hazard quotients for each target organ

In this example, an HQ of one was not equaled or exceeded for any individual substance. However, an HI (the sum of the hazard quotients for each target organ) of one was exceeded for the respiratory system and eyes. Exceeding a hazard index of one may indicate that there is the potential for adverse acute health impacts at this receptor location. The District and OEHHA should be consulted when a hazard index exceeds one (see Section 8.3).

I.3 Sample Calculation of Noncancer 8-Hour Hazard Indices

The 8-hour RELs are used to evaluate impacts to offsite workers. The 8-hour RELs also apply to exposure of children and teachers during school hours. Although not required in the HRA, 8-hour exposure modeling could also be performed at the discretion of the District to a residential scenario (i.e., the MEIR) where a facility operates only a portion of the day and exposure to residences are not adequately reflected by averaging concentrations over a 24 hour day.

The example below illustrates the approach for calculating a noncancer 8-hour HQ and HI at the maximum exposed individual worker (MEIW) from a noncontinuously emitting facility. An HQ expresses the noncancer 8-hour health impacts for an individual substance and an HI expresses the cumulative potential impacts for multiple substances. All HQs and hazard indices HIs must be determined by target organ system.

The steps involved in this sample calculation include: 1) estimating the daily 8-hour annual average concentration, determining the 8-hour reference exposure level (REL) and the target organ systems for each substance; 2) calculating the 8-hour HQ for each substance and applying the calculated HQ to the specified target organs for each substance; and 3) calculating the 8-hour HI by summing each HQ from each substance by target organ system. As discussed in Chapter 8, the following example is provided to assist the risk assessor in understanding the calculation of noncancer 8-hour HQ and HI. Using the HARP software, both the 8-hour HQ and HI will be automatically calculated at each receptor. Specific requirements for risk assessment under the Hot Spots Program can be found in Chapters 8 and 9.

In this example, the facility emits for a typical schedule of eight hours per day and five days per week and the offsite worker's shift coincides with the facility's emission schedule.

Step one – Estimate the daily 8-hour annual average concentrations at the MEIW from the annual average using an adjustment factor; determine the 8-hour REL and target organ systems for each emitted substance

The risk assessor would obtain the annual average concentrations from the air dispersion modeling results. See Chapter 4 or Appendix M for information on modeling and approximating 8-hour exposure concentrations. This example uses an adjustment factor to approximate the concentration the worker is breathing. Since this is a noncontinuously emitting facility, the annual average concentration is adjusted to represent daily 8-hour average concentration. These steps have been completed for this example. However for completeness, the following equation shows how to calculate the adjustment factor. See Chapter 4 for more explanation.

$$WAF = \frac{H_{residential}}{H_{source}} \times \frac{D_{residential}}{D_{source}} = \frac{24}{8} \times \frac{7}{5} = 4.2$$

Where:

WAF = the worker adjustment factor
$H_{residential}$ = the number of hours per day the long-term residential concentration is based on (always 24 hours)
H_{source} = the number of hours the source operates per day. In this example, we are assuming 8 hours per day.
$D_{residential}$ = the number of days per week the long-term residential concentration is based on (always 7 days).
D_{source} = the number of days the source operates per week. In this example, we are assuming 5 days per week.

The daily 8-hour annual average inhalation concentration is then estimated by multiplying the WAF with the annual average concentration:

$$\text{Concentration}_{\text{(8-hour average)}} = \text{Concentration}_{\text{(annual average)}} \times (\text{WAF})$$

Table I.8 shows the daily 8-hour annual average inhalation concentrations at a MEIW, target organ systems, and 8-hour RELs for seven substances. Note that where an 8-hour REL has not been developed for a substance, the tables in this example have been annotated with "N/A"; therefore, Table I.8 lists 8-hour RELs for arsenic and nickel. The 8-hour RELs and their corresponding target organ system(s) can be found in Table 6.2 (Chapter 6) and also in Appendix L.

TABLE I.8 ANNUAL AVERAGE CONCENTRATIONS AND ADJUSTED (AVERAGE DAILY) 8-HOUR CONCENTRATIONS FOR A FACILITY OPERATING 8 HRS/DAY, 5 DAYS/WEEK, AND THE 8-HOUR RELS AND TARGET ORGAN SYSTEM(S) FOR SUBSTANCES

Substance	Annual Average Conc. ($\mu g/m^3$)	Adjusted (Average Daily) 8-Hour Conc. ($\mu g/m^3$)	8-Hour REL ($\mu g/m^3$)	Target Organ System(s)
Ammonia	16	67	N/A	N/A
Arsenic	0.0015	0.0063	0.015	Cardiovascular System; Reproductive/Development; Nervous System: Respiratory System; Skin
Benzene	0.05	0.21	3	Hematologic system
Chlorine	0.08	0.3	N/A	N/A
Chlorobenzene	20	84	N/A	N/A
2,3,7,8-TCDD (dioxin)	0.000001	0.000004	N/A	N/A
Nickel	0.01	0.04	0.06	Respiratory System; Immune System

Step two - Determine the 8-hour hazard quotient for each substance.

Similar to the acute hazard quotient (HQ) calculation shown above, the 8-hour HQs are calculated by taking the noncontinuous source concentration, assumed to be based on an 8-hour facility operation from Table I.8, and dividing by the substance-specific 8-hour REL. The following equation shows how to calculate the hazard quotient for arsenic using the values presented in Table I.8:

$$\text{8-Hour Hazard Quotient} = \frac{\left(\text{Average daily concentration}\right)}{\left(\text{8-Hour REL}\right)} \Rightarrow \text{8-Hour Hazard Quotient}_{(\text{arsenic})} = \frac{\left(0.0063\ \mu g/m^3\right)}{\left(0.015\ \mu g/m^3\right)} = 0.4$$

Step three – Determine the HI for all emitted substances.

The 8-hour HQ calculated above for a substance applies to all the target organs listed under that substance. The 8-hour HIs are calculated by summing each HQ for each substance by target organ system. Similar to the example calculation for the acute HI, add the HQs for all substances that impact a specific organ system, then repeat this step for the next target organ system (e.g., respiratory system). This step is repeated until all target organs (for the substances emitted) are individually totaled. See Table 6.2 for target organ system information for 8-hour RELs. Note: do not add together the HQs or HIs for different target organ systems (e.g., do not add the impacts for the respiratory system to the reproductive/developmental system). Table I.9 shows individual hazard quotients for each substance and total hazard index for each organ system using the information presented in Table I.8.

In this example, an HQ of one was not equaled or exceeded for any individual substance. However, an HI (the sum of the hazard quotients for each target organ) of one was exceeded for the respiratory system. Exceeding a hazard index of one may indicate that there is the potential for an adverse health impact at this receptor location with repeated daily 8-hour exposures. The District and OEHHA should be consulted when a hazard index exceeds one (see Section 8.3).

For the MEIW, ideally only an 8-hour noncancer hazard assessment is required. However, development of 8-hour RELs is an ongoing process and many substances that have chronic RELs do not yet have 8-hour RELs. If 8-hour RELs have not been developed yet for all of the emitted chemicals with a chronic REL, as in the example below, then a chronic noncancer hazard assessment is also performed.

TABLE I.9 INDIVIDUAL HAZARD QUOTIENTS AND TOTAL HAZARD INDEX FOR 8-HOUR EXPOSURE

Substance	Respiratory System	Reproductive/ Development	Nervous System	Cardiovascular System	Skin	Immune System	Hematologic
Ammonia							
Arsenic	0.4	0.4	0.4	0.4	0.4		
Benzene							0.07
Chlorine							
Chloro-benzene							
2,3,7,8-TCDD (dioxin)							
Nickel	0.7					0.7	
Total Hazard Index*	**1.1**	**0.4**	**0.4**	**0.4**	**0.4**	**0.7**	**0.07**

* The total hazard index is the sum of the rounded individual hazard quotients for each target organ

I.4 Sample Calculation of Noncancer Chronic Hazard Indices

The example below illustrates the approach for calculating a noncancer chronic HQ and HI at the MEIR. An HQ expresses the noncancer health impacts for an individual substance and an HI expresses the potential impacts for multiple substances. All hazard quotients (HQ) and hazard indices (HI) must be determined by target organ system. The steps involved in this sample calculation include: 1) determining the annual average concentration, the inhalation and oral chronic RELs, and the target organ systems for each substance; 2) calculating the inhalation chronic HQ for each substance and applying the calculated HQ to all target organs for each substance; 3) calculating the noninhalation chronic HQ for each substance and applying the calculated HQ to all target organs for each substance; and 4) calculating the chronic HI by summing each HQ (inhalation and noninhalation) from each substance by target organ system.

As discussed in Chapter 8, the following example is provided to assist the risk assessor in understanding the calculation of a chronic HQ and HI. Using the HARP software, both the chronic HQ and HI will be automatically calculated at each receptor. No exposure adjustments are applied to chronic noncancer assessments. Specific requirements for risk assessment under the Hot Spots Program can be found in Chapters 4, 8 and 9.

Step one - Determine the annual average concentrations at the MEIR and inhalation and oral chronic RELs for each emitted substance.

The risk assessor would obtain the substance-specific annual average concentrations from the air dispersion modeling results. This step has been completed for this example. Table I.10 presents the annual average concentrations, target organ systems, and chronic RELs for seven substances. All of the substances have a chronic REL value associated with them. In addition, arsenic, dioxins, and nickel are multipathway substances; therefore, oral and dermal exposure must be included as potential exposure pathways. The chronic RELs and their corresponding target organ system(s) can be found in Tables 6.3 and 6.4 (Chapter 6) and also in Appendix L.

Step two – Determine the inhalation chronic hazard quotient for each substance.

For inhalation exposure, the individual hazard quotients for each substance are calculated by taking the annual average concentration and dividing by its corresponding chronic inhalation REL. Using the information contained in Table I.10, the equation below is used to calculate the inhalation hazard quotient for arsenic.

$$\text{Chronic Hazard Quotient} = \frac{\left(\begin{matrix}\text{Annual Avg.}\\ \text{Concentration}\end{matrix}\right)}{\left(\begin{matrix}\text{Chronic}\\ \text{REL}\end{matrix}\right)} \quad \Rightarrow \quad \text{Chronic Hazard Quotient}_{(\text{arsenic})} = \frac{\left(0.0015\ \mu g/m^3\right)}{\left(0.015\ \mu g/m^3\right)} = 0.1$$

The inhalation chronic HQ calculated above for a substance applies to all the target organs listed under that substance.

TABLE I.10 ANNUAL AVERAGE CONCENTRATIONS, CHRONIC RELS, AND TARGET ORGAN SYSTEMS FOR SUBSTANCES AT THE MEIR.

Substance	Annual Average Conc. ($\mu g/m^3$)	Chronic Inhalation REL ($\mu g/m^3$)	Target Organ System(s) (inhalation)	Chronic Oral REL (mg/kg-day)	Target Organ System(s) (oral/dermal)
Ammonia	160	200	Respiratory System	-	-
Arsenic	0.0015	0.015	Reproductive/ Development; Cardiovascular System; Nervous System; Respiratory System; Skin	0.0000035	Reproductive/ Development; Cardiovascular system; Nervous System; Respiratory System; Skin
Benzene	0.05	3	Hematologic System	-	-
Chlorine	0.08	0.2	Respiratory System	-	-
Chloro-benzene	20	1000	Alimentary System; Kidney; Reproductive/ Development	-	-
2,3,7,8-TCDD (dioxin)	0.000004	0.00004	Alimentary System (Liver); Reproductive/ Development; Endocrine System; Respiratory System; Hematologic System	0.00000001	Alimentary System (Liver); Reproductive/ Development; Endocrine System; Respiratory System; Hematologic System
Nickel	0.003	0.014	Respiratory System; Hematologic System	0.011	Reproductive/ Development

Step three – Determine the noninhalation hazard quotient for each substance.

For the substances that are subject to deposition, noninhalation (i.e., oral and dermal) exposure pathways need to be considered in the chronic hazard quotient evaluation. The point-estimates and algorithms for calculating the oral dose for all of the applicable exposure pathways and receptors (e.g., workers or residents) are explained in Chapter 5. Note, the HARP software uses the appropriate information and performs all the steps discussed in these examples.

As discussed in Section 8.3.3 for noncancer multipathway assessments, Tier I of the tiered approach to risk assessment states that the high-end point-estimates are used for the two dominant noninhalation exposure pathways and the non-dominant exposure pathways use the mean point-estimates to determine the dose and chronic health impacts at a residential receptor. To determine which exposure pathways are the two dominant ones, high-end point-estimates are used for all applicable noninhalation exposure pathways to see which two pathways provide the highest impacts for each substance. Once the two dominant noninhalation pathways are determined for each substance, the doses for the remaining noninhalation exposure pathway for that substance are recalculated using the average point-estimates. The 70-year dose (i.e., from calculating and adding the exposure contributions from the age 0 through 70 year bins) is used for residential receptors and the dose from the 16 through 70 year age bin is used for the worker evaluation. See Chapters 1, 4, 5, 6, and 8 for more information.

This example shows how to combine the impacts from multiple exposure pathways to obtain an oral (noninhalation) hazard quotient for a single substance. For each substance, the impacts for a specific exposure pathway are assessed by dividing the oral dose (derived from the annual average concentration) in milligrams per kilogram-day (mg/kg-day) by the oral chronic REL, expressed in units of (mg/kg-day) (Table 6.4). The next equation shows the HQ calculation for arsenic through the soil ingestion (SI) exposure pathway.

Note, prior to this point in this calculation, we are assuming several steps have taken place. These steps include: 1) the completion of air dispersion modeling to obtain the ground-level annual-average air concentration; 2) identification of the existing exposure pathways at the receptor location; 3) calculation of the concentration in the exposure media (e.g., for soil - Equation 5.3.2.A); 4) determination of the dominant noninhalation exposure pathway(s) for the substance; and 5) the calculation of the substance-specific dose for that exposure pathway (e.g., Equation 5.4.3.1 is used to calculate the dose from soil ingestion). See Chapter 5 for the algorithms for calculating the oral dose for all of the applicable exposure pathways and receptors.

For this example, the calculated dose for arsenic from soil ingestion is assumed to be 0.000000015 mg/kg-day.

$$\text{Chronic Oral Hazard Quotient} = \frac{SI\ dose}{\left(\frac{Chronic}{Oral\ REL}\right)} \Rightarrow \text{Chronic Oral Hazard Quotient}_{(SI\ arsenic)} = \frac{(0.000000015 mg/kg-day)}{(0.0000035 mg/kg-day)}$$

$$= 0.04$$

For each substance, this step is repeated for each applicable noninhalation exposure pathway. As illustrated below, the (total) oral HQ for a substance is calculated by summing the HQs for all applicable exposure pathways. In this example, the chronic oral HQ is assumed to equal 0.1 from all exposure pathways.

$$\text{Chronic Oral Hazard Quotient*}_{(arsenic)} = [HQ_{(SI)} + HQ_{(D)} + HQ_{(DW)} + HQ_{(MI)} + HQ_{(FI)} + HQ_{(HV)}]$$

$$\text{Chronic Oral Hazard} = 0.1$$

* Noninhalation pathways:

SI = soil ingestion
DW = drinking water
D = dermal absorption
MI = meat, milk & egg
FI = fisher-caught fish
HV = homegrown vegetables
BM = breast milk (not applicable for arsenic exposure)

The oral chronic HQ calculated above for a substance applies to all the target organs listed under that substance for the noninhalation pathway.

Step four – Determine the chronic HI

The chronic HI is calculated by summing each HQ (inhalation and noninhalation) for each substance by the target organ system(s). For example, add the HQs for all substances that impact the respiratory system, then repeat this step for the next target organ system (e.g., cardiovascular system). This step is repeated until all target organs (for the substances emitted) are individually totaled. See Tables 6.3 and 6.4 for target organ system information. Note, do not add together the HQs or HIs for different target organ systems (e.g., do not add the impacts for the respiratory system to the cardiovascular system). Table I.11 shows individual hazard quotients (inhalation and noninhalation) for each substance and the hazard index by target organ system. In this table, arsenic is highlighted in bold to identify how the information calculated above is presented and used.

In this example, an HQ of one was not equaled or exceeded for any individual substance. However, an HI (the sum of the hazard quotients for each target organ) of one was exceeded for the respiratory system. Exceeding a hazard index of one may indicate that there is the potential for adverse chronic health impacts at this receptor location. The District and OEHHA should be consulted when a hazard index exceeds one (see Section 8.3).

TABLE I.11 SUBSTANCE-SPECIFIC INHALATION AND NONINHALATION HAZARD QUOTIENTS AND THE HAZARD INDEX BY TARGET ORGAN SYSTEM

Substance	Respiratory System	Hematologic System	Alimentary System	Endocrine System	Reproductive/ Development	Kidney	Nervous System	Cardiovascular System	Skin
Ammonia	0.8								
Arsenic	***0.1(i)*** ***0.1(ni)***				***0.1(i)*** ***0.1(ni)***		***0.1(i)*** ***0.1(ni)***	***0.1(i)*** ***0.1(ni)***	***0.1(i)*** ***0.1(ni)***
Benzene		0.02							
Chlorine	0.4								
Chloro-benzene			0.02		0.02	0.02			
2,3,7,8-TCDD (dioxin)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)				
Nickel	0.2(i)	0.2(i)			0.1(ni)				
Chronic Hazard Index*	**1.9**	**0.52**	**0.32**	**0.3**	**0.62**	**0.02**	**0.2**	**0.2**	**0.2**

i = inhalation pathway contribution
ni = noninhalation pathway contribution
* The total hazard index is the sum of the rounded individual hazard quotients for each target organ.

I.5 References

OEHHA, 2012a. *Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis*. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA Available online at http://www.oehha.ca.gov

OEHHA, 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA. May 2009. Available online at: http://www.oehha.ca.gov

Page intentionally left blank

Appendix J:

Glossary of Acronyms and Definition of Selected Terms

Adverse Health Effect: As defined by U.S. EPA, an adverse health effect is a biochemical change, functional impairment, or pathologic lesion that affects the performance of the whole organism, or reduces an organism's ability to respond to an additional environmental challenge. A health effect from exposure to air contaminants may range from relatively mild temporary conditions, such as eye or throat irritation, shortness of breath, or headaches, to permanent and serious conditions, such as birth defects, cancer or damage to lungs, nerves, liver, heart, or other organs.

AERMOD: A steady-state, plume-based air dispersion model (developed by U.S. EPA) for estimating near-field impacts from a variety of industrial source types. This was designed to provide reasonable concentration estimates over a wide range of conditions with minimal discontinuities, to be easily implemented with reasonable input requirements and computer resource needs, to be based on up-to-date science that captures the essential physical processes while remaining simple, and to be easily revised as the science evolves. To the extent practicable, the structure of the input or the control file for AERMOD is the same as that for the previously used ISCST3 model.

Age Sensitivity Factor (ASF): ASFs are default weighting factors to account for potential increased sensitivity to carcinogens during early life stages including prenatal, postnatal and juvenile life stages. ASFs are applied to the cancer risk equation.

Air Dispersion Modeling: Algorithms, usually performed with a computer, that relate a mass emission rate, source configuration, and meteorological information to calculate ambient air concentrations.

Air District: The Air Pollution Control and Air Quality Management Districts are the agencies responsible for managing air quality on a regional or county basis. California is currently divided into 35 air districts.

Air monitoring: The periodic or continuous sampling and analysis of air pollutants in ambient air or from individual pollutant sources.

Air Toxics Hot Spots Act Emission Inventory Reports: Documents that contain information regarding emission sources, emitted substances, emission rates and release parameters, prepared under the Emission Inventory Criteria and Guidelines (also referred to as “Inventory Reports”).

Air Toxics Hot Spots Information and Assessment Act of 1987 (AB 2588): (Health and Safety Code, Section 44300-44394) - A state law which established the "Hot Spots" Program to develop a statewide inventory of site-specific air toxic emissions, to assess the risk to public health from exposure to these emissions, to notify the public of any significant health risks and to reduce emissions below the significant risk levels.

Algorithm: A set of rules for solving a problem in a finite number of steps

California Air Resources Board (ARB): The State's lead air quality management agency consisting of an eleven-member board appointed by the Governor; in addition, the Air Resources Board has an Executive Office and a large staff of scientists and engineers to evaluate air pollution control measures. The ARB is responsible for attainment and maintenance of the state and federal air quality standards, and is fully responsible for motor vehicle pollution control. It oversees county and regional air pollution management programs.

Asthma: A chronic inflammatory disorder of the lungs characterized by wheezing, breathlessness, bronchoconstriction (resulting in chest tightness), and cough.

Atmospheric half-life: The time required for the concentration of a pollutant or reactant to fall to one-half of its initial value.

Benchmark Dose: That dose derived from linear regression, using one or more models of one or more dose-response curves, associated with a specific response rate (such as 1, 5, or 10%) in the test population. This is the starting dose (point of departure) to which uncertainty factors are applied to determine a reference exposure level (REL) using the benchmark dose approach.

Urban Block Groups (BGs): A geographical unit smaller than a census tract used for reporting census data. BGs contain roughly 1,100 persons.

Bioaccumulation: The concentration of a substance in a body or part of a body or other living tissue in a concentration higher than that of the surrounding environment

Bioconcentrate: The process of increasing contaminant concentration in biota up the food chain as contaminants are ingested and concentrated in tissues of organisms higher up in the chain.

Cancer burden: The estimated number of theoretical cancer cases in a defined population resulting from lifetime exposure to pollutants emitted from a facility.

Cancer potency factor (CPF): The theoretical upper bound probability of extra cancer cases occurring in an exposed population assuming a lifetime exposure to the chemical when the chemical dose is expressed in units of milligrams/kilogram body weight-day (mg/kg-d). The CPF is thus expressed in inverse units of mg/kg-d ($(mg/kg\text{-}d)^{-1}$).

California Air Pollution Control Officers Association (CAPCOA): A non-profit association of the air pollution control officers from all 35 air quality districts throughout California. CAPCOA was formed in 1975 to promote clean air and to provide a forum for sharing knowledge, experience, and information among the air quality regulatory agencies around the state.

Cal/EPA: The California Environmental Protection Agency is charged with developing, implementing and enforcing the state's environmental protection laws that ensure clean air, clean water, clean soil, safe pesticides and waste recycling and reduction. Its departments are at the forefront of environmental science, using cutting-edge research to shape the state's environmental laws. The Agency's boards and departments are: the Air Resources Board, the Department of Pesticide Regulation, the Department of Resources Recycling and Recovery (CalRecycle), the Department of Toxics Substances Control, the Office of Environmental Health Hazard Assessment, and the State Water Resources Control Board.

Chemical Abstract Services Registry Number (CAS): The Chemical Abstracts Service Registry Number (CAS) is a numeric designation assigned by the American Chemical Society's Chemical Abstracts Service and uniquely identifies a specific chemical compound. This entry allows one to conclusively identify a material regardless of the name or naming system used.

CCR: California Code of Regulations

CERCLA: Comprehensive Environmental Response, Compensation and Liability Act (Superfund), a federal regulation providing direction and financial support for the clean-up of major hazardous waste sites

Centroid Locations: The location at which calculated ambient concentration is assumed to represent the entire subarea, typically the geometric centroid of an area, but possibly the population-weighted centroid of the area.

Census Tract: A physical area used by the U.S. Census Bureau to compile population and other statistical data.

Criteria Air Pollutant: A pollutant for which the U.S. Environmental Protection Agency or the Air Resources Board has established an Ambient Air Quality Standard (AAQS). Examples include ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, lead, and PM_{10} and $PM_{2.5}$.

Default: A value used to account for a factor when specific information on that factor that applies to a specific situation is not available.

Developmental toxicity: Adverse effects on the developing organism that may result from exposure prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism. Major manifestations of developmental toxicity include: death of the developing organism; induction of structural birth defects; altered growth; and functional deficiency.

Dilution factor (χ/Q): A site-specific quantity defined as a ratio of the ground level concentration in $\mu g/m^3$ to the mass emission rate in g/s and represented by χ/Q.

Dose: A calculated amount of a substance estimated to be received by the subject, whether human or animal, as a result of exposure. Doses are generally expressed in terms of amount of chemical per unit body weight; typical units are mg/kg-day.

Dose-response assessment: The process of characterizing the relationship between the exposure to an agent and the incidence of an adverse health effect in exposed populations.

DTSC: California Department of Toxic Substances Control—DTSC regulates hazardous waste, cleans-up existing contamination, and looks for ways to reduce the hazardous waste produced in California. Its scientists, engineers, and specialized support staff make sure that companies and individuals handle, transport, store, treat, dispose of, and clean-up hazardous wastes appropriately. Through these measures, DTSC contributes to greater safety for all Californians, and less hazardous waste reaches the environment.

ED: Rural Enumeration District. A geographical unit smaller than a census tract used to report census data. EDs contain roughly 1,100 persons.

Emission Inventory Criteria and Guidelines: Regulation and Report adopted by the California Air Resources Board specifying criteria and procedures for the preparation of Air Toxics Hot Spots Act Emission Inventory Reports (Title 17, California Code or Regulations, Sections 93300-93300.5).

Endpoint: An observable or measurable biological or biochemical event including cancer used as an index of the effect of a chemical on a cell, tissue, organ, organism, etc.

Epidemiology: The study of the occurrence and distribution of a disease or physiological condition in human populations and of the factors that influence this distribution.

Exposure: Contact of an organism with a chemical, physical, or biological agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, digestive tract) and available for absorption.

Exposure Pathway: A route of exposure by which xenobiotics enter the human body, (e.g., inhalation, ingestion, dermal absorption).

Fugitive Dust: Dust particles that are introduced into the air through certain activities such as soil cultivation, or vehicles operating on open fields or dirt roadways. A subset of fugitive emissions.

Fugitive Emissions: Emissions not caught by a capture system which are often due to equipment leaks, evaporative processes, and windblown disturbances.

Gaussian Model: An air dispersion model based on the assumption that the time-averaged concentration of a species emitted from a point source has a Gaussian distribution about the mean centerline.

Genotoxic: Having an adverse effect on the genetic material (DNA) resulting in a mutation or in chromosome damage.

GLC: Estimated ground level concentration, usually for a specified averaging time (e.g., annual average, 1 hour, etc.).

GRAF: The Gastrointestinal Relative Absorption Factor**,** defined as the fraction of contaminant absorbed by the GI tract relative to the fraction of contaminant absorbed from the matrix (feed, water, other) used in the study(ies) that is the basis of either the cancer potency factor (CPF) or the Reference Exposure Level (REL)

Hot Spots Analysis and Reporting Program (HARP): A single integrated software package designed to promote statewide consistency, efficiency, and cost-effective implementation of health risk assessments and the Hot Spots Program. The HARP software package consists of three modules that include: 1) the Emissions Inventory Database Module, 2) the Air Dispersion Modeling Module, and 3) the Risk Analysis and Mapping Module.

Health Risk Assessment (HRA): The name of a computer program developed by the ARB, the OEHHA, and the University of California which was designed to aid in the computation of risk in the Hot Spots program

HSC: Health and Safety Code of the State of California

Haber's Law: The product of the concentration (C) and time of exposure (t) required to produce a specific physiologic effect is equal to a constant level or severity of response (K), or C * t = K.

Hazard Identification: The process of determining whether exposure to an agent can cause an increase in the incidence of an adverse health effect including cancer.

Health Risk Assessment: Health risk assessment is the characterization of the potential adverse health effects of human exposures to environmental hazards. In the Air Toxics Hot Spots program, a health risk assessment (HRA) is an evaluation or report that a risk assessor (e.g., district, consultant, or facility operator) develops to describe the potential a person or population may have of developing adverse health effects from exposure to a facility's emissions. Some health effects that are evaluated could include cancer, developmental effects, or respiratory illness. The pathways that can be included in an HRA depend on the toxic air pollutants that a person (receptor) may be exposed to, and can include breathing, the ingestion of soil, water, crops, fish, meat, milk, and eggs, and dermal exposure.

Health Risk Guidance Value (HRGV): A numerical value with which to compare an exposure level in order to determine the probability of occurrence of an adverse health effect. In the Hot Spots program the toxicity criteria or toxicity values are known as Reference Exposure Levels (RELs) for noncancer effects and as inhalation unit risk factors and cancer potency values for cancer effects.

Hazard Index (HI): The sum of individual acute or chronic hazard quotients (HQs) for each substance affecting a particular toxicological endpoint.

Hazard Quotient (HQ): The estimated ground level concentration divided by the reference exposure level for a single substance and a particular endpoint. For an acute HQ the one hour maximum concentration is divided by the acute Reference Exposure Level (REL) for the substance. For a repeated 8 hr HQ, the 8 hr average concentration is divided by the 8 hr REL. For a chronic HQ, the annual concentration is divided by the chronic REL.

Hot Spot: A location where emissions from specific sources may expose individuals and population groups to elevated risks of adverse health effects, including but not limited to cancer, and contribute to the cumulative health risks of emissions from other sources in the area.

Individual Excess Cancer Risk: The theoretical probability of an individual person developing cancer as a result of lifetime exposure to carcinogenic substances. The Individual Excess Cancer Risk is calculated by summing the potential cancer risks due to both inhalation and noninhalation routes of exposure, generally at the off-site point of maximum impact. This "individual" is the maximally exposed individual (MEI).

Inhalation (Breathing) Rate: The amount of air inhaled in a specified time period (e.g., per minute, per hour, per day, etc.); also called breathing rate and ventilation rate. This is an example of an exposure variate.

Inhalation unit risk factor: The theoretical upper bound probability of extra cancer cases occurring in the exposed population assuming a lifetime exposure to the chemical when the air concentration is expressed in units of microgram/cubic meter ($\mu g/m^3$). The unit risk factor is thus expressed as $(\mu g/m^3)^{-1}$.

Initiator carcinogen: A substance which causes the first stage of carcinogenesis, the conversion of a normal cell to a neoplastic cell. Initiation is considered to be a rapid, irreversible change often involving a change in the DNA caused by the initiator.

Interspecies: Between different species.

Intraspecies: Within the same species.

Industrial Source Complex Dispersion model (ISC3): Air modeling software that was previously used by U.S. EPA and the Hot Spots program. It incorporates three sub-programs into a single program. These are the short-term model (ISCST), the long term model (ISCLT), and the complex terrain model (COMPLEX).

Isopleth: A line on a map connecting points of equal value (e.g., risk, concentration, etc.).

Lowest-observed adverse effect level (LOAEL): The lowest dose or exposure level of a chemical in a study at which there is a statistically or biologically significant increase in the frequency or severity of an adverse effect in the exposed population as compared with an appropriate, unexposed control group.

Margin of safety: The ratio of the no-observed-adverse-effect level (NOAEL) to the estimated human exposure.

Mean: The arithmetic average**.**

MEI: Maximum exposed individual (theoretical)

MEIR: Maximum exposed individual resident (actual)

MEIW: Maximum exposed individual worker (actual)

Meteorology: The science that deals with the phenomena of the atmosphere especially weather and weather conditions. In the area of air dispersion modeling, *meteorology* is used to refer to climatological data needed to run an air dispersion model including: wind speed, wind direction, stability class and ambient temperature.

Milligram: One one-thousandth (10^{-3}) of a gram.

Molecular formula: The formula which identifies the atoms and the number of each kind in the molecules of a compound. Elements in the molecular formula are listed according to the Hill convention (C, H, then other elements in alphabetical order).

Molecular weight: The sum of the atomic weights of the atoms in a molecule. For example, methane (CH_4) is 16.043, the atomic weights are carbon = 12.011, hydrogen = 1.008.

Monte Carlo simulation: Application of random sampling to obtain an approximate value of an expression. Monte Carlo simulation involves computational algorithms that rely on repeated random sampling to obtain numerical results by running simulations many times over in order to calculate probability of a value.

Multipathway substance: A substance or chemical that once airborne from an emission source can, under environmental conditions, be taken into a human receptor by multiple exposure routes, such as inhalation, skin contact with contaminated surfaces, ingestion of soil contaminated by the emission, etc.

No Observed Adverse Effect Level (NOAEL): The highest experimental dose at which there is no statistically or biologically significant increase in frequency or severity of adverse non-cancer health effects in the exposed population compared with an appropriate, unexposed population. Effects may be produced at this level, but they are not considered to be adverse.

Noncarcinogenic Effects: Noncancer health effects which may include birth defects, organ damage, morbidity, and death.

Office of Environmental Health Hazard Assessment (OEHHA): The office within the California Environmental Protection Agency that is responsible for evaluating chemicals for adverse health impacts and establishing safe exposure levels. OEHHA also assists in performing health risk assessments and developing risk assessment procedures for air quality management purposes.

PM_{10}, $PM_{2.5}$: PM_{10} is particulate matter less than 10 μm in diameter; $PM_{2.5}$ is particulate matter less than 2.5 μm in diameter.

PMI: Off-site point of maximum impact. A location, with or without people currently present, at which the total cancer risk, or the total noncancer risk, has the highest numerical value.

Point Estimate: A single value estimate for a given variate.

Potency: The relative effectiveness, or risk, of a standard amount of a substance to cause a toxic response. This term is used particularly to refer to carcinogens.

Potency Slope (also referred to as “Slope Factor” or “ Cancer Potency”)**:** Used to calculate the probability or risk of cancer associated with an estimated exposure, based on the assumption in cancer risk assessments that risk is directly proportional to dose and that there is no threshold for carcinogenesis. It is the slope of the dose-response curve extrapolated to low environmental exposures. It is expressed in per unit dose (usually mg per kg-day): thus cancer potency typically has the units $(mg/kg\text{-}day)^{-1}$.

Proposition 65: Safe Drinking Water and Toxic Enforcement Act of 1986, also known as Proposition 65. This Act is codified in California Health and Safety Code Section 25249.5, et seq. .The Proposition was intended by its authors to protect California citizens and the State's drinking water sources from chemicals known to cause cancer, birth defects or other reproductive harm, and to inform citizens about exposures to such chemicals.

Resource Conservation and Recovery Act (RCRA) of 1976: A federal law regulating disposal of hazardous waste.

Receptor: A location with or without people present at which the ground level concentration of an emitted chemical can be estimated.

Refined Models: Air dispersion models designed to provide more representative concentration estimates than screening models taking into account actual meteorological conditions.

Reference Concentration (RfC): An estimate, derived by the U.S. EPA (with an uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population, (including sensitive subgroups) that is likely to be without appreciable risk of deleterious non-cancer effects during a lifetime of exposure. The RfC is derived from a no or lowest observed adverse effect level from human or animal exposures, to which uncertainty factors are applied, and is expressed in units of mg or µg per m^3.

Reference Dose (RfD): An estimate delivered by the U.S. EPA (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subpopulations) that is likely to be without deleterious non-cancer effects during a lifetime. The RfD is reported in units of mg of substance/kg body weight/day for oral exposures.

Reference exposure level (REL): The REL is an exposure level at or below which no noncancer adverse health effect is anticipated to occur in a human population, including sensitive subpopulations, exposed for a specific duration. One hour acute RELs are designed to be protective for infrequent one hour maximum exposures. Eight-hour RELs are designed to be protective for repeated 8 hour exposures. Chronic RELs are designed to be protective for continuous long-term exposures. RELs are used to evaluate toxicity endpoints other than cancer. RELs are expressed in units of $\mu g/m^3$ for inhalation exposures and of mg/kg-d for noninhalation exposures.

Reproductive toxicity: Harmful effects on sexual function in males or females, fertility or gestation, caused by exposure of either parent to a substance. Reproductive toxicity also includes developmental effects on the offspring. See also developmental toxicity which refers to adverse effects on the offspring.

Risk: The estimated probability of adverse effects to human health, in this instance from the exposure to environmental hazards.

Risk Assessment: The characterization of the probability of adverse health effects to people from exposure to environmental hazards, in this case of exposure to chemical emissions.

Risk Management: An evaluation of the need for and feasibility of reducing risk. It includes consideration of magnitude of risk, available control technologies, and economic feasibility.

Risk Management and Prevention Program (RMPP): A program administered by the Office of Emergency Services (OES) and local agencies to reduce the frequency and severity of accidental releases of toxic materials.

SB 25 (Children's Environmental Health Protection Act): A state law (Senate Bill 25, Escutia, 1999) that amended the existing Toxic Air Contaminant and Criteria Air Pollutant laws and established requirements for the ARB and the OEHHA to examine the impacts of air pollution more explicitly on children's health. The act required the state to evaluate all ambient air quality standards to determine whether these standards adequately protect human health, particularly that of infants and children; to identify toxic air contaminants that disproportionately impact children, and to ensure that health assessments of toxic chemicals explicitly incorporate considerations of infants and children.

Scientific Review Panel on Toxic Air Contaminants or SRP: A nine-member panel appointed to advise the Air Resources Board, the Office of Environmental Health Hazard Assessment, and the Department of Pesticide Regulation in their evaluation of the adverse health effects and toxicity of substances being evaluated as Toxic Air Contaminants.

Screening Models: Dispersion models used to provide a maximum concentration that is likely to overestimate public exposure.

Sensitive Receptor: A location such as a hospital or daycare center where the human occupants are considered to be more sensitive to pollutants than “average”.

Spatial Averaging: The method used in the Hot Spots Program for determining an average air concentration from a grid of receptors over a specified area.

Stationary source: A non-mobile source of air pollutants which can be either a point or area source.

Stochastic: A process that involves random variation.

Synergism: A pharmacologic or toxicologic interaction in which the combined effect of two or more chemicals is greater than the sum of the effects of each chemical alone.

Subcensus Tract: Smaller population unit within a census tract.

Surrogate: As used in this document refers to a single substance category used to represent a family of related chemical compounds, e.g., benzo(a)pyrene in place of POM (polycyclic organic matter).

Threshold, Nonthreshold: A threshold dose is the minimally effective dose of any chemical that is observed in a population to produce a response (e.g., enzyme change, liver toxicity, death). For most toxic effects, except carcinogenesis, there appear to be threshold doses. (Exceptions include observed cardiovascular mortality in humans from exposure to particulate matter, and the neurotoxic effects of lead). Nonthreshold substances are those substances, including nearly all carcinogens, that are known or assumed to have some risk of response at any dose above zero.

Toxic air contaminant (TAC): As defined by California Health and Safety Code, Section 39655 (a): an air pollutant which may cause or contribute to an increase in mortality or in serious illness, or which may pose a present or potential hazard to human health. Substances, which have been identified by the United States Environmental Protection Agency as hazardous air pollutants (e.g. benzene, asbestos), shall be identified by the Board as toxic air contaminants.

Toxicology: The multidisciplinary study of toxicants, their harmful effects on biological systems, and the conditions under which these harmful effects occur. The mechanisms of action, detection, and treatment of the conditions produced by toxicants are studied.

Uncertainty: True uncertainty is that which is not known about a factor that influences its value.

URF: See inhalation unit risk factor.

UTM Coordinates: Universal Transverse Mercator Coordinates. Coordinates used to define a specific location on earth by means of two values (i.e., easting and northing coordinates).

United States Environmental Protection Agency (U.S. EPA): The mission of EPA is to protect human health and the environment. The agency sets national standards that are enforced by them or that states and tribes enforce through their own regulations. The agency also provide grants to state environmental programs, non-profits, educational institutions, and others for a wide variety of projects, from scientific studies that are used to make decisions to community cleanups. EPA's purpose is to ensure that: 1) All Americans are protected from significant risks to human health and the environment where they live, learn and work; 2) National efforts to reduce environmental risk are based on the best available scientific information; 3) Federal laws protecting human health and the environment are enforced fairly and effectively; 4) Environmental protection is an integral consideration in U.S. policies concerning natural resources, human health, economic growth, energy, transportation, agriculture, industry, and international trade, and these factors are similarly considered in establishing environmental policy; 5) All parts of society -- communities, individuals, businesses, and state, local and tribal governments -- have access to accurate information sufficient to effectively participate in managing human health and environmental risks; 6) Environmental protection contributes to making our communities and ecosystems diverse, sustainable and economically productive; and 7) The United States plays a leadership role in working with other nations to protect the global environment.

Vapor: The gaseous phase of materials at atmospheric temperature and pressure.

Vapor Pressure: The pressure exerted by a chemical vapor in equilibrium with its liquid or solid phase at any given temperature, used to calculate the rate of evaporation of a substance.

Variability: Ability to have different numerical values of a parameter, such as height or weight.

Variate: A variable quantity associated with a probability distribution, in the case of the Hot Spots program, for example, exposure factors (e.g. inhalation rate).

Volatile: Chemicals that rapidly pass off from the liquid state in the form of vapors.

Xenobiotic: A chemical or substance that is foreign to an organism or biological system. A chemical that is foreign to the species in which the chemical is being being studied.

Zone of impact: The area in the vicinity of the facility in which an individual is exposed to a specified cancer risk, usually one in a million or greater.

Appendix K:

HRA Forms and Maps Used With Air Dispersion Modeling

- Example of Census Tract Map
- Example of 7.5 minute Series Map
- Examples of Tables for Emissions Reporting

Figure 1
Census Tracts, View 1

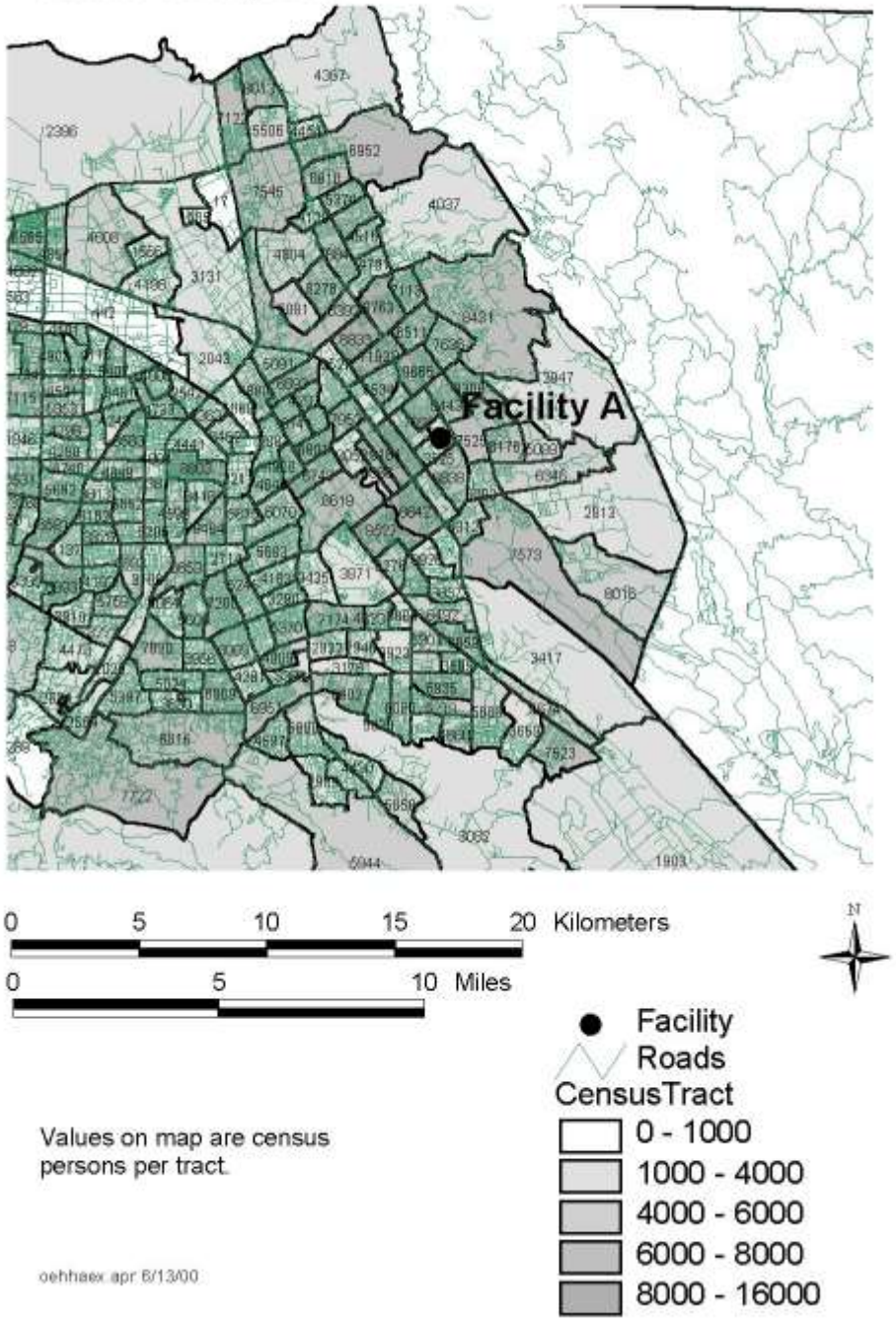


Figure 2
Census Tracts, View 2

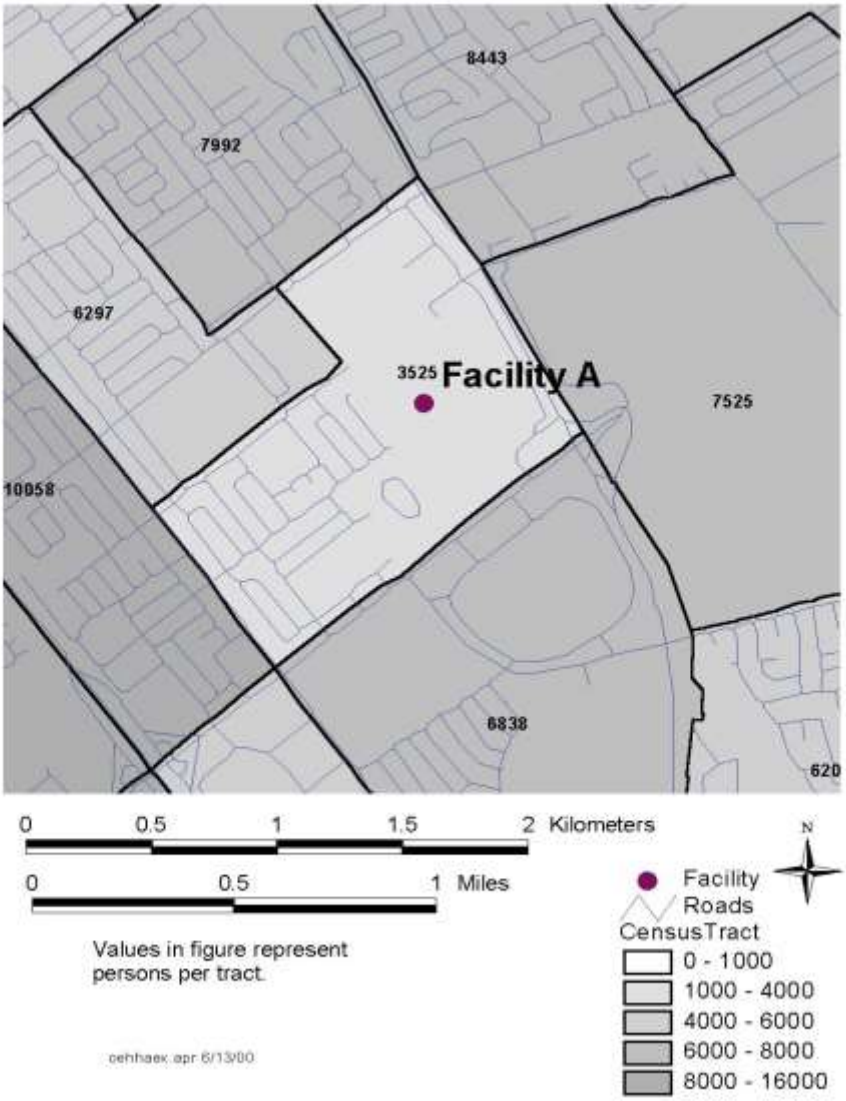
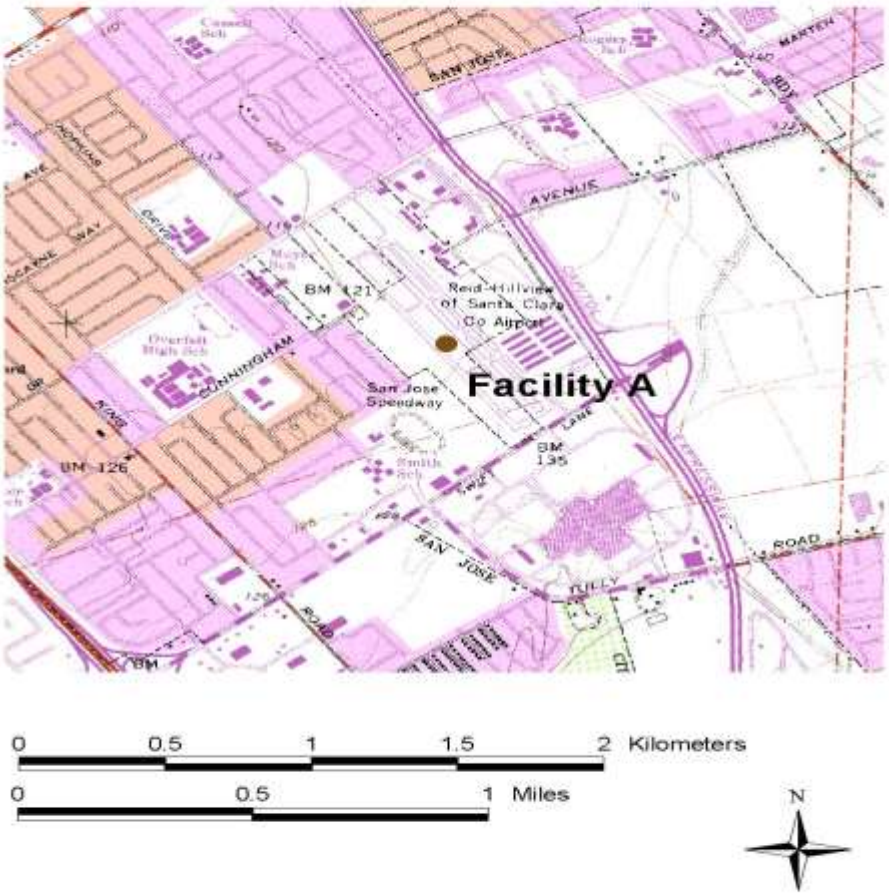


Figure 4
USGS 7.5 Minute Topographic Map



oehhaex.apr 6/13/00

HEALTH RISK ASSESSMENT

EMISSION RATE BY SUBSTANCE AND SOURCE RAG-001

FACILITY NAME / FACILITY ADDRESS / SITE ID#:

SOURCE ID No.	SOURCE NAME	SUBSTANCE NAME	CAS No.	1-HOUR MAXIMUM (lb/hr)	1-HOUR MAXIMUM (g/s)	ANNUAL AVERAGE (lb/yr)	ANNUAL AVERAGE (g/s)

HEALTH RISK ASSESSMENT

EMISSION RATE BY SUBSTANCE – TOTALS – RAG-002

FACILITY NAME / FACILITY
ADDRESS / SITE ID#

SUBSTANCE NAME	CAS No.	1-HOUR MAXIMUM (lb/hr)	1-HOUR MAXIMUM (g/s)	ANNUAL AVERAGE (lb/yr)	ANNUAL AVERAGE (g/s)

HEALTH RISK ASSESSMENT

SOURCE PARAMETERS – STACKS – RAG-003

FACILITY NAME / FACILITY ADDRESS / SITE ID#

SOURCE ID No.	STACK NAME	UTM Easting	UTM Northing	HEIGHT (m)	DIAMETER (m)	TEMP. (F) (K)		FLOW RATE (ACFM)	EXIT VEL. (m/s)

HEALTH RISK ASSESSMENT

SOURCE OPERATING HOURS – RAG-004

FACILITY NAME / FACILITY ADDRESS / SITE ID#

SOURCE ID No.	STACK NAME	AVERAGE OPERATING HOURS		MAXIMUM OPERATING HOURS	
		(hr/day)	(days/year)	(hr/day)	(days/year)

Appendix L:

OEHHA/ARB Approved Health Values for Use in Hot Spot Facility Risk Assessments

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical Abstract Number[b]	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
ACETALDEHYDE	75-07-0	4.7E+02	12/08	3.0E+02	12/08	1.4E+02	12/08			2.7E-06	1.0E-02	4/99 [5/93]			1
ACETAMIDE	60-35-5									2.0E-05	7.0E-02	4/99			1
ACROLEIN	107-02-8	2.5E+00	12/08	7.0E-01	12/08	3.5E-01	12/08								1
ACRYLAMIDE	79-06-1									1.3E-03	4.5E+00	4/99 [7/90]			1
ACRYLIC ACID	79-10-7	6.0E+03	4/99												1
ACRYLONITRILE	107-13-1					5.0E+00	12/01			2.9E-04	1.0E+00	4/99 [1/91]			1
ALLYL CHLORIDE	107-05-1									6.0E-06	2.1E-02	4/99			1
2-AMINOANTHRAQUINONE	117-79-3									9.4E-06	3.3E-02	4/99			1
AMMONIA	7664-41-7	3.2E+03	4/99			2.0E+02	2/00								1
ANILINE	62-53-3									1.6E-06	5.7E-03	4/99			1
ARSENIC AND COMPOUNDS (INORGANIC)[TAC]	7440-38-2 1016 [1015]	2.0E-01	12/08	1.5E-02	12/08	1.5E-02	12/08	3.5E-06	12/08	3.3E-03 [TAC]	1.2E+01	7/90	1.5E+00	10/00	1
ARSINE	7784-42-1	2.0E-01	12/08	1.5E-02	12/08	1.5E-02	12/08								1
ASBESTOS[TAC, f]	1332-21-4									1.9E-04 [TAC][f]	2.2E+02	3/86			333.33
BENZENE[TAC]	71-43-2	2.7E+01	6/14	3.0E+00	6/14	3.0E+00	6/14			2.9E-05[TAC]	1.0E-01	1/85			1
BENZIDINE (AND ITS SALTS) *values also apply to:*	92-87-5									1.4E-01	5.0E+02	4/99 [1/91]			1
Benzidine based dyes	*1020*									*1.4E-01*	*5.0E+02*	*4/99 [1/91]*			*1*
Direct Black 38	*1937-37-7*									*1.4E-01*	*5.0E+02*	*4/99 [1/91]*			*1*
Direct Blue 6	*2602-46-2*									*1.4E-01*	*5.0E+02*	*4/99 [1/91]*			*1*
Direct Brown 95 (technical grade)	*16071-86-6*									*1.4E-01*	*5.0E+02*	*4/99 [1/91]*			*1*
BENZYL CHLORIDE	100-44-7	2.4E+02	4/99							4.9E-05	1.7E-01	4/99			1
BERYLLIUM AND COMPOUNDS	7440-41-7 [1021]					7.0E-03	12/01	2.0E-03	12/01	2.4E-03	8.4E+00	4/99 [7/90]			1
BIS(2-CHLOROETHYL)ETHER (Dichloroethyl ether)	111-44-4									7.1E-04	2.5E+00	4/99			1
BIS(CHLOROMETHYL)ETHER	542-88-1									1.3E-02	4.6E+01	4/99 [1/91]			1
BROMINE AND COMPOUNDS	7726-95-6 [1040]														1
POTASSIUM BROMATE	7758-01-2									1.4E-04	4.9E-01	4/99 [10/93]			1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
1,3-BUTADIENE[TAC]	106-99-0	6.6E+02	7/13	9.0E+00	7/13	2.0E+00	7/13			1.7E-04 TAC	6.0E-01	7/92			1
CADMIUM AND COMPOUNDS[TAC]	7440-43-9 [1045]					2.0E-02	1/01	5.0E-04	10/00	4.2E-03 TAC	1.5E+01	1/87			1
CAPROLACTAM	105-60-2	5.0E+01	10/13	7.0E+00	10/13	2.2E+00	10/13								
CARBON DISULFIDE	75-15-0	6.2E+03	4/99			8.0E+02	5/02								1
CARBON MONOXIDE	630-08-0	2.3E+04	4/99												1
CARBON TETRACHLORIDE[TAC] (Tetrachloromethane)	56-23-5	1.9E+03	4/99			4.0E+01	1/01			4.2E-05 TAC	1.5E-01	9/87			1
CHLORINATED PARAFFINS	108171-26-2									2.5E-05	8.9E-02	4/99			1
CHLORINE	7782-50-5	2.1E+02	4/99			2.0E-01	2/00								1
CHLORINE DIOXIDE	10049-04-4					6.0E-01	1/01								1
4-CHLORO-O-PHENYLENEDIAMINE	95-83-0									4.6E-06	1.6E-02	4/99			1
CHLOROBENZENE	108-90-7					1.0E+03	1/01								1
CHLOROFORM[TAC]	67-66-3	1.5E+02	4/99			3.0E+02	4/00			5.3E-06 TAC	1.9E-02	12/90			1
Chlorophenols	*1060*														*1*
PENTACHLOROPHENOL	87-86-5									5.1E-06	1.8E-02	4/99			1
2,4,6-TRICHLOROPHENOL	88-06-2									2.0E-05	7.0E-02	4/99 [1/91]			1
CHLOROPICRIN	76-06-2	2.9E+01	4/99			4.0E-01	12/01								1
p-CHLORO-o-TOLUIDINE	95-69-2									7.7E-05	2.7E-01	4/99			1
CHROMIUM 6+[TAC] *values also apply to:*[g]	18540-29-9					2.0E-01	1/01	2.0E-02	10/00	1.5E-01 TAC	5.1E+02	1/86	5.0E-01	*1/14*	1
Barium chromate	*10294-40-3*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.2053*
Calcium chromate	*13765-19-0*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.3332*
Lead chromate	*7758-97-6*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.1609*
Sodium dichromate	*10588-01-9*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.397*
Strontium chromate	*7789-06-2*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.2554*
CHROMIUM TRIOXIDE (as chromic acid mist)	1333-82-0					2.0E-03	1/01	2.0E-02	10/00	1.5E-01 TAC	5.1E+02	1/86	5.0E-01	*1/14*	0.52
COPPER AND COMPOUNDS	7440-50-8 [1067]	1.0E+02	4/99												1
p-CRESIDINE	120-71-8									4.3E-05	1.5E-01	4/99			1
CRESOLS (mixtures of)	1319-77-3					6.0E+02	1/01								1
m-CRESOL	108-39-4					6.0E+02	1/01								1
o-CRESOL	95-48-7					6.0E+02	1/01								1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
p-CRESOL	106-44-5					6.0E+02	1/01								1
CUPFERRON	135-20-6									6.3E-05	2.2E-01	4/99			1
Cyanide Compounds (inorganic)	*57-12-5 1073*	*3.4E+02*	*4/99*			*9.0E+00*	*4/00*								*1*
HYDROGEN CYANIDE (Hydrocyanic acid)	74-90-8	3.4E+02	4/99			9.0E+00	4/00								1
2,4-DIAMINOANISOLE	615-05-4									6.6E-06	2.3E-02	4/99			1
2,4-DIAMINOTOLUENE	95-80-7									1.1E-03	4.0E+00	4/99			1
1,2-DIBROMO-3-CHLOROPROPANE (DBCP)	96-12-8									2.0E-03	7.0E+00	4/99 [1/92]			1
p-DICHLOROBENZENE	106-46-7					8.0E+02	1/01			1.1E-05	4.0E-02	4/99 [1/91]			1
3,3-DICHLOROBENZIDINE	91-94-1									3.4E-04	1.2E+00	4/99 [1/91]			1
1,1,-DICHLOROETHANE (Ethylidene dichloride)	75-34-3									1.6E-06	5.7E-03	4/99			1
1,1-DICHLOROETHYLENE … (see Vinylidene Chloride)															
DI(2-ETHYLHEXYL)PHTHALATE (DEHP)	117-81-7									2.4E-06	8.4E-03	4/99 [1/92]	8.4E-03	10/00	1
DIESEL EXHAUST … (see Particulate Emissions from Diesel-Fueled Engines)															
DIETHANOLAMINE	111-42-2					3.0E+00	12/01								
p-DIMETHYLAMINOAZOBENZENE	60-11-7									1.3E-03	4.6E+00	4/99			1
N,N-DIMETHYL FORMAMIDE	68-12-2					8.0E+01	1/01								1
2,4-DINITROTOLUENE	121-14-2									8.9E-05	3.1E-01	4/99			1
1,4-DIOXANE; (1,4-Diethylene dioxide)	123-91-1	3.0E+03	4/99			3.0E+03	4/00			7.7E-06	2.7E-02	4/99 [1/91]			1
EPICHLOROHYDRIN (1-Chloro-2,3-epoxypropane)	106-89-8	1.3E+03	4/99			3.0E+00	1/01			2.3E-05	8.0E-02	4/99 [1/92]			1
1,2-EPOXYBUTANE	106-88-7					2.0E+01	1/01								1
ETHYL BENZENE	100-41-4					2.0E+03	2/00			2.5E-06	8.7E-3	11/07			1
ETHYL CHLORIDE (Chloroethane)	75-00-3					3.0E+04	4/00								1
ETHYLENE DIBROMIDE[TAC] (1,2-Dibromoethane)	106-93-4					8.0E-01	12/01			7.1E-05 TAC	2.5E-01	7/85			1
ETHYLENE DICHLORIDE[TAC] (1,2-Dichloroethane)	107-06-2					4.0E+02	1/01			2.1E-05 TAC	7.2E-02	9/85			1
ETHYLENE GLYCOL	107-21-1					4.0E+02	4/00								1
ETHYLENE GLYCOL BUTYL ETHER … (see Glycol ethers)															
ETHYLENE OXIDE[TAC] (1,2-Epoxyethane)	75-21-8					3.0E+01	1/01			8.8E-05 TAC	3.1E-01	11/87			1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
ETHYLENE THIOUREA	96-45-7									1.3E-05	4.5E-02	4/99			1
Fluorides	1101	*2.4E+02*	*4/99*			1.3E+01	8/03	4.0E-02	8/03						*1*
HYDROGEN FLUORIDE (Hydrofluoric acid)	7664-39-3	2.4E+02	4/99			1.4E+01	8/03	4.0E-02	8/03						1
FORMALDEHYDE[TAC]	50-00-0	5.5E+01	12/08	9.0E+00	12/08	9.0E+00	12/08			6.0E-06 TAC	2.1E-02	3/92			1
GLUTARALDEHYDE	111-30-8					8.0E-02	1/01								1
GLYCOL ETHERS	1115														1
ETHYLENE GLYCOL BUTYL ETHER – EGBE	111-76-2	1.4E+04	4/99												1
ETHYLENE GLYCOL ETHYL ETHER – EGEE	110-80-5	3.7E+02	4/99[1/92]			7.0E+01	2/00								1
ETHYLENE GLYCOL ETHYL ETHER ACETATE – EGEEA	111-15-9	1.4E+02	4/99			3.0E+02	2/00								1
ETHYLENE GLYCOL METHYL ETHER – EGME	109-86-4	9.3E+01	4/99			6.0E+01	2/00								1
ETHYLENE GLYCOL METHYL ETHER ACETATE – EGMEA	110-49-6					9.0E+01	2/00								1
HEXACHLOROBENZENE	118-74-1									5.1E-04	1.8E+00	4/99 [1/91]			1
HEXACHLOROCYCLOHEXANES (mixed or technical grade)	608-73-1									1.1E-03	4.0E+00	4/99 [1/91]	4.0E+00	10/00 [1/92]	1
alpha-HEXACHLOROCYCLOHEXANE	319-84-6									1.1E-03	4.0E+00	4/99 [1/91]	4.0E+00	10/00 [1/92]	1
beta-HEXACHLOROCYCLOHEXANE	319-85-7									1.1E-03	4.0E+00	4/99 [1/91]	4.0E+00	10/00 [1/92]	1
gamma-HEXACHLOROCYCLOHEXANE (Lindane)	58-89-9									3.1E-04	1.1E+00	4/99	1.1E+00	10/00	1
n-HEXANE	110-54-3					7.0E+03	4/00								1
HYDRAZINE	302-01-2					2.0E-01	1/01			4.9E-03	1.7E+01	4/99 [7/90]			1
HYDROCHLORIC ACID (Hydrogen chloride)	7647-01-0	2.1E+03	4/99			9.0E+00	2/00								1
HYDROGEN BROMIDE … (see Bromine & Compounds)															
HYDROGEN CYANIDE … (see Cyanide & Compounds)															
HYDROGEN FLUORIDE … (see Fluorides & Compounds)															
HYDROGEN SELENIDE … (see Selenium & Compounds)															
HYDROGEN SULFIDE	7783-06-4	4.2E+01	4/99[7/90]			1.0E+01	4/00								1
ISOPHORONE	78-59-1					2.0E+03	12/01								

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
ISOPROPYL ALCOHOL (Isopropanol)	67-63-0	3.2E+03	4/99			7.0E+03	2/00								1
LEAD AND COMPOUNDS[TAC, h] (inorganic) *values also apply to:*	7439-92-1 1128 [1130]									1.2E-05 TAC	4.2E-02	4/97	8.5E-03	10/00	1
Lead acetate	*301-04-2*									*1.2E-05 TAC*	*4.2E-02*	*4/97*	*8.5E-03*	*10/00*	*0.637*
Lead phosphate	*7446-27-7*									*1.2E-05 TAC*	*4.2E-02*	*4/97*	*8.5E-03*	*10/00*	*0.7659*
Lead subacetate	*1335-32-6*									*1.2E-05 TAC*	*4.2E-02*	*4/97*	*8.5E-03*	*10/00*	*0.7696*
LINDANE ... (see gamma-Hexachlorocyclohexane)															
MALEIC ANHYDRIDE	108-31-6					7.0E-01	12/01								1
MANGANESE AND COMPOUNDS	7439-96-5 [1132]			1.7E-01	12/08	9.0E-02	12/08								1
MERCURY AND COMPOUNDS (INORGANIC)	7439-97-6 [1133]	6.0E-01	12/08	6.0E-02	12/08	3.0E-02	12/08	1.6E-04	12/08						1
Mercuric chloride	7487-94-7	6.0E-01	12/08	6.0E-02	12/08	3.0E-02	12/08	1.6E-04	12/08						1
METHANOL	67-56-1	2.8E+04	4/99			4.0E+03	4/00								1
METHYL BROMIDE (Bromomethane)	74-83-9	3.9E+03	4/99			5.0E+00	2/00								1
METHYL tertiary-BUTYL ETHER	1634-04-4					8.0E+03	2/00			2.6E-07	1.8E-03	11/99			1
METHYL CHLOROFORM (1,1,1-Trichloroethane)	71-55-6	6.8E+04	4/99			1.0E+03	2/00								1
METHYL ETHYL KETONE (2-Butanone)	78-93-3	1.3E+04	4/99												1
METHYL ISOCYANATE	624-83-9					1.0E+00	12/01								1
4,4'-METHYLENE BIS (2-CHLOROANILINE) (MOCA)	101-14-4									4.3E-04	1.5E+00	4/99			1
METHYLENE CHLORIDE[TAC] (Dichloromethane)	75-09-2	1.4E+04	4/99			4.0E+02	2/00			1.0E-06 TAC	3.5E-03	7/89			1
4,4'-METHYLENE DIANILINE (AND ITS DICHLORIDE)	101-77-9					2.0E+01	12/01			4.6E-04	1.6E+00	4/99	1.6E+00	10/00	1
METHYLENE DIPHENYL ISOCYANATE	101-68-8					7.0E-01	1/01								1
MICHLER'S KETONE (4,4'-Bis(dimethylamino)benzophenone)	90-94-8									2.5E-04	8.6E-01	4/99			1
N-NITROSODI-n-BUTYLAMINE	924-16-3									3.1E-03	1.1E+01	4/99 [1/92]			1
N-NITROSODI-n-PROPYLAMINE	621-64-7									2.0E-03	7.0E+00	4/99 [1/91]			1
N-NITROSODIETHYLAMINE	55-18-5									1.0E-02	3.6E+01	4/99 [1/91]			1
N-NITROSODIMETHYLAMINE	62-75-9									4.6E-03	1.6E+01	4/99 [1/91]			1
N-NITROSODIPHENYLAMINE	86-30-6									2.6E-06	9.0E-03	4/99			1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
N-NITROSO-N-METHYLETHYLAMINE	10595-95-6									6.3E-03	2.2E+01	4/99 [7/90]			1
N-NITROSOMORPHOLINE	59-89-2									1.9E-03	6.7E+00	4/99 [7/92]			1
N-NITROSOPIPERIDINE	100-75-4									2.7E-03	9.4E+00	4/99 [7/92]			1
N-NITROSOPYRROLIDINE	930-55-2									6.0E-04	2.1E+00	4/99 [7/90]			1
NAPHTHALENE ... (see Polycyclic aromatic hydrocarbons)															
NICKEL AND COMPOUNDS[TAC] *values also apply to:*	7440-02-0 [1145]	2.0E-01	3/12	6.0E-02	3/12	1.4E-02	3/12	1.1E-02	3/12	2.6E-04 TAC	9.1E-01	8/91			1
Nickel acetate	*373-02-4*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.3321*
Nickel carbonate	*3333-67-3*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.4945*
Nickel carbonyl	*13463-39-3*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.3438*
Nickel hydroxide	*12054-48-7*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.6332*
Nickelocene	*1271-28-9*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.4937*
NICKEL OXIDE	1313-99-1	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	2.0E-02	3/12	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			0.7859
Nickel refinery dust from the pyrometallurgical process	*1146*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*1*
Nickel subsulfide	*12035-72-2*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.2443*
NITRIC ACID	7697-37-2	8.6E+01	4/99												1
NITROGEN DIOXIDE	10102-44-0	4.7E+02	4/99[1/92]												1
p-NITROSODIPHENYLAMINE	156-10-5									6.3E-06	2.2E-02	4/99			1
OZONE	10028-15-6	1.8E+02	4/99[1/92]												1
PARTICULATE EMISSIONS FROM DIESEL-FUELED ENGINES[TAC, i]	9901					5.0E+00 TAC	8/98			3.0E-04 TAC	1.1E+00	8/98			1
PENTACHLOROPHENOL ... (see Chlorophenols)															
PERCHLOROETHYLENE[TAC] (Tetrachloroethylene)	127-18-4	2.0E+04	4/99			3.5E+01 TAC	10/91			5.9E-06 TAC	2.1E-02	10/91			1
PHENOL	108-95-2	5.8E+03	4/99			2.0E+02	4/00								1
PHOSGENE	75-44-5	4.0E+00	4/99												1
PHOSPHINE	7803-51-2					8.0E-01	9/02								1
PHOSPHORIC ACID	7664-38-2					7.0E+00	2/00								1
PHTHALIC ANHYDRIDE	85-44-9					2.0E+01	1/01								1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
PCB (POLYCHLORINATED BIPHENYLS) (unspeciated mixture)[j]	1336-36-3									2.0E-05 [lowest risk] 1.1E-04 [low risk] 5.7E-04 [high risk]	7.0E-02 [lowest risk] 4.0E-01 [low risk] 2.0E+00 [high risk]	4/99	7.0E-02 [lowest risk] 4.0E-01 [low risk] 2.0E+00 [high risk]	10/00	1
PCB (POLYCHLORINATED BIPHENYLS (speciated)[k]															
3,3',4,4'-TETRACHLOROBIPHENYL (PCB 77)	32598-13-3					4.0E-01	8/03	1.0E-04	8/03	3.8E-03	1.3E+01	8/03	1.3E+01	8/03	1
3,4,4',5-TETRACHLOROBIPHENYL (PCB 81)	70362-50-4					1.3E-01	1/11	3.3E-05	1/11	1.1E-02	3.9E+01	1/11	3.9E+01	1/11	1
2,3,3',4,4'-PENTACHLOROBIPHENYL (PCB 105)	32598-14-4					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
2,3,4,4',5-PENTACHLOROBIPHENYL (PCB 114)	74472-37-0					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
2,3',4,4',5-PENTACHLOROBIPHENYL (PCB 118)	31508-00-6					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
2,3',4,4',5'-PENTACHLOROBIPHENYL (PCB 123)	65510-44-3					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
3,3',4,4',5-PENTACHLOROBIPHENYL (PCB 126)	57465-28-8					4.0E-04	8/03	1.0E-07	8/03	3.8E+00	1.3E+04	8/03	1.3E+04	8/03	1
2,3,3',4,4',5-HEXACHLOROBIPHENYL (PCB 156)	38380-08-4					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
2,3,3',4,4',5'-HEXACHLOROBIPHENYL (PCB 157)	69782-90-7					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
2,3',4,4',5,5'-HEXACHLOROBIPHENYL (PCB 167)	52663-72-6					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
3,3',4,4',5,5'-HEXACHLOROBIPHENYL (PCB 169)	32774-16-6					1.3E-03	1/11	3.3E-07	1/11	1.1E+00	3.9E+03	1/11	3.9E+03	1/11	1
2,3,3',4,4',5,5'-HEPTACHLOROBIPHENYL (PCB 189)	39635-31-9					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
POLYCHLORINATED DIBENZO-*P*-DIOXINS (PCDD) (Treated as 2,3,7,8-TCDD for HRA)[TAC, k]	1085 1086					4.0E-05	2/00	1.0E-08	10/00	3.8E+01 TAC	1.3E+05	8/86	1.3E+05 TAC	8/86	1
2,3,7,8-TETRACHLORODIBENZO-*P*-DIOXIN[TAC]	1746-01-6					4.0E-05	2/00	1.0E-08	10/00	3.8E+01 TAC	1.3E+05	8/86	1.3E+05 TAC	8/86	1
1,2,3,7,8-PENTACHLORODIBENZO-*P*-DIOXIN	40321-76-4					4.0E-05	8/03	1.0E-08	8/03	3.8E+01	1.3E+05	8/03	1.3E+05	8/03	1
1,2,3,4,7,8-HEXACHLORODIBENZO-*P*-DIOXIN	39227-28-6					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,6,7,8-HEXACHLORODIBENZO-*P*-DIOXIN	57653-85-7					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,7,8,9-HEXACHLORODIBENZO-*P*-DIOXIN	19408-74-3					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,4,6,7,8-HEPTACHLORODIBENZO-*P*-DIOXIN	35822-46-9					4.0E-03	2/00	1.0E-06	10/00	3.8E-01	1.3E+03	4/99	1.3E+03	10/00	1
1,2,3,4,6,7,8,9-OCTACHLORODIBENZO-*P*-DIOXIN	3268-87-9					1.3E-01	1/11	3.3E-05	1/11	1.1E-02	3.9E+01	1/11	3.9E+01	1/11	1
POLYCHLORINATED DIBENZOFURANS (PCDF)[TAC, k] (Treated as 2,3,7,8-TCDD for HRA)	1080					4.0E-05	2/00	1.0E-08	10/00	3.8E+01 TAC	1.3E+05	8/86	1.3E+05 TAC	8/86	1
2,3,7,8-TETRACHLORODIBENZOFURAN	5120-73-19					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,7,8-PENTACHLORODIBENZOFURAN	57117-41-6					1.3E-03	1/11	3.3E-07	1/11	1.1E+00	3.9E +03	1/11	3.9E +03	1/11	1
2,3,4,7,8-PENTACHLORODIBENZOFURAN	57117-31-4					1.3E-04	1/11	3.3E-08	1/11	1.1E+01	3.9E +04	1/11	3.9E +04	1/11	1
1,2,3,4,7,8-HEXACHLORODIBENZOFURAN	70648-26-9					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,6,7,8-HEXACHLORODIBENZOFURAN	57117-44-9					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,7,8,9-HEXACHLORODIBENZOFURAN	72918-21-9					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
2,3,4,6,7,8-HEXACHLORODIBENZOFURAN	60851-34-5					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,4,6,7,8-HEPTACHLORODIBENZOFURAN	67562-39-4					4.0E-03	2/00	1.0E-06	10/00	3.8E-01	1.3E+03	4/99	1.3E+03	10/00	1
1,2,3,4,7,8,9-HEPTACHLORODIBENZOFURAN	55673-89-7					4.0E-03	2/00	1.0E-06	10/00	3.8E-01	1.3E+03	4/99	1.3E+03	10/00	1
1,2,3,4,6,7,8,9-OCTACHLORODIBENZOFURAN	39001-02-0					1.3E-01	1/11	3.3E-05	1/11	1.1E-02	3.9E +01	1/11	3.9E +01	1/11	1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
POLYCYCLIC AROMATIC HYDROCARBON (PAH)[l] [Treated as B(a)P for HRA][l]	1150 1151									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
BENZ(A)ANTHRACENE[l]	56-55-3									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
BENZO(A)PYRENE[l]	50-32-8									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
BENZO(B)FLUORANTHENE[l]	205-99-2									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
BENZO(J)FLUORANTHENE[l]	205-82-3									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
BENZO(K)FLUORANTHENE[l]	207-08-9									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
CHRYSENE[l]	218-01-9									1.1E-05	3.9E-02	4/99 [4/94]	1.2E-01	10/00 [4/94]	1
DIBENZ(A,H)ACRIDINE[l]	226-36-8									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
DIBENZ(A,H)ANTHRACENE[l]	53-70-3									1.2E-03	4.1E+00	4/99 [4/94]	4.1E+00	10/00 [4/94]	1
DIBENZ(A,J)ACRIDINE[l]	224-42-0									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
DIBENZO(A,E)PYRENE[l]	192-65-4									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
DIBENZO(A,H)PYRENE[l]	189-64-0									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
DIBENZO(A,I)PYRENE[l]	189-55-9									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
DIBENZO(A,L)PYRENE[l]	191-30-0									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
7H-DIBENZO(C,G)CARBAZOLE[l]	194-59-2									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
7,12-DIMETHYLBENZ(A)ANTHRACENE[l]	57-97-6									7.1E-02	2.5E+02	4/99 [4/94]	2.5E+02	10/00 [4/94]	1
1,6-DINITROPYRENE[l]	42397-64-8									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
1,8-DINITROPYRENE[l]	42397-65-9									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
INDENO(1,2,3-C,D)PYRENE[l]	193-39-5									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
3-METHYLCHOLANTHRENE[l]	56-49-5									6.3E-03	2.2E+01	4/99 [4/94]	2.2E+01	10/00 [4/94]	1
5-METHYLCHRYSENE[l]	3697-24-3									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
NAPHTHALENE	91-20-3					9.0E+00	4/00			3.4E-05	1.2E-01	8/04			1
5-NITROACENAPHTHENE[l]	602-87-9									3.7E-05	1.3E-01	4/99 [4/94]	1.3E-01	10/00 [4/94]	1
6-NITROCHRYSENE[l]	7496-02-8									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
2-NITROFLUORENE[l]	607-57-8									1.1E-05	3.9E-02	4/99 [4/94]	1.2E-01	10/00 [4/94]	1
1-NITROPYRENE[l]	5522-43-0									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
4-NITROPYRENE[l]	57835-92-4									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
POTASSIUM BROMATE.... ... (see Bromine & Compounds)															
1,3-PROPANE SULTONE	1120-71-4									6.9E-04	2.4E+00	4/99			1
PROPYLENE (PROPENE)	115-07-1					3.0E+03	4/00								1
PROPYLENE GLYCOL MONOMETHYL ETHER	107-98-2					7.0E+03	2/00								1
PROPYLENE OXIDE	75-56-9	3.1E+03	4/99			3.0E+01	2/00			3.7E-06	1.3E-02	4/99 [7/90]			1
SELENIUM AND COMPOUNDS[m]	7782-49-2 [1170]					2.0E+01	12/01	5.0E-03	12/01						1
HYDROGEN SELENIDE	7783-07-5	5.0E+00	4/99												1
Selenium sulfide	*7446-34-6*					*2.0E+01*	*12/01*	*5.0E-03*	*12/01*						*1*
SILICA [CRYSTALLINE, RESPIRABLE]	1175					3.0E+00	2/05								1
SODIUM HYDROXIDE	1310-73-2	8.0E+00	4/99												1
STYRENE	100-42-5	2.1E+04	4/99			9.0E+02	4/00								1
SULFATES	9960	1.2E+02	4/99												1
SULFUR DIOXIDE	7446-09-5	6.6E+02	4/99[1/92]												1
SULFURIC ACID	7664-93-9	1.2E+02	4/99			1.0E+00	12/01								1
SULFUR TRIOXIDE	*7446-71-9*	*1.2E+02*	*4/99*			*1.0E+00*	*12/01*								*1*
OLEUM	8014-95-7	1.2E+02	4/99												1
1,1,2,2-TETRACHLOROETHANE	79-34-5									5.8E-05	2.0E-01	4/99			1
TETRACHLOROPHENOLS ... (see Chlorophenols)															
2,4,5-TRICHLOROPHENOL ... (see Chlorophenols)															
2,4,6-TRICHLOROPHENOL ... (see Chlorophenols)															
THIOACETAMIDE	62-55-5									1.7E-03	6.1E+00	4/99			1
TOLUENE	108-88-3	3.7E+04	4/99			3.0E+02	4/00								1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
Toluene diisocyantates	*26471-62-5*					*7.0E-02*	*1/01*			*1.1E-05*	*3.9E-02*	*4/99*			*1*
TOLUENE-2,4-DIISOCYANATE	584-84-9					7.0E-02	1/01			1.1E-05	3.9E-02	4/99			1
TOLUENE-2,6-DIISOCYANATE	91-08-7					7.0E-02	1/01			1.1E-05	3.9E-02	4/99			1
1,1,2-TRICHLOROETHANE (Vinyl trichloride)	79-00-5									1.6E-05	5.7E-02	4/99			1
TRICHLOROETHYLENE[TAC]	79-01-6					6.0E+02	4/00			2.0E-06 TAC	7.0E-03	10/90			1
TRIETHYLAMINE	121-44-8	2.8E+03	4/99			2.0E+02	9/02								1
URETHANE (Ethyl carbamate)	51-79-6									2.9E-04	1.0E+00	4/99 [7/90]			1
Vanadium Compounds	*N/A*														*1*
Vanadium (fume or dust)	*7440-62-2*	*3.0E+01*	*4/99*												*1*
VANADIUM PENTOXIDE	1314-62-1	3.0E+01	4/99												1
VINYL ACETATE	108-05-4					2.0E+02	12/01								1
VINYL CHLORIDE[TAC] (Chloroethylene)	75-01-4	1.8E+05	4/99							7.8E-05 TAC	2.7E-01	12/90			1
VINYLIDENE CHLORIDE (1,1-Dichloroethylene)	75-35-4					7.0E+01	1/01								1
XYLENES (mixed isomers)	1330-20-7	2.2E+04	4/99			7.0E+02	4/00								1
m-XYLENE	108-38-3	2.2E+04	4/99			7.0E+02	4/00								1
o-XYLENE	95-47-6	2.2E+04	4/99			7.0E+02	4/00								1
p-XYLENE	106-42-3	2.2E+04	4/99			7.0E+02	4/00								1

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Purpose: The purpose of this reference table is to provide a quick list of all health values that have been approved by the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) for use in facility health risk assessments conducted for the AB 2588 Air Toxics Hot Spots Program. The OEHHA has developed and adopted new risk assessment guidelines that update and replace the 2003 version of the OEHHA *Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments*. The OEHHA has adopted three technical support documents for these guidelines, which can be found on their website (http://www.oehha.ca.gov/air/hot_spots/index.html). This table lists the OEHHA adopted inhalation and oral cancer slope factors, noncancer acute Reference Exposure Levels (RELs), and inhalation and oral noncancer chronic RELs. OEHHA is still in the process of adopting new health values. Therefore, new health values will periodically be added to, or deleted from, this table. Users of this table are advised to monitor the OEHHA website (*www.oehha.ca.gov*) for any updates to the health values.

May 2008 update: The Air Resources Board adopted amendments to the AB 2588 Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines Regulation (Title 17, California Code of Regulations, Section 93300.5) on November 16, 2006. The amendments became effective on September 26, 2007, after approval from the Office of Administrative Law. Under the new amendments, the substances previously listed in Appendix A-I *(Substances For Which Emissions Must Be Quantified*) and Appendix F (*Criteria For Inputs For Risk Assessment Using Screening Air Dispersion Modeling*) of the ARB's *Air Toxics* "*Hot Spots" Emission Inventory Criteria and Guidelines (EICG) (July 1997)* have been removed from this table.

a The *italic* font used in this table clarify applicability of OEHHA adopted health effects values to individual or grouped substances listed in the *Air Toxics* "*Hot Spots" Emission Inventory Criteria and Guidelines*, Appendix A-I list of *"Substances For Which Emissions Must Be Quantified"*.

b Chemical Abstract Service Number (CAS): For chemical groupings and mixtures where a CAS number is not applicable, the 4-digit code used in the *Air Toxics* "*Hot Spots" Emission Inventory Criteria and Guidelines (EICG) Report* is listed. The 4-digit codes enclosed in brackets [ ] are codes that have been phased out, but may still appear on previously reported Hot Spots emissions. For information on the origin and use of the 4-digit code, see the EICG report.

c Date Value Reviewed [Added]: These columns list the date that the health value was last reviewed by OEHHA, and/or the Scientific Review Panel, and/or approved for use in the AB 2588 Air Toxics Hot Spots Program. If the health value is unchanged since it was first approved for use in the Hot Spots Program, then the date that the value was first approved for use by CAPCOA is listed within the brackets [ ].

- April 1999 is listed for the cancer potency values and noncancer acute RELs, which have been adopted by the OEHHA as part of the AB 2588 Hot Spot Risk Assessment Guidelines.
- February 2000, April 2000, January 2001, and December 2001 are listed for the first set of 22, the second set of 16, the third set of 22, and the fourth set of 12 noncancer chronic RELs, respectively. The chronic REL for carbon disulfide was adopted in May 2002. Chronic RELs for phosphine and triethylamine were adopted in September 2002. Chronic RELs for fluorides including hydrogen fluoride were adopted August 2003. Chronic REL for silica [crystalline respirable] was adopted February 2005.
- October 2000 is listed for the oral chronic RELs and oral cancer slope factors.
- Cancer potency value adopted for naphthalene in August 2004. The inhalation and oral cancer potency values for ethyl benzene were adopted in November 2007.
- For the substances identified as Toxic Air Contaminants, the Air Resources Board hearing date is listed. The dates for acetaldehyde, benzo[a]pyrene, and methyl tertiary-butyl ether represent the dates the values were approved by the Scientific Review Panel.
- On December 19, 2008, OEHHA adopted new acute, 8-hour, and chronic RELs for acetaldehyde, acrolein, arsenic, formaldehyde, manganese, and mercury. The most current health values can be found at: http://www.oehha.ca.gov/air/allrels.html.

Note: 1. We present the new oral RELs only in milligrams (mg/kg-d), although OEHHA has presented them in other tables in either micrograms (μg/kg-d) or milligrams.

2. All acute RELs use a 1-hour averaging period (OEHHA, 2008). RELs which were developed using earlier guidelines and specified a different averaging time are unchanged in concentration value, but now refer to the 1-hour averaging period. As of 8/1/2013, the affected chemicals are: benzene, carbon disulfide, carbon tetrachloride, chloroform, ethylene glycol monoethyl ether, ethylene glycol monoethyl ether acetate, and ethylene glycol monomethyl ether: These may be replaced by updated RELs following the OEHHA (2008) guidelines in due course.

3. At OEHHA's direction, the chronic oral REL for arsenic does not apply to arsine because arsine is a gas and not particle associated.

- OEHHA's adoption of the World Health Organization's 2005 Toxicity Equivalency Factors for polychlorinated dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and dioxin-like polychlorinated biphenyls (PCBs) occurred in January 2011. See Appendix C of OEHHA's *Air Toxics Hot Spots Program Technical Support Document for Cancer Potencies* at http://www.oehha.ca.gov/air/hot_spots/pdf/AppCdioxinTEFs013111.pdf for more information.
- On March 23, 2012, OEHHA adopted revised acute, 8-hour and chronic RELs for nickel and nickel compounds. The values of the RELs are listed in the table at: http://www.oehha.ca.gov/air/chronic_rels/032312CREL.html.
- On July 29, 2013, OEHHA adopted an acute and 8-hour REL, and a revised chronic REL for 1,3-butadiene. The REL values and summary can be found online at: http://www.oehha.ca.gov/air/hot_spots/index.html.
- On October 18, 2013 (February 2014 table update), OEHHA adopted acute, 8-hour, and chronic RELs for caprolactam. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/pdf/Caprolactam2013.pdf. Changes have been made to target organs to the following substances with no change to health factors: Chloroform, Diethanolamine, Fluorides and Hydrogen Fluoride, Methylene Chloride, Styrene, Xylenes. The "date added" in this table reflects the date of the health factor only.
- On June 27, 2014, OEHHA adopted a new 8-hour REL and revised acute and chronic RELs for benzene. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/BenzeneJune2014.html.

d Inhalation cancer potency factor: The "unit risk factor" has been replaced in the new risk assessment algorithms by a factor called the "inhalation cancer potency factor". Inhalation cancer potency factors are expressed as units of inverse dose [i.e., $(mg/kg\text{-}day)^{-1}$]. They were derived from unit risk factors [units = $(ug/m^3)^{-1}$] by assuming that a receptor weighs 70 kilograms and breathes 20 cubic meters of air per day. The inhalation potency factor is used to calculate a potential inhalation cancer risk using the new risk assessment algorithms defined in the OEHHA, *Air Toxics Hot Spots Program; Technical Support Document for Exposure Assessment and Stochastic Analysis (August 2012)*.

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

e	Molecular Weight Adjustment Factor: Molecular weight adjustment factors (MWAF) are only to be used when a toxic metal has a cancer potency factor. For most of the Hot Spots toxic metals, the OEHHA cancer potency factor applies to the weight of the toxic metal atom contained in the overall compound. Some of the Hot Spots compounds contain various elements along with the toxic metal atom (e.g., "Nickel hydroxide", CAS number 12054-48-7, has a formula of H_2NiO_2). Therefore, an adjustment to the reported pounds of the overall compound is needed before applying the OEHHA cancer potency factor for "Nickel and compounds" to such a compound. This ensures that the cancer potency factor is applied only to the fraction of the overall weight of the emissions that are associated with health effects of the metal. In other cases, the Hot Spots metals are already reported as the metal atom equivalent (e.g., CAS 7440-02-0, "Nickel"), and these cases do not use any further molecular weight adjustment. (Refer to Note [7] in Appendix A, List of Substances in the EICG Report for further information on how the emissions of various Hot Spots metal compounds are reported.) The appropriate molecular weight adjustment factors (MWAF) to be used along with the OEHHA cancer potency factors for Hot Spots metals can be found in the MWAF column of this table. So, for example, assume 100 pounds of "Nickel hydroxide" emissions are reported under CAS number 12054-48-7. To get the Nickel atom equivalent of these emissions, multiply by the listed MWAF (0.6332) for Nickel hydroxide: a. 100 pounds x 0.6332 = 63.32 pounds of Nickel atom equivalent *This step should be completed prior to applying the OEHHA cancer potency factor for "Nickel and compounds" in a calculation for a prioritization score or risk assessment calculation.* (For more information see Chapter 4, Section 4.2.1.1.1 of OEHHA's 2014 document, *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments*.) Note: The value listed in the MWAF column for Asbestos is not a molecular weight adjustment. This is a conversion factor for adjusting mass to fibers or structures. See Appendix C of OEHHA's document *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (2014)* for more information on Asbestos, or see the EICG report for reporting guidance. Also see the Asbestos footnote (designated by the letter f)
TAC	Toxic Air Contaminant: The Air Resources Board has identified this substance as a Toxic Air Contaminant.
f	Asbestos: The units for the Inhalation Cancer Potency factor for asbestos are $(100 \text{ PCM fibers/m}^3)^{-1}$. A conversion factor of 100 fibers/0.003 µg can be multiplied by a receptor concentration of asbestos expressed in $\mu g/m^3$. Unless other information necessary to estimate the concentration ($fibers/m^3$) of asbestos at receptors of interest is available. A unit risk factor of $1.9 \text{ E } 10^{-4}$ $(\mu g/m^3)^{-1}$ and an inhalation cancer potency factor of $2.2 \text{ E } 10^{+2}$ $(\text{mg/kg BW} * \text{day})^{-1}$ are available. For more information on asbestos quantity conversion factors, see Appendix F of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II; Technical Support Document for Cancer Potency Factors (May 2009)*, and Appendix C of OEHHA's document *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (2014)*.
g	Hexavalent Chromium: In July 2011, OEHHA developed the oral cancer slope factor for chromium 6+ and compounds for the California Public Health Goal in drinking water. As of February 2014, OEHHA states it should also be used for the Hot Spots program.
h	Inorganic Lead: Inorganic Lead was identified by the Air Resources Board as a Toxic Air Contaminant in April 1997. Since information on noncancer health effects show no identified threshold, no Reference Exposure Level has been developed. The document, *Risk Management Guidelines for New, Modified, and Existing Sources of Lead, March 2001*, has been developed by ARB and OEHHA staff for assessing noncancer health impacts from sources of lead. See Appendix F of OEHHA's document *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments* (2014) for an overview of how to evaluate noncancer impacts from exposure to lead using these risk management guidelines.
i	Particulate Emissions from Diesel-Fueled Engines: The inhalation cancer potency factor was derived from whole diesel exhaust and should be used only for impacts from the inhalation pathway (based on diesel PM measurements). The inhalation impacts from speciated emissions from diesel-fueled engines are already accounted for in the inhalation cancer potency factor. However, at the discretion of the risk assessor, speciated emissions from diesel-fueled engines may be used to estimate acute noncancer health impacts or the contribution to cancer risk or chronic noncancer health impacts for the non-inhalation exposure pathway. See Appendix D of OEHHA's document *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (2014)* for more information. The noncancer chronic REL for diesel exhaust is based on assumptions of contributions of diesel PM to ambient PM. It should be used with diesel PM measurement.
j	Cancer Potency Factors (CPFs) for unspeciated mixtures of Polychlorinated Biphenyls: High Risk: For use in cases where congeners with more than four chlorines comprise more than one-half percent of total polychlorinated biphenyls. Use as default CPF for Tier 1 assessments. Low Risk: This number would not ordinarily be used in the Hot Spots program. Lowest Risk: For use in cases where congeners with more than four chlorines comprise less than one-half percent of total polychlorinated biphenyls. As of February, 2014, there is no approved method that can be used to assess the noncancer hazard of an unspeciated PCB mixture. Persons preparing HRAs for the Hot Spots Program should consult with OEHHA and the local Air Pollution Control or Air Quality Management District if an assessment of the noncancer hazard for unspeciated PCB mixtures is needed.
k	Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans (also referred to as chlorinated dioxins and dibenzofurans) and dioxin-like PCB congeners: The OEHHA has adopted the World Health Organization 2005 (WHO-05) Toxicity Equivalency Factor scheme for evaluating the risk due to exposure to samples containing mixtures of polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) and a number of dioxin-like PCB congeners. See Appendix A of OEHHA's Technical Support Document For Describing Available Cancer Potency Factors for more information about the scheme. See Appendix E of OEHHA's 2014 The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments for the methodology for calculating 2,3,7,8-equivalents, cancer risk, and noncancer Hazard Index for PCDD, PCDFs and a number of dioxin-like PCB congeners. The two numbers (i.e., 1085 and 1086) in the column listing Chemical Abstracts Numbers are used for reporting and risk assessment purposes. Be sure to input emissions under the proper code when using the HARP software. ID code 1085 has no health values associated with it in the HARP software; therefore, no health impacts will be calculated when using ID 1085. See the Emissions Inventory Criteria and Guidelines for more information on reporting emissions.
l	Polycyclic Aromatic Hydrocarbons (PAHs): These substances are PAH or PAH-derivatives that have OEHHA-developed Potency Equivalency Factors (PEFs) which were approved by the Scientific Review Panel in April 1994 (see ARB document entitled *Benzo[a]pyrene as a Toxic Air Contaminant*). PAH inhalation slope factors listed here have been adjusted by the PEFs. See OEHHA's Technical Support Document: Methodologies for Derivation, Listing of Available Values, and Adjustments to Allow for Early Life Exposures (2009) for more information about the scheme. Appendix G of OEHHA's *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (2014)* also contains information on PAHs. The two numbers (i.e., 1150 and 1151) in the column listing Chemical Abstracts Numbers are used for reporting and risk assessment purposes. Be sure to input emissions under the proper code when using the HARP software. ID code 1150 has no health values associated with it in the HARP software; therefore, no health impacts will be calculated when using ID 1150. See the Emissions Inventory Criteria and Guidelines for more information on reporting emissions.

Table 1: CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

m	SELENIUM AND COMPOUNDS: In February 2014, an oral REL was added to the consolidated table. The REL was adopted in Dec 2001, but could not be used by the Hot Spots Program (or HARP software) until transfer factors for the oral and dermal routes were adopted. Transfer factors are included in the OEHHA's Technical Support Document for Exposure Assessment and Stochastic Analysis (August 2012) and will be added to the HARP software in the future.
N/A	Not Applicable.
Other Changes:	
	• 10/18/2010, removed CHLORODIFLUOROMETHANE, which should have been removed in May 2008.
	February 2014:
	• Removed applicability of oleum to the sulfuric acid chronic inhalation REL because oleum represents only an acute health hazard. • Removed "METHYL MERCURY (see Mercury & Compounds)" entry because methyl mercury has different chemical properties, potency, and toxicity compared to elemental mercury and mercury salts, and it is not emitted directly from any California facilities.

Table 2: OEHHA/ARB APPROVED ACUTE REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Service Number (CAS)	Acute REL ($\mu g/m^3$)	Date[c] Value Reviewed	Target Organs								
				Alimentary	Cardiovascular	Reproductive/[d] Development	Eye	Hematologic	Immune	Nervous	Respiratory	Skin
ACETALDEHYDE	75-07-0	4.7E+02	12/08				X				X	
ACROLEIN	107-02-8	2.5E+00	12/08				X				X	
ACRYLIC ACID	79-10-7	6.0E+03	4/99				X				X	
AMMONIA	7664-41-7	3.2E+03	4/99				X				X	
ARSENIC AND COMPOUNDS (INORGANIC)[TAC]	7440-38-2 1016 [1015]	2.0E-01	12/08		X	X				X		
ARSINE	7784-42-1	2.0E-01	12/08		X	X				X		
BENZENE[TAC]	71-43-2	2.7E+01	6/14			X		X	X			
BENZYL CHLORIDE	100-44-7	2.4E+02	4/99				X				X	
1,3-BUTADIENE[TAC]	106-99-0	6.6E+02	7/13			X						
CAPROLACTAM	105-60-2	5.0E+01	10/13				X					
CARBON DISULFIDE	75-15-0	6.2E+03	4/99			X				X		
CARBON MONOXIDE	630-08-0	2.3E+04	4/99		X							
CARBON TETRACHLORIDE[TAC] (Tetrachloromethane)	56-23-5	1.9E+03	4/99	X		X				X		
CHLORINE	7782-50-5	2.1E+02	4/99				X				X	
CHLOROFORM[TAC]	67-66-3	1.5E+02	4/99			X				X	X	
CHLOROPICRIN	76-06-2	2.9E+01	4/99				X				X	
COPPER AND COMPOUNDS	7440-50-8 [1067]	1.0E+02	4/99								X	
Cyanide Compounds (inorganic)	*57-12-5 1073*	*3.4E+02*	*4/99*							✓		
HYDROGEN CYANIDE (Hydrocyanic acid)	74-90-8	3.4E+02	4/99							X		
1,4-DIOXANE (1,4-Diethylene dioxide)	123-91-1	3.0E+03	4/99				X				X	
EPICHLOROHYDRIN (1-Chloro-2,3-epoxypropane)	106-89-8	1.3E+03	4/99				X				X	
Fluorides and Compounds	*1101*	*2.4E+02*	*4/99*				✓				✓	
HYDROGEN FLUORIDE (Hydrofluoric acid)	7664-39-3	2.4E+02	4/99				X				X	
FORMALDEHYDE[TAC]	50-00-0	5.5E+01	12/08				X					

Table 2: OEHHA/ARB APPROVED ACUTE REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Service Number (CAS)	Acute REL ($\mu g/m^3$)	Date[c] Value Reviewed	Target Organs								
				Alimentary	Cardiovascular	Reproductive/[d] Development	Eye	Hematologic	Immune	Nervous	Respiratory	Skin
GLYCOL ETHERS	1115											
ETHYLENE GLYCOL BUTYL ETHER – EGBE	111-76-2	1.4E+04	4/99				X				X	
ETHYLENE GLYCOL ETHYL ETHER – EGEE	110-80-5	3.7E+02	4/99 [1/92]			X						
ETHYLENE GLYCOL ETHYL ETHER ACETATE - EGEEA	111-15-9	1.4E+02	4/99			X				X		
ETHYLENE GLYCOL METHYL ETHER – EGME	109-86-4	9.3E+01	4/99			X						
HYDROCHLORIC ACID (Hydrogen chloride)	7647-01-0	2.1E+03	4/99				X				X	
HYDROGEN CYANIDE (Hydrocyanic acid) (see Cyanide Compounds)												
HYDROGEN FLUORIDE (Hydrofluoric acid) (see Fluorides & Compounds)												
HYDROGEN SELENIDE (see Selenium & Compounds)												
HYDROGEN SULFIDE	7783-06-4	4.2E+01	4/99 [7/90]							X		
ISOPROPYL ALCOHOL (Isopropanol)	67-63-0	3.2E+03	4/99				X				X	
MERCURY AND COMPOUNDS (INORGANIC)	7439-97-6 [1133]	6.0E-01	12/08			X				X		
Mercuric chloride	*7487-94-7*	*6.0E-01*	*12/08*			✓				✓		
METHANOL	67-56-1	2.8E+04	4/99							X		
METHYL BROMIDE (Bromomethane)	74-83-9	3.9E+03	4/99			X				X	X	
METHYL CHLOROFORM (1,1,1-Trichloroethane)	71-55-6	6.8E+04	4/99							X		
METHYL ETHYL KETONE (2-Butanone)	78-93-3	1.3E+04	4/99				X				X	
METHYLENE CHLORIDE[TAC] (Dichloromethane)	75-09-2	1.4E+04	4/99		X					X		
NICKEL AND COMPOUNDS[TAC]	7440-02-0 [1145]	2.0E-01	3/12						X			
Nickel acetate	*373-02-4*	*2.0E-01*	*3/12*						✓			
Nickel carbonate	*3333-67-3*	*2.0E-01*	*3/12*						✓			
Nickel carbonyl	*13463-39-3*	*2.0E-01*	*3/12*						✓			
Nickel hydroxide	*12054-48-7*	*2.0E-01*	*3/12*						✓			
Nickelocene	*1271-28-9*	*2.0E-01*	*3/12*						✓			
NICKEL OXIDE	*1313-99-1*	*2.0E-01*	*3/12*						✓			

Table 2: OEHHA/ARB APPROVED ACUTE REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Service Number (CAS)	Acute REL ($\mu g/m^3$)	Date[c] Value Reviewed	Target Organs								
				Alimentary	Cardiovascular	Reproductive/ Development[d]	Eye	Hematologic	Immune	Nervous	Respiratory	Skin
Nickel refinery dust from the pyrometallurgical process	*1146*	*2.0E-01*	*3/12*						✓			
Nickel subsulfide	*12035-72-2*	*2.0E-01*	*3/12*						✓			
NITRIC ACID	7697-37-2	8.6E+01	4/99								X	
NITROGEN DIOXIDE	10102-44-0	4.7E+02	4/99 [1/92]								X	
OZONE	10028-15-6	1.8E+02	4/99 [1/92]				X				X	
PERCHLOROETHYLENE[TAC] (Tetrachloroethylene)	127-18-4	2.0E+04	4/99				X			X	X	
PHENOL	108-95-2	5.8E+03	4/99				X				X	
PHOSGENE	75-44-5	4.0E+00	4/99								X	
PROPYLENE OXIDE	75-56-9	3.1E+03	4/99			X	X				X	
Selenium and Compounds	*7782-49-2 [1170]*											
HYDROGEN SELENIDE	7783-07-5	5.0E+00	4/99				X				X	
SODIUM HYDROXIDE	1310-73-2	8.0E+00	4/99				X				X	X
STYRENE	100-42-5	2.1E+04	4/99			X	X				X	
SULFATES	9960	1.2E+02	4/99								X	
SULFUR DIOXIDE	7446-09-5	6.6E+02	4/99 [1/92]								X	
SULFURIC ACID	7664-93-9	1.2E+02	4/99								X	
SULFUR TRIOXIDE	*7446-71-9*	*1.2E+02*	*4/99*								✓	
OLEUM	8014-95-7	1.2E+02	4/99								X	
TOLUENE	108-88-3	3.7E+04	4/99			X	X			X	X	
TRIETHYLAMINE	121-44-8	2.8E+03	4/99				X			X		
Vanadium Compounds	*N/A*											
Vanadium (fume or dust)	*7440-62-2*	*3.0E+01*	*4/99*				✓				✓	
VANADIUM PENTOXIDE	1314-62-1	3.0E+01	4/99				X				X	
VINYL CHLORIDE[TAC] (Chloroethylene)	75-01-4	1.8E+05	4/99				X			X	X	
XYLENES (mixed isomers)	1330-20-7	2.2E+04	4/99				X			X	X	

Table 2: OEHHA/ARB APPROVED ACUTE REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Service Number (CAS)	Acute REL ($\mu g/m^3$)	Date[c] Value Reviewed	Target Organs								
				Alimentary	Cardiovascular	Reproductive/[d] Development	Eye	Hematologic	Immune	Nervous	Respiratory	Skin
m-Xylene	108-38-3	2.2E+04	4/99				X			X	X	
o-Xylene	95-47-6	2.2E+04	4/99				X			X	X	
p-Xylene	106-42-3	2.2E+04	4/99				X			X	X	

Purpose: The purpose of this reference table is to provide a quick list of all health values that have been approved by the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) for use in facility health risk assessments conducted for the AB 2588 Air Toxics "Hot Spots" Program. The OEHHA has developed and adopted new risk assessment guidelines that update and replace the 2003 version of the OEHHA *Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments*. The OEHHA has adopted three technical support documents for these guidelines, which can be found on their website (http://www.oehha.ca.gov/air/hot_spots/index.html). This table lists the OEHHA adopted noncancer acute Reference Exposure Levels (RELs). OEHHA is still in the process of adopting new health values. Therefore, new health values will periodically be added to, or deleted from, this table. Users of this table are advised to monitor the OEHHA website (*www.oehha.ca.gov*) for any updates to the health values.

May 2008 update: The Air Resources Board adopted amendments to the AB 2588 Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines Regulation (Title 17, California Code of Regulations, Section 93300.5) on November 16, 2006. The amendments became effective on September 26, 2007, after approval from the Office of Administrative Law. Under the new amendments, the substances previously listed in Appendix A-I *(Substances For Which Emissions Must Be Quantified*) and Appendix F (*Criteria For Inputs For Risk Assessment Using Screening Air Dispersion Modeling*) of the ARB's *Air Toxics* "*Hot Spots" Emission Inventory Criteria and Guidelines (EICG) (July 1997)* have been removed from this table.

a The checkmarks included in this table clarify applicability of OEHHA adopted heath effects values to individual or grouped substances listed in the *Air Toxics* "*Hot Spots" Emission Inventory Criteria and Guidelines*, Appendix A-I list of *"Substances For Which Emissions Must Be Quantified"*.

b Chemical Abstract Service Number (CAS): For chemical groupings and mixtures where a CAS number is not applicable, the 4-digit code used in the *Air Toxics* "*Hot Spots" Emission Inventory Criteria and Guidelines (EICG) Report* is listed. The 4-digit codes enclosed in brackets [ ] are codes that have been phased out, but may still appear on previously reported Hot Spots emissions. For information on the origin and use of the 4-digit code, see the EICG report.

Table 2: OEHHA/ARB APPROVED ACUTE REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

c	Date Value Reviewed [Added]: This column lists the date that the health value was last reviewed by OEHHA and the Scientific Review Panel, and/or approved for use in the AB 2588 Air Toxics Hot Spots Program. If the health value is unchanged since it was first approved for use in the "Hot Spots" Program, then the date that the value was first approved for use by CAPCOA is listed within the brackets [ ]. • April 1999 is listed for the noncancer acute RELs which have been adopted by the OEHHA as part of the AB 2588 Hot Spot Risk Assessment Guidelines. • On December 19, 2008, OEHHA adopted new acute RELs for acetaldehyde, acrolein, arsenic, formaldehyde, and mercury. The most current health values can be found at: http://www.oehha.ca.gov/air/allrels.html. Note: All acute RELs use a 1-hour averaging period (OEHHA, 2008). RELs which were developed using earlier guidelines and specified a different averaging time are unchanged in concentration value, but now refer to the 1-hour averaging period. As of 8/1/2013, the affected chemicals are: benzene, carbon disulfide, carbon tetrachloride, chloroform, ethylene glycol monoethyl ether, ethylene glycol monoethyl ether acetate, and ethylene glycol monomethyl ether: These may be replaced by updated RELs following the OEHHA (2008) guidelines in due course. • On March 23, 2012, OEHHA adopted revised acute, 8-hour and chronic RELs for nickel and nickel compounds. The values of the RELs are listed in the table at: http://www.oehha.ca.gov/air/chronic_rels/032312CREL.html. • On July 29, 2013, OEHHA adopted an acute and an 8-hour REL and a revised chronic REL for 1,3-butadiene. The REL value and summary can be found online at: http://www.oehha.ca.gov/air/hot_spots/index.html. • On October 18, 2013 (February 2014 table update), OEHHA adopted acute, 8-hour, and chronic RELs for caprolactam. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/pdf/Caprolactam2013.pdf. Changes have been made to target organs to the following substances with no change to health factors: Chloroform, Methylene Chloride, Styrene, and Xylenes. The "date added" in this table reflects the date of the health factor only. See footnotes below that discuss changes to substance target organs only. • On June 27, 2014, OEHHA adopted a new 8-hour REL and revised acute and chronic RELs for benzene. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/BenzeneJune2014.html
d	February 2014. Per OEHHA's current policy, substances with Reproductive System and/or Development as the hazard Index target organ(s) are represented under the single endpoint "Reproductive/Development"
TAC	Toxic Air Contaminant: The Air Resources Board has identified this substance as a Toxic Air Contaminant.
N/A	Not Applicable.

Other Changes:

February 2014 corrections based on original REL summaries:

- Chloroform – added respiratory system as a target organ.
- Methylene chloride – the cardiovascular system was added as a target organ.
- Entry of SULFURIC ACID AND OLEUM is removed to be consistent with Consolidated Table 1. This entry is removed from Table 1 because oleum represents only an acute health hazard.
- Styrene – added reproductive/development as a target organ.
- Xylenes – add nervous system as a target organ.

Table 3: OEHHA/ARB APPROVED 8-HOUR REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Number	8-Hour Inhalation REL ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Target Organs											
				Alimentary	Bone and Teeth	Cardiovascular	Reproductive/[d] Development	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Respiratory	Skin
ACETALDEHYDE	75-07-0	3.0E+02	12/08											X	
ACROLEIN	107-02-8	7.0E-01	12/08											X	
ARSENIC AND COMPOUNDS (INORGANIC)[TAC]	7440-38-2 1016	1.5E-02	12/08			X	X						X	X	X
ARSINE	7784-42-1	1.5E-02	12/08			X	X						X	X	X
BENZENE[TAC]	71-43-2	3.0E+00	6/14							X					
1,3-BUTADIENE[TAC]	106-99-0	9.0E+00	7/13				X								
CAPROLACTAM	105-60-2	7.0E+00	10/13											X	
FORMALDEHYDE[TAC]	50-00-0	9.0E+00	12/08											X	
MANGANESE AND COMPOUNDS	7439-96-5 [1132]	1.7E-01	12/08										X		
MERCURY AND COMPOUNDS (INORGANIC)	7439-97-6 [1133]	6.0E-02	12/08				X					X	X		
Mercuric chloride	*7487-94-7*	*6.0E-02*	*12/08*				✓					✓	✓		
NICKEL AND COMPOUNDS[TAC]	7440-02-0 [1145]	6.0E-02	3/12								X			X	
Nickel acetate	*373-02-4*	*6.0E-02*	*3/12*								✓			✓	
Nickel carbonate	*3333-67-3*	*6.0E-02*	*3/12*								✓			✓	
Nickel carbonyl	*13463-39-3*	*6.0E-02*	*3/12*								✓			✓	
Nickel hydroxide	*12054-48-7*	*6.0E-02*	*3/12*								✓			✓	
Nickelocene	*1271-28-9*	*6.0E-02*	*3/12*								✓			✓	
NICKEL OXIDE	*1313-99-1*	*6.0E-02*	*3/12*								✓			✓	
Nickel refinery dust from the pyrometallurgical process	*1146*	*6.0E-02*	*3/12*								✓			✓	
Nickel subsulfide	*12035-72-2*	*6.0E-02*	*3/12*								✓			✓	

Table 3: OEHHA/ARB APPROVED 8-HOUR REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Purpose:	The purpose of this reference table is to provide a quick list of all health values that have been approved by the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB). The OEHHA has developed and adopted new risk assessment guidelines that update and replace the 2003 version of the OEHHA *Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments*. The OEHHA has adopted three technical support documents for these guidelines, which can be found on their website (http://www.oehha.ca.gov/air/hot_spots/index.html). This table lists the OEHHA adopted 8-hour RELs. The methodology for the development and use of 8-hour RELs in Health Risk Assessments can be found in the OEHHA 2008 document *Air Toxics Hot Spots Program Technical Support Document for the Derivation of Noncancer Reference Exposure Levels* online at: http://oehha.ca.gov/air/hot_spots/rels_dec2008.html. OEHHA is still in the process of adopting new health values. Therefore, new health values will periodically be added to, or deleted from, this table. Users of this table are advised to monitor the OEHHA website (*www.oehha.ca.gov*) for any updates to the health values.
a	The checkmarks included in this table clarify applicability of OEHHA adopted health effects values to individual or grouped substances listed in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines*, Appendix A-I list of *"Substances For Which Emissions Must Be Quantified"*.
b	Chemical Abstract Service Number (CAS): For chemical groupings and mixtures where a CAS number is not applicable, the 4-digit code used in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG) Report* is listed. The 4-digit codes enclosed in brackets [ ] are codes that have been phased out, but may still appear on previously reported Hot Spots emissions. For information on the origin and use of the 4-digit code, see the EICG report.
c	Date Value Reviewed [Added]: This column lists the date that the health value was last reviewed by OEHHA and the Scientific Review Panel, and/or approved for use in the AB 2588 Air Toxics Hot Spots Program. If the health value is unchanged since it was first approved for use in the "Hot Spots" Program, then the date that the value was first approved for use by CAPCOA is listed within the brackets [ ]. • On December 19, 2008, OEHHA adopted new 8-hour RELs for acetaldehyde, acrolein, arsenic, formaldehyde, manganese, and mercury. The most current health values can be found at: http://www.oehha.ca.gov/air/allrels.html. • On March 23, 2012, OEHHA adopted revised acute, 8-hour and chronic RELs for nickel and nickel compounds. The values of the RELs are listed in the table at: http://www.oehha.ca.gov/air/chronic_rels/032312CREL.html. • On July 29, 2013, OEHHA adopted an acute and an 8-hour REL and a revised chronic REL for 1,3-butadiene. The REL value and summary can be found online at: http://www.oehha.ca.gov/air/hot_spots/index.html. • On October 18, 2013, OEHHA adopted acute, 8-hour, and chronic RELs for caprolactam. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/pdf/Caprolactam2013.pdf. • On June 27, 2014, OEHHA adopted a new 8-hour REL and revised acute and chronic RELs for benzene. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/BenzeneJune2014.html.
d	February 2014. Per OEHHA's current policy, substances with Reproductive System and/or Development as the hazard Index target organ(s) are represented under the single endpoint "Reproductive/Development".
TAC	Toxic Air Contaminant: The Air Resources Board has identified this substance as a Toxic Air Contaminant.

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Number	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL (mg/kg-d)	Date[c] Value Reviewed [Added]	Target Organs											
					Alimentary	Bone and Teeth	Cardiovascular	Reproductive/[d] Development	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Respiratory	Skin
ACETALDEHYDE	75-07-0	1.4E+02		12/08											X	
ACROLEIN	107-02-8	3.5E-01		12/08											X	
ACRYLONITRILE	107-13-1	5.0E+00		12/01											X	
AMMONIA	7664-41-7	2.0E+02		2/00											X	
ARSENIC AND COMPOUNDS (INORGANIC)[TAC]	7440-38-2 1016 [1015]	1.5E-02		12/08			X	X						X	X	X
			3.5E-06	12/08			X	X						X	X	X
ARSINE	7784-42-1	1.5E-02		12/08			X	X						X	X	X
BENZENE[TAC]	71-43-2	3.0E+00		6/14							X					
BERYLLIUM AND COMPOUNDS	7440-41-7 [1021]	7.0E-03		12/01								X			X	
			2.0E-03	12/01	X											
1,3-BUTADIENE[TAC]	106-99-0	2.0E+00		7/13				X								
CADMIUM AND COMPOUNDS[TAC]	7440-43-9 [1045]	2.0E-02		1/01									X		X	
			5.0E-04	10/00									X			
CAPROLACTAM	105-60-2	2.2E+00		10/13											X	
CARBON DISULFIDE	75-15-0	8.0E+02		5/02				X						X		
CARBON TETRACHLORIDE[TAC] (Tetrachloromethane)	56-23-5	4.0E+01		1/01	X			X						X		
CHLORINE	7782-50-5	2.0E-01		2/00											X	
CHLORINE DIOXIDE	10049-04-4	6.0E-01		1/01											X	
CHLOROBENZENE	108-90-7	1.0E+03		1/01	X			X					X			
CHLOROFORM[TAC]	67-66-3	3.0E+02		4/00	X			X					X			
CHLOROPICRIN	76-06-2	4.0E-01		12/01											X	
CHROMIUM 6+[TAC]	18540-29-9	2.0E-01		1/01											X	
			2.0E-02	10/00							X					
Barium chromate	*10294-40-3*	*2.0E-01*		*1/01*											✓	
			2.0E-02	*10/00*							✓					
Calcium chromate	*13765-19-0*	*2.0E-01*		*1/01*											✓	
			2.0E-02	*10/00*							✓					
Lead chromate	*7758-97-6*	*2.0E-01*		*1/01*											✓	

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Number	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL (mg/kg-d)	Date[c] Value Reviewed [Added]	Target Organs											
					Alimentary	Bone and Teeth	Cardiovascular	Reproductive/[d] Development	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Respiratory	Skin
			2.0E-02	*10/00*							✓					
Sodium dichromate	*10588-01-9*	*2.0E-01*		*1/01*											✓	
			2.0E-02	*10/00*							✓					
Strontium chromate	*7789-06-2*	*2.0E-01*		*1/01*											✓	
			2.0E-02	*10/00*							✓					
CHROMIUM TRIOXIDE (as chromic acid mist)	1333-82-0	2.0E-03		1/01											X	
			2.0E-02	*10/00*							✓					
CRESOLS (mixtures of)	1319-77-3	6.0E+02		1/01										X		
m-CRESOL	108-39-4	6.0E+02		1/01										X		
o-CRESOL	95-48-7	6.0E+02		1/01										X		
p-CRESOL	106-44-5	6.0E+02		1/01										X		
Cyanide Compounds (inorganic)	*57-12-5 1073*	*9.0E+00*		*4/00*			✓		✓					✓		
HYDROGEN CYANIDE (Hydrocyanic acid)	74-90-8	9.0E+00		4/00			X		X					X		
p-DICHLOROBENZENE	106-46-7	8.0E+02		1/01	X								X	X	X	
1,1,-DICHLOROETHYLENE … (see Vinylidene Chloride)																
DIESEL EXHAUST … (see Particulate Emissions from Diesel-Fueled Engines)																
DIETHANOLAMINE	111-42-2	3.0E+00		12/01							X				X	
N,N-DIMETHYL FORMAMIDE	68-12-2	8.0E+01		1/01	X										X	
1,4-DIOXANE; (1,4-Diethylene dioxide)	123-91-1	3.0E+03		4/00	X		X						X			
EPICHLOROHYDRIN (1-Chloro-2,3-epoxypropane)	106-89-8	3.0E+00		1/01						X					X	
1,2-EPOXYBUTANE	106-88-7	2.0E+01		1/01			X								X	
ETHYL BENZENE	100-41-4	2.0E+03		2/00	X			X	X				X			
ETHYL CHLORIDE (Chlorethane)	75-00-3	3.0E+04		4/00	X			X								
ETHYLENE DIBROMIDE[TAC] (1,2-Dibromoethane)	106-93-4	8.0E-01		12/01				X								
ETHYLENE DICHLORIDE[TAC] (1,2-Dichloroethane)	107-06-2	4.0E+02		1/01	X											
ETHYLENE GLYCOL	107-21-1	4.0E+02		4/00				X					X		X	

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Number	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL (mg/kg-d)	Date[c] Value Reviewed [Added]	Target Organs											
					Alimentary	Bone and Teeth	Cardiovascular	Reproductive/ Development[d]	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Respiratory	Skin
ETHYLENE OXIDE[TAC] (1,2-Epoxyethane)	75-21-8	3.0E+01		1/01										X		
Fluorides	1101	1.3E+01				X									X	
			4.0E-02	8/03		X										
HYDROGEN FLUORIDE (Hydrofluoric acid)	7664-39-3	1.4E+01				X									X	
			4.0E-02	8/03		X										
FORMALDEHYDE[TAC]	50-00-0	9.0E+00		12/08											X	
GLUTARALDEHYDE	111-30-8	8.0E-02		1/01											X	
GLYCOL ETHERS	1115															
ETHYLENE GLYCOL ETHYL ETHER – EGEE	110-80-5	7.0E+01		2/00				X			X					
ETHYLENE GLYCOL ETHYL ETHER ACETATE - EGEEA	111-15-9	3.0E+02		2/00				X								
ETHYLENE GLYCOL METHYL ETHER – EGME	109-86-4	6.0E+01		2/00				X								
ETHYLENE GLYCOL METHYL ETHER ACETATE - EGMEA	110-49-6	9.0E+01		2/00				X								
n-HEXANE	110-54-3	7.0E+03		4/00										X		
HYDRAZINE	302-01-2	2.0E-01		1/01	X				X							
HYDROCHLORIC ACID (Hydrogen chloride)	7647-01-0	9.0E+00		2/00											X	
HYDROGEN CYANIDE (Hydrocyanic acid) (see Cyanide Compounds)																
HYDROGEN BROMIDE ... (see Bromine & Compounds)																
HYDROGEN FLUORIDE (Hydrofluoric acid) (see Fluorides & Compounds)																
HYDROGEN SULFIDE	7783-06-4	1.0E+01		4/00											X	
ISOPHORONE	78-59-1	2.0E+03		12/01	X			X								
ISOPROPYL ALCOHOL (Isopropanol)	67-63-0	7.0E+03		2/00				X					X			
LINDANE ... (see gamma-Hexachlorocyclohexane)																
MALEIC ANHYDRIDE	108-31-6	7.0E-01		12/01											X	
MANGANESE AND COMPOUNDS	7439-96-5 [1132]	9.0E-02		12/08										X		

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Number	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL (mg/kg-d)	Date[c] Value Reviewed [Added]	Target Organs											
					Alimentary	Bone and Teeth	Cardiovascular	Reproductive/ Development[d]	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Respiratory	Skin
MERCURY AND INORGANIC COMPOUNDS	7439-97-6 [1133]	3.0E-02		12/08				X					X	X		
			1.6E-04	12/08				X					X	X		
Mercuric chloride	*7487-94-7*	*3.0E-02*		*12/08*				✓					✓	✓		
			1.6E-04	*12/08*				✓					✓	✓		
METHANOL	67-56-1	4.0E+03		4/00				X								
METHYL BROMIDE (Bromomethane)	74-83-9	5.0E+00		2/00				X						X	X	
METHYL tertiary-BUTYL ETHER	1634-04-4	8.0E+03		2/00	X					X			X			
METHYL CHLOROFORM (1,1,1-Trichloroethane)	71-55-6	1.0E+03		2/00										X		
METHYL ISOCYANATE	624-83-9	1.0E+00		12/01				X							X	
METHYLENE CHLORIDE[TAC] (Dichloromethane)	75-09-2	4.0E+02		2/00			X							X		
4,4'-METHYLENE DIANILINE (AND ITS DICHLORIDE)	101-77-9	2.0E+01		12/01	X					X						
METHYLENE DIPHENYL ISOCYANATE	101-68-8	7.0E-01		1/01											X	
NAPHTHALENE	91-20-3	9.0E+00		4/00											X	
NICKEL AND COMPOUNDS[TAC]	7440-02-0 [1145]	1.4E-02		3/12							X				X	
			1.1E-02	3/12				X								
Nickel acetate	*373-02-4*	*1.4E-02*		*3/12*							✓				✓	
			1.1E-02	*3/12*				✓								
Nickel carbonate	*3333-67-3*	*1.4E-02*		*3/12*							✓				✓	
			1.1E-02	*3/12*				✓								
Nickel carbonyl	*13463-39-3*	*1.4E-02*		*3/12*							✓				✓	
			1.1E-02	*3/12*				✓								
Nickel hydroxide	*12054-48-7*	*1.4E-02*		*3/12*							✓				✓	
			1.1E-02	*3/12*				✓								
Nickelocene	*1271-28-9*	*1.4E-02*		*3/12*							✓				✓	
			1.1E-02	*3/12*				✓								

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Number	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL (mg/kg-d)	Date[c] Value Reviewed [Added]	Target Organs											
					Alimentary	Bone and Teeth	Cardiovascular	Reproductive/[d] Development	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Respiratory	Skin
NICKEL OXIDE	1313-99-1	2.0E-02		3/12											X	
			1.1E-02	*3/12*				✓								
Nickel refinery dust from pyrometallurgical process	*1146*	*1.4E-02*		*3/12*							✓				✓	
			1.1E-02	*3/12*				✓								
Nickel subsulfide	*12035-72-2*	*1.4E-02*		*3/12*							✓				✓	
			1.1E-02	*3/12*				✓								
PARTICULATE EMISSIONS FROM DIESEL-FUELED ENGINES[TAC, e]	9901	5.0E+00 TAC		8/98											X	
PERCHLOROETHYLENE[TAC] (Tetrachloroethylene)	127-18-4	3.5E+01 TAC		10/91	X								X			
PHENOL	108-95-2	2.0E+02		4/00	X		X						X	X		
PHOSPHINE	7803-51-2	8.0E-01		9/02	X						X		X	X	X	
PHOSPHORIC ACID	7664-38-2	7.0E+00		2/00											X	
PHTHALIC ANHYDRIDE	85-44-9	2.0E+01		1/01											X	
DIOXIN-LIKE POLYCHLORINATED BIPHENYLS (PCBS)[f, g]	1336-36-3															
3,3',4,4'-TETRACHLOROBIPHENYL (PCB 77)	32598-13-3	4.0E-01		8/03	X			X	X		X				X	
			1.0E-04	8/03	X			X	X		X				X	
3,4,4',5-TETRACHLOROBIPHENYL (PCB 81)	70362-50-4	1.3E-01		1/11	X			X	X		X				X	
			3.3E-05	1/11	X			X	X		X				X	
2,3,3',4,4'-PENTACHLOROBIPHENYL (PCB 105)	32598-14-4	1.3E+00		1/11	X			X	X		X				X	
			3.3E-04	1/11	X			X	X		X				X	
2,3,4,4',5-PENTACHLOROBIPHENYL (PCB 114)	74472-37-0	1.3E+00		1/11	X			X	X		X				X	
			3.3E-04	1/11	X			X	X		X				X	
2,3',4,4',5-PENTACHLOROBIPHENYL (PCB 118)	31508-00-6	1.3E+00		1/11	X			X	X		X				X	
			3.3E-04	1/11	X			X	X		X				X	
2,3',4,4',5'-PENTACHLOROBIPHENYL (PCB 123)	65510-44-3	1.3E+00		1/11	X			X	X		X				X	
			3.3E-04	1/11	X			X	X		X				X	
3,3',4,4',5-PENTACHLOROBIPHENYL (PCB 126)	57465-28-8	4.0E-04		8/03	X			X	X		X				X	

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Number	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL (mg/kg-d)	Date[c] Value Reviewed [Added]	Target Organs											
					Alimentary	Bone and Teeth	Cardiovascular	Reproductive/[d] Development	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Respiratory	Skin
			1.0E-07	8/03	X			X	X		X				X	
2,3,3',4,4',5-HEXACHLOROBIPHENYL (PCB 156)	38380-08-4	1.3E+00		1/11	X			X	X		X				X	
			3.3E-04	1/11	X			X	X		X				X	
2,3,3',4,4',5'-HEXACHLOROBIPHENYL (PCB 157)	69782-90-7	1.3E+00		1/11	X			X	X		X				X	
			3.3E-04	1/11	X			X	X		X				X	
2,3',4,4',5,5'-HEXACHLOROBIPHENYL (PCB 167)	52663-72-6	1.3E+00		1/11	X			X	X		X				X	
			3.3E-04	1/11	X			X	X		X				X	
3,3',4,4',5,5'-HEXACHLOROBIPHENYL (PCB 169)	32774-16-6	1.3E-03		1/11	X			X	X		X				X	
			3.3E-07	1/11	X			X	X		X				X	
2,3,3',4,4',5,5'-HEPTACHLOROBIPHENYL (PCB 189)	39635-31-9	1.3E+00		1/11	X			X	X		X				X	
			3.3E-04	1/11	X			X	X		X				X	
POLYCHLORINATED DIBENZO-P-DIOXINS (PCDD) (Treated as 2,3,7,8-TCDD for HRA)[TAC, f]	1085 1086	4.0E-05		2/00	X			X	X		X				X	
			1.0E-08	10/00	X			X	X		X				X	
2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN[TAC]	1746-01-6	4.0E-05		2/00	X			X	X		X				X	
			1.0E-08	10/00	X			X	X		X				X	
1,2,3,7,8-PENTACHLORODIBENZO-P-DIOXIN	40321-76-4	4.0E-05		8/03	X			X	X		X				X	
			1.0E-08	8/03	X			X	X		X				X	
1,2,3,4,7,8-HEXACHLORODIBENZO-P-DIOXIN	39227-28-6	4.0E-04		2/00	X			X	X		X				X	
			1.0E-07	10/00	X			X	X		X				X	
1,2,3,6,7,8-HEXACHLORODIBENZO-P-DIOXIN	57653-85-7	4.0E-04		2/00	X			X	X		X				X	
			1.0E-07	10/00	X			X	X		X				X	
1,2,3,7,8,9-HEXACHLORODIBENZO-P-DIOXIN	19408-74-3	4.0E-04		2/00	X			X	X		X				X	
			1.0E-07	10/00	X			X	X		X				X	
1,2,3,4,6,7,8-HEPTACHLORODIBENZO-P-DIOXIN	35822-46-9	4.0E-03		2/00	X			X	X		X				X	
			1.0E-06	10/00	X			X	X		X				X	
1,2,3,4,6,7,8,9-OCTACHLORODIBENZO-P-DIOXIN	3268-87-9	1.3E-01		1/11	X			X	X		X				X	
			3.3E-05	1/11	X			X	X		X				X	

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Number	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL (mg/kg-d)	Date[c] Value Reviewed [Added]	Target Organs											
					Alimentary	Bone and Teeth	Cardiovascular	Reproductive/ Development[d]	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Respiratory	Skin
POLYCHLORINATED DIBENZOFURANS (PCDF) (Treated as 2,3,7,8-TCDD for HRA) [TAC, f]	1080	4.0E-05		2/00	X			X	X		X				X	
			1.0E-08	10/00	X			X	X		X				X	
2,3,7,8-TETRACHLORODIBENZOFURAN	5120-73-19	4.0E-04		2/00	X			X	X		X				X	
			1.0E-07	10/00	X			X	X		X				X	
1,2,3,7,8-PENTACHLORODIBENZOFURAN	57117-41-6	1.3E-03		1/11	X			X	X		X				X	
			3.3E-07	1/11	X			X	X		X				X	
2,3,4,7,8-PENTACHLORODIBENZOFURN	57117-31-4	1.3E-04		1/11	X			X	X		X				X	
			3.3E-08	1/11	X			X	X		X				X	
1,2,3,4,7,8-HEXACHLORODIBENZOFURAN	70648-26-9	4.0E-04		2/00	X			X	X		X				X	
			1.0E-07	10/00	X			X	X		X				X	
1,2,3,6,7,8-HEXACHLORODIBENZOFURAN	57117-44-9	4.0E-04		2/00	X			X	X		X				X	
			1.0E-07	10/00	X			X	X		X				X	
1,2,3,7,8,9-HEXACHLORODIBENZOFURAN	72918-21-9	4.0E-04		2/00	X			X	X		X				X	
			1.0E-07	10/00	X			X	X		X				X	
2,3,4,6,7,8-HEXACHLORODIBENZOFURAN	60851-34-5	4.0E-04		2/00	X			X	X		X				X	
			1.0E-07	10/00	X			X	X		X				X	
1,2,3,4,6,7,8-HEPTACHLORODIBENZOFURAN	67562-39-4	4.0E-03		2/00	X			X	X		X				X	
			1.0E-06	10/00	X			X	X		X				X	
1,2,3,4,7,8,9-HEPTACHLORODIBENZOFURAN	55673-89-7	4.0E-03		2/00	X			X	X		X				X	
			1.0E-06	10/00	X			X	X		X				X	
1,2,3,4,6,7,8,9-OCTACHLORODIBENZOFURAN	39001-02-0	1.3E-01		1/11	X			X	X		X				X	
			3.3E-05	1/11	X			X	X		X				X	
POTASSIUM BROMATE ... (see Bromine & Compounds)																
PROPYLENE (PROPENE)	115-07-1	3.0E+03		4/00											X	
PROPYLENE GLYCOL MONOMETHYL ETHER	107-98-2	7.0E+03		2/00	X											
PROPYLENE OXIDE	75-56-9	3.0E+01		2/00											X	

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Substance	Chemical[b] Abstract Number	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL (mg/kg-d)	Date[c] Value Reviewed [Added]	Target Organs											
					Alimentary	Bone and Teeth	Cardiovascular	Reproductive/ Development[d]	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Respiratory	Skin
SELENIUM AND COMPOUNDS (other than hydrogen selenide)[h]	7782-49-2 [1170]	2.0E+01		12/01	X		X							X		
			5.0E-03	12/01	X		X							X		
Selenium sulfide	*7446-34-6*	*2.0E+01*		*12/01*	✓		✓							✓		
			5.0E-03	*12/01*	✓		✓							✓		
SILICA [CRYSTALLINE, RESPIRABLE]	1175	3.0E+00		2/05											X	
STYRENE	100-42-5	9.0E+02		4/00										X		
Sulfuric Acid	7664-93-9	1.0E+00		12/01											X	
Sulfuric Trioxide	*7446-71-9*	*1.0E+00*		*12/01*											✓	
TOLUENE	108-88-3	3.0E+02		4/00				X						X	X	
Toluene diisocyanates	*26471-62-5*	*7.0E-02*		*1/01*											✓	
TOLUENE-2,4-DIISOCYANATE	584-84-9	7.0E-02		1/01											X	
TOLUENE-2,6-DIISOCYANATE	91-08-7	7.0E-02		1/01											X	
TRICHLOROETHYLENE[TAC]	79-01-6	6.0E+02		4/00						X				X		
TRIETHYLAMINE	121-44-8	2.0E+02		9/02						X						
VINYL ACETATE	108-05-4	2.0E+02		12/01											X	
VINYLIDENE CHLORIDE (1,1,-Dichloroethylene)	75-35-4	7.0E+01		1/01	X											
XYLENES (mixed isomers)	1330-20-7	7.0E+02		4/00						X				X	X	
m-XYLENE	108-38-3	7.0E+02		4/00						X				X	X	
o-XYLENE	95-47-6	7.0E+02		4/00						X				X	X	
p-XYLENE	106-42-3	7.0E+02		4/00						X				X	X	

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Purpose: The purpose of this reference table is to provide a quick list of all health values that have been approved by the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) for use in facility health risk assessments conducted for the AB 2588 Air Toxics "Hot Spots" Program. The OEHHA has developed and adopted new risk assessment guidelines that update and replace the 2003 version of the OEHHA *Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments*. The OEHHA has adopted three technical support documents for these guidelines, which can be found on their website (http://www.oehha.ca.gov/air/hot_spots/index.html). This table lists the OEHHA adopted inhalation and oral noncancer chronic RELs. OEHHA is still in the process of adopting new health values. Therefore, new health values will periodically be added to, or deleted from, this table. Users of this table are advised to monitor the OEHHA website (*www.oehha.ca.gov*) for any updates to the health values.

May 2008 update: The Air Resources Board adopted amendments to the AB 2588 Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines Regulation (Title 17, California Code of Regulations, Section 93300.5) on November 16, 2006. The amendments became effective on September 26, 2007, after approval from the Office of Administrative Law. Under the new amendments, the substances previously listed in Appendix A-I *(Substances For Which Emissions Must Be Quantified*) and Appendix F (*Criteria For Inputs For Risk Assessment Using Screening Air Dispersion Modeling*) of the ARB's *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG) (July 1997)* have been removed from this table.

a The checkmarks included in this table clarify applicability of OEHHA adopted health effects values to individual or grouped substances listed in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines*, Appendix A-I list of *"Substances For Which Emissions Must Be Quantified"*.

b Chemical Abstract Service Number (CAS): For chemical groupings and mixtures where a CAS number is not applicable, the 4-digit code used in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG) Report* is listed. The 4-digit codes enclosed in brackets [ ] are codes that have been phased out, but may still appear on previously reported Hot Spots emissions. For information on the origin and use of the 4-digit code, see the EICG report.

c Date Value Reviewed [Added]: This column lists the date that the health value was last reviewed by OEHHA and the Scientific Review Panel, and/or approved for use in the AB 2588 Air Toxics Hot Spots Program. If the health value is unchanged since it was first approved for use in the "Hot Spots" Program, then the date that the value was first approved for use by CAPCOA is listed within the brackets [ ].

- February 2000, April 2000, January 2001, and December 2001 are listed for the first set of 22, the second set of 16, the third set of 22, and the fourth set of 12 noncancer chronic RELs, respectively. The chronic REL for carbon disulfide was adopted in May 2002. Chronic RELs for phosphine and triethylamine were adopted in September 2002. Chronic RELs for fluorides including hydrogen fluoride were adopted August 2003. Chronic REL for silica [crystalline respirable] was adopted February 2005.
- October 2000 is listed for the oral chronic RELs.
- For the substances identified as Toxic Air Contaminants, the Air Resources Board hearing date is listed. The date for acetaldehyde represents the date the value was approved by the Scientific Review Panel.
- On December 19, 2008, OEHHA adopted new chronic RELs for acetaldehyde, acrolein, arsenic, formaldehyde, manganese, and mercury. The most current health values can be found at: http://www.oehha.ca.gov/air/allrels.html.

  Note: 1. We present the new oral RELs only in milligrams (mg/kg-d), although OEHHA has presented oral RELs in other tables in either micrograms (μg/kg-d) or mg/kg-d .

  2. At OEHHA's direction, the chronic oral REL for arsenic does not apply to arsine, because arsine is a gas and not particle associated.
- January 2011 is listed to reflect OEHHA's adoption of the World Health Organization's 2005 Toxicity Equivalency Factors for polychlorinated dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and dioxin-like polychlorinated biphenyls (PCBs). See Appendix C of OEHHA's *Air Toxics Hot Spots Program Technical Support Document for Cancer Potencies* at: http://www.oehha.ca.gov/air/hot_spots/pdf/AppCdioxinTEFs013111.pdf for more information.
- On March 23, 2012, OEHHA adopted revised acute, 8-hour and chronic RELs for nickel and nickel compounds, a separate chronic inhalation REL for nickel oxide, and a revised chronic oral REL for nickel and nickel compounds (including nickel oxide). The values of the RELs are listed in the table at: http://www.oehha.ca.gov/air/chronic_rels/032312CREL.html.
- On July 29, 2013, OEHHA adopted an acute and an 8-hour REL and a revised chronic REL for 1,3-butadiene. The REL value and summary can be found online at: http://www.oehha.ca.gov/air/hot_spots/index.html.
- On October 18, 2013 (February 2014 table update), OEHHA adopted acute, 8-hour, and chronic RELs for caprolactam. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/pdf/Caprolactam2013.pdf. Changes have been made to target organs to the following substances with no change to health factors: Diethanolamine, Fluorides and Hydrogen Fluoride, and Xylenes. The "date added" in this table reflects the date of the health factor only. See footnotes below that discuss changes to substance target organs only.
- On June 27, 2014, OEHHA adopted a new 8-hour REL and revised acute and chronic RELs for benzene. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/BenzeneJune2014.html.

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

d	February 2014. Per OEHHA's current policy, substances with Reproductive System and/or Development as the hazard Index target organ(s) are represented under the single endpoint "Reproductive/Development".
TAC	Toxic Air Contaminant: The Air Resources Board has identified this substance as a Toxic Air Contaminant.
e	Particulate Emissions from Diesel-Fueled Engines: The inhalation cancer potency factor was derived from whole diesel exhaust and should be used only for impacts from the inhalation pathway (based on diesel PM measurements). The inhalation impacts from speciated emissions from diesel-fueled engines are already accounted for in the inhalation cancer potency factor and REL. However, at the discretion of the risk assessor, speciated emissions from diesel-fueled engines may be used to estimate acute noncancer health impacts or the contribution to cancer risk or chronic noncancer health impacts for the non-inhalation exposure pathway. The noncancer chronic REL for diesel exhaust is based on assumptions of contributions of diesel PM to ambient PM. It should be used with diesel PM measurement. There is not an oral chronic REL for diesel exhaust. See Appendix D of OEHHA's document *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments* for more information.
f	Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans (also referred to as chlorinated dioxins and dibenzofurans) and dioxin-like PCB congeners: The OEHHA has adopted the World Health Organization 2005 (WHO-05) Toxicity Equivalency Factor scheme for evaluating the risk due to exposure to samples containing mixtures of polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) and a number of dioxin-like PCB congeners. See Appendix A of OEHHA's Technical Support Document For Describing Available Cancer Potency Factors for more information about the scheme. See Appendix E of OEHHA's 2014 The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments for the methodology for calculating 2,3,7,8-equivalents, cancer risk, and noncancer Hazard Index for PCDD, PCDFs and a number of dioxin-like PCB .
g	Polychlorinated Biphenyls (unspeciated): As of February, 2014, there is no approved method that can be used to assess the noncancer hazard of an unspeciated PCB mixture. Persons preparing HRAs for the Hot Spots Program should consult with OEHHA and the local Air Pollution Control or Air Quality Management District if an assessment of the noncancer hazard for unspeciated PCB mixtures is needed.
h	SELENIUM AND COMPOUNDS: In February 2014, an oral REL was added to the consolidated table. The REL was adopted in Dec 2001, but could not be used by the Hot Spots Program (or HARP software) until transfer factors for the oral and dermal routes were adopted. Transfer factors are included in the OEHHA's Technical Support Document for Exposure Assessment and Stochastic Analysis (August 2012) and will be added to the HARP software in the future.

Other Changes:

February 2014 corrections based on original REL summaries:

- Removed applicability of oleum to the sulfuric acid chronic inhalation REL because oleum represents only an acute health hazard.
- Diethanolamine – deleted cardiovascular and nervous system as target organs, and added hematologic and respiratory systems as target organs.
- Fluorides and Hydrogen Fluoride – target organ for these substances was reconfigured so that "Bone and Teeth" are a combined target organ.
- Xylenes (mixed isomers) – added eye as a target organ.
- Removed "METHYL MERCURY …(see Mercury & Compounds)" entry because methyl mercury has different chemical properties, potency, and toxicity compared to elemental mercury and mercury salts, and it is not emitted directly from any California facilities.

Table 4: OEHHA/ARB APPROVED CHRONIC REFERENCE EXPOSURE LEVELS AND TARGET ORGANS[a]

Page intentionally left blank

Appendix M:

How to Post-Process Offsite Worker Concentrations using the Hourly Raw Results from AERMOD

This appendix describes how to calculate refined offsite worker concentrations using the hourly raw results from the AERMOD air dispersion model. In some cases, a better representation of what the offsite worker breathes during their work shift is needed for the health risk analysis. To obtain a better representation, the hourly raw results contain enough information to allow the risk assessor to evaluate the concentrations that occurs during the offsite worker's shift. However, since the hourly raw results include all the concentrations for every hour of meteorological data at each receptor for each source in the air dispersion analysis, the results must be filtered and processed to obtain the refined offsite worker concentrations. The basic steps include: 1) determining the averaging periods needed for the offsite worker analysis; 2) outputting the hourly raw results from the AERMOD air dispersion model; 3) extracting the hourly concentrations based on when the receptor is present; and 4) identifying or calculating the required concentration. The calculation methods described in this appendix can be used for assessing acute, 8-hour non-cancer chronic, and inhalation cancer health impacts.

M.1 Determine the Averaging Periods Required for the Offsite Worker Health Risk Analysis

Before any refined offsite worker concentrations can be calculated, the first step is to determine which type of refined concentrations or averaging periods are needed for the health risk analysis. The refined averaging periods needed for the analysis are based on the pollutant-specific health values emitted by the source or sources. Specifically, refined offsite worker concentrations can only be used for pollutants that have inhalation cancer potency factors, 8-hour RELs, and/or acute RELs. This section describes the refined averaging periods required for assessing acute RELs, 8-hour RELs, and inhalation cancer potency factors.

M.1.1 Averaging Period Required for Acute RELs

The maximum 1-hour concentration is typically required for the acute health hazard index calculation. AERMOD can determine and output the maximum 1-hour concentration at each receptor location for each source in the air dispersion analysis. However, if more refined concentrations for the offsite worker are needed, the maximum1-hour concentration that occurs during the offsite worker's shift may be used.

This type of refinement can be processed using the hourly raw results from the air dispersion analysis.

If there are multiple sources in the analysis, an additional refinement step is to examine the coincident acute health impacts at each receptor from all sources at each hour during the offsite worker's shift and identify the total maximum acute health impacts from all sources. For example, if there are two sources that emit a single pollutant for ten hours per day and the offsite worker's shift is from hour three to hour seven, the risk assessor may evaluate the total acute risk from all sources during the offsite worker's shift. Assuming the acute REL is 50 $\mu g/m^3$, the highest acute health impact occurs at hour three with a Health Hazard Index of 0.3 (see Table M.1). This approach is also known as a refined acute analysis.

TABLE M.1. EXAMPLE OF A REFINED ACUTE CALCULATION

Hour	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
Source 1 Concentration ($\mu g/m^3$)	5	7	8	0	9	11	5	1	12	3
Source 2 Concentration($\mu g/m^3$)	4	6	7	0	2	1	3	4	5	2
Total Acute Health Hazard Index from All Sources	0.18	0.26	**0.3**	0	0.22	0.24	0.16	0.1	0.34	0.1

M.1.2 Averaging Period Required for Inhalation Cancer Potency Values

The period average is typically required for cancer risk assessments. AERMOD calculates this average by summing all the hourly concentrations and dividing it by the number of processed hours over the entire time period of the air dispersion analysis. However, the period averages calculated from AERMOD typically represent exposures for receptors (i.e., residential receptors) that are present 24 hours a day and seven days per week. For the offsite worker, the period average should represent what the worker breathes during their work shift when assessing the cancer inhalation pathway.

To estimate the offsite worker's concentration, there are two approaches. The simple approach is to obtain the period average concentration as calculated by AERMOD and approximate the worker's inhalation exposure using an adjustment factor (See Chapter 4.12.2. for more information). For a more representative concentration, the second approach is to calculate a refined period average using the hourly raw results from the air dispersion analysis. This refined period average should reflect only the concentrations that occur during the offsite worker's shift. It is calculated by summing all of the hourly concentrations that occurs during the offsite worker's shift and dividing it by the number of hours that occurs during the offsite worker's shift. The equation for calculating the refined offsite worker concentration is shown in Section 4.3.

M.1.3 Averaging Period Required for 8-Hour RELs

For 8-hour noncancer health impacts, we evaluate if the worker is exposed to a daily (e.g., 8-hour) average concentration that exceeds the 8-hour REL. The daily average concentration is intended to represent the long term average concentration the worker is breathing during their work shift. The long-term 8-hour daily average concentration is required for 8-hour health hazard index calculations. Specifically, this concentration represents the long-term average of repeated 8-hour daily averages that occur when the source's emission schedule and offsite worker's schedule overlap. For example, the 8-hour averages are first calculated for each day in the air dispersion analysis. The 8-hour averages should represent the eight hour sequential concentration for when the source's emission schedule and offsite worker's schedule overlap. All the 8-hour averages are then averaged over the entire time period of the air dispersion analysis.

There are two approaches for calculating the average 8-hour daily concentration. The simple approach is to obtain the long-term concentration (i.e., period average) as calculated by AERMOD and approximate the average 8-hour daily concentration using an adjustment factor. For a more representative concentration, the second approach is to calculate the offsite worker concentration using the hourly raw results from the air dispersion analysis.

Please note that although the duration of work shifts or period of overlap with the source's emission schedule can vary from eight hours, the calculated long-term daily average concentrations can still be applied to the 8-hour RELs. However, the risk assessor may wish to calculate the 8-hour hazard index using the adjustment factor approach as a screening assessment before proceeding with the post-processing approach. Based on the results of the screening assessment, the risk assessor can contact OEHHA for assistance in determining whether further evaluation may be necessary.

M.2 Output the Hourly Raw Results from AERMOD

The hourly raw results from the air dispersion analysis are needed to calculate the refined offsite worker concentrations as described above. AERMOD can output the hourly raw results to a file for post-processing. In order to output a file suitable for post-processing, the AERMOD input file must be modified. The AERMOD input file contains the modeling options, source location and parameter data, receptor locations, meteorological data file specifications, and output options. It is organized into five main sections that include the Control (CO), Source (SO), Receptor (RE), Meteorology (ME), and Output (OU) pathways (U.S. EPA, 2004). This section describes how to modify the pathways in the AERMOD input file to allow the hourly raw results to be saved to a file.

M.2.1 Modify the Control (CO) Pathway to Identify Calm and Missing Hours

By default, AERMOD disregards calm and missing hours when calculating the long-term and short-term averages. When calculating the refined offsite worker concentrations, the calm and missing hours must also be disregarded. However, the hourly raw results from AERMOD do not identify which hours are calm or missing. Since this is the case, an additional file from AERMOD must also be saved in order to post-process the hourly raw results correctly. The AERMOD Detailed Error Listing File will report all calm and missing hours from the air dispersion analysis. The syntax for creating a Detailed Error Listing File in the CO pathway is shown below. This modification in the CO pathway will create a file which will be used to assist with calculating the refined offsite worker concentrations. This process is described in the subsequent sections of this appendix.

Syntax for Creating the Detailed Error Listing File

CO ERRORFIL [Filename]

M.2.2 Modify the Source (SO) Pathway if Unit Emission Rates are used

In an air dispersion analysis, it is typical to use non-substance specific unit emission rates (e.g., 1 g/s) for evaluating multiple pollutants. This precludes modelers from having to run the air dispersion model for each individual pollutant that is emitted from a source. Unit emission rates allow the air dispersion modeling results to be expressed as dilution factors in $(\mu g/m^3)/(g/s)$. When these dilution factors are combined with the pollutant specific emission rate (g/s), it will yield the ground level concentrations ($\mu g/m^3$) for each pollutant in the analysis. When there are multiple sources in the air dispersion analysis and unit emission rates are used, the individual source contributions must be provided in the modeling results so the ground level concentrations can be correctly scaled for each pollutant. To do this, the air dispersion input file must be modified to create individual source groups for each source. The example below shows how individual source groups for two sources (S001 and S002) are specified in the SO pathway of an AERMOD input file. This modification in the SO pathway will allow the individual source contributions to be saved in the hourly raw results.

```
SO STARTING

**S001 and S002 location and source parameters are not shown.**

SRCGROUP SRCGP1 S001

SRCGROUP SRCGP2 S002

SO FINSHED
```

This parameter identifies the sources tied to the source group. Use only one source ID per source group.

This section specifies the name of your source group. The source group name is what is specified when you output the required concentrations files.

Please note that a separate input file is needed for evaluating acute health impacts when unit emission rates are used and the source has a variable emission schedule (e.g., emissions vary by hour-of-day and day-of-week). Acute health impacts are based on maximum hourly emissions whereas cancer and chronic health impacts are based on average hourly emissions. To correctly simulate unit emissions for the acute impacts, a duplicate source with a variable emission rate of "on" (1) or "off" (0) should be used so the maximum hourly inventory is correctly calculated separately from the emission factors placed in the annual file. The example below shows how the variable emission rates should be modified. Alternatively, a source can be duplicated in the same input file instead of rerunning the source using a separate input file.

First Run with Unmodified Emission Rate Factors for Long-Term

```
EMISFACT S002  HROFDY   0.000   0.000   0.000   0.000   0.000
         S002  HROFDY   0.000   2.667   2.667   2.667   2.667
         S002  HROFDY   2.667   2.667   1.333   1.333   1.333
         S002  HROFDY   1.333   1.333   1.333   0.000   0.000
         S002  HROFDY   0.000   0.000   0.000   0.000
```

Second Run with Modified Emission Rates Factors for Acute

```
EMISFACT S002  HROFDY   0.000   0.000   0.000   0.000   0.000
         S002  HROFDY   0.000   1.000   1.000   1.000   1.000
         S002  HROFDY   1.000   1.000   1.000   1.000   1.000
         S002  HROFDY   1.000   1.000   1.000   0.000   0.000
         S002  HROFDY   0.000   0.000   0.000   0.000
```

M.2.3 Modify the Receptor (RE) Pathway to Reduce the Processing Time

AERMOD is capable of outputting the hourly raw results from the air dispersion analysis. However, without taking appropriate precautions, outputting the hourly raw results can produce extremely large file sizes especially when evaluating multiple years of meteorological data, a large number of receptors, and short-term averaging periods (e.g., 1-hour). To minimize the amount of processing time and hard disk space, it is recommended to use only a single discrete receptor representing the off-site worker location. The proper syntax for specifying a discrete receptor is shown below.

Sample Syntax for Creating a Single Discrete Receptor

```
RE DISCCART XcoordYcoord (ZelevZhill) (Zflag)
```

M.2.4 Modify the Output (OU) Pathway to Output the Hourly Raw Results

To create a file containing the hourly raw results, modify the OU pathway to include the POSTFILE keyword and parameters. The sample below shows the syntax for outputting the hourly raw results for a single source. The POSTFILE will list in order the concentration for each receptor and for each hour of meteorological data regardless of the source's emission schedule. Use Table M.2 to help construct the proper syntax for the POSTFILE option. This step must be repeated for each source in the analysis which will result in additional files.

Please note that if the data are outputted as binary file (UNFORM), a separate computer program will be needed to read and parse the data.

Sample Syntax for Outputting the

Hourly Concentrations for a Single Source

```
OU POSTFILE 1 SRCGP1PLOT PSTS001.TXT
```

TABLE M.2. DESCRIPTIONS OF THE POSTFILE PARAMETERS

Keyword	**Parameters**	
POSTFILE	AveperGrpid Format Filnam (Funit)	
where:	Aveper	Specifies averaging period to be output to file. Set this value to 1 to output 1-hour raw results.
	Grpid	Specifies source group to be output to file. If there are multiple sources, you will need to repeat the POSTFILE option for each source. You can combine the different outputs to a single file using the Funit parameter.
	Format	Specifies format of file, either UNFORM for binary files or PLOT for formatted files. Unformatted files offer a smaller file size; however, this file requires programming expertise in order to view and parse the data. Selecting the PLOT option will allow you to view the file in any text editor.
	Filnam	Specifies filename for output file
	Funit (optional)	The file unit is an optional parameter. If the filename and the file unit number are the same, the results for different source groups can be combined into a single file.

M.3 Extract the Hourly Concentrations when the Offsite Worker is Present

To calculate the refined offsite worker concentrations, it is necessary to extract the hourly concentrations based on the offsite worker's schedule. This section provides information on how to extract the hourly concentrations for the offsite worker including the calm and missing hours that may occur during the offsite worker's shift.

At this point, it is recommended the hourly raw results be imported into a spreadsheet or database to assist with the extraction process. Spreadsheets and database contain preprogrammed functions to assist with deciphering data. **Use the information in Section M.3.1 as a guide to help import the hourly raw results into a database or spreadsheet.**

M.3.1 Description of the POSTFILE File Format

AERMOD was created using FORTRAN, a type of programming language. When the AERMOD output files are created, it is based on a specified FORTRAN format. The variables provided on each data record in the POSTFILE include the X and Y coordinates of the receptor location, the concentration value for that location, receptor terrain elevation, hill height scale, flagpole receptor height, the averaging period, the source group ID, and the date for the end of the averaging period (in the form of YYMMDDHH) (U.S. EPA, 2004). Table M.3 shows the equivalent data types based on the POSTFILE format. The POSTFILE will list in order the concentration for each receptor and for each hour of meteorological data regardless of the source's emission schedule (see Figure M.3.1). Use the information in this section as a guide to help import the hourly raw results into a database or spreadsheet.

TABLE M.3. POSTFILE VARIABLES AND EQUIVALENT DATA TYPES

Column Name	Fortran Format	Equivalent Data Type
X	F13.5	Number/Double Precision
Y	F13.5	Number/Double Precision
AVERAGE_CONC	F13.5	Number/Double Precision
ZELEV	F8.2	Number/Double Precision
ZHILL	F8.2	Number/Double Precision
ZFLAG	F8.2	Number/Double Precision
AVE	A6	6-Character String/Text
GRP	A8	8-Character String/Text
NUM_HRS OR DATE	I8.8	8-Character String/Text
NET_ID	A8	8-Character String/Text

FIGURE M.3.1. SAMPLE OF AN AERMOD POSTFILE

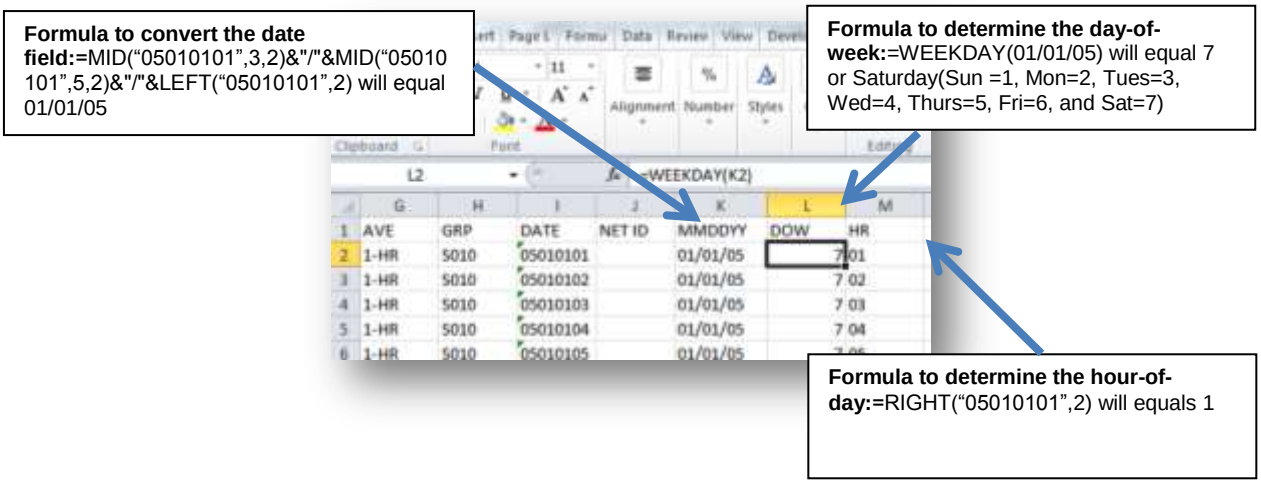
AERMOD (09292): LARGE PS 08/24/10
MODELING OPTIONS USED: 07:39:24
NonDFAULT CONC FLAT FLGPOL
POST/PLOT FILE OF CONCURRENT 1-HR VALUES FOR SOURCE GROUP: S010
FOR A TOTAL OF 1 RECEPTORS.
FORMAT: (3(1X,F13.5),3(1X,F8.2),2X,A6,2X,A8,2X,I8.8,2X,A8)

X	Y	AVERAGE CONC	ZELEV	ZHILL	ZFLAG	AVE	GRP	DATE	NET ID
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010101	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010102	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010103	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010104	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010105	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010106	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010107	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010108	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010109	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010110	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010111	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010112	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010113	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010114	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010115	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010116	
100.00000	0.00000	0.00000	10.00	10.00	1.20	1-HR	S010	05010117	

M.3.2 Determine the Day-of-Week and Hour-of-Day

In order to extract only the hourly concentrations that occur when an offsite worker is present, the risk assessor must first determine the day-of-week and hour-of-day for each hourly record using the date field. Since the date outputted by AERMOD cannot be directly interpreted by the day-of-week function in a database or spreadsheet, the date must be first converted. For example, the date field can be first converted using the LEFT and MID functions in Microsoft Excel (See Column K in Figure M.3.2). After which, the WEEKDAY function in Microsoft Excel can be used to determine the day-of-week (See Column L in Figure M.3.2). The hour-of-day can be extracted using the RIGHT function (See Column M in Figure M.3.2).

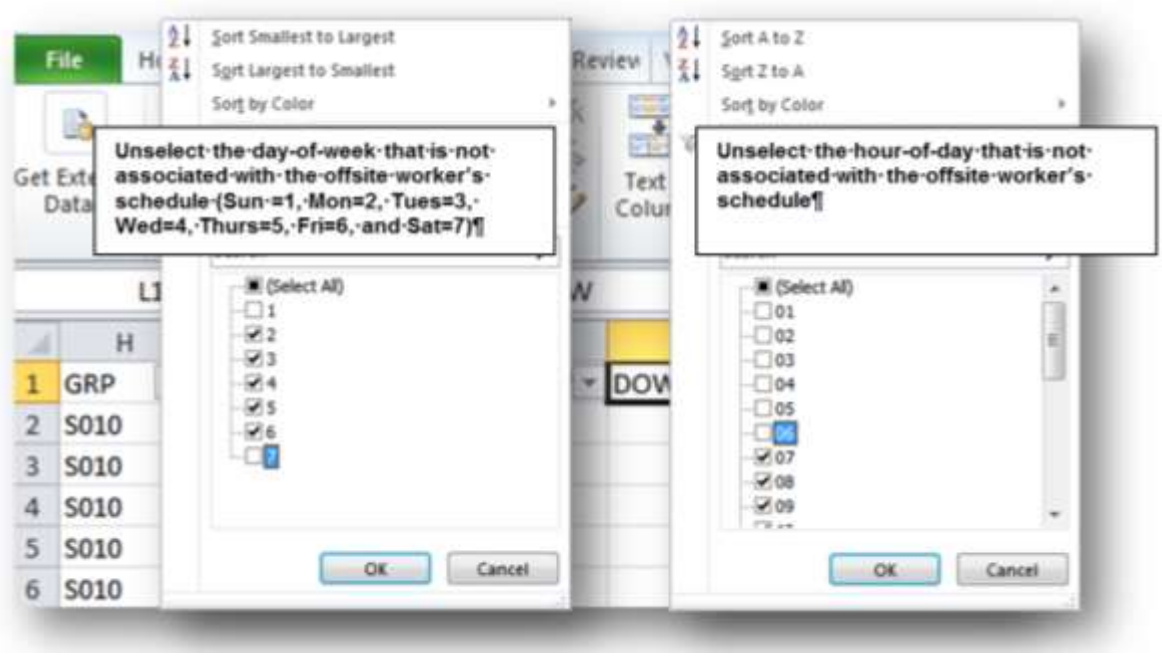
FIGURE M.3.2. HOW TO DETERMINE THE DAY-OF-WEEK AND HOUR-OF-DAY IN MICROSOFT EXCEL



M.3.3 Extract the Hourly Concentrations Based on the Offsite Worker's Schedule

After the day-of-week and hour-of-day have been determined, the concentrations can now be extracted or filtered. Based on the offsite worker's schedule, filter or query the hourly concentrations using a spreadsheet or database. For example, in Microsoft Excel, you can filter the data by selecting the data filter option (see Figure M.3.3). Then unselect the records that are not associated with the offsite worker's schedule using the day-of-week and hour-of-day fields that were created in previous section. If the data contains information for multiple receptors, filter the X and Y coordinates to get the concentrations that are specific to each receptor. The results from the filter will now only show hourly concentrations for times when the offsite worker is present.

FIGURE M.3.3. HOW TO FILTER THE DATA IN MICROSOFT EXCEL



M.3.4 Count the Number of Calm and Missing Hours that Occur During the Offsite Worker's Schedule

If calm hour processing was used in the air dispersion analysis, then calm and missing hours must also be considered when post-processing the long-term and short-term averages for the offsite worker. To assist in this calculation, the Detailed Error Listing File that was created from the air dispersion analysis (Section 2.1) can be used to count the number of calm and missing hours that occurred during the worker's shift.

To identify the calm and missing hours, it is recommended to import the Detailed Error Listing File into a spreadsheet or database. Then follow the instructions from Sections 3.2 and 3.3 to determine the number of calm and missing hours that occur during the offsite worker's schedule. This information is needed to calculate the averaging periods for the offsite worker.

M.4 How to Identify or Calculate the Refined Concentrations for the Offsite Worker Analysis

Depending on which averaging periods are needed (as determined by Section 1.0), use Sections 4.1 through 4.3 below to identify or calculate refined concentrations for estimating the acute, 8-hour, and cancer health impacts. The equations are based on how the long-term and short-term averages are calculated in AERMOD. These equations also account for how calm and missing hours are handled by AERMOD (U.S. EPA, 2005). After calculating the appropriate averaging periods, the refined concentrations can be used to assess the health impacts for the offsite worker's inhalation pathway.

Please note that if unit emission rates were used in the air dispersion analysis, each averaging period calculated using the methods below must be combined with the pollutant specific emission rate (g/s) to yield the actual ground level concentrations (µg/m^3) for each pollutant in the analysis before the health impacts can be assessed.

M.4.1 How to Determine the Maximum 1-Hour Average for a Simple Acute Assessment

The maximum 1-hour average concentration represents the highest concentration that occurs during the offsite worker's schedule. To determine the maximum 1-hour average, sort the extracted hourly concentrations in descending order using a spreadsheet or a database. The maximum hourly concentration will be at the top of the list (Figure M.4.1). This process must be repeated at each receptor for all sources of interest.

FIGURE M.4.1. IDENTIFYING THE MAXIMUM 1-HOUR CONCENTRATION

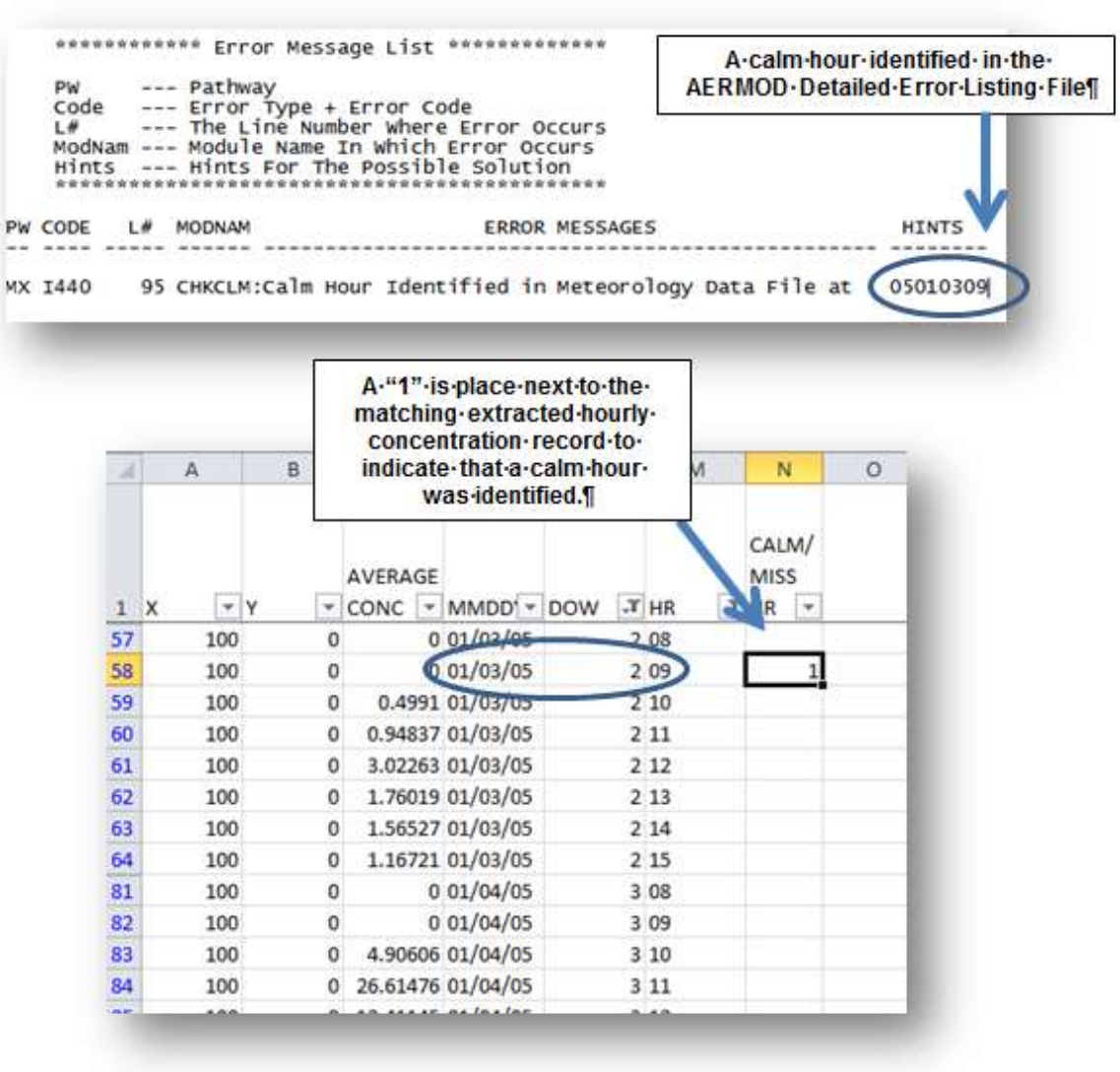
A	B	C	D	E	F	G	H	I	J	K	L
X	Y	AVERAGE CONC	ZELEV	ZHILL	ZFLAG	AVE	GRP	DATE	NET ID	MMDD'	DOW
100	0	110.2656	10	10	1.2	1-HR	S010	05082610		08/26/05	
100	0	105.365	10	10	1.2	1-HR	S010	05082315		08/23/05	
100	0	105.1168	10	10	1.2	1-HR	S010	05080512		08/05/05	
100	0	103.7613	10	10	1.2	1-HR	S010	05071310		07/13/05	
100	0	103.6595	10	10	1.2	1-HR	S010	05082314		08/23/05	
100	0	103.6498	10	10	1.2	1-HR	S010	05071113		07/11/05	
100	0	103.2635	10	10	1.2	1-HR	S010	05082413		08/24/05	
100	0	103.0836	10	10	1.2	1-HR	S010	05012012		01/20/05	
100	0	102.8738	10	10	1.2	1-HR	S010	05052310		05/23/05	

M.4.2 How to Determine the Long-Term Average of 8-Hour Daily Concentrations for an 8-Hour Assessment

To calculate the long-term 8-hour daily average concentration, the 8-hour averages are first calculated for each day in the air dispersion analysis. All the 8-hour averages are then averaged over the entire time period of the air dispersion analysis. However, since the 8-hour daily average is considered a short-term average, the total number of valid hours (i.e., not calm or not missing) must be considered. The total number of valid hours should be 75% of the 8-hour average. If the total number of valid hours in an 8-hour average is less than six (6), the 8-hour total concentration should be divided by six (6) (U.S. EPA, 2005). The following steps below are an example that shows how the average of 8-hour daily concentration is calculated.

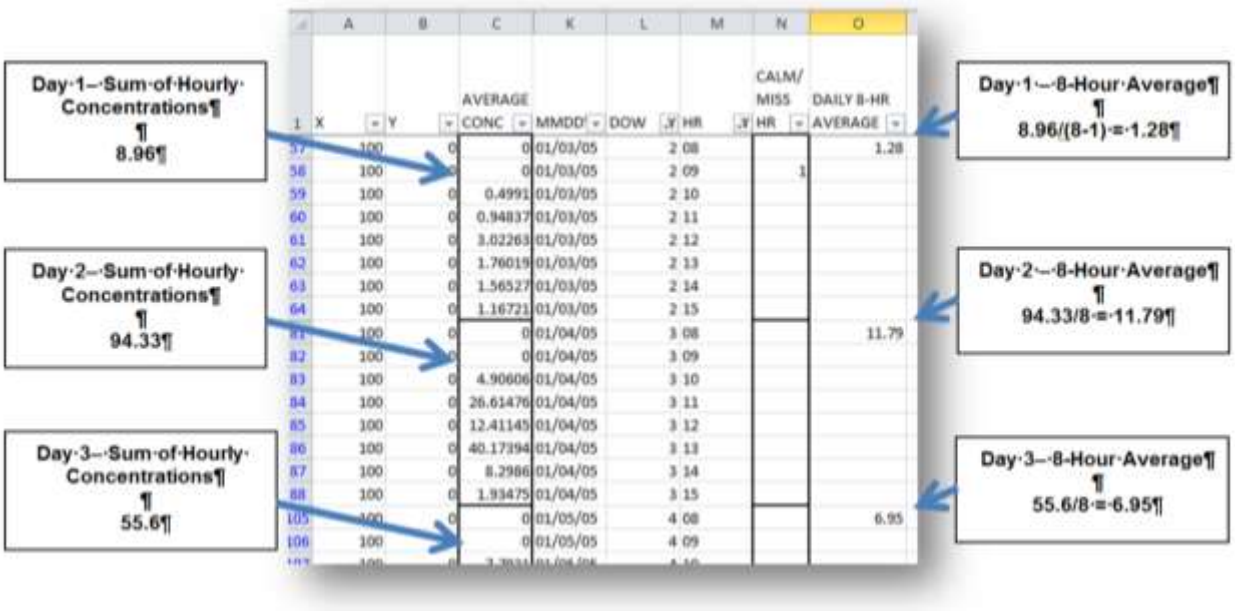
- Using the extracted hourly concentrations based on the steps from Section 3.0, identify any calm and missing hours with a "1". To do this, use the Detailed Error Listing File that was created from the air dispersion analysis (See Section 2.1 for more information).The Detailed Error Listing File will list the calm and missing hours by date. Place a "1" where the dates match up with the extracted hourly concentrations (See Column N in Figure M.4.2.1). Please note that some of the columns are hidden in Figure M.4.2.1 for presentation purposes.

FIGURE M.4.2.1. IDENTIFY CALM AND MISSING HOURS



- Then calculate the 8-hour average for each day throughout the file. The 8-hour average is the sum of the hourly concentrations in a day divided by eight (see Figure M.4.2.2). However, if there are any calm or missing hours in the time period, the sum of hourly concentrations should be divided by total number of valid hours. The total number of valid hours is eight minus the total number of calm and missing hours. If the total number of valid hours is less than six, then the sum of hourly concentrations should be divided by six.

FIGURE M.4.2.2. 8-HOUR DAILY AVERAGE CALCULATION



- Assuming that there were only three days in the entire time period of the air dispersion analysis, the average of 8-hour daily concentrations is (1.28 11.79 + 6.95) /3 = 6.78.

M.4.3 Equation for Calculating the Average Concentration for the Inhalation Cancer Pathway

Below is the equation for calculating the period average for the inhalation cancer pathway. This calculation must be repeated at each receptor for each source of interest.

$$C_{worker\_period\_average} = \frac{\sum C_{hourly}}{N_{total\_hrs} - N_{calm\_hrs} - N_{missing\_hrs}}$$

Where:

C_{hourly} = the concentration that occurs during the worker's shift. To obtain the sum of the hourly concentrations for the offsite worker, sum the extracted worker concentrations from Section 3.0.

N_{total_hrs} = the number of processed hours that occur during worker's shift. To obtain the number of processed hours, use the COUNT function to return the total number of extracted worker concentrations from Section 3.0.

N_{calm_hrs} = the number of calm hours that occur during the worker's shift. To obtain the number of calm and missing hours, use the COUNT function to return the total number of missing and calm hours from Section 3.0. Since the total will include missing hours, it is not necessary to repeat this step for the variable below.

$N_{missing\_hrs}$ = the number of missing hours that occur during worker's shift.

M.5 References

U.S. EPA (2004). User's Guide for the AMS/EPA Regulatory Model – AERMOD. EPA-454/B-03-001.U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2005). Guideline on Air Quality Models (Revised). 40 CFR 51, Appendix W.

Page intentionally left blank

Appendix N:

Sensitivity Study of the Worker Adjustment Factor using AERMOD

N.1. Introduction

The offsite worker health risk analysis begins with estimating the pollutant concentration at a receptor location. To estimate this concentration, the typical approach is to use the residential annual concentration that is modeled based on the adjacent facility's emission schedule. However, if the facility emissions are non-continuous, the residential concentration may not represent what the worker breathes during their work shift. In lieu of conducting additional special case modeling which can be time-consuming, the residential annual concentration is adjusted upwards using a worker adjustment factor based on the facility's emission schedule with respect to the worker's schedule. For an 8-hour work shift that coincides with an adjacent facility that emits eight hours per day, a worker adjustment factor of 4.2 (24 hours / 8 hours * 7 days / 5 days) is typically used for cancer risk assessment.

A possible problem with using this approach is that wind direction, wind speed, and atmospheric stability can vary throughout the day and night and straight scaling as above may skew the results. If the diurnal variation is considerable, the 4.2 adjustment could be an under- or overestimate depending on the time of day that the offsite worker shift begins and ends. The goal of this study is to test the validity of the 4.2 adjustment using five meteorological data sets from five different locations in California and with three different size point sources. The modeling is performed with 8-hour emissions coinciding with the offsite workers' schedule. The 8-hour shifts are modeled as starting every hour around the clock.

To perform this study, the AERMOD air dispersion model, meteorological data from five locations (i.e., Kearny Mesa, Palomar, Pomona, Redlands, and San Bernardino), and three different size point sources (small, medium, and large) are used. The AERMOD-ready meteorological datasets are selected to represent a range of meteorological conditions around the state. To mirror the assumptions used in the 4.2 worker adjustment factor, the emission rate of each source is simulated for eight continuous hours with 24 different start times for five days a week (Monday through Friday). This will simulate the conditions that result during an 8-hour work schedule starting any hour of the day. In addition, the emitting source and offsite worker are assumed to have coincident schedules.

Using the AERMOD air dispersion modeling results, the Point of Maximum Impact (PMI) is identified and the hourly raw concentrations are post-processed to calculate the long-term offsite worker concentration for each scenario. To test the validity of the worker adjustment factor, the calculated long-term offsite worker concentration is divided by the long term residential average to obtain a quotient that is unique to each

meteorological data location. The quotient is then compared to the 4.2 worker adjustment factor to see which is higher or more health protective.

Although this study is primarily based on an 8-hour work schedule, the actual duration that an offsite worker is present near the emitting source may vary when considering a lunch break or a longer work shift. Thus, 10-hour scenarios are also evaluated. The worker adjustment factor for ten hours is 3.4 (24 hours / 10 hours * 7 days / 5 days).

N.2. Background on the Worker Adjustment Factor for Inhalation Cancer Assessments

There are basically two approaches that can be used to calculate the offsite worker inhalation exposure for cancer assessments. One approach is to post-process the hourly dispersion modeling results and examine the coincident hours between the source's emission schedule and the worker's schedule. The second, and more commonly used approach, is to apply a worker adjustment factor to the modeled long-term residential concentration. While post-processing the hourly modeling output will offer a more representative worker concentration, it is very time consuming and requires the management of large amounts of data. Thus, the simplistic approach of applying a worker adjustment factor to estimate the worker inhalation exposure is typically used.

The worker adjustment factor is used together with the long-term residential concentration to estimate the offsite worker's inhalation exposure. This calculation is summarized below.

a. Obtain the long-term concentrations from air dispersion modeling as is typical for residential receptors (all hours of a year or multi-year analysis are used).

b. Determine the coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule.

c. Calculate the worker adjustment factor using Equation N.1. When assessing inhalation cancer health impacts, a discount factor (*DF*) may also be applied if the offsite worker's schedule partially overlaps with the source's emission schedule. The discount factor is based on the number of coincident hours per day and days per week between the source's emission schedule and the offsite worker's schedule (see Equation N.2).

Please note that worker adjustment factor does not apply if the source's emission schedule and the offsite worker's schedule do not overlap. Since the worker is not around during the time that the source is emitting, the worker is not exposed to the source's emission (i.e., the DF in Equation N.2 becomes 0).

$$WAF = \frac{H_{residentid}}{H_{source}} \times \frac{D_{residentid}}{D_{source}} \times DF \qquad \textbf{Eq. N.1}$$

Where:

WAF = the worker adjustment factor

$H_{residential}$ = the number of hours per day the long-term residential concentration is based on (24)

H_{source} = the number of hours the source operates per day

$D_{residential}$ = the number of days per week the long-term residential concentration is based on (7).

D_{source} = the number of days the source operates per week.

DF = a discount factor for when the offsite worker's schedule partially overlaps the source's emission schedule. Use 1 if the offsite worker's schedule occurs within the source's emission schedule. If the offsite worker's schedule partially overlaps with the source's emission schedule, then calculate the discount factor using Equation N.2 below.

$$DF = \frac{H_{coincident}}{H_{worker}} \times \frac{D_{coincident}}{D_{worker}} \qquad \textbf{Eq. N.2}$$

Where:

DF = *the discount factor for assessing cancer impacts*

$H_{coincident}$ = *the number of hours per day the offsite worker's schedule and the source's emission schedule overlap*

$D_{coincident}$ = *the number of days per week the offsite worker's schedule and the source's emission schedule overlap.*

H_{worker} = *the number of hours the offsite worker works per day*

D_{worker} = *the number of days the offsite worker works per week.*

d. The final step is to estimate the offsite worker inhalation exposure by multiplying the worker adjustment factor with the long-term residential concentration.

N.3. Method and Modeling Parameters

For this study, all scenarios are simulated using the AERMOD (Version 09292) air dispersion model. The modeling parameters input to AERMOD and methods used to process the model outputs are discussed below.

N.3.1. Point Source Release Parameters

This study uses three different size point sources representing small, medium, and large. The point source release parameters are shown in Table N.1.

TABLE N.1. POINT SOURCE MODELING PARAMETERS

Source Size	Emission Rate (g/s)	Release Ht (m)	Diameter (m)	Exit Temp (K)	Exit Vel (m/s)	Building Dimensions L (m) x W (m) x H (m)	XBADJ YBADJ [1]
Large	1	30	3	400	10	15 x 15 x 6	7.5
Medium	1	10	1	400	10	12 x 12 x 6	6
Small	1	2.15	0.1	400	10	6 x 6 x 2	3

1 – The XBADJ and YBADJ are keywords defining the along-flow and across-flow distances from the stack to the center of the upwind face of the projected building, respectively (U.S. EPA, 2004).

N.3.2. Temporal Emission Rate

Each point source (i.e., small, medium, and large) is simulated with continuous emissions for eight hours a day from Monday through Friday. In addition, all starting hour combinations (24 scenarios) are evaluated by duplicating each source 24 times with unique start times. Table N.2 shows the 8-hour operating schedule for each scenario. All emissions for Saturday and Sunday are set at zero. This process will also be repeated for the 10-hour evaluation. Table N.3 shows the 10-hour operating schedule for each scenario.

TABLE N.2. 8-HOUR OPERATING SCHEDULE

Time	Scenario																							
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
12:00 AM	ON																	ON	ON	ON	ON	ON	ON	ON
1:00 AM	ON	ON																	ON	ON	ON	ON	ON	ON
2:00 AM	ON	ON	ON																	ON	ON	ON	ON	ON
3:00 AM	ON	ON	ON	ON																	ON	ON	ON	ON
4:00 AM	ON	ON	ON	ON	ON																	ON	ON	ON
5:00 AM	ON	ON	ON	ON	ON	ON																	ON	ON
6:00 AM	ON	ON	ON	ON	ON	ON	ON																	ON
7:00 AM	ON	ON	ON	ON	ON	ON	ON	ON																
8:00 AM		ON	ON	ON	ON	ON	ON	ON	ON															
9:00 AM			ON	ON	ON	ON	ON	ON	ON	ON														
10:00 AM				ON	ON	ON	ON	ON	ON	ON	ON													
11:00 AM					ON	ON	ON	ON	ON	ON	ON	ON												
12:00 PM						ON	ON	ON	ON	ON	ON	ON	ON											
1:00 PM							ON	ON	ON	ON	ON	ON	ON	ON										
2:00 PM								ON	ON	ON	ON	ON	ON	ON	ON									
3:00 PM									ON	ON	ON	ON	ON	ON	ON	ON								
4:00 PM										ON	ON	ON	ON	ON	ON	ON	ON							
5:00 PM											ON	ON	ON	ON	ON	ON	ON	ON						
6:00 PM												ON	ON	ON	ON	ON	ON	ON	ON					
7:00 PM													ON	ON	ON	ON	ON	ON	ON	ON				
8:00 PM														ON	ON	ON	ON	ON	ON	ON	ON			
9:00 PM															ON	ON	ON	ON	ON	ON	ON	ON		
10:00 PM																ON	ON	ON	ON	ON	ON	ON	ON	
11:00 PM																	ON	ON	ON	ON	ON	ON	ON	ON

TABLE N.3. 10-HOUR OPERATING SCHEDULE

Time	Scenario																							
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
12:00 AM	ON															ON	ON	ON	ON	ON	ON	ON	ON	ON
1:00 AM	ON	ON															ON	ON	ON	ON	ON	ON	ON	ON
2:00 AM	ON	ON	ON															ON	ON	ON	ON	ON	ON	ON
3:00 AM	ON	ON	ON	ON															ON	ON	ON	ON	ON	ON
4:00 AM	ON	ON	ON	ON	ON															ON	ON	ON	ON	ON
5:00 AM	ON	ON	ON	ON	ON	ON															ON	ON	ON	ON
6:00 AM	ON	ON	ON	ON	ON	ON	ON															ON	ON	ON
7:00 AM	ON	ON	ON	ON	ON	ON	ON	ON															ON	ON
8:00 AM	ON	ON	ON	ON	ON	ON	ON	ON	ON															ON
9:00 AM	ON	ON	ON	ON	ON	ON	ON	ON	ON	ON														
10:00 AM		ON	ON	ON	ON	ON	ON	ON	ON	ON	ON													
11:00 AM			ON	ON	ON	ON	ON	ON	ON	ON	ON	ON												
12:00 PM				ON	ON	ON	ON	ON	ON	ON	ON	ON	ON											
1:00 PM					ON	ON	ON	ON	ON	ON	ON	ON	ON	ON										
2:00 PM						ON	ON	ON	ON	ON	ON	ON	ON	ON	ON									
3:00 PM							ON	ON	ON	ON	ON	ON	ON	ON	ON	ON								
4:00 PM								ON	ON	ON	ON	ON	ON	ON	ON	ON	ON							
5:00 PM									ON	ON	ON	ON	ON	ON	ON	ON	ON	ON						
6:00 PM										ON	ON	ON	ON	ON	ON	ON	ON	ON	ON					
7:00 PM											ON	ON	ON	ON	ON	ON	ON	ON	ON	ON				
8:00 PM												ON	ON	ON	ON	ON	ON	ON	ON	ON	ON			
9:00 PM													ON	ON	ON	ON	ON	ON	ON	ON	ON	ON		
10:00 PM														ON	ON	ON	ON	ON	ON	ON	ON	ON	ON	
11:00 PM															ON	ON	ON	ON	ON	ON	ON	ON	ON	ON

N.3.3. Receptor Grid Parameters

A 1000 meter by 1000 meter receptor grid is centered over each source. The receptors are spaced in 50 meter increments resulting in 441 receptor points. All receptor flagpole heights are set at 1.2 meters above ground.

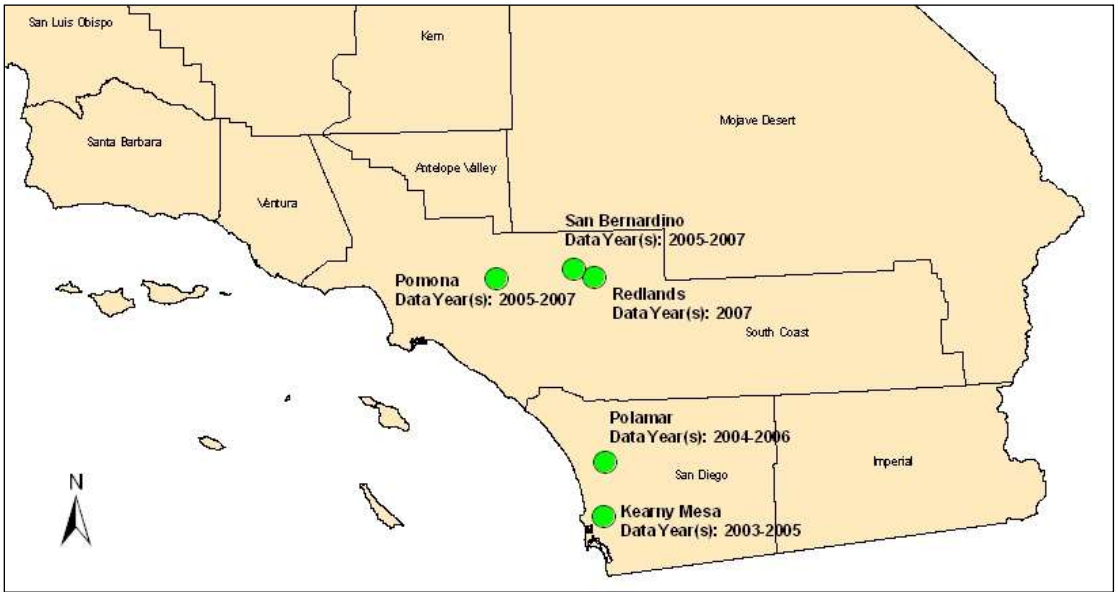
N.3.4. Meteorological Data

The meteorological data input to AERMOD were requested from two local air districts in California (ARB 2009a and ARB 2009b). The meteorological data that were provided by the Districts are, based on the Districts' observations and expertise, datasets that were likely to result in higher than average long-term impacts. The data includes four multi-year files and one single year file. Table N.4 shows the meteorological datasets used in this study. Figure N.1 shows the location of the meteorological station. The AERMOD profile base is defaulted to 10 meters above mean sea level for each meteorological file.

TABLE N.4. METEOROLOGICAL DATASETS

Data Provider	Area	Data Year(s)	Total Hours	Percent of Calm and Missing Hours	Avg. Wind Speed (m/s)
San Diego Air Pollution Control District	Kearny Mesa	2003-2005	26304	6.9	1.36
	Palomar	2004-2006	26304	8.7	1.36
South Coast Air Quality Management District	Pomona	2005-2007	26280	1.6	1.18
	Redlands	2007	8760	5.5	0.94
	San Bernardino	2005-2007	26280	4.9	1.44

FIGURE N.1. METEOROLOGICAL DATA SET LOCATIONS



N.3.5. Post-Processing the Period Average Concentrations for the Offsite Worker

The period average concentration represents the average concentration of all hours processed within the meteorological set. Equation N.3 shows how the period average is calculated in AERMOD including how calm and missing hours are processed (U.S. EPA, 2005).

$$C_{period\_average} = \frac{\sum C_{hourly}}{N_{total\_hrs} - N_{calm\_hrs} - N_{missing\_hrs}} \qquad \textbf{Eq. N.3}$$

Where:

C_{hourly} = the concentration that occurs at a given hour

N_{total_hrs} = the number of processed hours reported by AERMOD (e.g., 1 yr = 8760 hours)

N_{calm_hrs} = the number of calm hours reported by AERMOD

$N_{missing\_hrs}$ = the number of missing hours reported by AERMOD

Normally to post-process hourly data, the off-site worker hours are extracted from the hourly model output files and then averaged. However, this sensitivity study assumes the hourly emissions are coincident with the off-site worker schedule. Since this is the case, the 8-hour period average for the offsite worker can simply be scaled from the period average reported by AERMOD (see Equation N.4). To make sure this calculation is accurate, a check was performed by processing the hourly concentrations

for one receptor with the Pomona data. If the emission schedule was not 100% coincident with the offsite worker, then all post-processing would have to be completed on an hourly basis. See Appendix M for more information on how to post-process worker concentrations using hourly raw results.

$$C_{worker\_period\_average} = C_{period\_average} \times \frac{N_{total\_hrs} - N_{calm\_hrs} - N_{missing\_hrs}}{N_{worker\_hrs} - N_{worker\_calm\_hrs} - N_{worker\_missing\_hrs}} \quad \textbf{Eq. N.4}$$

Where:

$C_{period\_average}$ = the period concentration reported by AERMOD

N_{total_hrs} = the total number of processed hours reported by AERMOD

N_{calm_hrs} = the total number of calm hours reported by AERMOD

$N_{missing\_hrs}$ = the total number of missing hours reported by AERMOD

N_{worker_hrs}[a] = the total number of hours that occurred during the worker's shift

$N_{worker\_calm\_\_hrs}$[b] = the number of calm hours that occurs during the worker's shift

$N_{worker\_missing\_hrs}$[b] = the number of missing hours that occurred during the worker's shift

a. The worker hours are determined by multiplying the number of weekdays (Monday through Friday) that occurs in the meteorological data set by the work shift duration (8 hours). For example, a meteorological data set ranging from 1/1/2003 to 12/31/2005 contains 783 weekdays. If you multiply the number weekdays by the work shift duration (8 hour/day), this will equal 6264 worker hours. The number of weekdays varies depending on the day of the week January 1st starts on.

b. Calm and missing hours are reported in the AERMOD Detailed Message Listing File. To determine the number of worker calm and missing hours, the calm and missing hours that occur during the worker shift are isolated and summed.

N.4. Results

To test the validity of the worker adjustment factor, the post-processed period average concentration for the offsite worker was divided by the modeled period residential average to obtain a quotient. This calculation was performed at the PMI of each scenario. If the quotient is smaller or equal to the worker adjustment factor, the worker adjustment factor is considered a suitable health protective approximation. If the quotient is greater, the worker adjustment factor will underestimate the long-term average concentration and would not be the most conservative estimation of what the worker breathes. For these scenarios, the 8-hour and 10-hour worker adjustment factors are 4.2 and 3.4, respectively. The results for this study are summarized in the figures and tables below. To view the details for every scenario, see Appendix N-1.

Figure N.2 shows how the post-processed period averages changes over 8-hour rolling work shifts. The value at each 8-hour work shift represents the quotient average across the five meteorological data sets. Values that fall on or below the thick dashed line (i.e., the 4.2 worker adjustment factor) indicate that the worker adjustment factor would be a health protective value. Based on the five metrological data sets, the worker adjustment factor is health protective for work shifts that start approximately between 8 am and 3 pm (i.e., 8-hour work shifts starting at 8 am and ending by 11 pm).

FIGURE N.2. SUMMARY OF THE 8-HOUR SCENARIOS

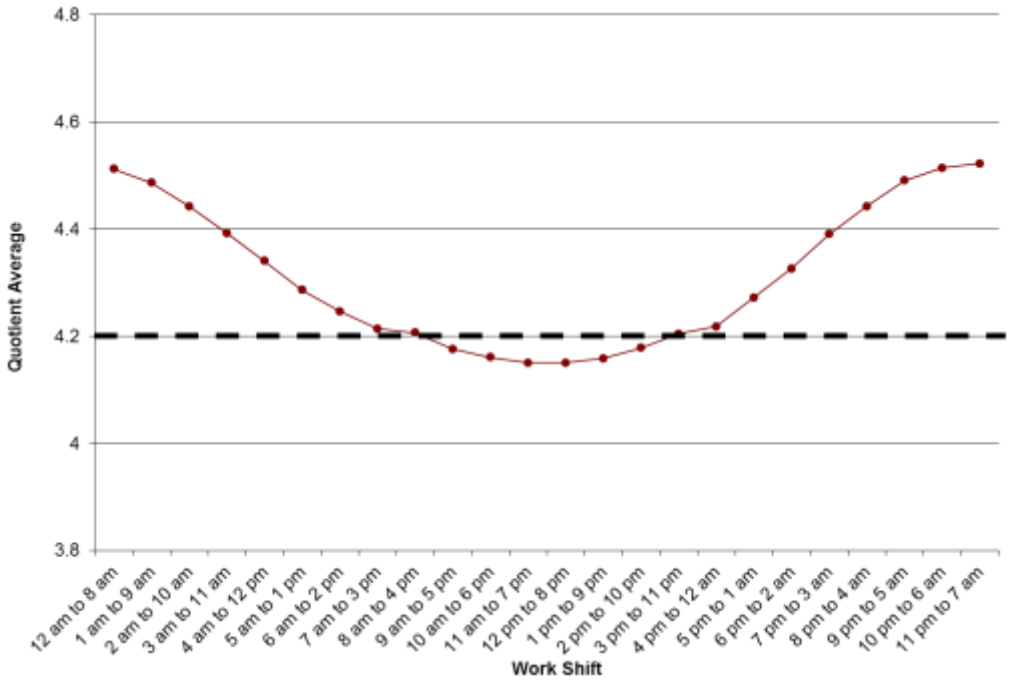


Figure N.3 shows relationship between the worker schedule and the percent of calm and missing hours that occurred during 8-hr work shifts. The figure shows the percent of calm and missing hours are higher during the early morning and evening hour start hours.

FIGURE N.3. AVERAGE PERCENT OF CALM AND MISSING HOURS FOR 8-HOUR WORK SHIFTS

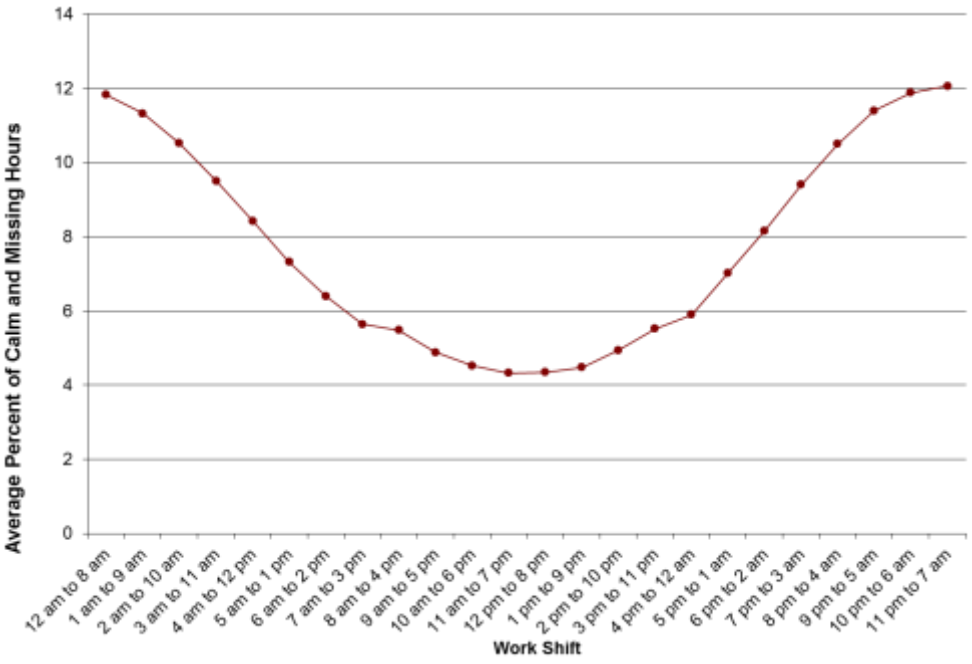


Figure N.4 shows how the post-processed period averages change over 10-hour rolling work shifts. The value at each 10-hour work shift represents the quotient average across the five meteorological data sets. Values that fall on or below the thick dashed line (i.e., the 3.4 worker adjustment factor) indicate that the worker adjustment factor would be a health protective value. Based on the five metrological data sets, the worker adjustment factor is health protective for work shifts that start approximately between 5 am and 4 pm (i.e., 10-hour work shifts starting at 5 am and ending by 2 am).

FIGURE N.4. SUMMARY OF THE 10-HOUR SCENARIOS

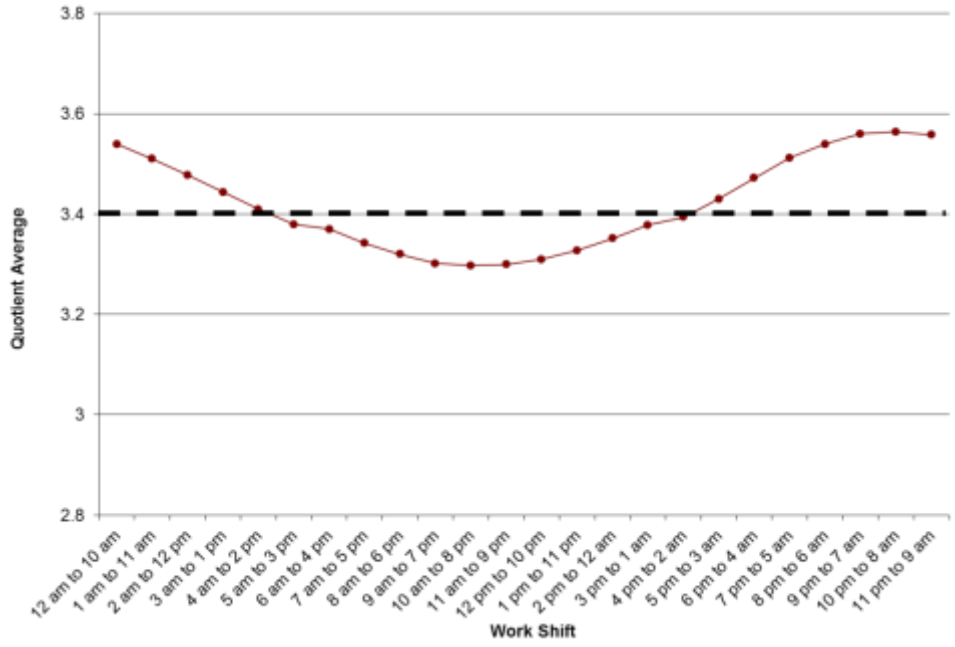


Figure N.5 shows relationship between the worker schedule and the percent of calm and missing hours that occurred during 10-hr work shifts. The figure shows the percent of calm and missing hours are higher during the early morning and evening hour start hours.

FIGURE N.5. AVERAGE PERCENT OF CALM AND MISSING HOURS FOR 10-HOUR WORK SHIFTS

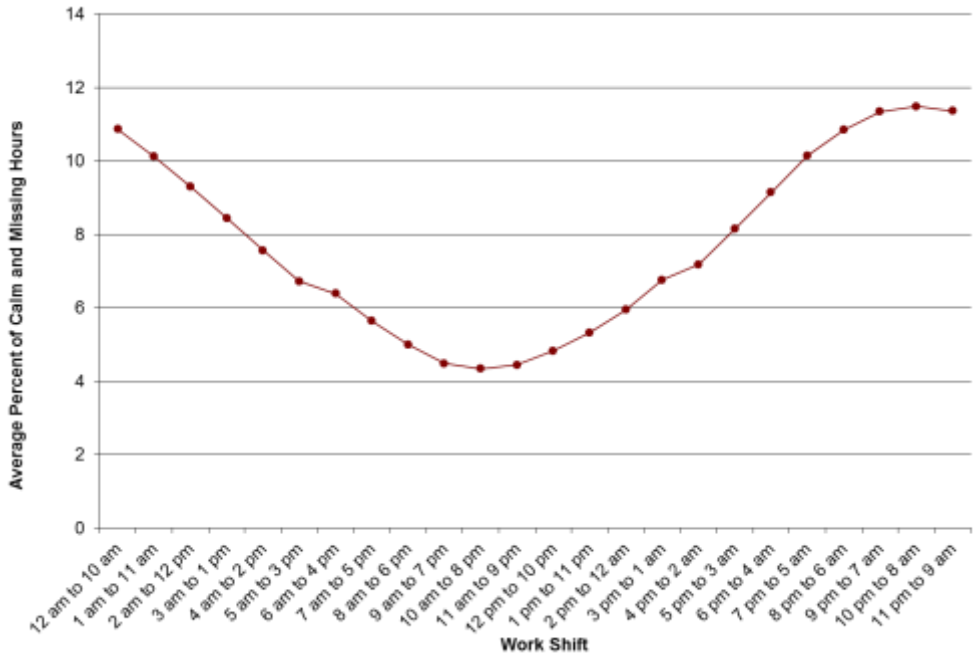


Table N.5 shows the average, minimum, and maximum quotients across all 24 8-hour work shifts for each point source size (i.e., small, medium, and large). The values in the parentheses are the range across the 24 work shifts for each meteorological data set.

TABLE N.5. SUMMARY OF THE AVERAGE 8-HOUR SCENARIOS BY POINT SOURCE SIZE

Meteorological Set	Point Source Size			% Calm/Missing Hours During the Worker's Shift
	Small	Medium	Large	
Kearny Mesa	4.33 (4.19 to 4.43)	4.33 (4.19 to 4.43)	4.33 (4.19 to 4.43)	9.6 (6.8 to 11.8)
Palomar	4.38 (4.18 to 4.65)	4.38 (4.18 to 4.65)	4.38 (4.18 to 4.65)	12.2 (8.2 to 17.5)
Pomona	4.24 (4.23 to 4.25)	4.24 (4.23 to 4.25)	4.24 (4.23 to 4.25)	2.3 (2.1 to 2.5)
Redlands	4.31 (4.00 to 4.75)	4.31 (4.00 to 4.75)	4.31 (4.00 to 4.75)	7.6 (1.0 to 16.5)
San Bernardino	4.31 (4.06 to 4.65)	4.31 (4.06 to 4.65)	4.31 (4.06 to 4.65)	6.9 (1.4 to 14.1)

Table N.6 shows the average, minimum, and maximum quotients across all 24 10-hour work shifts for each point source size (i.e., small, medium, and large). The values in the parentheses are the range across the 24 work shifts for each meteorological data set.

TABLE N.6. SUMMARY OF THE AVERAGE 10-HOUR SCENARIOS BY POINT SOURCE SIZE

Meteorological Set	Point Source Size			% Calm/Missing Hours During the Worker's Shift
	Small	Medium	Large	
Kearny Mesa	3.46 (3.38 to 3.54)	3.46 (3.38 to 3.54)	3.46 (3.38 to 3.54)	9.6 (7.5 to 11.6)
Palomar	3.50 (3.34 to 3.70)	3.50 (3.34 to 3.70)	3.50 (3.34 to 3.70)	12.2 (8.0 to 17.1)
Pomona	3.39 (3.38 to 3.39)	3.39 (3.38 to 3.39)	3.39 (3.38 to 3.39)	2.3 (2.2 to 2.5)
Redlands	3.45 (3.21 to 3.74)	3.45 (3.21 to 3.74)	3.45 (3.21 to 3.74)	7.6 (1.1 to 15.2)
San Bernardino	3.31 (3.12 to 3.54)	3.31 (3.12 to 3.54)	3.31 (3.12 to 3.54)	6.9 (1.5 to 13.1)

N.5. Conclusions

The goal of this study was to determine if the worker adjustment factor of 4.2 (8 hours/day, 5 days/week) or 3.4 (10 hours/day, 5 days/week) would always yield a more conservative or health protective approximation using five meteorological data sets. This study demonstrated that the worker adjustment factor does not always represent the most health protective approximation of long-term hourly model predictions. This is primarily observed during night conditions. Air Districts may wish to evaluate their meteorological data to determine an appropriate worker adjustment factor for their area using the methods described in this appendix.

Although the meteorological data used in this study are site-specific, several general conclusions and recommendations can be made. These conclusions and recommendations are summarized below.

- ***The worker adjustment factor is generally a suitable health protective approximation for daytime work shifts.***

  For the meteorological data used in this study, the results show that the worker adjustment factor is a suitable health protective approximation for work shifts that occur during the daytime hours. When comparing the 8-hour and 10-hour scenarios, the results show that the range of work shifts that were considered a more health protective approximation increased with the longer work shift duration.

- ***The size of the emitting source did not affect the long-term concentration approximated with the worker adjustment factor.***

  The size of the source was inconsequential in determining whether the worker adjustment factor is health protective. This is because the worker adjustment factor is applied to the modeling results after the air dispersion analysis has been completed. However, it should be noted that the size of the source does affect the location of the PMI during a specific time of day. This is shown in the scenario details in Appendix N-1.

- ***The worker adjustment factor may not represent the most conservative estimation of the worker's inhalation exposure for nighttime work shifts.***

  In most cases, the worker adjustment factor will represent a health protective approximation for work shifts that occur during the daytime. However, the worker adjustment factor may not represent the most conservative estimation when the source's emission schedule and offsite worker's schedules are 100% coincident at night. It is recommended that the offsite worker long-term average concentrations be post-processed using the hourly dispersion modeling results when examining work shifts occurring at night. Alternatively, a more conservative worker adjustment factor can be used to account for the calm hours (see the next bullet point below).

- ***Recommended worker adjustment factor for 8 and 10-hour work shifts***

  Based on the five meteorological data sets used in this study, the range of worker adjustment factors (WAF) was between 4.2 and 4.8. We recommend using the 4.2 WAF for most cases. In the event of predominant night time emissions and worker schedule or if only one year of meteorological data are available, then we recommend using 4.8 for the 8-hour WAF.

N.6. References

ARB (2009a). Harris, Gregory. "Aermod met data in San Diego." Email to Ralph Desina, San Diego Air Pollution Control District.

ARB (2009b). Harris, Gregory. "Aermod met data in SC." Email to Tom Chico, South Coast Air Quality Management District.

U.S. EPA (2004). User's Guide for the AMS/EPA Regulatory Model – AERMOD. EPA-454/B-03-001. U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. EPA (2005). Guideline on Air Quality Models (Revised). 40 CFR 51, Appendix W.

APPENDIX N-1 – SCENARIO DATA DETAILS

KEARNY MESA - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	500	0.02584	26304	1813	632.84744	6264	723	11.5	0.11421	**4.42**
2	0	300	0.05638	26304	1813	1380.80258	6264	739	11.8	0.24992	**4.43**
3	150	-150	0.10366	26304	1813	2538.73706	6264	729	11.6	0.45867	**4.42**
4	150	-100	0.19993	26304	1813	4896.48563	6264	718	11.5	0.88289	**4.42**
5	200	-100	0.33363	26304	1813	8170.93233	6264	700	11.2	1.46854	**4.40**
6	200	-100	0.48136	26304	1813	11788.98776	6264	688	11.0	2.11424	**4.39**
7	200	-100	0.62685	26304	1813	15352.18335	6264	684	10.9	2.75129	**4.39**
8	200	-100	0.76245	26304	1813	18673.16295	6264	681	10.9	3.34465	**4.39**
9	200	-100	0.85443	26304	1813	20925.84513	6264	665	10.6	3.73743	**4.37**
10	250	-100	0.89012	26304	1813	21799.92892	6264	618	9.9	3.86113	**4.34**
11	250	-100	0.85448	26304	1813	20927.06968	6264	568	9.1	3.67399	**4.30**
12	250	-100	0.76187	26304	1813	18658.95817	6264	517	8.3	3.24673	**4.26**
13	250	-100	0.63409	26304	1813	15529.49819	6264	488	7.8	2.68863	**4.24**
14	250	-100	0.48738	26304	1813	11936.42358	6264	467	7.5	2.05907	**4.22**
15	300	-150	0.34902	26304	1813	8547.84882	6264	454	7.2	1.47123	**4.22**
16	300	-150	0.20978	26304	1813	5137.72198	6264	433	6.9	0.88110	**4.20**
17	300	-150	0.09739	26304	1813	2385.17849	6264	425	6.8	0.40849	**4.19**
18	350	-200	0.02843	26304	1813	696.27913	6264	456	7.3	0.11988	**4.22**
19	0	500	0.00479	26304	1813	117.31189	6264	516	8.2	0.02041	**4.26**
20	-50	500	0.00491	26304	1813	120.25081	6264	578	9.2	0.02115	**4.31**
21	0	500	0.00512	26304	1813	125.39392	6264	625	10.0	0.02224	**4.34**
22	0	500	0.00513	26304	1813	125.63883	6264	658	10.5	0.02241	**4.37**
23	0	500	0.00528	26304	1813	129.31248	6264	675	10.8	0.02314	**4.38**
24	0	500	0.01002	26304	1813	245.39982	6264	699	11.2	0.04410	**4.40**

KEARNY MESA - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	100	0.48213	26304	1813	11807.84583	6264	723	11.5	2.13100	**4.42**
2	0	100	0.99949	26304	1813	24478.50959	6264	739	11.8	4.43050	**4.43**
3	50	50	1.69544	26304	1813	41523.02104	6264	729	11.6	7.50190	**4.42**
4	50	50	2.6458	26304	1813	64798.28780	6264	718	11.5	11.68379	**4.42**
5	50	50	3.51528	26304	1813	86092.72248	6264	700	11.2	15.47317	**4.40**
6	50	50	4.24949	26304	1813	104074.25959	6264	688	11.0	18.66468	**4.39**
7	100	-50	5.33685	26304	1813	130704.79335	6264	684	10.9	23.42380	**4.39**
8	100	-50	6.51541	26304	1813	159568.90631	6264	681	10.9	28.58121	**4.39**
9	100	-50	7.325	26304	1813	179396.57500	6264	665	10.6	32.04082	**4.37**
10	100	-50	7.60514	26304	1813	186257.48374	6264	618	9.9	32.98928	**4.34**
11	100	-50	7.28086	26304	1813	178315.54226	6264	568	9.1	31.30540	**4.30**
12	100	-50	6.51093	26304	1813	159459.18663	6264	517	8.3	27.74651	**4.26**
13	100	-50	5.53256	26304	1813	135497.92696	6264	488	7.8	23.45878	**4.24**
14	100	-50	4.37499	26304	1813	107147.88009	6264	467	7.5	18.48333	**4.22**
15	100	-50	3.13098	26304	1813	76680.83118	6264	454	7.2	13.19808	**4.22**
16	100	-50	1.92339	26304	1813	47105.74449	6264	433	6.9	8.07850	**4.20**
17	150	-50	0.97341	26304	1813	23839.78431	6264	425	6.8	4.08285	**4.19**
18	200	-100	0.37344	26304	1813	9145.91904	6264	456	7.3	1.57471	**4.22**
19	0	150	0.19509	26304	1813	4777.94919	6264	516	8.2	0.83124	**4.26**
20	0	150	0.18348	26304	1813	4493.60868	6264	578	9.2	0.79029	**4.31**
21	0	150	0.17623	26304	1813	4316.04893	6264	625	10.0	0.76539	**4.34**
22	0	150	0.16448	26304	1813	4028.27968	6264	658	10.5	0.71857	**4.37**
23	0	150	0.16295	26304	1813	3990.80845	6264	675	10.8	0.71405	**4.38**
24	0	150	0.22443	26304	1813	5496.51513	6264	699	11.2	0.98769	**4.40**

KEARNY MESA - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	50	56.94704	26304	1813	1394689.95664	6264	723	11.5	251.70366	**4.42**
2	0	50	63.90855	26304	1813	1565184.29805	6264	739	11.8	283.29128	**4.43**
3	0	50	72.78622	26304	1813	1782607.31402	6264	729	11.6	322.06094	**4.42**
4	0	50	80.59339	26304	1813	1973812.71449	6264	718	11.5	355.89843	**4.42**
5	0	50	86.44869	26304	1813	2117214.86679	6264	700	11.2	380.52029	**4.40**
6	50	0	96.25147	26304	1813	2357294.75177	6264	688	11.0	422.75731	**4.39**
7	50	0	117.66867	26304	1813	2881823.39697	6264	684	10.9	516.45581	**4.39**
8	50	0	138.64904	26304	1813	3395653.63864	6264	681	10.9	608.21308	**4.39**
9	50	0	156.76654	26304	1813	3839369.33114	6264	665	10.6	685.72412	**4.37**
10	50	0	172.75048	26304	1813	4230832.00568	6264	618	9.9	749.35034	**4.34**
11	50	0	184.10847	26304	1813	4509000.53877	6264	568	9.1	791.60824	**4.30**
12	50	0	190.80885	26304	1813	4673099.54535	6264	517	8.3	813.13721	**4.26**
13	50	0	183.97723	26304	1813	4505786.33993	6264	488	7.8	780.08766	**4.24**
14	50	0	168.91026	26304	1813	4136781.17766	6264	467	7.5	713.60724	**4.22**
15	50	0	150.42213	26304	1813	3683988.38583	6264	454	7.2	634.07717	**4.22**
16	50	-50	146.48297	26304	1813	3587514.41827	6264	433	6.9	615.24857	**4.20**
17	50	-50	144.08415	26304	1813	3528764.91765	6264	425	6.8	604.34405	**4.19**
18	50	-50	130.6006	26304	1813	3198539.29460	6264	456	7.3	550.71269	**4.22**
19	50	-50	111.9118	26304	1813	2740831.89380	6264	516	8.2	476.83227	**4.26**
20	50	-50	86.25428	26304	1813	2112453.57148	6264	578	9.2	371.51839	**4.31**
21	50	-50	65.37008	26304	1813	1600978.62928	6264	625	10.0	283.91180	**4.34**
22	0	50	56.60048	26304	1813	1386202.35568	6264	658	10.5	247.27120	**4.37**
23	0	50	53.20196	26304	1813	1302969.20236	6264	675	10.8	233.13101	**4.38**
24	-100	-100	54.24037	26304	1813	1328400.90167	6264	699	11.2	238.70636	**4.40**

PALOMAR - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	250	0.02363	26304	2291	567.42719	6256	1096	17.5	0.10997	**4.65**
2	100	150	0.0631	26304	2291	1515.22030	6256	1090	17.4	0.29331	**4.65**
3	150	50	0.14317	26304	2291	3437.94121	6256	1050	16.8	0.66038	**4.61**
4	150	50	0.27432	26304	2291	6587.24616	6256	971	15.5	1.24640	**4.54**
5	200	50	0.42859	26304	2291	10291.73167	6256	879	14.1	1.91403	**4.47**
6	200	50	0.58751	26304	2291	14107.87763	6256	788	12.6	2.58008	**4.39**
7	200	0	0.73867	26304	2291	17737.68271	6256	701	11.2	3.19310	**4.32**
8	200	0	0.87304	26304	2291	20964.30952	6256	628	10.0	3.72500	**4.27**
9	250	0	0.96493	26304	2291	23170.86409	6256	679	10.9	4.15472	**4.31**
10	250	0	0.99791	26304	2291	23962.81283	6256	589	9.4	4.22848	**4.24**
11	250	0	0.9484	26304	2291	22773.92920	6256	540	8.6	3.98424	**4.20**
12	250	0	0.83614	26304	2291	20078.22982	6256	518	8.3	3.49917	**4.18**
13	250	0	0.68595	26304	2291	16471.71735	6256	517	8.3	2.87014	**4.18**
14	250	0	0.51501	26304	2291	12366.93513	6256	523	8.4	2.15715	**4.19**
15	300	0	0.34888	26304	2291	8377.65544	6256	550	8.8	1.46822	**4.21**
16	300	-50	0.20229	26304	2291	4857.58977	6256	596	9.5	0.85823	**4.24**
17	300	-100	0.10109	26304	2291	2427.47417	6256	516	8.2	0.42290	**4.18**
18	300	-150	0.0311	26304	2291	746.80430	6256	612	9.8	0.13232	**4.25**
19	-450	-200	0.00583	26304	2291	139.99579	6256	701	11.2	0.02520	**4.32**
20	-400	-150	0.00576	26304	2291	138.31488	6256	802	12.8	0.02536	**4.40**
21	-400	-200	0.00503	26304	2291	120.78539	6256	895	14.3	0.02253	**4.48**
22	-400	-200	0.00427	26304	2291	102.53551	6256	980	15.7	0.01943	**4.55**
23	-400	-200	0.00323	26304	2291	77.56199	6256	1040	16.6	0.01487	**4.60**
24	-500	-500	0.0081	26304	2291	194.50530	6256	1067	17.1	0.03748	**4.63**

PALOMAR - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	50	0.39916	26304	2291	9585.02908	6256	1096	17.5	1.85756	**4.65**
2	50	50	1.1355	26304	2291	27266.76150	6256	1090	17.4	5.27812	**4.65**
3	50	50	2.23922	26304	2291	53770.38986	6256	1050	16.8	10.32854	**4.61**
4	50	50	3.46481	26304	2291	83200.48253	6256	971	15.5	15.74276	**4.54**
5	100	0	5.01511	26304	2291	120427.83643	6256	879	14.1	22.39685	**4.47**
6	100	0	7.1387	26304	2291	171421.60310	6256	788	12.6	31.34996	**4.39**
7	100	0	9.3361	26304	2291	224187.76930	6256	701	11.2	40.35783	**4.32**
8	100	0	11.30065	26304	2291	271362.50845	6256	628	10.0	48.21651	**4.27**
9	100	0	12.55274	26304	2291	301428.94562	6256	679	10.9	54.04858	**4.31**
10	100	0	12.9907	26304	2291	311945.67910	6256	589	9.4	55.04600	**4.24**
11	100	0	12.32253	26304	2291	295900.91289	6256	540	8.6	51.76713	**4.20**
12	100	0	10.99232	26304	2291	263958.58016	6256	518	8.3	46.00184	**4.18**
13	100	0	9.16435	26304	2291	220063.53655	6256	517	8.3	38.34528	**4.18**
14	100	0	7.04288	26304	2291	169120.67744	6256	523	8.4	29.49951	**4.19**
15	100	0	4.85232	26304	2291	116518.76016	6256	550	8.8	20.42039	**4.21**
16	100	0	2.83666	26304	2291	68116.71658	6256	596	9.5	12.03476	**4.24**
17	150	0	1.4789	26304	2291	35512.82570	6256	516	8.2	6.18690	**4.18**
18	150	0	0.51952	26304	2291	12475.23376	6256	612	9.8	2.21035	**4.25**
19	500	100	0.16252	26304	2291	3902.59276	6256	701	11.2	0.70254	**4.32**
20	-100	-50	0.13578	26304	2291	3260.48514	6256	802	12.8	0.59782	**4.40**
21	-100	-50	0.12284	26304	2291	2949.75692	6256	895	14.3	0.55023	**4.48**
22	-100	-50	0.10491	26304	2291	2519.20383	6256	980	15.7	0.47748	**4.55**
23	-150	-50	0.08895	26304	2291	2135.95635	6256	1040	16.6	0.40950	**4.60**
24	-100	0	0.15313	26304	2291	3677.11069	6256	1067	17.1	0.70864	**4.63**

PALOMAR - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	0	62.23758	26304	2291	1494511.00854	6256	1096	17.5	289.63392	**4.65**
2	-50	0	67.07392	26304	2291	1610646.04096	6256	1090	17.4	311.77817	**4.65**
3	-50	0	69.58692	26304	2291	1670990.70996	6256	1050	16.8	320.97401	**4.61**
4	50	0	76.6273	26304	2291	1840051.35490	6256	971	15.5	348.16487	**4.54**
5	50	0	101.35151	26304	2291	2433753.80963	6256	879	14.1	452.62299	**4.47**
6	50	0	132.881	26304	2291	3190871.45300	6256	788	12.6	583.55367	**4.39**
7	50	0	166.85749	26304	2291	4006748.90737	6256	701	11.2	721.28693	**4.32**
8	50	0	199.35655	26304	2291	4787148.83515	6256	628	10.0	850.59503	**4.27**
9	50	0	227.0465	26304	2291	5452067.60450	6256	679	10.9	977.59864	**4.31**
10	50	0	258.20597	26304	2291	6200299.95761	6256	589	9.4	1094.10622	**4.24**
11	50	0	284.95975	26304	2291	6842738.47675	6256	540	8.6	1197.12010	**4.20**
12	50	0	306.84919	26304	2291	7368369.59947	6256	518	8.3	1284.13552	**4.18**
13	50	0	305.48615	26304	2291	7335638.91995	6256	517	8.3	1278.20856	**4.18**
14	50	0	284.9321	26304	2291	6842074.51730	6256	523	8.4	1193.45448	**4.19**
15	50	0	255.29701	26304	2291	6130447.10113	6256	550	8.8	1074.38610	**4.21**
16	50	0	222.46841	26304	2291	5342133.92933	6256	596	9.5	943.83992	**4.24**
17	50	0	190.65477	26304	2291	4578192.99201	6256	516	8.2	797.59460	**4.18**
18	50	0	149.99496	26304	2291	3601828.97448	6256	612	9.8	638.16956	**4.25**
19	50	0	109.43689	26304	2291	2627908.03957	6256	701	11.2	473.07075	**4.32**
20	50	0	71.34752	26304	2291	1713267.99776	6256	802	12.8	314.13055	**4.40**
21	50	0	47.98635	26304	2291	1152296.22255	6256	895	14.3	214.94054	**4.48**
22	-50	50	46.33971	26304	2291	1112755.45623	6256	980	15.7	210.90892	**4.55**
23	-50	0	48.61618	26304	2291	1167420.33034	6256	1040	16.6	223.81525	**4.60**
24	-50	0	55.01306	26304	2291	1321028.60978	6256	1067	17.1	254.58250	**4.63**

POMONA - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	300	-100	0.0378	26280	432	977.05440	6248	138	2.2	0.15991	**4.23**
2	200	-50	0.08941	26280	432	2311.06968	6248	140	2.2	0.37837	**4.23**
3	200	-50	0.18145	26280	432	4690.11960	6248	142	2.3	0.76812	**4.23**
4	200	-50	0.30538	26280	432	7893.46224	6248	145	2.3	1.29337	**4.24**
5	200	-50	0.4489	26280	432	11603.16720	6248	147	2.4	1.90185	**4.24**
6	200	0	0.59344	26280	432	15339.23712	6248	152	2.4	2.51628	**4.24**
7	200	0	0.72765	26280	432	18808.29720	6248	154	2.5	3.08636	**4.24**
8	250	0	0.84968	26280	432	21962.52864	6248	157	2.5	3.60573	**4.24**
9	250	0	0.93127	26280	432	24071.46696	6248	159	2.5	3.95327	**4.25**
10	250	0	0.9478	26280	432	24498.73440	6248	158	2.5	4.02278	**4.24**
11	250	0	0.89255	26280	432	23070.63240	6248	157	2.5	3.78766	**4.24**
12	250	0	0.7753	26280	432	20039.95440	6248	154	2.5	3.28847	**4.24**
13	300	0	0.63398	26280	432	16387.11504	6248	149	2.4	2.68685	**4.24**
14	300	0	0.49462	26280	432	12784.93776	6248	145	2.3	2.09486	**4.24**
15	300	50	0.35974	26280	432	9298.55952	6248	142	2.3	1.52286	**4.23**
16	350	50	0.22753	26280	432	5881.19544	6248	139	2.2	0.96271	**4.23**
17	350	50	0.11619	26280	432	3003.27912	6248	135	2.2	0.49129	**4.23**
18	400	0	0.03912	26280	432	1011.17376	6248	134	2.1	0.16539	**4.23**
19	0	-50	0.0042	26280	432	108.56160	6248	133	2.1	0.01775	**4.23**
20	0	-50	0.00468	26280	432	120.96864	6248	133	2.1	0.01978	**4.23**
21	0	-50	0.0052	26280	432	134.40960	6248	136	2.2	0.02199	**4.23**
22	0	-50	0.00567	26280	432	146.55816	6248	135	2.2	0.02397	**4.23**
23	0	-50	0.00623	26280	432	161.03304	6248	136	2.2	0.02635	**4.23**
24	500	-250	0.01616	26280	432	417.70368	6248	136	2.2	0.06834	**4.23**

POMONA - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	-50	0.59146	26280	432	15288.05808	6248	138	2.2	2.50214	**4.23**
2	100	0	1.20437	26280	432	31130.55576	6248	140	2.2	5.09669	**4.23**
3	100	0	2.08811	26280	432	53973.46728	6248	142	2.3	8.83941	**4.23**
4	100	0	3.14746	26280	432	81355.54608	6248	145	2.3	13.33042	**4.24**
5	100	0	4.34608	26280	432	112337.47584	6248	147	2.4	18.41296	**4.24**
6	100	0	5.57952	26280	432	144219.43296	6248	152	2.4	23.65804	**4.24**
7	100	0	6.79151	26280	432	175546.95048	6248	154	2.5	28.80652	**4.24**
8	100	0	7.82163	26280	432	202173.49224	6248	157	2.5	33.19217	**4.24**
9	100	0	8.41525	26280	432	217517.38200	6248	159	2.5	35.72301	**4.25**
10	100	0	8.44758	26280	432	218353.04784	6248	158	2.5	35.85436	**4.24**
11	100	0	7.8987	26280	432	204165.59760	6248	157	2.5	33.51922	**4.24**
12	100	0	6.84909	26280	432	177035.27832	6248	154	2.5	29.05075	**4.24**
13	100	0	5.65066	26280	432	146058.25968	6248	149	2.4	23.94790	**4.24**
14	100	0	4.41875	26280	432	114215.85000	6248	145	2.3	18.71471	**4.24**
15	100	0	3.20379	26280	432	82811.56392	6248	142	2.3	13.56233	**4.23**
16	150	0	2.10868	26280	432	54505.16064	6248	139	2.2	8.92211	**4.23**
17	150	0	1.168	26280	432	30190.46400	6248	135	2.2	4.93873	**4.23**
18	200	0	0.48016	26280	432	12411.17568	6248	134	2.1	2.02996	**4.23**
19	500	-200	0.19471	26280	432	5032.86408	6248	133	2.1	0.82304	**4.23**
20	500	0	0.07366	26280	432	1903.96368	6248	133	2.1	0.31136	**4.23**
21	0	-50	0.04644	26280	432	1200.38112	6248	136	2.2	0.19640	**4.23**
22	0	-50	0.05041	26280	432	1302.99768	6248	135	2.2	0.21315	**4.23**
23	0	-50	0.05369	26280	432	1387.77912	6248	136	2.2	0.22706	**4.23**
24	100	-50	0.21115	26280	432	5457.80520	6248	136	2.2	0.89297	**4.23**

POMONA - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	-50	65.9476	26280	432	1704613.56480	6248	138	2.2	278.98749	**4.23**
2	50	0	58.23568	26280	432	1505275.85664	6248	140	2.2	246.44333	**4.23**
3	50	0	70.24739	26280	432	1815754.53672	6248	142	2.3	297.37218	**4.23**
4	50	0	88.80241	26280	432	2295364.69368	6248	145	2.3	376.10432	**4.24**
5	50	0	111.03137	26280	432	2869938.85176	6248	147	2.4	470.40466	**4.24**
6	50	0	135.13711	26280	432	3493024.01928	6248	152	2.4	573.00263	**4.24**
7	50	0	158.47651	26280	432	4096300.83048	6248	154	2.5	672.18589	**4.24**
8	50	0	179.27428	26280	432	4633881.58944	6248	157	2.5	760.77517	**4.24**
9	50	0	197.23857	26280	432	5098222.55736	6248	159	2.5	837.28405	**4.25**
10	50	0	218.81575	26280	432	5655949.50600	6248	158	2.5	928.72734	**4.24**
11	50	0	244.03622	26280	432	6307848.21456	6248	157	2.5	1035.60141	**4.24**
12	50	0	270.93265	26280	432	7003067.13720	6248	154	2.5	1149.17413	**4.24**
13	50	0	285.34864	26280	432	7375691.64672	6248	149	2.4	1209.32803	**4.24**
14	50	0	285.77704	26280	432	7386764.92992	6248	145	2.3	1210.34982	**4.24**
15	50	0	275.07823	26280	432	7110222.08904	6248	142	2.3	1164.46480	**4.23**
16	50	0	256.69684	26280	432	6635099.92032	6248	139	2.2	1086.11883	**4.23**
17	50	0	236.76058	26280	432	6119787.47184	6248	135	2.2	1001.11033	**4.23**
18	50	0	207.98698	26280	432	5376047.45904	6248	134	2.1	879.30119	**4.23**
19	50	0	170.7548	26280	432	4413670.07040	6248	133	2.1	721.77761	**4.23**
20	100	-50	154.35448	26280	432	3989754.59904	6248	133	2.1	652.45374	**4.23**
21	100	-50	130.80712	26280	432	3381102.43776	6248	136	2.2	553.19084	**4.23**
22	100	-50	109.58201	26280	432	2832475.79448	6248	135	2.2	463.35282	**4.23**
23	100	-50	93.63298	26280	432	2420225.26704	6248	136	2.2	395.97926	**4.23**
24	100	-50	78.6095	26280	432	2031898.35600	6248	136	2.2	332.44410	**4.23**

REDLANDS - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-500	0	0.04181	8760	478	346.27042	2088	291	13.9	0.19269	**4.61**
2	150	-100	0.08511	8760	478	704.88102	2088	250	12.0	0.38350	**4.51**
3	150	-100	0.18241	8760	478	1510.71962	2088	209	10.0	0.80400	**4.41**
4	150	-100	0.31173	8760	478	2581.74786	2088	167	8.0	1.34396	**4.31**
5	150	-100	0.45602	8760	478	3776.75764	2088	125	6.0	1.92397	**4.22**
6	200	-100	0.60555	8760	478	5015.16510	2088	84	4.0	2.50258	**4.13**
7	200	-50	0.75634	8760	478	6264.00788	2088	51	2.4	3.07511	**4.07**
8	200	-100	0.88379	8760	478	7319.54878	2088	31	1.5	3.55836	**4.03**
9	200	-50	0.9679	8760	478	8016.14780	2088	25	1.2	3.88568	**4.01**
10	250	-50	0.99231	8760	478	8218.31142	2088	20	1.0	3.97404	**4.00**
11	250	-50	0.94769	8760	478	7848.76858	2088	20	1.0	3.79534	**4.00**
12	250	-50	0.83365	8760	478	6904.28930	2088	21	1.0	3.34025	**4.01**
13	250	-50	0.69935	8760	478	5792.01670	2088	35	1.7	2.82125	**4.03**
14	300	-50	0.54905	8760	478	4547.23210	2088	53	2.5	2.23451	**4.07**
15	300	-50	0.40803	8760	478	3379.30446	2088	83	4.0	1.68544	**4.13**
16	300	-50	0.27569	8760	478	2283.26458	2088	120	5.7	1.16020	**4.21**
17	350	-50	0.15386	8760	478	1274.26852	2088	162	7.8	0.66161	**4.30**
18	400	-50	0.05645	8760	478	467.51890	2088	208	10.0	0.24868	**4.41**
19	-50	0	0.00342	8760	478	28.32444	2088	249	11.9	0.01540	**4.50**
20	-50	0	0.00391	8760	478	32.38262	2088	290	13.9	0.01801	**4.61**
21	-50	0	0.0043	8760	478	35.61260	2088	318	15.2	0.02012	**4.68**
22	-50	0	0.0046	8760	478	38.09720	2088	341	16.3	0.02181	**4.74**
23	-50	0	0.00521	8760	478	43.14922	2088	344	16.5	0.02474	**4.75**
24	-500	50	0.01975	8760	478	163.56950	2088	327	15.7	0.09288	**4.70**

REDLANDS - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	WORKER PERIOD AVE CONC	% WORKER CALM & MISSING HRS	QUOTIENT (FACTOR)
1	-50	0	0.52894	8760	478	4380.68108	2088	291	2.43777	13.9	**4.61**
2	50	-50	1.22841	8760	478	10173.69162	2088	250	5.53520	12.0	**4.51**
3	50	-50	2.14057	8760	478	17728.20074	2088	209	9.43491	10.0	**4.41**
4	50	-50	3.12441	8760	478	25876.36362	2088	167	13.47026	8.0	**4.31**
5	100	-50	4.19282	8760	478	34724.93524	2088	125	17.68973	6.0	**4.22**
6	100	-50	5.31036	8760	478	43980.40152	2088	84	21.94631	4.0	**4.13**
7	100	-50	6.45196	8760	478	53435.13272	2088	51	26.23227	2.4	**4.07**
8	100	-50	7.43242	8760	478	61555.30244	2088	31	29.92479	1.5	**4.03**
9	100	-50	7.96745	8760	478	65986.42090	2088	25	31.98566	1.2	**4.01**
10	100	-50	7.90056	8760	478	65432.43792	2088	20	31.64044	1.0	**4.00**
11	100	-50	7.20298	8760	478	59655.08036	2088	20	28.84675	1.0	**4.00**
12	100	-50	6.14084	8760	478	50858.43688	2088	21	24.60495	1.0	**4.01**
13	100	0	5.07104	8760	478	41998.35328	2088	35	20.45706	1.7	**4.03**
14	150	-50	4.07763	8760	478	33770.93166	2088	53	16.59505	2.5	**4.07**
15	150	0	3.14168	8760	478	26019.39376	2088	83	12.97725	4.0	**4.13**
16	150	0	2.23696	8760	478	18526.50272	2088	120	9.41387	5.7	**4.21**
17	150	0	1.32077	8760	478	10938.61714	2088	162	5.67945	7.8	**4.30**
18	150	0	0.517	8760	478	4281.79400	2088	208	2.27755	10.0	**4.41**
19	500	-100	0.07352	8760	478	608.89264	2088	249	0.33110	11.9	**4.50**
20	-50	0	0.04779	8760	478	395.79678	2088	290	0.22013	13.9	**4.61**
21	-50	0	0.05202	8760	478	430.82964	2088	318	0.24341	15.2	**4.68**
22	-50	0	0.05512	8760	478	456.50384	2088	341	0.26131	16.3	**4.74**
23	-50	0	0.05897	8760	478	488.38954	2088	344	0.28004	16.5	**4.75**
24	-50	0	0.18742	8760	478	1552.21244	2088	327	0.88144	15.7	**4.70**

REDLANDS - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-300	50	45.47894	8760	478	376656.58108	2088	291	13.9	209.60299	**4.61**
2	-50	0	45.80464	8760	478	379354.02848	2088	250	12.0	206.39501	**4.51**
3	-50	0	53.94402	8760	478	446764.37364	2088	209	10.0	237.76710	**4.41**
4	50	0	74.29323	8760	478	615296.53086	2088	167	8.0	320.30012	**4.31**
5	50	0	96.44381	8760	478	798747.63442	2088	125	6.0	406.90149	**4.22**
6	50	0	123.94464	8760	478	1026509.50848	2088	84	4.0	512.23029	**4.13**
7	50	0	151.19332	8760	478	1252183.07624	2088	51	2.4	614.71923	**4.07**
8	50	0	175.86202	8760	478	1456489.24964	2088	31	1.5	708.06478	**4.03**
9	50	0	200.54185	8760	478	1660887.60170	2088	25	1.2	805.08367	**4.01**
10	50	0	230.43001	8760	478	1908421.34282	2088	20	1.0	922.83431	**4.00**
11	50	0	263.81094	8760	478	2184882.20508	2088	20	1.0	1056.51944	**4.00**
12	50	0	299.22627	8760	478	2478191.96814	2088	21	1.0	1198.93177	**4.01**
13	50	0	298.91289	8760	478	2475596.55498	2088	35	1.7	1205.84343	**4.03**
14	50	0	277.77399	8760	478	2300524.18518	2088	53	2.5	1130.47872	**4.07**
15	50	0	252.24911	8760	478	2089127.12902	2088	83	4.0	1041.95867	**4.13**
16	50	0	224.21967	8760	478	1856987.30694	2088	120	5.7	943.59111	**4.21**
17	50	0	190.84881	8760	478	1580609.84442	2088	162	7.8	820.66970	**4.30**
18	50	0	147.20039	8760	478	1219113.62998	2088	208	10.0	648.46470	**4.41**
19	50	0	96.70574	8760	478	800916.93868	2088	249	11.9	435.51764	**4.50**
20	100	-50	65.67926	8760	478	543955.63132	2088	290	13.9	302.53372	**4.61**
21	100	-50	44.74535	8760	478	370580.98870	2088	318	15.2	209.36779	**4.68**
22	-300	50	46.41385	8760	478	384399.50570	2088	341	16.3	220.03406	**4.74**
23	-300	50	48.26296	8760	478	399713.83472	2088	344	16.5	229.19371	**4.75**
24	-300	50	48.06504	8760	478	398074.66128	2088	327	15.7	226.05035	**4.70**

SAN BERNARDINO - 8-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	200	350	0.04085	26280	1292	1020.75980	6248	872	14.0	0.18987	**4.65**
2	100	200	0.09946	26280	1292	2485.30648	6248	823	13.2	0.45812	**4.61**
3	100	150	0.20057	26280	1292	5011.84316	6248	744	11.9	0.91058	**4.54**
4	100	150	0.33332	26280	1292	8329.00016	6248	636	10.2	1.48414	**4.45**
5	150	150	0.48464	26280	1292	12110.18432	6248	526	8.4	2.11643	**4.37**
6	150	150	0.64456	26280	1292	16106.26528	6248	414	6.6	2.76076	**4.28**
7	150	150	0.79252	26280	1292	19803.48976	6248	312	5.0	3.33617	**4.21**
8	150	150	0.92034	26280	1292	22997.45592	6248	206	3.3	3.80627	**4.14**
9	200	200	1.02323	26280	1292	25568.47124	6248	138	2.2	4.18469	**4.09**
10	200	200	1.0794	26280	1292	26972.04720	6248	99	1.6	4.38641	**4.06**
11	200	200	1.04725	26280	1292	26168.68300	6248	87	1.4	4.24747	**4.06**
12	200	200	0.92541	26280	1292	23124.14508	6248	91	1.5	3.75575	**4.06**
13	200	200	0.78218	26280	1292	19545.11384	6248	92	1.5	3.17497	**4.06**
14	250	250	0.6348	26280	1292	15862.38240	6248	109	1.7	2.58387	**4.07**
15	250	250	0.49254	26280	1292	12307.58952	6248	150	2.4	2.01830	**4.10**
16	250	250	0.34312	26280	1292	8573.88256	6248	208	3.3	1.41952	**4.14**
17	300	300	0.19921	26280	1292	4977.85948	6248	282	4.5	0.83437	**4.19**
18	300	300	0.08024	26280	1292	2005.03712	6248	370	5.9	0.34111	**4.25**
19	500	500	0.0042	26280	1292	104.94960	6248	461	7.4	0.01814	**4.32**
20	500	-400	0.00275	26280	1292	68.71700	6248	565	9.0	0.01209	**4.40**
21	-50	0	0.00279	26280	1292	69.71652	6248	674	10.8	0.01251	**4.48**
22	-50	0	0.00305	26280	1292	76.21340	6248	769	12.3	0.01391	**4.56**
23	500	-450	0.00363	26280	1292	90.70644	6248	830	13.3	0.01674	**4.61**
24	500	-400	0.01549	26280	1292	387.06412	6248	878	14.1	0.07208	**4.65**

SAN BERNARDINO - 8-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	100	0.61923	26280	1292	15473.31924	6248	872	14.0	2.87822	**4.65**
2	50	50	1.30694	26280	1292	32657.81672	6248	823	13.2	6.01987	**4.61**
3	50	50	2.2765	26280	1292	56885.18200	6248	744	11.9	10.33524	**4.54**
4	50	50	3.33493	26280	1292	83333.23084	6248	636	10.2	14.84911	**4.45**
5	50	50	4.37187	26280	1292	109244.28756	6248	526	8.4	19.09198	**4.37**
6	50	50	5.37512	26280	1292	134313.49856	6248	414	6.6	23.02254	**4.28**
7	50	100	6.31892	26280	1292	157897.17296	6248	312	5.0	26.59993	**4.21**
8	100	100	7.24372	26280	1292	181006.07536	6248	206	3.3	29.95797	**4.14**
9	100	100	8.1813	26280	1292	204434.32440	6248	138	2.2	33.45897	**4.09**
10	100	100	8.82249	26280	1292	220456.38012	6248	99	1.6	35.85240	**4.06**
11	100	100	8.99277	26280	1292	224711.33676	6248	87	1.4	36.47319	**4.06**
12	100	100	8.30546	26280	1292	207536.83448	6248	91	1.5	33.70746	**4.06**
13	100	100	7.26975	26280	1292	181656.51300	6248	92	1.5	29.50886	**4.06**
14	100	100	6.13035	26280	1292	153185.18580	6248	109	1.7	24.95279	**4.07**
15	100	100	4.96832	26280	1292	124148.38016	6248	150	2.4	20.35887	**4.10**
16	100	100	3.72613	26280	1292	93108.53644	6248	208	3.3	15.41532	**4.14**
17	100	100	2.45722	26280	1292	61401.01336	6248	282	4.5	10.29182	**4.19**
18	150	150	1.45646	26280	1292	36394.02248	6248	370	5.9	6.19157	**4.25**
19	250	300	0.78676	26280	1292	19659.55888	6248	461	7.4	3.39719	**4.32**
20	400	500	0.34453	26280	1292	8609.11564	6248	565	9.0	1.51489	**4.40**
21	400	500	0.1543	26280	1292	3855.64840	6248	674	10.8	0.69172	**4.48**
22	150	-100	0.09964	26280	1292	2489.80432	6248	769	12.3	0.45443	**4.56**
23	150	-100	0.1332	26280	1292	3328.40160	6248	830	13.3	0.61432	**4.61**
24	150	-100	0.22779	26280	1292	5692.01652	6248	878	14.1	1.05997	**4.65**

SAN BERNARDINO - 8-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	100	63.46595	26280	1292	1585887.15860	6248	872	14.0	294.99389	**4.65**
2	0	50	55.96467	26280	1292	1398445.17396	6248	823	13.2	257.77791	**4.61**
3	0	50	65.81835	26280	1292	1644668.92980	6248	744	11.9	298.81340	**4.54**
4	0	50	76.94855	26280	1292	1922790.36740	6248	636	10.2	342.62123	**4.45**
5	0	50	88.11255	26280	1292	2201756.39940	6248	526	8.4	384.78791	**4.37**
6	0	50	98.59945	26280	1292	2463803.05660	6248	414	6.6	422.31797	**4.28**
7	0	50	107.32754	26280	1292	2681900.56952	6248	312	5.0	451.80266	**4.21**
8	0	50	112.73519	26280	1292	2817026.92772	6248	206	3.3	466.24080	**4.14**
9	50	50	120.54293	26280	1292	3012126.73484	6248	138	2.2	492.98310	**4.09**
10	50	50	141.77071	26280	1292	3542566.50148	6248	99	1.6	576.12075	**4.06**
11	50	50	169.40463	26280	1292	4233082.89444	6248	87	1.4	687.07724	**4.06**
12	50	50	207.02118	26280	1292	5173045.24584	6248	91	1.5	840.18926	**4.06**
13	50	50	237.14305	26280	1292	5925730.53340	6248	92	1.5	962.59430	**4.06**
14	50	50	260.28953	26280	1292	6504114.77564	6248	109	1.7	1059.47463	**4.07**
15	50	50	274.82077	26280	1292	6867221.40076	6248	150	2.4	1126.14323	**4.10**
16	50	50	274.32052	26280	1292	6854721.15376	6248	208	3.3	1134.88761	**4.14**
17	50	50	267.24594	26280	1292	6677941.54872	6248	282	4.5	1119.33315	**4.19**
18	50	50	247.00929	26280	1292	6172268.13852	6248	370	5.9	1050.06263	**4.25**
19	50	50	216.76584	26280	1292	5416544.80992	6248	461	7.4	935.98493	**4.32**
20	50	100	173.1904	26280	1292	4327681.71520	6248	565	9.0	761.51359	**4.40**
21	50	100	149.39248	26280	1292	3733019.29024	6248	674	10.8	669.72000	**4.48**
22	50	100	121.76981	26280	1292	3042784.01228	6248	769	12.3	555.35390	**4.56**
23	50	100	100.07427	26280	1292	2500655.85876	6248	830	13.3	461.54593	**4.61**
24	50	100	79.55709	26280	1292	1987972.56492	6248	878	14.1	370.19973	**4.65**

KEARNY MESA - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	150	-150	0.08297	26304	1813	2032.01827	7830	910	11.6	0.29364	**3.54**
2	150	-100	0.15998	26304	1813	3918.07018	7830	907	11.6	0.56595	**3.54**
3	200	-100	0.26694	26304	1813	6537.62754	7830	886	11.3	0.94148	**3.53**
4	200	-100	0.38512	26304	1813	9431.97392	7830	872	11.1	1.35556	**3.52**
5	200	-100	0.50152	26304	1813	12282.72632	7830	856	10.9	1.76122	**3.51**
6	200	-100	0.61064	26304	1813	14955.18424	7830	848	10.8	2.14196	**3.51**
7	200	-100	0.69021	26304	1813	16903.93311	7830	849	10.8	2.42142	**3.51**
8	250	-100	0.73932	26304	1813	18106.68612	7830	817	10.4	2.58187	**3.49**
9	250	-100	0.75042	26304	1813	18378.53622	7830	755	9.6	2.59767	**3.46**
10	250	-100	0.72932	26304	1813	17861.77612	7830	685	8.7	2.49990	**3.43**
11	250	-100	0.68371	26304	1813	16744.74161	7830	645	8.2	2.33051	**3.41**
12	250	-100	0.60961	26304	1813	14929.95851	7830	621	7.9	2.07102	**3.40**
13	250	-100	0.50731	26304	1813	12424.52921	7830	610	7.8	1.72085	**3.39**
14	250	-100	0.38994	26304	1813	9550.02054	7830	593	7.6	1.31961	**3.38**
15	300	-150	0.27924	26304	1813	6838.86684	7830	590	7.5	0.94459	**3.38**
16	300	-150	0.16786	26304	1813	4111.05926	7830	592	7.6	0.56798	**3.38**
17	300	-150	0.07795	26304	1813	1909.07345	7830	606	7.7	0.26427	**3.39**
18	350	-200	0.02278	26304	1813	557.90498	7830	645	8.2	0.07765	**3.41**
19	0	500	0.00482	26304	1813	118.04662	7830	702	9.0	0.01656	**3.44**
20	0	500	0.00483	26304	1813	118.29153	7830	762	9.7	0.01674	**3.47**
21	0	500	0.00496	26304	1813	121.47536	7830	797	10.2	0.01727	**3.48**
22	-50	500	0.00874	26304	1813	214.05134	7830	825	10.5	0.03056	**3.50**
23	-50	500	0.02154	26304	1813	527.53614	7830	859	11.0	0.07568	**3.51**
24	0	300	0.04544	26304	1813	1112.87104	7830	898	11.5	0.16054	**3.53**

KEARNY MESA - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	50	1.35817	26304	1813	33262.94147	7830	910	11.6	4.80678	**3.54**
2	50	50	2.11813	26304	1813	51875.12183	7830	907	11.6	7.49316	**3.54**
3	50	50	2.81323	26304	1813	68898.81593	7830	886	11.3	9.92206	**3.53**
4	50	50	3.40099	26304	1813	83293.64609	7830	872	11.1	11.97092	**3.52**
5	100	-50	4.27704	26304	1813	104748.98664	7830	856	10.9	15.01993	**3.51**
6	100	-50	5.2404	26304	1813	128342.63640	7830	848	10.8	18.38193	**3.51**
7	100	-50	6.03015	26304	1813	147684.40365	7830	849	10.8	21.15519	**3.51**
8	100	-50	6.5101	26304	1813	159438.85910	7830	817	10.4	22.73476	**3.49**
9	100	-50	6.57622	26304	1813	161058.20402	7830	755	9.6	22.76441	**3.46**
10	100	-50	6.3076	26304	1813	154479.43160	7830	685	8.7	21.62063	**3.43**
11	100	-50	5.84464	26304	1813	143141.07824	7830	645	8.2	19.92221	**3.41**
12	100	-50	5.22149	26304	1813	127879.51159	7830	621	7.9	17.73887	**3.40**
13	100	-50	4.43399	26304	1813	108592.84909	7830	610	7.8	15.04056	**3.39**
14	100	-50	3.50471	26304	1813	85833.85261	7830	593	7.6	11.86042	**3.38**
15	100	-50	2.50936	26304	1813	61456.73576	7830	590	7.5	8.48850	**3.38**
16	100	-50	1.54547	26304	1813	37850.10577	7830	592	7.6	5.22936	**3.38**
17	150	-50	0.78926	26304	1813	19329.76666	7830	606	7.7	2.67577	**3.39**
18	200	-100	0.30774	26304	1813	7536.86034	7830	645	8.2	1.04897	**3.41**
19	0	150	0.18342	26304	1813	4492.13922	7830	702	9.0	0.63021	**3.44**
20	0	150	0.16993	26304	1813	4161.75563	7830	762	9.7	0.58882	**3.47**
21	0	150	0.16545	26304	1813	4052.03595	7830	797	10.2	0.57615	**3.48**
22	0	150	0.21125	26304	1813	5173.72375	7830	825	10.5	0.73858	**3.50**
23	0	100	0.41536	26304	1813	10172.58176	7830	859	11.0	1.45927	**3.51**
24	0	100	0.83705	26304	1813	20500.19155	7830	898	11.5	2.95733	**3.53**

KEARNY MESA - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	50	68.76835	26304	1813	1684205.65985	7830	910	11.6	243.38232	**3.54**
2	0	50	74.07187	26304	1813	1814094.16817	7830	907	11.6	262.03874	**3.54**
3	0	50	78.4778	26304	1813	1921999.79980	7830	886	11.3	276.78569	**3.53**
4	50	0	81.98311	26304	1813	2007848.34701	7830	872	11.1	288.56688	**3.52**
5	50	0	99.45639	26304	1813	2435786.44749	7830	856	10.9	349.26677	**3.51**
6	50	0	117.63254	26304	1813	2880938.53714	7830	848	10.8	412.62368	**3.51**
7	50	0	134.71148	26304	1813	3299218.85668	7830	849	10.8	472.59975	**3.51**
8	50	0	151.26253	26304	1813	3704570.62223	7830	817	10.4	528.24335	**3.49**
9	50	0	164.57775	26304	1813	4030673.67525	7830	755	9.6	569.70653	**3.46**
10	50	0	175.05832	26304	1813	4287353.31512	7830	685	8.7	600.04945	**3.43**
11	50	0	176.15086	26304	1813	4314110.71226	7830	645	8.2	600.43295	**3.41**
12	50	0	169.94269	26304	1813	4162066.42079	7830	621	7.9	577.34310	**3.40**
13	50	0	158.91434	26304	1813	3891971.10094	7830	610	7.8	539.05417	**3.39**
14	50	0	144.4592	26304	1813	3537950.26720	7830	593	7.6	488.86973	**3.38**
15	50	-50	129.79889	26304	1813	3178904.61499	7830	590	7.5	439.07522	**3.38**
16	50	-50	127.14583	26304	1813	3113928.52253	7830	592	7.6	430.21947	**3.38**
17	50	-50	122.72119	26304	1813	3005564.66429	7830	606	7.7	416.05269	**3.39**
18	50	-50	111.89165	26304	1813	2740338.40015	7830	645	8.2	381.39713	**3.41**
19	50	-50	97.37192	26304	1813	2384735.69272	7830	702	9.0	334.55888	**3.44**
20	50	-50	76.25987	26304	1813	1867680.47617	7830	762	9.7	264.24455	**3.47**
21	0	50	59.92054	26304	1813	1467513.94514	7830	797	10.2	208.66116	**3.48**
22	0	50	56.81233	26304	1813	1391390.77403	7830	825	10.5	198.62823	**3.50**
23	0	50	58.33987	26304	1813	1428801.75617	7830	859	11.0	204.96367	**3.51**
24	0	50	63.14546	26304	1813	1546495.46086	7830	898	11.5	223.09513	**3.53**

PALOMAR - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	150	50	0.11461	26304	2291	2752.12993	7820	1313	16.8	0.42295	**3.69**
2	150	50	0.21952	26304	2291	5271.33376	7820	1235	15.8	0.80051	**3.65**
3	200	50	0.34291	26304	2291	8234.29783	7820	1156	14.8	1.23564	**3.60**
4	200	50	0.47006	26304	2291	11287.55078	7820	1071	13.7	1.67248	**3.56**
5	200	0	0.59099	26304	2291	14191.44287	7820	985	12.6	2.07629	**3.51**
6	200	0	0.70014	26304	2291	16812.46182	7820	902	11.5	2.43025	**3.47**
7	250	0	0.78328	26304	2291	18808.90264	7820	951	12.2	2.73823	**3.50**
8	250	0	0.83593	26304	2291	20073.18709	7820	858	11.0	2.88325	**3.45**
9	250	0	0.84409	26304	2291	20269.13317	7820	757	9.7	2.86976	**3.40**
10	250	0	0.8161	26304	2291	19597.00930	7820	663	8.5	2.73816	**3.36**
11	250	0	0.75885	26304	2291	18222.26505	7820	623	8.0	2.53193	**3.34**
12	250	0	0.66899	26304	2291	16064.45687	7820	623	8.0	2.23210	**3.34**
13	250	0	0.54882	26304	2291	13178.81466	7820	656	8.4	1.83959	**3.35**
14	250	0	0.41206	26304	2291	9894.79678	7820	710	9.1	1.39167	**3.38**
15	300	0	0.27978	26304	2291	6718.35714	7820	766	9.8	0.95242	**3.40**
16	300	-50	0.16245	26304	2291	3900.91185	7820	842	10.8	0.55903	**3.44**
17	300	-100	0.08094	26304	2291	1943.61222	7820	779	10.0	0.27604	**3.41**
18	300	-150	0.02496	26304	2291	599.36448	7820	876	11.2	0.08631	**3.46**
19	-450	-200	0.00494	26304	2291	118.62422	7820	978	12.5	0.01734	**3.51**
20	-400	-150	0.00466	26304	2291	111.90058	7820	1085	13.9	0.01661	**3.57**
21	-400	-200	0.00408	26304	2291	97.97304	7820	1179	15.1	0.01475	**3.62**
22	-500	-250	0.00734	26304	2291	176.25542	7820	1254	16.0	0.02684	**3.66**
23	-50	250	0.01896	26304	2291	455.28648	7820	1312	16.8	0.06996	**3.69**
24	100	150	0.05053	26304	2291	1213.37689	7820	1336	17.1	0.18713	**3.70**

PALOMAR - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	50	1.79401	26304	2291	43079.56213	7820	1313	16.8	6.62050	**3.69**
2	50	50	2.7745	26304	2291	66624.06850	7820	1235	15.8	10.11755	**3.65**
3	100	0	4.02097	26304	2291	96555.55261	7820	1156	14.8	14.48913	**3.60**
4	100	0	5.71297	26304	2291	137185.54861	7820	1071	13.7	20.32680	**3.56**
5	100	0	7.47105	26304	2291	179402.32365	7820	985	12.6	26.24760	**3.51**
6	100	0	9.08402	26304	2291	218134.57226	7820	902	11.5	31.53145	**3.47**
7	100	0	10.25315	26304	2291	246208.89095	7820	951	12.2	35.84348	**3.50**
8	100	0	10.98429	26304	2291	263765.75577	7820	858	11.0	37.88649	**3.45**
9	100	0	11.11226	26304	2291	266838.69938	7820	757	9.7	37.77980	**3.40**
10	100	0	10.70486	26304	2291	257055.80318	7820	663	8.5	35.91670	**3.36**
11	100	0	9.8762	26304	2291	237157.19060	7820	623	8.0	32.95223	**3.34**
12	100	0	8.79903	26304	2291	211291.10739	7820	623	8.0	29.35822	**3.34**
13	100	0	7.34081	26304	2291	176274.87053	7820	656	8.4	24.60565	**3.35**
14	100	0	5.64239	26304	2291	135490.71107	7820	710	9.1	19.05636	**3.38**
15	100	0	3.89019	26304	2291	93415.13247	7820	766	9.8	13.24286	**3.40**
16	100	0	2.28302	26304	2291	54822.15926	7820	842	10.8	7.85643	**3.44**
17	150	0	1.19218	26304	2291	28627.81834	7820	779	10.0	4.06587	**3.41**
18	150	0	0.42743	26304	2291	10263.87659	7820	876	11.2	1.47809	**3.46**
19	500	100	0.13519	26304	2291	3246.31747	7820	978	12.5	0.47447	**3.51**
20	-100	-50	0.11603	26304	2291	2786.22839	7820	1085	13.9	0.41369	**3.57**
21	-100	-50	0.1019	26304	2291	2446.92470	7820	1179	15.1	0.36846	**3.62**
22	-100	0	0.13253	26304	2291	3182.44289	7820	1254	16.0	0.48469	**3.66**
23	-50	50	0.32155	26304	2291	7721.38015	7820	1312	16.8	1.18644	**3.69**
24	50	50	0.91054	26304	2291	21864.79702	7820	1336	17.1	3.37212	**3.70**

PALOMAR - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	0	64.60191	26304	2291	1551285.66483	7820	1313	16.8	238.40259	**3.69**
2	50	0	67.16566	26304	2291	1612848.99358	7820	1235	15.8	244.92771	**3.65**
3	50	0	86.7754	26304	2291	2083737.68020	7820	1156	14.8	312.68573	**3.60**
4	50	0	111.35187	26304	2291	2673892.45431	7820	1071	13.7	396.19091	**3.56**
5	50	0	139.09175	26304	2291	3340010.19275	7820	985	12.6	488.66279	**3.51**
6	50	0	167.58523	26304	2291	4024224.12799	7820	902	11.5	581.70340	**3.47**
7	50	0	194.22411	26304	2291	4663903.55343	7820	951	12.2	678.97853	**3.50**
8	50	0	224.85236	26304	2291	5399379.72068	7820	858	11.0	775.55009	**3.45**
9	50	0	252.42285	26304	2291	6061429.89705	7820	757	9.7	858.19480	**3.40**
10	50	0	275.34655	26304	2291	6611896.70515	7820	663	8.5	923.83634	**3.36**
11	50	0	282.82242	26304	2291	6791414.77146	7820	623	8.0	943.64524	**3.34**
12	50	0	277.9957	26304	2291	6675510.74410	7820	623	8.0	927.54075	**3.34**
13	50	0	262.24815	26304	2291	6297364.82595	7820	656	8.4	879.02915	**3.35**
14	50	0	239.25516	26304	2291	5745234.15708	7820	710	9.1	808.04981	**3.38**
15	50	0	213.26193	26304	2291	5121058.72509	7820	766	9.8	725.97941	**3.40**
16	50	0	185.3631	26304	2291	4451124.12030	7820	842	10.8	637.87964	**3.44**
17	50	0	158.33517	26304	2291	3802102.43721	7820	779	10.0	539.99467	**3.41**
18	50	0	125.85979	26304	2291	3022271.13727	7820	876	11.2	435.23490	**3.46**
19	50	0	93.2437	26304	2291	2239060.96810	7820	978	12.5	327.25241	**3.51**
20	50	0	62.12509	26304	2291	1491809.78617	7820	1085	13.9	221.50108	**3.57**
21	-50	0	47.17899	26304	2291	1132909.08687	7820	1179	15.1	170.59315	**3.62**
22	-50	0	51.9114	26304	2291	1246548.44820	7820	1254	16.0	189.84899	**3.66**
23	-50	0	57.95502	26304	2291	1391673.89526	7820	1312	16.8	213.84049	**3.69**
24	-50	0	62.2143	26304	2291	1493951.98590	7820	1336	17.1	230.40592	**3.70**

POMONA - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	0	1.67498	26280	432	43294.88304	7810	175	2.2	5.67058	**3.39**
2	100	0	2.52254	26280	432	65202.61392	7810	179	2.3	8.54444	**3.39**
3	100	0	3.48087	26280	432	89973.52776	7810	183	2.3	11.79671	**3.39**
4	100	0	4.46874	26280	432	115507.99152	7810	188	2.4	15.15455	**3.39**
5	100	0	5.44049	26280	432	140625.78552	7810	189	2.4	18.45241	**3.39**
6	100	0	6.37933	26280	432	164892.92184	7810	192	2.5	21.64517	**3.39**
7	100	0	7.16963	26280	432	185320.59624	7810	193	2.5	24.32987	**3.39**
8	100	0	7.58985	26280	432	196182.44280	7810	193	2.5	25.75587	**3.39**
9	100	0	7.54073	26280	432	194912.78904	7810	194	2.5	25.59254	**3.39**
10	100	0	7.03831	26280	432	181926.23688	7810	193	2.5	23.88424	**3.39**
11	100	0	6.33091	26280	432	163641.36168	7810	190	2.4	21.47524	**3.39**
12	100	0	5.48577	26280	432	141796.18296	7810	188	2.4	18.60354	**3.39**
13	100	0	4.52666	26280	432	117005.10768	7810	184	2.4	15.34292	**3.39**
14	100	0	3.53869	26280	432	91468.05912	7810	179	2.3	11.98638	**3.39**
15	100	0	2.56683	26280	432	66347.42184	7810	174	2.2	8.68877	**3.39**
16	150	0	1.68973	26280	432	43676.14104	7810	170	2.2	5.71677	**3.38**
17	150	0	0.93943	26280	432	24282.38664	7810	168	2.2	3.17749	**3.38**
18	200	0	0.38972	26280	432	10073.48256	7810	168	2.2	1.31817	**3.38**
19	500	-200	0.15933	26280	432	4118.36184	7810	169	2.2	0.53898	**3.38**
20	500	0	0.06427	26280	432	1661.25096	7810	169	2.2	0.21741	**3.38**
21	0	-50	0.04922	26280	432	1272.23856	7810	171	2.2	0.16655	**3.38**
22	100	-50	0.17372	26280	432	4490.31456	7810	170	2.2	0.58774	**3.38**
23	100	-50	0.47768	26280	432	12347.07264	7810	170	2.2	1.61611	**3.38**
24	100	0	0.96732	26280	432	25003.28736	7810	171	2.2	3.27311	**3.38**

POMONA - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	200	-50	0.14539	26280	432	3758.04072	7810	175	2.2	0.49221	**3.39**
2	200	-50	0.24454	26280	432	6320.86992	7810	179	2.3	0.82831	**3.39**
3	200	-50	0.35936	26280	432	9288.73728	7810	183	2.3	1.21788	**3.39**
4	200	0	0.475	26280	432	12277.80000	7810	188	2.4	1.61084	**3.39**
5	200	0	0.58245	26280	432	15055.16760	7810	189	2.4	1.97548	**3.39**
6	250	0	0.68649	26280	432	17744.39352	7810	192	2.5	2.32927	**3.39**
7	250	0	0.77125	26280	432	19935.27000	7810	193	2.5	2.61721	**3.39**
8	250	0	0.81936	26280	432	21178.81728	7810	193	2.5	2.78047	**3.39**
9	250	0	0.82376	26280	432	21292.54848	7810	194	2.5	2.79577	**3.39**
10	250	0	0.78241	26280	432	20223.73368	7810	193	2.5	2.65508	**3.39**
11	250	0	0.7142	26280	432	18460.64160	7810	190	2.4	2.42266	**3.39**
12	250	0	0.62035	26280	432	16034.80680	7810	188	2.4	2.10375	**3.39**
13	300	0	0.50729	26280	432	13112.43192	7810	184	2.4	1.71944	**3.39**
14	300	0	0.39583	26280	432	10231.41384	7810	179	2.3	1.34077	**3.39**
15	300	50	0.28793	26280	432	7442.41464	7810	174	2.2	0.97465	**3.39**
16	350	50	0.18215	26280	432	4708.21320	7810	170	2.2	0.61626	**3.38**
17	350	50	0.09308	26280	432	2405.93184	7810	168	2.2	0.31483	**3.38**
18	400	0	0.03142	26280	432	812.14416	7810	168	2.2	0.10627	**3.38**
19	0	-50	0.00464	26280	432	119.93472	7810	169	2.2	0.01570	**3.38**
20	0	-50	0.00508	26280	432	131.30784	7810	169	2.2	0.01718	**3.38**
21	0	-50	0.00569	26280	432	147.07512	7810	171	2.2	0.01925	**3.38**
22	500	-250	0.01302	26280	432	336.54096	7810	170	2.2	0.04405	**3.38**
23	300	-100	0.0304	26280	432	785.77920	7810	170	2.2	0.10285	**3.38**
24	200	-50	0.07176	26280	432	1854.85248	7810	171	2.2	0.24281	**3.38**

POMONA - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKE HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	0	66.88293	26280	432	1728789.97464	7810	175	2.2	226.42960	**3.39**
2	50	0	78.93616	26280	432	2040341.86368	7810	179	2.3	267.37542	**3.39**
3	50	0	94.94525	26280	432	2454144.82200	7810	183	2.3	321.77066	**3.39**
4	50	0	113.62804	26280	432	2937057.57792	7810	188	2.4	385.33949	**3.39**
5	50	0	133.76259	26280	432	3457495.42632	7810	189	2.4	453.68002	**3.39**
6	50	0	155.21512	26280	432	4012000.42176	7810	192	2.5	526.64747	**3.39**
7	50	0	174.83572	26280	432	4519153.69056	7810	193	2.5	593.29837	**3.39**
8	50	0	196.43289	26280	432	5077397.34072	7810	193	2.5	666.58755	**3.39**
9	50	0	221.2805	26280	432	5719658.36400	7810	194	2.5	751.00556	**3.39**
10	50	0	249.09373	26280	432	6438574.73304	7810	193	2.5	845.29011	**3.39**
11	50	0	267.02625	26280	432	6902094.51000	7810	190	2.4	905.78668	**3.39**
12	50	0	271.20773	26280	432	7010177.40504	7810	188	2.4	919.72939	**3.39**
13	50	0	265.00007	26280	432	6849721.80936	7810	184	2.4	898.20637	**3.39**
14	50	0	252.4629	26280	432	6525661.03920	7810	179	2.3	855.15149	**3.39**
15	50	0	237.46298	26280	432	6137943.10704	7810	174	2.2	803.81654	**3.39**
16	50	0	219.40304	26280	432	5671129.77792	7810	170	2.2	742.29447	**3.38**
17	50	0	200.09348	26280	432	5172016.27104	7810	168	2.2	676.78831	**3.38**
18	50	0	174.28381	26280	432	4504887.92088	7810	168	2.2	589.49070	**3.38**
19	100	-50	148.72624	26280	432	3844275.85152	7810	169	2.2	503.11162	**3.38**
20	100	-50	136.06151	26280	432	3516917.91048	7810	169	2.2	460.26932	**3.38**
21	100	-50	116.42089	26280	432	3009247.16472	7810	171	2.2	393.93208	**3.38**
22	100	-50	95.89973	26280	432	2478816.22104	7810	170	2.2	324.45238	**3.38**
23	100	-50	79.98215	26280	432	2067378.61320	7810	170	2.2	270.59929	**3.38**
24	100	-50	67.81091	26280	432	1752776.40168	7810	171	2.2	229.45103	**3.38**

REDLANDS - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	150	-100	0.14613	8760	478	1210.24866	2610	303	11.6	0.52460	**3.59**
2	150	-100	0.24958	8760	478	2067.02156	2610	258	9.9	0.87884	**3.52**
3	150	-100	0.36502	8760	478	3023.09564	2610	216	8.3	1.26278	**3.46**
4	200	-100	0.4846	8760	478	4013.45720	2610	172	6.6	1.64621	**3.40**
5	200	-50	0.6053	8760	478	5013.09460	2610	128	4.9	2.01978	**3.34**
6	200	-100	0.71152	8760	478	5892.80864	2610	86	3.3	2.33471	**3.28**
7	200	-50	0.79696	8760	478	6600.42272	2610	54	2.1	2.58233	**3.24**
8	250	-50	0.85358	8760	478	7069.34956	2610	36	1.4	2.74645	**3.22**
9	250	-50	0.87022	8760	478	7207.16204	2610	32	1.2	2.79564	**3.21**
10	250	-50	0.82892	8760	478	6865.11544	2610	29	1.1	2.65987	**3.21**
11	250	-50	0.75826	8760	478	6279.90932	2610	42	1.6	2.44545	**3.23**
12	250	-50	0.66701	8760	478	5524.17682	2610	58	2.2	2.16465	**3.25**
13	250	-50	0.55959	8760	478	4634.52438	2610	86	3.3	1.83618	**3.28**
14	300	-50	0.43933	8760	478	3638.53106	2610	122	4.7	1.46243	**3.33**
15	300	-50	0.32652	8760	478	2704.23864	2610	165	6.3	1.10603	**3.39**
16	300	-50	0.22066	8760	478	1827.50612	2610	213	8.2	0.76241	**3.46**
17	350	-50	0.12319	8760	478	1020.25958	2610	256	9.8	0.43342	**3.52**
18	400	-50	0.04524	8760	478	374.67768	2610	299	11.5	0.16213	**3.58**
19	-50	0	0.0038	8760	478	31.47160	2610	340	13.0	0.01386	**3.65**
20	-50	0	0.00417	8760	478	34.53594	2610	378	14.5	0.01547	**3.71**
21	-50	0	0.00479	8760	478	39.67078	2610	395	15.1	0.01791	**3.74**
22	-500	50	0.01591	8760	478	131.76662	2610	396	15.2	0.05952	**3.74**
23	-500	0	0.03356	8760	478	277.94392	2610	373	14.3	0.12425	**3.70**
24	150	-100	0.06827	8760	478	565.41214	2610	343	13.1	0.24941	**3.65**

REDLANDS - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	-50	1.71658	8760	478	14216.71556	2610	303	11.6	6.16243	**3.59**
2	50	-50	2.50366	8760	478	20735.31212	2610	258	9.9	8.81603	**3.52**
3	100	-50	3.35706	8760	478	27803.17092	2610	216	8.3	11.61369	**3.46**
4	100	-50	4.25095	8760	478	35206.36790	2610	172	6.6	14.44068	**3.40**
5	100	-50	5.1653	8760	478	42779.01460	2610	128	4.9	17.23570	**3.34**
6	100	-50	6.01292	8760	478	49799.00344	2610	86	3.3	19.73019	**3.28**
7	100	-50	6.72041	8760	478	55658.43562	2610	54	2.1	21.77560	**3.24**
8	100	-50	7.11772	8760	478	58948.95704	2610	36	1.4	22.90169	**3.22**
9	100	-50	7.01506	8760	478	58098.72692	2610	32	1.2	22.53636	**3.21**
10	100	-50	6.50262	8760	478	53854.69884	2610	29	1.1	20.86583	**3.21**
11	100	-50	5.76643	8760	478	47757.57326	2610	42	1.6	18.59719	**3.23**
12	100	-50	4.91534	8760	478	40708.84588	2610	58	2.2	15.95174	**3.25**
13	100	0	4.05934	8760	478	33619.45388	2610	86	3.3	13.31991	**3.28**
14	150	-50	3.26436	8760	478	27035.42952	2610	122	4.7	10.86633	**3.33**
15	150	0	2.51516	8760	478	20830.55512	2610	165	6.3	8.51965	**3.39**
16	150	0	1.79145	8760	478	14836.78890	2610	213	8.2	6.18973	**3.46**
17	150	0	1.05852	8760	478	8766.66264	2610	256	9.8	3.72416	**3.52**
18	150	0	0.41545	8760	478	3440.75690	2610	299	11.5	1.48886	**3.58**
19	500	-100	0.05953	8760	478	493.02746	2610	340	13.0	0.21719	**3.65**
20	-50	0	0.05022	8760	478	415.92204	2610	378	14.5	0.18635	**3.71**
21	-50	0	0.05482	8760	478	454.01924	2610	395	15.1	0.20497	**3.74**
22	-50	0	0.15882	8760	478	1315.34724	2610	396	15.2	0.59410	**3.74**
23	-50	0	0.43321	8760	478	3587.84522	2610	373	14.3	1.60386	**3.70**
24	50	-50	0.98664	8760	478	8171.35248	2610	343	13.1	3.60448	**3.65**

REDLANDS - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	-50	0	45.3508	8760	478	375595.32560	2610	303	11.6	162.80682	**3.59**
2	50	0	60.52773	8760	478	501290.65986	2610	258	9.9	213.13378	**3.52**
3	50	0	78.2791	8760	478	648307.50620	2610	216	8.3	270.80514	**3.46**
4	50	0	100.35242	8760	478	831118.74244	2610	172	6.6	340.90186	**3.40**
5	50	0	123.30279	8760	478	1021193.70678	2610	128	4.9	411.43985	**3.34**
6	50	0	147.25117	8760	478	1219534.18994	2610	86	3.3	483.17519	**3.28**
7	50	0	173.53484	8760	478	1437215.54488	2610	54	2.1	562.29090	**3.24**
8	50	0	204.41071	8760	478	1692929.50022	2610	36	1.4	657.70377	**3.22**
9	50	0	237.08429	8760	478	1963532.08978	2610	32	1.2	761.64938	**3.21**
10	50	0	270.99063	8760	478	2244344.39766	2610	29	1.1	869.56389	**3.21**
11	50	0	274.80034	8760	478	2275896.41588	2610	42	1.6	886.25250	**3.23**
12	50	0	263.13703	8760	478	2179300.88246	2610	58	2.2	853.95803	**3.25**
13	50	0	247.94703	8760	478	2053497.30246	2610	86	3.3	813.58847	**3.28**
14	50	0	227.47119	8760	478	1883916.39558	2610	122	4.7	757.20112	**3.33**
15	50	0	205.25923	8760	478	1699956.94286	2610	165	6.3	695.27891	**3.39**
16	50	0	181.48141	8760	478	1503029.03762	2610	213	8.2	627.04591	**3.46**
17	50	0	154.0154	8760	478	1275555.54280	2610	256	9.8	541.86727	**3.52**
18	50	0	118.85346	8760	478	984344.35572	2610	299	11.5	425.93871	**3.58**
19	50	0	78.48865	8760	478	650042.99930	2610	340	13.0	286.36255	**3.65**
20	100	-50	55.02469	8760	478	455714.48258	2610	378	14.5	204.17316	**3.71**
21	-300	50	46.19985	8760	478	382627.15770	2610	395	15.1	172.74364	**3.74**
22	-300	50	45.56241	8760	478	377347.87962	2610	396	15.2	170.43716	**3.74**
23	-300	50	43.32203	8760	478	358793.05246	2610	373	14.3	160.39028	**3.70**
24	-300	50	40.49639	8760	478	335391.10198	2610	343	13.1	147.94491	**3.65**

SAN BERNARDINO - 10-HOUR ANALYSIS - LARGE POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	100	150	0.16062	26280	2291	3853.11318	7810	945	12.1	0.56127	**3.49**
2	100	150	0.26681	26280	2291	6400.50509	7810	857	11.0	0.92054	**3.45**
3	150	150	0.38784	26280	2291	9303.89376	7810	768	9.8	1.32120	**3.41**
4	150	150	0.51578	26280	2291	12373.04642	7810	659	8.4	1.73025	**3.35**
5	150	150	0.63431	26280	2291	15216.46259	7810	547	7.0	2.09507	**3.30**
6	150	150	0.74255	26280	2291	17813.03195	7810	433	5.5	2.41467	**3.25**
7	200	200	0.84805	26280	2291	20343.87145	7810	332	4.3	2.72050	**3.21**
8	200	200	0.92818	26280	2291	22266.11002	7810	229	2.9	2.93709	**3.16**
9	200	200	0.95389	26280	2291	22882.86721	7810	160	2.0	2.99122	**3.14**
10	200	200	0.91165	26280	2291	21869.57185	7810	125	1.6	2.84575	**3.12**
11	200	200	0.83833	26280	2291	20110.69837	7810	116	1.5	2.61382	**3.12**
12	200	200	0.74042	26280	2291	17761.93538	7810	132	1.7	2.31335	**3.12**
13	200	200	0.6259	26280	2291	15014.71510	7810	171	2.2	1.96553	**3.14**
14	250	250	0.50812	26280	2291	12189.29068	7810	227	2.9	1.60745	**3.16**
15	250	250	0.39411	26280	2291	9454.30479	7810	302	3.9	1.25923	**3.20**
16	250	250	0.27457	26280	2291	6586.65973	7810	393	5.0	0.88805	**3.23**
17	300	300	0.15944	26280	2291	3824.80616	7810	483	6.2	0.52202	**3.27**
18	300	300	0.06426	26280	2291	1541.53314	7810	591	7.6	0.21354	**3.32**
19	500	500	0.00341	26280	2291	81.80249	7810	703	9.0	0.01151	**3.38**
20	-50	0	0.00273	26280	2291	65.48997	7810	810	10.4	0.00936	**3.43**
21	500	-400	0.00355	26280	2291	85.16095	7810	909	11.6	0.01234	**3.48**
22	500	-400	0.01276	26280	2291	306.09964	7810	996	12.8	0.04492	**3.52**
23	200	350	0.03276	26280	2291	785.87964	7810	1024	13.1	0.11581	**3.54**
24	100	200	0.07971	26280	2291	1912.16319	7810	1008	12.9	0.28112	**3.53**

SAN BERNARDINO - 10-HOUR ANALYSIS - MEDIUM POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	50	50	1.82487	26280	2291	43776.80643	7810	945	12.1	6.37681	**3.49**
2	50	50	2.67182	26280	2291	64094.28998	7810	857	11.0	9.21822	**3.45**
3	50	50	3.50148	26280	2291	83997.00372	7810	768	9.8	11.92800	**3.41**
4	50	50	4.30472	26280	2291	103265.92808	7810	659	8.4	14.44077	**3.35**
5	50	100	5.06866	26280	2291	121592.08474	7810	547	7.0	16.74130	**3.30**
6	100	100	5.91978	26280	2291	142009.60242	7810	433	5.5	19.25032	**3.25**
7	100	100	6.91876	26280	2291	165974.13364	7810	332	4.3	22.19499	**3.21**
8	100	100	7.75458	26280	2291	186024.61962	7810	229	2.9	24.53827	**3.16**
9	100	100	8.257	26280	2291	198077.17300	7810	160	2.0	25.89244	**3.14**
10	100	100	8.0456	26280	2291	193005.89840	7810	125	1.6	25.11463	**3.12**
11	100	100	7.431	26280	2291	178262.25900	7810	116	1.5	23.16900	**3.12**
12	100	100	6.66787	26280	2291	159955.53343	7810	132	1.7	20.83297	**3.12**
13	100	100	5.82847	26280	2291	139819.16683	7810	171	2.2	18.30333	**3.14**
14	100	100	4.91446	26280	2291	117892.98094	7810	227	2.9	15.54701	**3.16**
15	100	100	3.97902	26280	2291	95452.71078	7810	302	3.9	12.71347	**3.20**
16	100	100	2.9845	26280	2291	71595.17050	7810	393	5.0	9.65285	**3.23**
17	100	100	1.96987	26280	2291	47255.21143	7810	483	6.2	6.44946	**3.27**
18	150	150	1.16932	26280	2291	28050.81748	7810	591	7.6	3.88569	**3.32**
19	250	300	0.63256	26280	2291	15174.48184	7810	703	9.0	2.13515	**3.38**
20	400	500	0.28079	26280	2291	6735.87131	7810	810	10.4	0.96227	**3.43**
21	400	500	0.14007	26280	2291	3360.13923	7810	909	11.6	0.48691	**3.48**
22	150	-100	0.19283	26280	2291	4625.79887	7810	996	12.8	0.67887	**3.52**
23	50	100	0.50387	26280	2291	12087.33743	7810	1024	13.1	1.78122	**3.54**
24	50	50	1.0492	26280	2291	25169.25880	7810	1008	12.9	3.70027	**3.53**

SAN BERNARDINO - 10-HOUR ANALYSIS - SMALL POINT SOURCE

SCENARIO	X	Y	MODELED PERIOD AVE CONC	TOTAL HRS PROCESSED REPORTED BY AERMOD	NO. CALM & MISSING HRS REPORTED BY AERMOD	SUM HRLY CONC	TOTAL WORKER HRS PROCESSED	WORKER NO. CALM & MISSING HRS	% WORKER CALM & MISSING HRS	WORKER PERIOD AVE CONC	QUOTIENT (FACTOR)
1	0	50	60.43292	26280	2291	1449725.31788	7810	945	12.1	211.17630	**3.49**
2	0	50	69.41259	26280	2291	1665138.62151	7810	857	11.0	239.48492	**3.45**
3	0	50	77.69048	26280	2291	1863716.92472	7810	768	9.8	264.65733	**3.41**
4	0	50	85.534	26280	2291	2051875.12600	7810	659	8.4	286.93541	**3.35**
5	0	50	93.35436	26280	2291	2239477.74204	7810	547	7.0	308.34060	**3.30**
6	0	50	100.18756	26280	2291	2403399.37684	7810	433	5.5	325.79631	**3.25**
7	50	50	106.42361	26280	2291	2552995.98029	7810	332	4.3	341.40091	**3.21**
8	50	50	125.22838	26280	2291	3004103.60782	7810	229	2.9	396.26746	**3.16**
9	50	50	150.67387	26280	2291	3614515.46743	7810	160	2.0	472.48568	**3.14**
10	50	50	184.43774	26280	2291	4424476.94486	7810	125	1.6	575.72895	**3.12**
11	50	50	211.62126	26280	2291	5076582.40614	7810	116	1.5	659.81055	**3.12**
12	50	50	232.56731	26280	2291	5579057.19959	7810	132	1.7	726.62897	**3.12**
13	50	50	246.19103	26280	2291	5905876.61867	7810	171	2.2	773.12169	**3.14**
14	50	50	248.55743	26280	2291	5962644.18827	7810	227	2.9	786.31731	**3.16**
15	50	50	246.83969	26280	2291	5921437.32341	7810	302	3.9	788.68371	**3.20**
16	50	50	238.7665	26280	2291	5727769.56850	7810	393	5.0	772.24883	**3.23**
17	50	50	227.65219	26280	2291	5461148.38591	7810	483	6.2	745.34576	**3.27**
18	50	50	209.04015	26280	2291	5014664.15835	7810	591	7.6	694.64803	**3.32**
19	50	50	182.12183	26280	2291	4368920.57987	7810	703	9.0	614.73485	**3.38**
20	50	100	150.39433	26280	2291	3607809.58237	7810	810	10.4	515.40137	**3.43**
21	50	100	130.14718	26280	2291	3122100.70102	7810	909	11.6	452.41280	**3.48**
22	50	100	105.33813	26280	2291	2526956.40057	7810	996	12.8	370.84773	**3.52**
23	50	100	85.36188	26280	2291	2047746.13932	7810	1024	13.1	301.76041	**3.54**
24	50	100	68.96638	26280	2291	1654434.48982	7810	1008	12.9	243.22765	**3.53**

EXHIBIT 8

Final Report

Ultra-Low NO_x Natural Gas Vehicle Evaluation ISL G NZ

November 2016

Submitted by:

Author: Dr. Kent Johnson (PI)
PhD. Candidate Yu (Jade) Jiang
PhD. Candidate Jiacheng (Joey) Yang

College of Engineering-Center for Environmental Research and Technology
University of California
Riverside, CA 92521
(951) 781-5791
(951) 781-5790 fax

Disclaimer

This report was prepared as a result of work sponsored in part by the California Energy Commission (Commission), the South Coast Air Quality Management District (SCAQMD), Southern California Gas Company (SoCalGas). It does not necessarily represent the views of the Commission, SCAQMD, or SoCal Gas, their employees, or the State of California. The Commission, SCAQMD, SoCalGas, the State of California, their employees, contractors, and subcontractors make no warranty, express or implied, and assume no legal liability for the information in this report; nor does any party represent that the use of this information will not infringe upon privately owned rights. This report has not been approved or disapproved by the Commission nor has the Commission passed upon the accuracy or adequacy of the information in this report.

The statements and conclusions in this report are those of the author and not necessarily those of Cummins Westport, Inc. The mention of commercial products, their source, or their use in connection with material reported herein is not to be construed as actual or implied endorsement of such products.

Inquiries related to this final report should be directed to Kent Johnson (951) 781 5786, kjohnson@cert.ucr.edu.

Acknowledgments

The work reported herein was performed for Cummins Westport, Inc., as part of SCAQMD Contract No. 15626 with Cummins Westport, Inc. The work was partially funded by SCAQMD and by CEC Contract 600-13-008 with SCAQMD and SoCalGas Agreement No. 5662230866 with SCAQMD.

The authors acknowledge Mr. Don Pacocha, Mr. Eddie O'Neil, Mr. Mark Villa, and Mr. Daniel Gomez of CE-CERT for performing the tests and preparing the equipment for testing and Ms. Rachael Hirst for her analytical support for the particulate matter laboratory measurements.

Table of Contents

List of Tables

List of Figures

Abstract

Heavy duty on-road vehicles represent one of the largest sources of NO_x emissions and fuel consumption in North America. Heavy duty vehicles are predominantly diesels, with the recent interest in natural gas (NG) systems. As emissions and greenhouse gas regulations continue to tighten new opportunities for advanced fleet specific heavy duty vehicles are becoming available with improved fuel economy. NO_x emissions have dropped 90% for heavy duty vehicles with the recent 2010 certification limit. Additional NO_x reductions of another 90% are desired for the South Coast Air basin to meet its 2023 NOx inventory requirements.

Although the 2010 certification standards were designed to reduce NO_x emissions, the in-use NO_x emissions are actually much higher than certification standards. The main reason is a result of the poor performance of aftertreatment systems for diesel vehicles during low duty cycle operation. Recent studies by UCR suggest 99% of the operation within 10 miles of the ports represented by up to 1 g/bhp-hr. Thus, a real NO_x success will not only be providing a solution that is independent of duty cycle, but one that also reduces the emissions an additional 90% from the current 2010 standard.

The ISL G NZ 8.9 liter NG engine met and exceeded the target NOx emissions of 0.02 g/bhp-hr and maintained those emissions during a full ration of duty cycles found in the South Coast Air Basin. The other gaseous, particulate matter, particle number and selected non regulated emissions were similar to previous levels. It is expected NG vehicles could play a role in the reduction of the south coast NOx inventory problem given their near zero emission factors demonstrated.

Acronyms and Abbreviations

ARB	Air Resources Board
bs	brake specific
CE-CERT	College of Engineering-Center for Environmental Research and Technology (University of California, Riverside)
CFR	Code of Federal Regulations
CO	carbon monoxide
CO_2	carbon dioxide
CNG	compressed natural gas
CWI	Cummins Westport Inc.
FID	flame ionization detector
NH_3	ammonia
g/bhp-hr	grams per brake horsepower hour
lpm	liters per minute
MEL	mobile emission laboratory
NO_x	nitrogen oxides
N_2O	nitrous oxides
OEM	original equipment manufacturer
PM	particulate matter
$PM_{2.5}$	ultra-fine particulate matter less than 2.5 µm (certification gravimetric reference method)
PN	particle number
PSD	particle size distribution
RPM	revolutions per minute
scfm	standard cubic feet per minute
THC	total hydrocarbons
UCR	University of California at Riverside
FE	Fuel economy
GDE	gallons diesel equivalent
NG	natural gas
LNG	liquid natural gas

Executive Summary

Heavy duty on-road vehicles represent one of the largest sources of NO_x emissions and fuel consumption in North America. Heavy duty vehicles are predominantly diesels, with the recent penetration of natural gas (NG) engines in refuse collection, transit, and local delivery where vehicles are centrally garaged and fueled. As emissions and greenhouse gas regulations continue to tighten, new opportunities to use advanced fleet specific heavy duty vehicles with improved fuel economy are becoming available. NO_x emissions have dropped 90% for heavy duty vehicles with the recent 2010 certification limit. Additional NO_x reductions of another 90% are desired for the South Coast Air basin to meet its 2023 NO_x inventory requirements.

Although the 2010 certification standards were designed to reduce NO_x emissions, the in-use NO_x emissions are actually much higher than certification standards. The main reason is a result of the poor performance of aftertreatment systems for diesel vehicles during low duty cycle operation. Recent studies by UCR suggest 99% of the operation within 10 miles of the ports are up to 1 g/bhp-hr NO_x. Stoichiometric natural gas engines with three-way catalysts tend to have better low duty cycle NO_x emissions than diesel engines with SCR aftertreatment systems. Thus, a real NO_x success will not only be providing a solution that is independent of duty cycle, but one that also reduces the emissions an additional 90% from the current 2010 standard.

Goals: The goals of project are to evaluate the ISL G NZ (near zero) 8.9 liter ultra-low NO_x NG vehicle emissions, global warming potential, and fuel economy during in-use conditions. This report presents a summary of the results and conclusions of ultra-low NO_x NG vehicle evaluation.

Approach: The testing was performed on UC Riverside's chassis dynamometer integrated with its mobile emissions laboratory (MEL) located in Riverside CA just east of the South Coast Air Quality Management District (AQMD). The cycles selected for this study are representative of operation in the South Coast Air Basin and included the urban dynamometer driving schedule, the near dock, local, and regional port cycles, the AQMD refuse cycle, and the central business district cycle.

One of the difficulties in quantifying NO_x emissions at 90% of the 2010 certification level (~ 0.02 g/bhp-hr), is the measurement method is approaching its detection limit. Three upgraded NO_x measurement methods were considered which include a raw NO_x measurement integrated with real time exhaust flow, a real-time ambient correction approach, and a trace level ambient analyzer for accurate bag analysis. In summary the improved methods varied in their success where the raw sampling approach showed to be the most accurate and precise over the range of conditions tested.

In addition to the regulated emissions, the laboratory was equipped to measure particle size distribution, particle number, soot PM mass, ammonia, and nitrous oxide emissions to investigate any dis-benefit resulting from the ISL G NZ engine and aftertreatment system.

Results: The ISL G NZ 8.9 liter NG engine showed NO_x emissions below the proposed 0.02 g/bhp-hr emission target and averaged between 0.014 and 0.002 g/bhp-hr for the various hot start tests, see Figure ES-1. The NO_x emissions (g/bhp-hr) decreased as the duty cycle was decreased

which was the opposite trend for the diesel vehicles (where emissions increased as duty cycle decreased). The large error bars (represented by 1 standard deviation) may suggest measurement variability, but when the real-time data was investigated, one can see the variability was a result of test-to-test differences from a few isolated NO_x events during rapid throttle tip-in at idle, see Figure ES-2. This suggests possible driver behavior may impact the overall NO_x in-use performance of the vehicle where more gradual accelerations are desired. This is also evident with the more gradual accelerations of the near dock and local port cycles which showed smaller error bars and lower average emission factors, see Figure ES-1.

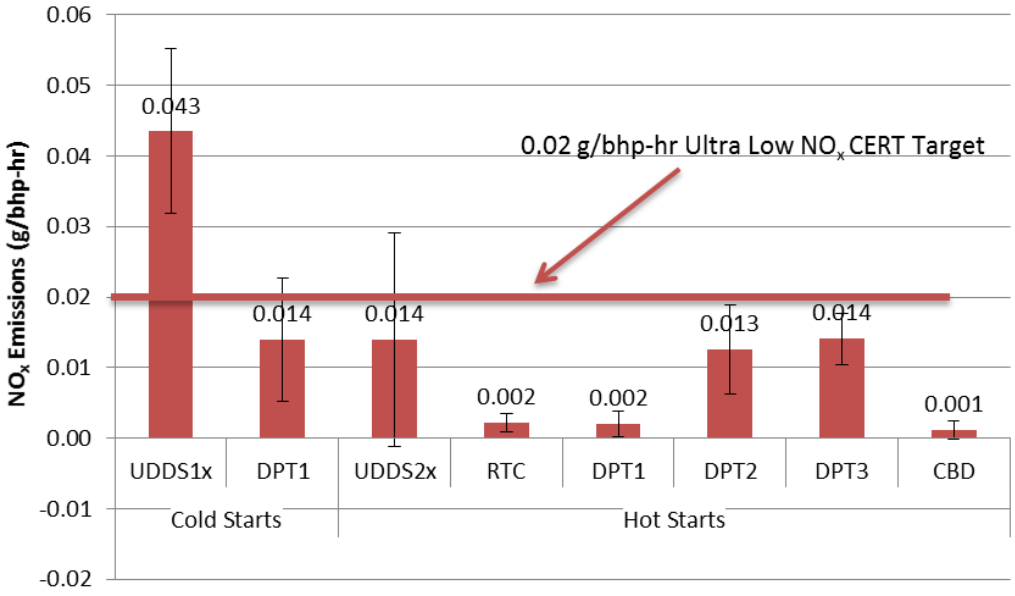


Figure ES-1 Cycle averaged NO_x emissions for the ISL G NZ 8.9 liter equipped vehicle

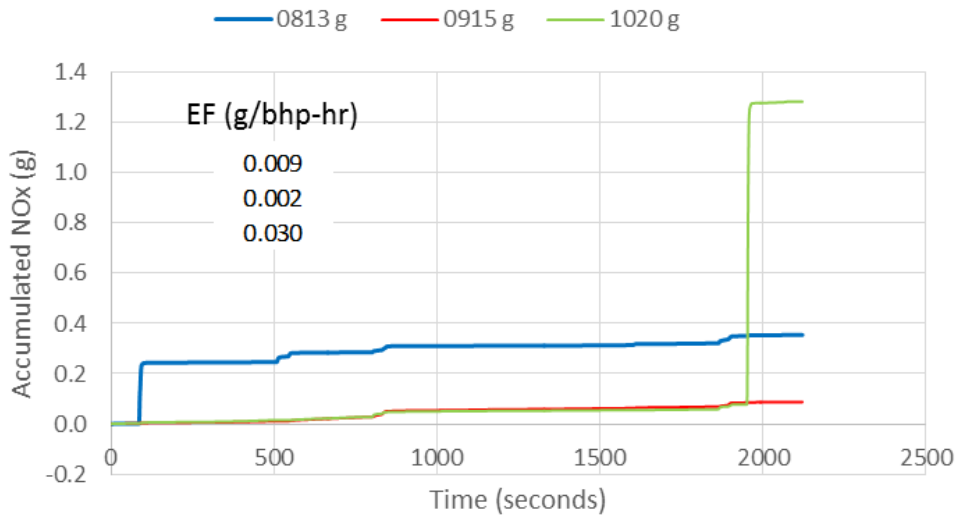


Figure ES-2 Real-time NO_x accumulated mass for the three UDDS hot cycles

[1] Individual accumulated and integrated EF for the UDDS cycle is shown in the figure above. The average of these tests is represented in Figure ES-1, UDDS cycle (0.14 g/bhp-hr).

Cold start emissions represented a significant part of the total NO_x emissions where 90% of the NO_x emissions occurred in the first 200 seconds of the cold UDDS test. Once the catalyst was warmed up, the remaining portions of the cold UDDS test showed low NO_x emissions similar to the hot UDDS test. The hot/cold UDDS weighted emission was 0.0181 g/bhp-hr (weighted as $1/7^{th}$ of the hot cycle) which is below the 0.02 g/bhp-hr standard. Once the TWC catalyst lights off, its NO_x reduction potential remains at a high performance unlike diesel SCR equipped engines where low duty cycles (associated with SCR temperatures below 250C) will cause the SCR performance to decline.

The other emission such as carbon monoxide, particulate matter, particle number, particle size distribution, nitrous oxide, and ammonia were similar to previous versions of the same stoichiometric 8.9 liter engine certified to 0.2 g/bhp-hr NO_x. For example PM was typically below 0.001 g/bhp-h (90% below the standard), ammonia was typically above 200 ppm. This suggests the reduced NO_x emissions did not come at the expense of an increase in other species. The methane emissions were notably lower than the 0.2 g/bhp-hr NOx version of the same engine. The lower methane emissions may be a result of the closed crankcase ventilation system. The fuel economy also appeared to be similar to previous versions of the same engine displacement where the UDDS showed the lowest CO_2 emissions and were below the current FTP standard of 555 g/bhp-hr for both the cold start and hot start tests during in-use chassis testing.

Summary: In general the ISL G NZ 8.9 liter engine hot/cold emissions were within the 0.02 g/bhp-hr certification standard for all the cycles tested. Ironically these emissions factors were maintained for the full range of hot-start duty cycles found in the South Coast Air Basin unlike other heavy duty diesel fueled technologies and certification standards. The other gaseous and PM emissions were similar to previous levels. It is expected NG vehicles with the ISL G NZ could play a role in the reduction of the south coast NO_x inventory in future years given the near zero emission factors demonstrated on each test cycle. Additional research is needed to see if the on-road behavior is similar to test cycles and if there are any deviations as the vehicles age.

1 Background

1.1 Introduction

Heavy duty on-road vehicles represent one of the largest sources of NO_x emissions and fuel consumption in North America. Heavy duty vehicles are predominantly diesels, although there is increasing interest in natural gas (NG) systems. As emissions and greenhouse gas regulations continue to tighten new opportunities for advanced fleet specific heavy duty vehicles are becoming available with improved fuel economy. At the same time NO_x emissions have dropped 90% for heavy duty vehicles with the recent 2010 certification limit. Additional NO_x reductions of another 90% are desired for the South Coast Air basin to meet its 2023 NO_x inventory requirements. Thus, an approach to reduce emissions also needs lower fuel consumption to the extent possible.

1.2 NO_x Emissions

Although the 2010 certification standards were designed to reduce NO_x emissions, the in-use NO_x emissions are actually much higher than certification standards for certain fleets. The magnitude is largely dependent on the duty cycle. Since engines are certified at moderate to high engine loads, low load duty cycle can show different emission rates. For diesel engines low load duty cycles have a significant impact in the NO_x emissions. The NO_x cold start emissions for the first 100 seconds were over 2.2 g/hp-h where for the same time frame with the hot cycle it was 0.006 g/hp-h[1], see Figure 1-1. The cold start emissions were ten times higher than the certification standard and much higher than the corresponding hot start emissions. Additionally the stabilized emission of the two systems over the same time period was very similar at 0.05 g/hp-h (about 75% below the standard). The main cause for the high NO_x emissions is low selective catalytic reduction (SCR) inlet temperatures resulting from low power operation.

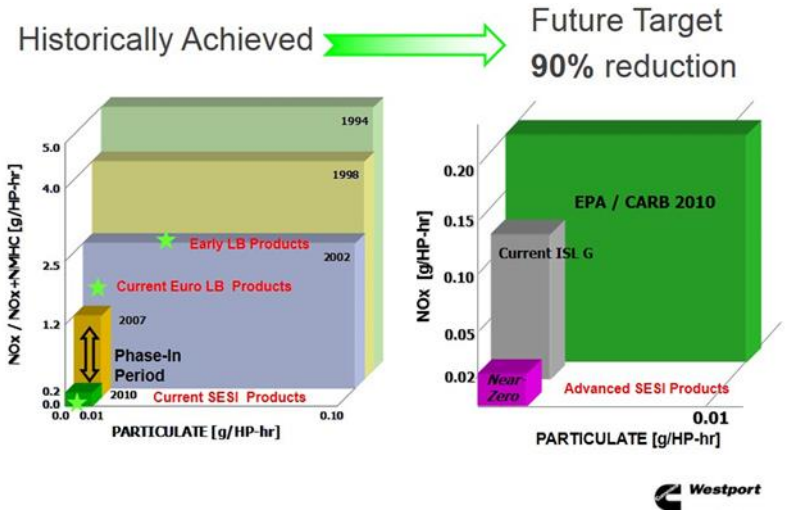


Figure 1-1 Engine dynamometer NO_x and PM certification emissions standards (source CWI)

[1] Wayne Miller, Kent C. Johnson, Thomas Durbin, and Ms. Poornima Dixit 2013, In-Use Emissions Testing and Demonstration of Retrofit Technology, Final Report Contract #11612 to SCAQMD September 2013.

These same trucks were tested on cycles designed to simulate port activity[2]. The port driving schedule represents near dock (2-6 miles), local (6-20 miles), and regional (20+ miles) drayage port operation. The SCR was inactive for 100% of the near dock cycle, 95% of the local cycle, and 60% of the regional cycle, see Figure 1-2. The NO_x emissions were on the order of 0.3 to 2 g/hp-h (1 to 9 g/mi) as much as 10 times higher than the 2010 standards. It has been show that the SCR system also becomes inactive even after hours of operation due to low loads and lean compression ignition combustion. Thus, the current diesel 2010 solution for low duty cycle activity (like at ports) is very poor where a NG solution can make significant improvements for NO_x emissions, and a reduction in carbon emissions (carbon dioxide), but at a slight penalty in equivalent gallon diesel fuel economy.

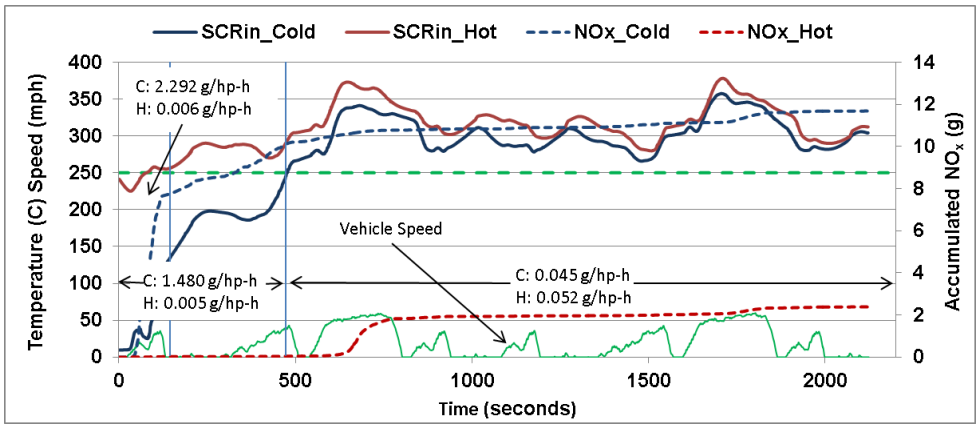
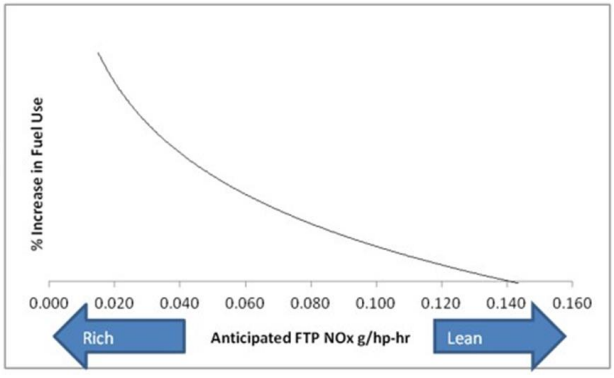


Figure 1-2 In-use emissions from a heavy duty truck tested on UCR's chassis dyno

1.3 Fuel economy

Fuel consumption and emissions are a tradeoff due to the science of combustion. Figure 1-3 shows the NO_x emissions change with changes in fuel consumption for a typical spark ignited engine. As NO_x is reduced from 0.14 to 0.02 g/hp-h fuel consumption increases a known amount. This is a result of the stoichiometric combustion of fuels. Advanced catalysts can be used to reduce NO_x from its baseline levels, but trying to reduce NO_x within a fixed SI combustion system will come at a penalty of increased fuel consumption.



(Source CWI)

Figure 1-3 NOx emissions versus fuel consumption tradeoffs during certification testing

[2] Patrick Couch, John Leonard, TIAX Development of a Drayage Truck Chassis Dynamometer Test Cycle, Port of Long Beach/ Contract HD-7188, 2011

1.4 Objectives

The goals of project are to evaluate the ISL G NZ 8.9 liter ultra-low NO_x NG vehicle emissions, global warming potential, and fuel economy during in-use conditions. Given the low NO_x concentrations expected, additional measures were implemented to quantify NO_x emissions at and below 0.02 g/bhp-hr emissions levels. This report is a summary of the approach, results, and conclusions of ultra-low NO_x NG vehicle evaluation.

2 Approach

The approach for this demonstration vehicle evaluation includes in-use testing on a chassis dynamometer, emissions measurements with UCRs mobile emission laboratory (MEL), improvements to the NO_x measurement method and a representative selection of in-use test cycles. One of the difficulties in quantifying NO_x emissions at the levels proposed in this project (90% lower than the 2010 certification level ~ 0.02 g/bhp-hr) is the measurement methods are approaching their detection limit to accurately quantify NO_x emissions. This section describes the test article, laboratories and the upgrades performed to quantify NO_x emissions at and below 90% of the 2010 emission standard.

2.1 Test article

2.1.1 Engine

The test article is the ISL G NZ 320 Cummins Westport Inc. (CWI) Natural Gas engine (SN = 73779339), see Table 2-1 for specifics and Appendix F for additional details. The engine was initially certified as a 0.2 g/bhp-hr NO_x and 0.01 g/bhp-hr PM based on the family number ECEXH0540LBH found on the engine label and the executive order (EO) published on the ARB website, see Figure F-1 Appendix F. CWI developed this engine as a ultra-low NO_x demonstration engine where the NO_x emissions have been further reduced to 0.02 g/bhp-hr (90% below the 2010 NO_x emissions standard). A second, recently released EO for the near zero configuration with engine family GCEXH0540BH, also on the CARB website and provided from CWI shows the lower NO_x standard is 0.02 g/bhp-hr and the actual certified value was 0.01 g/bhp-hr, see Figure F4 Appendix F. This evaluation is to quantify the in-use NO_x emissions in relationship to the 0.02 g/bhp-hr demonstration level.

Table 2-1 Summary of selected main engine specifications

Mfg	Model	Year	Eng. Family	Rated Power (hp @ rpm)	Disp. (liters)	Adv NO_x Std g/bhp-h [1]	PM Std. g/bhp-h
CWI	ISL G NZ	2014	ECEXH0540LBH	320 @ 2100	8.9	0.02	0.01

[1] The family ECEXH0540LBH is on the engine label given its year of manufacture. The engine tested was produced under the ECEX… label but was later certified and upgraded to the GCEX… label. The engine tested is thus, based on the GCEX label and represents a 0.02 g/bhp-hr NO_x standard, see Appendix F Figure 4 for details.

2.1.2 Test Fuel

California pipeline fuel was used for this study which represents typical Natural Gas available in Southern California. The fuel properties were measured during the emissions testing and are presented in Table 2-2. Fuel samples were collected from the vehicle prior to testing. Three vehicle refuelings (Agua Mansa Station, Riverside CA) were required to complete the work and three fuel samples were collected. Due to sample container issues, only the November 20th sample collected was analyzed as presented in Table 2-2. It is expected the pump NG fuel was consistent over the five days of testing.

Table 2-2 Fuel properties for the local NG test fuels utilized

Property	Molar %	Property	Molar %
Methane	94.65	Pentane	0.01
Ethane	3.87	Carbon dioxide	0.35
Propane	0.41	Oxygen	0.00
Butane	0.08	Nitrogen	0.63

1 Based on these fuel properties the HHV is 1-42.5 BTU/ft3 and the LHV is 939.9 BTU/ft3 with a H/C ratio of 3.905, a MON of 132.39 and a carbon weight fraction of 0.745 and a SG = 0.58, see Appendix E for laboratory results. Note these results meets the US EPA 40 CFR Part 1065.715fuel specification for NG fueled vehicles.

2.1.3 Vehicle inspection

Prior to testing, the vehicle was inspected for proper tire inflation and condition, vehicle condition, vehicle securing, and the absence of any engine code emission faults. The vehicle inspection and securing met UCR's specifications. Cummins Westport Inc. had a service person on site to make sure fault codes were absent prior to and during emissions testing. All tests were performed with-in specification and without any engine code faults. Thus, the results presented in this report are representative of a properly operating vehicle, engine, and aftertreatment system.

2.1.4 Test cycles

The test vehicle utilized an 8.9 liter NG engine which is available for three typical vocations in the South Coast Air Basin, 1) goods movement, 2) bus, and 3) refuse[3]. The engine was provided to UCR in its refuse hauler application which is one of the more common uses for the 8.9 liter engine, see Figure 2-4. In order to characterize emissions from this engine over the rage of in-use applications, goods movement and bus cycles were also tested. UCR tested the vehicle following the three port cycles (Near Dock, Local, and Regional), the Urban Dynamometer Driving Schedule (UDDS), the Central Business District (CBD) bus cycle, and the AQMD Refuse cycle, see Appendix B for details. These cycles are representative of Sothern California driving. Some cycles are short (less than 15 minutes) where double or triple cycles (2x or 3x) cycles are recommended in order capture enough PM mass to quantify emissions near 1 mg/bhp-hr. The UDDS was performed twice (UDDSx2) and the CBD was performed three times (CBDx3) where the emissions represent the average of the cycle.

Table 4 Summary of statistics for the various proposed driving cycles

Day	Distance (mi)	Average Speed (mph)	Duration (sec)
Near Dock	5.61	6.6	3046
Local	8.71	9.3	3362
Regional	27.3	23.2	3661
UDDSx2	11.1	18.8	2122
CBDx3	3.22	20.2	560
AQMD Refuse	4.30	7.31	2997

[1] Hot UDDS was performed as a double cycle (2x) and a single (1x) for the cold tests. The CBD was performed as a triple (3x) test. The refuse cycle includes a compaction element where no distance is accumulated, but emissions are counted with a simulated compaction cycle, see Appendix B for details.

[3] Cummins Westport, California Energy Commission Merit Review- ISL G Near Zero, December 2, 2015

2.1.5 Work calculation

The reported emission factors presented are based on a g/bhp-hr and g/mi basis (g/mi are provided in Appendix E). The engine work is calculated utilizing actual torque, friction torque, and reference torque from broadcast J1939 ECM signals. The following two formulas show the calculation used to determine engine brake horse power (bhp) and work (bhp-hr) for the tested vehicle. Distance is measured by the chassis dynamometer and the vehicle broadcast J1939 vehicle speed signal. A representative ISL G NZ 320 engine lug curve is provided in Figure 2-1.

$$Hp_{\_i} = \frac{RPM_{\_i}\left(Torque_{actual\_i} - Torque_{friction\_i}\right)}{5252} * Torque_{reference}$$

Where:

Hp_i	instantaneous power from the engine. Negative values set to zero
RPM_i	instantaneous engine speed as reported by the ECM (J1939)
Torque_actual_i	instantaneous engine actual torque (%): ECM (J1939)
Torque_friction_i	instantaneous engine friction torque (%): ECM (J1939)
Torque_reference	reference torque (ft-lb) as reported by the ECM (J1939)

$$Work = \sum_{i=0}^{n} \frac{Hp_{\_i}}{3600}$$

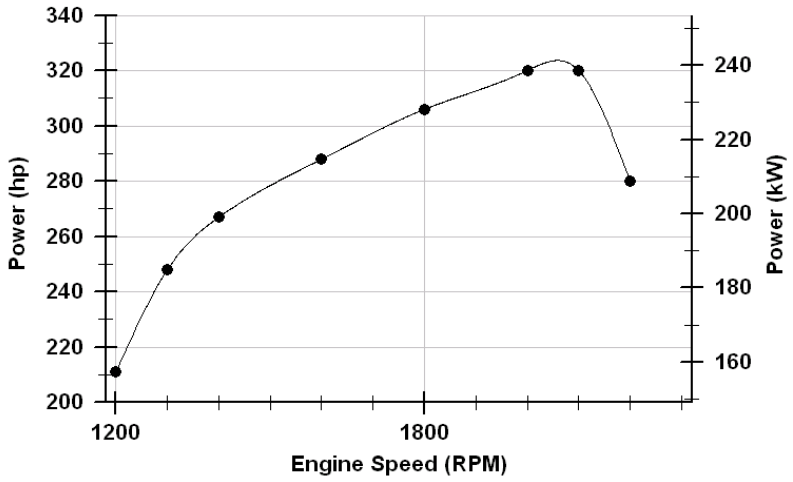


Figure 2-1 Published ISLG 8.9 Natural Gas engine power curve

Figure 2-2 and Figure 2-3 show the measured power and work for each of the tests performed on the refuse vehicle. The engine is certified on the FTP type of cycle where the average power is around 82 Hp and estimated at 24.7 bhp-h, also shown in Figure 2-2 and Figure 2-3. The UDDS, regional (DPT3) and the CBD test cycles represent power near (but lower) than the FTP certification cycle. The near dock (DPT1), local (DPT2), and refuse (RTC) cycles showed much lower power with the DPT1 being the lowest (as shown by previous studies). Previous testing of the low power from the DPT1 cycle resulted in high diesel NO_x emissions because the SCR operating temperatures were never obtained.

The measured work for the all the cycles (except the CBD (lower), RTC, and the regional (DPT3 much higher)) were close to the certification FTP estimated work (Note the hot-UDDS was higher because a double cycle was performed where the cold-UDDS was performed as a single UDDS test). In general the cycles selected are representative of in-use conditions and certification testing. It is expected the results from this study will be very representative for real world emission factors for the test article.

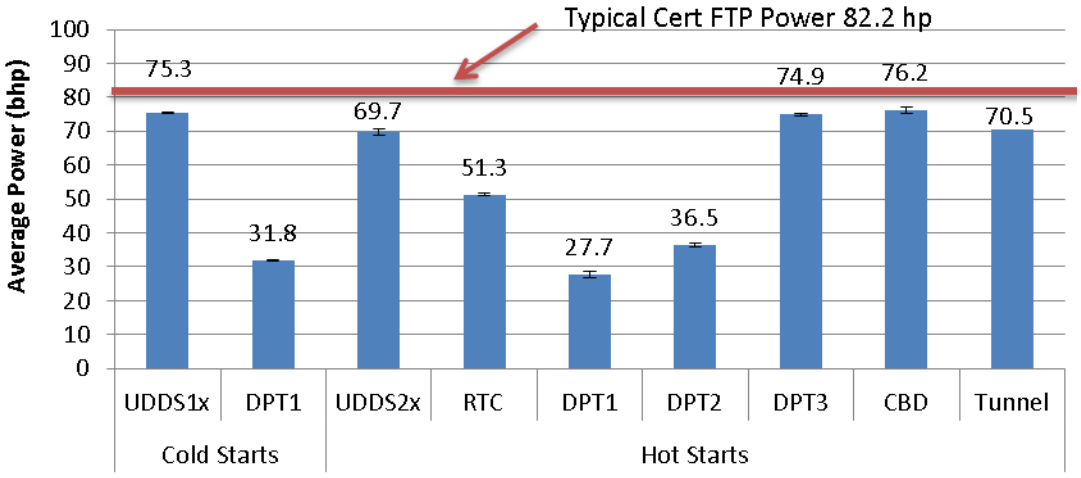


Figure 2-2 Power from the various tests with 1 stdev error bars

[1] The tunnel blank (TB) was performed without the vehicle operating. To calculate a work specific TB comparison, the TB test utilized the power and work value of a single hot-UDDS test to provide context of the measurement detection limits.

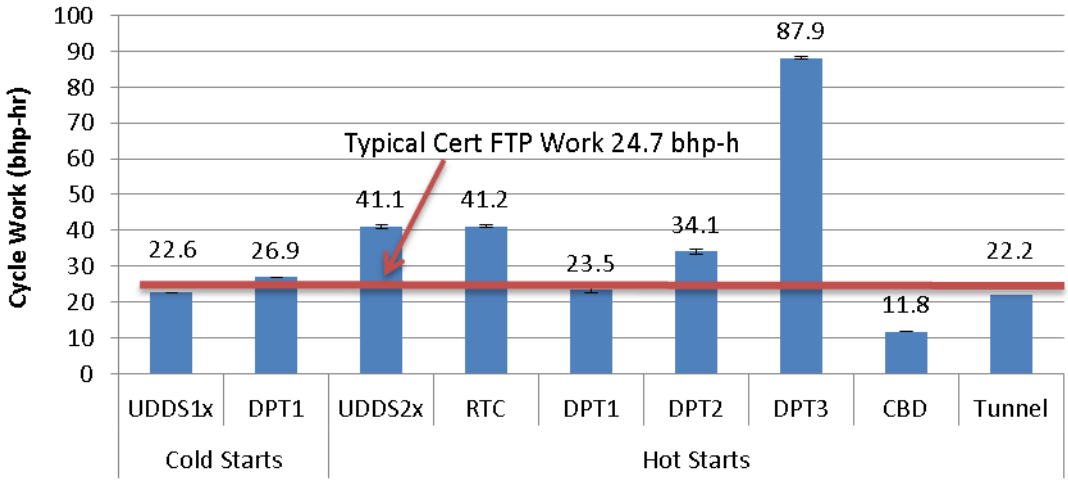


Figure 2-3 Work from the various tests with 1 stdev error bars

[1] The TB was performed without the vehicle operating. To calculate a work specific TB comparison, the TB test utilized the power and work value of a single hot-UDDS test to provide context of the measurement detection limits.

2.2 Laboratories

The testing was performed on UC Riverside's chassis dynamometer integrated with its mobile emissions laboratory (MEL) located in Riverside CA just east of the South Coast Air Quality

Management District (AQMD). This section describes the chassis dynamometer and emissions measurement laboratories used for evaluating the in-use emissions from the demonstration vehicle. Due to challenges of NO_x measurement at 0.02 g/bhp-hr, additional sections are provided to introduce the measurement improvements.

2.2.1 Chassis dynamometer

UCR's chassis dynamometer is an electric AC type design that can simulate inertia loads from 10,000 lb to 80,000 lb which covers a broad range of in-use medium and heavy duty vehicles, see Figure 2-4. The design incorporates 48" rolls, vehicle tie down to prevent tire slippage, 45,000 lb base inertial plus two large AC drive motors for achieving a range of inertias. The dyno has the capability to absorb accelerations and decelerations up to 6 mph/sec and handle wheel loads up to 600 horse power at 70 mph. This facility was also specially geared to handle slow speed vehicles such as yard trucks where 200 hp at 15 mph is common. See Appendix D for more details.

2.2.1.1 Test weight

The ISL G NZ 320 engine is installed in a refuse hauler chassis with a GVW of 62,000 lb, VIN 3BPZX20X6FF100173. The representative test weight for refuse haulers operating in the south coast air basin is 56,000 lb[4]. The testing weight of 56,000 lb was also utilized during previous testing of refuse haulers with diesel and NG engines by UC Riverside and WVU [4 and 5]. For this testing program UCR utilized a testing weight of 56,000 lb for all test cycles (refuse, CBD, UDDS, and port cycles).

Figure 2-4 UCR's heavy duty chassis eddy current transient dynamometer

[4] Wayne Miller, Kent C. Johnson, Thomas Durbin, and Ms. Poornima Dixit 2014, In-Use Emissions Testing and Demonstration of Retrofit Technology, Final Report Contract #11612 to SCAQMD September 2014.

[5] Daniel K Carder, Mridul Gautam, Arvind Thiruvengada,m Marc C. Besch (2013) In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines, Final Report Contract #11611 to SCAQMD July 2014.

2.2.1.2 Coast down

UCR utilizes a calculation approach for the coast down settings of the chassis dynamometer. This approach is also used by other testing facilities and has been shown to be representative of in-use operation, see Appendix G for a more detailed discussion. The test weight of 56,000 lb resulted in a power of 117.42 Hp at 50 mph with the calculated dynamometer loading coefficients of A = 397.73642, B = -2.43E-14 and C = 0.193166. See calculation methods in Appendix G for more details.

2.2.2 Emissions measurements

The proposed NO_x measurement (at 0.02 g/bhp-hr) are approaching the detection limits for the traditional dilute CVS measurement method. This section discussed the traditional and upgraded methods recommended for the ultra-low NO_x evaluation. This section also provides a section on the calculations utilized, additional measurements needed (ie. Trace analyzers and exhaust flow) and an evaluation of the upgraded methods in comparison to the tradition methods.

2.2.2.1 Traditional method

The approach used for measuring the emissions from a vehicle or an engine on a dynamometer is to connect UCR's heavy-duty mobile emission lab (MEL) to the total exhaust of the diesel engine, see Appendix C for more details. The details for sampling and measurement methods of mass emission rates from heavy-duty diesel engines are specified in Section 40, Code of Federal Regulations (CFR): Protection of the Environment, Part 1065. UCR's unique heavy-duty diesel MEL is designed and operated to meet those stringent specifications. MEL is a complex laboratory and a schematic of the major operating subsystems for MEL are shown in Figure 2-4. The accuracy of MEL's measurements has been checked/verified against ARB's[6] and Southwest Research Institute's [7, 8] heavy-duty diesel laboratories. MEL routinely measures Total Hydrocarbons (THC), Methane (CH_4), Carbon Monoxide (CO), Carbon Dioxide (CO_2), Nitrogen Oxides (NO_x), and Particulate Matter (PM) emissions from diesel engines. Design capabilities and details of MEL are described in Cocker et al[4,9]. Samples can be collected for more detailed analyses such as hydrocarbon speciation, carbonyl emissions, polynuclear aromatic hydrocarbons, etc.

The traditional NO_x measurements include a 600 heated chemiluminescent detector (HCLD) from California Analytical Inc. (CAI) configured to sample from the CVS tunnel during real time and ambient and dilute bag measurements following automated routines of the MEL laboratory. The samples are collected from the CVS dilute tunnel through an acid treated filter to

---

[6] Cocker III, D. R., Shah, S. D., Johnson, K. C., Zhu, X., Miller, J. W., Norbeck, J. M., Development and Application of a Mobile Laboratory for Measuring Emissions from Diesel Engines. 2. Sampling for Toxics and Particulate Matter, Environ. Sci. Technol. **2004**, 38, 6809-6816

[7] Cocker III, D. R, Shah, S. D., Johnson, K. C., Miller, J. W., Norbeck, J. M., Measurement Allowance Project – On-Road Validation. Final Report to the Measurement Allowance steering Committee.

[8] Johnson, K.C., Durbin, T.D., Cocker, III, D.R., Miller, W.J., Bishnu, D.K., Maldonado, H., Moynahan, N., Ensfield, C., Laroo, C.A. (2009) On-road comparison of a portable emission measurement system with a mobile reference laboratory for a heavy-duty diesel vehicle, Atmospheric Environment 43 (2009) 2877–2883

[9] Cocker III, D. R, Shah, S. D., Johnson, K. C., Miller, J. W., Norbeck, J. M., *Development and Application of a Mobile Laboratory for Measuring Emissions From Diesel Engines I. Regulated Gaseous Emissions*, Environmental Science and Technology. **2004**, 38, 2182-2189

prevent measurement interferences from ammonia (NH_3) concentrations. The acid treated filters were replaced daily.

In addition to the regulated emissions, the laboratory was equipped to measure particle size distribution (PSD) with TSI's Engine Exhaust Particle Sizer (EEPS) model 3090, particle number (PN) with a TSI 3776 condensation particle counter (CPC), soot PM mass with AVL's Micro Soot Sensors (MSS 483), NH_3 emissions with an integrated real-time tunable diode laser (TDL) from Unisearch Associates Inc. LasIR S Series, and a batched low level nitrous oxide (N_2O) emissions with a Fourier Transform Infrared Spectrometer (FTIR). The PN measurement system used a low cut point CPC (2.5 nm D50) because of the large PN concentrations reported below the PMP protocol CPC 23 nm measurement system (10, 11, and 12). The EEPS spectrometer displays measurements in 32 channels total (16 channels per decade) and operates over a wide particle concentration range, including down to 200 particles/cm3.

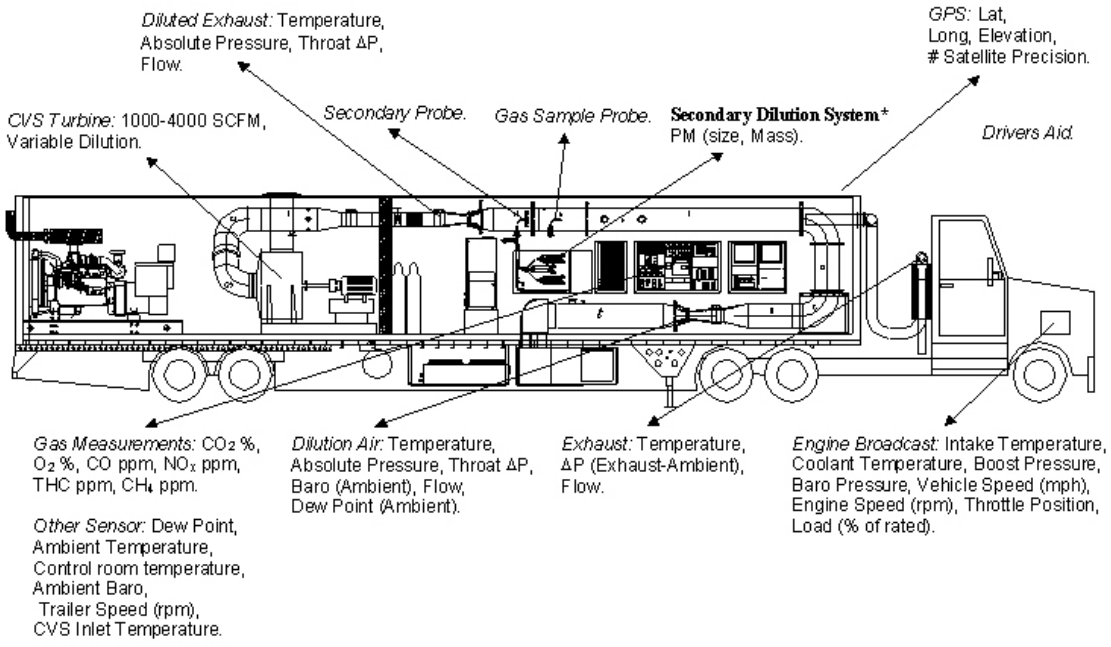


Figure 2-5 Major Systems within UCR's Mobile Emission Lab (MEL)

2.2.2.2 NO_x Method upgrades

Three NO_x upgrade methods were considered for this project. These included 1) real-time raw sampling and exhaust flow measurements, 2) real-time ambient second by second corrections, and 3) advanced trace type analyzer bag measurements. The new measurement methods required instrumentation upgrades which are discussed below.

Raw NOx measurements

The raw NO_x measurements utilized a 300 HCLD CAI analyzer which sampled raw exhaust through a low volume heated filter and heated sample line. The low volume design was considered to improve the response time of the analyzer with the exhaust flow measurement. The heated filter was acid treated to minimize NH_3 interference with the NO_x measurement. A real-time high speed exhaust flow meter (100 Hz model EFM-HS Sensors Inc) was used to align NO_x

concentration with real time exhaust flow measurements. The EFM-HS was correlated with UCR dual CVS system prior to testing to improve the accuracy between the raw and dilute CVS methods and eliminate exhaust flow biases from propagating through the comparison.

Trace level NOx analyzer

A trace level chemiluminescence NO-NO_2-NO_x analyzer model 42C manufactured by Thermo Environmental Instruments Inc (TECO) was used for the real-time ambient measurements and the low level bag analysis. This analyzer has been operating with-in CE-CERT's atmospheric research laboratories for ambient NO_x quantification for several years. This analyzer was calibrated and integrated specially for this ultra-low NO_x project. The span on the instrument was set to 600 ppb and showed a signal to noise ratio about an order in magnitude lower than the traditional (600 HCLD) analyzer. The signal averaging was reduced from 30 seconds to 1 second and showed a $T_{10\text{-}90}$ and a $T_{90\text{-}10}$ just over 10 seconds (slightly higher than the specifications of 40 CFR Part 1065). The slightly slower time constant should not impact the gradual transients expected during real-time ambient measurements or bag concentrations. Although this trace analyzer does not meet the requirements of 1065, it does provide a good assessment of NO_x emissions below 1 ppm with an ambient trace type NO_x analyzer.

2.2.2.3 Calculation upgrades

The calculations for the traditional and improved methods are presented in this section. The calculations are in agreement with 40 CFR Part 1065, but are presented in a condensed version to draw observation differences without the details of working in molar flow rates as per 40 CFR Part 1065.

Table 2-3 NO_x measurement methods traditional and upgraded

Type	Analyzer	Meth. ID	Description
Traditional	600 HCLD dil 600 HCLD amb	M1	Modal NO_x with ambient bag correction
Traditional	600 HCLD dil 600 HCLD amb	M2	Dilute bag NO_x with ambient bag correction
Upgrade	300 HCLD raw	M3	Raw NO_x no ambient bag correction
Upgrade	600 HCLD dil TECO amb	M4	Modal dilute NOx with ambient real time correction
Upgrade	TECO dil TECO amb	M5	Trace analyzer dilute bag with trace ambient bag correction

Traditional Methods:

The traditional NO_x measurement methods are described in the next two equations. The first equation is the real-time modal measurement corrected for the ambient bag concentration and real time dilution factor, Method 1 (M1). The second traditional equation (M2) is based on dilute bag and ambient bag concentrations and an integrated dilution factor over the cycle.

$$NO_{x\_m1} = \sum_{i=1}^{n} (Q_{cvs_i} * \Delta t_{\_i}) * \rho_{NO_x} * \left(C_{m\_i} - C_a * \left(1 - \frac{1}{DF_i}\right)\right)$$

Where:

NO_{x_m1} the Method 1 NO_x measurement method (g/cycle)
Q_{cvs_i} is the instantaneous CVS flow
ρ_{NOx} is the density of NO_x from 40 CFR Part 1065
C_{m_i} is the instantaneous NO_x concentration measured with the dilute NO_x 600 HCLD CAI analyzer
C_a is the ambient bag NO_x concentration measured by the 600 HCLD CAI analyzer
DF_i instantaneous dilution factor

$$NO_{x\_m2} = (Q_{cvs\_ave} * \Delta t) * \rho_{NO_x} * \left(C_d - C_a * \left(1 - \frac{1}{DF_{\_ave}} \right) \right)$$

Where:
NO_{x_m2} the Method 2 NO_x measurement method (g/cycle)
Q_{cvs_ave} is the average CVS flow
ρ_{NOx} is the density of NO_x from 40 CFR Part 1065
C_d is the dilute bag NO_x concentration measured with the dilute NOx 600 HCLD CAI analyzer
C_a is the ambient bag NO_x concentration measured by the 600 HCLD CAI analyzer
DF_{ave} average dilution factor

Upgraded Methods:
The upgraded NOx measurement methods are presented in the next three equations. These upgrades included new analyzers, sample lines, sample filters, and exhaust flow measurement systems. For Method 3 (M3) there is no ambient correction. For Method 4 (M4) the real time dilute NOx is corrected for ambient real time NOx on a second by second basis. For Method 5 (M5) the trace NOx analyzer is used to measure the dilute bag and ambient bags (similar to Method 2).

$$NO_{x\_m3} = \sum_{i=1}^{n} (Q_{exh_i} * \Delta t_{\_i}) * \rho_{NO_x} * \left(C_{m\_i} \right)$$

Where:
NO_{x_m3} the Method 3 NO_x measurement method (g/cycle)
Q_{exh_i} is the instantaneous exhaust flow measured in the tail pipe
ρ_{NOx} is the density of NO_x from 40 CFR Part 1065
C_{m_i} is the dilute bag NO_x concentration measured with the dilute NO_x 300 HCLD CAI analyzer

$$NO_{x\_m4} = \sum_{i=1}^{n} (Q_{cvs_i} * \Delta t_{\_i}) * \rho_{NO_x} * \left(C_{m\_i} - C_{a\_adv\_i} * \left(1 - \frac{1}{DF_i} \right) \right)$$

Where:
NO_{x_m4} the Method 4 NO_x measurement method (g/cycle)
Q_{cvs_i} is the instantaneous CVS flow
ρ_{NOx} is the density of NO_x from 40 CFR Part 1065

C_{m_i} is the dilute bag NO_x concentration measured with the dilute NO_x 600 HCLD CAI analyzer

C_{a_adv} is the trace ambient bag NOx concentration measured by the TECO trace NO_x analyzer

DF_i instantaneous dilution factor

$$NO_{x\_m5} = (Q_{cvs\_ave} * \Delta t) * \rho_{NO_x} * \left(C_{d\_adv} - C_{a\_adv} * \left(1 - \frac{1}{DF_{\_ave}} \right) \right)$$

Where:

NO_{x_m5} the Method 5 NO_x measurement method (g/cycle)

Q_{cvs_ave} is the average CVS flow

ρ_{NOx} is the density of NO_x from 40 CFR Part 1065

C_{d_adv} is the dilute bag NO_x concentration measured by the TECO trace NO_x analyzer

C_{a_adv} is the ambient bag NO_x concentration measured by the TECO trace NO_x analyzer

DF_{ave} average dilution factor

2.2.3 Method evaluation

One of the main contributing factors to the issue with the traditional CVS sampling system is the magnitude of the ambient concentration has on the calculation. Table 2-4 lists the 10th, 50th, and 90th average ambient, dilute modal, and raw tailpipe measured percentile concentrations. The 50th percentile raw, dilute, and ambient NO_x concentration were 0.55 ppm, 0.17 ppm, and 0.07 ppm respectively.

As discussed previously, the ambient concentration is subtracted from the dilute concentration prior to calculating the mass based emissions. This subtraction is typically a larger number minus a small number. At the 0.02 g/bhp-hr emission level, the ambient concentration is now at the same levels as the dilute measured value. The ambient concentration was found to be 54% of the total measured dilute concentration at the 50th percentile measured concentration, see Table 2-4. The ambient corrected NOx concentration (C_{a_cor}) utilized in the dilution measurements is the product of ambient NO_x concentration and an inverse ratio of the dilution factor, see equation below. If we divide the C_{a_cor} by the dilute NOx measured we get a ratio that is representative of the ambient percent of total NOx. Figure 2-6 shows the ratio in a histogram plot and more than half the data is above 0.6 suggest that most of the measurements meeting the 0.02 g/bhp-hr were only twice that of ambient concentrations. This ratio gives the reader a feel for the influence ambient has at and below 0.02 g/bhp-hr NOx emissions.

Table 2-4 Cycle averaged raw, dilute, and ambient measured concentrations (ppm) statistics

Percentile	Amb	Dilute [1]	Raw [1]	C_{a_cor}/Dil %
10th	0.234	0.632	6.533	105%
50th	0.070	0.168	0.554	54%
90th	0.021	0.033	0.070	10%

[1] With the cold starts removed, the dilute and raw 10th, 50th, and 90th would be 0.326, 0.146, and 0.031 ppm for the dilute concentration and 2.115, 0.450, and 0.069 ppm for the raw concentration, respectively.

The results show a 10th, 50th and 90th percentile (C_{a_cor}/C_d) ratio of 10%, 54% and 105%, respectively. This suggest more than ½ of the measurements were sampled where the dilute concentration was 50% of the ambient corrected (C_{a_cor}) concentration. The low concentrations measured by dilute methods will impact all the methods except for M3 that utilizes the raw sampling approach where no dilution correction is needed.

$$C_{a\_cor} = C_a * \left(1 - \frac{1}{DF_{\_ave}}\right)$$

Where:

C_{a_cor}	is the ambient NOx concentration factor used in M1
C_a	is the ambient bag NOx concentration
DF_{ave}	cycle average dilution factor (typically 20-30)
$\left(1 - \frac{1}{DF_{\_ave}}\right)$	dilution factor term (varied from 0.95 to 0.98 in this study)

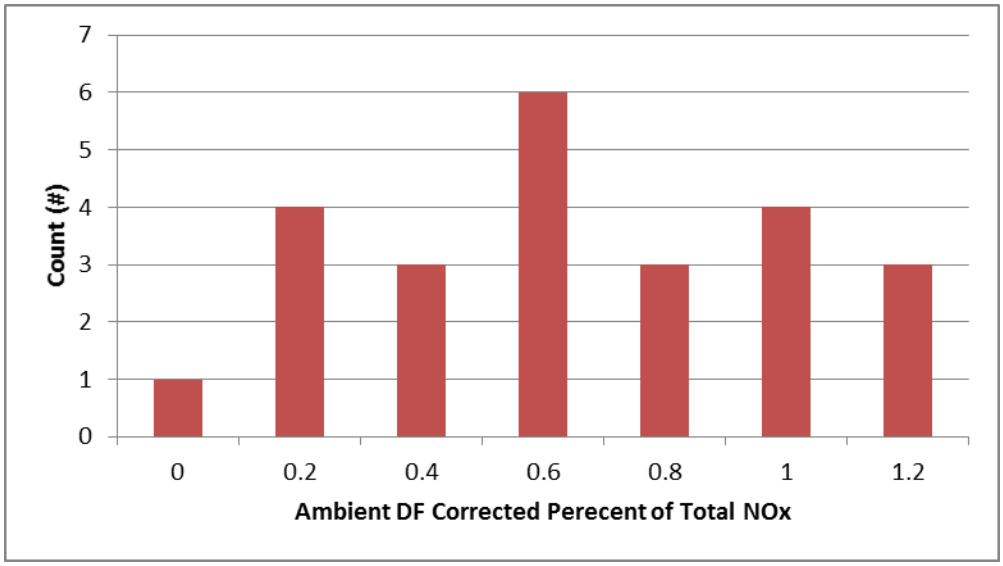


Figure 2-6 Ambient fraction of dilute NO_x concentration distribution

The real-time concentrations for each cycle is also important where observations suggest a few NO_x spikes of 20-30 times the average values were the basis of the cycle average concentrations. Section 4 provides additional discussions on the real-time transient NO_x measurements. It is important to understand that the real-time NO_x spikes will impact the M1, M3, and M4 measurements since these utilize real-time signals where M2 and M5 are integrated bag signals.

The average mean difference in average emissions between the methods is shown in Table 2-5 with M1 as the reference method. For M2 the average NO_x emissions was very similar to M1 (only 5% higher on average, but varied from higher to lower from cycle to cycle). M3 was slightly lower (-18% on average), but was consistently lower except for the CBD tests. Further investigation of the CBD tests shows one of the M1 tests had a negative emission rate due a high ambient bag concentration compared to the modal dilute concentration. This negative value was

not an outlier, but a real measurement difficulty at these emission levels. The M4 average NO_x emission rate was notably higher (and relatively more variable) and for M5 the average was significantly lower for all tests compared to the M1 traditional method.

The M4 utilized real time ambient concentrations for real time correction of the background calculation. The trace analyzer utilized show some short term drift that didn't appear to be related to ambient concentration changes. Additional investigation is needed, but is outside the scope of this effort. The researchers suggest the M4 method will have more variability as a result and could be the cause for the higher mean difference.

The M5 utilized the trace NO_x analyzer for bag measurements. Surprisingly the M5 method showed a much lower mean value. Investigations were carried out to see about analyzer drift or stability and no issues were found during the bag analysis time spans.

Table 2-5 NO_x emission average percent difference from Method 1

Trace	M2	M3	M4	M5
UDDS1x	-17%	-40%	96%	-87%
DPT1	31%	-42%	-8%	-99%
UDDS2x	7%	-13%	21%	-70%
RTC	4%	-21%	111%	-7%
DPT1	-21%	-11%	25%	-14%
DPT2	3%	-20%	25%	-61%
DPT3	12%	-22%	27%	-72%
CBD	19%	23%	32%	16%
Ave	5%	-18%	41%	-49%
Stdev	17%	20%	40%	42%

A comparison of the statistical significance between the traditional M1 and other methods is provided in Table 2-6. The two tailed paired t-test and f-test results suggest the two traditional methods do not have statistically different means or different variances at 95% confidence, see Table 2-6 (M2 p-value >> 0.05 for both). The upgraded methods showed a different result that varies. The M3 (raw exhaust flow approach) mean difference is not statistically significant at 95% confidence (M3 p-value > 0.06) but is at the 90% confidence. The M4 (RT ambient correction) and M5 (trace bag evaluation) upgraded methods both have statistically different means (p-value < 0.05 for both).

Table 2-6 Comparison to traditional Method 1 measurement (modal dilute NO_x)

Method	t-test	f-test
M2	0.521	0.998
M3	0.060	0.152
M4	0.021	0.141
M5	0.001	0.104

Each of the added methods (M3, M4, and M5) may have some possible implementation issues that need to be considered in order to evaluate the comparative results. The M3 measurement showed good alignment between the measured NOx signal and the exhaust flow signal. The majority of the NOx mass emissions resulted from a few large spikes, as discussed in Section 4. These NOx spikes were found to represent more than 80% of the total emission factor. Closer inspection shows that the NOx concentration and exhaust flow spike occurred simultaneously and were usually a result of a rapid acceleration from idle.

For the M4 approach (real-time NOx ambient correction) the analyzer had a slight zero stability issue over the 20-40 minute test cycle not found during the short 3 minute bag analysis. As such, the drift may be the result of the M4 poor method comparison.

The low M5 method may represent the best approach with very accurate bag measurements for both the ambient and dilute bag measurements with a trace type NOx analyzer with a larger sample cell. The drift issue suggested for the M4 measurement didn't appear to be a factor during the short bag analysis, but additional tests should be performed to evaluation. As such, this method may have performed the best, but additional testing is suggested to evaluate this method on future testing opportunities at 0.02 g/bhp-hr.

In summary the M1, M2, and M3 appear to be the most reliable where the M3 results are more consistent at the extremely low concentrations measured. M4 and M5 require further investigation with lower zero drift instrumentation.

3 Results

This section describes the results from the ISL G NZ 8.9 liter ultra-low NO_x NG engine. The results are organized by gaseous emissions followed by PM, particle size distribution, greenhouse gases, and fuel economy. The emission factors presented in g/bhp-hr for comparison to the certification standard. Emissions in g/mile are provided in Appendix E. Error bars are represented by single standard deviations due to the relatively large magnitude of the error bars in relationship to the low emission levels measured for several species (three repeats were performed where the 95% confidence interval multiplier for the single standard deviation is 3.182).

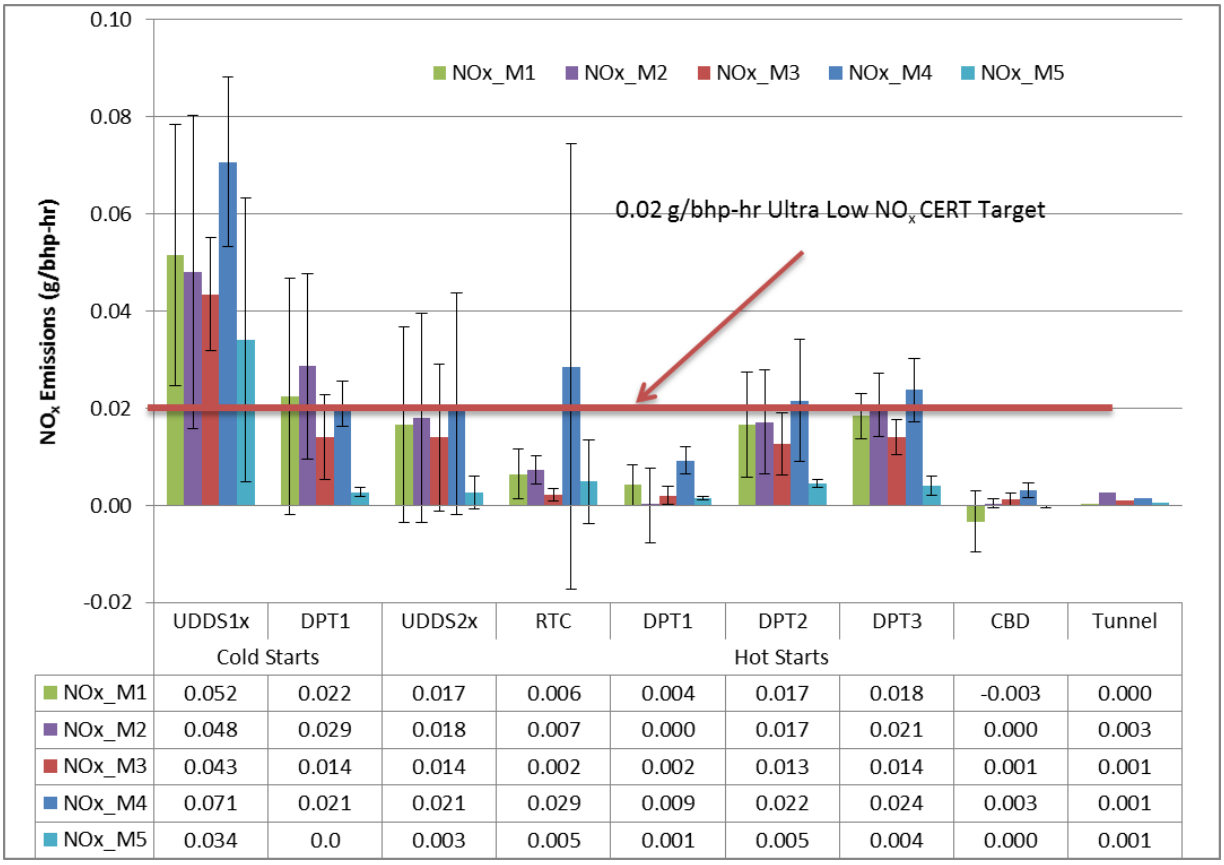
The UDDS cycle is the representative test cycle for comparisons to the engine certification FTP cycle where the other cycles (port, refuse, and bus) provide the reader a feel for the in-use comparability to low duty cycles, cruise conditions, and other vocational specifics of the real world. As such, the results will be presented in each sub-section within the context of the test cycle.

3.1 Gaseous emissions

3.1.1 NOx emissions

The NO_x emissions are presented in Figure 3-1 for each of the methods evaluated and for all the test cycles performed. The NO_x emissions were below the demonstration 0.02 g/bhp-hr emissions targets for the UDDS, DPT1 (hot and cold), and the CBD for all measurement methods. The local and regional port cycles (DPT2 and DPT3) NO_x emissions were below the improved methods but at and below the standard for the traditional methods. The cold start emissions were higher than the hot tests when comparing between like tests (UDDS cold vs hot and DPT1 cold vs hot) and averaged at 0.043 g/bhp-hr for the UDDS test cycle (M3).

In general, the NO_x emissions are below the ISL G NZ 2016 NO_x certification standard of 0.02 g/bhp-hr for all tests and below the in-use NTE standard of 0.03 g/bhp-hr. The reported certification value listed on the ARB EO is 0.01 g/bhp-hr which is slightly lower than the M3 measurements (0.014 g/bhp-hr) shown for the UDDS hot test cycle, Figure 3-1. Deeper investigation shows one of the three hot UDDS tests was statistically higher (M3 = 0.009, 0.002, 0.030 g/bhp-hr). A similar trend was also found for the other four methods where the third point was much higher than the other two points. If the third point was eliminated the average for the hot UDDS would be just under the EO certification value reported by CWI (M3 = 0.005 g/bhp-hr). The test-to-test variability shown by the large error bars in Figure 3-1 was investigated where real-time analysis suggest the variability is not from low measurement issues, but appears to be the results of the vehicle variability. Section 4 provides a discussion on real-time investigation.

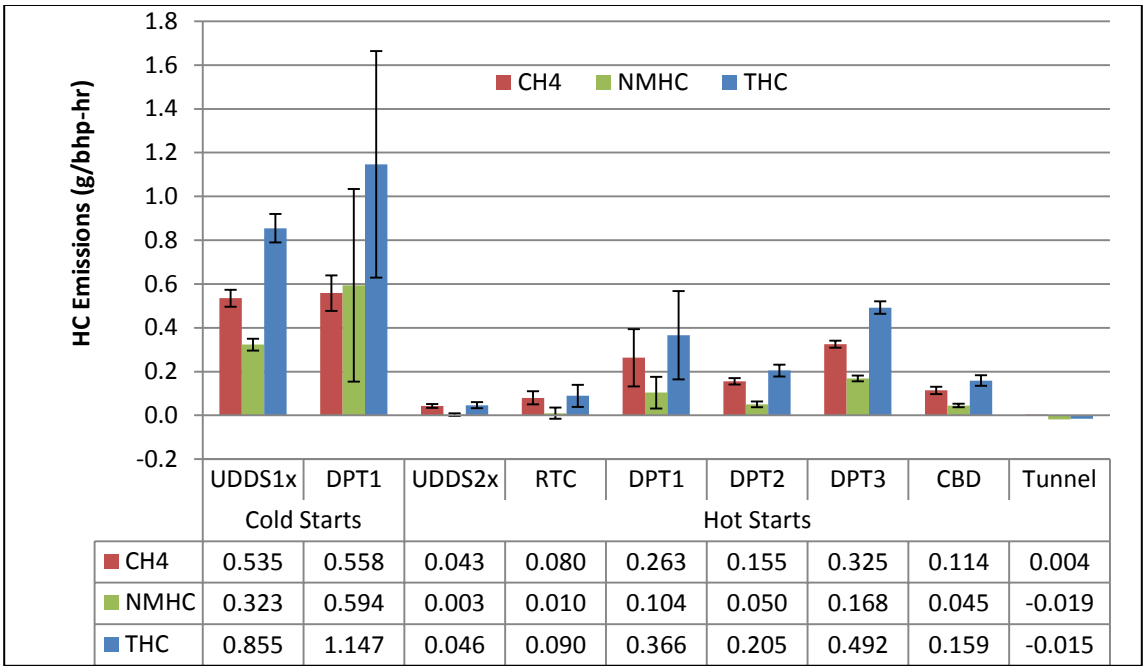


	UDDS1x	DPT1	UDDS2x	RTC	DPT1	DPT2	DPT3	CBD	Tunnel
	Cold Starts		Hot Starts						
NOx_M1	0.052	0.022	0.017	0.006	0.004	0.017	0.018	-0.003	0.000
NOx_M2	0.048	0.029	0.018	0.007	0.000	0.017	0.021	0.000	0.003
NOx_M3	0.043	0.014	0.014	0.002	0.002	0.013	0.014	0.001	0.001
NOx_M4	0.071	0.021	0.021	0.029	0.009	0.022	0.024	0.003	0.001
NOx_M5	0.034	0.0	0.003	0.005	0.001	0.005	0.004	0.000	0.001

Figure 3-1 Measured NOx emission for the various test cycles

3.1.2 Other gaseous emissions

The hydrocarbon emissions (THC, CH_4, and NMHC) are presented in Figure 3-2. The HC are highest for the cold start tests compared to the hot tests where the regional port cycle (PDT3) showed the highest HC emissions. For all the hot tests the NMHC was below the standard but just above the reported certification value except for the regional port cycle. The NMHC was typically lower than CH_4 emission as one would expect for a NG fueled vehicle. The CH_4 emissions are lower than the certification results presented in Appendix F Figure F-4 (0.04 vs the FEL level of 0.65 g/bhp-hr). Also the CH_4 emissions for the refuse hauler are significantly lower (6.4 g/mi vs 0.26 g/mi) than previously tested NG reuse haulers with the 2010 certified NG 8.9 liter engine. The lower CH_4 emissions may be a result of the closed crankcase ventilation (CCV) improvement over previous versions of this engine, see Appendix F for details.



	UDDS1x	DPT1	UDDS2x	RTC	DPT1	DPT2	DPT3	CBD	Tunnel
	Cold Starts		Hot Starts						
CH4	0.535	0.558	0.043	0.080	0.263	0.155	0.325	0.114	0.004
NMHC	0.323	0.594	0.003	0.010	0.104	0.050	0.168	0.045	-0.019
THC	0.855	1.147	0.046	0.090	0.366	0.205	0.492	0.159	-0.015

Figure 3-2 Hydrocarbon emission factors (g/bhp-hr)

Figure 3-3 shows the CO emissions on a g/bhp-hr basis and Figure 3-4 shows the un-regulated NH_3 emissions on a g/bhp-hr basis. The CO emissions ranged between 1.3 to 5.3 g/bhp-hr for the cold start near dock (PDT1) and regional (DPT3) test cycles, respectively. The distance specific emissions ranged from 4.2 to 24.3 g/mi for the regional (PDT3) and the cold start UDDS test cycles. Previous testing of the ISG vehicle show similar CO emissions ranging from 14.4 to 19.2 g/mi (CBD and UDDS test cycles and same test weights).

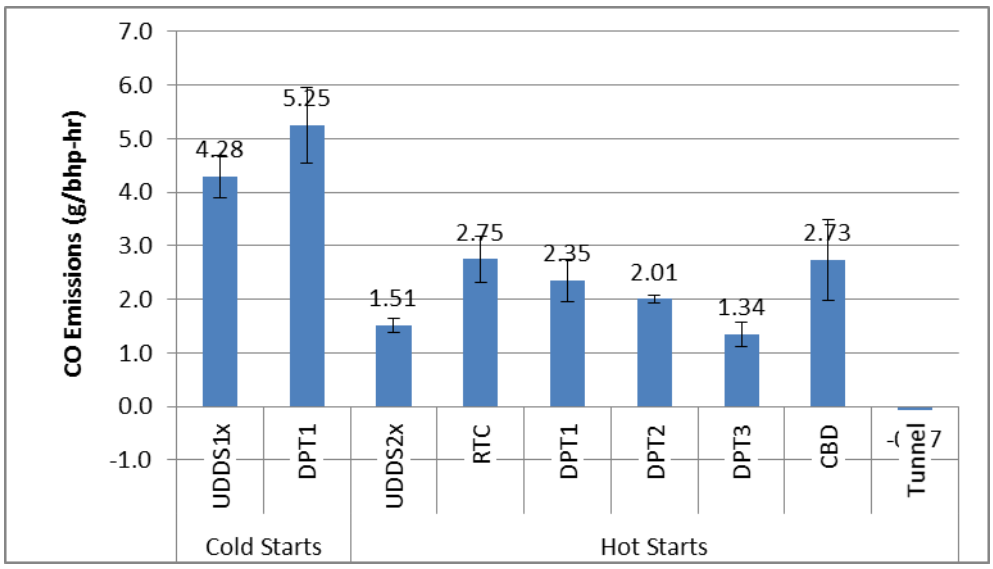


Figure 3-3 CO emission factors (g/bhp-hr)

The NH_3 emissions ranged from 0.43 to 0.94 g/bhp-hr for the hot UDDS and regional (DPT3) cycles. The distance specific emissions varied from 1.16 g/mi to 5.27 g/mi for the regional and CBD test cycles. The NH_3 emissions are slightly higher than previous ISL G vehicle where the NH_3 ranged from 1.17 to 2.8 g/mi for the UDDS and RTC cycle as compared to 1.19 and 4.09 g/mi for the ISL G NZ, respectively. The NH_3 concentration varied from 118 ppm (UDDS) to 305 ppm (CBD), see Figure 3-5.

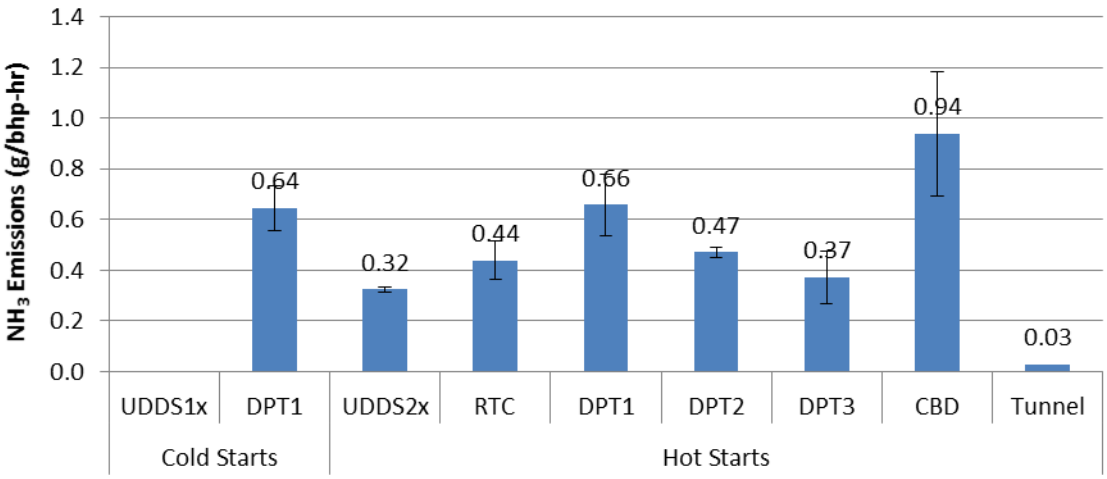


Figure 3-4 Ammonia emission factors (g/bhp-hr)

[1] NH_3 measurements for the cold UDDS test stopped working during the first hill where the system may have over ranged. The cold start UDDS NH_3 results are estimated at 20% higher than the hot-UDDS test.

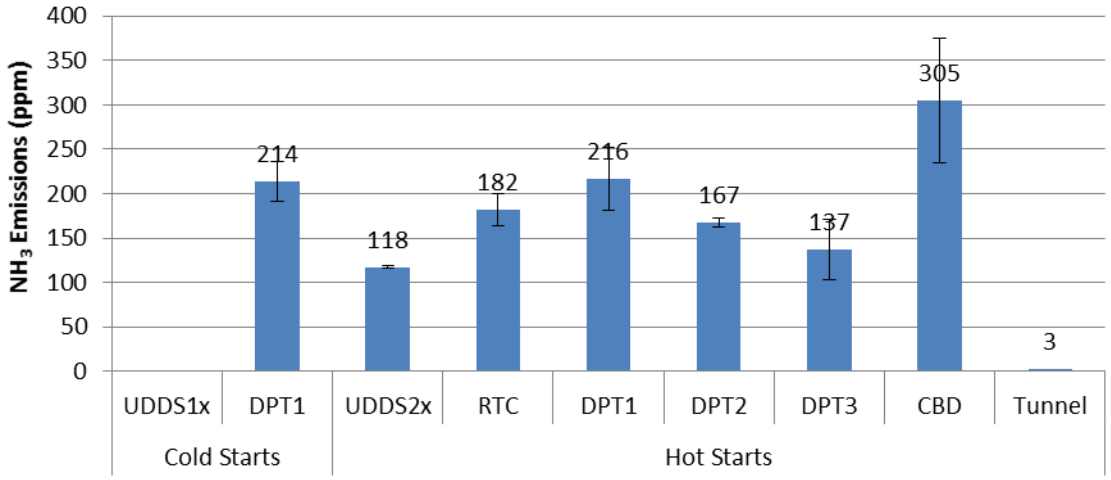


Figure 3-5 Ammonia measured tail pipe concentration (ppm)

[1] NH_3 measurements for the cold UDDS test stopped working during the first hill where the system may have over ranged. The cold start UDDS NH_3 results are estimated at 20% higher than the hot-UDDS test.

3.2 PM emissions

The PM emissions for all the tests including the cold start tests was typically 90% below the certification standard and close to UCR tunnel blank value of 0.42 g/bhp-hr (based on UDDS sample time and work), see Figure 3-6. The first regional PM filter weight was statistically higher than the other three (80, 21, 20 ug) where it is suggested something may have burned off

the exhaust system that test that may be artifact of previous vehicle operation. If the first PM results was eliminated the DPT3 EF would be reduced from 1.01 mg/bhp-hr to 0.5 mg/bhp-hr. In either case all the EF were well below the certification standard of 10 mg/bhp-hr. Low PM results are expected for a NG fueled engine where previous studies showed similar PM emissions well below 10 mg/bhp-hr.

The measured filter weights were 13 ug with a single standard deviation of 3 ug where the tunnel blank was measured at 5 ug (representative of 0.42 g/bhp-hr using the UDDS sample conditions). As such, the PM emission rates are very low and the shown variability may be a result of measurement detection capability more than vehicle performance between cycles.

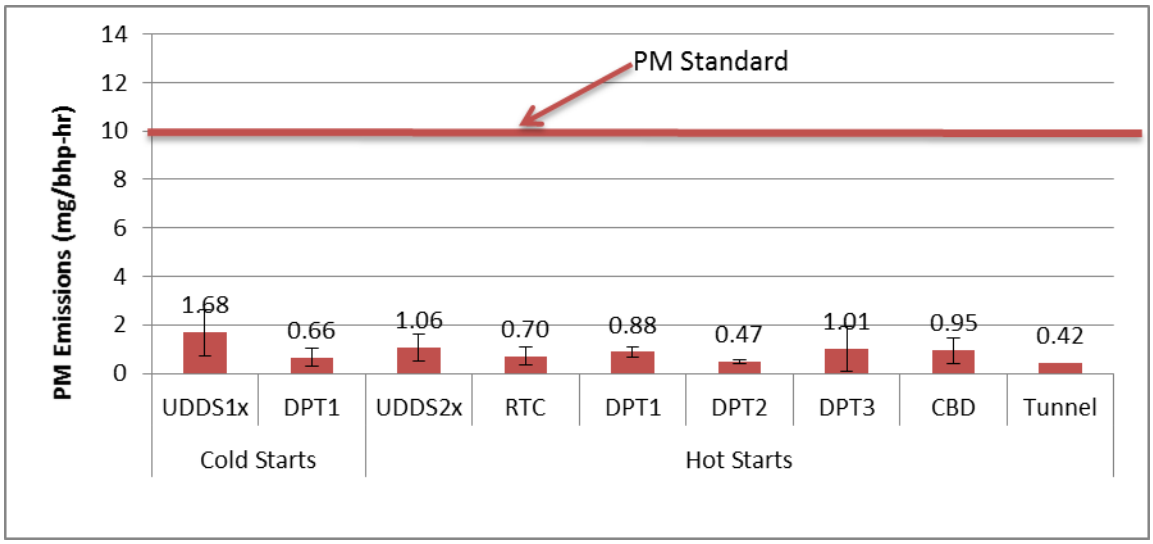


Figure 3-6 PM emission factors (mg/bhp-hr)

[1] Tunnel PM emission factor was based on a tunnel blank and test conditions of the UDDS 2x load conditions for the ISL G NZ test engine.

3.3 PN emissions

The PN emissions (CPC 3772) are shown in Figure 3-7 and Table 3-1 for the test cycles performed. The PN were highest for the high speed regional cycle (DPT3) on a total # basis, but were highest on a #/mi basis for the cold start near dock cycle (PDT1). Since the UDDS cycle is representative of the FTP certification cycle, comparisons to the hot UDDS cycle are presented in Table 3-2 (#/mi basis). The statistical analyses in Table 3-2 were conducted using a 2-tailed, 2 sample equal variance t-test. For the statistical analyses, results are considered to be statistically significant for $p < 0.05$, or marginally statistically significant for $0.05 < p < 0.1$. The near dock port cycle (DPT1) and the UDDS cold start showed statistically significant mean differences where the regional port cycle (DPT 3) showed marginally significant mean difference to the UDDS hot test. The cold start UDDS showed about three times the PN compared to the hot UDDS. The regional cycle showed about 82% more PN compared to the UDDS cycle and the near dock (DPT 1) showed 92% fewer PN. The trash compaction cycle (RTC) and the local port cycle (PDT 2) had similar PN emission rates and did not show statistically different means.

During previous studies with 0.2 g/bhp-hr certified NO_x ISL G engine tested on the near dock and regional port cycles, the PN emissions were $1.9 \times 10^{12} \pm 3.8 \times 10^{11}$ #/mi (11) which was about

92% lower than the ISL G NZ UDDS test cycle results, but about the same as the near dock port cycle. In a second study with the ISL G 8.9 liter engine, the PN emissions were $4x10^{12}$ for the CBD test cycle (10) which agrees well with the results in this study for the near dock and CBD test cycles. During a similar refuse hauler application of the ISL G engine, the PN emissions for the RTC cycle were $2.5x10^{13}$, $5.8x10^{12}$, and $2.0x10^{12}$ #/mi for the curbside, transit, and compaction portions of the RTC test cycle, respectively (12) which compare well with the PN from the ISL G NZ results. Late model diesel engines equipped with DPFs show PN emissions that range from $1.3x10^{11}$ to $0.7x10^{11}$ for on-road UDDS and cruise type of tests (18). In general the PN emissions for the ISL G NZ are mixed in comparison to the ISL G with some higher and some about the same. The ISL G NZ and ISL G both show higher PN emissions compared to diesel vehicles equipped with DPFs.

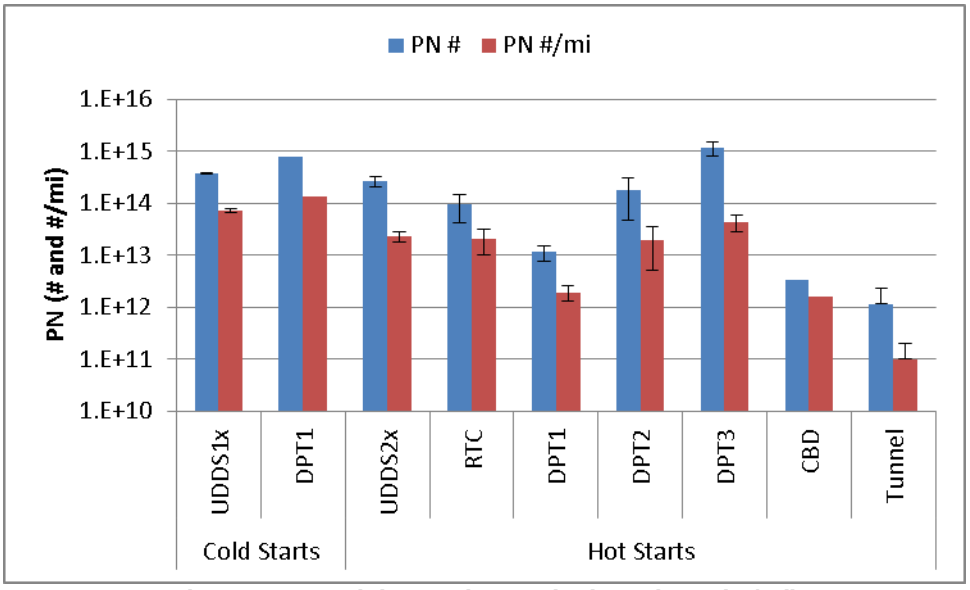


Figure 3-7 Particle number emissions (# and #/mi)

[1] Note the PN presented are based on CVS dilute measurements without sample conditioning (no volatile particle catalytic stripper system) and a D50 of 3 nm (CPC 3776). These PN values will be higher than those presented by the PMP system which uses a 3790A counter (24 nm D50 cut diameter and a volatile particle catalytic stripper system).

Table 3-1 PN Emissions from the ISL-G NZ 8.9 liter engine for various cycles

	PN #		PN #/mi	
Trace	ave	stdev	ave	stdev
CS_UDDS1x	3.80E+14	1.90E+13	7.25E+13	5.22E+12
CS_DPT1	7.87E+14		1.36E+14	
UDDS2x	2.66E+14	6.21E+13	2.37E+13	5.39E+12
RTC	9.49E+13	5.20E+13	2.12E+13	1.12E+13
DPT1	1.16E+13	3.83E+12	1.96E+12	6.25E+11
DPT2	1.83E+14	1.35E+14	2.01E+13	1.50E+13
DPT3	1.16E+15	3.46E+14	4.30E+13	1.51E+13
CBD	3.42E+12		1.62E+12	
Tunnel	1.15E+12	1.15E+12	1.02E+11	1.02E+11

[1] CS stands for cold start and Tunnel stands for tunnel blank. Stdev is a single standard deviation.

Table 3-2 Statistical comparison to the UDDSx2 test cycle

Cycle	t-test	f-test	mean % dif
CS UDDS	0.012	0.870	206%
RTC	0.492	0.388	-11%
DPT1	0.002	0.027	-92%
DPT2	0.721	0.230	-15%
DPT3	0.104	0.227	82%

[1] Unpaired two tailed sample equal variance t-test and mean % difference from the UDDSx2 test cycle

3.4 Ultrafines

The ultrafine PSD (as measured by the EEPS) are shown in Figure 3-8 on a log-log scale concentration basis as measured in the dilute CVS. The cold start UDDS and the regional (DPT3) cycles showed the highest particle number concentration at 10 nm particle diameter of all the traces. The higher PSD for the cold UDDS and regional cycle are a result of PN spikes under different conditions. The cold start UDDS PSD PN spike occurred during the cold portion and for the hot regional cycle (DPT3) the spike occurred during the cruise. The secondary peak at 105 nm particle diameter was highest for the same two cycles and the CBD. DPT1 showed the lowest PSD and was typically below the tunnel blank concentrations. During previous testing on the ISL G 8.9 liter engine the PSD showed a similar bi-modal PSD at 10 nm and 110 nm (10, 11, and 12). Diesel vehicles equipped with a DPF only show a single mode of operation (when not in a DPF regeneration) for the same UDDS and port cycles tested on the ISL G NZ vehicle (2).

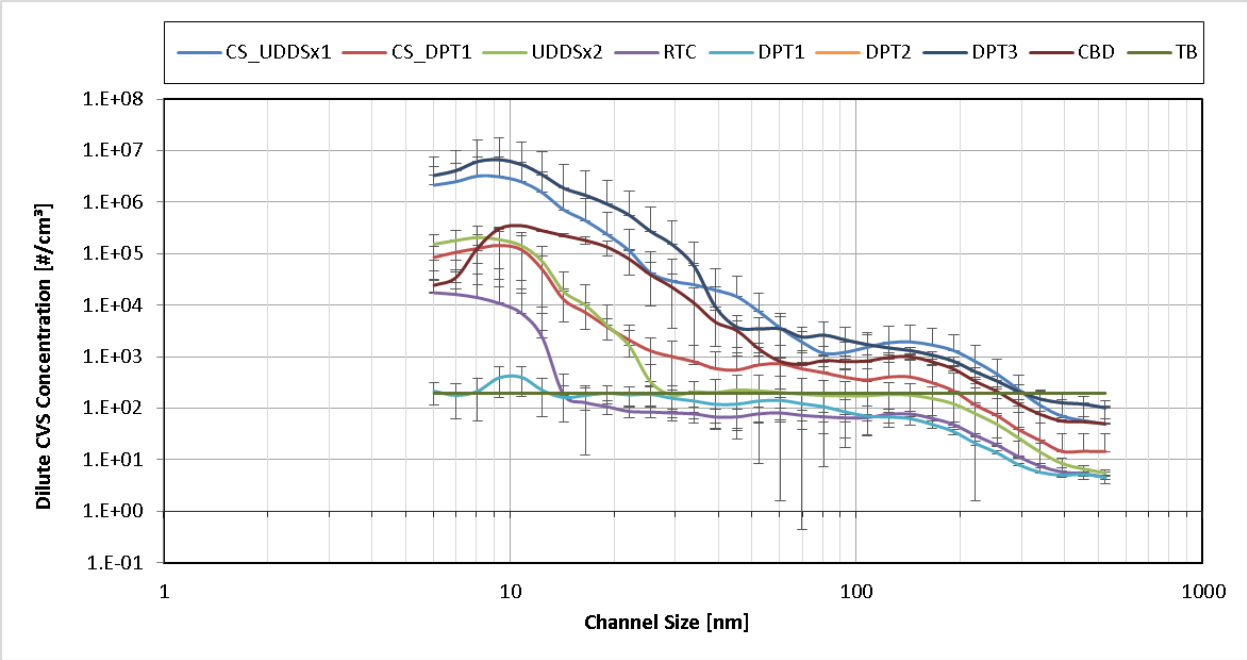


Figure 3-8 EEPS ultrafine PSD measurements for each of the test cycles

3.5 Greenhouse gases

The greenhouse gases include CO_2, CH_4 and N_2O and are reported here to characterize the vehicles global warming potential (GWP). The GWP calculations are based on the

intergovernmental panel on climate change (IPCC) values of 25 times CO_2 equivalent for CH_4 and 298 times CO_2 equivalent for nitrous oxide (N_2O), IPCC fourth assessment report - 2007. The global warming potential is provided in Table 3-3 on a g/bhp-hr basis (see Appendix E for g/mi basis). The CH_4 and N_2O emissions are low and represent 5% for the cold start tests and around 1-2% for the hot start tests.

Greenhouse gases from vehicles are also found in PM emissions for their absorption of solar radiation. The main species of the PM responsible for solar absorption is called black carbon (BC). BC is a short lived climate forcer and is not grouped with the CO_2 equivalent method, and is treated here separately. UCR quantified the BC emissions (referred to as equivalent black carbon eBC) from the vehicle with its AVL micro soot sensor 483 (MSS) which measures the PM soot or eBC. Table 3-3 lists the soot PM for each cycle and the ratio of soot/total PM emissions. The results suggest less than 10% of the PM measured for all the cycles except the regional port cycle are BC and during the regional cycle up to 22% of the total PM measured is BC. Additional analysis showed that the measured average concentration ranged between 2-3 ug/m3 when corrected for water interferences (as reported by manufacturer) the concentration was~ 1ug for all tests. The low concentrations are at the detection limits of the MSS instrument and suggests the measured BC cannot be quantified accurately, but may suggest BC is not significant for the ISL G NZ NG engine.

Table 3-3 Global warming potential for the ISLG NZ vehicle tested (g/bhp-hr)

Trace	CO_2	CH_4	N_2O	GWP ($CO_{2\,eq}$)	CO_2/GWP	Soot	Soot/$PM_{2.5}$
UDDS1x	546.8	0.53	0.062	578.5	0.95	0.05	3%
DPT1	627.0	0.56	0.090	667.7	0.94	0.02	3%
UDDS2x	548.9	0.04	-	555.0	0.99	0.06	5%
RTC	577.0	0.08	-	584.0	0.99	0.01	1%
DPT1	649.8	0.26	-	661.4	0.98	0.07	8%
DPT2	597.0	0.16	0.027	608.9	0.98	0.1	22%
DPT3	549.3	0.33	0.024	564.4	0.97	0.01	1%
CBD	576.1	0.11	0.034	589.0	0.98	0.04	4%

[1] N_20 samples were not collected on the hot UDDS, RTC, and DPT1 due to scheduling details. PM Soot measurements were near the detection limits of the MSS-483 measurement system. The MSS soot signal was corrected for a 1 ug/1% water interference factor as reported by AVL.

3.6 Fuel economy

The fuel economy of the NG vehicle is evaluated by comparing the CO_2 emissions between cycles where the higher the CO_2 the higher the fuel consumption. CO_2 is also regulated by EPA with a standard as performed with the FTP and SET test cycles. The certification like cycle (UDDS) showed the lowest CO_2 emissions and were below 555 g/bhp-hr (FTP standard) for both the cold start and hot start tests. The NG vehicle CO_2 emissions varied slightly between cycles where only the near dock cycle (DPT1) showed a statistically higher CO_2 emission rate. The average CO_2 for all the cycles was 584 g/bhp-hr, and 565 g/bhp-hr with the PDT1 cycle removed. The CO_2 standard and certification value is 555 g/bhp-hr and 465 g/bhp-hr respectively for this displacement engine, see Figure F1 Appendix F. The standard is the target and the certification value is the value measured by the manufacturer. It is suggested the higher in-use CO_2 value (ie in the chassis vs on a test stand) could be a result of additional losses in the chassis where the certification test occurs with the engine on a test stand.

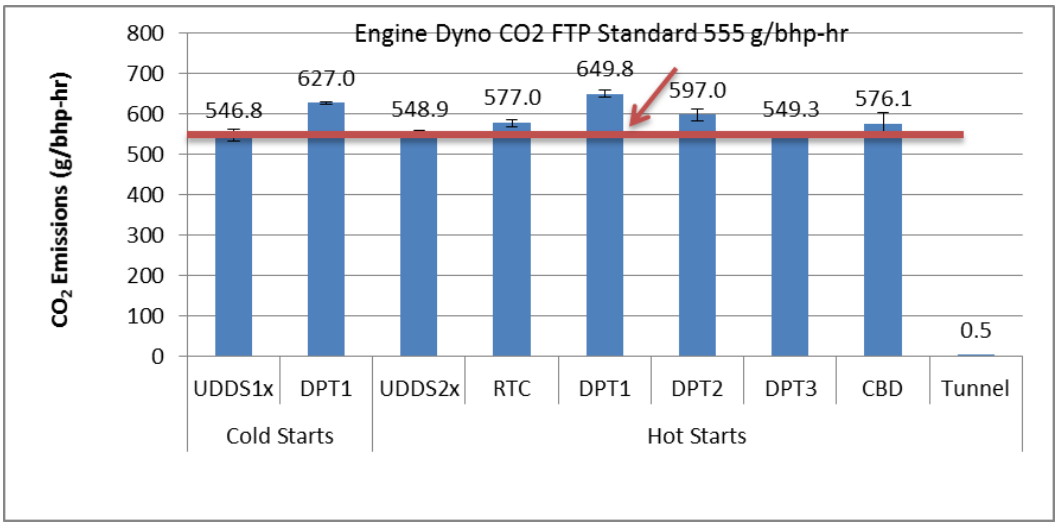


Figure 3-9 CO_2 emission factors (g/bhp-hr)

The ISL G-NZ MPG on a diesel gallon equivalent (MPG_{de}) basis (assuming 2863gNG/gallon diesel (14)) ranges from 4.5 MPG_{de} for the regional port cycle (DPT3) to 2.5 MPG_{de} for the CBD cycle. During previous testing, the previous ISL G 8.9 L fuel economy was found to be 2365 g/mi on a chassis dynamometer at 56,000 GVW following the UDDS test cycle.

4 Discussion

This section discusses investigation into the real-time data to characterize the impact of the cold start and transient NO_x emissions.

4.1 Transient emissions

Figure 4-1 shows the real-time NO_x mass emission rate (g/sec) for the three repeated UDDS cycles. Test 0813 and 1020 had large NO_x spikes, one near the beginning of the test and one near the end of the test where test ID 0915 had only small spikes which are not apparent in Figure 4-1. This indicates that NOx emissions are essentially zero except during sharp accelerations. Figure 4-2 shows the accumulated NO_x emissions as a function of time. The results in Figure 4-2 show the impacts the large and small spikes have on the accumulated NO_x emissions. Test 0915 and 1020 were very similar except for the large spike near the end of the 1020 test.

Figure 4-3 shows the percent of total NOx accumulate as a function of time. The one large spike for test 1020 represented 90% of the total emissions. If the single NOx spike did not occur, the EF for the triplicate cycle would have been close to 0.005 g/bhp-hr instead of the 0.014 g/bhp-hr reported. Figure 4-4 shows the real time NOx emission rate (g/s) exhaust flow, engine RPM, and engine power at the time where the spike occurred. The NOx spike appears to be occurring at the transition from idle to loaded conditions. The figure shows that NOx emission rate and exhaust flow are lined up well suggesting there is not a measurement issue but a real event. In general the transient nature of the emissions suggest the NOx emission are low and are typically below 0.02 g/bhp-hr when good control of the engine stoichiometry is maintained.

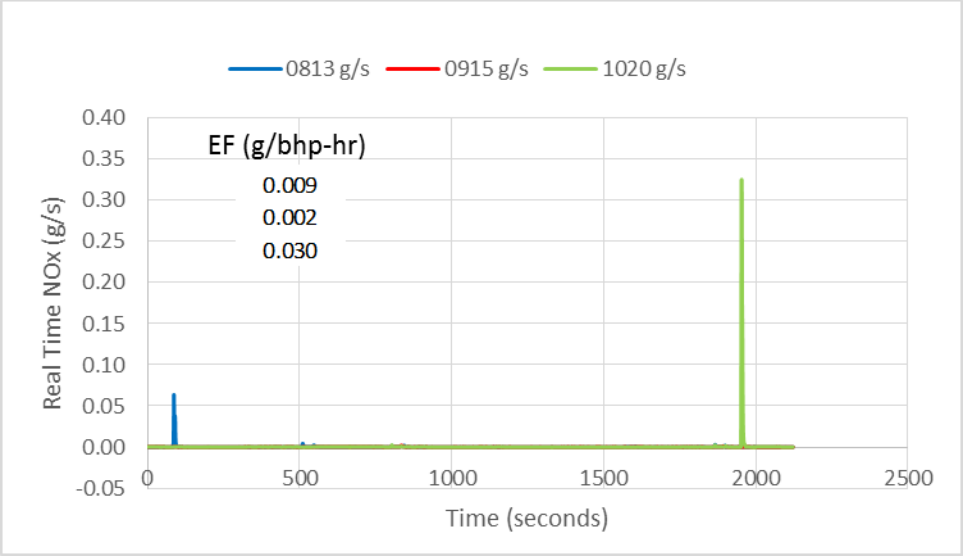


Figure 4-1 Real-time mass rate NOx emissions (g/sec) UDDS cycles

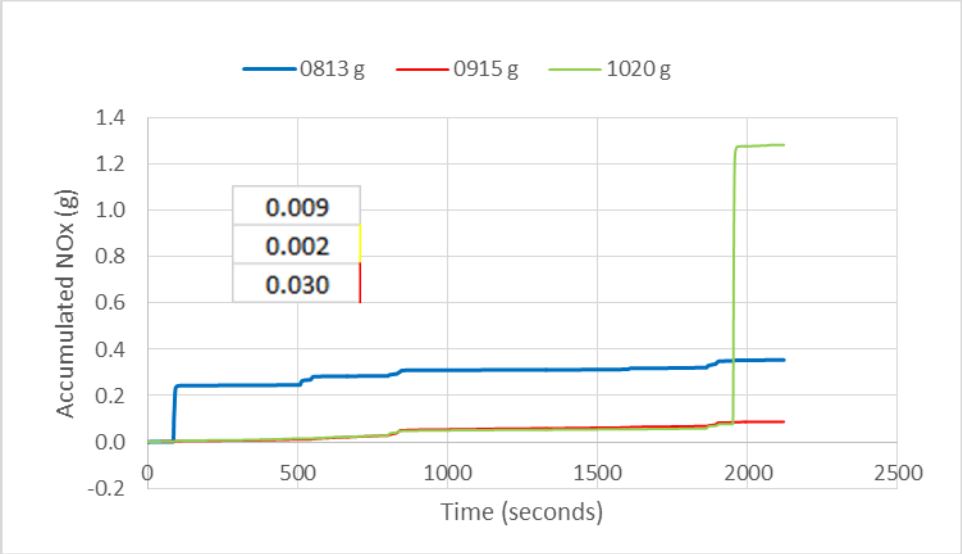


Figure 4-2 Accumulated mass NOx emissions UDDS cycles

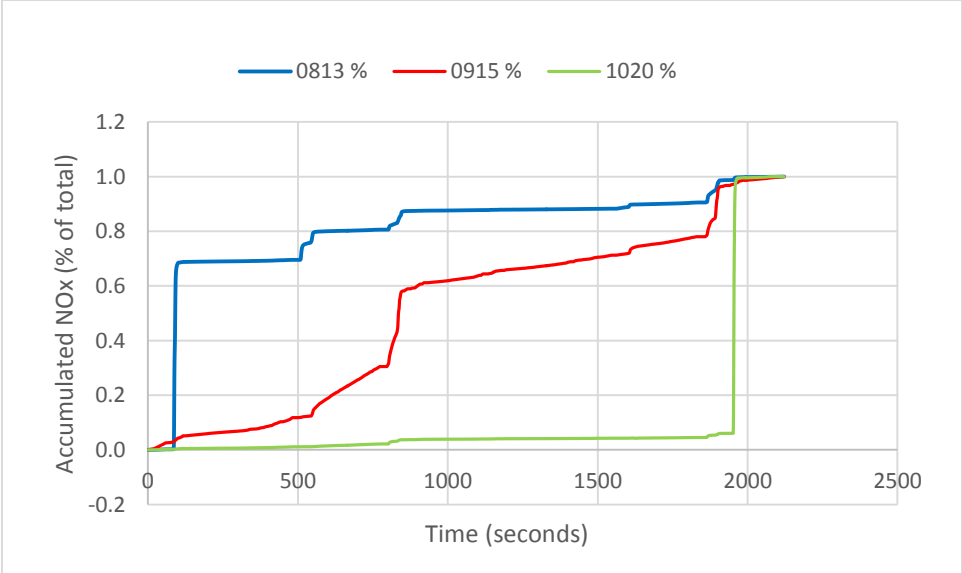


Figure 4-3 Real time NOx emissions (percent of total)

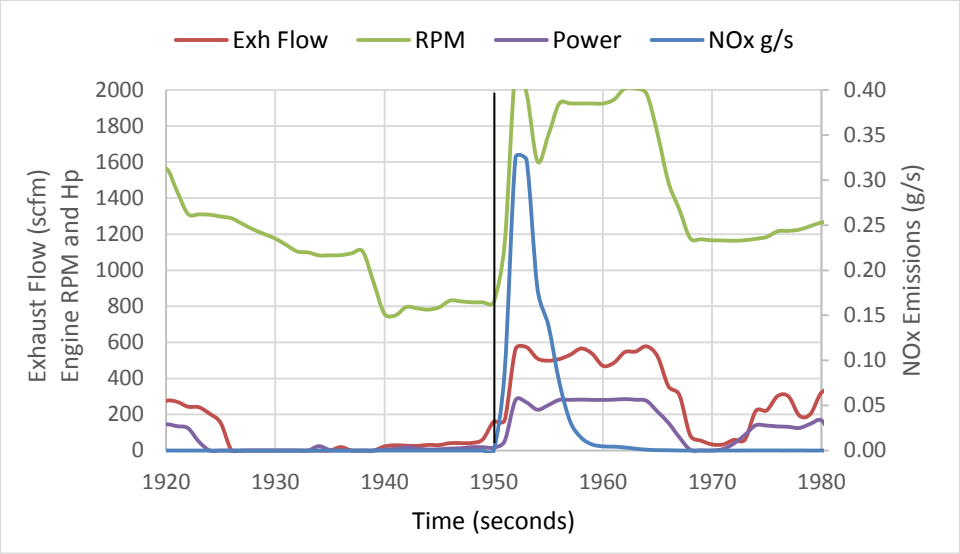


Figure 4-4 Real time NOx emissions large spike evaluation

4.2 Cold start emissions

Cold start emissions represented a significant part of the total emissions as one would expect. Figure 4-5 shows the accumulated NO_x (g) and exhaust temperature as a function of time. 90% of the NO_x emissions occurred in the first 200 seconds of the cold start test. The remaining part of the cold UDDS test was very similar to the hot UDDS test. The UDDS hot/cold weighted emissions is 0.0181 g/bhp-hr (weighted as $1/7^{th}$ of the hot cycle). Given that the cold start lasted 200 seconds out of 1080 seconds (total cycle length) the weighted cold start emissions ($1/7^{th}$ of the hot test) are, thus, based on 200sec/1080sec/7 = 2.6%. This suggests 2.6% of this vehicles in-use emissions are represented by a cold start as defined by how the certification process computes its impact for the regulation process. Also unique to the NG solution, once the catalyst performance is achieved it remains at this high performance unlike the diesel SCR equipped engines where low duty cycle will cause the NOx emissions to increase again.

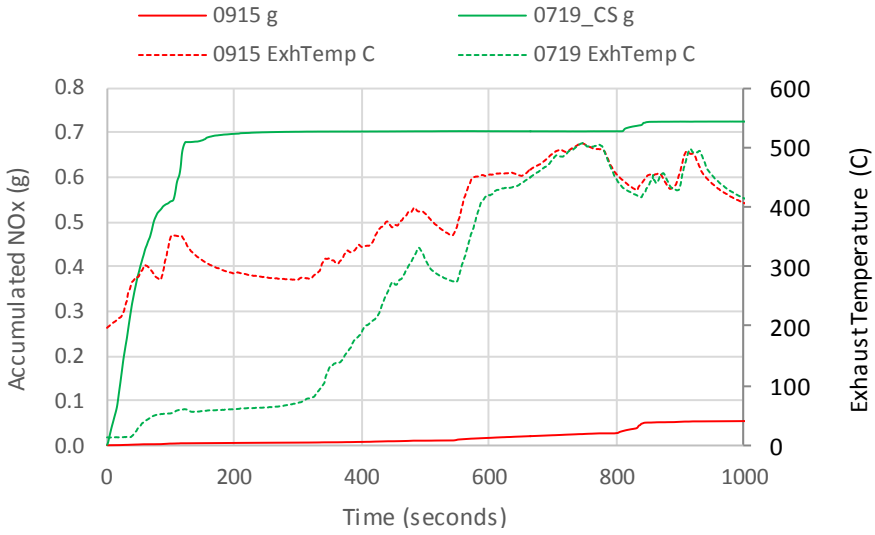


Figure 4-5 Accumulated NOx emissions hot vs cold UDDS comparison

5 Summary and Conclusions

The testing was performed on UC Riverside's chassis dynamometer integrated with its mobile emissions laboratory (MEL) located in Riverside CA just east of the South Coast Air Quality Management District (SCAQMD). The cycles selected for this study are representative of operation in the South Coast Air Basin and included the urban dynamometer driving schedule, the near dock, local, and regional port cycles, the AQMD refuse cycle, and the central business district cycle.

One of the difficulties in quantifying NO_x emissions at the levels proposed in this research (90% below the 2010 certification level ~ 0.02 g/bhp-hr) is the dilute measurement methods are too close to the detection limit to quantify NO_x emissions at the 5% accuracy expected from the emissions industry. Three upgraded NO_x measurement methods were considered which include a raw NO_x measurement integrated with real time exhaust flow, a real-time ambient correction approach, and a trace level ambient analyzer for accurate bag analysis. In summary the improved methods varied in their success; however, the raw sampling approach was the most accurate and precise over the range of conditions tested.

In general the ISL G NZ 8.9 met and exceeded the target NO_x emissions of 0.02 g/bhp-hr and maintained those emissions during a range of duty cycles found in the South Coast Air Basin. It is expected NG vehicles could play a role in the reduction of the south coast NOx inventory problem given their near zero emission factors demonstrated

The main conclusions can be summarized as (conclusions are based on the Method 2 results unless noted otherwise):

1. The ILS G NZ 8.9 liter NG engine showed NO_x emissions below the 0.02 g/bhp-hr emission target and averaged between 0.014 and 0.002 g/bhp-hr for hot start tests.
2. The cold start tests ranged from 0.043 to 0.014 g/bhp-hr for the UDDS and DPT2 cycles. The UDDS hot/cold weighted emissions was 0.0181 g/bhp-hr for all test cycles performed which is below the certified 0.02 g/bhp-hr emission factor.
3. The NO_x emissions did not increase with lower power duty cycles and showed the opposite trend where the lower power duty cycles showed lower NO_x emissions unlike the diesel counterparts
4. The large NO_x error bars suggest measurement variability, but real-time data shows the variability is isolated to a few NO_x events during rapid tip-in events from accelerations from idle. This suggests possible driver behavior may impact the overall NO_x in-use performance of the vehicle and more gradual accelerations are desired for minimum emissions.
5. This suggests possible driver behavior may impact the overall NO_x in-use performance of the vehicle where more gradual accelerations are desired.
6. The other gaseous and PM emissions were similar to previously measured levels from the 0.2 g/bhp-hr ISL G engine and should not add to any unknown impacts for the use of the NZ engine in the heavy duty fleet.

References

1. AQMD 2 October 2015 see: http://www.aqmd.gov/docs/default-source/Agendas/aqmp/white-paper-working-groups/wp-blueprint-revdf.pdf?sfvrsn=2
2. Wayne Miller, Kent C. Johnson, Thomas Durbin, and Ms. Poornima Dixit 2013, In-Use Emissions Testing and Demonstration of Retrofit Technology, Final Report Contract #11612 to SCAQMD December 2013
3. Hesterberg T., Lapin C., Bunn A., Navistar, Inc. 4201 Winfield Road, P.O. Box 1488, Warrenville, Illinois 60555, VOL. 42, NO. 17, 2008 / ENVIRONMENTAL SCIENCE & TECHNOLOGY 9 6437
4. Thirubvengadam A., Besch M., Pradhan S., Carder D., and Emission Rates of Regulated Pollutants from Current Technology Heavy-Duty Diesel and Natural Gas Goods Movement Vehicles. ENVIRONMENTAL SCIENCE & TECHNOLOGY 2015, 49, 5236−5244
5. Patrick Couch, John Leonard, TIAX Development of a Drayage Truck Chassis Dynamometer Test Cycle, Port of Long Beach/ Contract HD-7188, 2011.
6. Results from UC Riverside's Chassis Dyno while testing an 8.9 liter heavy duty vehicle at transient and state operating modes.
7. Chatterjee, D., Deutschmann, O., and Warnatz, J., Detailed surface reaction mechanism in a three-way catalyst, Faraday Discussions, 119, pg 371-384 (2001).
8. Cocker III, D. R., Shah, S. D., Johnson, K. C., Zhu, X., Miller, J. W., Norbeck, J. M., Development and Application of a Mobile Laboratory for Measuring Emissions from Diesel Engines. 2. Sampling for Toxics and Particulate Matter, Environ. Sci. Technol. 2004, 38, 6809-6816.
9. Cocker III, D. R, Shah, S. D., Johnson, K. C., Miller, J. W., Norbeck, J. M., Measurement Allowance Project – On-Road Validation. Final Report to the Measurement Allowance steering Committee.
10. George Karavalakis, Yu Jiang, Jiacheng Yang, Maryam Hajbabaei, Kent Johnson, Thomas Durbin, 2016, Gaseous and Particulate Emissions from a Waste Hauler Equipped with a Stoichiometric Natural Gas Engine on Different Fuel Compositions, SAE Technical Paper No. 2016-01-0799, Society of Automotive Engineers, World Congress 2016.
11. Hajbabaei, M., Karavalakis, G., Johnson, K.C, Lee, L., and Durbin, T.D., 2013, Impact of natural gas fuel composition on criteria, toxic, and particle emissions from transit buses equipped with lean burn and stoichiometric engines, Energy, 62, 425-434.
12. George Karavalakis, Maryam Hajbabaei, Yu Jiang, Jiacheng Yang, Kent C. Johnson, David R. Cocker; Thomas D. Durbin, 2016, Regulated, Greenhouse Gas, and Particulate Emissions from Lean-Burn and Stoichiometric Natural Gas Heavy-Duty Vehicles on Different Fuel Compositions, Fuel, 175, 146-156.

13. Johnson, K., C., Durbin, T., Khan, Y., M., Jung, H., Cocker, D., (2010). Validation Testing for the PM-PEMS Measurement Allowance Program. California Air Resources Board, November 2010, Contract No. 07-620

14. Johnson, K.C., Durbin, T.D., Cocker, III, D.R., Miller, W.J., Bishnu, D.K., Maldonado, H., Moynahan, N., Ensfield, C., Laroo, C.A. (2009) On-road comparison of a portable emission measurement system with a mobile reference laboratory for a heavy-duty diesel vehicle, Atmospheric Environment 43 (2009) 2877–2883

15. Cocker III, D. R, Shah, S. D., Johnson, K. C., Miller, J. W., Norbeck, J. M., Development and Application of a Mobile Laboratory for Measuring Emissions From Diesel Engines I. Regulated Gaseous Emissions, Environmental Science and Technology. 2004, 38, 2182-2189.

16. Miller W., Johnson K., C., Durbin T., Dixit P., (2013) In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines.

17. L&R Committee 2014 Final Report Appendix A ¬– Items: 232-2, 232-3, 237-1, 237-3, and 237-5: GGE of Natural Gas as Vehicular Fuel.

18. Zhongqing Zheng, Thomas D. Durbin, Jian Xue, Kent C. Johnson, Yang Li, Shaohua Hu, Tao Huai, Alberto Ayala, David B. Kittelson, and Heejung S. Jung, Comparison of Particle Mass and Solid Particle Number (SPN) Emissions from a Heavy-Duty Diesel Vehicle under On-Road Driving Conditions and a Standard Testing Cycle, Environ. Sci. Technol. 2014, 48, 1779 – 1786.

Appendix A. Test Log

This Appendix contains detailed test logs recorded during engine and chassis dynamometer testing. The testing was performed on Vehicle ID 2015_016, Project Low NOx 2015, Vehicle VIN = 3BPZX20X6FF100173 with the test mode in Conventional mode. The chassis and vehicle operators were Eddie and Don for all the testing and the instrument operators were Mark, Jade, Danny and Joey.

Date	Test Time	Test Cycle	Test ID	Hp @ 50	Weight	A	B	C
11/16/2015	14:43	Refuse	201511161358	117.42	56000	397.73642	-2.43E-14	0.193166
11/16/2015	14:56	Compaction Cycle	201511161358	117.42	56000	397.73642	-2.43E-14	0.193166
11/18/2015	7:33	UDDS_CS_1x	201511180727	117.42	56000	397.73642	-2.43E-14	0.193166
11/18/2015	8:17	UDDS_x2	201511180813	117.42	56000	397.73642	-2.43E-14	0.193166
11/18/2015	9:22	UDDS_x2	201511180915	117.42	56000	397.73642	-2.43E-14	0.193166
11/18/2015	10:23	UDDS_x2	201511181020	117.42	56000	397.73642	-2.43E-14	0.193166
11/18/2015	12:14	RTC_DPF_NG	201511181280	117.42	56000	397.73642	-2.43E-14	0.193166
11/19/2015	7:22	UDDS_CS_1x	201511190719	117.42	56000	397.73642	-2.43E-14	0.193166
11/19/2015	7:48	Compaction Cycle	warmup	117.42	56000	397.73642	-2.43E-14	0.193166
11/19/2015	8:13	RTC_DPF_NG	201511190809	117.42	56000	397.73642	-2.43E-14	0.193166
11/19/2015	8:54	RTC_DPF_NG	201511190809	117.42	56000	397.73642	-2.43E-14	0.193166
11/19/2015	9:35	RTC_DPF_NG	201511190929	117.42	56000	397.73642	-2.43E-14	0.193166
11/19/2015	10:16	RTC_DPF_NG	201511190929	117.42	56000	397.73642	-2.43E-14	0.193166
11/19/2015	10:58	DTP_1	201511191051	117.42	56000	397.73642	-2.43E-14	0.193166
11/19/2015	12:58	DTP_1	201511191255	117.42	56000	397.73642	-2.43E-14	0.193166
11/19/2015	14:16	DTP_1	201511191412	117.42	56000	397.73642	-2.43E-14	0.193166
11/20/2015	7:19	DTP_1_CS	201511200716	117.42	56000	397.73642	-2.43E-14	0.193166
11/20/2015	8:41	DTP_2	201511200838	117.42	56000	397.73642	-2.43E-14	0.193166
11/20/2015	10:04	DTP_2	201511200959	117.42	56000	397.73642	-2.43E-14	0.193166
11/20/2015	11:24	DTP_2	201511201122	117.42	56000	397.73642	-2.43E-14	0.193166
11/23/2015	7:24	DTP_1_CS	201511230717	117.42	56000	397.73642	-2.43E-14	0.193166
11/23/2015	8:45	DTP_3	201511230840	117.42	56000	397.73642	-2.43E-14	0.193166
11/23/2015	10:18	DTP_3	201511231015	117.42	56000	397.73642	-2.43E-14	0.193166
11/23/2015	12:35	DTP_3	201511231225	117.42	56000	397.73642	-2.43E-14	0.193166
11/23/2015	2:10	CBD	201511231408	117.42	56000	397.73642	-2.43E-14	0.193166

Date	Test Time	Test Cycle	Test ID	Hp @ 50	Weight	A	B	C
11/25/2015	8:27	UDDS_CS_1x	201511250820	117.42	56000	397.73642	-2.43E-14	0.193166
11/25/2015	9:13	CBD	201511250907	117.42	56000	397.73642	-2.43E-14	0.193166
11/25/2015	9:48	CBD	201511250946	117.42	56000	397.73642	-2.43E-14	0.193166

Appendix B. Test Cycle Description

The test vehicle utilizes an 8.9 liter NG engine which is available for three typical vocations in the South Coast Air Basin, 1) goods movement, 2) transit bus, and 3) refuse. As such UCR tested the vehicle following the three drayage type port cycles (Near Dock, Local, and Regional), the Urban Dynamometer Driving Schedule (UDDS), the Central Business District (CBD) bus cycle, and the AQMD Refuse cycle. These cycles are representative of Sothern California driving vocations used. Some cycles are very short (less than 30 minutes) where double or triple cycles (2x or 3x) cycles are recommended in order capture enough PM mass to quantify emissions near 1 mg/bhp-hr.

Drayage Truck Port (DTP) cycle

TIAX, the Port of Long Beach and the Port of Los Angeles developed the port cycle. Over 1,000 Class 8 drayage trucks at these ports were data logged for trips over a four-week period in 2010. Five modes were identified based on several driving behaviors: average speed, maximum speed, energy per mile, distance, and number of stops. These behaviors are associated with different driving conditions such as queuing or on-dock movement, near-dock, local or regional movement, and highway movements (see Table B-1 for the phases). The data was compiled and analyzed to generate a best fit trip (combination of phases). The best-fit trip data was then additionally filtered (eliminating accelerations over 6 mph/s) to allow operation on a chassis dynamometer.

The final driving schedule is called the drayage port tuck (DPT) cycle and is represented by 3 modes where each mode has three phases to best represent near dock, local, and regional driving as shown in Table B-1, B-2 and Figure B-1. The near-dock (DTP-1) cycle is composed of phase 1, 2, and 3a from Table B-1. This gives the complete near-dock cycle listed in Table B-2. Similarly, for the Local and Regional cycles (DPT-2 and DPT-3) the main difference is phase 3, which changes to 4 and 5 respectively. Phase 1 and 2 remain the same for all three cycles where creep and low speed transient are considered common for all the port cycles. For this testing it is recommended to perform phase 1 through 5 individually and to calculate the weighted emissions from the combined phases for an overall weighing impact.

Table B-1. Drayage Truck Port cycle by phases

Description	Phase #	Distance mi	Ave Speed mph	Max Speed mph	Cycle length
Creep	1	0.0274	0.295	4.80	335
low speed transient	2	0.592	2.67	16.8	798
short high speed transient	3	4.99	9.39	40.6	1913
Long high speed transient	4	8.09	13.07	46.4	2229
High speed cruise	5	24.6	35.04	59.3	2528

Table B-2. Drayage Truck Port cycle by mode and phases

Description	Distance mi	Ave Speed mph	Max Speed Mph	Mode 1	Mode 2	Mode 3
Near-dock PDT1	5.61	6.6	40.6	Creep	Low Speed Transient	Short High Speed Transient
Local PDT2	8.71	9.3	46.4	Creep	Low Speed Transient	Long High Speed Transient
Regional PDT3	27.3	23.2	59.3	Creep	Low Speed Transient	High Speed Cruise

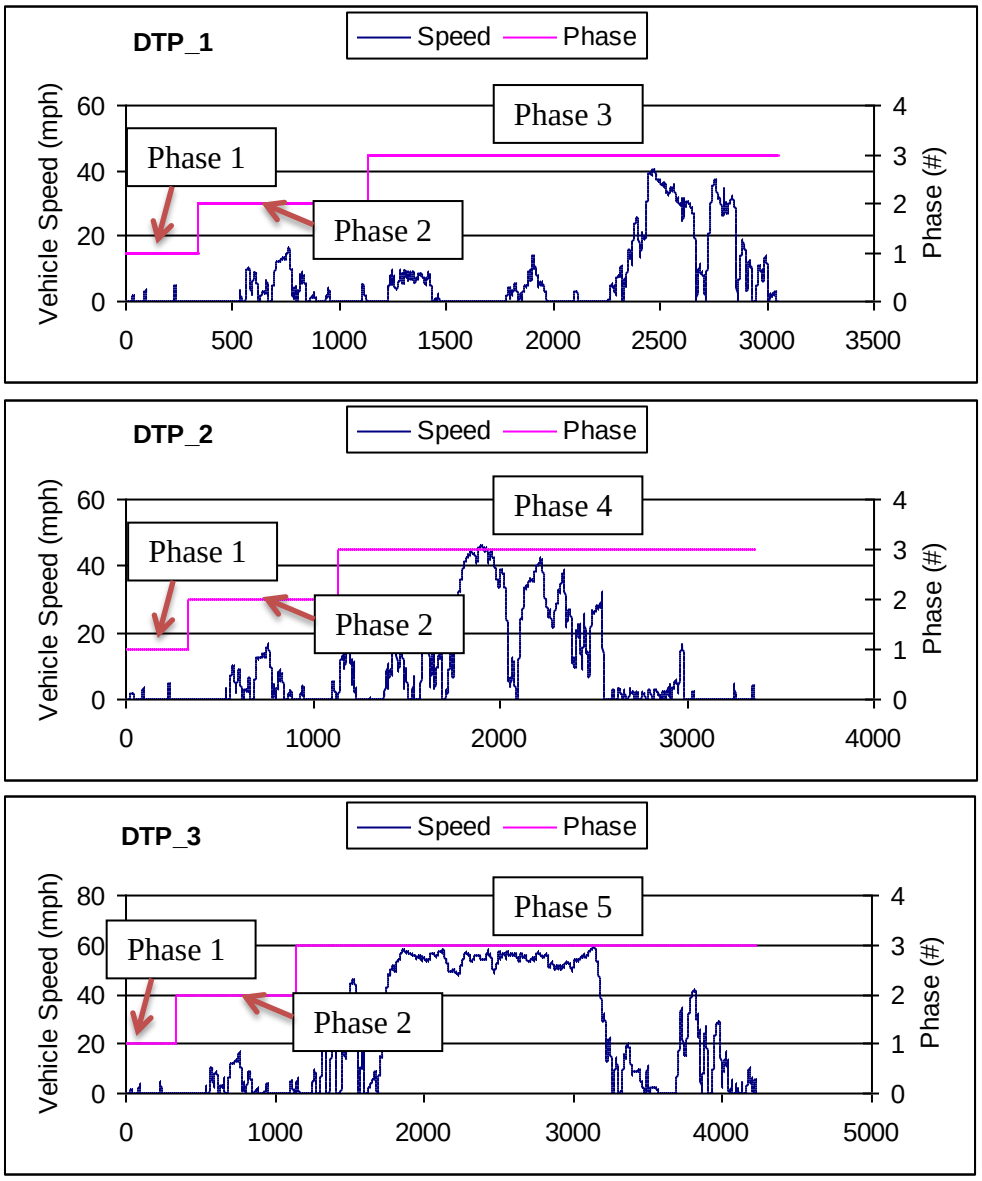


Figure B-1 Drayage truck port cycle near dock, local, and regional

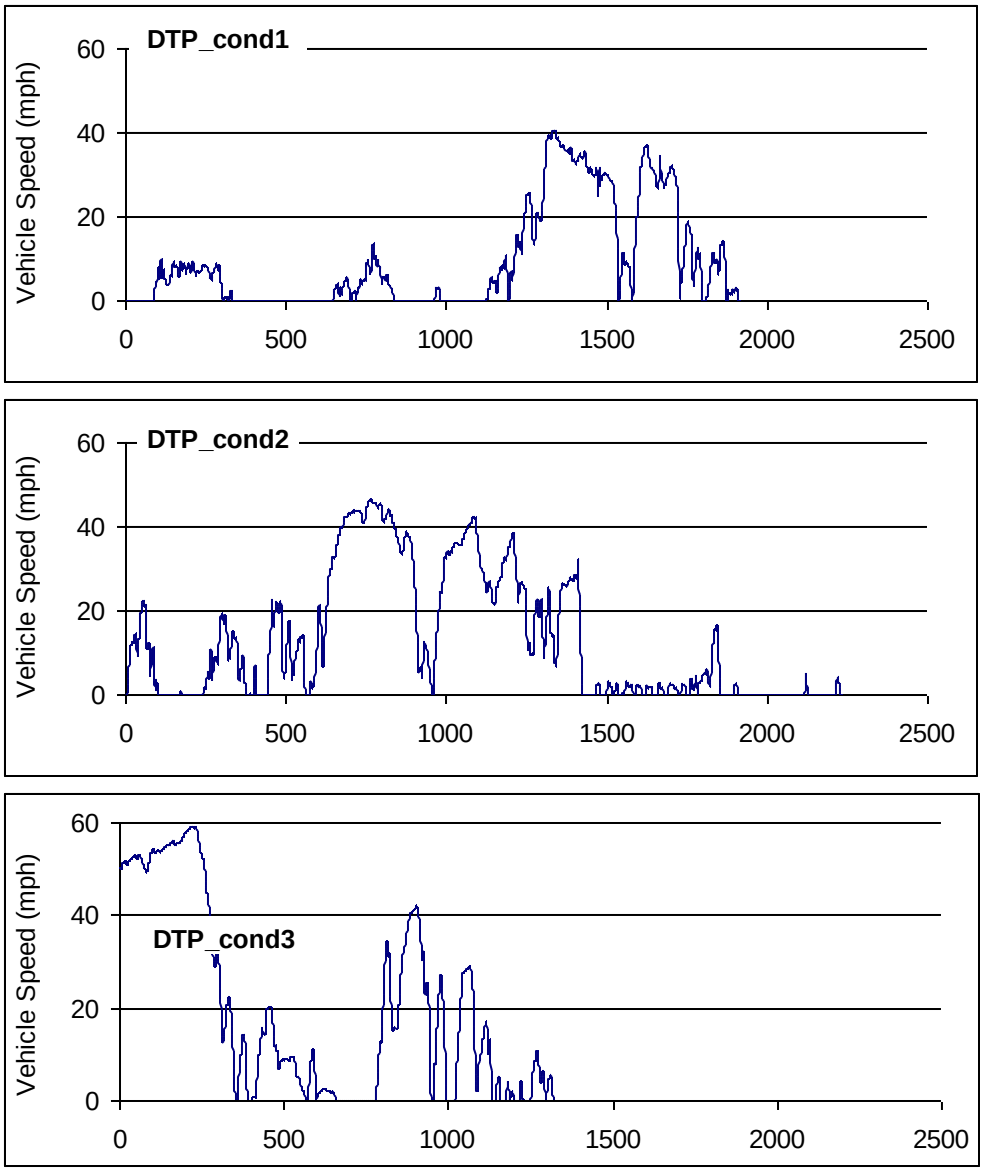


Figure B-2 Drayage truck port cycle conditioning segments consisting of phase 3 parts

Urban Dynamometer Driving Schedule (UDDS) description

The Federal heavy-duty vehicle Urban Dynamometer Driving Schedule (UDDS) is a cycle commonly used to collect emissions data on engines already in heavy, heavy-duty diesel (HHD) trucks. This cycle covers a distance of 5.55 miles with an average speed of 18.8 mph, sample time of 1061 seconds, and maximum speed of 58 mph. The speed/time trace for the HUDDS is provided below in Figures B-3. This cycle was used for all cold start tests as a single test and was performed in duplicate for all hot tests. Duplicates were used to accumulate sufficient mass for the gravimetric measurement method.

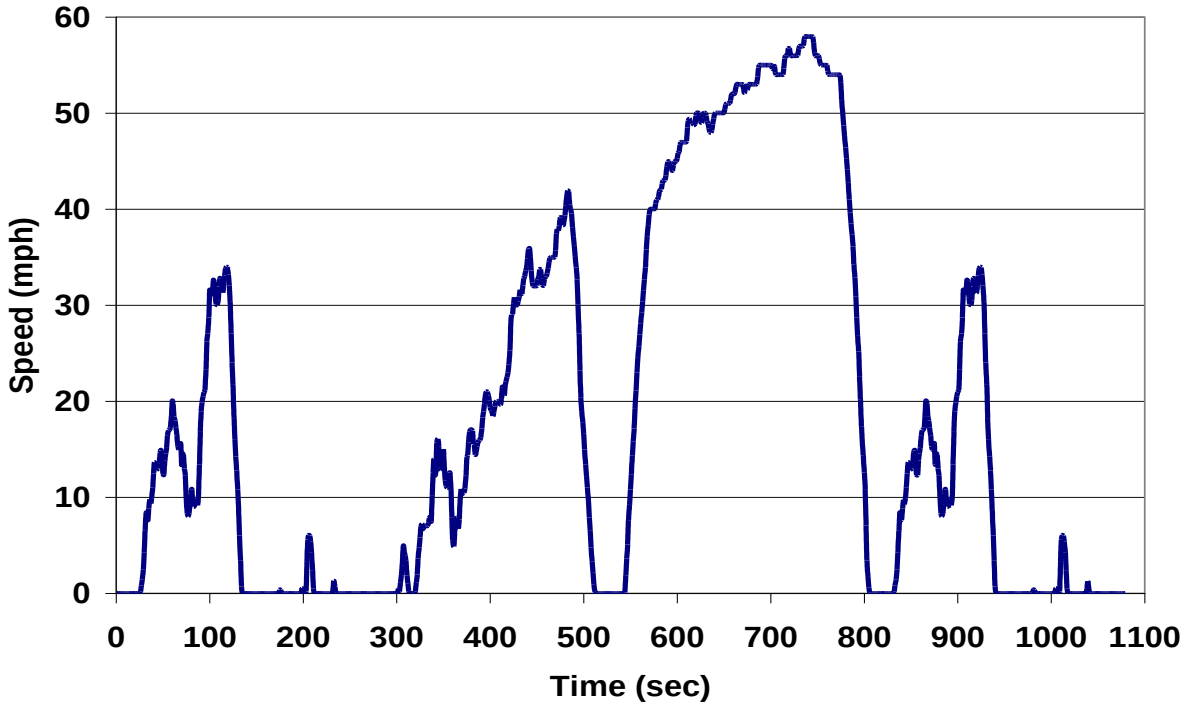


Figure B-3. Speed/Time Trace for a 1xHUDDS cycle for the chassis dynamometer.

The AQMD refuse truck cycle

The AQMD refuse truck cycle (AQMD-RTC) is the same as the WHM-RTC in that the cycle consists of a transport, curbside and compaction operation, with the main difference being the length of time and arrangement of the individual modes. The duration of the AQMD-RTC transport and curbside is 2127 seconds, representing a distance of 4.56 miles and the compaction adds another 760 seconds for a total of 2887 seconds. Figure A-4 shows the vehicle speed vs. time trace for the cycle preparation, transport (phase 1) and curbside (phase 2) portion of the cycle. The curb side pick-up mode is representative of multiple short idle times with frequent stop-and-go operation. The cycle is characterized by frequent accelerations and decelerations. The frequent stop-and-go operation could lead to lower catalytic activity and higher mass tailpipe emissions rates.

Real-world compaction operation was obtained from ECU engine load. It was observed that the engine load varied from 80 to 20 hp in a cyclical manner. The compaction cycle is simulated with the vehicle operating at steady-state speed of 30 mph with an intermittent engine of 80 hp and 20 hp. The total duration of the compaction cycle (phase 3) is 880 seconds, see Figure A-5 for the vehicle speed vs. time trace and axle power loading of the compaction cycle. The emissions are collected for only the stabilized speed which occurs 80 seconds into the trace and ends 40 seconds before the end of the trace for a total of 760 seconds.

Since, the compaction operation does not accrue any driving miles in real-world, the emissions from the compaction cycle are represented on a time-specific basis. Further, in order to represent the distance-specific emissions of the refuse truck operation as a whole, the total mass of emissions from the compaction cycle is added to the transport and curbside emissions divided by

the distance of the transport and curbside portion. Thus, it is expected the distance specific emissions on the refuse cycle will be higher than the transport plus curbside emissions since the compaction cycle didn't accumulate any distance.

UCR's MEL was configured with the conditioning and transport plus triple curbside into a signal cycle where the sampling was started at second 526 (Start of Transport Phase 1). After completing Phase 2 (Curbside), the compaction cycle was loaded and the driver brought the vehicle speed up to 30 mph and then the dyno was put in a load cycle mode that oscillated from 20 to 80 hp as shown in Figure B-5.

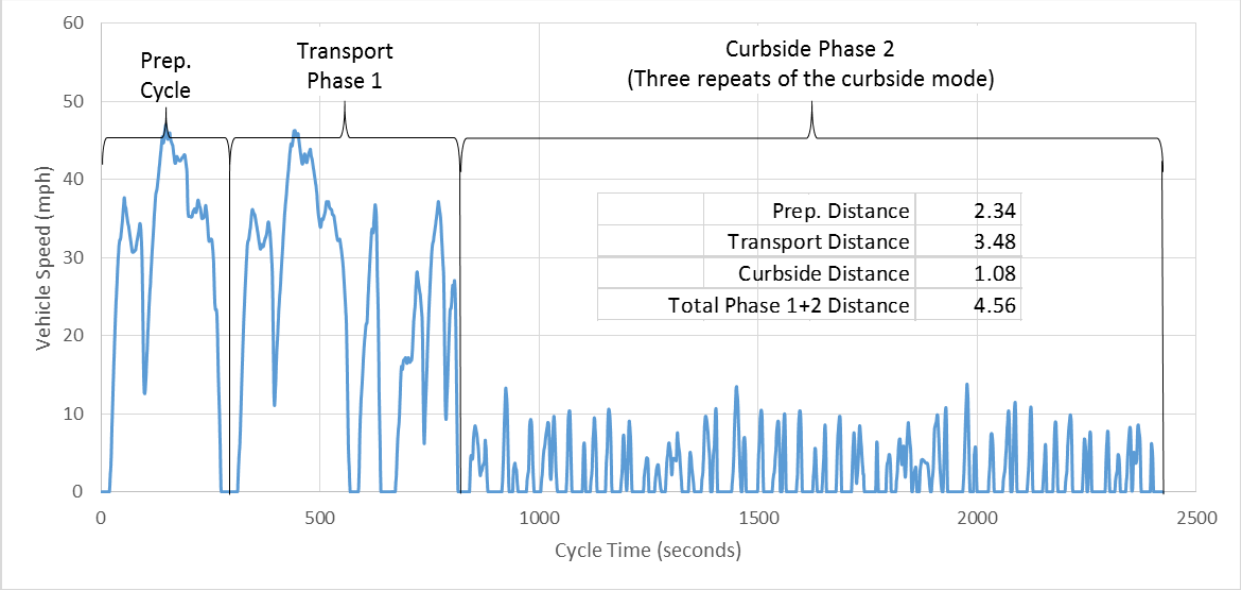


Figure B-4 Speed trace for AQMD refuse truck driving cycle

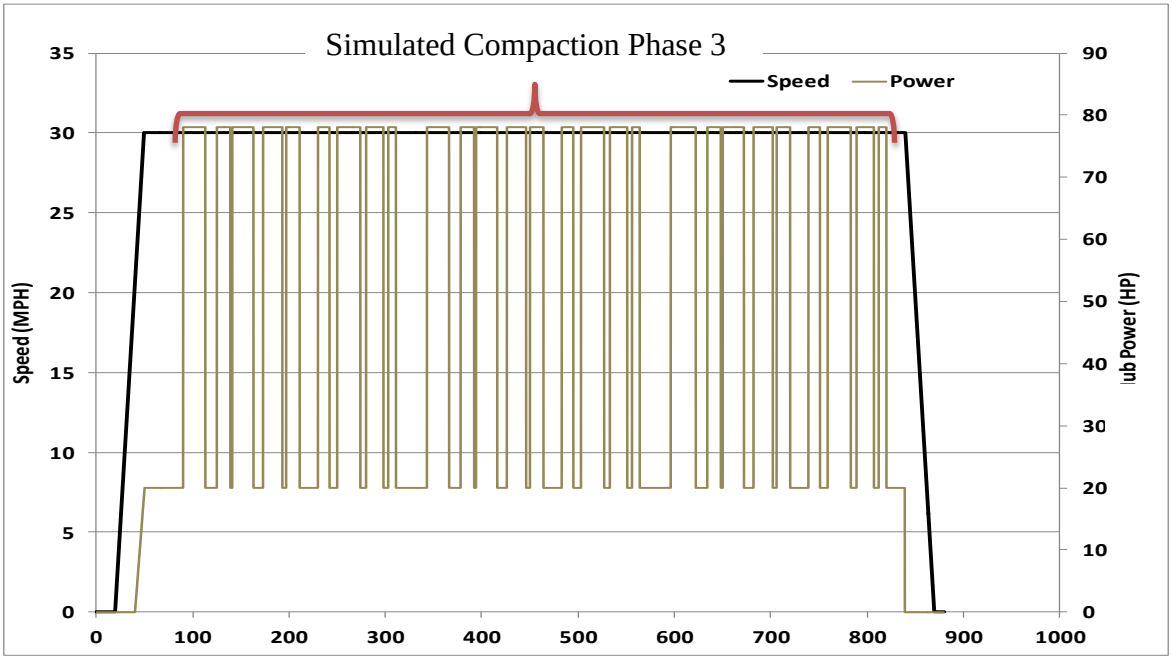


Figure B-5 Speed trace for AQMD refuse truck compaction cycle

Central Business District (CBD) Cycle

The Central Business District (CBD) Cycle is a chassis dynamometer testing procedure for heavy-duty vehicles (*SAE J1376*). The CBD cycle represents a "sawtooth" driving pattern, which includes 14 repetitions of a basic cycle composed of idle, acceleration, cruise, and deceleration modes. The following are characteristic parameters of the cycle:

- Duration: 560 s
- Average speed: 20.23 km/h
- Maximum speed: 32.18 km/h (20 mph)
- Driving distance: 3.22 km
- Average acceleration: 0.89 m/s^2
- Maximum acceleration: 1.79 m/s^2

Vehicle speed over the duration of the CBD cycle is shown in Figure A-1.

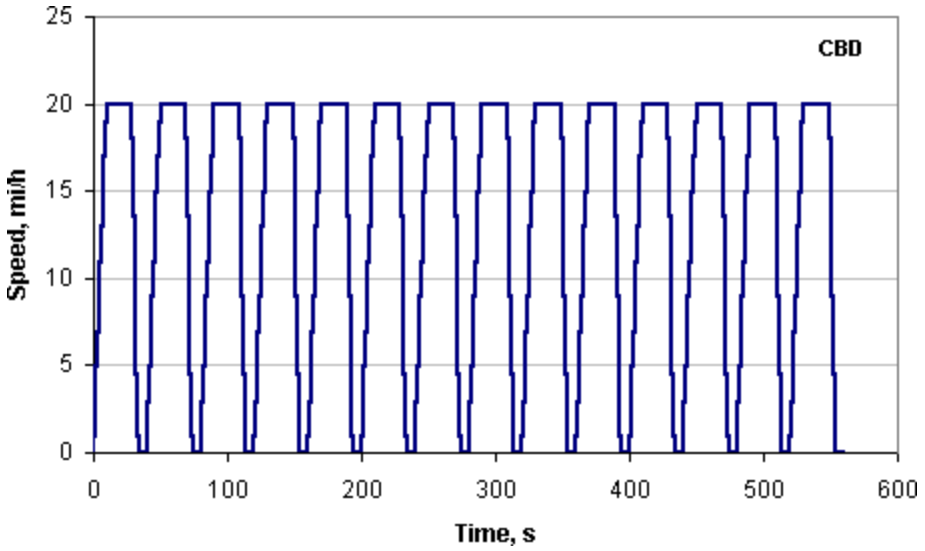


Figure B-6. CBD Driving Cycle

The standard CBD test cycle will be used for bus testing where three cycles will be combined for a triple CBD for a total sample time of 30 minutes. Performing the CBD cycle three times in one test allows for additional sample volumes to be collected for all batched type analysis (filters, DNPH, BETEX and N_2O). Preconditioning is defined as performing a previous triple CBD and a 20 minute soak to improve repeatability between hot repeats. Emissions analyses for gaseous emissions will also be collected over the triple CBD cycles. This cycle is shown in Figure A-2. The triple CBD cycle will be repeated in triplicate for repeatability metrics as described earlier.

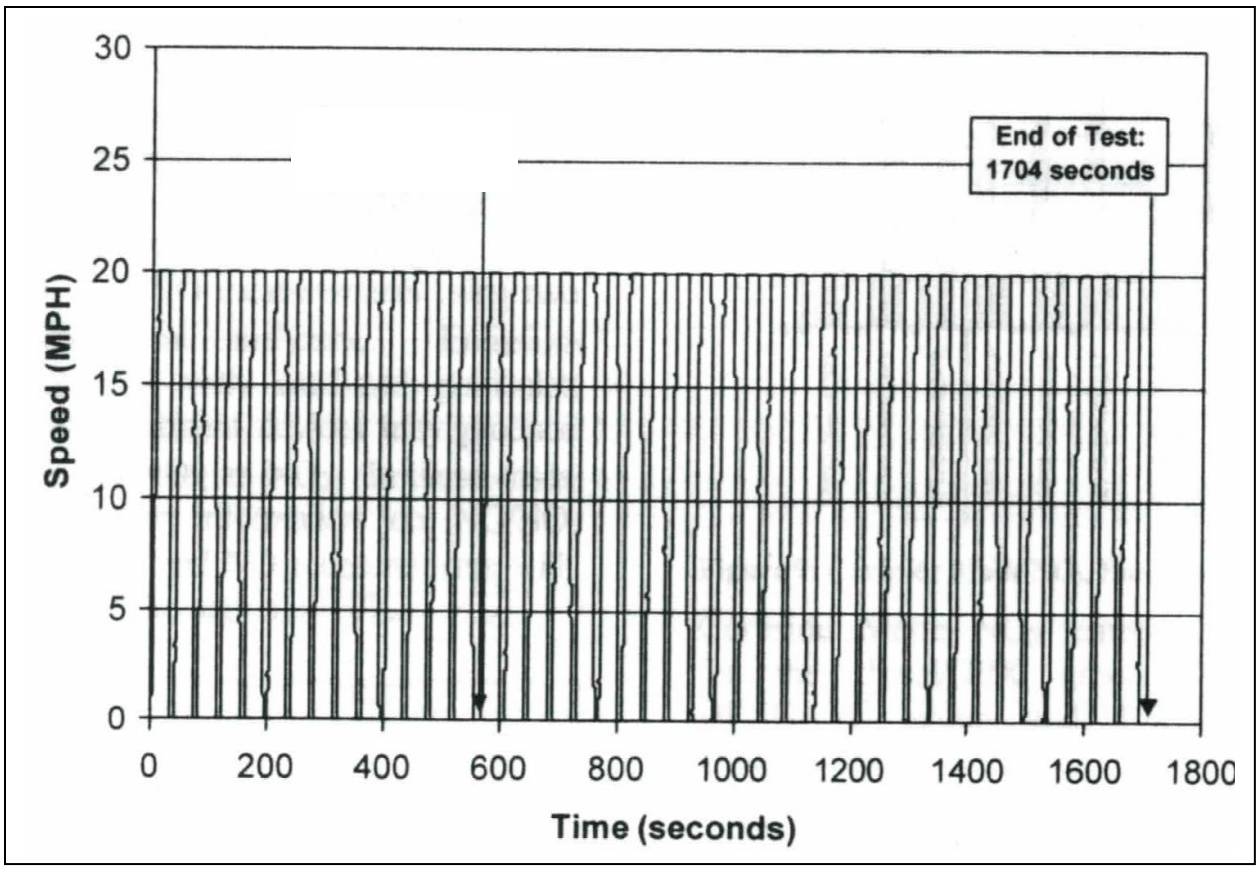


Figure B-7. Triple CBD Cycle

Appendix C. UCR Mobile Emission Laboratory

The approach used for measuring the emissions from a vehicle or an engine on a dynamometer is to connect UCR's heavy-duty mobile emission lab (MEL) to the total exhaust of the diesel engine. The details for sampling and measurement methods of mass emission rates from heavy-duty diesel engines are specified in Code of Federal Regulations (CFR): Protection of the Environment, Section 40, Part 1065. UCR's unique heavy-duty diesel mobile emissions laboratory (MEL) is designed and operated to meet those stringent specifications. MEL is a complex laboratory and a schematic of the major operating subsystems for MEL are shown in Figure C-1. The accuracy of MEL's measurements have been checked/verified against ARB's[10] and Southwest Research Institute's[11,12] heavy-duty diesel laboratories. MEL routinely measures Total Hydrocarbons (THC), Methane, Carbon Monoxide, Carbon Dioxide, Nitrogen Oxides, and Particulate Matter (PM) emissions from diesel engines. Design capabilities and details of MEL are described in Cocker et al[1,13]. Samples can be collected for more detailed analyses such as hydrocarbon speciation, carbonyl emissions, polynuclear aromatic hydrocarbons, etc.

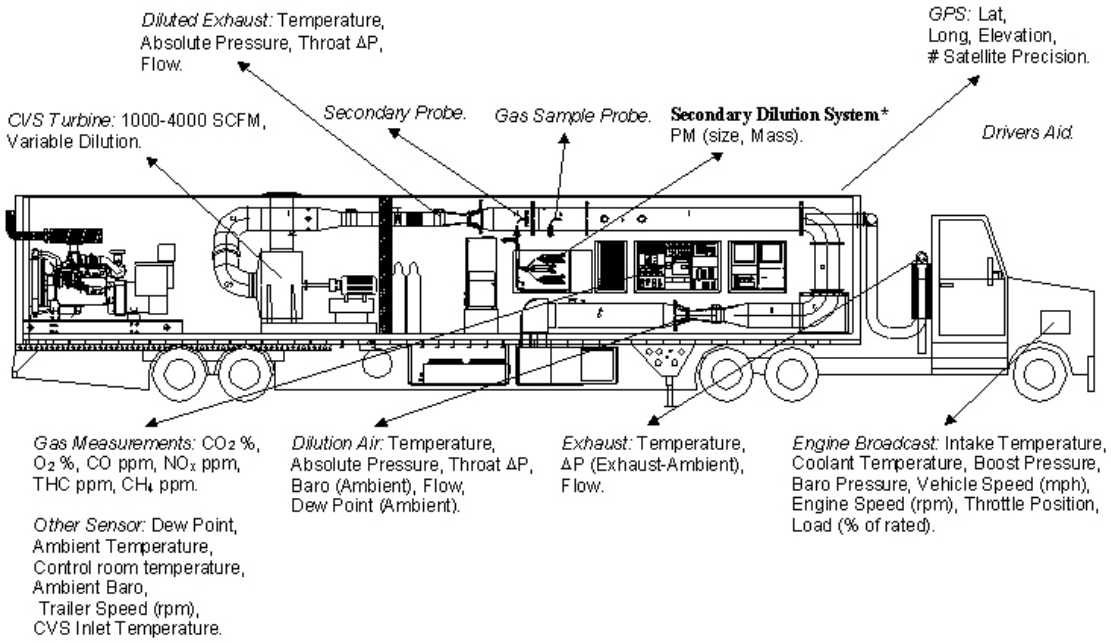


Figure C-1: Major Systems within UCR's Mobile Emission Lab (MEL)

[10] Cocker III, D. R., Shah, S. D., Johnson, K. C., Zhu, X., Miller, J. W., Norbeck, J. M., Development and Application of a Mobile Laboratory for Measuring Emissions from Diesel Engines. 2. Sampling for Toxics and Particulate Matter, Environ. Sci. Technol. **2004**, 38, 6809-6816

[11] Cocker III, D. R, Shah, S. D., Johnson, K. C., Miller, J. W., Norbeck, J. M., Measurement Allowance Project – On-Road Validation. Final Report to the Measurement Allowance steering Committee.

[12] Johnson, K.C., Durbin, T.D., Cocker, III, D.R., Miller, W.J., Bishnu, D.K., Maldonado, H., Moynahan, N., Ensfield, C., Laroo, C.A. (2009) On-road comparison of a portable emission measurement system with a mobile reference laboratory for a heavy-duty diesel vehicle, Atmospheric Environment 43 (2009) 2877–2883

[13] Cocker III, D. R, Shah, S. D., Johnson, K. C., Miller, J. W., Norbeck, J. M., *Development and Application of a Mobile Laboratory for Measuring Emissions From Diesel Engines I. Regulated Gaseous Emissions*, Environmental Science and Technology. **2004**, 38, 2182-2189

Appendix D. Heavy-Duty Chassis Dynamometer Laboratory

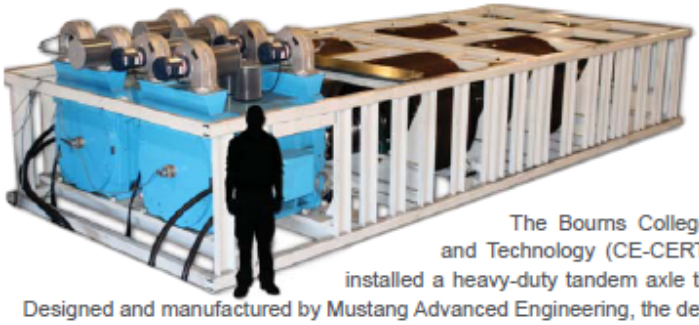
UCR's chassis dynamometer is an electric AC type design that can simulate inertia loads from 10,000 lb to 80,000 lb which covers a broad range of in-use medium and heavy duty vehicles, see Figure D-1. The design incorporates 48" rolls, axial loading to prevent tire slippage, 45,000 lb base inertial plus two large AC drive for achieving a range of inertias. The dyno has the capability to absorb accelerations and decelerations up to 6 mph/sec and handle wheel loads up to 600 horse power at 70 mph. This facility was also specially geared to handle slow speed vehicles such as yard trucks where 200 hp at 15 mph is common.

The chassis dynamometer was designed to accurately perform the new CARB 4 mode cycle, urban dynamometer driving schedule (UDDS), refuse drive schedule (WHM), bus cycles (CBD), as well as any speed vs time trace that do not exceed the acceleration and deceleration rates. The load measurement uses state of the art sensing and is accurate to 0.05% FS and has a response time of less than 100 ms which is necessary for repeatable and accurate transient testing. The speed accuracy of the rolls is ± 0.01 mph and has acceleration accuracy of ± 0.02 mph/sec which are both measured digitally and thus easy to maintain their accuracy. The torque transducer is calibrated as per CFR 1065 and is a standard method used for determining accurate and reliable wheel loads.

Figure D-1. UCR's heavy duty chassis eddy current transient dynamometer

Mustang Publication “Project Spotlights” March 2010

Mustang Advanced Engineering delivers a newly designed 48” Electric AC Heavy-Duty Truck Chassis Dynamometer with dual, direct-connected 300-hp AC motors to The University of California - Riverside, College of Engineering - Center for Environmental Research and Technology (CE-CERT).



The science of measuring emissions from mobile and other sources has evolved significantly over the past several years. The most important changes in the nature of emissions measurement science has been a shift to examining emissions from diesel sources and to understanding emissions under in-use driving conditions.

The Bourns College of Engineering – Center for Environmental Research and Technology (CE-CERT) at The University of California Riverside has recently installed a heavy-duty tandem axle truck chassis dynamometer in the facility’s research area. Designed and manufactured by Mustang Advanced Engineering, the development of this chassis dynamometer design was based on targeting vehicles in the medium to heavy-duty diesel vehicle range. Heavy-duty applications that can be tested at the facility include on-highway trucks, buses, waste haulers, yard tractors, and more - under test conditions representative of their specific in-use operations. The facility couples the new heavy-duty chassis dynamometer from Mustang Advanced Engineering with CE-CERT’s Mobile Emissions Laboratory (MEL), to perform precise vehicle simulation and in-operation emissions measurements.

The first research conducted on the new facility will be a comparison of federally mandated diesel fuel formulas versus the stricter formulation required in California. The program calls for 10 heavy-duty trucks to be tested with several different fuels.

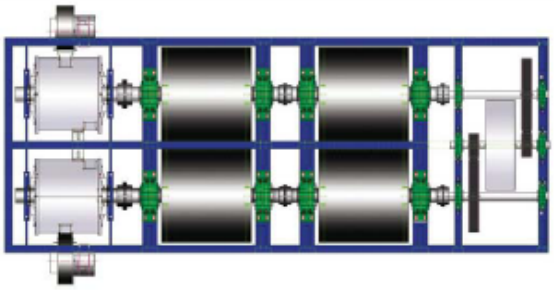
The new dynamometer will simulate on-road driving conditions for any big rig using its 48" precision rollers with dual, direct connected, 300 horsepower motors attached to each roll set. The dynamometer applies the appropriate loading to a vehicle to simulate factors such as the friction of the roadway and wind resistance that it would experience under typical driving conditions. An additional large inertia weight was incorporated into the dynamometer to increase the base mechanical inertia and enable the dynamometer to provide precise on-road simulation for a wide range of vehicle weights. The driver accelerates and decelerates according to a driving trace which specifies the speed and time over a wide range of vehicle simulation cycles. As the on-road driving conditions are being simulated on the dynamometer, emissions measurements will be collected with CE-CERT’s Mobile Emissions Laboratory (MEL).

“This adds new capabilities in California that are only available at a limited number of facilities around the country," said Tom Durbin, who with J. Wayne Miller, are the principle investigators for the project. At both the state and federal levels, scientific requirements for emissions testing are trending away from steady state engine testing in favor of transient conditions found in typical driving, Durbin explained. “This addition will significantly expand our laboratory and measurement capabilities and help us continue our role as leading experts in the field of emissions research," said CE-CERT Director Matthew Barth.

CE-CERT's new heavy-duty chassis dynamometer will allow the testing of a variety of heavy vehicles under loaded and transient in-use conditions with corresponding emissions measurements. The dynamometer configuration is capable of meeting the inertia simulation range requirements of 10,000 to 80,000 lb for each of the cycles listed below. This includes acceleration rates up-to 6 mph/sec, as found in the UDDS Section D Drive Schedule and deceleration rates of up to 7 mph/sec as required for the WHM Refuse Drive Schedule. The dynamometer can also provide a load in excess of 600 HP @ 70 mph. The dynamometer also has the ability to continuously handle 200 Hp @ 15 mph for applications such as yard tractors.

The Dynamometer system is designed to meet the Heavy Duty Drive Schedules for diesel trucks in the weight range of 10,000 to 80,000 lb with acceleration rates for the following cycles:

- CARB HHDDT Cruise Mode Drive Schedule
- UDDS (Urban Dynamometer Drive Schedule)
- CARB 50 mph HHDDT Cruise Cycle
- HHDDT Transient Mode Drive Schedule
- WHM Refuse Drive Schedule
- Bus cycles such as, the CBD, OC Bus cycle, NY bus cycle
- In-use cycles for applications such as yard tractors.



"As part of our strategic plan, Mustang has developed a cost effective series of diesel, petroleum and hybrid certification grade dynamometer systems to address the needs of the global emissions and R&D market. There is a clear and present demand for a full performance cost effective dynamometer systems that offer all of the capabilities and confidence of a certification system at a price point that makes it no longer cost-prohibitive for organization to perform critical emissions studies, hybrid system calibration development, performance evaluation and other cutting edge research technologies. Researchers are in need of dynamometer systems to develop the next generation technologies which mimic the capabilities of the certification requirements, but at a fraction of the cost of a true certification system. That is what we are developing with this series of dynamometers and universities are lining up for them", said Executive Vice President, Donald Ganzhorn.

Appendix E. Additional Test Data and Results

This appendix includes some additional results not presented in the main report, but can be used to support the assumptions and decisions made for the results presented. Following Tables E-1 through E-4 are fuel sample analysis reports.

Table E-1 Average emission factors for all cycles (g/bhp-hr)

	Duration	Engine		Ave Modal Emission Factor (g/bhp-hr)							PM (mg/bhp-hr)		NOx Emissions (mg/bhp-hr)				
Trace	sec	bhp	bhp-hr	THC	CH_4	NMHC	CO	N_2O	CO_2	NH_3	$PM_{2.5}$	Soot	M1	M2	M3	M4	M5
CS_UDDS	1081	75.3	22.6	0.85	0.53	0.32	4.28	0.062	546.8		1.7	0.05	51.5	48.0	43.5	70.7	34.1
CS_DPT1	3049	31.8	26.9	1.15	0.56	0.59	5.25	0.090	627.0	0.64	0.7	0.02	22.5	28.6	14.0	20.9	2.7
UDDS	2122	69.7	41.1	0.05	0.04	0.00	1.51	-	548.9	0.32	1.1	0.06	16.6	18.0	13.9	20.9	2.6
RTC	2889	51.3	41.2	0.09	0.08	0.01	2.75	-	577.0	0.44	0.7	0.00	6.4	7.2	2.2	28.6	4.9
DPT1	3049	27.7	23.5	0.37	0.26	0.10	2.35	-	649.8	0.66	0.9	0.07	4.2	0.0	2.0	9.2	1.4
DPT2	3365	36.5	34.1	0.20	0.16	0.05	2.01	0.027	597.0	0.47	0.5	0.10	16.6	17.2	12.6	21.6	4.5
DPT3	4228	74.9	87.9	0.49	0.33	0.17	1.34	0.024	549.3	0.37	1.0	0.01	18.4	20.8	14.1	23.7	4.1
CBD	560	76.2	11.8	0.16	0.11	0.05	2.73	0.034	576.1	0.94	0.9	0.04	-3.3	0.4	1.2	3.1	-0.2
Tunnel	1134	70.5	22.2	-0.01	0.00	-0.02	-0.07		0.5	0.03	0.4	0.04	0.2	2.6	1.0	1.4	0.6

Table E-2 Standard deviation of the emission factors for all cycles (g/bhp-hr)

	Duration	Engine		Stdev Modal Emission Factor (g/bhp-hr)							PM (mg/bhp-hr)		NOx Emissions (mg/bhp-hr)				
Trace	sec	bhp	bhp-hr	THC	CH_4	NMHC	CO	N_2O	CO_2	NH_3	$PM_{2.5}$	Soot	M1	M1	M1	M1	M1
CS_UDDS	0.0	0.2	0.1	0.07	0.04	0.03	0.39	-	14.1		0.9	0.01	26.9	32.3	11.7	17.5	29.2
CS_DPT1	0.0	0.1	0.1	0.52	0.08	0.44	0.71	-	2.9	0.09	0.4	0.07	24.4	19.2	8.7	4.7	0.9
UDDS	0.0	1.1	0.6	0.01	0.01	0.01	0.13	-	9.1	0.01	0.5	0.03	20.1	21.6	15.1	22.8	3.5
RTC	0.0	0.4	0.3	0.05	0.03	0.03	0.44	-	8.1	0.08	0.4	0.10	5.1	2.9	1.3	45.9	8.6
DPT1	0.0	1.1	0.9	0.20	0.13	0.07	0.39	-	8.3	0.12	0.2	0.02	4.1	7.8	1.9	2.8	0.4
DPT2	0.0	0.7	0.6	0.03	0.01	0.01	0.06	0.004	13.7	0.02	0.1	0.01	10.8	10.7	6.3	12.5	0.8
DPT3	0.0	0.3	0.4	0.03	0.02	0.01	0.23	0.003	7.7	0.10	0.9	0.01	4.7	6.5	3.6	6.5	1.9
CBD	0.0	0.8	0.1	0.02	0.02	0.01	0.75	0.012	25.9	0.24	0.5	0.01	6.3	1.0	1.3	1.5	0.3
Tunnel	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Table E-3 Average emission factors for all cycles (g/mi)

		Vehicle		Ave Modal Emission Factor (g/mi)							PM (mg/mi)		NOx Emissions (mg/mi)				
Trace	sec	bhp	mi	THC	CH_4	NMHC	CO	N_2O	CO_2	NH_3	$PM_{2.5}$	Soot	M1	M1	M1	M1	M1
CS_UDDS	1081	75.3	5.2	3.70	2.31	1.40	18.5	0.27	2367	-	7.3	0.21	223	208	189	306	164
CS_DPT1	3049	31.8	5.8	5.29	2.58	2.74	24.3	0.41	2895	2.98	3.1	0.10	104	132	65	96	13
UDDS	2122	69.7	11.2	0.17	0.16	0.01	5.5	-	2005	1.19	3.9	0.20	61	66	51	77	10
RTC	2889	51.3	4.4	0.82	0.74	0.09	25.4	-	5348	4.09	6.5	0.02	61	67	20	274	47
DPT1	3049	27.7	5.9	1.45	1.04	0.41	9.4	-	2589	2.64	3.5	0.29	17	-1	8	37	6
DPT2	3365	36.5	9.1	0.77	0.58	0.19	7.5	0.10	2236	1.77	1.7	0.39	63	65	48	81	17
DPT3	4228	74.9	28.1	1.54	1.02	0.53	4.2	0.07	1718	1.16	3.2	0.05	58	65	44	74	13
CBD	560	76.2	2.1	0.89	0.64	0.25	15.3	0.19	3226	5.27	5.3	0.23	-19	2	7	17	0
Tunnel	1134	70.5	6.0	-0.07	0.02	-0.08	-0.3	-	1.9	0.10	1.6	0.14	1	10	4	5	2

Table E-4 Standard deviation of the emission factors for all cycles (g/mi)

		Vehicle		Stdev Modal Emission Factor (g/mi)							PM (mg/mi)		NOx Emissions (mg/mi)				
Trace	sec	bhp	bhp-hr	THC	CH_4	NMHC	CO	N_2O	CO_2	NH_3	$PM_{2.5}$	Soot	M1	M1	M1	M1	M1
CS_UDDS	0	0.2	0.1	0.22	0.13	0.09	1.4	-	93	-	4.2	0.06	118	141	53	79	156
CS_DPT1	0	0.1	0.0	2.37	0.37	2.02	3.4	-	3	0.39	1.6	0.35	112	88	40	22	4
UDDS	0	1.1	0.1	0.05	0.03	0.02	0.4	-	8	0.06	2.0	0.12	75	81	56	85	13
RTC	0	0.4	0.1	0.44	0.26	0.23	3.4	-	146	0.78	3.2	0.96	49	30	12	444	83
DPT1	0	1.1	0.1	0.77	0.50	0.28	2.0	-	88	0.61	0.6	0.08	17	31	7	11	2
DPT2	0	0.7	0.1	0.11	0.06	0.05	0.3	0.01	16	0.11	0.3	0.04	41	41	24	47	3
DPT3	0	0.3	0.1	0.08	0.04	0.04	0.7	0.01	27	0.31	2.8	0.04	14	20	11	20	6
CBD	0	0.8	0.0	0.14	0.10	0.05	4.4	0.07	187	1.44	3.1	0.04	36	5	7	9	0
Tunnel	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Atmospheric Analysis & Consulting, Inc.

Laboratory Analysis Report
ASTM-D3588 (BTU and F-Factor)

CLIENT	**University of Riverside**	**SAMPLING DATE**	**11/20/2015**
PROJECT NO.	**160033**	**ANALYSIS DATE**	**1/11/2016**

	Client ID:	CNG 1501	
	AAC ID:	160033-86526	
	Component	Mole %	Weight %
FIXED GASES	H_2	0.00	0.00
	O_2	0.00	0.00
	N_2	0.63	1.04
	CO	0.00	0.00
	CO_2	0.35	0.90
	CH_4	94.65	89.77
	He	NM	NM
	Ar	NM	NM
HYDROCARBONS	C_2 (as Ethane)	3.8675	6.8752
	C_3 (as Propane)	0.4110	1.0715
	C_4 (as Butane)	0.0757	0.2602
	C_5 (as Pentane)	0.0112	0.0480
	C_6 (as Hexane)	0.0025	0.0126
	C_{6+} (as Hexane)	0.0032	0.0163
TRS	TRS as H2S	NM	NM
H2O	Moisture content	NM	NM

All results have been normalized to 100% on a dry weight basis.

Fuel Gas Specifications				
Atomic Breakdown - (scf/lb) / %			*HHV Btu/lb*	23286
Carbon (C)	74.1		*LHV Btu/lb*	20987
Hydrogen (H)	24.2		*HHV Btu/dscf*	1038
Oxygen (O)	0.7		*LHV Btu/dscf*	936
Nitrogen (N)	1.0		*F-Factor*	8645
Helium (He)	0.00		*Relative Denisty*	0.5841
Argon (Ar)	0.00		*C2-C6+ Weight %*	8.2837
Sulfur (S)	0.00		*MW lb/lb-mole*	16.915
Motor Octane Number	131.32		*Methane Number*	94.17

Marcus Hueppe
Laboratory Director

1534 Eastman Ave., Ste. A • Ventura, • CA 93003 www.aaclab.com • (805) 650-1642 • FAX (805) 650-1644

Atmospheric Analysis & Consulting, Inc.

Quality Control/Quality Assurance Report

Date Analyzed : 01/11/2016
Analyst : DJ
Units : %

Instrument ID : TCD#1
Calb Date : 01/06/2016
Reporting Limit : 0.1%

I - Opening Continuing Calibration Verification - ASTM D-1945/1946

AAC ID	Analyte	H_2	O_2	N_2	CO_2	CH_4	CO
CCV	Spike Conc	9.5	10.0	20.4	10.2	10.1	10.2
	Result	9.4	10.4	20.6	10.4	10.0	10.1
	% Rec *	98.8	103.3	100.8	101.6	99.4	98.8

II - Method Blank - ASTM D-1945/1946

AAC ID	Analyte	H_2	O_2	N_2	CO_2	CH_4	CO
MB	Concentration	ND	ND	ND	ND	ND	ND

III - Laboratory Control Spike & Duplicate - ASTM D-1945/1946

AAC ID	Analyte	H_2	O_2	N_2	CO_2	CH_4	CO
Lab Control Standards	Sample Conc	0.0	0.0	0.0	0.0	0.0	0.0
	Spike Conc	9.5	10.0	20.4	10.2	10.1	10.2
	LCS Result	8.8	9.9	19.9	10.3	9.8	9.9
	LCSD Result	9.2	10.4	21.0	10.8	10.4	10.5
	LCS % Rec *	92.7	98.2	97.5	100.3	96.8	96.5
	LCSD % Rec *	96.9	104.1	102.9	106.1	103.4	102.5
	% RPD ***	4.5	5.8	5.4	5.6	6.7	6.0

IV - Sample & Sample Duplicate - ASTM D-1945/1946

AAC ID	Analyte	H_2	O_2	N_2	CO_2	CH_4	CO
151755-86178	Sample	0.0	12.2	44.2	0.0	0.0	0.0
	Sample Dup	0.0	12.2	44.1	0.0	0.0	0.0
	Mean	0.0	12.2	44.1	0.0	0.0	0.0
	% RPD ***	0.0	0.2	0.3	0.0	0.0	0.0

V - Matrix Spike & Duplicate - ASTM D-1945/1946

AAC ID	Analyte	H_2	N_2	CO_2	CH_4	CO
151755-86178	Sample Conc	0.0	22.1	0.0	0.0	0.0
	Spike Conc	9.5	9.9	10.2	10.1	10.2
	MS Result	9.3	33.1	10.7	10.1	10.2
	MSD Result	8.6	33.0	10.3	9.6	9.7
	MS % Rec **	98.1	110.9	104.2	100.4	99.9
	MSD % Rec **	90.7	109.4	101.1	95.2	94.9
	% RPD ***	7.9	1.3	3.0	5.3	5.0

VI - Closing Continuing Calibration Verification - ASTM D-1945/1946

AAC ID	Analyte	H_2	O_2	N_2	CO_2	CH_4	CO
CCV	Spike Conc	9.5	10.0	20.4	10.2	10.1	10.2
	Result	9.5	10.8	21.5	10.9	10.4	10.5
	% Rec *	100.5	107.7	104.9	106.3	103.3	102.4

** Must be 85-115%*
*** Must be 75-125%*
**** Must be < 25%*
ND = Not Detected
<RL = less than Reporting Limit

Marcus Hueppe
Laboratory Director

Atmospheric Analysis & Consulting, Inc.

Quality Control/Quality Assurance Report

Date Analyzed : 01/11/2015
Analyst : DJ
Units : ppmv

Instrument ID : FID #3
Calb Date : 01/06/16
Reporting Limit : 0.5 ppmv

I - Opening Continuing Calibration Verification - ASTM D-1945/1946

AAC ID	Analyte	Methane	Ethane	Propane	Butane	Pentane	Hexane
CCV	Spike Conc	102.0	98.6	95.2	100.8	99.6	98.8
	Result	105.4	103.9	100.1	105.8	104.5	104.7
	% Rec *	103.3	105.3	105.1	104.9	104.9	106.0

II - Method Blank - ASTM D-1945/1946

AAC ID	Analyte	Methane	Ethane	Propane	Butane	Pentane	Hexane
MB	Concentration	ND	ND	ND	ND	ND	ND

III - Laboratory Control Spike & Duplicate - ASTM D-1945/1946

AAC ID	Analyte	Methane	Ethane	Propane	Butane	Pentane	Hexane
Lab Control Standards	Sample Conc	0.0	0.0	0.0	0.0	0.0	0.0
	Spike Conc	102.0	98.6	95.2	100.8	99.6	98.8
	LCS Result	101.2	99.8	96.9	102.3	101.8	102.8
	LCSD Result	102.4	101.4	98.2	103.9	104.2	108.0
	LCS % Rec *	99.2	101.2	101.8	101.5	102.2	104.1
	LCSD % Rec *	100.4	102.9	103.2	103.1	104.6	109.4
	% RPD ***	1.2	1.6	1.3	1.6	2.3	5.0

IV - Sample & Sample Duplicate - ASTM D-1945/1946

AAC ID	Analyte	Methane	Ethane	Propane	Butane	Pentane	Hexane
151755-86178	Sample	2.6	0.0	0.0	0.0	0.0	0.0
	Sample Dup	2.6	0.0	0.0	0.0	0.0	0.0
	Mean	2.6	0.0	0.0	0.0	0.0	0.0
	% RPD ***	0.8	0.0	0.0	0.0	0.0	0.0

V - Matrix Spike & Duplicate - ASTM D-1945/1946

AAC ID	Analyte	Methane	Ethane	Propane	Butane	Pentane	Hexane
151755-86178	Sample Conc	1.3	0.0	0.0	0.0	0.0	0.0
	Spike Conc	51.0	49.3	47.6	50.4	49.8	49.4
	MS Result	53.4	50.2	49.2	51.9	51.6	52.4
	MSD Result	53.1	50.9	49.3	52.2	55.1	53.3
	MS % Rec **	102.2	101.9	103.5	103.0	103.6	106.1
	MSD % Rec **	101.5	103.2	103.6	103.7	110.7	107.8
	% RPD ***	0.7	1.3	0.2	0.6	6.6	1.6

VI - Closing Continuing Calibration Verification - ASTM D-1945/1946

AAC ID	Analyte	Methane	Ethane	Propane	Butane	Pentane	Hexane
CCV	Spike Conc	102.0	98.6	95.2	100.8	99.6	98.8
	Result	101.3	99.5	96.4	101.3	99.3	98.4
	% Rec *	99.3	100.9	101.2	100.5	99.7	99.6

** Must be 85-115%*

*** Must be 75-125%*

**** Must be < 25%*

ND = Not Detected

<RL = less than Reporting Limit

Marcus Hueppe
Laboratory Director

Appendix F. Engine certification data, labels, and upgrades

This appendix includes the engine executive order Figure F-1 as listed on the ARB website for the family number listed on the engine name plate see Figure F-2 and F-3, Family number ECEXH0540LBH. The ISL G NZ certification is provided in the recently released documents as presented in Figure F-4, 5, and 6.

MODEL YEAR	ENGINE FAMILY	ENGINE SIZES (L)	FUEL TYPE [1]	STANDARDS & TEST PROCEDURE	INTENDED SERVICE CLASS [2]	ECS & SPECIAL FEATURES [3]	DIAGNOSTIC [6]
2014	ECEXH0540LBH	8.9	CNG/LNG	Diesel	HHDD	TBI, TC, CAC, ECM, EGR, TWC, HO2S	EMD

PRIMARY ENGINE'S IDLE EMISSIONS CONTROL [5]	ADDITIONAL IDLE EMISSIONS CONTROL [5]
EXEMPT	N/A

ENGINE (L)	ENGINE MODELS / CODES (rated power, in hp)
8.9	See attachment for engine models and ratings

* =not applicable; **GVWR**=gross vehicle weight rating; **13 CCR xyz**=Title 13, California Code of Regulations, Section xyz; **40 CFR 86.abc**=Title 40, Code of Federal Regulations, Section 86.abc; **L**=liter; **hp**=horsepower; **kw**=kilowatt; **hr**=hour;

[1] **CNG/LNG**=compressed/liquefied natural gas; **LPG**=liquefied petroleum gas; **E85**=85% ethanol fuel; **MF**=multi fuel a.k.a. **BF**=bi fuel; **DF**=dual fuel; **FF**=flexible fuel;

[2] **L/M/H HDD**=light/medium/heavy heavy-duty diesel; **UB**=urban bus; **HDO**=heavy duty Otto;

[3] **ECS**=emission control system; **TWC/OC**=three-way/oxidizing catalyst; **NAC**=NOx adsorption catalyst; **SCR-U / SCR-N**=selective catalytic reduction – urea / – ammonia; **WU (prefix)** =warm-up catalyst; **DPF**=diesel particulate filter; **PTOX**=periodic trap oxidizer; **HO2S/O2S**=heated/oxygen sensor; **HAFS/AFS**=heated/air-fuel-ratio sensor (a.k.a., universal or linear oxygen sensor); **TBI**=throttle body fuel injection; **SFI/MFI**=sequential/multi port fuel injection; **DGI**=direct gasoline injection; **GCARB**=gaseous carburetor; **IDI/DDI**=indirect/direct diesel injection; **TC/SC**=turbo/super charger; **CAC**=charge air cooler; **EGR / EGR-C**=exhaust gas recirculation / cooled EGR; **PAIR/AIR**=pulsed/secondary air injection; **SPL**=smoke puff limiter; **ECM/PCM**=engine/powertrain control module; **EM**=engine modification; **2 (prefix)**=parallel; **(2) (suffix)**=in series;

[5] **ESS**=engine shutdown system (per 13 CCR 1956.8(a)(6)(A)(1); **30g**=30 g/hr NOx (per 13 CCR 1956.8(a)(6)(C); **APS** =internal combustion auxiliary power system; **ALT**=alternative method (per 13 CCR 1956.8(a)(6)(D); **Exempt**=exempted per 13 CCR 1956.8(a)(6)(B) or for CNG/LNG fuel systems; **N/A**=not applicable (e.g., Otto engines and vehicles);

[6] **EMD**=engine manufacturer diagnostic system (13 CCR 1971); **OBD**=on-board diagnostic system (13 CCR 1971.1);

Following are: 1) the FTP exhaust emission standards, or family emission limit(s) as applicable, under 13 CCR 1956.8; 2) the EURO and NTE limits under the applicable California exhaust emission standards and test procedures for heavy-duty diesel engines and vehicles (Test Procedures); and 3) the corresponding certification levels, for this engine family. "Diesel" CO, SET and NTE certification compliance may have been demonstrated by the manufacturer as provided under the applicable Test Procedures in lieu of testing. (For flexible- and dual-fueled engines, the CERT values in brackets [ ] are those when tested on conventional test fuel. For multi-fueled engines, the STD and CERT values for default operation permitted in 13 CCR 1956.8 are in parentheses.).[4]

in g/bhp-hr	NMHC		NOx		NMHC+NOx		CO		PM		HCHO	
	FTP	SET	FTP	SET	FTP	SET	FTP	SET	FTP	SET	FTP	SET
STD	0.14	0.14	0.20	0.20	*	*	15.5	15.5	0.01	0.01	*	*
FEL	*	*			*	*	*	*	*	*	*	*
CERT	0.09	0.08	0.13	0.01	*	*	14.2	11.6	0.002	0.001	*	*
NTE	0.21		0.30		*		19.4		0.02		*	

[4] **g/bhp-hr**=grams per brake horsepower-hour; **FTP**=Federal Test Procedure; **SET**= Supplemental emissions testing; **NTE**=Not-to-Exceed; **STD**=standard or emission test cap; **FEL**=family emission limit; **CERT**=certification level; **NMHC/HC**=non-methane/hydrocarbon; **NOx**=oxides of nitrogen; **CO**=carbon monoxide; **PM**=particulate matter; **HCHO**=formaldehyde

Engine Family	1.Engine Code	2.Engine Model	3.BHP@RPM (SAE Gross)	4.Fuel Rate: mm/stroke @ peak HP (for diesel only)	5.Fuel Rate: (lbs/hr) @ peak HP (for diesels only)	6.Torque @ RPM (SEA Gross)	7.Fuel Rate: mm/stroke@peak torque	8.Fuel Rate: (lbs/hr)@peak torque	9.Emission Control Device Per SAE J1930
ECEXH0540LBH	3519;FR93287	ISL G 250	250@2200	N/A	N/A	730@1300	N/A	N/A	H02S, PCM, TWC,
ECEXH0540LBH	3519;FR93284	ISL G 260	260@2200	N/A	N/A	660@1300	N/A	N/A	H02S, PCM, TWC,
ECEXH0540LBH	3519;FR93282	ISL G 280	280@2200	N/A	N/A	900@1300	N/A	N/A	H02S, PCM, TWC,
ECEXH0540LBH	3519;FR93279	ISL G 300	300@2100	N/A	N/A	860@1300	N/A	N/A	H02S, PCM, TWC,
ECEXH0540LBH	3519;FR93276	ISL G 320	320@2100	N/A	N/A	1000@1300	N/A	N/A	H02S, PCM, TWC,
ECEXH0540LBH	3519;FR94391	ISL G 250	250@2200	N/A	N/A	730@1300	N/A	N/A	H02S, PCM, TWC,
ECEXH0540LBH	3519;FR94388	ISL G 260	260@2200	N/A	N/A	660@1300	N/A	N/A	H02S, PCM, TWC,
ECEXH0540LBH	3519;FR94386	ISL G 280	280@2200	N/A	N/A	900@1300	N/A	N/A	H02S, PCM, TWC,
ECEXH0540LBH	3519;FR94383	ISL G 300	300@2100	N/A	N/A	860@1300	N/A	N/A	H02S, PCM, TWC,
ECEXH0540LBH	3519;FR94380	ISL G 320	320@2100	N/A	N/A	1000@1300	N/A	N/A	H02S, PCM, TWC,

Figure F-1 Engine certification order for the ISL G (not ISL G NZ) NG engine (ARB source)

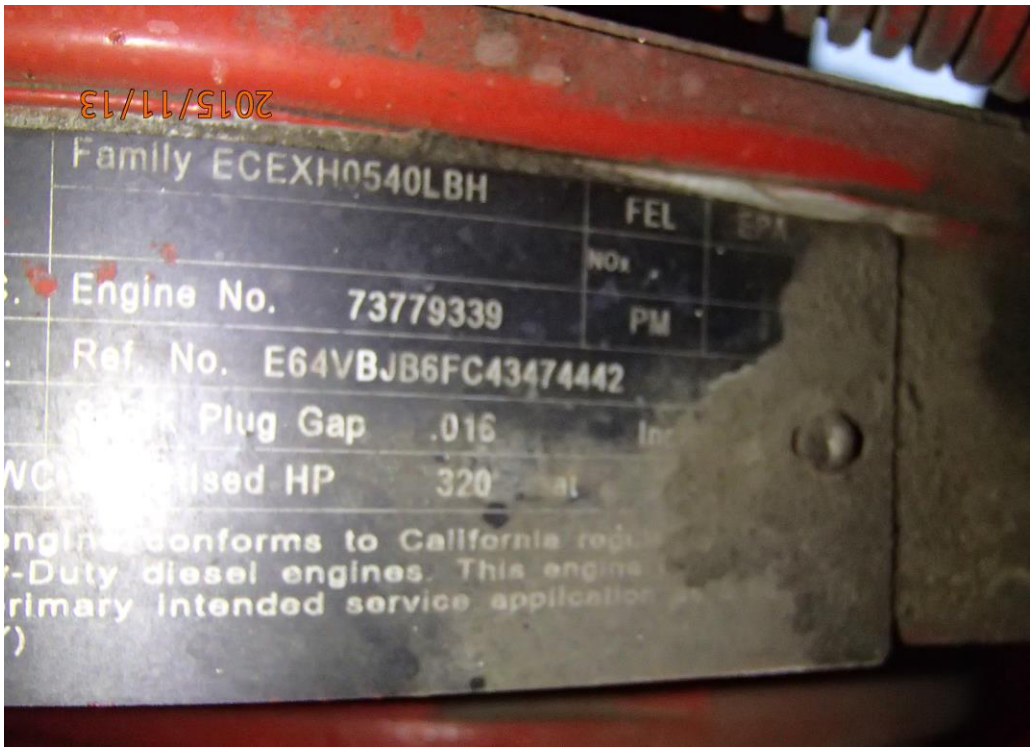


Figure F-2 Engine label for the ISL G NZ 320 NG engine

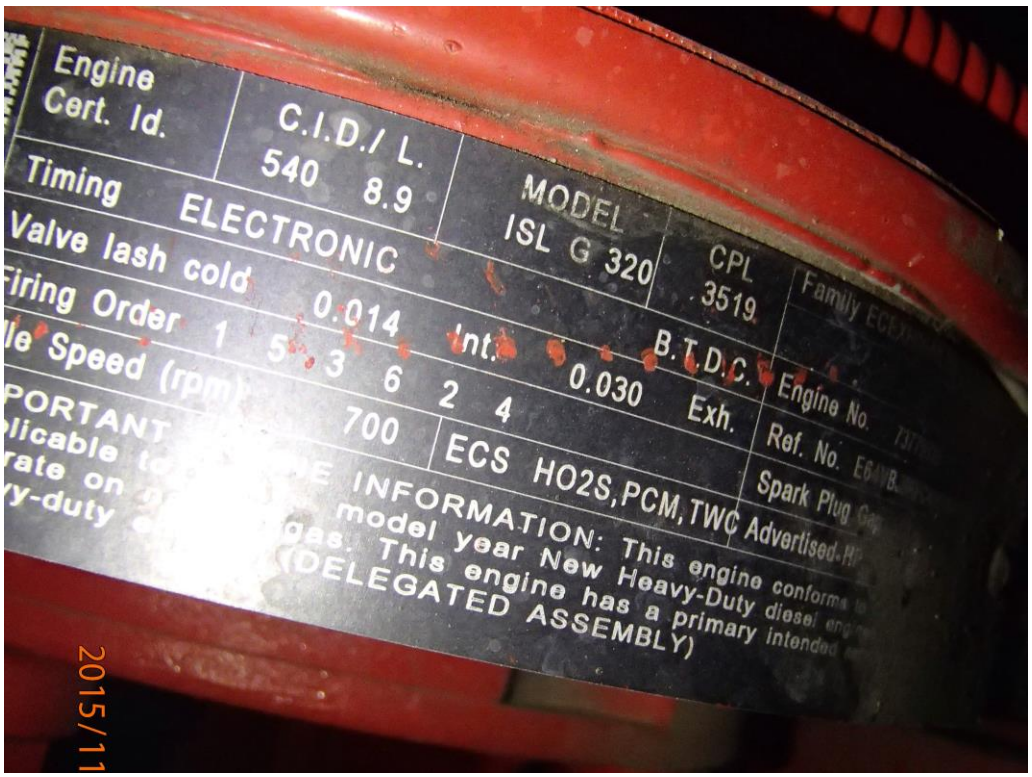


Figure F-3 Engine label for the ISL G NZ 320 NG engine

ARB

ENGINE MODEL	ADVERTISED HP(KW) @ RPM	PEAK TORQUE LB-FT @ RPM	GOVERNED SPEED
ISL G NZ 320	320 (239) @ 2000	1000 (1356) @ 1300	2200 RPM
ISL G NZ 300	300 (224) @ 2100	860 (1166) @ 1300	2200 RPM
ISL G NZ 280	280 (209) @ 2000	900 (1220) @ 1300	2200 RPM
ISL G NZ 260	260 (194) @ 2200	660 (895) @ 1300	2200 RPM
ISL G NZ 250	250 (186) @ 2200	730 (990) @ 1300	2200 RPM

California Environmental Protection Agency

CUMMINS INC. | EXECUTIVE ORDER A-021-0629 Now On-Road Heavy-Duty Engines Page 1 of 2 Pages

rces Board by Health and Safety Code Division 26, Part 5, Chapter 2; signed by Health and Safety Code Sections 39515 and 39516 and

l emission control systems produced by the manufacturer are certified cles with a manufacturer's GVWR over 14,000 pounds. Production t as those for which certification is granted.

TYPE	STANDARDS & TEST PROCEDURE	INTENDED SERVICE CLASS	ECS & SPECIAL FEATURES	DIAGNOSTIC
NG	Diesel	UB	TBI, TC, CAC, ECM, EGR, TWC, HO2S	EMD+

ADDITIONAL IDLE EMISSIONS CONTROL

N/A

MDH/F MODELS / CODES (rated power, in hp)

350 FRS8354 (280), ISL G 320 / 4836 FRS8351 (300), ISL G 320 / 4836 FRS8348 (320)

Following are: 1) the FTP exhaust emission standards, or family emission limit(s) as applicable, under 13 CCR 1956.8

in g/bhp-hr	NMHC		NOx		NMHC+NOx		CO		PM		HCHO	
	FTP	SET	FTP	SET	FTP	SET	FTP	SET	FTP	SET	FTP	SET
STD	0.14	0.14	0.02	0.02	*	*	15.5	15.5	0.01	0.01	*	*
CERT	0.01	0.000	0.01	0.004	*	*	1.5	0.3	0.001	0.000	*	*
NTE	0.21		0.03		*		19.4		0.02		*	

[4] g/bhp-hr=grams per brake horsepower-hour; FTP=Federal Test Procedure; SET= Supplemental emissions testing; NTE=Not-to-Exceed; STD=standard or emission test cap; FEL=family emission limit; CERT=certification level; NMHC/HC=non-methane/hydrocarbon; NOx=oxides of nitrogen; CO=carbon monoxide; PM=particulate matter; HCHO=formaldehyde

EPA

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF TRANSPORTATION AND AIR QUALITY
WASHINGTON, DC 20460

CERTIFICATE OF CONFORMITY
2016 MODEL YEAR

Manufacturer: CUMMINS INC

Engine Family: GCEXH0540LBH
Certificate Number: CEX-ONHWY-16-01
Intended Service Class: URBAN BUS
Fuel Type: NATURAL GAS
FELs: G/BHP: NMHC +NOx: N/A; NOx: N/A; PM: N/A

Greenhouse Gas Info.
Primary Intended Service Class: VOCATIONAL
Primary Test Configuration FTP (if applicable)
CO_2 FCL value (g/hp-hr) 476
CO_2 FEL value (g/hp-hr) 490
N_2O FEL value (g/hp-hr) 0.10
CH_4 FEL value (g/hp-hr) 0.65
Primary Test Configuration Exempted-(model(if applicable)
CO_2 FCL value (g/hp-hr)
CO_2 FEL value (g/hp-hr)

Effective Date: 9/28/2015

Byron J. Bunker, Director
Compliance Division
Office of Transportation and Air Quality

R Part 86, and subject to the terms and conditions with respect to the test engines which represent the and conditions prescribed in those provisions.

hich conform in all material respects to the design quired by 40 CFR Part 86 and which are produced during 40 CFR Part 86.

ions described in 40 CFR 86.096-7, 86.606, and 86.1006 ments of such a warrant or court order may lead to rt 86. It is also a term of this certificate that this easons specified in 40 CFR Part 86.

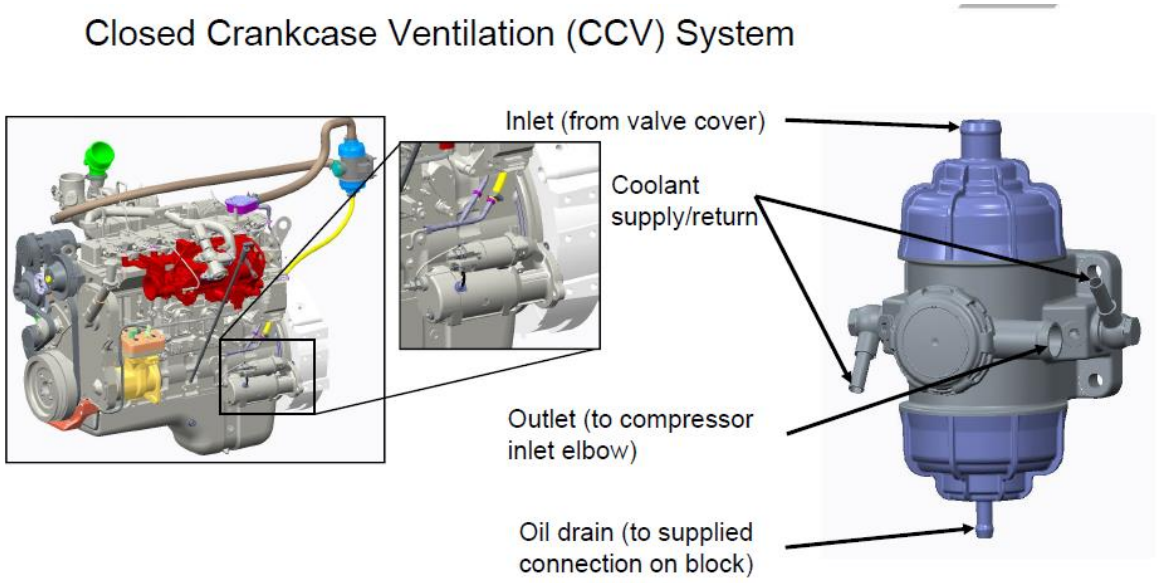
delivered for introduction into commerce in the U.S. prior to the effective date of the certificate.

In g/bhp-hr	EPA CERTIFICATE OF CONFORMITY * CO_2 FTP	CO_2 SET	PRIMARY INTENDED SERVICE CLASS VOCATIONAL CH_4	N_2O
STD	555	*	0.10	0.10
FCL	476	*	*	*
FEL	490	*	0.65	*
CERT	465	*	0.56	0.02

[4] g/bhp-hr=grams per brake horsepower-hour; FTP=Federal Test Procedure; SET=Supplemental emissions testing; STD = standard or emission test cap; FEL=family emission limit; FCL=family certification level; CERT=certification level; CO_2=carbon dioxide; CH_4=methane; N_2O=nitrous oxide; VOCATIONAL=vocational engine; TRACTOR=tractor engine

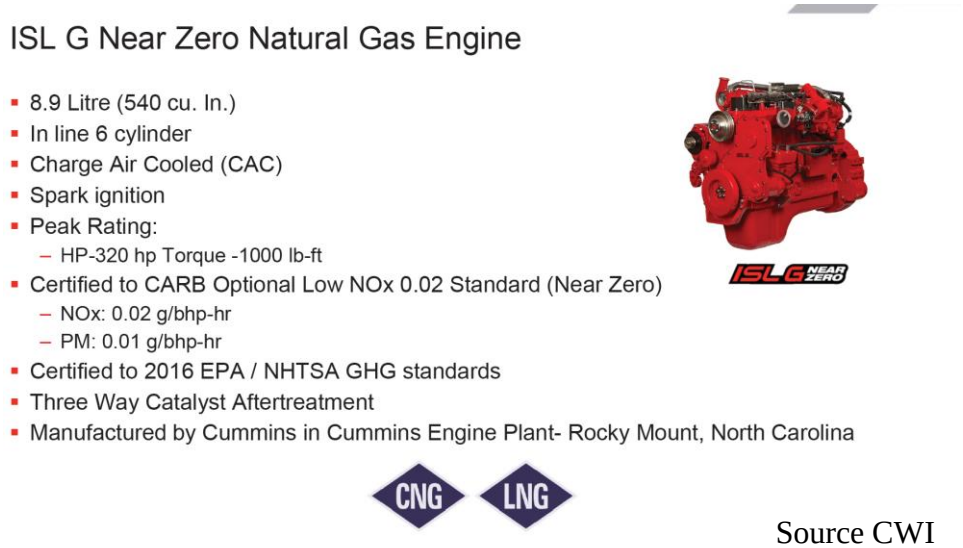
Source CWI
Fam# GCEXH0540LBH

Figure F-4 2016 ISL G NZ certification executive order with engine ratings (ARB and EPA)



Source CWI

Figure F-5 Cummins methane blow by capture improvement



Source CWI

Figure F-6 Cummins specifications for the ISL G NZ

Changes from ISL G EPA 2013

- **Certification**
  - new Agency Approval (AP) option
- **ECM Calibration**
  - 0.02g NOx calibration
  - Delegated Assembly protected via catalyst / ECM connection
- **Three Way Catalyst (TWC)**
  - Same as ISX12 G and ISL G Euro VI
  - Has extra mid bed temperature sensor that must be added to OEM harness
- **New Closed Crankcase Ventilation (CCV) System**
  - Remote mount CCV filter – to be installed by OEMs
  - Similar to ISL G Euro VI, but with coolant heating (same as ISB6.7 G)
  - Requires OEM installed air/oil and coolant plumbing to and from the engine
- **Crankcase Pressure Sensor**
  - New for diagnostic and OBD purposes

Source CWI

Figure F-7 ISL G NZ emission enhancements

Appendix G. Coastdown methods

Road load coefficients are important where at 65 mph the aerodynamic term accounts for 53% of the resisting force, rolling resistance 32%, driveline losses 6% and auxiliary loads at 9%. These load fractions vary with speed and the square of the speed where a properly configured dynamometer is needed to simulate the loads from 0 to 70 mph. The method for determining coastdown coefficients was published and evaluated as part of a study submitted to the South Coast Air Quality Management District[14]. Typical coastdown procedures assume that vehicle loading force is a function of vehicle speed, drag coefficient, frontal area and tire rolling resistance coefficient and takes the form of equation 1:

$$M\frac{dV}{dt} = \frac{1}{2}\rho A C_D V^2 + \mu M g cos(\theta) + Mgsin(\theta) \qquad \text{(Equation 1)}$$

Where:
M = mass of vehicle in lb (tractor + payload + trailer+ 125lb/tire)
ρ = density of air in kg/m^3.
A = frontal area of vehicle in square feet, see Figure G-1 below
C_D = aerodynamic drag coefficient (unit less).
V = speed vehicle is traveling in mph.
μ = tire rolling resistance coefficient (unit less).
g = acceleration due to gravity = 32.1740 ft/sec^2.
θ = angle of inclination of the road grade in degrees (this becomes zero).

Assuming that the vehicle loading is characteristic of this equation, speed-time data collected during the coastdown test can be used with static measurements (ZET/NZET mass, air density, frontal area, and grade) to solve for drag coefficient (C_d) and tire rolling resistance coefficient (μ). The frontal area is measured based on the method described in Figure G-1 below. However, experience performing in-use coastdowns is complex and requires grades of less than 0.5% over miles of distance, average wind speeds < 10 mph ± 2.3 mph gusts and < 5 mph cross wind[15]. As such, performing in-use coastdowns in CA where grade and wind are unpredictable are unreliable where a calculated approach is more consistent and appropriate. Additionally vehicles equipped with automatic transmissions have shown that on-road loading is also affected by the characteristics of the vehicle transmission, especially when reverse pumping losses at low speed begin to dominate.

UCR's and others recommend a road load determination method that uses a characteristic coastdown equation, with a measured vehicle frontal area (per SAE J1263 measurement recommendations), a tire rolling resistance μ, and a coefficient of drag (Cd) as listed in Table G-1. If low rolling resistant tires are used then the fuel savings can be employed with a slightly improved coefficient as listed. Similarly if an aerodynamic tractor design is utilized (ie a certified SmartWay design) then a lower drag coefficient can be selected. Table G-1 lists the

[14] Draft Test Plan Re: SCAQMD RFP#P2011-6, "In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines", October 2011
[15] EPA Final rulemaking to establish greenhouse gas emissions standards and fuel efficiency standards for medium and heavy duty engines and vehicles, Office of Transportation and Air Quality, August 2011 (Page 3-7) and J1263 coast down procedure for fuel economy measurements

coefficients to use based on different ZET/NZET configurations. Once the coefficients are selected then they can be used in the above equation to calculate coastdown times to be used for calculating the A, B, C coefficients in Equation 2 for the dynamometer operation parameters. From these equations calculate the coastdown times from based on the coefficients in Table G-1 as shown in Table G-2 (65,000 lb, ustd, Cdstd and Table G-1). From Table G-2 one can plot the force (lb) vs average speed bin to get the ABC coefficients for the chassis dynamometer (see Figure G-2). These are the coefficients to enter into the chassis dynamometer then validate via the details of Appendix C. Repeat process until validation criteria is met. Typically one or two iterations is needed to meet the validation criteria.

Table G-1 Constants and parameters for Class 8 heavy duty trucks

Variable	Value	Description
θ	0	no grade in these tests
ρ	1.202	standard air density kg/m^3
μ_{std}	0.00710	standard tires
μ_{adv}	0.00696	low rolling resistant tires
C_{D_std}	0.750	for non-SmartWay tractor
C_{D_adv}	0.712	for SmartWay tractor
g	9.806	nominal value m/sec^2
M	Varies	mass: final test weight kg

[1] The tire rolling resistance, μ, for low rolling resistant tires shows a 1-2% savings (ref SmartWay). As such utilize 0.00686 fpr low rolling resistant tires. In this document the tractors may vary, but the trailers will be assumed similar. As such, if the tractor utilizes the certified SmartWay tractor type then coefficient of drag can be reduced by up to 10% (5% fuel savings) depending on the technology. As such in this guidance document utilize the Cd_adv for SmartWay tractors and Cd_std for non-SmartWay tractors. Additionally, for reference other vocations show higher Cd's, such as the C_D = 0.79 for buses and 0.80 for refuse trucks. Nominal value of gravity is used in this document where actual value can be found by following 40CFR 1065.630 or at http://www.ngs.noaa.gov

$$\frac{dV}{dt} = \frac{1}{2}\frac{\rho A C_D V^2}{M} + \mu g cos(\theta) + g sin(\theta) \qquad \text{(Equation 2)}$$

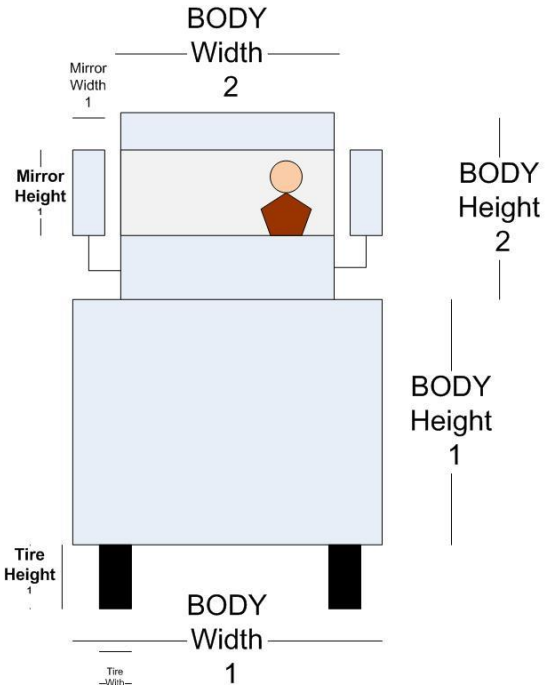


Figure G-1 Vehicle frontal area dimensions method

Using Equation 2 (solution for $\frac{dV}{dt}$ or deceleration), one can calculate the deceleration for each average speed bin (60, 50, … down to 20 mph), see Table G-2. From the deceleration time one can calculate the desired time which is the target for the coast down simulation on the chassis dynamometer. Using the final test weight (M), the total simulated force can be calculated using Equation 1 at each speed bin, see values Table G-2. Plot the simulated force (lb) on the y-axis vs truck speed (mph) on the x-axis. Using a best fit polynomial of order two, calculate the polynomial coefficients A (0^{th} order term), B (1^{st} order term), and C (2^{nd} order term), see Figure G-2. Enter these ABCs into your chassis dynamometer and verify the coast down times match your desired coast down times to within 5%.

The calculation approach is consistent and has proven very reliable for chassis testing heavy duty vehicle and has been used for years by UCR and others. For detailed evaluation of aerodynamic modifications and body styles, UCR recommends investing the time perform in-use coastdowns where sufficient program resources will be needed as per 40 CFR Part 1066, SAE J2263, and J1263.

Table G-2 Desired coastdown times for a Class 8 truck with standard components

Data Point	Avg Speed MPH	Calc Time sec	Decel MPH/Sec	Desired Decel ft/sec^2	Decel Gs	Force lb
65-55	60	25.67	0.38954	0.57	0.018	1154
55-45	50	31.44	0.31806	0.47	0.014	942
45-35	40	38.51	0.25965	0.38	0.012	769
35-25	30	46.68	0.21422	0.31	0.010	635
25-15	20	55.02	0.18177	0.27	0.008	539

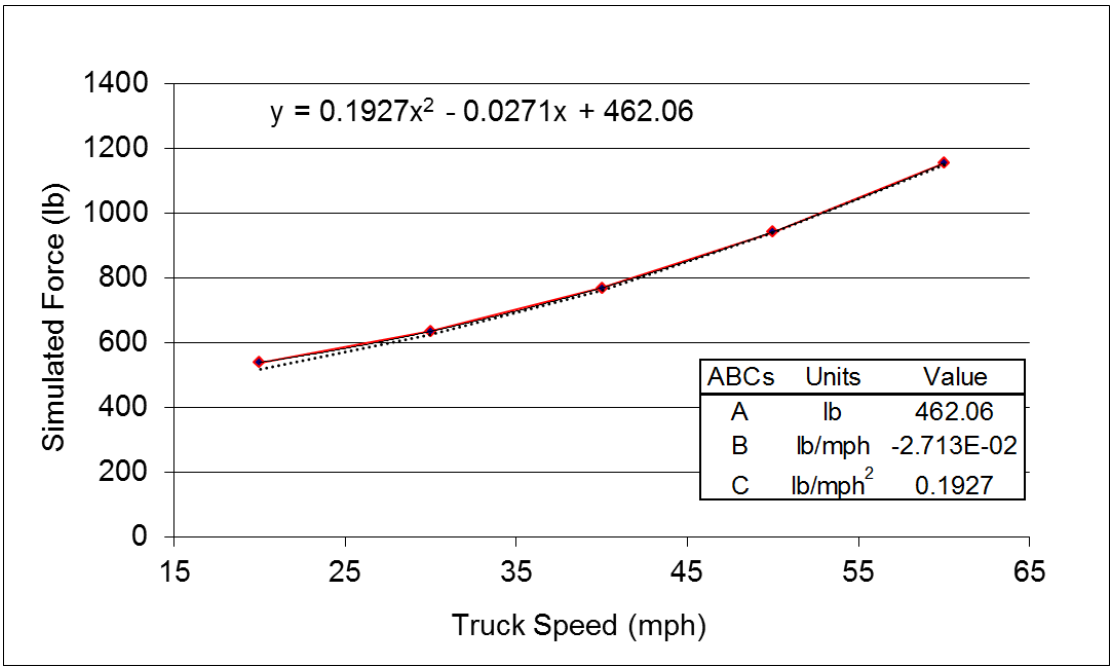


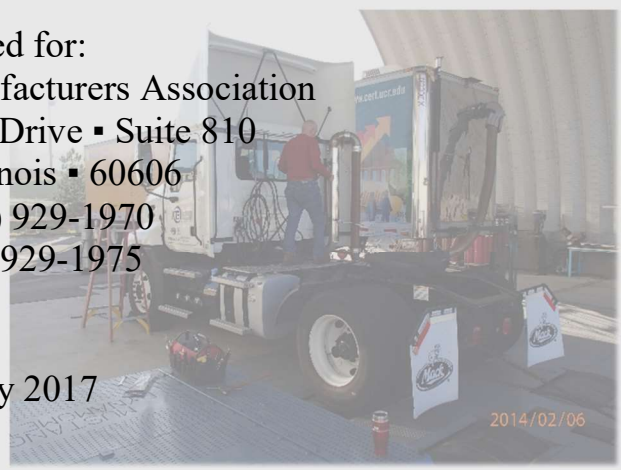
Figure G-2 Resulting ABCs based on Table G-2 results

EXHIBIT 9

Final Report

Heavy-Duty Chassis Dynamometer Test Program



Prepared for:
Truck & Engine Manufacturers Association
333 West Wacker Drive ▪ Suite 810
Chicago ▪ Illinois ▪ 60606
Main: (312) 929-1970
Fax: (312) 929-1975

February 2017

Submitted by:
Thomas D. Durbin
Kent C. Johnson
Georgios Karavalakis
Yu Jiang
Jiacheng Yang
University of California
CE-CERT
Riverside, CA 92521
951-781-5791
951-781-5790 (fax)

Acknowledgments

The authors thank the following organizations and individuals for their valuable contributions to this project.

We acknowledge funding from the Truck and Engine Manufacturers Association.

We acknowledge the participation of the California Air Resources Board (ARB) in this program, including involvement in the development of the test plan, the selection of the test vehicles, and their own chassis dynamometer testing of three of the five selected vehicles.

We acknowledge Mr. Don Pacocha, Mr. Eddie O'Neal, and Mr. Joe Valdez of the University of California, Riverside for their contributions in conducting the emissions testing for this program.

Table of Contents

Table of Tables

Table of Figures

Acronyms and Abbreviations

ARB .....Air Resources Board
bhp ……. brake horse power
bhp-hr …… brake horse power - hour
CAFEE .....West Virginia University Center for Alternative Fuels Engines and Emissions Laboratory
CAI.....California Analytical Instruments
CARB.....California Air Resources Board
CE-CERT.....College of Engineering-Center for Environmental Research and Technology (University of California, Riverside)
CFR.....Code of Federal Regulations
CH_4…….methane
CNG.....compressed natural gas
CO.....carbon monoxide
COV.....coefficient of variation
CO_2.....carbon dioxide
CPC.....condensation particle counter
CVS.....constant volume sampling
DOC.....diesel oxidation catalyst
Dp.....particle diameter
DPF.....diesel particle filter
ECM……engine control module
EEPS……...Engine Exhaust Particle Sizer
EGR……exhaust gas recirculation
EMA.....Truck and Engine Manufacturers Association
EMFAC.....EMission FACtors inventory model
EPA.....United States Environmental Protection Agency
FID.....flame ionization detector
g/mi.....grams per mile
HDDE.....heavy-duty diesel engine
HDV.....heavy-duty vehicle
lbs.....pounds
MEL.....CE-CERT's Mobile Emissions Laboratory
mpg.....miles per gallon
m/s^2.....meters per second squared
NDIR……..non-dispersive infrared detector
nm…….. nanometer
NMHC.....non-methane hydrocarbons
NO_x.....oxides of nitrogen
PM.....particulate matter
PN.....particle number
THC.....total hydrocarbons
SCAQMD.....South Coast Air Quality Management District
SCR.....Selective Catalytic Reduction

UCR……University of California, Riverside
ZMR.....zero mile emission rates

Executive Summary

Characterization of the in-use emissions from on-road heavy-duty trucks is an important element of developing accurate emissions inventory estimates. Current-technology diesel engines are equipped with aftertreatment systems designed to meet stringent emissions standards that were put in place in 2007 for particulate matter (PM) and fully phased-in for oxides of nitrogen (NO_x) in 2010. Data from real-world in-use emissions measurements of those modern diesel engines are scarce, however, and show some variation depending on the type of truck tested and the testing conditions. The goal of this work was to obtain data on Class 8 trucks equipped with the newest emission control strategies and operated over in-use cycles on a heavy-duty chassis dynamometer. A particular emphasis was on gathering data that can be used to improve estimates of "zero mile" emission rates (ZMRs) for 2010 and later model year heavy-duty engines/trucks. This information could be analyzed to augment the data being used in the development of emissions inventory models utilized in different levels of the regulatory process, and in particular the California Air Resources Board's (ARB's) EMFAC2014 model.

Five vehicles were tested in this program. The vehicles were all Class 8 heavy-duty diesel trucks equipped with the latest generation of emissions control technology, including a diesel particulate filter (DPF) for PM emissions control and a selective catalystic reduction (SCR) system for NOx emissions control. The vehicles tested ranged in model year from 2012 to 2015, with 4 of the 5 engines being 2014 or newer. The vehicle matrix included 5 engines from heavy-duty engine manufacturers representing the majority of trucks operating in California, with two engines being from the same manufacturer. The engines/vehicles were certified to a 0.20 g/bhp-hr or lower NOx certification limit, with the exception of one credit-using engine that was certified to a 0.35 g/bhp-hr NO_x standard. Each vehicle was tested on UCR's heavy-duty chassis dynamometer over the four phases of ARB's Heavy Heavy-Duty Diesel Truck (HHDDT) test cycle (i.e., idle, creep, transient, and cruise), the HHDDT-short or (HHDDT-S) cycle (which is a high-speed cruise schedule), and the Urban Dynamometer Driving Schedule (UDDS) (which is a cycle considered to be the chassis dynamometer equivalent of the Federal Test Procedure (FTP) engine dynamometer transient test). Three of the five trucks were retested at the ARB's Heavy-Duty Chassis Dynamometer Testing Facility in Los Angeles.[1]

The results of this study are summarized below. As the primary emphasis for this study is on the ZMRs for NOx emissions, NOx emissions are the main emphasis of the executive summary. It should be noted that other emissions, such as PM, total hydrocarbons (THC), and carbon monoxide (CO), were found to be very low under most of the testing conditions. Those emissions are described in greater detail in the main body of the report.

NOx emissions results are presented in Figures ES-1 and ES-2 on a g/mi and g/bhp-hr basis, respectively. The emissions on a g/bhp-hr basis were derived using emissions on a mass basis divided by the engine work. The engine work is obtained by integration of the engine torque and

[1] This report is based on data and analysis from the truck testing conducted at CE-CERT. It is important to note that additional testing was also carried out on a subset of the trucks at the ARB's heavy-duty chassis-dynamometer test facility as part of the agreed upon study effort. The goal was to include all of the data and analysis from both testing efforts in this report, however the ARB data have not been provided for analysis or reporting. The additional ARB data would provide additional insight to the overall project results. It is hoped that ARB will consider their data, in addition to what is reported here, as they improve the EMFAC model.

engine speed channel from the J1939 signal from the engine control module (ECM). The torque signal is estimated using engine speed and instantaneous fuel flow. Note that in both of the figures the emissions for the Creep cycle are divided by 5 to allow the emissions over all 5 cycles to be presented on the same graph. NOx emissions varied depending on the test cycle and the test truck. It should be noted that the NOx emissions for the manufacturer D truck appear to be an outlier with noticeably higher NOx emissions. Some additional testing was conducted on this vehicle subsequent to the main testing of this study after the vehicle's aftertreatment system was regenerated. Given anomalies for the manufacturer D truck, this truck is discussed separately from and in more detail than the results for the manufacturer A1, manufacturer A2, manufacturer B and manufacturer C engine-powered trucks below.

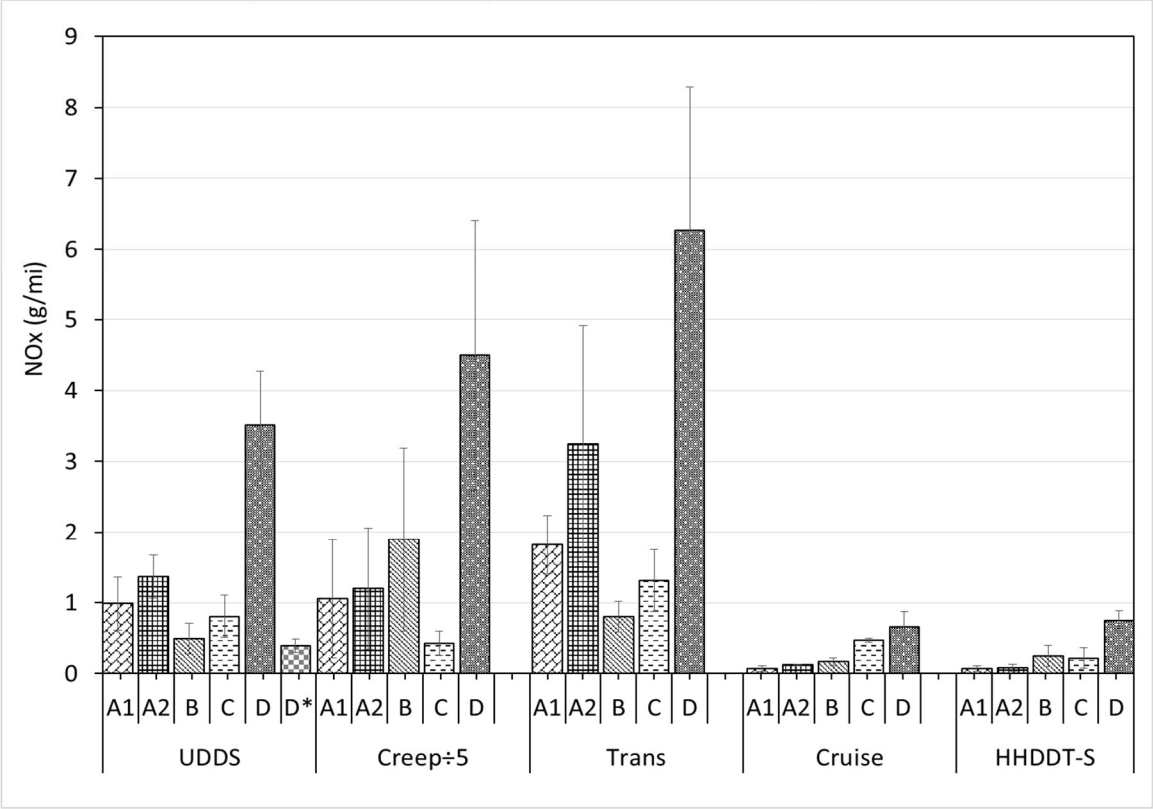
For the manufacturer A1, manufacturer A2, manufacturer B and manufacturer C trucks, NOx emissions over the UDDS cycle ranged from 0.495 to 1.363 g/mi (0.136 to 0.387 g/bhp-hr). On a bhp-hr basis, those emission levels are comparable to the level to which the engines were certified to on an engine dynamometer, with some trucks being below and other being above the 0.20/0.35 NOx g/bhp-hr level. NOx emissions for the ARB transient cycle were slightly higher than for the UDDS, with emission rates ranging from 0.803 to 3.252 g/mi (0.194 to 0.762 g/bhp-hr). The lowest emissions were found over the two cruise cycles. For the HHDDT-S, NOx emissions ranged from 0.067 to 0.249 g/mi (0.023 to 0.078 g/bhp-hr). For the Cruise cycle, NOx emissions ranged from 0.068 to 0.471 g/mi (0.030 to 0.225 g/bhp-hr). The highest NOx emissions were seen for the Creep cycle, which showed NOx emissions ranging from 2.131 to 9.468 g/mi (0.910 to 3.613 g/bhp-hr). The higher NOx emissions for the Creep cycle can be attributed to the fact that the cycle is comprised of short, low-speed accelerations between periods of idle that cover a very short distance (0.124 miles). Such stop-and-go type of driving tends to create high emissions when evaluated on a per mile or per unit of work done basis.

As discussed above, the manufacturer D truck seemed to be an outlier with noticeably higher NOx emissions relative to the other vehicles. In this study, on a g/mi basis, its NOx emissions were 3.519 over the UDDS, 22.505 over the Creep cycle, 6.270 over the Transient cycle, 0.656 over the Cruise cycle, and 0.751 for the HHDDT-S. The lowest NOx emissions for the manufacturer D engine were recorded over the Cruise and HHDDT-S cycles. On a g/bhp-hr basis, NOx emissions for the manufacturer D vehicle were 0.967 for the UDDS, 3.832 for the Creep cycle, 1.430 for the Transient cycle, 0.257 for the Cruise cycle, and 0.234 over the HHDDT-S.

Subsequent to the dynamometer testing conducted at UCR, the manufacturer D vehicle was more extensively evaluated by the manufacturer. The vehicle was initially outfitted with a PEMS, which confirmed the poor NOx conversion efficiency found in the UCR testing. Upon further investigation, it was determined that this specific vehicle had served its entire life as a dealer demonstrator, and as such rarely or never operated with a loaded trailer, and spent a considerable amount of time operating in an idle mode. This type of low temperature, high proportion idle operation is known to cause significant exposure of the aftertreatment system to unburned hydrocarbon in the exhaust stream. An examination of the logged electronic history revealed no OBD faults or other indications of failure or system malfunction. A clear anomaly, however, was that due to its unusual, unladen duty cycle, the engine had never undergone a regeneration event, despite being approximately 2.5 years in service (albeit with only 12,000 miles on the odometer). A series of conventional parked regenerations were performed. After further operation, there was a significant recovery of the aftertreatment NOx-conversion efficiency, as

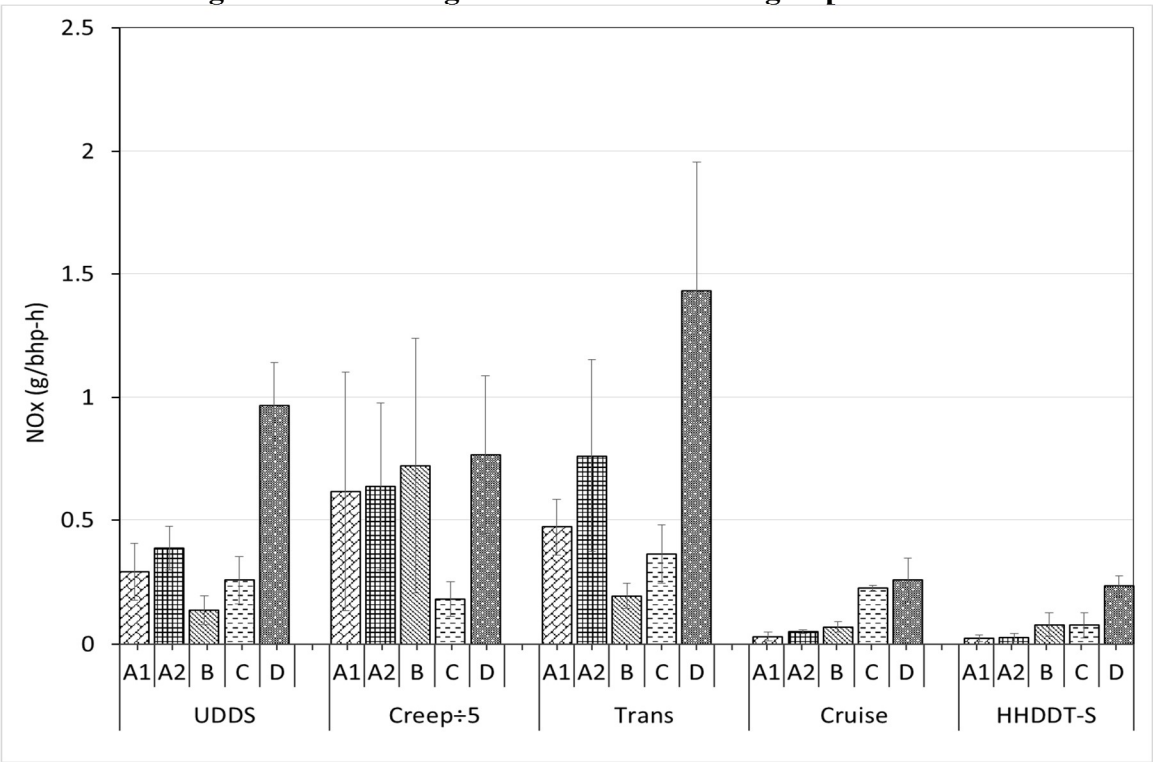
revealed through PEMS measurements. The regeneration intervention is believed to have been fully effective in driving off the accumulated unburned hydrocarbons that were hindering catalytic reaction. Additional chassis dynamometer testing of the manufacturer D vehicle was subsequently conducted at the West Virginia University (WVU) Center for Alternative Fuels Engines and Emissions (CAFEE) Laboratory, replicating the testing that had been performed at UCR, with the exception of a 70,000 lbs. test weight. The results of that testing indicated a NOx emission rate of 0.39 g/mi over the UDDS cycle, near the lower end of the NOx emission rates found in the current study.

Figure ES-1. Average NOx Emissions on a g/mi Basis



Note that D represents the UDDS emissions level found after retesting the manufacturer D1 truck after a regeneration.

Figure ES-2. Average NOx Emissions on a g/bhp-hr Basis.



The results of our study can be compared to the emission factors being used in the EMFAC2014 model. For engines certified to the 0.20 g/bhp-hr NOx level, EMFAC2014 utilizes a ZMR of 1.89. This ZMR is adjusted by a fuel correction factor of 0.93 to account for clean CARB diesel used in California, such that a ZMR of 1.76 was used for the comparisons in this study for 0.20 g/bhp-hr NOx engines. EMFAC2014 does not specifically utilize a ZMR for engines certified to the 0.35 g/bhp-hr NOx level, but the ZMR for those engines can be most directly compared with the 2011 model year ZMR of 3.26. That ZMR also is adjusted by the fuel correction factor of 0.93 to account for the use of clean CARB diesel used in California, resulting in a ZMR of 3.03, which was used for the comparisons in this study relating to 0.35 g/bhp-hr NOx engines. EMFAC2014 is a relatively complicated model, including speed correction factors, tampering, malmaintenance and deterioration rates, and other factors. ZMRs, however, are best approximated by the emissions obtained over the UDDS cycle from low mileage vehicles that are well maintained and checked for any evidence of tampering, such as those selected for inclusion in this study. Consequently, that cycle is used as a comparison point between the current EMFAC2014 model ZMRs and our study results.

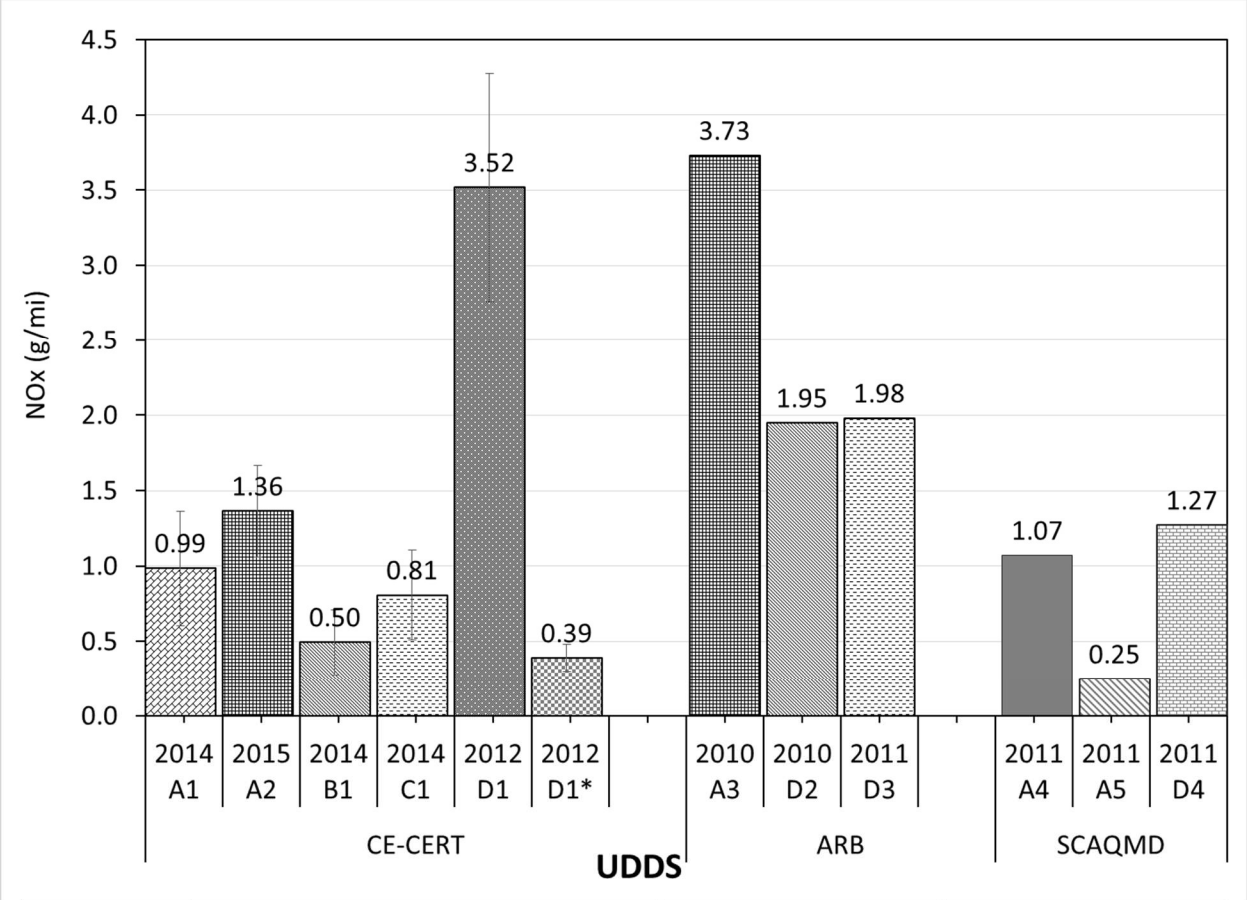
The UDDS emission rates from this study, as well as other studies of 2010 and newer heavy-duty vehicles, are presented in Figure ES-3 and ES-4, along with the current EMFAC2014 ZMRs and the NOx Family Emission Limits (FELs) to which the engines are certified. The other studies include a study by the ARB that was utilized to update the emissions factors for its EMFAC model and another UCR study funded by the South Coast Air Quality Management District (SCAQMD), which included several SCR and DPF equipped 2010+ model year vehicles in the test fleets. For the UDDS for the current study, the emissions levels for all of the 0.20 g/bhp-hr NOx trucks, except the manufacturer D truck prior to regeneration, are below the EMFAC2014

value. The NOx emissions for the manufacturer D truck after regeneration restored its NOx-conversion efficiency are also well below the EMFAC2014 value. The average UDDS values for the current study for the 0.20 g/bhp-hr NOx trucks are 0.89 g/mi if the outlier manufacturer D truck is excluded, or 0.77 g/mi if the UDDS emission rate from the manufacturer D truck as tested at WVU, after ensuring regeneration, is included. In both cases, those values are well below the EMFAC2014 value, with the lower value being more representative of the in-use fleet. Similarly, the manufacturer A1 truck that was certified to a 0.35 g/bhp-hr NOx FEL has a UDDS emissions level of 0.99 g/mi, which is well below the 2011 model year ZMR approximation of 3.03 g/mi for 0.35 g/bhp-hr NOx trucks.

The UDDS results for the earlier SCAQMD study, with an average NOx level of 0.86 g/mi, are also well below the EMFAC2014 0.20 g/bhp-hr fuel-adjusted ZMR of 1.76 g/mi. The two manufacturer D trucks from the previous ARB study are comparable to but slightly higher than the EMFAC2014 ZMR, as these two vehicles were the primary source of information used in developing that ZMR. They are higher than all of the other trucks certified to the 0.20 g/bhp-hr, with the exception of the manufacturer D truck under the current study, which is again from the same manufacturer. The other manufacturer A3 truck in the earlier ARB study has NOx emissions higher than the 2011 model year EMFAC2014 ZMR value that approximates the value for 0.35 g/bhp-hr NOx level engines.

As the name implies, the ZMR is the estimated emissions rate at zero miles (i.e., for a new truck). Normally, an adjustment factor would be applied to account for the mileage accumulated on the vehicle. As the five vehicles in this study had low mileages (one of the selection criteria for the study as shown in Figure ES-3), no adjustment factor has been applied for mileage. If an adjustment factor were to be applied, the ZMR estimates based on the UDDS emission results would go down slightly. Overall, this comparison analysis suggests that lower ZMRs for future EMFAC model updates may better represent the in-use emission rates of heavy-duty diesel trucks. This study suggests ZMRs may be better represented by the UDDS emission rates of 0.77 g/mi, perhaps ranging to 0.89 g/mi, for engines certified to a 0.20 NOx standard. A lower possible ZMR value of 0.99 g/mi was also found for an engine certified to a 0.35 NOx standard, but only a single vehicle with this certification level was tested in this study.

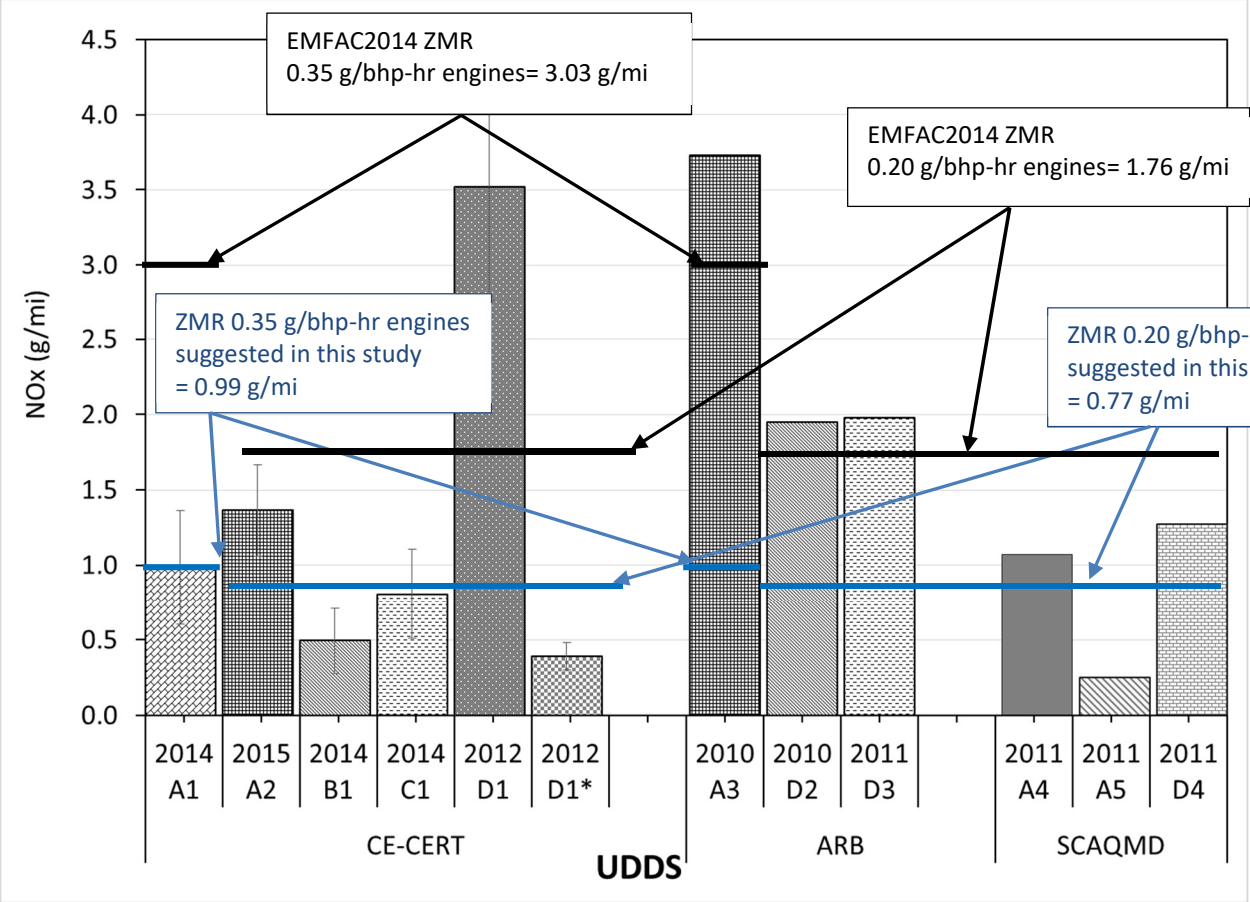
Figure ES-3 Comparison of NOx emission rates from this study, and the ARB and SCAQMD studies, for SCR-equipped 2010+ vehicles over the UDDS



	CE-CERT					ARB			SCAQMD		
Maker	A1	A2	B1	C1	D1	A3	D2	D3	A4	A5	D4
ODO miles	28,611	2,924	15,914	7,686	12,640	13,500	68,000	36,900	14,269	4,769	36,982
STD/FEL g/bhp-hr	0.35	0.20	0.20	0.20	0.20	0.35	0.20	0.20	0.20	0.20	0.20

*Note that D1 represents the UDDS emissions level found after retesting the manufacturer D1 truck after a regeneration.

Figure ES-4 Comparison of NOx Zero Mile Rates Between EMFAC2014 and this Study



1 Introduction

Heavy-duty diesel trucks can be significant sources of oxides of nitrogen (NOx) and particulate matter (PM) emissions in urban areas. In order to reduce emissions of NOx and particulate matter (PM) from HDDTs, a series of regulations for heavy heavy-duty diesel engines (HDDE) were implemented starting in 1974, and were last made more stringent in 2007 and 2010. Those rules have required that emissions of NOx and PM be reduced from an estimated unregulated emission level of 16 g/bhp-hr to 0.20 g/bhp-hr and from 1.0 g/bhp-hr to 0.01 g/bhp-hr, respectively. Current-technology diesel engines are now equipped with diesel particulate filters (DPFs) to meet the PM standards for 2007 and newer engines, and selective catalytic reduction (SCR) systems to meet the NOx standards for 2010 and newer engines.

While there are extensive data on the effectiveness of DPFs and SCR systems over certification test cycles run in emissions laboratories, data on in-use emissions from modern diesel engines are scarce and show some variation depending on the type of truck tested and the testing conditions (Miller et al., 2013; Carder et al., 2014; Misra et al., 2015; California Air Resources Board 2015). The need for in-use emissions data is particularly important because HDD engines are certified to meet emission standards before the engines are integrated into a vehicle chassis for commercial use, which commercial uses can span a broad range of applications. Characterization of in-use emissions from on-road heavy-duty trucks is an important element of developing accurate emissions inventory estimates.

The California Air Resources Board (ARB) has been utilizing in-use emissions testing results in the development of emissions factors for its EMFAC model for a number of years (California Air Resources Board, 2015a, 2015b). Those emissions factors are developed from "zero-mile" emissions rates (ZMRs) that can be adjusted to account for engine deterioration with age and for variations in vehicle speed. For the EMFAC2007 model, in-use emissions data were primarily obtained from the CRC E-55/59 study (Clark et al. 2006, 2007) coupled with estimates for 2007 and newer model year vehicles.

For the EMFAC2014 model, a greater emphasis was placed on developing emission factors for vehicles equipped with newer PM and NOx aftertreatment control devices, and incorporating in-use emissions data from 2007 and newer engines/vehicles. Those data were derived from studies conducted by the ARB and the South Coast Air Quality Management District (SCAQMD), which included a limited amount of chassis dynamometer testing over multiple cycles, and over-the-road testing with portable emissions measurement systems (PEMS) over two routes and at three different loads. While this represents an important step in better quantifying emissions from 2007-2009 and 2010 and later model year vehicles, the data are still relatively scarce to serve as the basis for making important emissions inventory projections out to 2020 and beyond. In particular, for the 2010 and later model year technology engines, only 5 vehicles/engines were included in the ARB/SCAQMD studies, with all the engines being in the 2010-2011 model year range, which only covers the earliest implementation years for advanced NOx control strategies. More importantly, of those 5 engines, only 2 were certified to the 0.20 g/bhp-hr NOx standard, and both of those engines were from the same manufacturer. Additionally, 2 of the 5 engines utilized only exhaust gas recirculation (EGR) for NOx control, an approach that had a very limited production run.

The goal of this study is to provide additional information regarding modern heavy-duty diesel vehicles equipped with the newest emission control strategies for reducing NOx. Testing was conducted on 5 HDDTs with model year 2012 to 2015 engines equipped with DPFs and SCR systems. The vehicle matrix included 5 engines from heavy-duty engine manufacturers representing the majority of trucks operating in California, with two engines being from the same manufacturer. The engines/vehicles were certified to a 0.20 g/bhp-hr certification limit, with the exception of one credit-using engine that was certified to a 0.35 g/bhp-hr standard. Each vehicle was tested on UCR's heavy-duty chassis dynamometer over the four phases of ARB's Heavy Heavy-Duty Diesel Truck (HHDDT) cycle (i.e., idle, creep, transient, and cruise), the HHDDT-short or (HHDDT-S) cycle (which is a high-speed cruise cycle), and the Urban Dynamometer Driving Schedule (UDDS) (which is a cycle considered to be the chassis dynamometer equivalent of the engine dynamometer transient test). Three of the HDDTs were also retested at the ARB's Heavy-Duty Chassis Dynamometer Testing Facility in Los Angeles.[2] The results obtained from this study, can augment the data being used in the development of future emissions inventory models that are relied on throughout ARB's regulatory process.

[2] This report is based on data and analysis from the truck testing conducted at CE-CERT. It is important to note that additional testing was also carried out on a subset of the trucks at the ARB's heavy-duty chassis-dynamometer test facility as part of the agreed upon study effort. The goal was to include all of the data and analysis from both testing efforts in this report, however the ARB data have not been provided for analysis or reporting. The additional ARB data would provide additional insight to the overall project results. It is hoped that ARB will consider their data, in addition to what is reported here, as they improve the EMFAC model.

2 Experimental Procedures

2.1 Test Vehicles and Fuels

Five vehicles were tested in this program. All of the vehicles were heavy-duty Class 8 trucks powered by diesel engines equipped with the latest generation of emissions control technology, including a diesel particulate filter (DPF) for PM emissions control and a selective catalytic reduction (SCR) system for NO_x emissions control. The selected vehicles were equipped with engines that were at least model year 2012 and newer, with a preference for 2014 and newer engines. The vehicle matrix included 5 engines from heavy-duty engine manufacturers representing the majority of trucks operating in California, with two engines being from the same manufacturer. The engines/vehicles were certified to a 0.20 g/bhp-hr certification limit, with the exception of one engine that was certified to a 0.35 g/bhp-hr standard. The engines selected have reasonably high sales percentages in the California market. The vehicles all had mileage less than 30,000 miles, with mileage ranging from 2,924 to 28,611 miles. The selection and approval of each test vehicle was done in consultation with both the Truck and Engine Manufacturers Association (EMA) and the ARB. A description of the vehicles/engines is provided in Table 2-1. Vehicles were obtained from different entities, including local truck rental agencies, dealers, and other entities. Three of the five vehicles were also subsequently tested at the ARB Heavy-Duty Chassis Dynamometer Testing Facility in Los Angeles, CA. The results from the testing at the ARB facility are not included in this report, but are expected to be considered and incorporated as part of the next update to the EMFAC model, and will be made publically available at that time.

Table 2-1. Engine/Vehicle Specifications

Engine	*A1*	*A2*	*B*	*C*	*D*
Model Year	2014	2015	2014	2014	2012
Engine Type	Compression-ignition	Compression-ignition	Compression-ignition	Compression-ignition	Compression-ignition
Horsepower	400 HP	550 HP	450 HP	450 HP	415 HP
Vehicle Mileage	28,611	2,924	15,914	7,686	12,640
Displacement	14.9 L	14.9 L	12.8 L	12.4 L	12.8 L
Peak Torque	1450 ft-lbs. @ 1650 rpm	2050 ft-lbs. @ 1200 rpm	450 hp. @ 1800 rpm	1700ft-lbs.@ 1000 rpm	415 hp. @ 1500 rpm
Aftertreatment	DOC/DPF/SCR	DOC/DPF/SCR	DOC/DPF/SCR	DOC/DPF/SCR	DOC/DPF/SCR
Test Period	November 2014	Sept./Oct, 2015	January 2015	December 2014	February 2015
Standard/FEL Level (g/bhp-hr)	NO_x:0.35 PM:0.01	NO_x:0.20 PM:0.01	NO_x:0.20 PM:0.01	NO_x:0.20 PM:0.01	NO_x:0.20 PM:0.01
Certification Level (g/bhp-hr)	NO_x:0.22 PM:0.001	NO_x:0.18 PM:0.000	NO_x:0.17 PM:0.004	NO_x:0.12 PM:0.003	NO_x:0.12 PM:0.003

After acquisition, each vehicle was inspected using a standard checklist to ensure the vehicle was safe to drive and testable on a chassis dynamometer. The checklist utilized for this program is provided in Appendix A.

The test fuel was the fuel in the tank of the vehicle, provided it could be reasonably ascertained to be California No. 2 diesel. The vehicles were topped off, if needed, with a commercially available California No. 2 diesel fuel, prior to testing, to ensure that adequate fuel was available for the full test program.

2.2 Test Cycles

Each vehicle was tested over the four phases of ARB's Heavy Heavy-Duty Diesel Truck (HHDDT) cycle (i.e., idle, creep, transient, and cruise) (Gautam, et al. 2002), the HHDDT short or (HHDDT-S) cycle (Clark et al., 2004) (which is a high-speed cruise schedule), and the Urban Dynamometer Driving Schedule (UDDS) (U.S. Environmental Protection Agency, 2005). The characteristics of each test cycle are provided in Table 2-2 and Appendix B. Two tests were conducted for each of the three test modes. For each test over each of the different test cycles, different numbers of replicates were run. The number of iterations that were conducted on each cycle is provided in Table 2-3, along with the preconditioning sequence and total test time. Conducting multiple iterations of each driving cycle helped to ensure that a sufficient mass of PM was collected for weighing. Note that the preconditioning and number of test iterations were designed to be consistent with the procedures utilized in the earlier testing program the ARB

conducted to update its emission factors for EMFAC2014. The ARB was also involved in the review and approval of this test sequence. The ARB followed the same testing protocol in its retesting of 3 of the 5 vehicles utilized in this study.

Table 2-2. Description of Test Cycles

Schedule	Pre-conditioning	Time (s)	Avg Speed (mph)	Distance (mi)	Number of Iterations	Total Test Time (s)	Description
UDDS	One UDDS	1060	18.86	5.55	3	3180	comparable to FTP engine dynamometer transient cycle
HHDDT Idle	15 minutes at 45 mph	900	0	0	3	2700	Idle of vehicle
HHDDT Creep	15 minutes at 45 mph	256	1.7	0.124	10	2560	Stop and go modes (congestion)
HHDDT Transient	15 minutes at 45 mph	688	14.9	2.9	4	2752	Local street driving
HHDDT Cruise	15 minutes at 45 mph	2083	39.9	23.1	1	2083	Freeway driving
HHDDT-Short	15 minutes at 45 mph	760	49.9	10.5	2	1520	High speed driving

The vehicles were warmed up in the morning by driving them for 15 minutes at 45 mph to bring the vehicles out of a cold-start mode. The vehicles then went through a test sequence that included each of the test cycles with the associated preconditioning cycle. An outline of a typical test day is provided in Table 2-3. Between each test sequence, there was a soak period to allow for analysis of the emissions from the just-completed test, to replace the PM-collection filters for the upcoming test, and to prepare the laboratory instrumentation for the next test. The soak period was typically on the order of 10 to 20 minutes with the engine off. Once those activities were completed, the vehicle went into the preconditioning for the next test, and then, immediately following, was run on the test sequence where emissions were collected. It should be noted that the vehicles were monitored throughout testing to ensure that a DPF regeneration event did not occur during any of the emissions tests. For this study, the only observed regeneration occurred while testing the manufacturer C engine during a preconditioning warm-up. In that one case, it was ensured that the regeneration was completed prior to starting the actual emissions testing.

Table 2-3. Test Sequence for Typical Test Day

Activity		
Vehicle warm up (15 minutes at 45 mph)	Blue is full testing	
Soak (engine off)		
UDDS	Yellow is soak/break	
UDDS x 3		
Soak (engine off)	Red is prep/Conditioning	
15 minutes @ 45 mph		
HHDDT Idle x 3		
Soak (engine off)		
15 minutes @ 45 mph		
HHDDT Creep x 10		
Testing break/soak (engine off)		
15 minutes @ 45 mph		
HHDDT Transient x 4		
Soak (engine off)		
15 minutes @ 45 mph		
HHDDT Cruise		
Soak (engine off)		
15 minutes @ 45 mph		
HHDDT-S x 2		

The road-load coefficients were calculated based on the frontal area of the vehicle and a factor accounting for the general shape of a class 8 tractor. A description of the road-load calculation methodology is provided in Appendix C, along with the specific road-load coefficients used for each tractor. In comparing between vehicles, the primary difference in road-loads between different test tractors is in the measured frontal area of each tractor, with the most significant difference between vehicles being that the manufacturer A2 tractor had a smaller frontal area than the other tractors since it did not have an aerodynamic fairing on the roof of the cab. Note in this case, since the frontal area is based on the area of a bare cab (tractor) without a trailer, the aerodynamic drag for the tractor without the aerodynamic fairing is actually less than that for the other tractors. The actual aerodynamic drag on the road for a truck carrying a box trailer would likely be greater if the tractor did not have a fairing. Road loads can also be determined more directly by on-road coast-downs. This requires finding a roadway that is sufficiently flat, long, and safe to provide a consistent coast down time. This was not done as part of this test program. However, prior to testing, a coast-down was performed was for each tractor on the chassis dynamometer to ensure the coast down time on the dynamometer was similar to that calculated based on the road load coefficients.

The vehicles were tested at a weight of 65,000 lbs. That weight was selected because the Federal Highway Administration estimates that a typical 5-axle semi-truck with a trailer is loaded to an approximately 65,000 lbs. gross combined weight (GCW) (Couch and Leonard, 2011; Federal Highway Administration, 2000). Note that this test weight is slightly greater than the test weight

of 56,000 ±1,000 lbs. used by the ARB in its earlier testing to update EMFAC2014. The choice of a test weight of 65,000 lbs. was reviewed with and accepted by ARB.

2.3 Emissions Testing and Measurements

The chassis dynamometer testing was conducted at the University of California, Riverside (UCR) Center for Environmental Research and Technology's (CE-CERT's) heavy-duty chassis dynamometer facility. A picture of a typical vehicle set-up on the chassis dynamometer at CE-CERT is provided in Figure 2-1. The ARB's retesting of 3 of the 5 subject vehicles was conducted at its Heavy-Duty Chassis Dynamometer Testing Facility in Los Angeles.

Figure 2-1. Typical Setup of Test Vehicles on the Chassis Dynamometer

The emissions measurements were made using CE-CERT's Mobile Emissions Laboratory (MEL). For all tests, standard emissions measurements of total hydrocarbons (THC), non-methane hydrocarbons (NMHC), methane (CH_4), carbon monoxide (CO), NO_x, carbon dioxide (CO_2), and PM, were measured. CO and CO_2 emissions were measured with a 602P nondispersive infrared (NDIR) analyzer from California Analytical Instruments (CAI). THC, NMHC, and CH_4 emissions were measured with 600HFID flame ionization detector (FID) from CAI. NO_x emissions were measured with 600HPLC chemiluminescence analyzer from CAI. A schematic of the experimental setup is provided in Figure 2-2. Fuel consumption was derived from the CO_2, CO, and THC emissions by the carbon balance, using typical densities and carbon weight fractions for California ULSD.

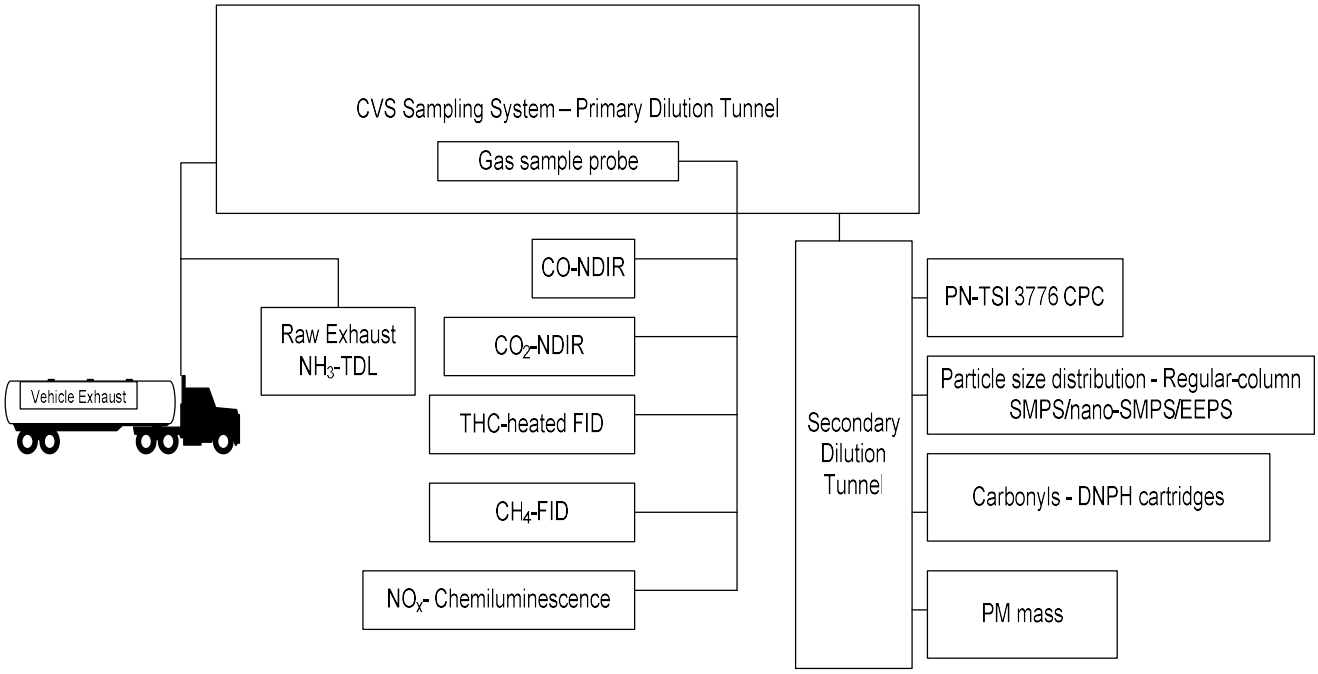
The mass concentrations of $PM_{2.5}$ were obtained by analysis of particulates collected on 47mm diameter 2µm pore Teflo filters (Whatman brand). The filters were measured for net gains using a UMX2 ultra precision microbalance with buoyancy correction in accordance with the weighing procedure guidelines of the Code of Federal Regulations (CFR). Sampling for PM was done cumulatively over the entire duration of the cycles due to the very low mass levels expected for PM.

Solid particle number counts were measured with a TSI 3776 ultrafine-Condensation Particle Counter (CPC) with a 2.5 nm cut point coupled with a catalysis striper (CS). The operating temperature of the CS was 300°C, which is a temperature that is sufficiently high to remove most volatile particles, leaving only solid particles to be detected by the CPC.

An Engine Exhaust Particle Sizer (EEPS) spectrometer (TSI 3090, firmware version 8.0.0) was used for measuring particle size distributions on a concentration and mass basis. The EEPS was used to obtain real-time second-by-second size distributions between 5.6 to 560 nm. The EEPS has a scan time of one second and provides a size range from 6 to 423 nm in electrical mobility. Particles were sampled at a flow rate of 10 L/min, which is considered to be sufficiently high to minimize diffusional losses. Particles were charged with a corona charger and sized based on their electrical mobility in an electrical field. Concentrations were determined through the use of multiple electrometers.

Engine brake power was calculated using ECM broadcast J1939 standardized information, including the engine speed in revolutions per minute (rpm), ECM broadcast actual torque in (%) estimated using engine speed and instantaneous fuel flow, ECM broadcast friction torque in (%), and ECM broadcast reference torque in (ft-lb). These signals are the same signals used for in-use compliance testing according to the in-use test procedures in 40 CFR Part 1065.

Figure 2-2. Schematic of the Sampling Systems and Instruments



3 Heavy-Duty Vehicle Chassis Dynamometer Testing Results

The emissions test results are presented in this section. The figures for each pollutant show the results for each vehicle/fuel/cycle combination based on the average of tests conducted on that particular test combination. The error bars on the figures are the standard deviation over all tests for each test combination. Note that in many figures the emissions for the Creep cycle are divided by 5 to allow the emissions over all 5 cycles to be more clearly presented on the same graph.

3.1 NOx Emissions

NOx emissions for the test trucks are shown on a g/mi and g/bhp-hr basis in Figure 3-1 and Figure 3-2, respectively. NOx emissions varied depending on the test cycle and the test truck. It should be noted that the NOx emissions for the manufacturer D vehicle were an outlier with noticeably higher emissions levels. Additional testing was performed on this vehicle subsequent to the main testing conducted under this study after the vehicle's aftertreatment system was regenerated, and the results are included in Figure 3-1. Given the anomalies relating to the manufacturer D truck, this truck is discussed separately from and in more detail than the results for the manufacturer A1, manufacturer A2, manufacturer B and manufacturer C engine-powered trucks.

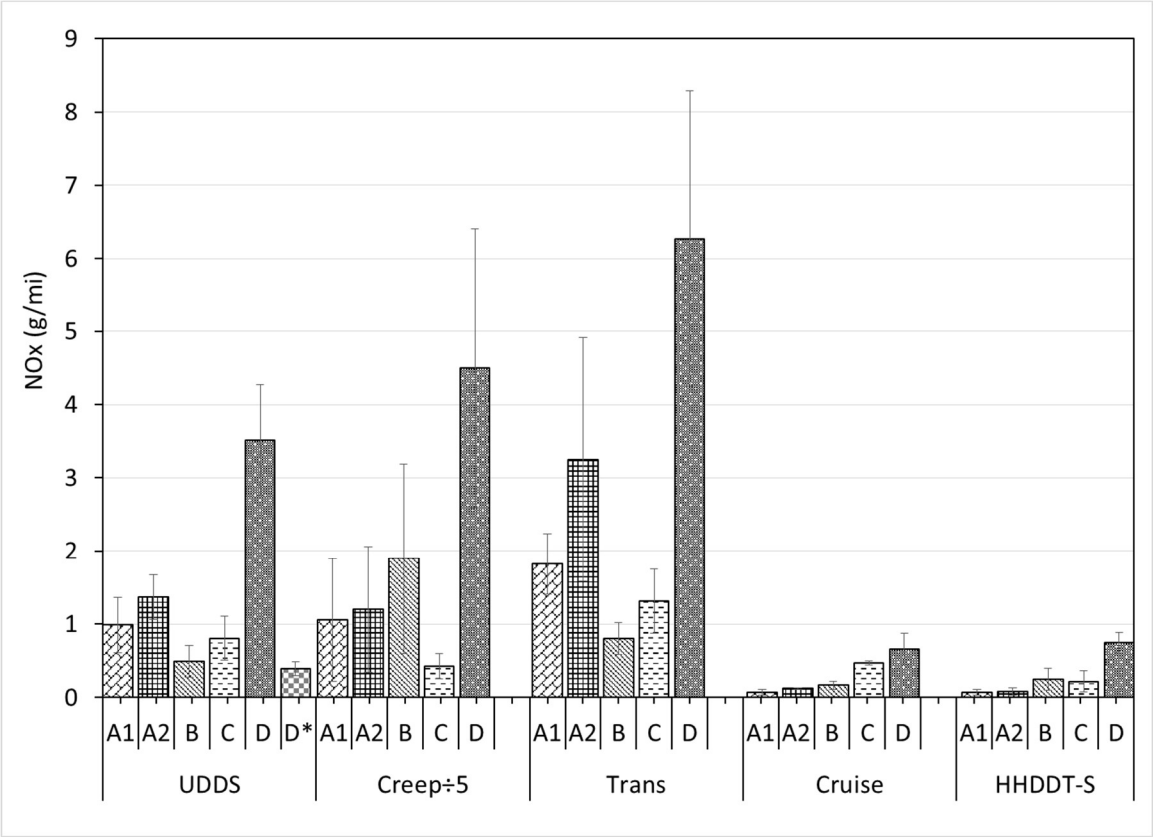
For the manufacturer A1, manufacturer A2, manufacturer B and manufacturer C engine-powered trucks, on a g/mi basis, NOx emissions ranged from 0.495 to 1.363 over the UDDS (the cycle most relevant to ZMRs), from 2.131 to 9.468 over the Creep cycle, from 0.803 to 3.252 over the Transient cycle, from 0.068 to 0.471 over the Cruise cycle, and from 0.067 to 0.249 over the HHDDT-S. The lowest NOx emissions were recorded over the Cruise and HHDDT-S cycles, which produce the highest speeds, loads, and exhaust temperatures. Under those conditions, SCR catalysts are expected to operate at temperatures where NOx conversion efficiencies are robust, leading to relatively low tailpipe NOx emission, even though engine-out NOx levels are likely highest. Higher emissions were observed over the other cycles, which include more transient and lower average speed operation, with different vehicles showing higher or lower emissions on different cycles. The Creep cycle showed the highest NOx emissions on a g/mile or g/bhp basis, since it is comprised of short, low-speed accelerations between periods of idle that yield lower loads and exhaust temperatures, and that cover a very short distance (0.124 miles). Absolute values of NOx are likely lower.

For the manufacturer A1, manufacturer A2, manufacturer B and manufacturer C trucks, on a g/bhp-hr basis, NOx emissions ranged from 0.136 to 0.387 over the UDDS, from 0.910 to 3.613 over the Creep cycle, from 0.194 to 0.762 over the Transient cycle, from 0.030 to 0.225 over the Cruise cycle, and from 0.023 to 0.078 over the HHDDT-S. Emissions were generally comparable to their respective emissions standards over the UDDS. Emissions on a g/bhp-hr basis were higher for the Transient cycle, although the manufacturer B engine on the Transient cycle showed emissions comparable to the UDDS. For the Cruise and HHDDT-S cycles, the emissions for most vehicles were below the NTE standard of 0.30 g/bhp-hr, which is the standard most relevant for regulatory in-use emissions testing of heavy-duty trucks under highway cruise conditions. Emissions over the Creep cycle were the highest for all vehicles. For the Creep cycle, the NOx emissions for the manufacturer A1, manufacturer A2 and manufacturer B truck engines were comparable, with the manufacturer C engine recording the lowest NO_x emissions.

As discussed above, the manufacturer D truck was an outlier with noticeably higher NOx emissions relative to the other vehicles. In this study, on a g/mi basis, its NOx emissions were 3.519 over the UDDS, 22.505 over the Creep cycle, 6.270 over the Transient cycle, 0.656 over the Cruise cycle, and 0.751 over the HHDDT-S. The lowest NOx emissions for the manufacturer D engine were recorded over the Cruise and HHDDT-S cycles. On a g/bhp-hr basis, NOx emissions for the D vehicle were 0.967 for the UDDS, 3.832 for the Creep cycle, 1.430 for the Transient cycle, 0.257 for the Cruise cycle, and 0.234 over the HHDDT-S.

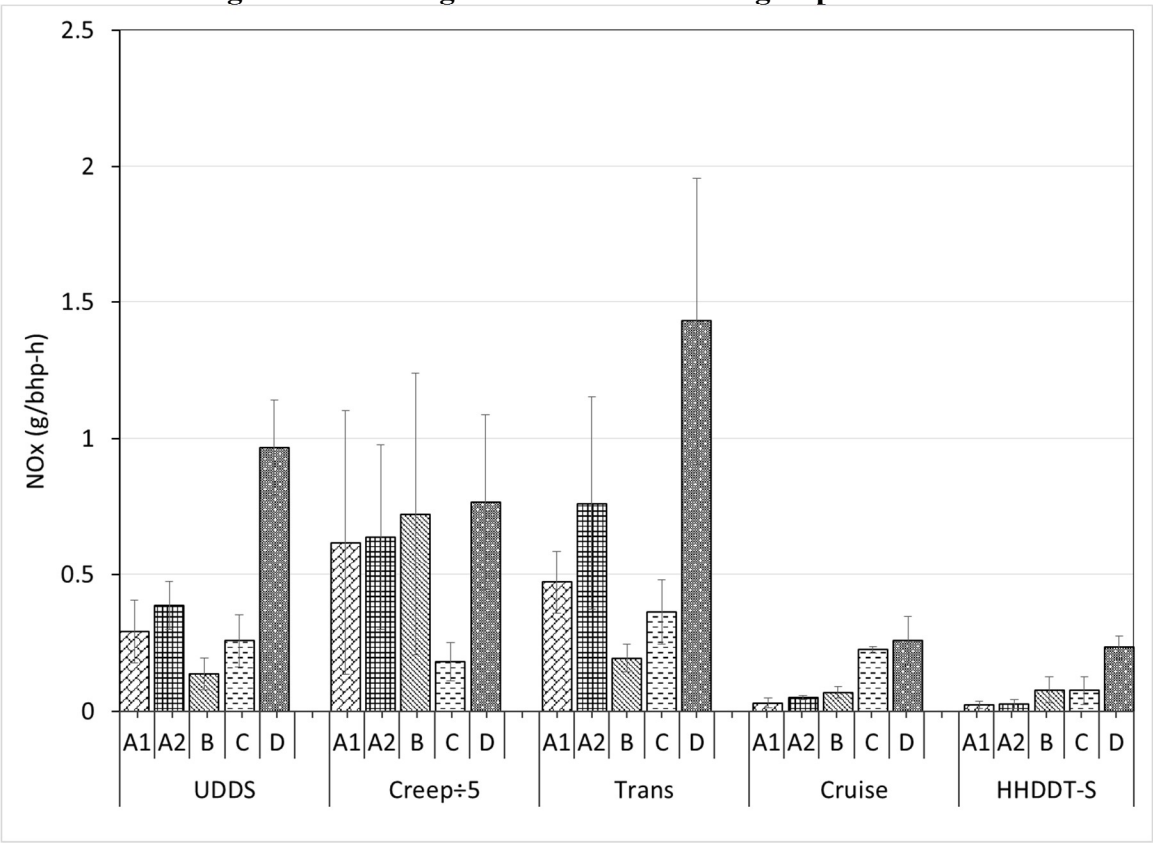
Subsequent to the dynamometer testing conducted at UCR, the manufacturer D vehicle was more extensively evaluated by the manufacturer. The vehicle was initially outfitted with a PEMS, which confirmed the poor NOx conversion efficiency observed from the UCR testing. Upon further investigation, it was found that this specific vehicle had served its entire life as a dealer demonstrator, and as such rarely or ever operated with a loaded trailer, and spent a considerable amount of time operating in an idle mode. This type of low-temperature, high proportion idle operation is known to cause significant exposure of the aftertreatment system to unburned hydrocarbons in the exhaust stream. An examination of the logged electronic history revealed no OBD faults or other indications of failure or system malfunction. A clear anomaly, however, was that due to its unusual, unladen service history and duty cycle, the engine had never undergone a regeneration event, despite being approximately 2.5 years in service (albeit with only 12,000 miles on the odometer). A series of conventional parked regenerations were performed. After further operation, there was a significant recovery of the aftertreatment NOx-conversion efficiency, as revealed through PEMS measurements. The regeneration intervention is believed to have been fully effective in driving off the accumulated unburned hydrocarbons that were hindering catalytic reaction. Additional chassis dynamometer testing of manufacturer D vehicle was conducted at the West Virginia University (WVU) Center for Alternative Fuels Engines and Emissions (CAFEE) Laboratory, replicating the testing that had been performed at UCR, with the exception of a 70,000 lbs. test weight. The results of that testing indicated a NOx emission rate of 0.39 g/mi over the UDDS cycle, near the lower end of the NOx emission rates found in the current study.

Figure 3-1 Average NO_x Emissions on a g/mi Basis



Note that D represents the UDDS emissions level measured upon retesting the manufacturer D truck after ensuring a regeneration event.

Figure 3-2. Average NO_x Emissions on a g/bhp-hr Basis



The NOx emissions from this study can be compared to those from previous studies. Studies that have included SCR-equipped 2010 and later model year production Class 8 trucks include studies conducted by the ARB as part of the development of the EMFAC2014 model (California Air Resources Board, 2015a, 2015b), and a study sponsored by the SCAQMD that included testing by UC Riverside (Miller, et al. 2013) and West Virginia University (WVU) (Carder et al., 2014). The earlier ARB study included a truck equipped with a manufacturer A 2010 engine certified to the 0.35 g NOx/bhp-hr level, and two manufacturer D engines certified to the 0.20 g NOx/bhp-hr level (California Air Resources Board, 2015a, 2015b). In that ARB study, the 2010 manufacturer A truck produced NO_x emissions of 3.73 g/mi over the UDDS, 16.78 g/mi over the Creep cycle, 6.33 g/mi over the Transient cycle, 0.60 g/mi over the Cruise cycle, and 0.74 g/mi over the high-speed Cruise cycle. All of those values are significantly higher than those obtained in this study for trucks tested at CE-CERT. In that same ARB study, a 2010 manufacturer D truck produced NOx emissions of 1.95 g/mi over the UDDS, 16.08 g/mi over the Creep cycle, 6.33 g/mi over the Transient cycle, 0.60 g/mi for the Cruise cycle, and 0.74 g/mi for the high-speed Cruise, while a 2011 manufacturer D truck produced NOx emissions of 1.98 g/mi over the UDDS. The emissions reported for those manufacturer D trucks are lower than that values for the manufacturer D truck tested in this study (before retesting at WVU). In the previous UCR SCAQMD study, UDDS emissions for trucks equipped a manufacturer A 8.3 liter engine (A4), a manufacturer A 11.9 liter engine (A5), and a manufacturer D 12.8 liter engine (D4) were found to be 1.07, 0.25, and 1.27 g/mi, respectively (Miller et al., 2013). Those values are comparable to or lower than the values observed in the present study. In that same UCR SCAQMD study, WVU found slightly NOx higher UDDS emissions of 1.98 g/mi for the manufacturer D vehicle

(D4) (Carder et al., 2015). Misra et al. (2013 and 2016) also conducted some in-use testing with PEMS on 2010 to 2014 model year SCR-equipped trucks on roads in the Sacramento area. They found NOx emissions rates of 0.07 to 0.27 g/bhp-hr for highway driving, consistent with the relatively low results obtained in this study, and NOx emission rates of 0.22 to 1.07 g/bhp-hr for arterial driving, which is more comparable to the UDDS and Transient cycles for this study.

The results of our study can be compared to the emission factors being used in the EMFAC model. For engines certified to the 0.20 g/bhp-hr NOx level, EMFAC2014 utilizes a ZMR of 1.89. This ZMR is adjusted by a fuel correction factor of 0.93 to account for the clean CARB diesel fuel used in California, such that a ZMR of 1.76 was used for the comparisons in this study for the 0.20 g/bhp-hr NOx engines. EMFAC2014 does not specifically utilize a ZMR for engines certified to the 0.35 g/bhp-hr NOx level, but the ZMR for those engines can be most directly compared with the 2011 model year ZMR of 3.26. That ZMR also is adjusted by a fuel correction factor of 0.93 to account for the use of clean CARB diesel fuel in California, resulting in a ZMR of 3.03, which was used for the comparisons in this study relating to the 0.35 g/bhp-hr NOx engines. EMFAC2014 is a relatively complicated model, including speed correction factors, tampering, malmaintenance and deterioration rates, and other factors. ZMRs, however, are best approximated by the emissions obtained over the UDDS cycle from low mileage vehicles that are well maintained and checked for any evidence of tampering, such as those selected for inclusion in this study. Consequently, that cycle is used as a comparison point between the current EMFAC model ZMRs and our study results. The UDDS emission rates from this study, as well as other studies of 2010 and newer model year heavy-duty vehicles, are presented in Figure 3-3 and Figure 3-4, along with the current EMFAC2014 ZMRs and the NOx Family Emission Limits (FELs) to which the engines are certified.

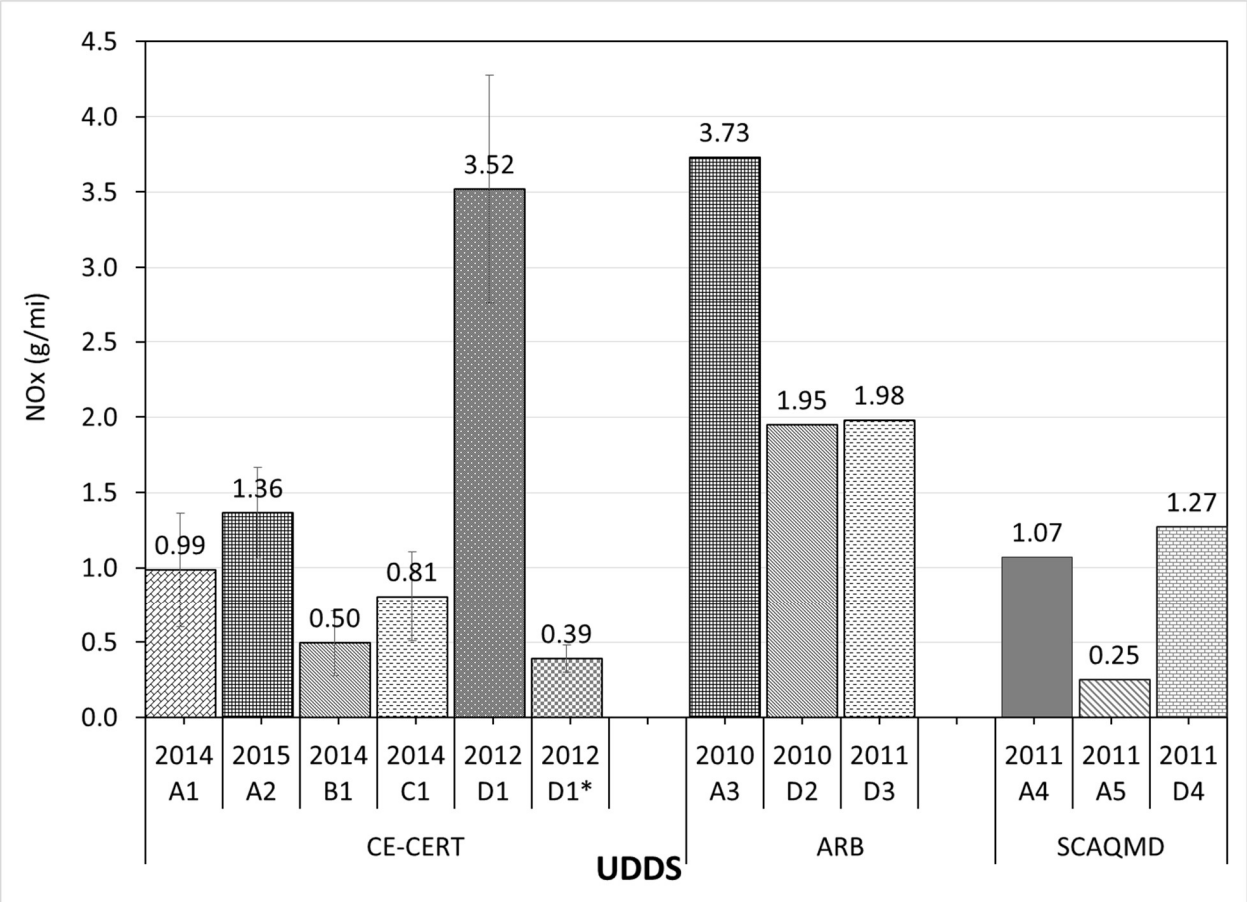
The other earlier studies include a study by the ARB that was utilized to update the emissions factors for the EMFAC model, and another UCR study funded by the South Coast Air Quality Management District (SCAQMD), which included several SCR-equipped 2010+ model year vehicles in the test fleets. For the UDDS under the current study, the emission levels for all of the 0.20 g/bhp-hr NOx trucks, except the manufacturer D truck prior to regeneration, are below the EMFAC2014 value. The NOx emissions for the manufacturer D truck after regeneration restored it NOx-conversion efficiency are also well below the EMFAC2014 value. The average UDDS values for the current study for the 0.20 g/bhp-hr NOx trucks are 0.89 g/mi if the outlier manufacturer D truck is excluded, or 0.77 g/mi if the UDDS emission rate from the manufacturer D truck as tested at WVU, after ensuring regeneration, is included. In all cases, those values are well below the EMFAC2014 value, with the lowest value being more representative of the in-use fleet. Similarly, the manufacturer A1 truck that was certified to a 0.35 g/bhp-hr NOx FEL has a UDDS emissions level of 0.99 g/mi, which is well below the 2011 model year ZMR approximation of 3.03 g/mi for 0.35 g/bhp-hr NOx trucks.

The UDDS results for the earlier SCAQMD study, with an average NOx level of 0.86 g/mi, are also well below the EMFAC2014 0.20 g/bhp-hr fuel-adjusted ZMR of 1.76 g/mi. The two manufacturer D trucks from the previous ARB study are comparable to but slightly higher than the EMFAC2014 ZMR, as those two vehicles were the primary source of information used in developing the ZMR. They are higher than all of the other trucks certified to the 0.20 g/bhp-hr, with the exception of the manufacturer D truck under the current study, which is again from the same manufacturer. The other manufacturer A3 truck in the earlier ARB study has NOx

emissions higher than the 2011 model year EMFAC2014 ZMR value that approximates the value for 0.35 g/bhp-hr NOx level engines.

Also, as the name implies, the ZMR is the estimated emissions rate at zero miles (i.e., for a new truck). Normally, an adjustment factor would be applied to account for the mileage accumulated on the vehicle. As the five vehicles in this study had low mileages (one of the selection criteria for the study, as shown in Figure 3-3), no adjustment factor has been applied for mileage. If an adjustment factor were to be applied, the ZMR estimates based on the UDDS emission results would go down slightly. Overall, this comparison analysis suggests that lower ZMRs for future EMFAC model updates may better represent the in-use emission rates of heavy-duty diesel trucks. This study suggests ZMRs may be better represented by the UDDS emission rates of 0.77 g/mi, perhaps ranging to 0.89 g/mi, for engines certified to a 0.20 NOx standard. A lower possible ZMR value of 0.99 g/mi was also found for an engine certified to a 0.35 NOx standard, but only a single vehicle with this certification level was tested in this study.

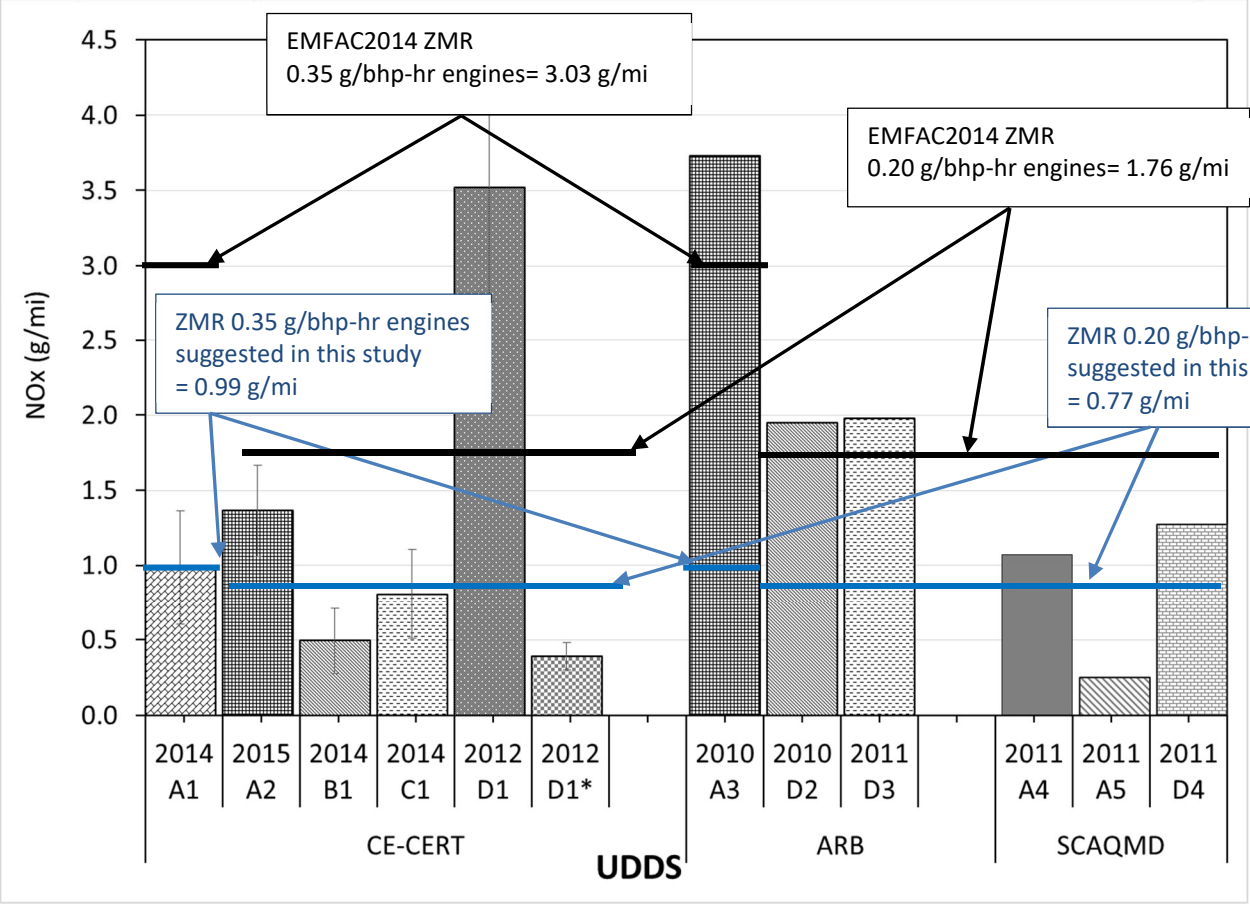
Figure 3-3 Comparison of NOx emission rates from this study, and the ARB and SCAQMD studies, for SCR-equipped 2010+ vehicles over the UDDS



	CE-CERT					ARB			SCAQMD		
Maker	A1	A2	B1	C1	D1	A3	D2	D3	A4	A5	D4
ODO miles	28,611	2,924	15,914	7,686	12,640	13,500	68,000	36,900	14,269	4,769	36,982
STD/FEL g/bhp-hr	0.35	0.20	0.20	0.20	0.20	0.35	0.20	0.20	0.20	0.20	0.20

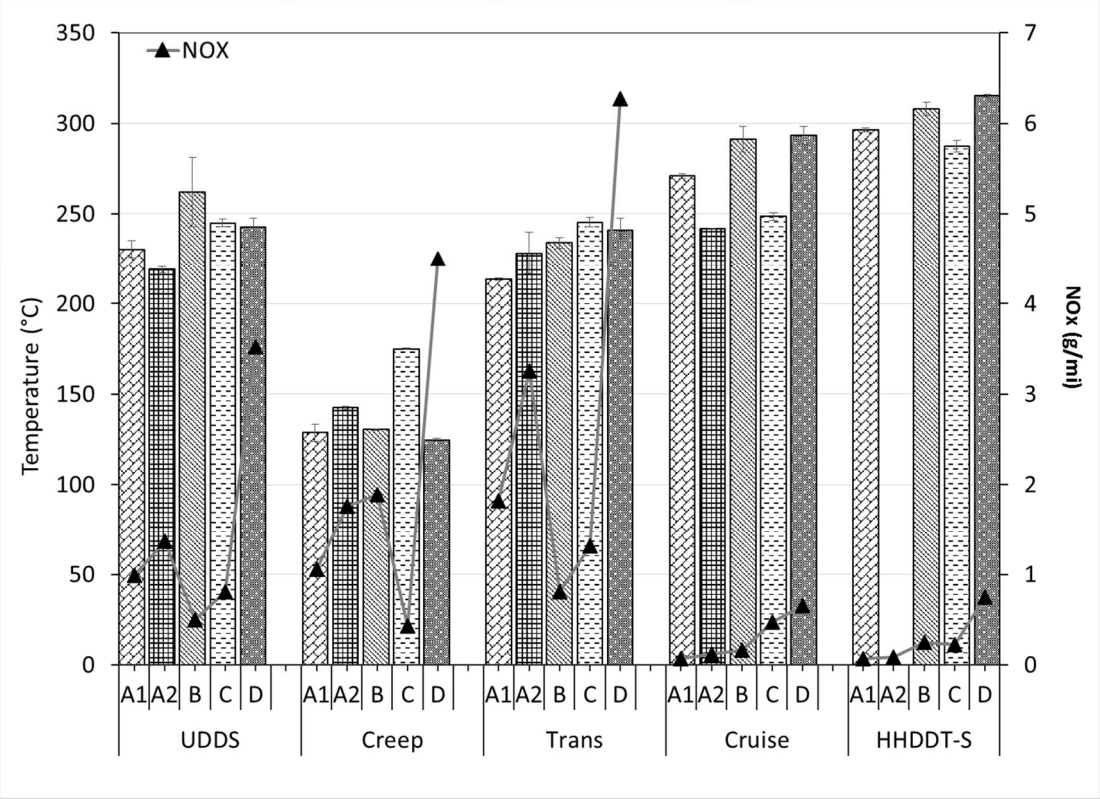
*Note that D1 represents the UDDS emissions level found after retesting the manufacturer D1 truck after a regeneration.

Figure 3-4 Comparison of NOx Zero Mile Rates Between EMFAC2014 and this Study



For SCR-equipped vehicles, NOx emissions are typically strongly correlated to the SCR temperature. Specifically, a minimum exhaust temperature is needed to promote hydrolysis of urea into ammonia (NH_3), which then reduces NOx into nitrogen (N_2) and water (H_2O) (Majewski, 2006). That requisite conversion temperature is typically around 250°C. The SCR inlet temperature for all vehicles in this study is provided in Figure 3-5. The results show that the average SCR inlet temperature is at or above 250°C for the Cruise and HHDDT-S cycles for all of the vehicles, except for the manufacturer A2 vehicle over the Cruise cycle. Note that although the SCR inlet temperature is not available for the manufacturer A2 engine, the SCR outlet temperature for that engine over the HHDDT-S cycle was above 250°C, indicating that the average SCR inlet would be above 250°C, as the inlet temperature was higher than the outlet temperature for all test combinations.

Figure 3-5. Average SCR Inlet Temperature



The average measured NOx emissions are also shown in Figure 3-5, to provide an additional comparison between NOx emissions and SCR inlet temperature. NOx emissions were lowest in most cases for the Cruise and HHDDT-S cycles, consistent with the highly effective conversion rate of NOx when the SCR has reached its effective operational temperature, with the increased NOx reduction efficiency more than making up for the increased NOx engine out emissions at high speed, high load operation. For the UDDS and Transient cycles, the average SCR inlet temperature was in the range of 213 to 261°C. This suggests that the SCR is at or above its operational temperature for only part of those cycles, which is consistent with the higher average NOx emissions observed over the UDDS and Transient cycles compared to the two Cruise mode cycles. The lowest temperature was found over the Creep cycle, where the SCR inlet temperature ranged from approximately 124°C to 174°C. At those lower temperatures, the SCR would not be reducing NOx emissions as effectivity and the denominators in term of g/mi and g/bhp-hr would be very low, so that is the cycle where the highest g/mi NOx emissions were observed.

Additional comparisons between NOx emissions and SCR inlet temperatures are provided in Figure 3-6, Figure 3-7, and Figure 3-8. These Figures show the average SCR inlet temperature for the UDDS, the HHDDT-S, and the all cycles except the Creep, respectively. For these figures the data from the manufacturer D truck have been removed, since it is an outlier, and no SCR temperature data is available for the manufacturer A2 truck over the HHDDT-S. The Creep data are eliminated from Figure 3-8, since the temperature for the Creep cycle are all below those needed for the urea dosing. Linear regression lines are shown in Figure 3-6 and Figure 3-7 for the UDDS and HHDDT-S cycles, respectively. For the UDDS cycle, these results are consistent with the expected trends for NOx emissions as a function of SCR inlet temperature. NOx

emissions are generally found to decrease as the SCR inlet temperature goes from approximately 200 to 250°C. For the HHDDT-S cycle, NOx emissions are very low as the average SCR temperature is well above 250°C. The minor variations in NOx emissions shown on the HHDDT-S chart are likely due to factors other than SCR temperature, as the SCR temperatures are already well above the optimal temperatures needed for good SCR conversion efficiencies.

Figure 3-6. Average SCR Inlet Temperature vs NOx emissions over UDDS

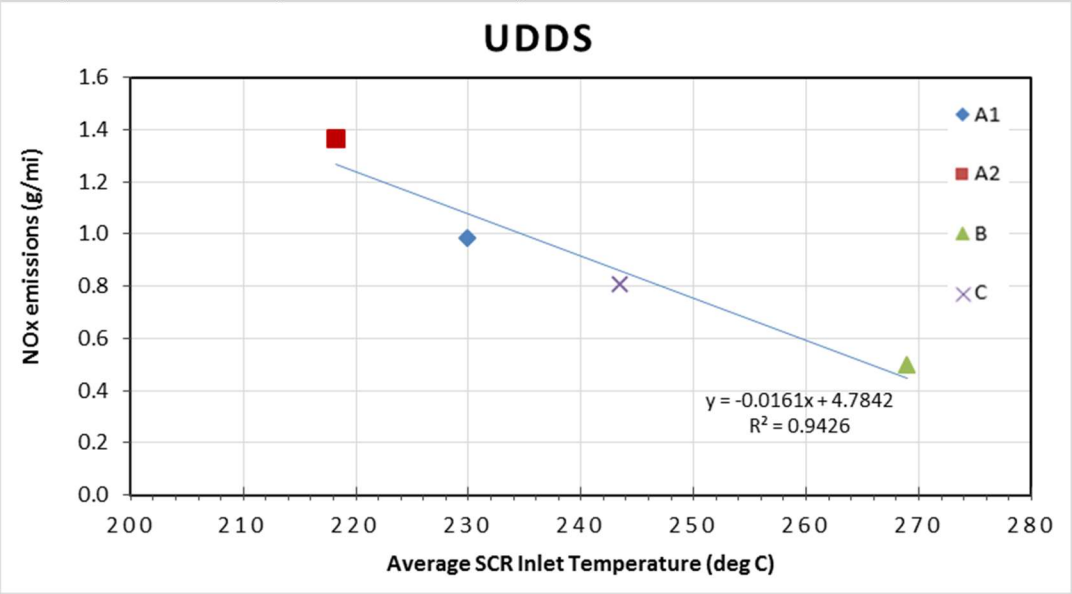


Figure 3-7 Average SCR Inlet Temperature vs NOx emissions over HHDDT-S

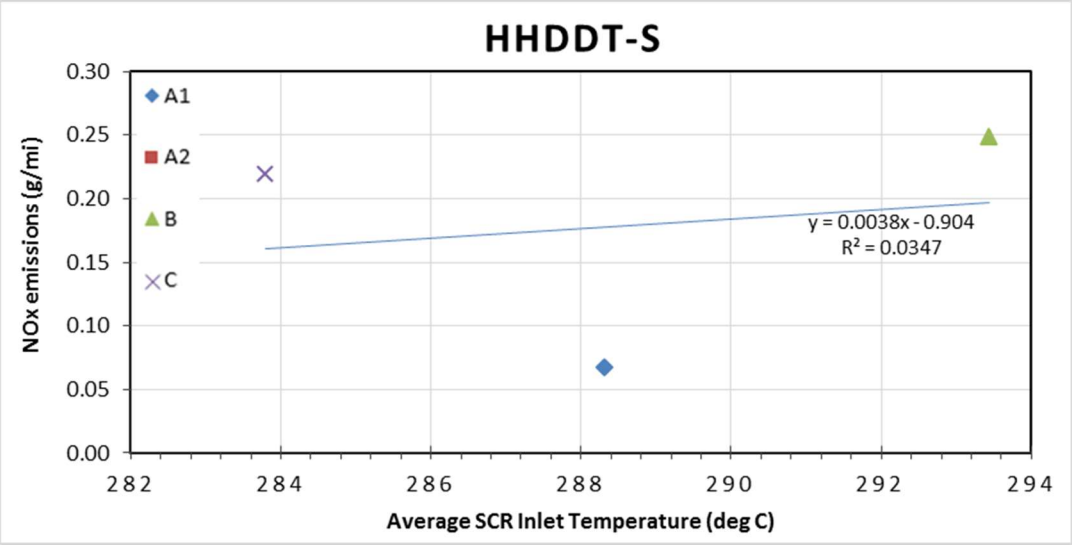
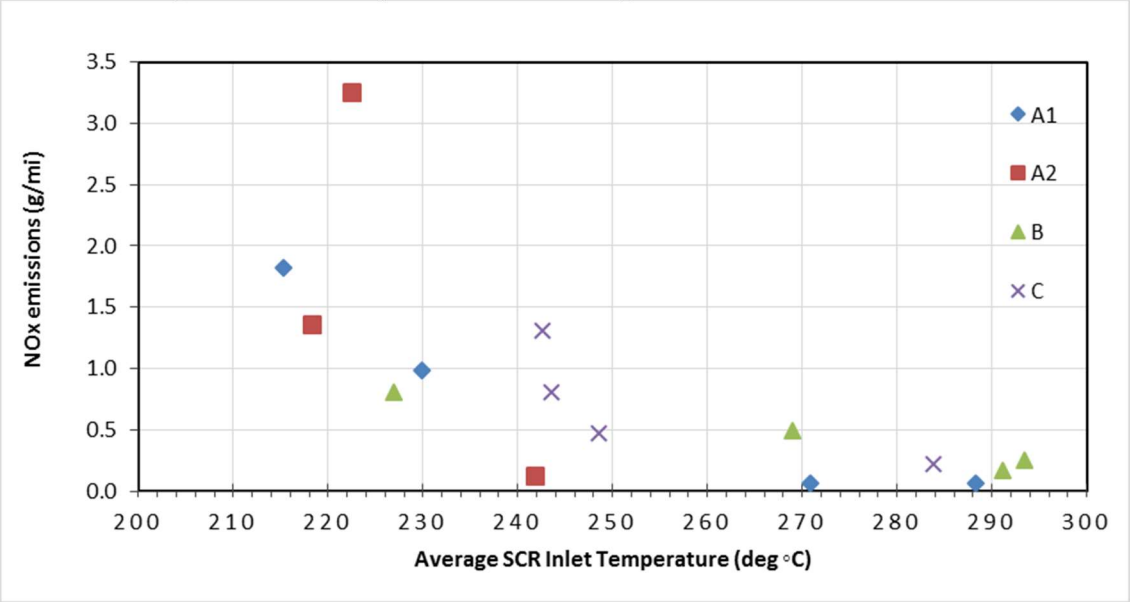


Figure 3-8 Average SCR Inlet Temperature vs NOx emissions



Plots of real-time cumulative NOx emissions over the UDDS are provided in Figure 3-9 to Figure 3-13, along with the corresponding exhaust temperatures measured before the aftertreatment system, before the DPF, and before the SCR. The cumulative NO_x emission show that NO_x emissions for the UDDS cycles are primarily formed during the second and third peaks for the cycle (see Appendix B). At that point, the vehicles are accelerating, while at the same time the exhaust temperature is below 250°C, which is below the temperature where the SCR provides more effective reduction of NOx emissions.

Figure 3-9. Real-time NOx Emissions of UDDS for Manufacturer A1 (201411140659)

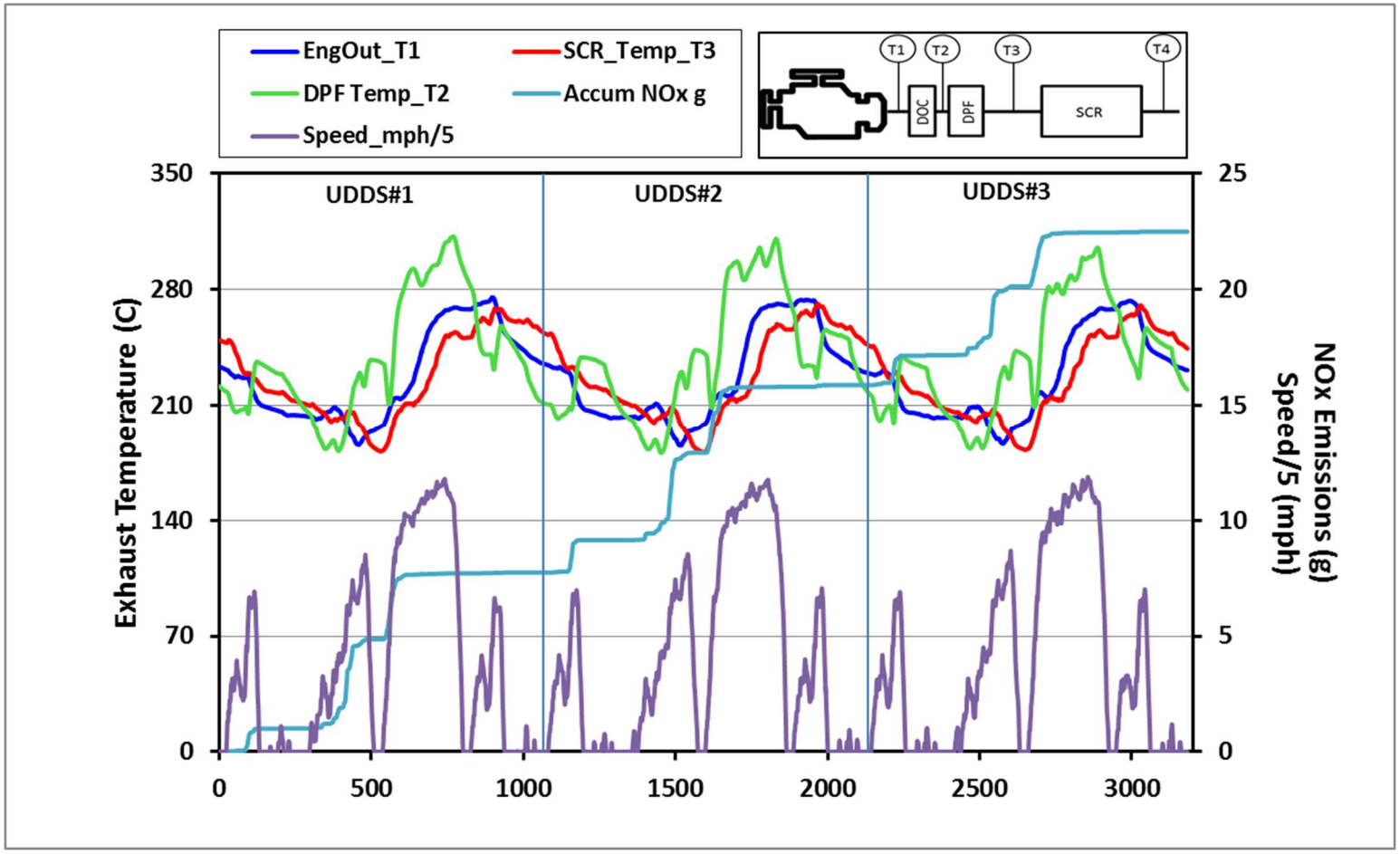


Figure 3-10. Real-time NOx Emissions of UDDS for Manufacturer A2 (201509300820)

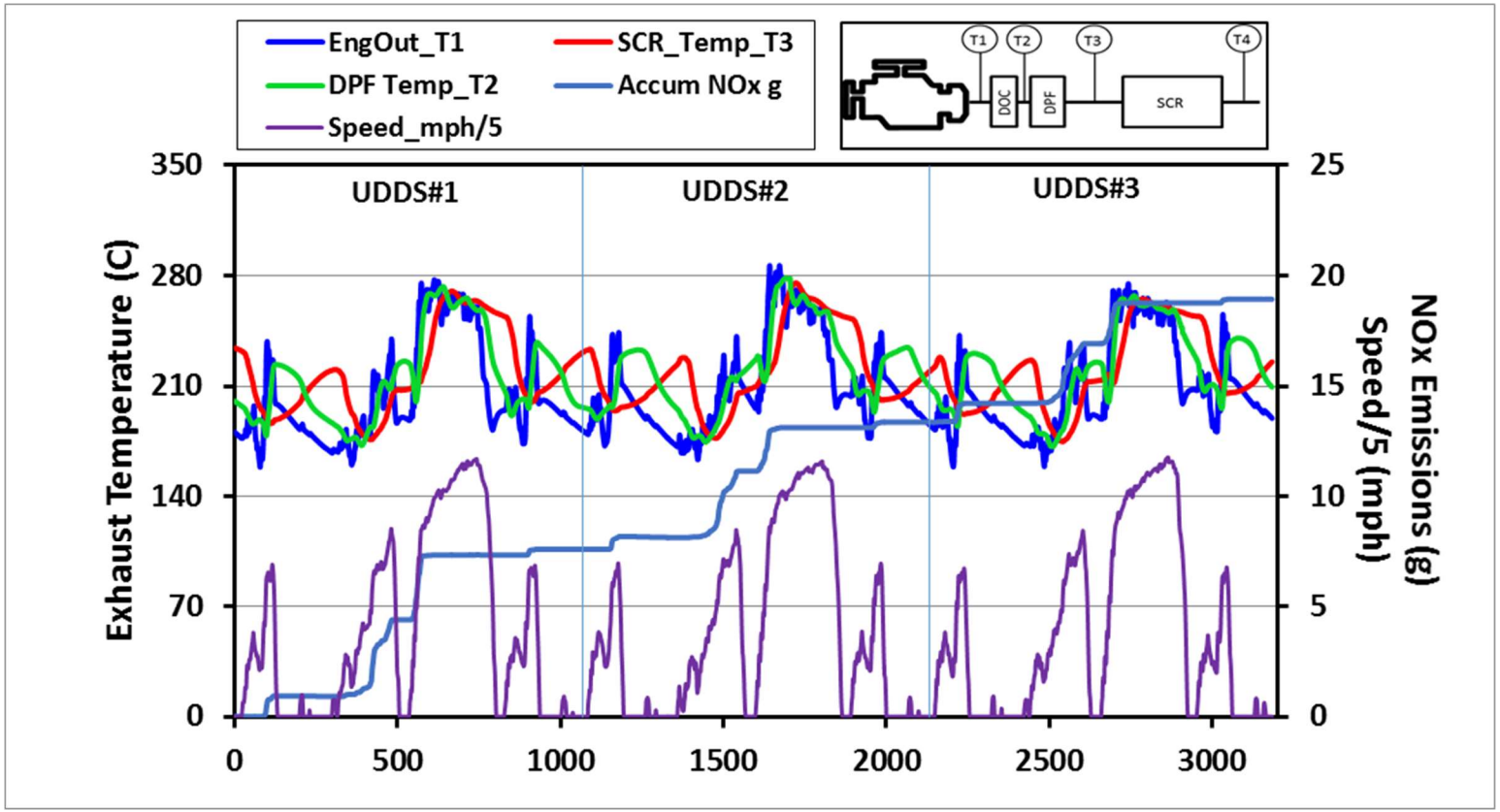


Figure 3-11. Real-time NOx Emissions of UDDS for Manufacturer B (201501140658)

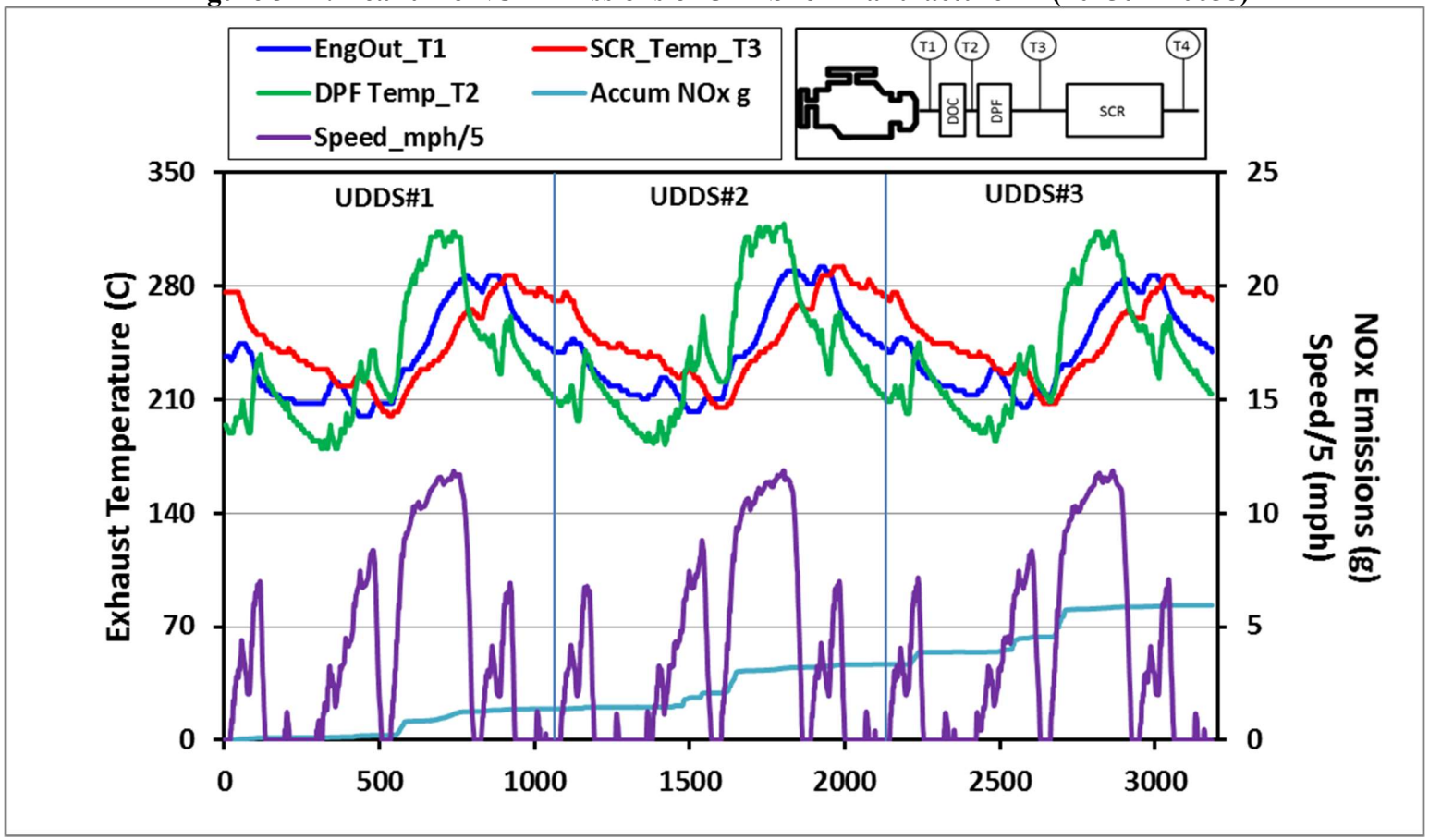


Figure 3-12. Real-time NOx Emissions of UDDS for Manufacturer C (201412180709)

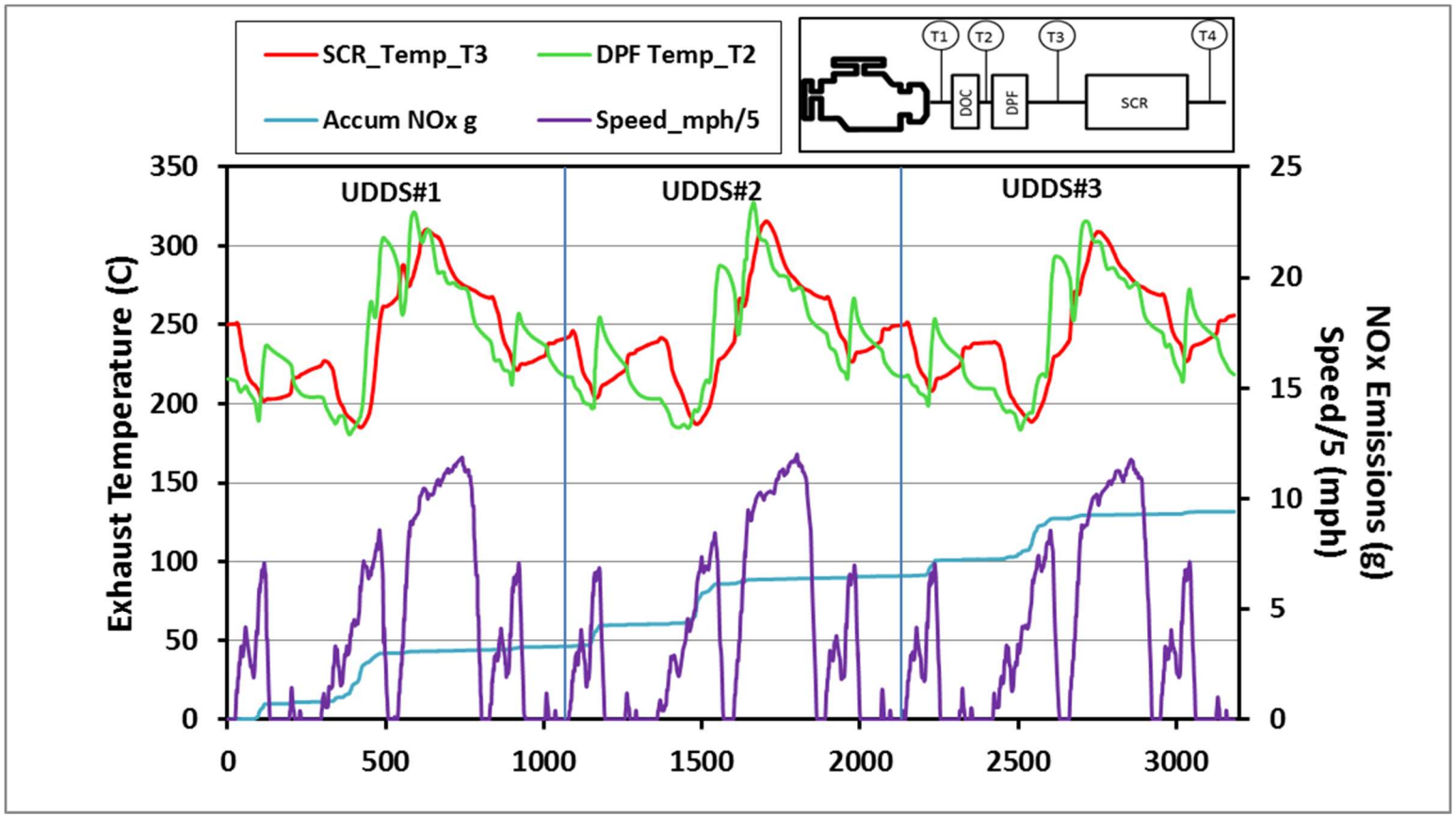
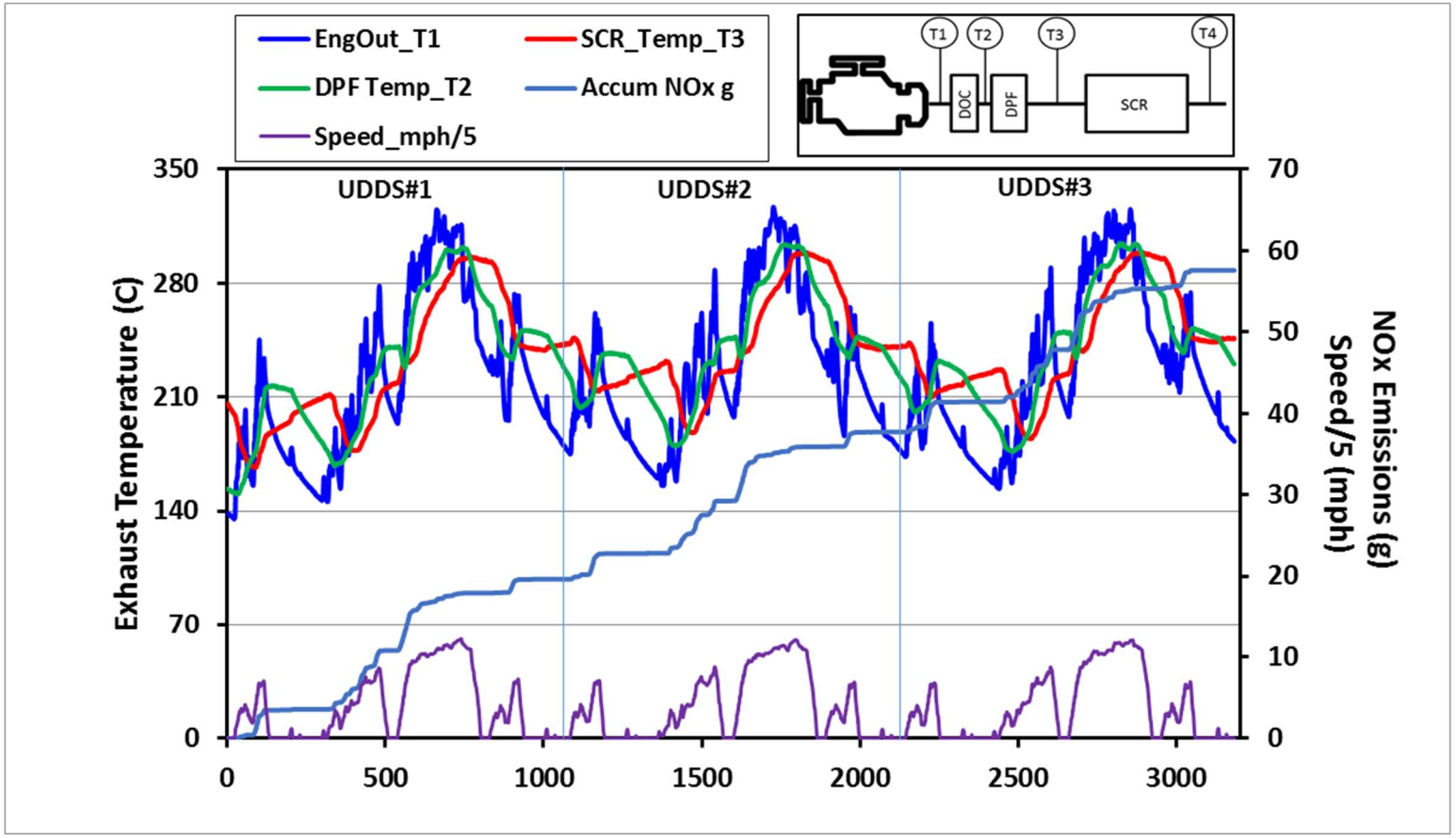


Figure 3-13. Real-time NOx Emissions of UDDS for Manufacturer D (201502100726)



3.2 PM Mass Emissions

Measured PM mass emissions for the test trucks are shown on a g/mi and g/bhp-hr basis in Figure 3-14 and Figure 3-15, respectively.

PM mass emissions were very low for most of the test cycles. PM emissions were below 0.015 g/mi and 0.006 g/bhp-hr for all vehicles over all cycles, except for the manufacturer C truck over the HHDDT-S cycle and the manufacturer A2 truck over the Creep cycle. The PM levels are significantly below the 0.01 g/bhp-hr PM standard under all test conditions, except the manufacturer C truck over the HHDDT-S cycle.

Figure 3-14. Average PM Mass Emissions on a g/mi Basis

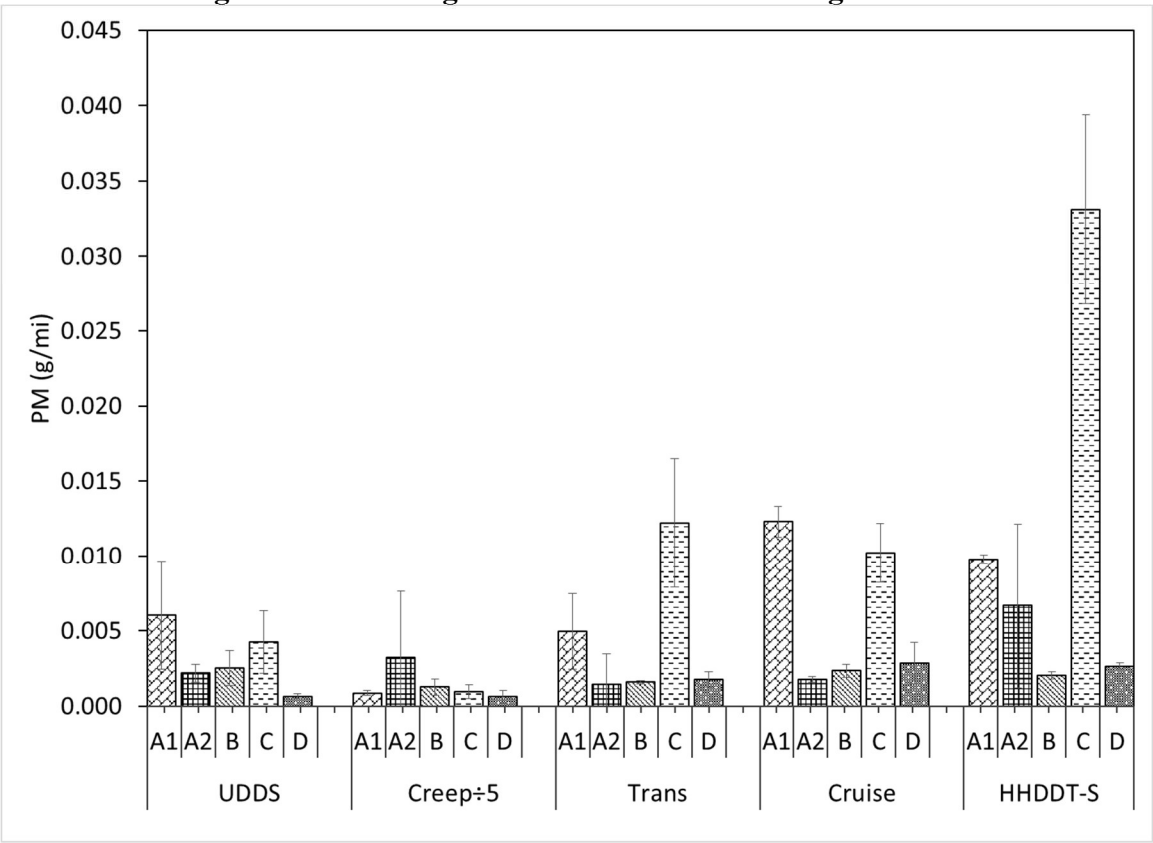
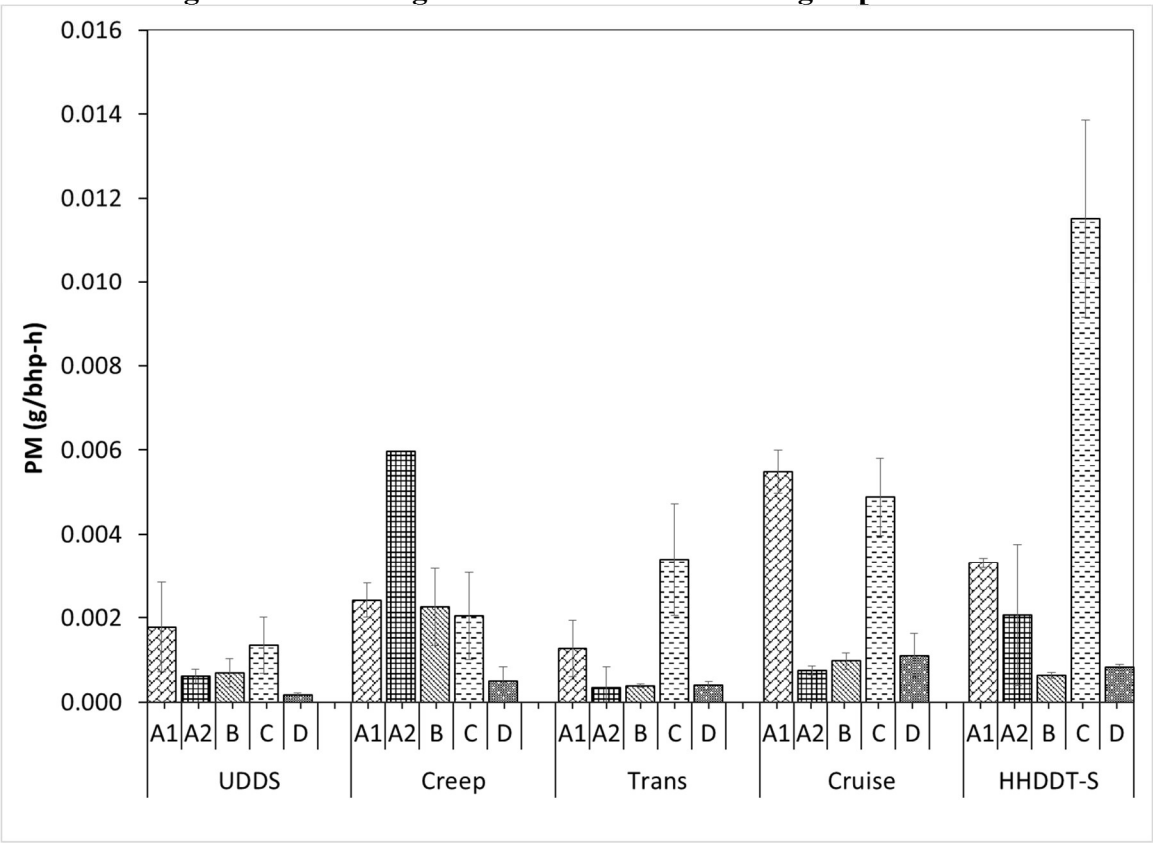


Figure 3-15. Average PM Mass Emissions on a g/bhp-hr Basis



The very low PM levels are consistent with the results of previous studies. For the previous ARB EMFAC2014 study, PM emission rates were below 0.015 g/mi for most vehicle/cycle combinations as well, although three vehicle/cycle points did show PM emission rates ranging from 0.028 to 0.055 g/mi (California Air Resources Board, 2015a, 2015b). In the earlier UCR-SCAQMD study (Miller et al., 2013) of goods movement trucks, PM emissions were ≤0.002 g/mi for most vehicle/cycle combinations, although there were a few vehicle/cycle combinations above 0.002 g/mi for some of the 2010+ vehicles on the Regional and near-dock drayage cycles. Carder et al. (2014) found PM emissions of <0.010 g/mi for a 2010+ SCR-equipped truck over a range of cycles including a UDDS, and near-dock, local, and regional drayage cycles.

3.3 THC Emissions

THC emissions for the test trucks are shown on a g/mi and g/bhp-hr basis in Figure 3-16 and Figure 3-17, respectively.

As expected, THC emissions were very low for most of the test cycles, due to the presence of the DPF/SCR aftertreatment system, and are comparable to background levels in some cases, as indicated by the negative values or the error bars extending below zero for some tests. THC emissions were below 0.034 g/mi and 0.011 g/bhp-hr for all test vehicles over the UDDS, Transient, Cruise, and HHDDT-S cycles, and were below 0.458 g/mi and 0.220 g/bhp-hr for all vehicles over all test cycles. The Creep cycle did show considerably higher THC emissions on a per-mile basis, ranging from 0.241 to 0.458 g/mi, and from 0.015 to 0.046 g/bhp-hr, due its short, low-speed accelerations and longer idle periods.

In comparison with other studies, the ARB EMFAC2014 study showed HC emissions that were below 0.050 g/mi for many vehicle/cycle combinations, although some vehicle/cycle points ranged from 0.117 to 1.442 g/mi, and one vehicle had an emission rate of 2.740 g/mi over the Creep cycle (California Air Resources Board, 2015a, 2015b). In the UCR-SCAQMD study (Miller et al., 2013), THC emission rates over the UDDS were 0.030 g/mi or less for all diesel trucks, except for one 2011 model year manufacturer C truck. Carder et al. (2014) found NMHC emissions of ≤0.005 g/mi for a 2010+ SCR-equipped truck over a range of cycles including a UDDS, and near-dock, local, and regional drayage cycles.

Figure 3-16. Average THC Emissions on a g/mi Basis

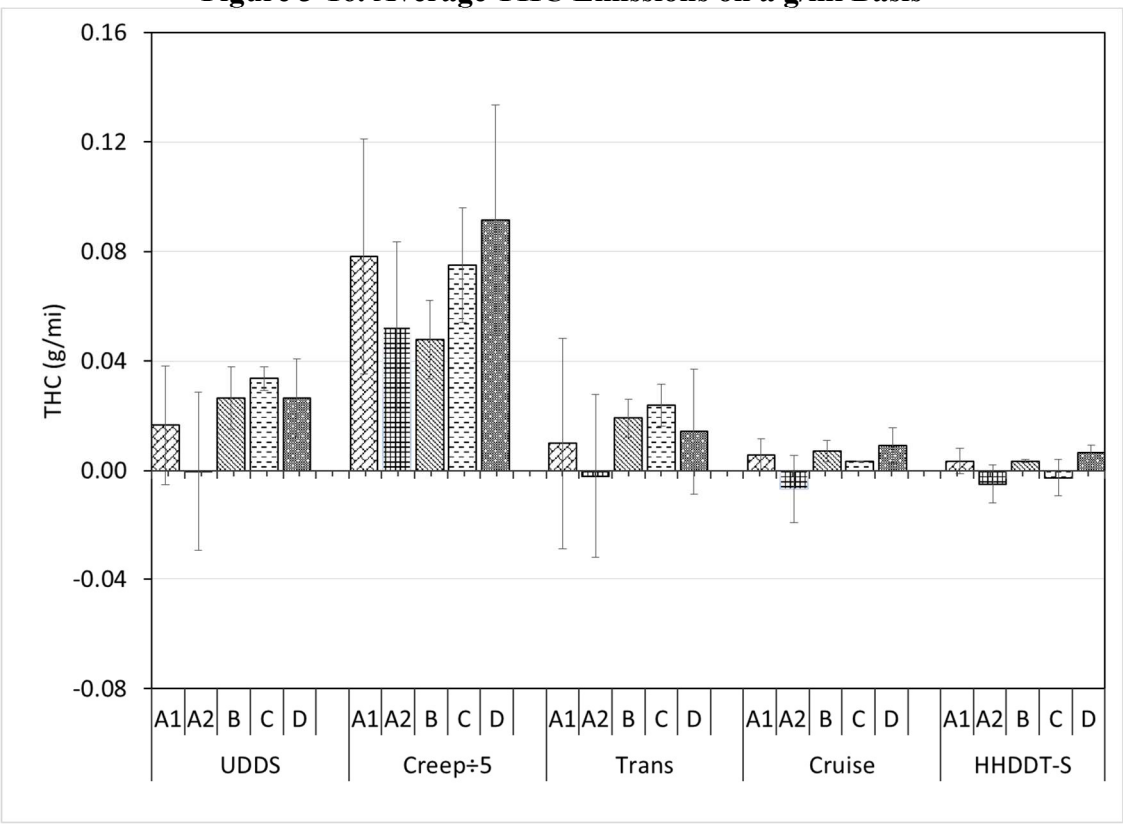
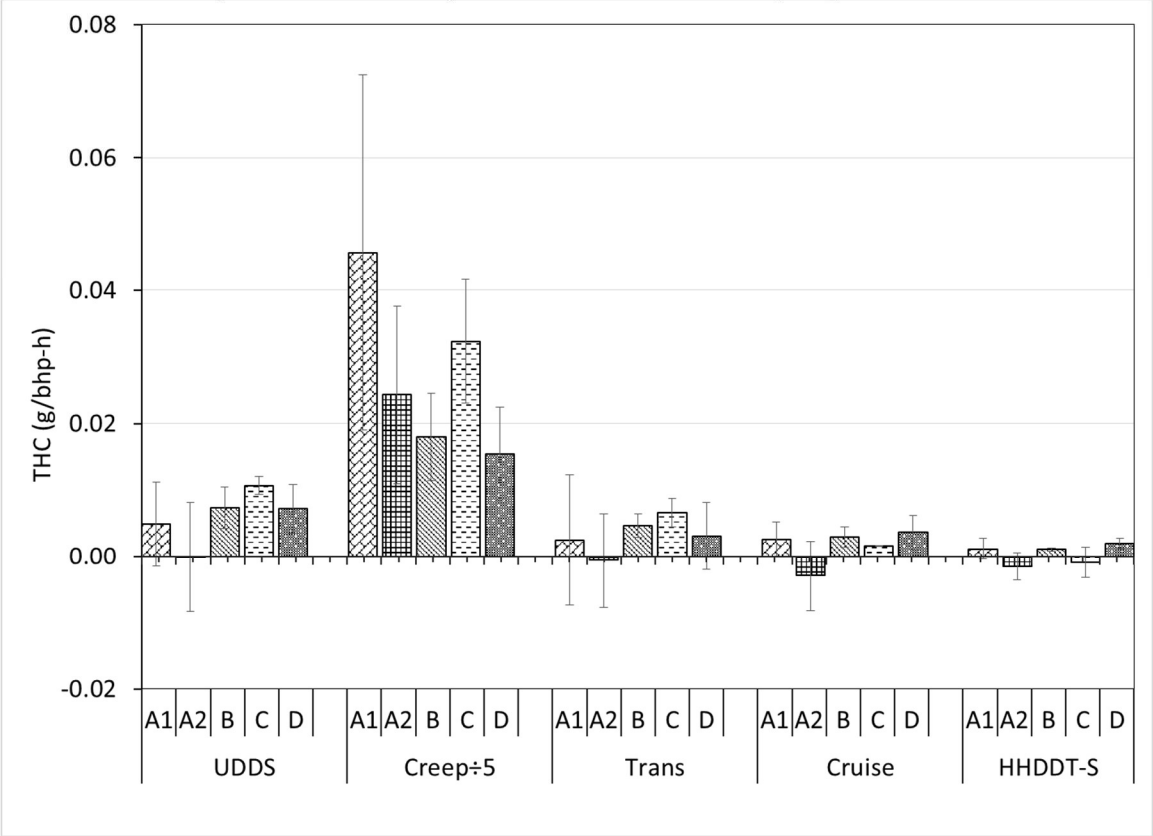


Figure 3-17. Average THC Emissions on a g/bhp-hr Basis



3.4 CO Emissions

CO emissions for the test trucks are shown on a g/mi and g/bhp-hr basis in Figure 3-18 and Figure 3-19, respectively.

CO emissions were very low for most of the test cycles. CO emissions were below 0.2 g/mi for all vehicles over all cycles, except over the Creep cycle and the manufacturer D truck over the Transient cycle. Similarly, on a g/bhp-hr basis, CO emissions were below 0.06 g/bhp-hr for all vehicles, except over the Creep cycle. Emissions over the Creep cycle ranged from 0.004 to 5.042 g/mi and from 0.001 to 1.011 g/bhp-hr. Overall, the CO emission rates were considerably below the 15.5 g/bhp-hr and 14.0 g/bhp-hr standards established by EPA and ARB, respectively, for all vehicles and cycles.

Figure 3-18. Average CO Emissions on a g/mi Basis

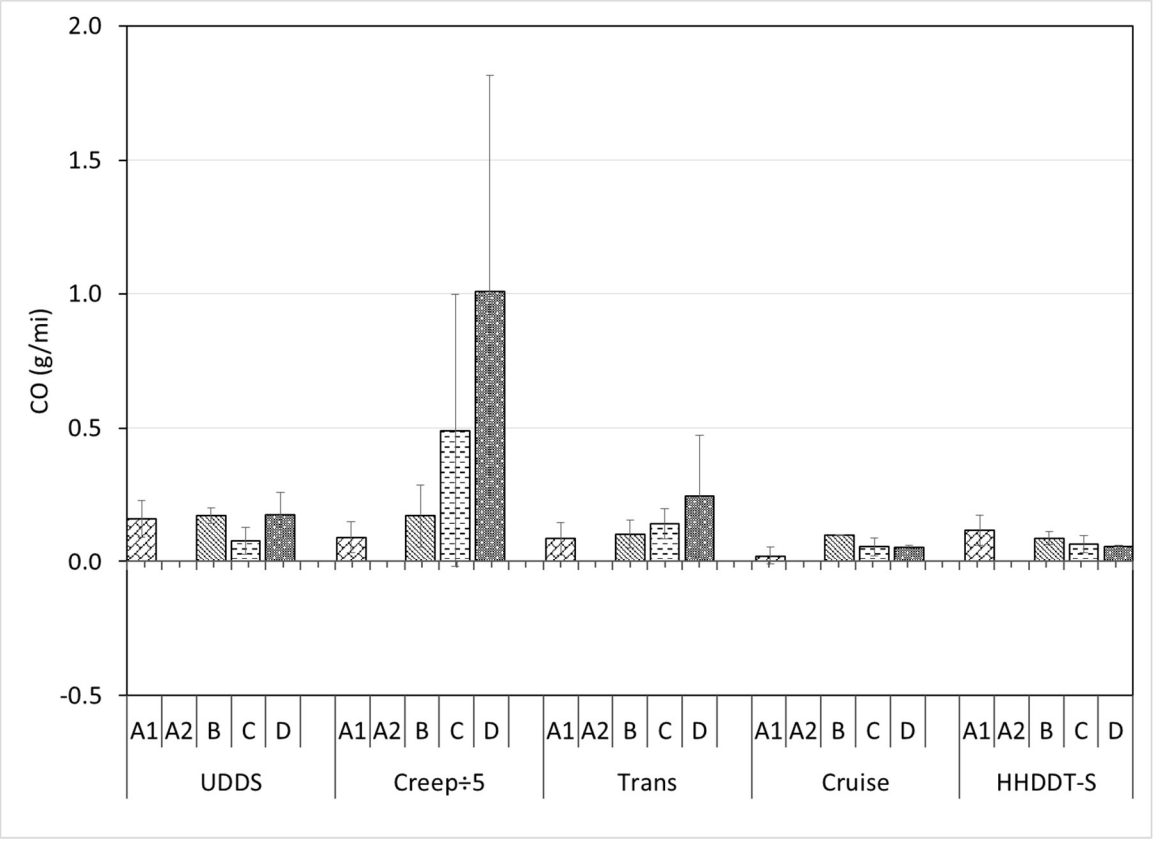
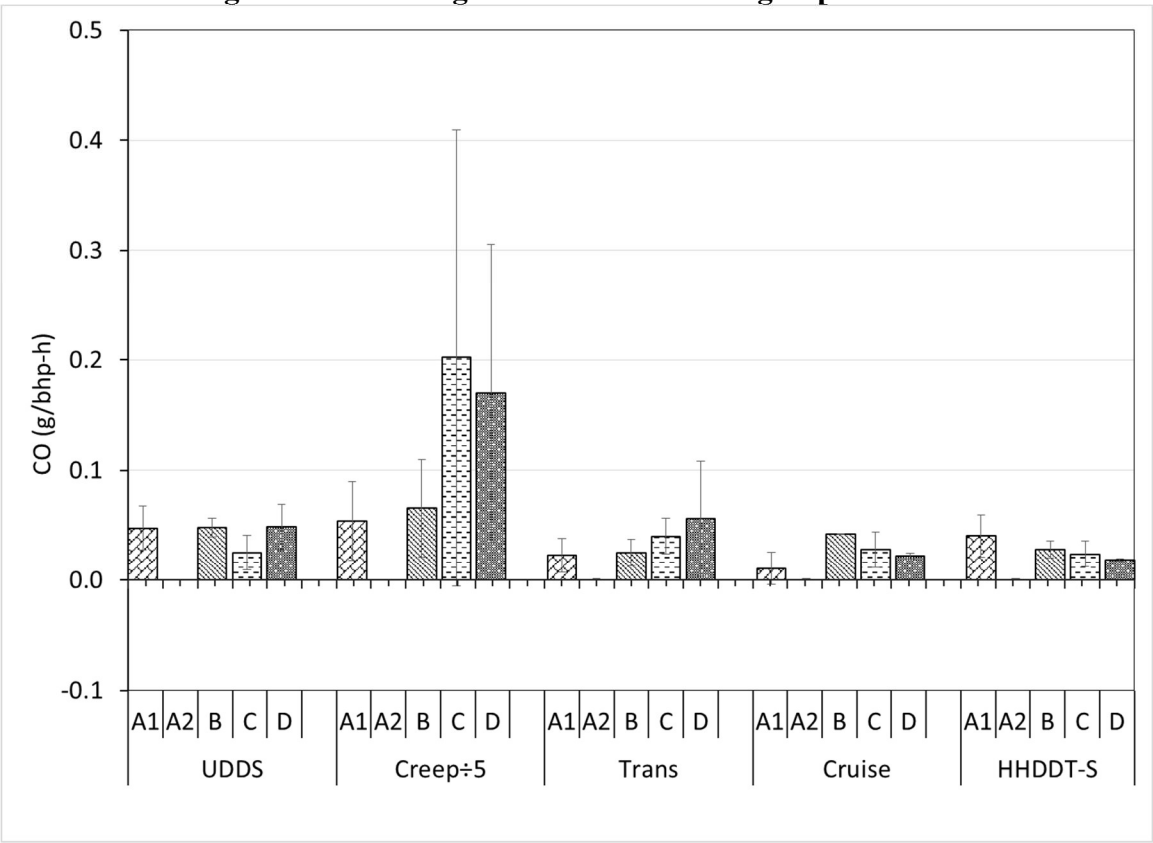


Figure 3-19. Average CO Emissions on a g/bhp-hr Basis



In comparison with other studies, the ARB EMFAC2014 study showed that CO emissions were below 0.500 g/mi for a majority of the vehicle/cycle combinations, although some vehicle/cycle points ranged from 1.64 to 4.75 g/mi, and one vehicle had an emission rate of 9.09 g/mi over the Creep cycle (California Air Resources Board, 2015a, 2015b). In the UCR-SCAQMD study (Miller et al., 2013), CO emission rates over the UDDS were 0.064 g/mi or below for all diesel trucks, except for one 2011 model year manufacturer C truck, with many of those levels being at or below the background level. Carder et al. (2014) found CO emissions of 0.216, 0.749, 0.169, and 0.854 g/mi for a 2010+ SCR-equipped truck over UDDS, and near-dock, local, and regional drayage cycles, respectively.

3.5 Fuel Economy/Consumption and CO_2 Emissions

CO_2 emissions for the five test trucks are shown on a g/mi and a g/bhp-hr basis in Figure 3-20 and Figure 3-21, respectively.

CO_2 emissions over the UDDS cycles ranged from 1864 to 2219 g/mi and 548 to 678 g/bhp-hr. CO_2 emissions over the Transient cycle were similar to those over the UDDS, ranging from 2260 to 2624 g/mi and 584 to 724 g/bhp-hr. CO_2 emissions over the Cruise and HHDDT-S cycles were slightly lower on a g/mi basis. For the Cruise cycle, CO_2 emissions ranged from 1160 to 1443 g/mi and from 518 to 589 on a g/bhp-hr basis. For the HHDDT-S cycle, CO_2 emissions ranged from 1450 to 1743 g/mi and from 491 to 572 on a g/bhp-hr basis. CO_2 emissions were highest over the Creep cycle, where loads are lowest, ranging from 3748.7 to 5095.0 g/mi and 816.6 to 2337.4 g/bhp-hr.

Figure 3-20. Average CO_2 Emissions on a g/mi Basis

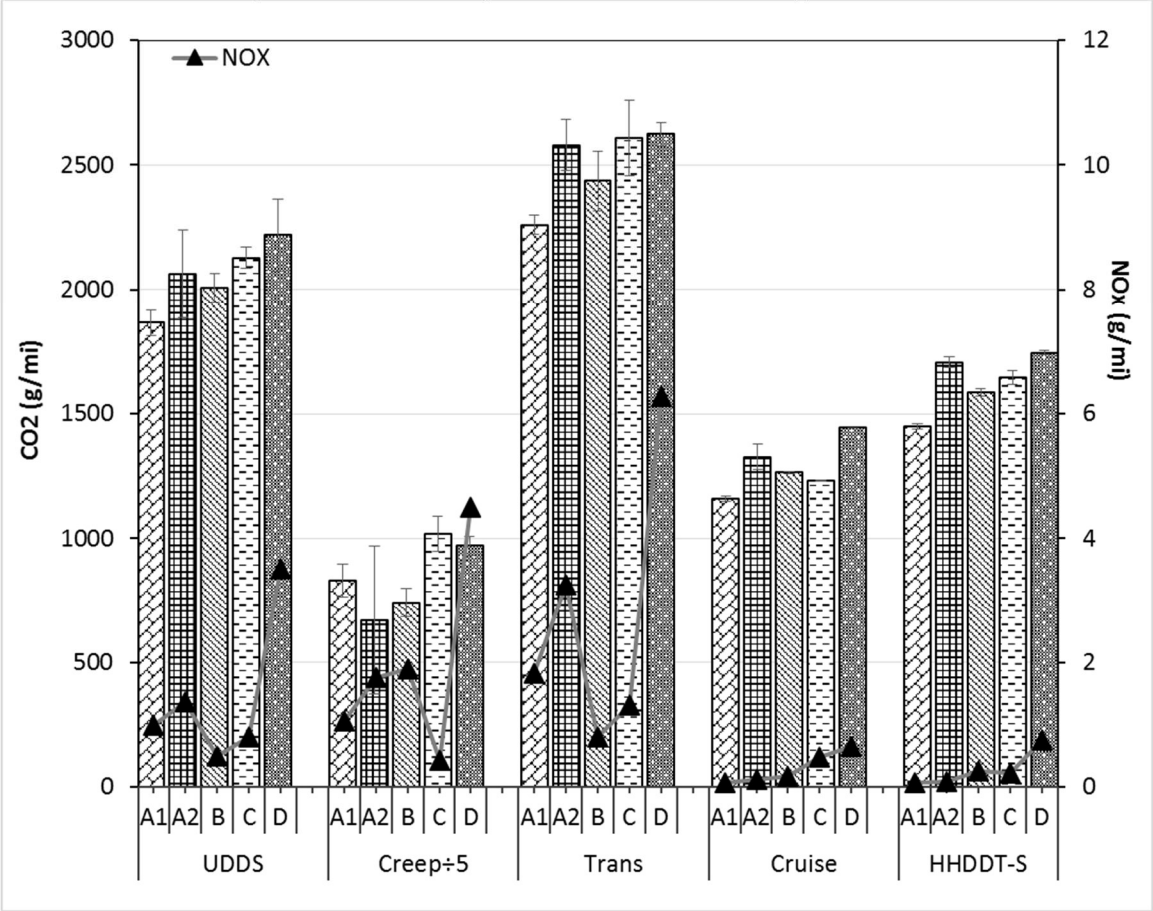
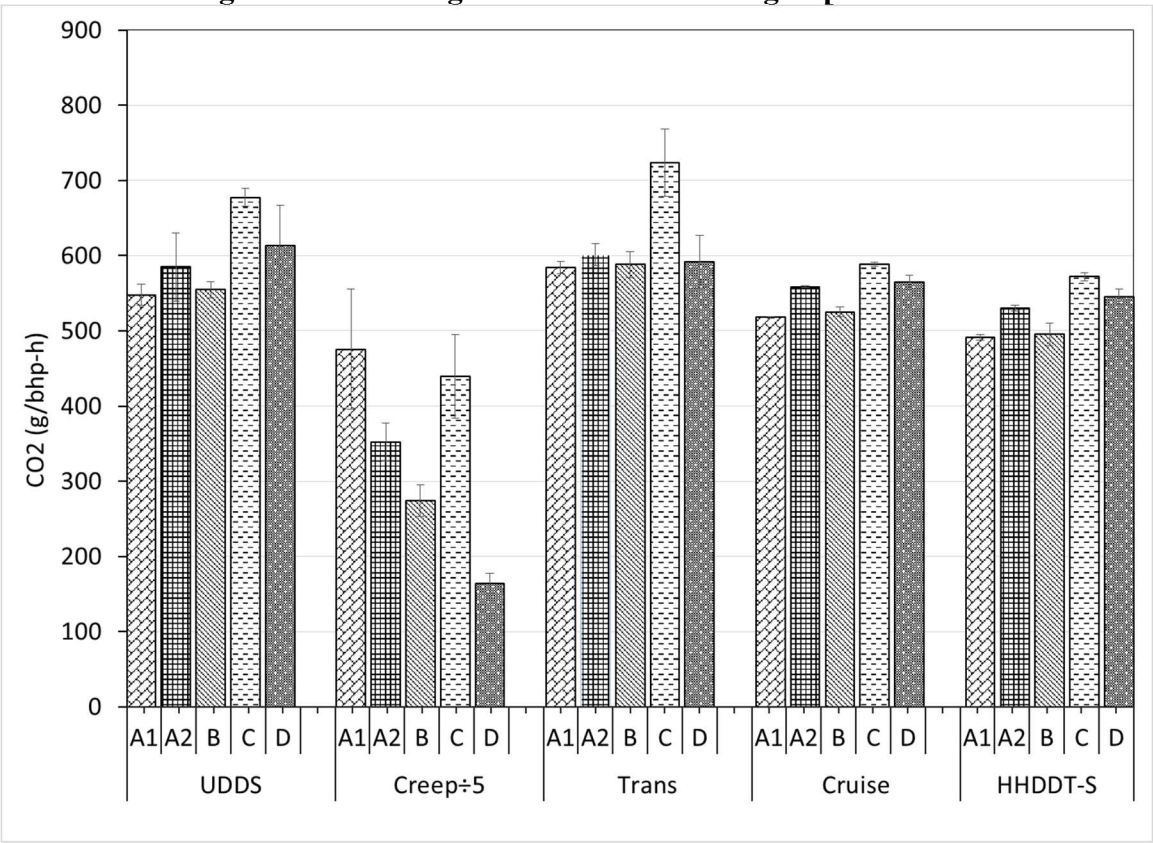


Figure 3-21. Average CO_2 Emissions on a g/bhp-hr Basis



The differences in CO_2 emissions between different vehicles over the same cycle at the same test weight could be more a function of the differences in the dynamometer loading between trucks or differences in the different engine/chassis technologies. Table 3-1 provides a comparison between the chassis dynamometer work in units of bhp-hr and CO_2 emissions in g/mi units. Overall, the results show that the chassis dynamometer work was relatively comparable between different vehicles for the same test cycle, and showed smaller differences than the CO_2 emissions. The biggest differences in the chassis dynamometer work is the lower work for the manufacturer A2 vehicles over the Cruise and HHDDT-S cycles, due to the lack of a roof fairing on the cab, as discussed in section 2.2 and Appendix C.

A closer examination of CO_2 emissions as a function of chassis dynamometer work is provided in Figure 3-22 to 3-25. For those figures, the data for the manufacturer A vehicle have been excluded to provide a closer comparison among the four trucks that had more similar dynamometer loadings. The figures include a dashed line representing the linear regression based on the actual data and a solid line representing the theoretical percentage increase in CO_2 emissions that would be expected for a given percentage increase in work, if the BSFC values for all of the engines were held constant. Interestingly, the slope of the regression for the CO_2 emissions is steeper than what would be expected for a given percentage increase in load, suggesting that the differences in CO_2 emissions for the different trucks over a given cycle are related to more than just simple differences in work performed.

Table 3-1. Comparison of Chassis Dynamometer Work vs. CO_2 Emissions

Cycles	A1		A2		B1		C1		D1	
	Chassis Dyno bhp-hr	CO2 g/mi	Chassis Dyno bhp-hr	CO2 g/mi	Chassis Dyno bhp-hr	CO2 g/mi	Chassis Dyno bhp-hr	CO2 g/mi	Chassis Dyno bhp-hr	CO2 g/mi
UDDS	56.21	1864.9	53.14	2063.4	57.07	2005.7	59.18	2128.3	57.70	2219.0
HHDDT_Creep	2.60	4147.6	2.69	4781.2	3.01	3707.4	3.34	5095.5	2.78	4850.3
HHDDT_Tranient	42.14	2260.1	43.06	2579.9	42.69	2436.2	44.70	2607.3	43.97	2624.7
HHDDT_Cruise	58.60	1160.2	54.45	1326.6	59.70	1264.4	60.68	1232.1	61.01	1443.4
HHDDT-S	66.76	1449.9	59.47	1706.7	68.00	1586.7	70.41	1645.9	68.22	1744.0

Figure 3-22. Correlations of Chassis Dynamometer Work and CO_2 Emissions over the UDDS Cycle

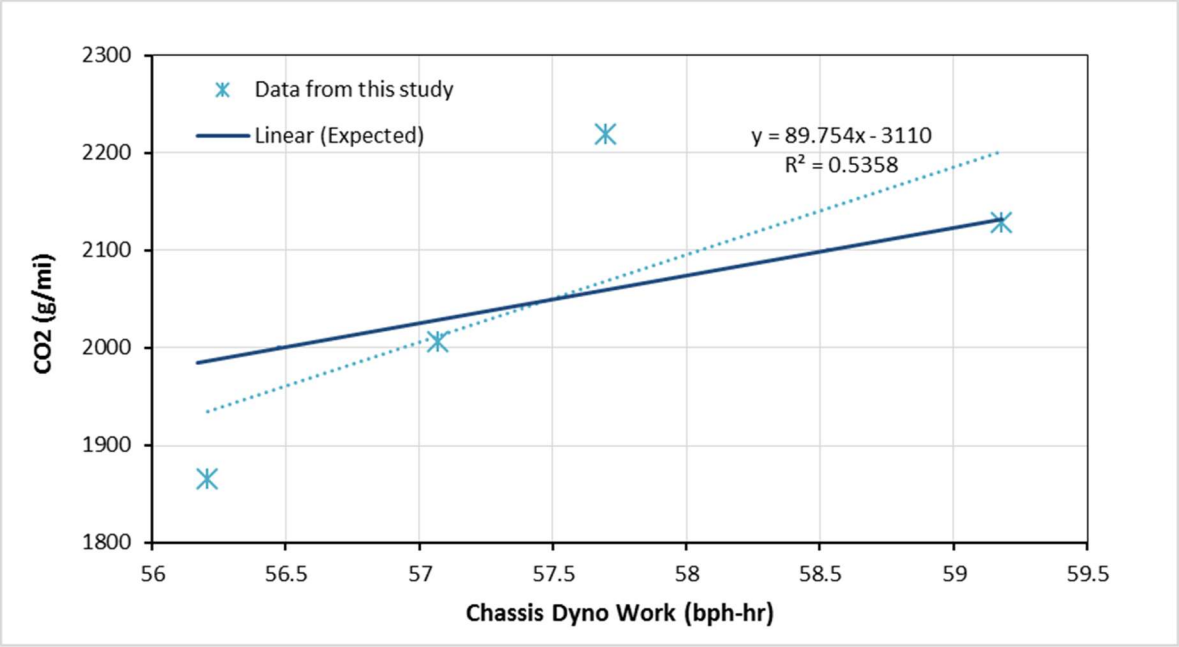


Figure 3-23. Correlations of Chassis Dynamometer Work and CO_2 Emissions over the Transient Cycle

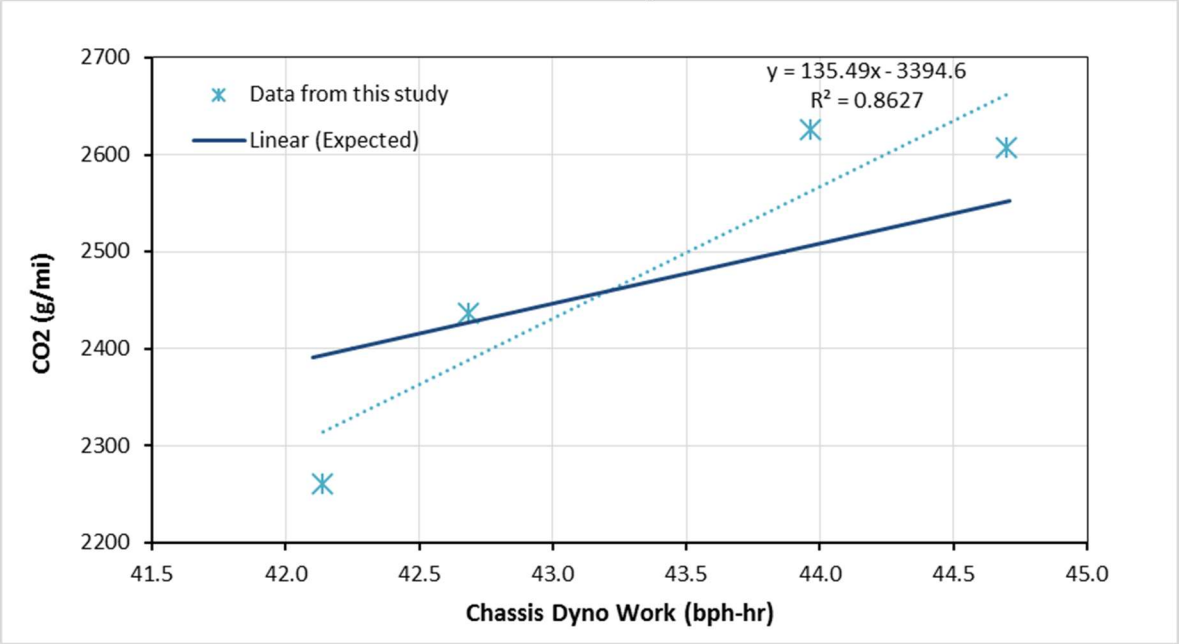


Figure 3-24. Correlations of Chassis Dynamometer Work and CO_2 Emissions over the Cruise Cycle

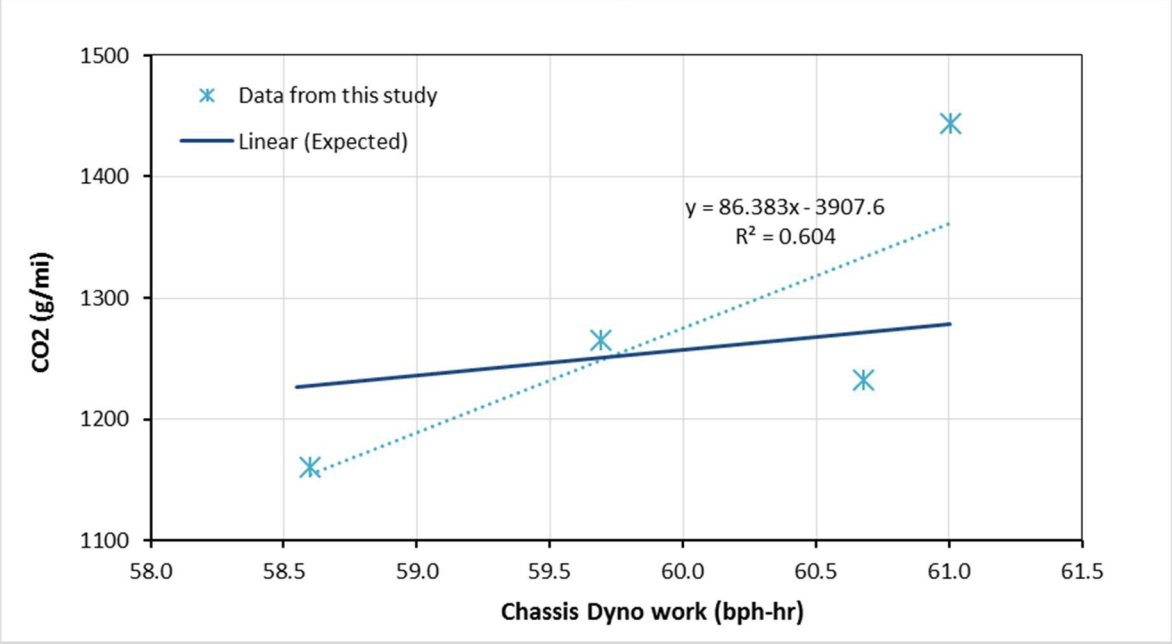
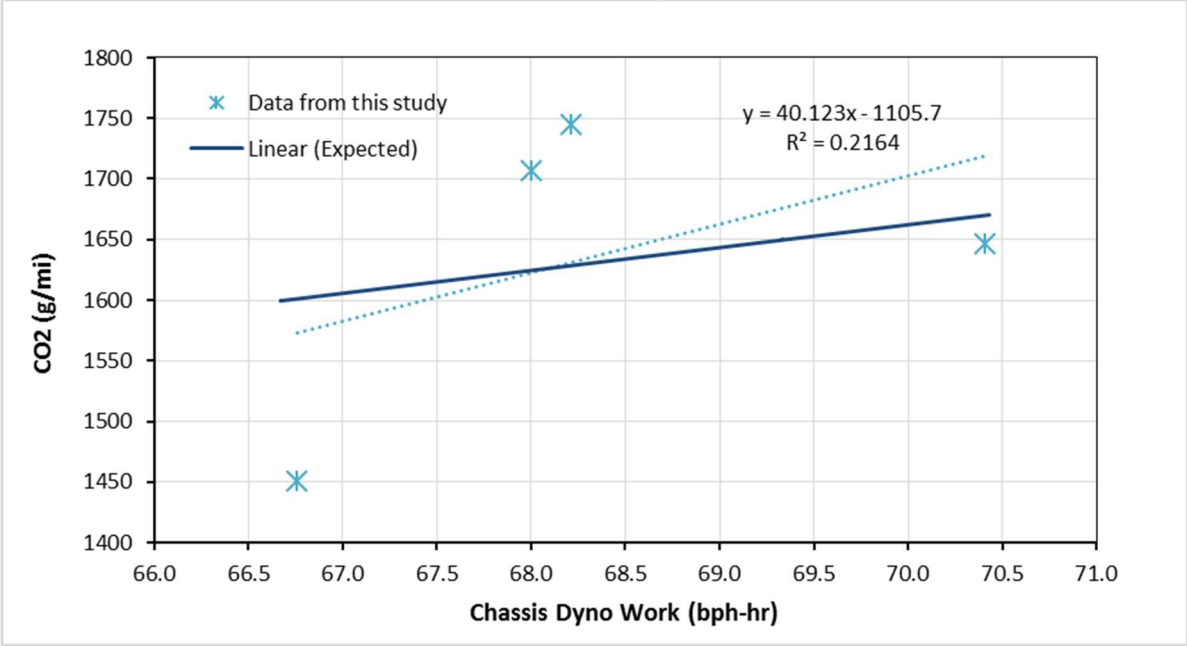


Figure 3-25. Correlations of Chassis Dynamometer Work and CO_2 Emissions over the HHDDT-S Cycle



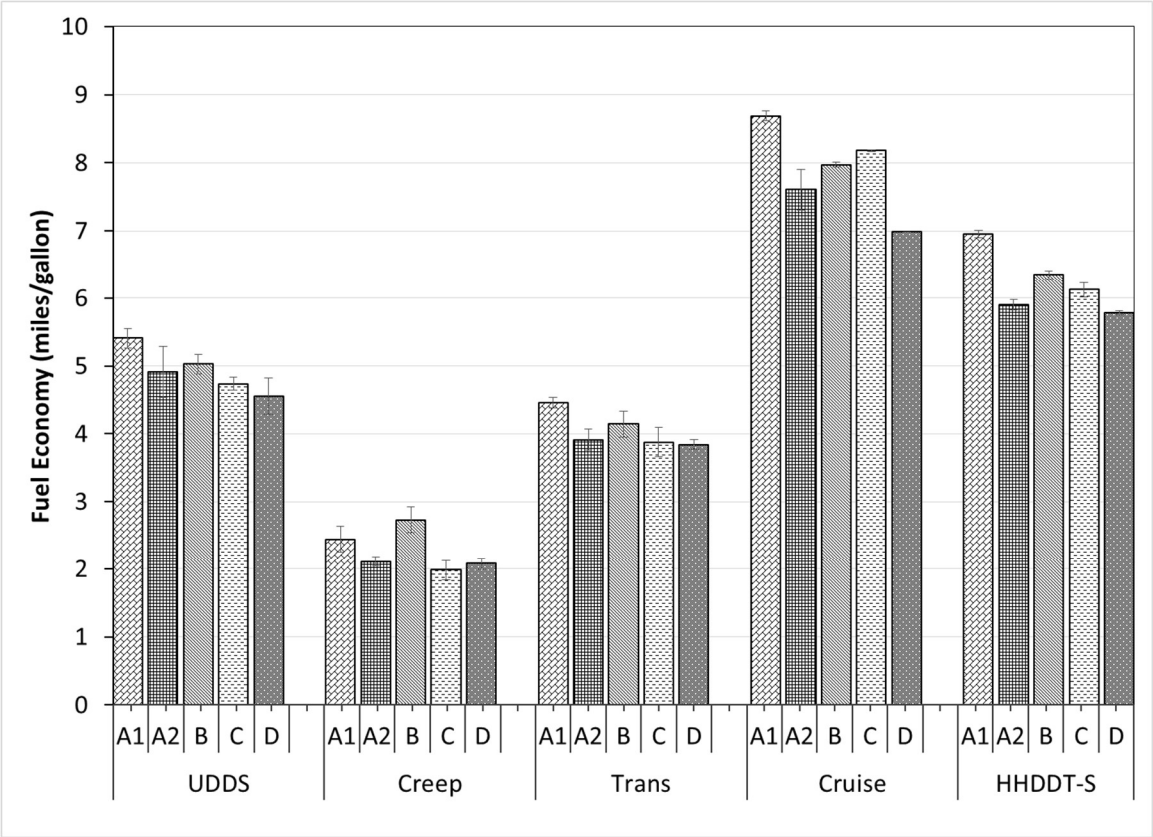
In comparison, CO_2 emissions as measured in the earlier ARB study ranged from 1,831 to 2,964 g/mi over the UDDS, from 2,034 to 2,432 g/mi over the Transient cycle, from 1,014 to 1,558 g/mi over the Cruise cycle, from 1,310 to 1,898 g/mi for the High Speed Cruise cycle, and from 3,805 to 5,006 g/mi over the Creep cycle (California Air Resources Board, 2015a, 2015b). For the previous UCR-SCAQMD study (Miller et al., 2013), CO_2 emissions for the Class 8 diesel trucks ranged from 2,379 to 3,117 g/mi over the hot UDDS cycle. For the previous WVU-SCAQMD study (Carder et al., 2014), CO_2 emissions for 2009 model year and newer Class 8 goods movement diesel trucks ranged from 2,115 to 2,757 g/mi over the UDDS cycle. Note that some of the differences between those various studies could be due to differences in test weight loading, as the ARB study used a weight of 56,000 lbs., the SCAQMD study used a test weight of 69,500 lbs, and the present study used 65,000 lbs. It should also be noted that the range in CO_2 emissions for trucks tested over the same cycle in those two earlier studies is similar to that found in the current study.

For comparison, average NOx emissions for each vehicle/cycle combination are also included in Figure 3-20, as there can be tradeoffs between lowering CO_2 emissions and resultant increases in NO_x emissions. Overall, there do not appear to be significant trends with respect to a CO_2/NO_x emissions tradeoff. In particular, vehicles/engines with lower CO_2 emissions do not necessarily have higher levels of NO_x emissions. Similarly, vehicles/engines with higher NO_x emissions do not exhibit significantly lower CO_2 emissions.

Fuel economy for the five test trucks is shown in Figure 3-26. Fuel economy was similar over the UDDS and Transient cycles. Fuel economy over the UDDS ranged from 4.56 to 5.41 g/mi, while fuel economy over the Transient ranged from 3.84 to 4.46 mi/gal. Fuel economy over the Cruise and HHDDT-S cycles was slightly better, ranging from 6.98 to 8.69 mi/gal for the Cruise cycle, and from 5.78 to 6.95 mi/gal for the HHDDT-S cycle. The lowest fuel economy was found over

the Creep cycle, and ranged from 1.978 to 2.689 mi/gal., due to the slow speeds and stop-and-go nature of the cycle. Again, it should be noted that the differences in fuel economy between different vehicles for the same cycle at the same test weight could be more a function of the differences in the dynamometer loading between trucks due to different frontal areas, as opposed to differences in engine technologies/manufacturers.

Figure 3-26. Average Fuel Economy on a miles/gallon Basis



3.6 Idle Emissions Results

Idle emissions in g/hr units are provided in Table 3-2. For NO_x, idle emission levels varied from 4.7 to 21 g/hr. The manufacturer D truck showed the highest idle NOx emissions, consistent with its higher overall emissions over the other test cycles. PM emissions were relatively low at idle, ranging from background levels to 0.008 g/hr. THC, NMHC, CH_4, and CO idle emissions ranged from 0.09 to 1.37 g/hr, from 0.04 to 0.94 g/hr, from background levels to 0.57 g/hr, and from 0.10 to 6.79 g/hr, respectively. CO_2 idle emissions ranged from 3,552 to 6,820 g/hr.

Idle NOx emissions in EMFAC11 and EMFAC2014 are 30 g/hr and 12 g/hr, respectively, for 2010 and newer model year vehicles, with our results suggesting that EMFAC2014 represents a more realistic value. PM idle emissions in EMFAC11 and EMFAC2014 are 0.072 g/hr and 0.001 g/hr, respectively, for 2010 and newer vehicles, with the EMFAC2014 values being closer to the values found in our study. CO_2 idle emissions in EMFAC11 and EMFAC2014 are 4934 g/hr and 4547 g/hr, respectively, for 2010 and newer vehicles.

The idle emission values observed in this study are generally lower than those that have been found in previous studies of older vehicles. Khan et al. (2006) reviewed idle emissions from 2005 and older engines. They found that idle NO_x emissions varied from near 0 g/hr to 200 g/hr, with a majority of the vehicles equipped with diesel engines and electronic fuel injection having idle emissions between 40 to 160 g/hr. They found that average idle NO_x emissions ranged between 20 and 26 g/hr with the EGR on, but jumped up to 80–90 g/hr when the EGR valve was shut off. PM idle emissions for the electronically controlled engines were typically below 3 g/hr, with PM idle emissions for many of the later generation engines/vehicles (as of 2005) being near the detection limits.

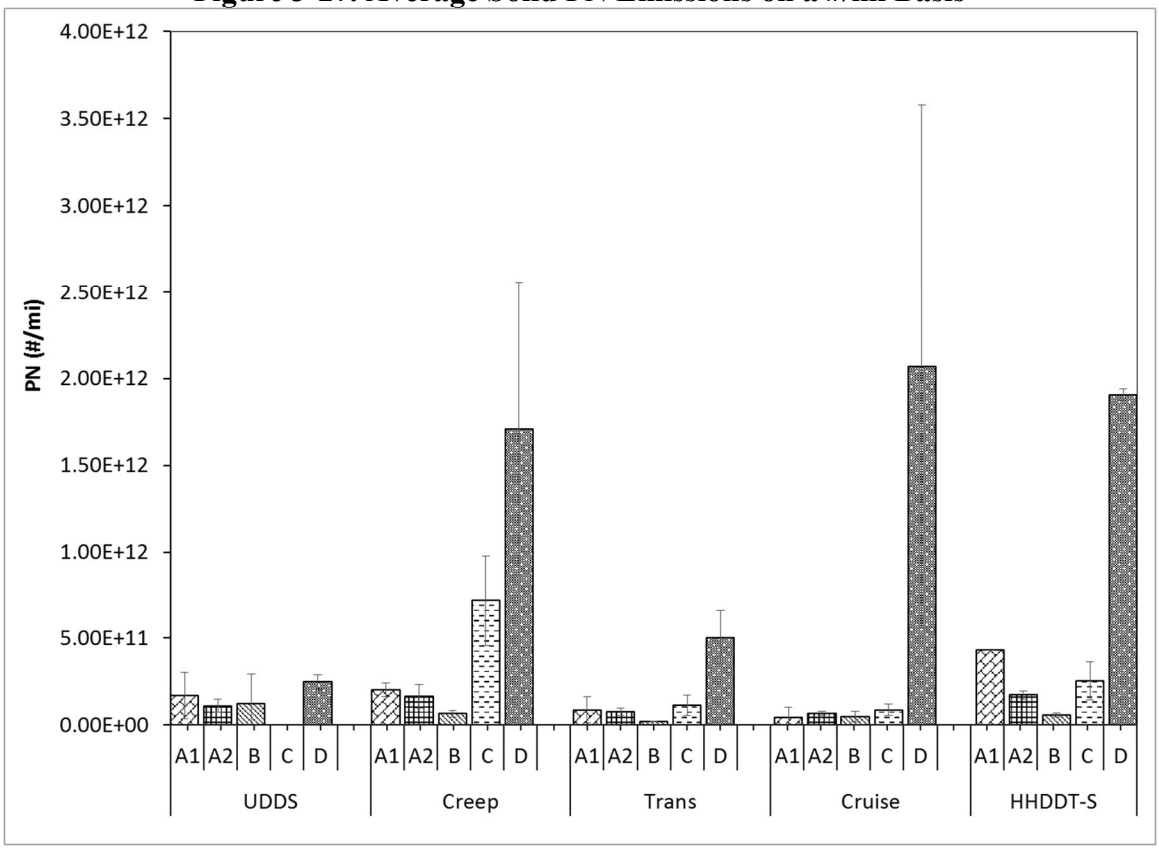
Table 3-2. Idle Emissions

	THC		CH_4		NMHC		CO		NOx		CO_2		NH_3		PM	
	g/hr															
Trace	Ave	Stdev	Ave	Stdev	Ave	Stdev	Ave	Stdev	Ave	Stdev	Ave	Stdev	Ave	Stdev	Ave	Stdev
A1	0.09	0.19	-0.01	0.12	0.10	0.11	3.73	4.87	4.67	5.66	5453	586	0.02	0.07	0.008	0.004
A2	0.65	0.49	0.26	0.19	0.42	0.44	0.10	0.08	12.38	8.82	6820	291	0.43	0.19	0.005	0.013
B	0.43	0.10	0.11	0.05	0.33	0.12	3.97	3.28	11.13	9.38	3552	72	0.27	0.13	0.007	0.004
C	1.37	0.93	0.18	0.03	1.23	0.94	3.14	2.85	5.89	5.54	4466	346	0.26	0.05	0.005	0.003
D	0.54	0.24	0.57	0.24	0.04	0.41	6.79	5.40	20.99	10.10	4856	299	0.39	0.21	-0.002	0.001

3.7 Solid Particle Number Emissions

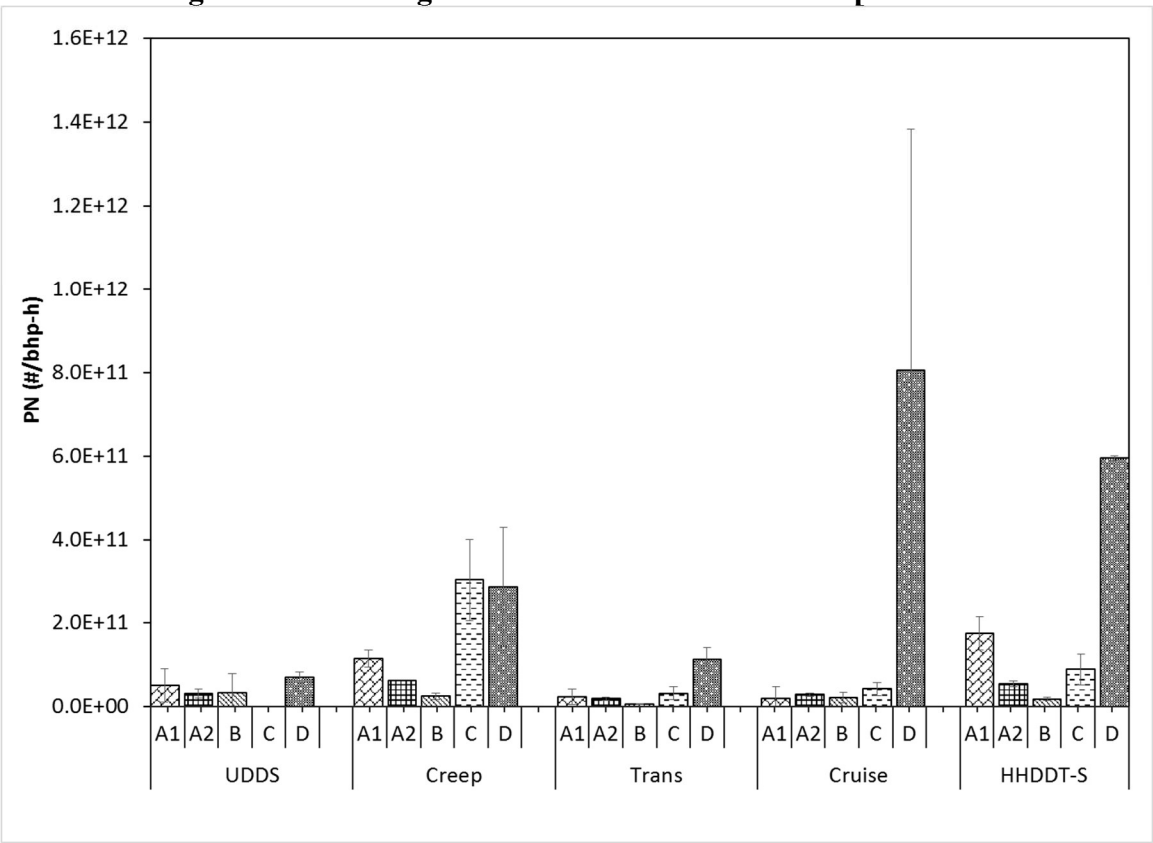
Solid PN emissions for the test trucks are shown on a #/mi and #/bhp-hr basis in Figure 3-27 and Figure 3-28, respectively. Figure 3-27 shows that all the solid PN emissions were under $2.1x10^{12}$ #/mi over all the cycles. On a #/mi basis, solid PN emissions ranged from 1.1 $x10^{11}$ to 2.5 $x10^{11}$ over the UDDS, from $6.5x10^{10}$ to $1.7x10^{12}$ over the Creep cycle, from 1.9 $x10^{10}$ to 5.0 $x10^{11}$ over the Transient cycle, from 4.3 $x10^{10}$ to $2.1x10^{12}$ over the Cruise cycle, and from 5.7 $x10^{10}$ to 1.9 $x10^{12}$ over the HHDDT-S cycle. The lowest solid PN emissions were found over the Transient and Cruise cycles. Solid PN emissions also varied depending on the different trucks. The manufacturer D truck exhibited significantly higher solid PN emissions compared to the other trucks over all the HHDDT cycles. The manufacturer B truck showed the lowest solid PN emissions over the Creep, Transient and HHDDT-S cycles. On a #/bhp-hr basis, solid PN emissions were under 8 x 10^{11} #/bhp-hr for all the cycles, while the solid PN emissions of the manufacturer D vehicle were relatively higher than those of the other trucks, especially over the Cruise and HHDDT-S cycles.

Figure 3-27. Average Solid PN Emissions on a #/mi Basis



* For A2, CS-CPC sampled from second dilution tunnel and results were corrected with dilution factor, others sampled from CVS. The data of C for UDDS was not available.

Figure 3-28. Average Solid PN Emissions on a #/bhp-hr Basis

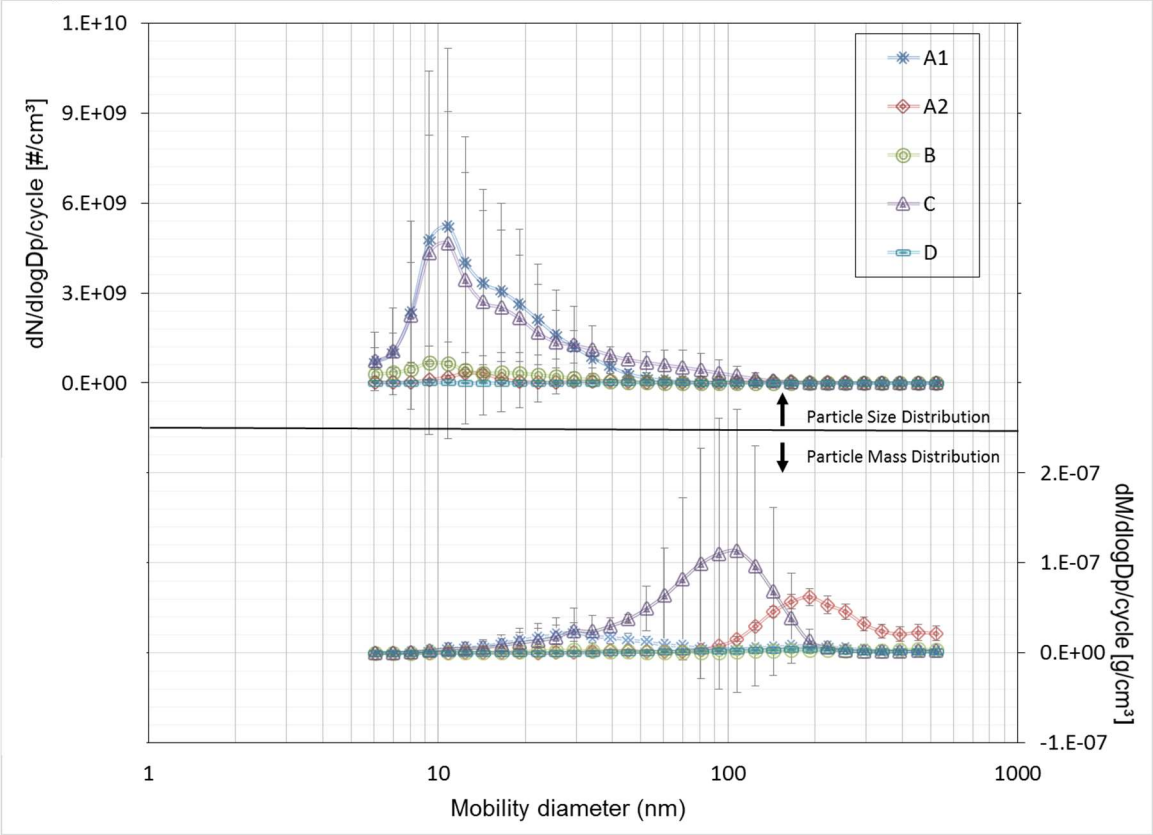


3.8 Particle Size Distributions

The number and mass-based particle size distributions (PSD) for all the cycles are provided in Figure 3-29 through Figure 3-34 (based on EEPS measurements). The number-based PSDs show the average concentration (#/cm^3) as a function of particle diameter over a complete test cycle, and the mass-based PSDs show the distribution of particles by mass (g/cm^3) where the peak diameter represents the largest influence on the PM mass. The particle mass distributions were based on two assumptions from a study by Li et al. (2014). Those assumptions are that all particles were spherical and that the density of particles was 1.46 g/cm^3 for particles smaller than 30 nm and had a functional dependence for particles larger than 30nm. This section is divided into number and mass-based PSD discussions.

For the UDDS cycle, the PSDs for all trucks show a consistent nucleation mode, with a peak particle diameter around 20 nm. The peak concentrations for the manufacturer A1's and manufacturer C engines were relatively higher than for the other trucks. Even though the manufacturer D truck showed higher solid PN emissions over the UDDS, the manufacturer D truck did not have a higher PN concentration for any size bin. This could be attributed to a higher soot component fraction of PM for the manufacturer D truck, whereas the PSDs are based on total PN measurements. Particle mass distributions were dominated by accumulation mode particles, with a peak particle diameter ranging from 30 nm to 200 nm for all trucks.

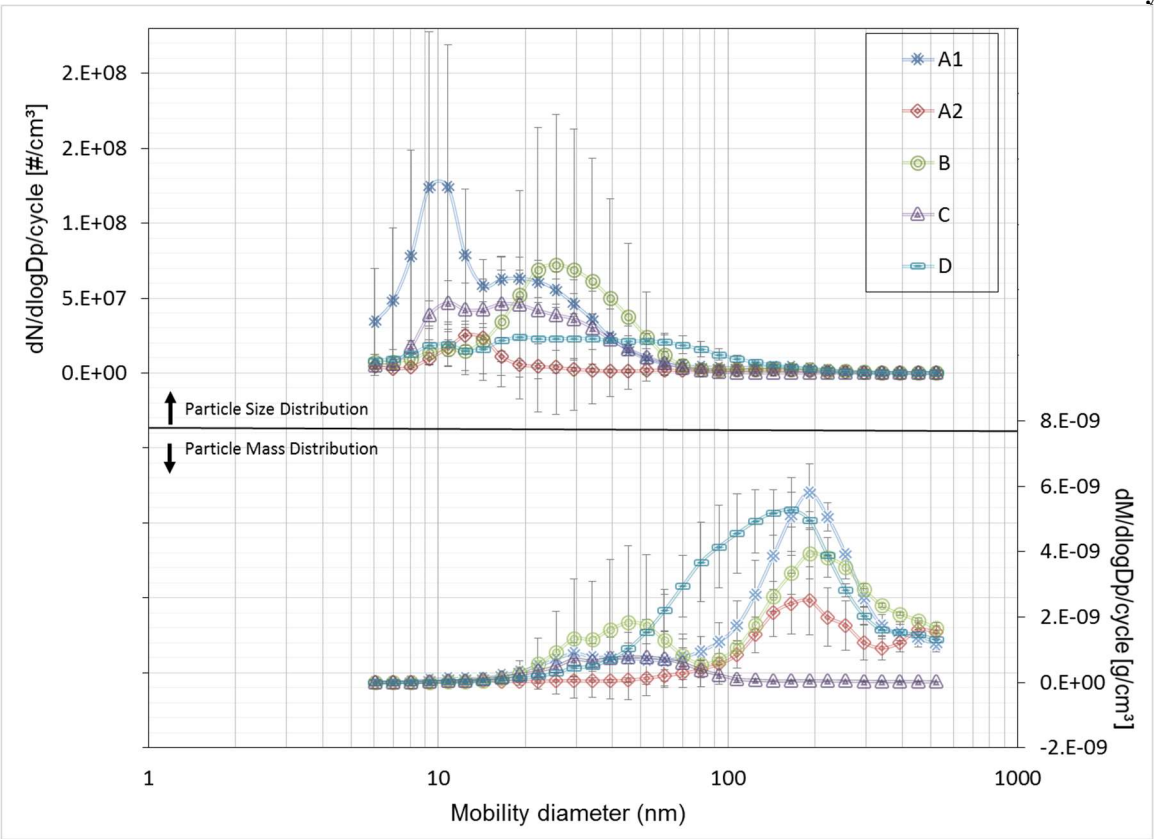
Figure 3-29. Particle Size Distributions and Particle Mass Distributions for UDDS



* For A2, EEPS sampled from second dilution tunnel and results were corrected with dilution factor, others sampled from CVS.

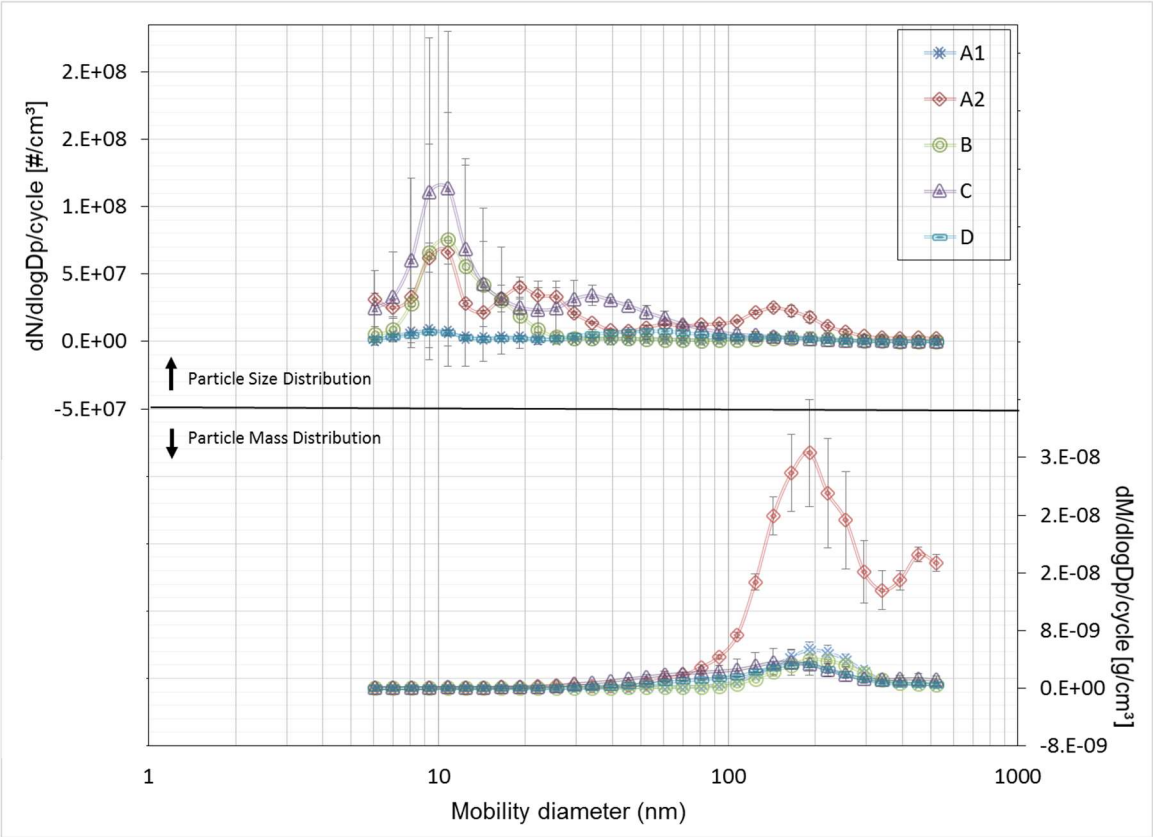
For the Transient cycle, the PSD of the manufacturer C vehicle showed a significantly larger peak compared to the other trucks in the nucleation mode. For the other trucks, the PSDs were predominantly in the nanometer size range, with a peak particle diameter around 20 nm to 50 nm. The particle mass distributions were predominantly accumulation particles with a peak particle diameter around 200 nm, while the manufacturer C vehicle showed a peak around 40 nm-100 nm.

Figure 3-30. Particle Size Distributions and Particle Mass Distributions for Transient Cycle



For the Creep cycle, the PSDs were bimodal for all the trucks, with the highest peak concentrations at particle diameters of 10 nm and lower depending on the different trucks. The manufacturer A2 truck showed another peak around 200 nm particle diameter. For the particle mass distributions, all of the trucks showed significant mass contributions in the accumulation mode particle size range (Dp=200 nm).

Figure 3-31. Particle Size Distributions and Particle Mass Distributions for Creep Cycle



PSD and particle mass distribution results for the Cruise and HHDDT-S cycles were similar, as both were high-speed driving cycles. For the Cruise cycle, the PSDs for the manufacturer A1 and manufacturer C trucks showed significantly higher nucleation mode peaks compared to the other trucks. For particle mass distributions, the manufacturer A1 and manufacturer C trucks showed a significant mass contribution between particle diameters of 20 nm to 50 nm, which suggests that nucleation mode particles contribute considerably higher mass fractions than the accumulation mode over this cycle. For the HHDDT-S cycle, the PSDs and particle mass distributions for the manufacturer A1 and manufacturer C trucks showed a larger peak compared to other trucks in nanoparticle size range (10 nm-50 nm). The higher PM PSDs for the manufacturer C truck over the HHDDT-S cycle is consistent with the higher PM mass seen for this vehicle/cycle combination, as discussed above in section 3.2.

Figure 3-32. Particle Size Distributions and Particle Mass Distributions for Cruise Cycle

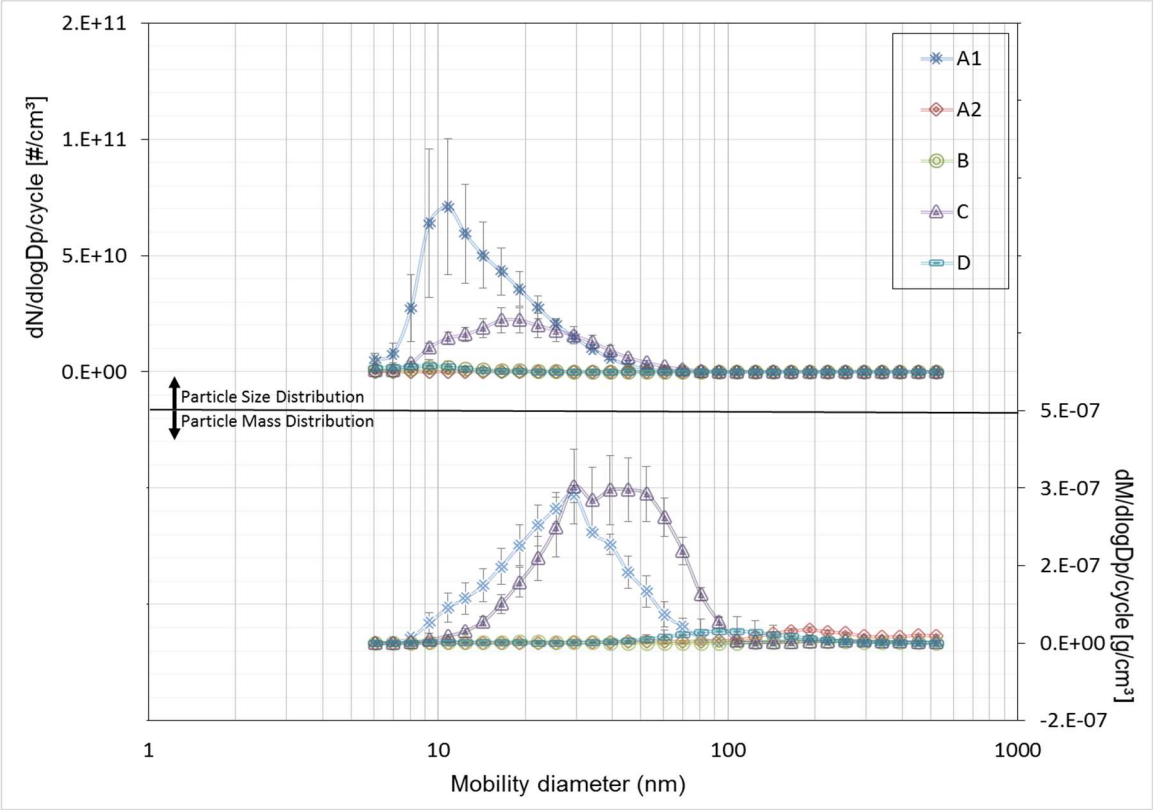
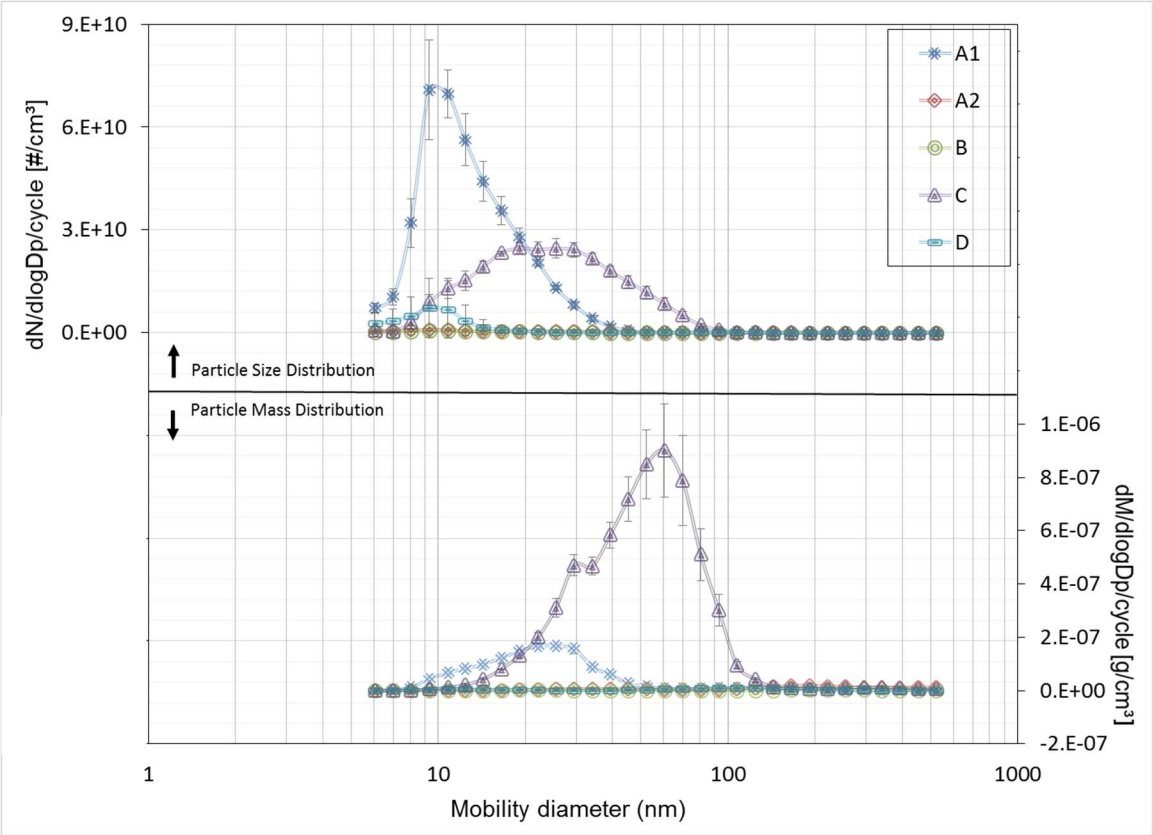
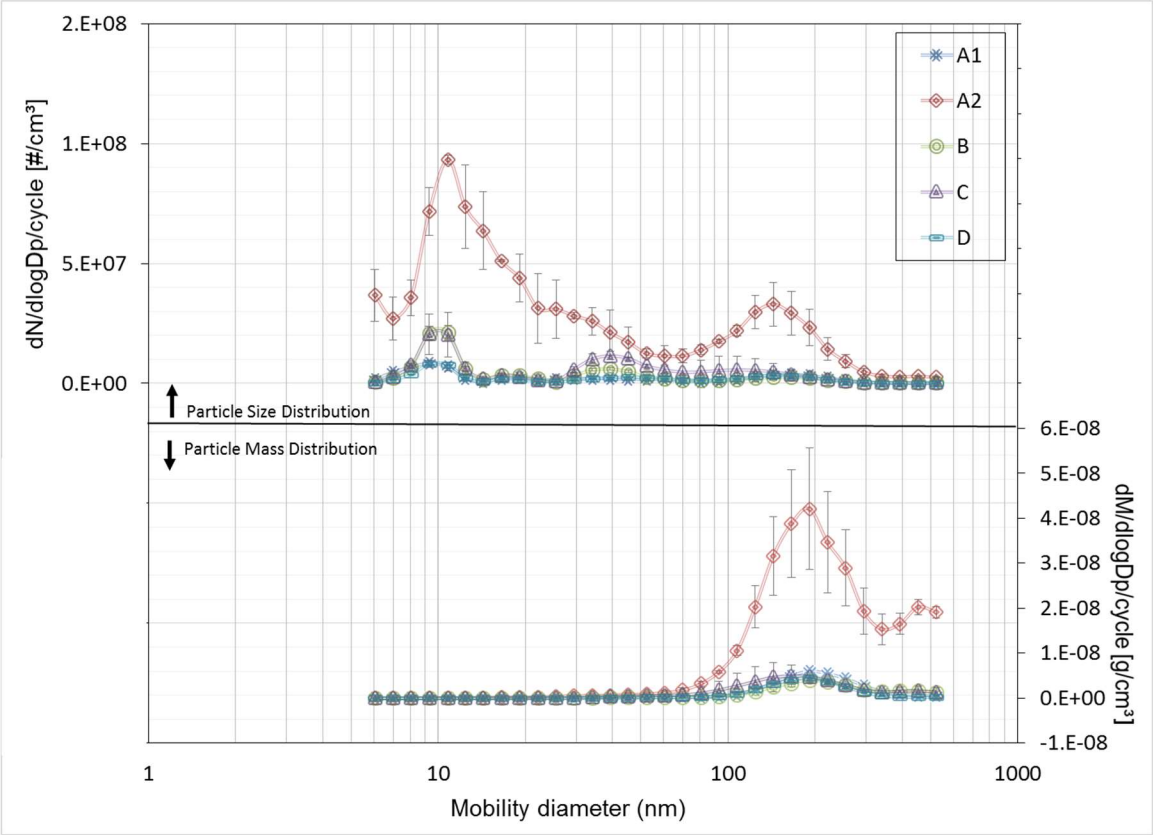


Figure 3-33. Particle Size Distributions and Particle Mass Distributions for HHDDT_S Cycle



For the Idle cycle, the PSDs were bimodal for all the trucks, with the highest peak concentrations at a particle diameter of 20 nm, and a smaller peak around 40 nm. The manufacturer A2 truck showed a third peak around 200 nm particle diameter. For the particle mass distributions, all of the trucks showed a significant mass contribution at 200 nm particle diameter, which suggests that accumulation mode particles contributed considerably higher mass fractions than the nucleation mode.

Figure 3-34. Particle Size Distributions and Particle Mass Distributions for Idle Cycle



4 Summary and Conclusions

Characterization of in-use emissions from on-road heavy-duty trucks is an important element of developing accurate emissions inventory estimates. The goal of this work was to obtain emissions data on low mileage 2010 and later model year Class 8 diesel trucks equipped with the newest emission control strategies, with a special emphasis on data that can be used to improve estimates of "zero-mile" emission rates (ZMRs). We tested five heavy-duty Class 8 diesel trucks equipped with DPFs for PM emissions and SCR systems for NO_x emissions. The vehicles tested ranged in model year from 2012 to 2015, with 4 of the 5 engines being 2014 or newer. The vehicle matrix included 5 engines from heavy-duty engine manufacturers representing the majority of trucks operating in California, with two engines being from the same manufacturer. The engines/vehicles were certified to a 0.2 g/bhp-hr or a lower certification limit, with the exception of one engine that was certified to a 0.35 g/bhp-hr standard. Each vehicle was tested on UCR's heavy-duty chassis dynamometer over the four phases of ARB's Heavy Heavy-Duty Diesel Truck (HHDDT) cycle (i.e., idle, creep, transient, and cruise), the HHDDT short or (HHDDT-S) cycle (which is a high-speed cruise schedule), and the Urban Dynamometer Driving Schedule (UDDS) (which is a cycle considered to be the chassis dynamometer equivalent of the engine dynamometer transient test). Three of the five trucks were retested using the same testing protocol at the ARB's Heavy-Duty Chassis Dynamometer Testing Facility in Los Angeles.[3] The results of this study are summarized below.

NOx emissions varied depending on the test cycle and the test truck. The NOx emissions for the manufacturer D vehicle appear to be an outlier with noticeably higher emission levels, and subsequent additional testing vehicle on this vehicle was conducted at WVU after determining that the vehicle's service history and lack of regeneration events needed to be taken into account. Given anomalies for the manufacturer D truck, this truck is discussed separately from and in more detail than the results for the manufacturer A1, manufacturer A2, manufacturer B and manufacturer C engine-powered trucks.

For the manufacturer A1, manufacturer A2, manufacturer B and manufacturer C trucks, NOx emissions over the UDDS cycle ranged from 0.495 to 1.363 g/mi (0.136 to 0.387 g/bhp-hr). On a bhp-hr basis, those emission levels are comparable to the level to which the engines were certified to on an engine dynamometer, with some trucks being below and other being above the 0.20/0.35 NOx g/bhp-hr level. NOx emissions over the ARB transient cycle were slightly higher than for the UDDS, with emission rates from ranging 0.803 to 3.252 g/mi (0.194 to 0.762 g/bhp-hr). The lowest emissions were found over the two cruise cycles. For the HHDDT-S, NOx emissions ranged from 0.067 to 0.249 g/mi (0.023 to 0.078 g/bhp-hr). For the Cruise cycle, NOx emissions ranged from 0.068 to 0.471 g/mi (0.030 to 0.225 g/bhp-hr). The highest NOx emissions were seen for the Creep cycle, which showed NOx emission ranging from 2.131 to 9.468 g/mi (0.910 to 3.613 g/bhp-hr). The higher NOx emissions for the Creep cycle can be attributed to the fact that the cycle is comprised of short, low-speed accelerations between periods of idle that cover a very short distance (0.124 miles). Such stop-and-go type of driving tends to create high emissions when evaluated on a per mile or per unit of work done basis.

[3] This report is based on data and analysis from the truck testing conducted at CE-CERT. It is important to note that additional testing was also carried out on a subset of the trucks at the ARB's heavy-duty chassis-dynamometer test facility as part of the agreed upon study effort. The goal was to include all of the data and analysis from both testing efforts in this report, however the ARB data have not been provided for analysis or reporting. The additional ARB data would provide additional insight to the overall project results. It is hoped that ARB will consider their data, in addition to what is reported here, as they improve the EMFAC model.

As discussed above, the manufacturer D truck was an outlier with noticeably higher NOx emissions relative to the other vehicles. In this study, on a g/mi basis, its NOx emissions were 3.519 over the UDDS, 22.505 over the Creep cycle, 6.270 over the Transient cycle, 0.656 over the Cruise cycle, and 0.751 over the HHDDT-S. The lowest NOx emissions for the manufacturer D engine were recorded over the Cruise and HHDDT-S cycles. On a g/bhp-hr basis, NOx emissions for the manufacturer D vehicle were 0.967 over the UDDS, 3.832 over the Creep cycle, 1.430 over the Transient cycle, 0.257 over the Cruise cycle, and 0.234 over the HHDDT-S.

Subsequent to the dynamometer testing conducted at UCR, the manufacturer D vehicle was more extensively evaluated by the manufacturer. The vehicle was initially outfitted with a PEMS, which confirmed the poor NOx conversion efficiency observed from the UCR testing. Upon further investigation, it was determined that this specific vehicle had served its entire life as a dealer demonstrator, and as such rarely or never operated with a loaded trailer, and spent a considerable amount of time operating in an idle mode. This type of low-temperature, high proportion idle operation is known to cause significant exposure of the aftertreatment system to unburned hydrocarbons in the exhaust stream. An examination of the logged electronic history revealed no OBD faults or other indications of failure or system malfunction. A clear anomaly, however, was that due to its unusual, unladen service history and duty cycle, the engine had never undergone a regeneration event, despite being approximately 2.5 years in service (albeit with only 12,000 miles on the odometer). A series of conventional parked regenerations were performed. After further operation, there was a significant recovery of the aftertreatment NOx-conversion efficiency, as revealed through PEMS measurements. The regeneration intervention is believed to have been fully effective in driving off the accumulated unburned hydrocarbons that were hindering catalytic reaction. Additional chassis dynamometer testing of the manufacturer D vehicle was subsequently conducted at the West Virginia University CAFEE Laboratory, replicating the testing that had been performed at UCR, with the exception of a 70,000 lbs. test weight. The results of that testing indicated a NOx emission rate of 0.39 g/mi over the UDDS cycle, near the lower end of the NOx emission rates found in the current study.

The results of our study can be compared to the emission factors being used in the EMFAC2014 model. For engines certified to the 0.20 g/bhp-hr NOx level, EMFAC2014 utilizes a ZMR of 1.89. This ZMR is adjusted by a fuel correction factor of 0.93 to account for the clean CARB diesel fuel used in California, such that a ZMR of 1.76 was used for the comparisons in this study for the 0.20 g/bhp-hr NOx engines. EMFAC2014 does not specifically utilize a ZMR for engines certified to the 0.35 g/bhp-hr NOx level, but the ZMR for those engines can be most directly compared with the 2011 model year ZMR of 3.26. That ZMR also is adjusted by the fuel correction factor of 0.93 to account for the use of clean CARB diesel fuel used in California, resulting in a ZMR of 3.03, which was used for the comparisons in this study relating to 0.35 g/bhp-hr NOx engines. EMFAC2014 is a relatively complicated model, including speed correction factors, tampering, malmaintenance and deterioration rates, and other factors. ZMRs, however, are best approximated by the emissions obtained over the UDDS cycle from low mileage vehicles that are well maintained and checked for any evidence of tampering, such as those selected for inclusion in this study. Consequently, that cycle is used as a comparison point between the current EMFAC2014 model ZMRs and our study results.

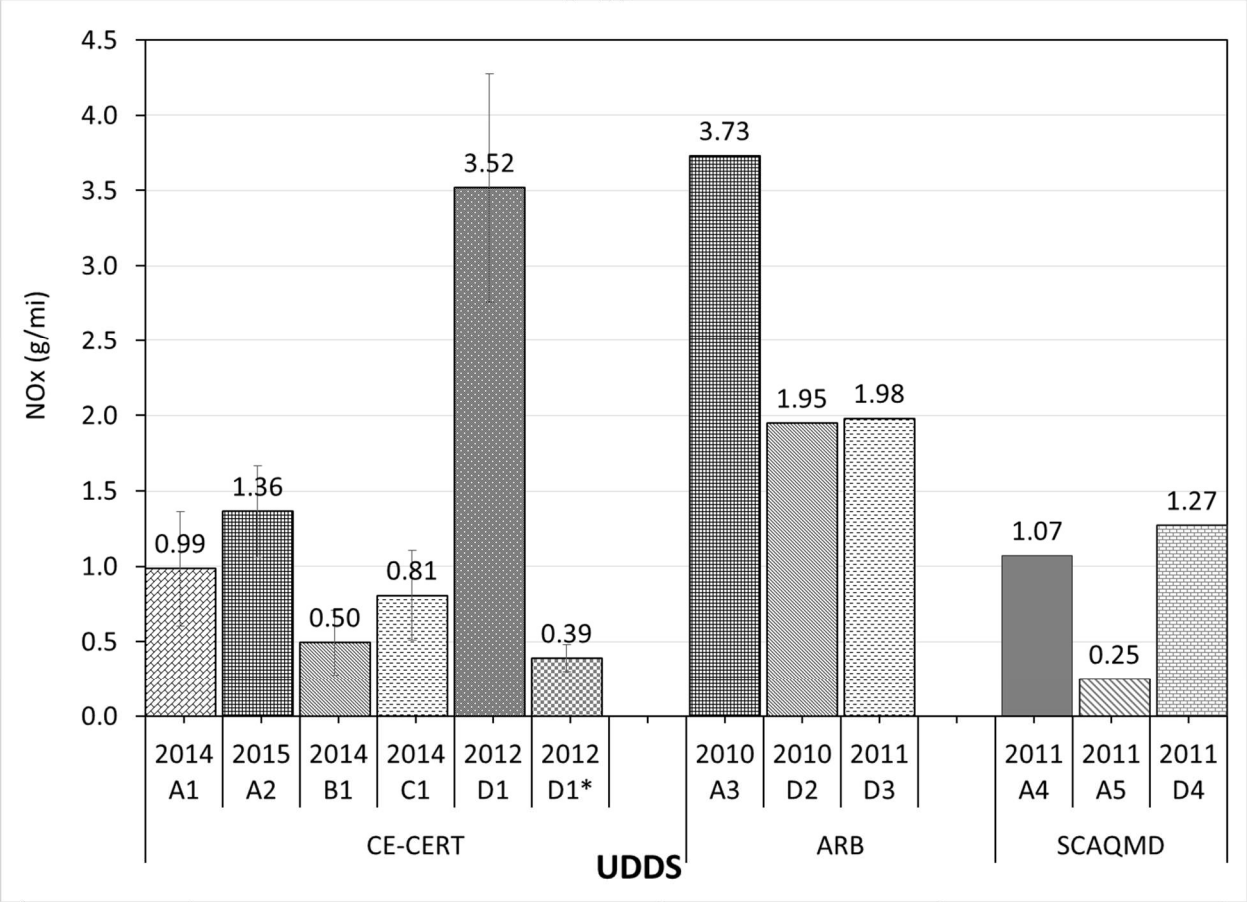
The UDDS emission rates from this study, as well as other studies of 2010 model year and newer heavy-duty vehicles, are presented in Figure 4-1 and Figure 4-2, along with the current EMFAC2014 ZMRs and the NOx Family Emission Limits (FELs) to which the engines are certified. The other earlier studies include a study by the ARB that was utilized to update the emissions factors for the EMFAC model, and another UCR study funded by the South Coast Air

Quality Management District (SCAQMD), which included several SCR and DPF equipped 2010+ model year vehicles in the test fleets. For the UDDS under the current study, the emissions levels for all of the 0.20 g/bhp-hr NOx trucks, except the manufacturer D truck prior to regeneration, are below the EMFAC2014 value. The NOx emissions for the manufacturer D truck after regeneration restored its NOx-conversion efficiency are also well below the EMFAC2014 value. The average UDDS values for the current study for the 0.20 g/bhp-hr NOx trucks are 0.89 g/mi if the outlier manufacturer D truck is excluded, or 0.77 g/mi if the UDDS emission rate from the manufacturer D truck as tested at WVU, after ensuring regeneration, is included. In both cases, those values are well below the EMFAC2014 value, with the lower value being more representative of the in-use fleet. Similarly, the manufacturer A1 truck that was certified to a 0.35 g/bhp-hr NOx FEL has a UDDS emissions level of 0.99 g/mi, which is well below the 2011 model year ZMR approximation of 3.03 g/mi for 0.35 g/bhp-hr NOx trucks.

The UDDS results for the earlier SCAQMD study, with an average NOx level of 0.86 g/mi, are also well below the EMFAC2014 0.20 g/bhp-hr fuel-adjusted ZMR of 1.76 g/mi. The two manufacturer D trucks from the previous ARB study are comparable to but slightly higher than the EMFAC2014 ZMR, as those two vehicles were the primary source of information used in developing that ZMR. They are higher than all of the other trucks certified to the 0.20 g/bhp-hr, with the exception of the manufacturer D truck under the current study, which is again from the same manufacturer. The other manufacturer A3 truck in the earlier ARB study has NOx emissions higher than the 2011 model year EMFAC2014 ZMR value that approximates the value for 0.35 g/bhp-hr NOx level engines.

Also, as the name implies, the ZMR is the estimated emissions rate at zero miles (i.e., for a new truck). Normally, an adjustment factor would be applied to account for the mileage accumulated on the vehicle. As the five vehicles in this study had low mileages (one of the selection criteria for the study as shown in Figure 4-1), no adjustment factor has been applied for mileage. If an adjustment factor were to be applied, the ZMR estimates based on the UDDS emission results would go down slightly. Overall, this comparison analysis suggests that lower ZMRs for future EMFAC model updates may better represent the in-use emission rates of heavy-duty diesel trucks. This study suggests ZMRs may be better represented by the UDDS emission rates of 0.77 g/mi, perhaps ranging to 0.89 g/mi, for engines certified to a 0.20 NOx standard. A lower possible ZMR value of 0.99 g/mi was also found for an engine certified to a 0.35 NOx standard, but only a single vehicle with this certification level was tested in this study.

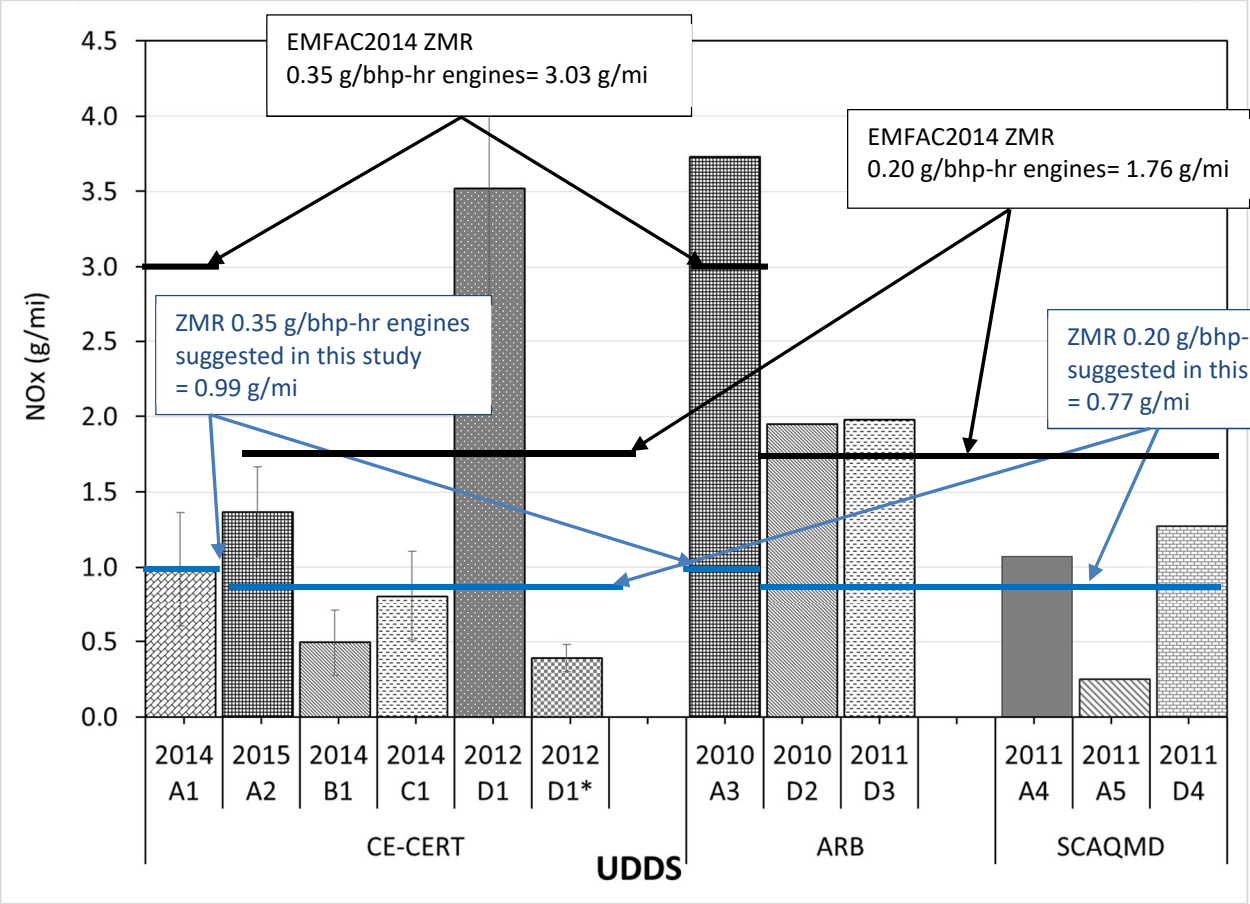
Figure 4-1. Comparison of NOx emission rates from this study, and the ARB and SCAQMD studies, for SCR-equipped 2010+ vehicles over the UDDS



	CE-CERT					ARB			SCAQMD		
Maker	A1	A2	B1	C1	D1	A3	D2	D3	A4	A5	D4
ODO miles	28,611	2,924	15,914	7,686	12,640	13,500	68,000	36,900	14,269	4,769	36,982
STD/FEL g/bhp-hr	0.35	0.20	0.20	0.20	0.20	0.35	0.20	0.20	0.20	0.20	0.20

*Note that D1 represents the UDDS emissions level found after retesting the manufacturer D1 truck after a regeneration.

Figure 4-2 Comparison of NOx Zero Mile Rates Between EMFAC2014 and this Study



PM, THC, and CO emissions were found to be very low under most of the testing conditions. PM emissions were below 0.015 g/mi and 0.006 g/bhp-hr for all vehicles and cycles, except for the manufacturer C truck over the HHDDT-S cycle, and the manufacturer A2 truck over the Creep cycle. Over the UDDS, Transient, Cruise, and HHDDT-S cycles, THC emissions were below 0.05 g/mi for all test vehicles. The Creep cycle did show higher THC emissions on a per-mile and bhp-hr basis, ranging from 0.241 to 0.458 g/mi and from 0.015 to 0.046 g/bhp-hr, due its short, low-speed accelerations and longer idle periods. CO emissions were below 0.2 g/mi and 0.06 g/bhp-hr for all vehicles and cycles, except over the Creep cycle, and for one vehicle over the Transient cycle on a g/mi basis. Overall, the CO emission rates are considerably below the standards of 15.5 and 14.0 g/bhp-hr established by EPA and ARB, respectively, for all vehicles and cycles.

The solid PN emissions for all the cycles were under 2.1×10^{12} #/mi for all the cycles. On a g/mi basis, solid PN emissions ranged from 1.1×10^{11} to 2.5×10^{11} over the UDDS, from 6.5×10^{10} to 1.7×10^{12} over the Creep cycle, from 1.9×10^{10} to 5.0×10^{11} over the Transient cycle, from 4.3×10^{10} to 2.1×10^{12} over the Cruise cycle, and from 5.7×10^{10} to 1.9×10^{12} for the HHDDT-S cycle. The PSD for all trucks as assessed with the EEPS instrument showed a consistent nucleation mode, with a peak particle diameter ranging from 20 nm to 50 nm. Particle mass distributions were dominated by an accumulation mode with a peak around 200 nm particle diameter for most of the test cycles.

CO_2 emissions ranged from 1864 to 2219 g/mi over the UDDS, and from 2260 to 2624 g/mi for the Transient cycle. CO_2 emissions ranged from 1160 to 1443 g/mi for the Cruise cycle, and from 1450 to 1743 g/mi for the HHDDT-S cycle. CO_2 emissions were highest over the Creep cycle, where loads are lowest, ranging from 3748.7 to 5095.0 g/mi.

Fuel economy for the UDDS ranged from 4.56 to 5.41, while fuel economy for the Transient ranged from 3.84 to 4.46 mi/gal. Fuel economy over the Cruise and HHDDT-S cycles was slightly better, ranging from 6.98 to 8.69 mi/gal for the Cruise cycle, and from 5.78 to 6.95 mi/gal for the HHDDT-S cycle. The lowest fuel economy was found over the Creep cycle, and ranged from 1.978 to 2.689 mi/gal., due to the slow speeds and stop-and-go nature of the cycle.

5 References

California Air Resources Board. 2015a. Mobile Source Emissions Inventory and Assessment. Los Angeles. http://www.arb.ca.gov/msei/msei.htm

California Air Resources Board. 2015b. EMFAC2014 Volume III Technical Documentation. https://www.arb.ca.gov/msei/downloads/emfac2014/emfac2014-vol3-technical-documentation-052015.pdf.

Carder, D. K., Gautam, M., Thiruvengadam, A., Besch, M. C., 2014. In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines. Final Report by West Virginia University to the South Coast Air Quality Management District under contract No. 11611, July.

Clark, N. N., Gautam, M., Wayne, W. S., Lyons, D. W., Thompson, G., Zielinska, B., 2007. Heavy-duty vehicle chassis dynamometer testing for emissions inventory, air quality modeling, source apportionment and air toxics emissions inventory, E-55/59 all phases. Final Report by West Virginia University to the Coordinating Research Council. August. Available at ‹www.crcao.com›.

Clark, N. N., Gautam, M., Wayne, W. S., Thompson, G. J., Lyons, D. W., 2004. California heavy heavy-duty diesel truck emissions characterization for project E-55/E-59 phase 1.5. by West Virginia University to the Coordinating Research Council.

Clark, N., Gautam, M., Wayne, W. S., Thompson, G. J., Nine, R. D., Lyons, D. W., Buffamonte, T., Xu, S., Maldonado, H., 2006. Regulated emissions from heavy heavy-duty diesel trucks operating in the south coast air basin. SAE Technical Paper No. 2006-01-3395. Society of Automotive Engineers, Warrendale, PA.

Couch, P., Leonard. J., 2011. Characterization of Drayage Truck Duty Cycles at the Port of Long Beach and Port of Los Angeles. Final Report prepared by TIAX for the Ports of Long Beach and Los Angeles, March.

Environmental Protection Agency, 2015. Heavy Trucks, Buses, and Engines. http://www.epa.gov/otaq/hd-hwy.htm#regs.

Federal Highway Administration, 2000. Table III-4, Comprehensive Truck Size and Weight Study. http://www.fhwa.dot.gov/reports/tswstudy/Vol3-Chapter3.pdf

Gautam, M., Clark, N., Riddle, W., Nine, R., Wayne, W. S., Maldonado, H., Agrawal A., Carlock, M., 2002. Development and initial use of a heavy-duty diesel truck test schedule for emissions characterization. SAE Technical Paper No. 2002-01-1753. Society of Automotive Engineers, Warrendale, PA.

Khan, A. S., Clark, N. N., Thompson, G. J., Wayne, W. S., Gautam, M., Lyon, D. W., Hawelti, D., 2006. Idle emissions from heavy-duty diesel vehicles: review and recent data. Journal of the Air & Waste Management Association, 56(10), 1404-1419.

Khair, M. K., Majewski, W. A., 2006. Diesel emissions and their control. Society of Automotive Engineers, Warrendale, PA.

Li, Y., Xue, J., Johnson, K., Durbin, T., Villela, M., Pham, L., Hosseini, S., Zheng, Z., Short, D., Karavalakis, G. and Asa-Awuku, A., 2014. Determination of suspended exhaust PM mass for light-duty vehicles. SAE Technical Paper No. 2014-01-1594. Society of Automotive Engineers, Warrendale, PA.

Miller, W., Johnson, K.C., Durbin, T., P. Dixit., 2013. In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines. Final Report by University of California, Riverside to the South Coast Air Quality Management District under contract no. 11612.

Misra, C., Collins, J. F., Herner, J. D., Sax, T., Krishnamurthy, M., Sobieralski, W., Burntizki, m., Chernich, D., 2013. In-Use NOx Emissions from Model Year 2010 and 2011 Heavy-

Duty Diesel Engines Equipped with Aftertreatment Devices. Environmental Science & Technology, 47(14), 7892-7898.
Part, C. F. R. 86," Control of Emissions from New and In-Use Highway Vehicles and Engines.". Code of Federal Regulations.

Appendix A. Vehicle Inspection Checklist/Form

Veh. No.:_____ VIN:

ARRIVAL DATE:	ARRIVAL TIME:
AGENCY RELEASE SIGNATURE:	
DELIVERED BY:	

DEPARTURE DATE:	DEPARTURE TIME:
UCR ENGINEER RELEASE SIGNATURE:	
RETURNED TO:	

Retest? ☐Yes ☐No. If Yes, reason for retest:

Engine Compartment

			REMARKS
OIL LEVEL:	☐ FULL	☐ LOW	
COOLANT LEVEL:	☐ FULL	☐ LOW	
POWER STEERING FLUID:	☐ FULL	☐ LOW	
CONDITION OF BELTS:	☐ GOOD	☐ WORN	
CONDITION OF AIR FILTER:	☐ CLEAN	☐ DIRTY	
VISIBLE EXHAUST LEAKS:	☐ YES	☐ NO	
VISIBLE FLUID LEAKS:	☐ YES	☐ NO	
ENGINE APPEARANCE:	☐ CLEAN	☐ GREASY	

Equipment

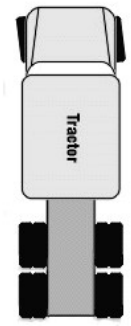
SERVICE BRAKES:	☐ GOOD	☐ POOR	☐ TOUCHY	
PARKING BRAKES:	☐ GOOD	☐ POOR		
POWER DIVIDER:	☐ GOOD	☐ DEFECTIVE	☐ NOT EQUIPPED	
TRANSMISSION:	☐ NORMAL	☐ SHIFTS HARD	☐ NOISY	
LUG NUT COVERS:	☐ YES	☐ NO	NUMBER MISSING:	
TIRE CONDITION:	FRONT		REAR	
	☐ GOOD	☐ WORN	☐ GOOD	☐ WORN
REMARKS:				

Vehicle Interior

UPHOLSTERY:	☐ CLEAN ☐ DIRTY ☐ STAINED ☐ DAMAGED	REMARKS:
CARPET:	☐ CLEAN ☐ DIRTY ☐ STAINED ☐ DAMAGED	REMARKS:
GENERAL APPEARANCE:	☐ CLEAN ☐ DIRTY	REMARKS:
GAUGES AND CONTROLS:	☐ OPERATE PROPERLY ☐ DEFECTIVE	REMARKS:

Vehicle Exterior (mark the location and describe any dents, scratches, damaged lights, mirrors etc. when the vehicle was received by UCR):

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____
7. _____
8. _____
9. _____



10. _____
11. _____
12. _____
13. _____
14. _____
15. _____
16. _____
17. _____
18. _____

Was this vehicle damaged while in UCR custody?☐☐Yes ☐No. If Yes, explain:

General Remarks

_____

_____

_____

_____

Vehicle Information Form

☐ Agency:

☐ Address:

☐ Contact Person:

☐ Phone Number/Email:

☐ Vehicle Manufacturer/ChassisType:

☐ Vehicle Occupancy Capacity: Seated _____ Standing

☐ Agency Vehicle #:_____Licence Plate # :

☐ Vehicle Model Year: _____VIN #:(17 DIGIT)

☐ GVWR Front: Middle: Rear:

☐ Curb Weight:Front: Middle: Rear:

☐ Vehicle Dimensions: Length: Width: Height:

☐ Mileage Odometer: Hub Meter:

☐ Engine Manufacturer: _____ Model: _____Year:

☐ Engine Serial#: _____EPA Family Cert. #:

☐ Engine Displacement:_____ # of Cylinders: Configuration:

☐ Max. Engine Power (hp) _____ hp @ RPM

☐ Max. Engine Torque:(ft-lb.) _____ ft-lbs @ RPM

☐ Idle Speed:_____ Governed Speed: High Idle:

☐ Electronic Engine Control (☐Y/☐N) If Yes, Rebuild:

☐ Engine Rebuilt (☐Y/☐N) If Yes, Year of Rebuild:

☐ Primary Fuel Type: ☐D1 ☐D2 ☐CNG ☐LNG ☐ BD (%): Other (Specify):

☐ Number of Fuel Tanks: _____Capacity: _____

☐ Oil Type: Weight _____Brand

Aftertreatment Configuration:

☐ Oxidation Catalyst (☐Y/☐N) Manufacturer

☐ PM Trap (☐Y/☐N) Manufacturer

☐ SCR (☐Y/☐N) Manufacturer_____

☐ NOx Absorber (☐Y/☐N) Manufacturer

☐ NH3 Catalyst (☐Y/☐N) Manufacturer

☐ Other (☐Y/☐N) Manufacturer

☐ Total Number of Axles:_____ Number of Drive Axles:

☐ Transmission Type: Auto/Manual _____Speeds:

☐ Transmission Manufacturer

☐ Hybrid Technology (☐Y/☐N) Comment:

☐ Tire Size:_____Tire Manufacturer:_____Type(☐Bias ☐Radial ☐Other)

☐ Tailpipe Size:_____Location/Configuration:

Appendix B: Test Cycles

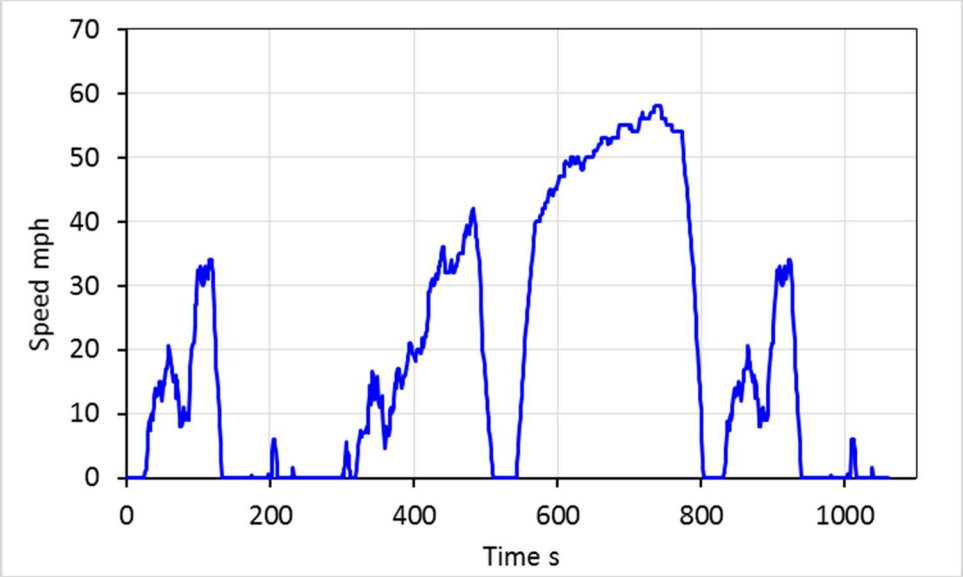


Figure B-1. Speed/Time Trace for a 1xUDDS cycle for the chassis dynamometer.

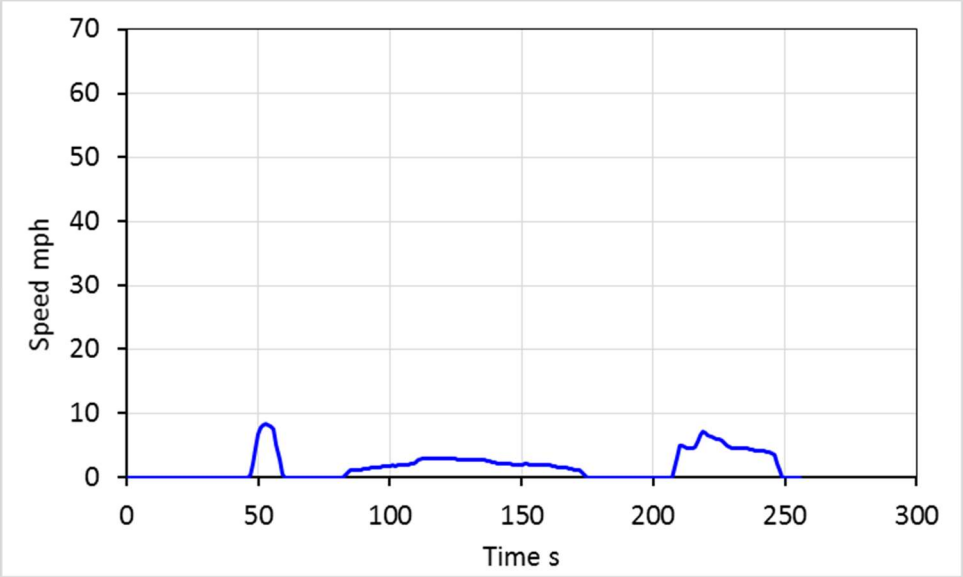


Figure B-2. Speed/Time Trace for a HHDDT-Creep cycle for the chassis dynamometer.

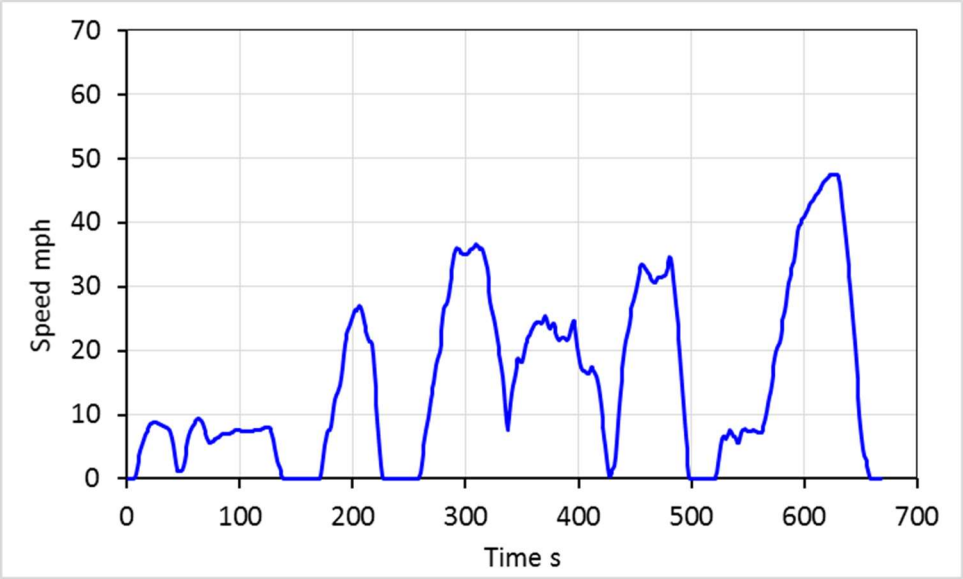


Figure B-3. Speed/Time Trace for a HHDDT-Transient cycle for the chassis dynamometer.

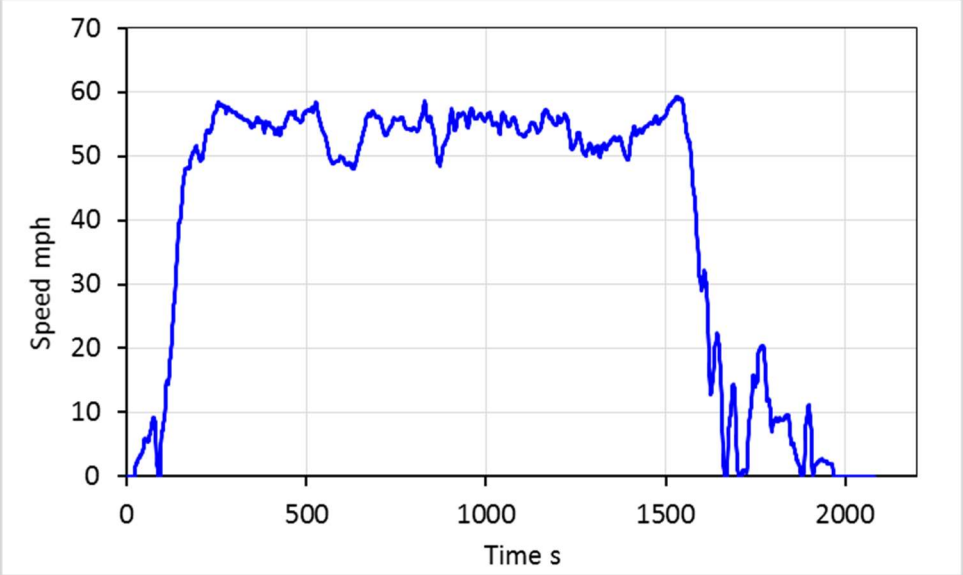


Figure B-4. Speed/Time Trace for a HHDDT-Cruise cycle for the chassis dynamometer.

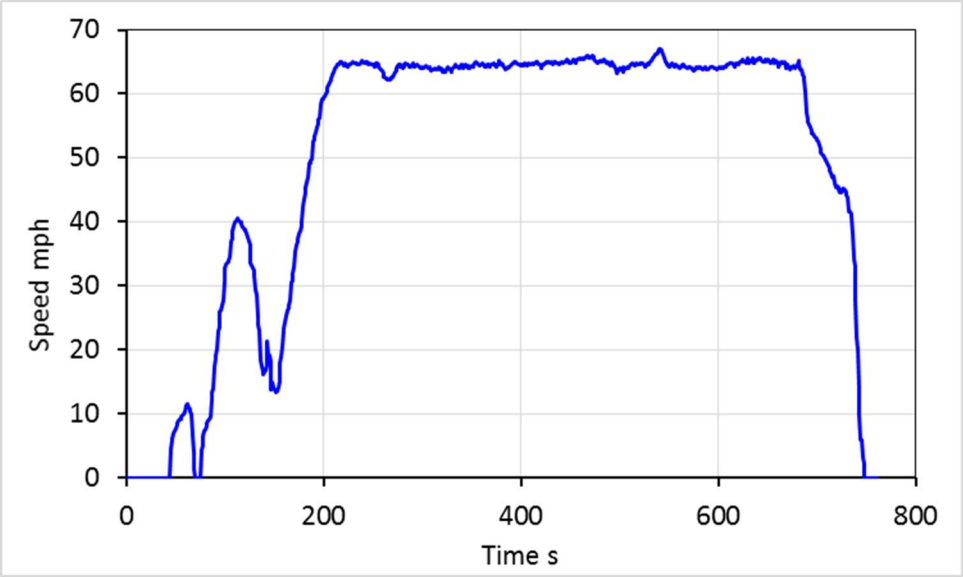


Figure B-5. Speed/Time Trace for a HHDDT-Short cycle for the chassis dynamometer.

Appendix C: Road Load Calculations

The method for determining coast down coefficients at UCR was published and evaluated as part of a previous report to the South Coast Air Quality Management District[4]. Typical coastdown procedures assume that vehicle loading force is a function of vehicle speed, drag coefficient, frontal area and tire rolling resistance coefficient and takes the form of equation 1:

$$M\frac{dV}{dt} = \frac{1}{2}\rho A C_D V^2 + \mu M g cos(\theta) + Mgsin(\theta) \quad \text{(Equation 1)}$$

Where:
M = mass of vehicle in lbs
ρ = density of air in kg/m^3.
A = frontal area of vehicle in square feet, see Figure 1
C_D = aerodynamic drag coefficient (unitless).
V = speed vehicle is traveling in mph.
μ = tire rolling resistance coefficient (unitless).
g = acceleration due to gravity = 32.1740 ft/sec^2.
θ = angle of inclination of the road grade in degrees.

Constant parameters for equation 1	
μ	0.007
C_D	0.75 for Truck 0.79 for Bus 0.80 for Refuse Truck
g	32.1740 ft/sec^2

Assuming that the vehicle loading is characteristic of this equation, speed-time data collected during the coast down test can be used with static measurements (Mass, air density, frontal area, and grade) to solve for drag coefficient (CD) and tire rolling resistance coefficient (μ). The frontal area is measured based on the method described in Figure C-1 below.
However, experience performing in-use coast downs is complex and requires grades of less than 0.5% over miles of distance, average wind speeds < 10 mph ± 2.3 mph gusts and < 5 mph cross wind5. As such, performing in-use coast downs in CA where grade and wind are unpredictable are unreliable where a calculated approach is more consistent and appropriate. Additionally vehicles equipped with automatic transmissions have shown that on-road loading is also affected by the characteristics of the vehicle transmission, especially when reverse pumping losses at low speed begin to dominate.
UCR's and others recommend a coast down method that uses a characteristic coast down equation, with a measured vehicle frontal area (per SAE J1263 measurement recommendations), a tire rolling resistance of 0.007, and a Cd 0.75 (Truck) 0.79 (Bus) and 0.80 (Refuse Truck) in the above equation to calculate coast down times to be used for calculating the A, B, C

[4] Draft Test Plan Re: SCAQMD RFP#P2011-6, "In-Use Emissions Testing and Demonstration of Retrofit Technology for Control of On-Road Heavy-Duty Engines", October 2011
[5] EPA Final rulemaking to establish greenhouse gas emissions standards and fuel efficiency standards for medium and heavy duty engines and vehicles, Office of Transportation and Air Quality, August 2011 (Page 3-7) and J1263 coast down procedure for fuel economy measurements

coefficients in equation 2 for the dyno operation parameters. This approach is consistent and has proven very reliable for chassis testing heavy duty vehicle and has been used for years. For evaluation of aerodynamic modifications and body styles, UCR recommends investing the time perform in-use coast downs.

$$Y = C(x^2) + B(x) + A \qquad \text{(equation 2)}$$

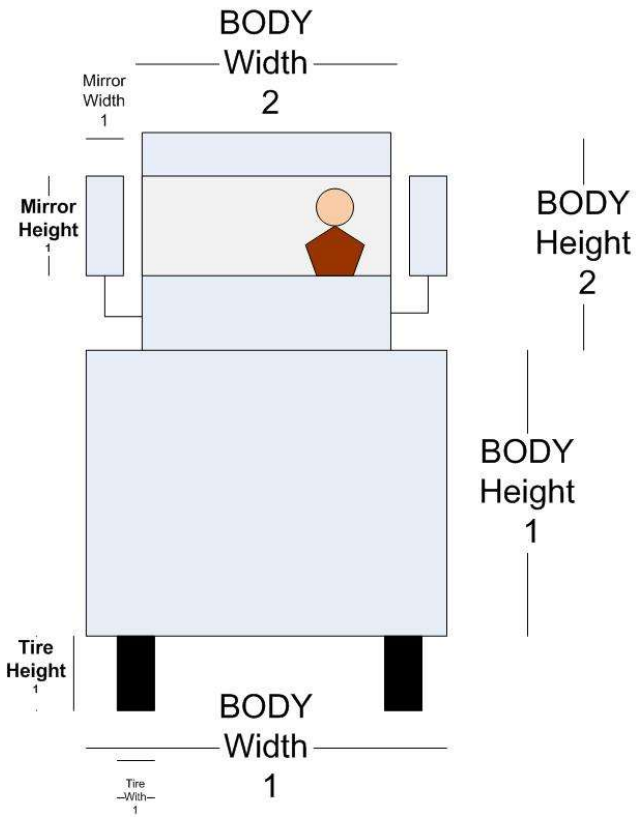


Figure C-1 Vehicle Frontal Area Dimensions Method

The road load coefficients for the specific test vehicles are provided in Table C-1. For comparison, the force applied to the vehicle based on these sets of road load coefficients are also provided in Table C-1. It should be noted that the forces applied for different vehicles were generally more similar for vehicles A1, B1, C1, and D1 compared to A2. This can be attributed to the frontal area calculation, as the manufacturer A2 truck did not have roof fairings on the cab, whereas the other vehicles did, the manufacturer A2 truck had the smallest calculated frontal area, leading to lower loadings at the different speeds. The data for the actual chassis dynamometer verification coast downs compared to the coast down times that would be expected based on the road load coefficients for each vehicle are shown in Table C-2.

Table C-1 Coast Down Coefficient and Road Loads

	MPH	Force (Pounds)				
		A1	A2	B1	C1	D1
	60	1018	895	1023	1069	1065
	50	848	762	852	883	880
	40	709	654	711	732	730
	30	601	570	602	613	612
	20	523	510	524	529	529
	10	477	474	477	479	478
	0	462	462	462	462	462
ABC Coefficients	A	461.658	461.658	461.658	461.658	461.658
	B	-1.529E-14	1.7822E-14	-6.79E-15	8.5018E-15	1.752E-14
	C	0.1544778	0.1203313	0.1559971	0.16870221	0.1675234

Table C-2 Coast Down Times (Calculated Target vs. Actual)

MPH	Coastdown Time (sec)									
	A1		A2		B1		C1		D1	
	Calculated	Actual	Calculated	Actual	Calculated	Actual	Calculated	Actual	Calculated	Actual
65-55	29.1	28.3	33.1	32.6	29.0		27.7		27.8	27.8
55-45	34.9	34.5	38.9	40.2	34.8	35.0	33.5	32.9	33.7	34.2
45-35	41.8	43.0	45.3	46.7	41.7	41.4	40.5	39.9	40.6	41.4
35-25	49.3	51.0	52.0	53.8	49.2	50.1	48.3	49.4	48.4	49.3
25-15	56.6	58.4	58.1	60.1	56.5	57.3	56.0	57.5	56.0	57.6
Total Time (s)	211.8	215.2	227.4	233.4	211.2	183.8*	206.1	179.7*	206.5	210.3

* total time from 55mph to 15 mph

Note that the coast down times represent the amount of time it took to coast the vehicle down from the highest speed to the lowest speed in each row.

Appendix D. Emission Test Results for Individual Vehicles

Table D-1 Emission factors for A1 on distance specific basis

Test Date	Trace	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	Fuel Economy	Total PM
			Emission Factors (g/mi)							MPG	g/mi
2014113	UDDS_x3	Phase 1	0.001	0.009	-0.007	0.09	1.36	1890	0.07	5.33	
		Phase 2	-0.002	0.009	-0.010	0.10	1.40	1853	0.11	5.44	
		Phase 3	0.006	0.012	-0.005	0.20	1.16	1955	0.12	5.16	
											0.009
2014114	UDDS_x3	Phase 1	0.049	0.057	0.001	0.27	0.69	1843	0.03	5.47	
		Phase 2	0.039	0.038	0.006	0.17	0.83	1815	0.01	5.55	
		Phase 3	0.007	0.005	0.003	0.15	0.47	1833	0.05	5.50	
											0.004
2014113	Creep_x10	Phase 1	0.124	0.124	0.017	0.17	0.38	3512	0.07	2.87	
		Phase 2	0.165	0.131	0.053	0.31	0.43	3986	0.07	2.53	
		Phase 3	0.208	0.115	0.109	0.42	1.20	3918	0.07	2.57	
		Phase 4	0.295	0.111	0.201	0.10	3.21	4007	0.06	2.51	
		Phase 5	0.359	0.096	0.279	0.11	6.45	3976	0.07	2.53	
		Phase 6	0.420	0.088	0.347	0.24	7.40	3816	0.06	2.64	
		Phase 7	0.513	0.099	0.432	0.14	8.21	3837	0.07	2.63	
		Phase 8	0.673	0.114	0.581	0.13	10.57	4166	0.08	2.42	
		Phase 9	0.694	0.097	0.616	0.38	10.96	3875	0.08	2.60	
		Phase 10	0.737	0.101	0.657	0.10	12.33	4034	0.10	2.50	
											0.005
2014114	Creep_x10	Phase 1	0.120	0.106	0.029	0.81	0.31	4115	0.16	2.45	
		Phase 2	0.120	0.093	0.041	0.81	0.20	4045	0.12	2.49	
		Phase 3	0.161	0.103	0.074	0.64	0.41	4267	0.15	2.36	
		Phase 4	0.211	0.095	0.131	0.71	1.74	4618	0.18	2.18	
		Phase 5	0.292	0.094	0.213	0.86	3.37	4440	0.21	2.27	
		Phase 6	0.408	0.104	0.322	0.79	4.79	4584	0.25	2.20	
		Phase 7	0.481	0.096	0.402	0.69	6.55	4465	0.25	2.26	
		Phase 8	0.568	0.082	0.501	0.70	9.13	4902	0.23	2.06	
		Phase 9	0.632	0.076	0.572	0.76	8.01	4348	0.18	2.32	
		Phase 10	0.638	0.070	0.583	0.46	9.89	4038	0.12	2.49	
											0.004
2014113	Tansient_x4	Phase 1	-0.027	-0.001	-0.027	0.07	1.79	2285	0.04	4.41	
		Phase 2	-0.030	0.003	-0.034	0.19	1.67	2290	0.04	4.40	
		Phase 3	-0.023	0.005	-0.027	0.15	1.45	2226	0.07	4.53	
		Phase 4	-0.017	0.013	-0.029	0.08	1.86	2230	0.13	4.52	
											0.007
2014114	Tansient_x4	Phase 1	0.025	0.017	0.011	0.00	2.78	2251	0.05	4.48	
		Phase 2	0.037	0.032	0.009	0.04	1.78	2202	0.05	4.58	
		Phase 3	0.056	0.051	0.012	0.08	1.47	2286	0.10	4.41	
		Phase 4	0.059	0.052	0.015	0.09	1.78	2311	0.27	4.36	
											0.003
2014113	Cruise	Phase 1	0.002	0.006	-0.004	0.05	0.10	1153	0.10	8.74	
											0.013
2014114	Cruise	Phase 1	0.010	0.005	0.005	0.00	0.04	1167	0.12	8.64	
											0.012
2014113	HHDDT-S_x2	Phase 1	0.007	0.005	0.003	0.18	0.12	1440	0.04	7.00	
		Phase 2	0.004	0.002	0.002	0.15	0.04	1440	0.05	7.00	
											0.010
2014114	HHDDT-S_x2	Phase 1	0.005	0.009	-0.003	0.09	0.08	1459	0.04	6.91	
		Phase 2	-0.003	0.001	-0.004	0.06	0.04	1461	0.05	6.90	
											0.010

Table D-2 Emission factors for A1 on brake specific basis

Test Date	Trace	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	Total PM
			Emission Factors (g/bhp-hr)							
2014113	UDDS_x3	Phase 1	0.000	0.003	-0.002	0.03	0.40	555	0.02	
		Phase 2	-0.001	0.003	-0.003	0.03	0.42	559	0.03	
		Phase 3	0.002	0.004	-0.002	0.06	0.33	565	0.04	
										0.003
2014114	UDDS_x3	Phase 1	0.014	0.017	0.000	0.08	0.20	542	0.01	
		Phase 2	0.011	0.011	0.002	0.05	0.24	535	0.00	
		Phase 3	0.002	0.001	0.001	0.04	0.14	530	0.02	
										0.001
2014113	Creep_x10	Phase 1	0.069	0.069	0.010	0.10	0.21	1949	0.04	
		Phase 2	0.092	0.073	0.029	0.17	0.24	2213	0.04	
		Phase 3	0.109	0.061	0.058	0.22	0.63	2063	0.03	
		Phase 4	0.166	0.062	0.113	0.06	1.80	2248	0.03	
		Phase 5	0.206	0.055	0.160	0.06	3.70	2281	0.04	
		Phase 6	0.223	0.047	0.185	0.13	3.94	2031	0.03	
		Phase 7	0.292	0.057	0.246	0.08	4.67	2183	0.04	
		Phase 8	0.395	0.067	0.341	0.08	6.21	2446	0.04	
		Phase 9	0.396	0.055	0.351	0.22	6.25	2211	0.05	
		Phase 10	0.387	0.053	0.345	0.05	6.48	2121	0.05	
										0.003
2014114	Creep_x10	Phase 1	0.054	0.048	0.013	0.37	0.14	1863	0.07	
		Phase 2	0.061	0.047	0.021	0.41	0.10	2046	0.06	
		Phase 3	0.078	0.050	0.036	0.31	0.20	2061	0.07	
		Phase 4	0.135	0.061	0.084	0.45	1.11	2956	0.12	
		Phase 5	0.188	0.060	0.137	0.55	2.16	2848	0.14	
		Phase 6	0.255	0.065	0.202	0.50	3.00	2871	0.16	
		Phase 7	0.296	0.059	0.248	0.43	4.04	2751	0.15	
		Phase 8	0.316	0.046	0.279	0.39	5.08	2730	0.13	
		Phase 9	0.486	0.059	0.440	0.58	6.15	3342	0.14	
		Phase 10	0.368	0.040	0.336	0.26	5.71	2329	0.07	
										0.002
2014113	Tansient_x4	Phase 1	-0.007	0.000	-0.007	0.02	0.46	591	0.01	
		Phase 2	-0.008	0.001	-0.009	0.05	0.42	582	0.01	
		Phase 3	-0.006	0.001	-0.007	0.04	0.38	589	0.02	
		Phase 4	-0.005	0.003	-0.007	0.02	0.48	579	0.03	
										0.002
2014114	Tansient_x4	Phase 1	0.007	0.004	0.003	0.00	0.74	597	0.01	
		Phase 2	0.010	0.008	0.002	0.01	0.47	574	0.01	
		Phase 3	0.014	0.013	0.003	0.02	0.37	575	0.02	
		Phase 4	0.015	0.013	0.004	0.02	0.45	586	0.07	
										0.001
2014113	Cruise	Phase 1	0.001	0.003	-0.002	0.02	0.04	518	0.04	
										0.006
2014114	Cruise	Phase 1	0.004	0.002	0.002	0.00	0.02	517	0.05	
										0.005
2014113	HHDDT-S_x2	Phase 1	0.003	0.002	0.001	0.06	0.04	487	0.01	
		Phase 2	0.001	0.001	0.001	0.05	0.01	490	0.02	
										0.003
2014114	HHDDT-S_x2	Phase 1	0.002	0.003	-0.001	0.03	0.03	493	0.01	
		Phase 2	-0.001	0.000	-0.001	0.02	0.01	496	0.02	
										0.003

Table D-3 Emission factors for A2 on distance specific basis

Test Date	Trace	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	Fuel Economy	PM
			Emission Factors (g/mi)							MPG	
		Phase 1	0.026	0.012	0.015	0.00	1.34	1981	0.08	5.09	
20150930	UDDS_x3	Phase 2	0.014	0.015	0.001	0.00	1.04	1991	0.04	5.06	
		Phase 3	0.010	0.018	-0.005	0.00	0.99	2037	0.07	4.95	
											0.0026
		Phase 1	-0.056	-0.054	-0.010	0.00	1.55	1932	0.02	5.22	
20151001	UDDS_x3	Phase 2	0.000	-0.005	0.004	0.00	1.49	2414	0.02	4.18	
		Phase 3	0.005	-0.001	0.005	0.00	1.77	2024	0.02	4.98	
											0.0018
		Phase 1	0.063	0.070	0.003	0.00	0.03	4580	0.21	2.20	
		Phase 2	0.120	0.068	0.062	0.00	1.52	4632	0.21	2.18	
		Phase 3	0.203	0.059	0.154	0.00	5.16	4894	0.19	2.06	
		Phase 4	0.281	0.054	0.236	0.00	7.42	4794	0.18	2.10	
20150930	Creep_x10	Phase 5	0.289	0.028	0.267	0.00	8.72	4746	0.18	2.12	
		Phase 6	0.413	0.031	0.391	0.00	11.75	4944	0.19	2.04	
		Phase 7	0.448	0.021	0.435	0.00	10.68	4827	0.18	2.09	
		Phase 8	0.512	0.013	0.505	0.00	10.88	4946	0.18	2.04	
		Phase 9	0.546	0.023	0.530	0.00	14.16	4815	0.20	2.09	
		Phase 10	0.527	0.034	0.503	0.01	13.17	4840	0.20	2.08	
											0.0006
		Phase 1	-0.031	0.061	-0.084	0.00	0.26	4474	0.18	2.25	
		Phase 2	0.063	0.068	0.005	0.00	1.43	4441	0.17	2.27	
		Phase 3	0.219	0.029	0.195	0.00	6.99	4764	0.19	2.12	
		Phase 4	0.326	-0.007	0.335	0.00	11.41	5116	0.20	1.97	
20151001	Creep_x10	Phase 5	0.362	0.034	0.336	0.00	10.22	4768	0.19	2.11	
		Phase 6	0.343	-0.044	0.385	0.00	13.55	4939	0.19	2.04	
		Phase 7	0.390	-0.031	0.421	0.00	10.86	4795	0.18	2.10	
		Phase 8	0.477	-0.037	0.515	0.00	11.68	4859	0.19	2.07	
		Phase 9	0.514	-0.054	0.566	0.00	13.67	4720	0.18	2.14	
		Phase 10	0.630	-0.035	0.667	0.00	12.47	4728	0.18	2.13	
											0.0316
		Phase 1	0.036	0.038	0.004	0.00	4.58	2584	0.13	3.90	
20150930	Tansient_x4	Phase 2	0.025	0.045	-0.014	0.00	2.39	2429	0.11	4.15	
		Phase 3	0.022	0.039	-0.011	0.00	1.05	2563	0.09	3.93	
		Phase 4	0.011	0.019	-0.005	0.00	1.25	2759	0.11	3.65	
											n/a
		Phase 1	-0.010	-0.021	0.008	0.00	5.36	2591	0.03	3.89	
20151001	Tansient_x4	Phase 2	-0.024	-0.020	-0.007	0.00	5.09	2651	0.03	3.80	
		Phase 3	-0.033	-0.022	-0.014	0.00	3.46	2586	0.03	3.90	
		Phase 4	-0.045	-0.033	-0.016	0.00	2.84	2475	0.03	4.07	
											0.0029
20150930	Cruise	Phase 1	0.002	0.018	-0.014	0.00	0.11	1363	0.09	7.40	
											0.0017
20151001	Cruise	Phase 1	-0.016	-0.010	-0.007	0.00	0.13	1290	0.01	7.81	
											0.0019
20150930	HHDDT-S_x2	Phase 1	0.000	0.003	-0.003	0.00	0.10	1685	0.10	5.98	
		Phase 2	0.001	0.006	-0.004	0.00	0.03	1723	0.09	5.85	
											0.0105
20151001	HHDDT-S_x2	Phase 1	-0.008	-0.010	0.000	0.00	0.15	1690	0.02	5.96	
		Phase 2	-0.013	-0.015	0.000	0.00	0.06	1729	0.02	5.83	
											0.0028

Table D-4 Emission factors for A2 on brake specific basis

Test Date	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	PM
		Emission Factors (g/bhp-hr)							
20150930	Phase 1	0.007	0.004	0.004	0.00	0.39	569	0.02	
	Phase 2	0.004	0.004	0.000	0.00	0.29	566	0.01	
	Phase 3	0.003	0.005	-0.002	0.00	0.28	569	0.02	
									0.0007
20151001	Phase 1	-0.016	-0.016	-0.003	0.00	0.45	557	0.01	
	Phase 2	0.000	-0.001	0.001	0.00	0.42	677	0.01	
	Phase 3	0.001	0.000	0.001	0.00	0.50	573	0.01	
									0.0005
20150930	Phase 1	0.025	0.028	0.001	0.00	0.01	1833	0.09	
	Phase 2	0.042	0.024	0.022	0.00	0.53	1612	0.07	
	Phase 3	0.067	0.020	0.051	0.00	1.71	1623	0.06	
	Phase 4	0.106	0.021	0.090	0.00	2.81	1817	0.07	
	Phase 5	0.120	0.012	0.111	0.00	3.60	1962	0.07	
	Phase 6	0.154	0.011	0.146	0.00	4.38	1844	0.07	
	Phase 7	0.147	0.007	0.143	0.00	3.51	1585	0.06	
	Phase 8	0.194	0.005	0.191	0.00	4.12	1872	0.07	
	Phase 9	0.215	0.009	0.209	0.00	5.57	1895	0.08	
	Phase 10	0.168	0.011	0.160	0.00	4.20	1544	0.06	
									0.0002
20151001	Phase 1	-0.012	0.023	-0.032	0.00	0.10	1715	0.07	
	Phase 2	0.026	0.028	0.002	0.00	0.59	1833	0.07	
	Phase 3	0.088	0.012	0.079	0.00	2.82	1921	0.08	
	Phase 4	0.108	-0.002	0.111	0.00	3.78	1692	0.07	
	Phase 5	0.144	0.013	0.133	0.00	4.05	1889	0.08	
	Phase 6	0.111	-0.014	0.125	0.00	4.39	1601	0.06	
	Phase 7	0.134	-0.011	0.144	0.00	3.73	1646	0.06	
	Phase 8	0.180	-0.014	0.194	0.00	4.40	1831	0.07	
	Phase 9	0.189	-0.020	0.208	0.00	5.03	1738	0.07	
	Phase 10	0.226	-0.013	0.239	0.00	4.47	1697	0.07	
									0.0117
20150930	Phase 1	0.008	0.009	0.001	0.00	1.06	597	0.03	
	Phase 2	0.006	0.011	-0.003	0.00	0.57	581	0.03	
	Phase 3	0.005	0.009	-0.003	0.00	0.24	578	0.02	
	Phase 4	0.002	0.004	-0.001	0.00	0.28	608	0.02	
									n/a
20151001	Phase 1	-0.002	-0.005	0.002	0.00	1.26	608	0.01	
	Phase 2	-0.006	-0.005	-0.002	0.00	1.17	612	0.01	
	Phase 3	-0.008	-0.005	-0.003	0.00	0.83	619	0.01	
	Phase 4	-0.011	-0.008	-0.004	0.00	0.70	607	0.01	
									0.0007
20150930	Phase 1	0.001	0.008	-0.006	0.00	0.05	560	0.04	
									0.0007
20151001	Phase 1	-0.007	-0.004	-0.003	0.00	0.06	558	0.01	
									0.0008
20150930	Phase 1	0.000	0.001	-0.001	0.00	0.03	532	0.03	
	Phase 2	0.000	0.002	-0.001	0.00	0.01	525	0.03	
									0.0033
20151001	Phase 1	-0.003	-0.003	0.000	0.00	0.05	533	0.01	
	Phase 2	-0.004	-0.005	0.000	0.00	0.02	532	0.01	
									0.0009

Table D-5 Emission factors for B on distance specific basis

Tset Date	Trace	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	Fuel Economy	PM
			Emission Factors (g/mi)							MPG	g/mi
20150113	UDDS_x3	Phase 1	0.004	0.008	-0.003	0.18	0.75	2097	0.10	4.81	
		Phase 2	0.033	0.029	0.007	0.22	0.76	2038	0.09	4.95	
		Phase 3	0.035	0.031	0.008	0.13	0.42	2024	0.10	4.98	
											0.0017
20150114	UDDS_x3	Phase 1	0.032	0.035	0.003	0.19	0.24	1954	0.08	5.16	
		Phase 2	0.027	0.027	0.003	0.16	0.34	1950	0.08	5.17	
		Phase 3	0.028	0.027	0.004	0.16	0.46	1971	0.07	5.11	
											0.0033
20150113	Creep_x10	Phase 1	0.157	0.162	0.018	0.29	0.34	3579	0.21	2.82	
		Phase 2	0.147	0.137	0.030	0.32	0.15	3313	0.20	3.04	
		Phase 3	0.169	0.134	0.054	0.22	3.47	3683	0.27	2.74	
		Phase 4	0.209	0.143	0.087	0.55	10.38	3867	0.29	2.61	
		Phase 5	0.272	0.182	0.116	0.68	14.67	4473	0.31	2.25	
		Phase 6	0.228	0.149	0.101	1.21	11.13	3858	0.24	2.61	
		Phase 7	0.256	0.142	0.135	1.28	14.92	3740	0.26	2.69	
		Phase 8	0.287	0.163	0.147	1.01	16.10	3627	0.17	2.78	
		Phase 9	0.344	0.177	0.193	1.43	17.39	3601	0.13	2.80	
		Phase 10	0.333	0.159	0.199	1.32	16.62	3571	0.13	2.82	
											0.0082
20150114	Creep_x10	Phase 1	0.136	0.158	0.000	0.37	0.41	3738	0.18	2.70	
		Phase 2	0.124	0.118	0.022	-0.02	0.18	3562	0.16	2.83	
		Phase 3	0.213	0.186	0.053	0.16	0.65	4422	0.17	2.28	
		Phase 4	0.193	0.150	0.065	0.47	4.89	3634	0.11	2.77	
		Phase 5	0.222	0.146	0.097	0.62	9.62	3507	0.00	2.87	
		Phase 6	0.245	0.148	0.119	1.32	11.69	3623	0.09	2.78	
		Phase 7	0.289	0.147	0.164	1.59	13.86	3705	0.16	2.72	
		Phase 8	0.280	0.137	0.163	0.89	11.54	3426	0.15	2.94	
		Phase 9	0.343	0.150	0.216	2.09	15.88	3757	0.15	2.68	
		Phase 10	0.337	0.143	0.216	1.47	15.47	3463	0.13	2.91	
											0.0043
20150113	Tansient_x4	Phase 1	0.007	0.035	-0.024	0.10	1.06	2490	0.06	4.05	
		Phase 2	0.014	0.046	-0.026	0.13	0.60	2699	0.02	3.74	
		Phase 3	0.018	0.043	-0.020	0.20	0.62	2364	0.01	4.26	
		Phase 4	0.016	0.040	-0.019	0.14	1.05	2416	0.01	4.17	
											0.0015
20150114	Tansient_x4	Phase 1	0.026	0.032	-0.001	0.07	1.05	2324	0.11	4.34	
		Phase 2	0.024	0.033	-0.004	0.05	0.61	2449	0.11	4.12	
		Phase 3	0.026	0.033	-0.003	0.09	0.68	2368	0.10	4.26	
		Phase 4	0.022	0.030	-0.004	0.05	0.74	2381	0.09	4.23	
											0.0017
20150113	Cruise	Phase 1	0.010	0.008	0.003	0.10	0.20	1268	0.04	7.95	
											0.0020
20150114	Cruise	Phase 1	0.004	0.007	-0.002	0.10	0.13	1260	0.05	8.00	
											0.0027
20150113	HHDDT-S_x2	Phase 1	0.004	0.004	0.001	0.12	0.44	1605	0.05	6.28	
		Phase 2	0.003	0.004	-0.001	0.10	0.13	1589	0.05	6.34	
											0.0022
20150114	HHDDT-S_x2	Phase 1	0.003	0.006	-0.002	0.08	0.30	1582	0.04	6.37	
		Phase 2	0.004	0.006	-0.002	0.07	0.13	1608	0.09	6.27	
											0.0019

Table D-6 Emission factors for B on brake specific basis

Tset Date	Trace	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	PM
			Emission Factors (g/bhp-hr)							
20150113	UDDS_x3	Phase 1	0.001	0.002	-0.001	0.05	0.20	567	0.03	
		Phase 2	0.009	0.008	0.002	0.06	0.21	566	0.03	
		Phase 3	0.009	0.008	0.002	0.04	0.11	540	0.03	
										0.0005
20150114	UDDS_x3	Phase 1	0.009	0.010	0.001	0.05	0.07	553	0.02	
		Phase 2	0.007	0.008	0.001	0.04	0.10	546	0.02	
		Phase 3	0.008	0.008	0.001	0.05	0.13	558	0.02	
										0.0009
20150113	Creep_x10	Phase 1	0.056	0.057	0.006	0.10	0.12	1269	0.07	
		Phase 2	0.059	0.055	0.012	0.13	0.06	1331	0.08	
		Phase 3	0.053	0.042	0.017	0.07	1.09	1159	0.08	
		Phase 4	0.076	0.052	0.031	0.20	3.75	1397	0.11	
		Phase 5	0.088	0.059	0.037	0.22	4.74	1445	0.10	
		Phase 6	0.071	0.046	0.031	0.37	3.44	1192	0.07	
		Phase 7	0.092	0.051	0.048	0.46	5.34	1340	0.09	
		Phase 8	0.117	0.067	0.060	0.41	6.57	1480	0.07	
		Phase 9	0.148	0.076	0.083	0.62	7.48	1549	0.06	
		Phase 10	0.142	0.068	0.085	0.56	7.08	1521	0.05	
										0.0029
20150114	Creep_x10	Phase 1	0.047	0.055	0.000	0.13	0.14	1294	0.06	
		Phase 2	0.044	0.042	0.008	-0.01	0.07	1270	0.06	
		Phase 3	0.067	0.059	0.017	0.05	0.20	1390	0.05	
		Phase 4	0.073	0.057	0.024	0.18	1.85	1379	0.04	
		Phase 5	0.091	0.060	0.040	0.25	3.93	1434	0.00	
		Phase 6	0.089	0.054	0.043	0.48	4.25	1316	0.03	
		Phase 7	0.112	0.057	0.064	0.62	5.38	1439	0.06	
		Phase 8	0.119	0.058	0.069	0.38	4.90	1455	0.06	
		Phase 9	0.117	0.051	0.073	0.71	5.40	1278	0.05	
		Phase 10	0.141	0.060	0.090	0.61	6.46	1445	0.06	
										0.0016
20150113	Tansient_x4	Phase 1	0.002	0.008	-0.006	0.02	0.25	596	0.01	
		Phase 2	0.003	0.010	-0.006	0.03	0.14	610	0.00	
		Phase 3	0.004	0.011	-0.005	0.05	0.15	576	0.00	
		Phase 4	0.004	0.009	-0.004	0.03	0.24	554	0.00	
										0.0004
20150114	Tansient_x4	Phase 1	0.007	0.008	0.000	0.02	0.26	583	0.03	
		Phase 2	0.006	0.008	-0.001	0.01	0.15	595	0.03	
		Phase 3	0.007	0.008	-0.001	0.02	0.17	603	0.02	
		Phase 4	0.005	0.007	-0.001	0.01	0.18	588	0.02	
										0.0004
20150113	Cruise	Phase 1	0.004	0.003	0.001	0.04	0.08	521	0.02	
										0.0008
20150114	Cruise	Phase 1	0.002	0.003	-0.001	0.04	0.05	530	0.02	
										0.0011
20150113	HHDDT-S_x2	Phase 1	0.001	0.001	0.000	0.04	0.14	500	0.02	
		Phase 2	0.001	0.001	0.000	0.03	0.04	473	0.01	
										0.0007
20150114	HHDDT-S_x2	Phase 1	0.001	0.002	-0.001	0.02	0.10	504	0.01	
		Phase 2	0.001	0.002	-0.001	0.02	0.04	505	0.01	
										0.0006

Table D-7 Emission factors for C on distance specific basis

Test Date	Trace	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	Fuel Economy	PM
			Emission Factors (g/mi)							MPG	g/mi
20141217	UDDS_x3	Phase 1	0.041	0.029	0.017	0.13	1.10	2164	0.09	4.66	
		Phase 2	0.031	0.021	0.013	0.11	1.18	2104	0.09	4.79	
		Phase 3	0.036	0.020	0.019	0.14	0.92	2181	0.10	4.62	
											0.006
20141218	UDDS_x3	Phase 1	0.031	0.024	0.010	0.04	0.57	2108	0.14	4.78	
		Phase 2	0.031	0.019	0.015	0.05	0.56	2068	0.14	4.87	
		Phase 3	0.031	0.015	0.018	0.01	0.51	2144	0.17	4.70	
											0.003
20141217	Creep_x10	Phase 1	0.168	0.109	0.075	0.06	0.66	4240	0.31	2.38	
		Phase 2	0.272	0.141	0.151	3.75	3.51	5023	0.35	2.00	
		Phase 3	0.353	0.202	0.181	6.18	3.16	5102	0.36	1.97	
		Phase 4	0.351	0.193	0.186	5.75	1.79	5228	0.37	1.92	
		Phase 5	0.317	0.181	0.163	4.99	1.23	4775	0.34	2.11	
		Phase 6	0.286	0.121	0.184	-0.01	1.63	4714	0.29	2.14	
		Phase 7	0.304	0.116	0.206	-0.13	1.76	4595	0.31	2.19	
		Phase 8	0.401	0.165	0.261	0.06	2.65	5183	0.33	1.94	
		Phase 9	0.506	0.242	0.300	5.55	2.38	5553	0.33	1.81	
		Phase 10	0.435	0.203	0.263	3.82	2.00	5008	0.36	2.01	
											0.003
20141218	Creep_x10	Phase 1	0.203	0.148	0.076	0.47	0.32	4868	0.53	2.07	
		Phase 2	0.258	0.143	0.136	0.54	1.08	4921	0.51	2.05	
		Phase 3	0.451	0.232	0.254	6.88	3.83	5685	0.54	1.77	
		Phase 4	0.350	0.156	0.217	0.37	2.26	5268	0.50	1.91	
		Phase 5	0.416	0.156	0.285	0.40	2.40	5395	0.49	1.87	
		Phase 6	0.462	0.152	0.334	0.54	2.46	5214	0.46	1.93	
		Phase 7	0.493	0.185	0.337	4.69	2.47	5466	0.45	1.84	
		Phase 8	0.486	0.164	0.348	0.50	2.08	5336	0.41	1.89	
		Phase 9	0.498	0.190	0.339	3.92	2.23	5292	0.38	1.90	
		Phase 10	0.501	0.144	0.382	0.63	2.69	5047	0.37	2.00	
											0.006
20141217	Tansient_x4	Phase 1	0.022	0.008	0.016	0.14	1.56	2583	0.06	3.90	
		Phase 2	0.017	0.003	0.015	0.14	1.32	2501	0.06	4.03	
		Phase 3	0.022	0.009	0.014	0.05	1.91	2633	0.07	3.83	
		Phase 4	0.028	0.018	0.012	0.10	1.28	2736	0.09	3.68	
											0.009
20141218	Tansient_x4	Phase 1	0.041	0.032	0.014	0.23	0.45	2904	0.01	3.47	
		Phase 2	0.019	0.020	0.002	0.12	1.11	2461	0.03	4.10	
		Phase 3	0.021	0.023	0.001	0.17	1.64	2469	0.03	4.08	
		Phase 4	0.021	0.025	-0.001	0.20	1.22	2573	0.04	3.92	
											0.015
20141217	Cruise	Phase 1	0.003	0.003	0.001	0.08	0.45	1231	0.04	8.19	
											0.009
20141218	Cruise	Phase 1	0.003	0.007	-0.003	0.03	0.49	1233	0.06	8.17	
											0.012
20141217	HHDDT-S_x2	Phase 1	0.003	0.003	0.000	0.03	0.38	1671	0.08	6.03	
		Phase 2	0.002	0.004	-0.002	0.06	0.11	1665	0.08	6.06	
											0.029
20141218	HHDDT-S_x2	Phase 1	-0.004	0.012	-0.014	0.10	0.30	1641	0.12	6.14	
		Phase 2	-0.011	0.007	-0.017	0.09	0.08	1608	0.09	6.27	
											0.038

Table D-8 Emission factors for C on brake specific basis

Test Date	Trace	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	PM
			Emission Factors (g/bhp-hr)							
20141217	UDDS_x3	Phase 1	0.013	0.009	0.005	0.04	0.35	683	0.03	
		Phase 2	0.010	0.007	0.004	0.03	0.38	671	0.03	
		Phase 3	0.012	0.006	0.006	0.04	0.30	700	0.03	
										0.002
20141218	UDDS_x3	Phase 1	0.010	0.008	0.003	0.01	0.18	671	0.04	
		Phase 2	0.010	0.006	0.005	0.02	0.18	667	0.04	
		Phase 3	0.010	0.005	0.006	0.00	0.16	674	0.05	
										0.001
20141217	Creep_x10	Phase 1	0.069	0.045	0.031	0.02	0.27	1738	0.13	
		Phase 2	0.097	0.051	0.054	1.34	1.26	1802	0.12	
		Phase 3	0.139	0.080	0.071	2.44	1.25	2012	0.14	
		Phase 4	0.141	0.078	0.075	2.31	0.72	2098	0.15	
		Phase 5	0.147	0.084	0.075	2.32	0.57	2218	0.16	
		Phase 6	0.133	0.056	0.086	0.00	0.76	2194	0.14	
		Phase 7	0.145	0.056	0.099	-0.06	0.84	2197	0.15	
		Phase 8	0.162	0.067	0.106	0.02	1.07	2094	0.13	
		Phase 9	0.196	0.094	0.117	2.16	0.92	2155	0.13	
		Phase 10	0.175	0.081	0.106	1.53	0.80	2010	0.15	
										0.001
20141218	Creep_x10	Phase 1	0.071	0.052	0.026	0.16	0.11	1693	0.18	
		Phase 2	0.150	0.083	0.079	0.31	0.63	2862	0.30	
		Phase 3	0.186	0.096	0.105	2.84	1.58	2347	0.22	
		Phase 4	0.172	0.077	0.107	0.18	1.11	2583	0.25	
		Phase 5	0.182	0.068	0.125	0.18	1.05	2358	0.21	
		Phase 6	0.205	0.068	0.148	0.24	1.10	2318	0.20	
		Phase 7	0.214	0.080	0.147	2.04	1.08	2376	0.19	
		Phase 8	0.193	0.065	0.138	0.20	0.83	2119	0.16	
		Phase 9	0.216	0.082	0.147	1.70	0.96	2291	0.16	
		Phase 10	0.242	0.069	0.184	0.30	1.30	2435	0.18	
										0.003
20141217	Tansient_x4	Phase 1	0.006	0.002	0.004	0.04	0.43	708	0.02	
		Phase 2	0.005	0.001	0.004	0.04	0.36	682	0.02	
		Phase 3	0.006	0.002	0.004	0.01	0.52	712	0.02	
		Phase 4	0.007	0.005	0.003	0.03	0.34	733	0.02	
										0.002
20141218	Tansient_x4	Phase 1	0.012	0.009	0.004	0.07	0.13	829	0.00	
		Phase 2	0.006	0.006	0.000	0.03	0.32	709	0.01	
		Phase 3	0.006	0.006	0.000	0.05	0.46	700	0.01	
		Phase 4	0.006	0.007	0.000	0.06	0.34	715	0.01	
										0.004
20141217	Cruise	Phase 1	0.002	0.001	0.001	0.04	0.22	591	0.02	
										0.004
20141218	Cruise	Phase 1	0.002	0.003	-0.001	0.02	0.23	587	0.03	
										0.006
20141217	HHDDT-S_x2	Phase 1	0.001	0.001	0.000	0.01	0.13	577	0.03	
		Phase 2	0.001	0.001	-0.001	0.02	0.04	568	0.03	
										0.010
20141218	HHDDT-S_x2	Phase 1	-0.002	0.004	-0.005	0.03	0.11	576	0.04	
		Phase 2	-0.004	0.002	-0.006	0.03	0.03	566	0.03	
										0.013

Table D-9 Emission factors for D on distance specific basis

Test Date	Trace	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	Fuel Economy	PM
			Emission Factors (g/mi)							MPG	
20150209	UDDS_x3	Phase 1	0.049	0.091	-0.029	0.30	5.01	2238	0.04	4.50	
		Phase 2	0.017	0.059	-0.034	0.13	3.07	2169	0.04	4.65	
		Phase 3	0.011	0.040	-0.023	0.12	2.97	2501	0.02	4.03	
											0.0005
20150210	UDDS_x3	Phase 1	0.037	0.049	-0.005	0.27	3.41	2138	0.10	4.71	
		Phase 2	0.018	0.041	-0.017	0.14	3.17	2127	0.11	4.74	
		Phase 3	0.025	0.033	-0.004	0.11	3.48	2141	0.10	4.71	
											0.0008
20150209	Creep_x10	Phase 1	0.180	0.229	-0.017	0.13	3.27	4934	0.47	2.04	
		Phase 2	0.241	0.239	0.036	0.34	12.75	4741	0.39	2.13	
		Phase 3	0.293	0.243	0.085	1.25	20.70	4820	0.39	2.09	
		Phase 4	0.373	0.254	0.155	2.22	18.84	4879	0.36	2.06	
		Phase 5	0.446	0.203	0.274	3.76	26.81	4529	0.32	2.22	
		Phase 6	0.550	0.216	0.368	5.30	22.85	4949	0.30	2.03	
		Phase 7	0.611	0.202	0.441	6.24	29.69	4847	0.32	2.07	
		Phase 8	0.747	0.242	0.543	9.01	26.31	4863	0.30	2.07	
		Phase 9	0.705	0.226	0.515	8.78	33.11	4845	0.32	2.07	
		Phase 10	0.825	0.239	0.625	12.47	32.02	5140	0.33	1.95	
											0.0016
20150210	Creep_x10	Phase 1	0.176	0.346	-0.124	0.94	1.03	4908	0.43	2.05	
		Phase 2	0.157	0.315	-0.115	1.01	7.36	4847	0.42	2.08	
		Phase 3	0.225	0.268	-0.005	1.17	23.44	5157	0.45	1.95	
		Phase 4	0.283	0.246	0.072	3.20	23.34	4470	0.39	2.25	
		Phase 5	0.374	0.223	0.184	2.82	28.20	4766	0.42	2.11	
		Phase 6	0.518	0.240	0.315	5.03	28.34	4783	0.38	2.10	
		Phase 7	0.572	0.230	0.377	8.05	21.66	4672	0.35	2.15	
		Phase 8	0.688	0.245	0.481	13.11	26.18	5228	0.41	1.92	
		Phase 9	0.534	0.167	0.394	6.39	32.70	4819	0.38	2.09	
		Phase 10	0.642	0.162	0.508	9.61	31.50	4809	0.37	2.09	
											0.0045
20150209	Tansient_x4	Phase 1	0.045	0.066	-0.012	0.11	5.12	2705	0.11	3.73	
		Phase 2	0.027	0.051	-0.017	0.17	4.51	2636	0.11	3.82	
		Phase 3	0.034	0.040	0.000	0.12	4.62	2631	0.11	3.83	
		Phase 4	0.029	0.032	0.002	0.14	4.59	2621	0.11	3.85	
											0.0022
20150210	Tansient_x4	Phase 1	-0.022	0.098	-0.108	0.80	6.16	2619	0.10	3.85	
		Phase 2	-0.002	0.091	-0.081	0.20	6.62	2540	0.09	3.97	
		Phase 3	-0.003	0.076	-0.069	0.19	8.81	2651	0.09	3.80	
		Phase 4	0.005	0.045	-0.034	0.23	9.72	2594	0.10	3.88	
											0.0014
20150209	Cruise	Phase 1	0.005	0.012	-0.005	0.05	0.50	1443	0.03	6.99	
											0.0038
20150210	Cruise	Phase 1	0.014	0.011	0.004	0.06	0.81	1444	0.03	6.98	
											0.0019
20150209	HHDDT-S_x2	Phase 1	0.005	0.010	-0.004	0.06	0.87	1733	0.04	5.82	
		Phase 2	0.004	0.006	-0.002	0.06	0.61	1742	0.04	5.79	
											0.0025
20150210	HHDDT-S_x2	Phase 1	0.010	0.010	0.001	0.06	0.87	1747	0.03	5.77	
		Phase 2	0.008	0.007	0.002	0.06	0.66	1754	0.03	5.75	
											0.0028

Table D-10 Emission factors for D on brake specific basis

Test Date	Trace	Phase	THC	CH4	NMHC	CO	NOx	CO2	NH3	PM
			Emission Factors (g/bhp-hr)							
20150209	UDDS_x3	Phase 1	0.013	0.024	-0.008	0.08	1.32	588	0.01	
		Phase 2	0.005	0.017	-0.010	0.04	0.89	631	0.01	
		Phase 3	0.003	0.011	-0.007	0.03	0.85	713	0.01	
										0.0002
20150210	UDDS_x3	Phase 1	0.010	0.013	-0.001	0.07	0.90	566	0.03	
		Phase 2	0.005	0.011	-0.005	0.04	0.89	596	0.03	
		Phase 3	0.007	0.009	-0.001	0.03	0.96	588	0.03	
										0.0002
20150209	Creep_x10	Phase 1	0.030	0.038	-0.003	0.02	0.55	824	0.08	
		Phase 2	0.047	0.046	0.007	0.07	2.46	915	0.08	
		Phase 3	0.052	0.043	0.015	0.22	3.64	848	0.07	
		Phase 4	0.061	0.041	0.025	0.36	3.07	795	0.06	
		Phase 5	0.080	0.036	0.049	0.67	4.79	809	0.06	
		Phase 6	0.096	0.038	0.064	0.93	4.00	865	0.05	
		Phase 7	0.103	0.034	0.074	1.05	5.00	816	0.05	
		Phase 8	0.120	0.039	0.087	1.45	4.22	781	0.05	
		Phase 9	0.117	0.038	0.085	1.46	5.50	804	0.05	
		Phase 10	0.135	0.039	0.102	2.04	5.24	841	0.05	
										0.0003
20150210	Creep_x10	Phase 1	0.027	0.053	-0.019	0.15	0.16	756	0.07	
		Phase 2	0.023	0.046	-0.017	0.15	1.07	702	0.06	
		Phase 3	0.036	0.043	-0.001	0.19	3.79	833	0.07	
		Phase 4	0.057	0.050	0.015	0.65	4.72	904	0.08	
		Phase 5	0.073	0.043	0.036	0.55	5.49	928	0.08	
		Phase 6	0.086	0.040	0.052	0.84	4.71	795	0.06	
		Phase 7	0.100	0.040	0.066	1.41	3.81	821	0.06	
		Phase 8	0.120	0.043	0.084	2.28	4.56	910	0.07	
		Phase 9	0.073	0.023	0.054	0.88	4.50	663	0.05	
		Phase 10	0.110	0.028	0.087	1.64	5.38	821	0.06	
										0.0007
20150209	Tansient_x4	Phase 1	0.010	0.014	-0.003	0.02	1.11	586	0.02	
		Phase 2	0.005	0.010	-0.003	0.03	0.88	512	0.02	
		Phase 3	0.008	0.009	0.000	0.03	1.06	605	0.03	
		Phase 4	0.007	0.007	0.000	0.03	1.03	589	0.02	
										0.0005
20150210	Tansient_x4	Phase 1	-0.005	0.023	-0.025	0.18	1.41	599	0.02	
		Phase 2	0.000	0.022	-0.019	0.05	1.57	603	0.02	
		Phase 3	-0.001	0.018	-0.016	0.04	2.03	612	0.02	
		Phase 4	0.001	0.011	-0.008	0.06	2.35	627	0.02	
										0.0003
20150209	Cruise	Phase 1	0.002	0.005	-0.002	0.02	0.19	558	0.01	
										0.0015
20150210	Cruise	Phase 1	0.005	0.004	0.002	0.02	0.32	571	0.01	
										0.0007
20150209	HHDDT-S_x2	Phase 1	0.002	0.003	-0.001	0.02	0.27	548	0.01	
		Phase 2	0.001	0.002	-0.001	0.02	0.19	545	0.01	
										0.0008
20150210	HHDDT-S_x2	Phase 1	0.003	0.003	0.000	0.02	0.26	532	0.01	
		Phase 2	0.002	0.002	0.001	0.02	0.21	557	0.01	
										0.0009

EXHIBIT 10

EMFAC2011

Technical Documentation

California Environmental Protection Agency

Air Resources Board

September 19, 2011

(Updated January 2013)

CONTENTS

LIST OF FIGURES

LIST OF TABLES

1 INTRODUCTION

EMFAC2011 represents the next step in the ongoing improvement of the EMFAC series of emissions estimation models. The EMFAC2011 release is needed to support the Air Resources Board's (ARB) regulatory and air quality planning efforts and to meet the Federal Highway Administration's transportation planning requirements. EMFAC2011 includes the latest data on California's car and truck fleets and travel activity. The model also reflects the emissions benefits of ARB's recent rulemakings including on-road diesel fleet rules, Pavley Clean Car Standards, and the Low Carbon Fuel Standard.

In order to incorporate the new detailed data and methods to estimate emissions from diesel trucks and buses and future improvements, staff used a modular emissions modeling approach for EMFAC2011 that departs from past EMFAC versions. The first module, named EMFAC2011-LDV, estimates passenger vehicles emissions. A second module, called EMFAC2011-HD, estimates emissions from diesel trucks and buses. A third module integrates the output of EMFAC2011-LDV and EMFAC2011-HD and provides users with the ability to conduct scenario assessments for air quality and transportation planning. This third module is called EMFAC2011-SG. Together the three modules comprise EMFAC2011.

This section describes and summarizes the major updates to this version of the model. The rest of the document provides a complete discussion of all the updates, revisions, and additions to EMFAC2011, and to the new ARB on-line data tool. Most importantly, the on-line tool is designed to cover most data needs for both summarized and detailed emissions and emission rates. The vast majority of EMFAC users will no longer have to download, learn, and run the model to get the data they need. Much simpler data queries across the internet will suffice.

In July 2012 EMFAC2011 was re-released to highlight new web-database tools that provided improved capabilities for project level assessment. In addition EMFAC2011-LDV and EMFAC2011-SG were updated to correct the number of starts in Santa Clara County for several small categories of gasoline vehicle classes which led to an overestimate of emissions in Santa Clara County. No other counties were affected, and no model algorithms were changed in the update.

New Data

The most important improvement in EMFAC2011 is the integration of the new data and methods to estimate emissions from diesel trucks and buses. EMFAC2011 uses the

same diesel truck and bus vehicle populations, miles traveled and other emissions-related factors developed for the Truck and Bus Rule approved by the Air Resources Board in 2010. EMFAC2011 still provides the same traditional emissions rate detail for these vehicles that user are familiar with in previous EMFAC versions. The model includes the emissions benefits of the truck and bus rule and the previously adopted rules for other on-road diesel equipment. Finally, the impacts of the recession on emissions that were quantified as part of the truck and bus rulemaking are included.

Light-duty motor vehicle fleet age, vehicle type, and vehicle population in EMFAC2011 is based on 2009 California Department of Motor Vehicles data. These data along with the new diesel truck and bus data, satisfies guidance issued by the U.S. Department of Transportation, Federal Highway Administration that requires that vehicle fleet data used in transportation conformity analyses be no older than 5 years.

As in previous releases of EMFAC, travel activity data (which includes vehicle miles traveled, trips, and distributions of vehicle miles traveled by speed and time period) are provided by metropolitan planning organizations (MPOs) that conduct travel demand modeling for the transportation planning process. For EMFAC2011, updated travel activity data was provided by the Southern California Association of Governments (SCAG) for the greater Los Angeles region, by the San Diego Association of Governments (SANDAG) for San Diego County, by the eight San Joaquin Valley MPOs, by the Metropolitan Transportation Commission (MTC) for the Bay Area, and by the Sacramento Area Council of Governments (SACOG) for the greater Sacramento region.

New Modular Model Structure

The new EMFAC2011 comprises a suite of three modules that estimates emissions from diesel trucks, buses, and gasoline powered vehicles. Staff has moved to a modular model structure to accommodate more detailed information about the truck and bus fleet than has been in prior EMFAC versions. The modular structure will also more easily accommodate future model enhancements that are necessary to support on-going program development associated with criteria and greenhouse gas emissions.

The first module, named EMFAC2011-LDV, is used as the basis for estimating emissions from gasoline powered on-road vehicles, diesel vehicles below 14,000 pounds gross vehicle weight rating, and urban transit buses. The algorithms used in EMFAC2011-LDV for passenger cars are the same as used in EMFAC2007

The second module, called EMFAC2011-HD, is the basis for emissions estimates for diesel trucks and buses with a gross vehicle weight rating greater than 14,000 pounds operating in California. This model is based upon the Statewide Truck and Bus Rule emissions inventory that was developed between 2007 and 2010 and approved by the Air Resources Board in December 2010. The truck and bus population and vehicle

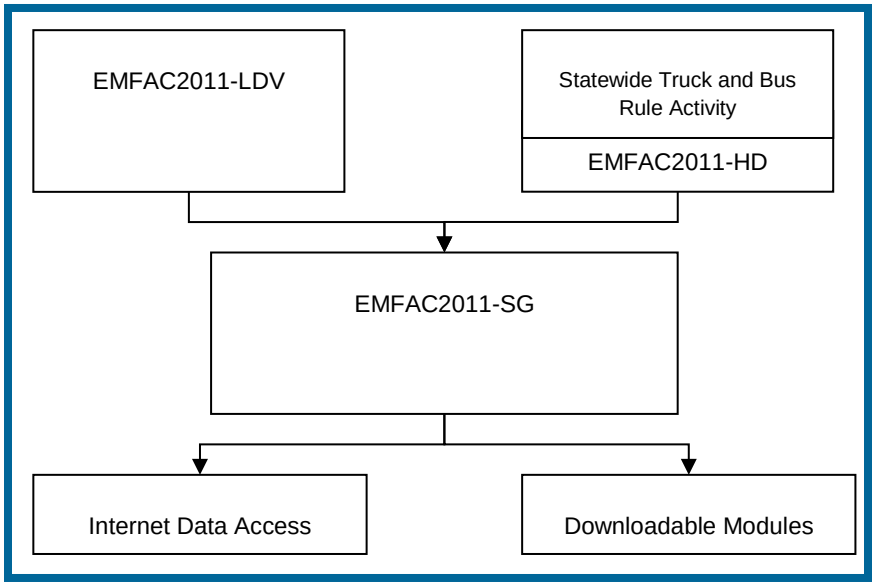
miles traveled estimates in EMFAC2011-HD are identical to those presented to the Board. Emission factors in the Statewide Truck and Bus Rule inventory were based on statewide, annual average emission factors and adjusted for improvements to fuel economy, medium-heavy duty truck emission factors, and other new information. EMFAC2011-HD emissions estimates include all of the improvements made as part of the Statewide Truck and Bus Rule inventory and then add the traditional detailed EMFAC methods that provide emission rates for annual, summer, and winter seasons on a regional basis.

Finally, EMFAC2011 contains a new simplified tool, called EMFAC2011-SG, that provides air quality planners, transportation planners, and other EMFAC users a tool for assessing emissions under different future growth scenarios. This includes conformity analyses of transportation plans and programs with the State Implementation Plans required by federal law, State Implementation Plan inventories, alternative growth scenarios associated with regional transportation planning for greenhouse gas reductions (SB375), and the like.

EMFAC2011-SG takes the output from EMFAC2011-LDV and EMFAC2011-HD and applies scaling factors to estimate emissions consistent with user-defined vehicle miles of travel (VMT) and speeds. The EMFAC2011-SG module also estimates the benefits of Pavley and Low Carbon Fuel Standard regulations.

EMFAC2011's enhanced modular structure and computation requirements can make running the model more resource-intensive than the previous EMFAC release. Therefore, ARB is enhancing data availability by providing a new database through the ARB mobile source emissions inventory web site that provides regional population, activity, emissions, and emission rates at varying levels of detail. Most current EMFAC users will find the new database sufficient for obtaining emissions data to support California Environmental Quality Act (CEQA) assessments and other types of analyses for which the EMFAC model is currently used and will not need to run the model. The model executable files will also remain publicly available on our website, which is located at **http://www.arb.ca.gov/msei/msei.htm**. In July 2012 these tools were updated based on user comments provided to our web site. Figure 1-1 provides a schematic that describes how the modules fit together.

Figure 1-1. EMFAC2011 Schematic



Beyond EMFAC2011

EMFAC2011 is a transition step to a future EMFAC emissions modeling system under development to meet the increasingly demanding needs for data in both the regulatory and planning arenas. Some of the areas of focus for the future EMFAC system that staff is developing over the next two to three years include:

Population, activity, and forecasting:

- Improved capture of recession and fuel price spikes.
- Reflect planned GHG emissions standards and their impact on future year fleet mix.

New Smog Check Algorithms

- Reflect the new Smog Check mandated by State law starting in 2013.

Flexibility for air quality planning better support for long-term scenario assessment

- Scenario development support for tighter National Ambient Air Quality Standards.
- SB375 –Model structure to better support for smart growth planning.
- Better conformity methods to support MPO conformity determinations.

Improved documentation and public availability of model output:

Future model output will be available in raw and processed form over the internet in ways that require minimal processing to understand.

2 USING THE MODULES AND ACCESSING DATA

The EMFAC model has always been used by a wide cross-section of air quality professionals. Typical uses include:

- ARB and air district staff routinely develop emissions inventories to support air quality planning and to meet air quality standards;
- Regional transportation planning agencies that use EMFAC to test whether or not new transportation plans fall within conformity budgets;
- Statewide and Regional transportation planning agencies evaluating land-use changes that impact regional VMT estimates and their impact on emissions;
- ARB staff developing assessments to support regulatory program development;
- ARB and regional air district staff developing emissions inventories to support health risk assessments; and
- Consultants developing fleet average or by model year emission rates for local scale modeling to support project level assessments.

With EMFAC2011, staff has tried to anticipate data needs and to serve common data requests in the form of a web site data serving tool instead of relying solely upon executable models. These tools provide several different ways to access and summarize both emissions and emission rates to the desired level of detail.

The mobile source emissions inventory website is located at

http://www.arb.ca.gov/msei/msei.htm

On the website the user will find EMFAC2011 including each inventory module, technical documentation, and training resources. The website also contains similar information associated with the myriad of different types of mobile sources from cars and trucks to lawnmowers, construction equipment, and other sources.

Instructions on operating each EMFAC2011 module as well as technical documentation of the modules and data improvements are available in this document; locations and information are described in documentation below for each module in the EMFAC2011 suite.

3 EMFAC2011-LDV

3.1 WHEN THE MODULE SHOULD BE USED

EMFAC2011-LDV estimates emissions for gasoline vehicles, urban transit buses, and diesel vehicles less than 14,000 pounds GVWR. This represents a change from EMFAC2007 when a single tool covered all on-road categories. As a result, EMFAC2011-LDV cannot be used by itself to develop a comprehensive statewide on-road vehicle emissions inventory.

To obtain a comprehensive inventory, a user must use additional modules in the EMFAC2011 suite. The easiest way to access EMFAC2011 emissions is to use the new web based data tools available through our website. Alternatively, a user may choose to run EMFAC2011-LDV and EMFAC2011-SG separately, or to run EMFAC2011-SG. In most cases, data served through the internet should be sufficient to answer many user questions without running the model.

EMFAC2011-LDV may be run to obtain emission rates for specific meteorological and speed conditions, and to generate input files for photochemical modeling.

3.2 USER'S GUIDE TO RUNNING THE MODULE

The fundamental design of the EMFAC2011-LDV executable file is the same as EMFAC2007. A comprehensive user's guide was developed for EMFAC2007 and may be used; it is available on our website.

3.3 MODULE IMPROVEMENTS

Many improvements have been made to the EMFAC model between the release of EMFAC2007 and the updated EMFAC2011-LDV. Population, vehicle miles traveled (VMT), speed distributions, vehicle survival curves, and portions of emission rates have all been updated. This section provides an overview to improvements; major improvements are discussed in greater detail in the technical appendices to this document.

3.3.1 POPULATION

In the EMFAC2011-LDV module, vehicle populations are estimated using registration data from the Department of Motor Vehicles. Data from the 2009 registration year was used to update the populations in each vehicle class for 45 age groups and 69 geographic areas. Based on the 2009 registration data, there are approximately 25 million registered vehicles operating in California.

Staff developed an improved methodology for classifying vehicles among vehicle classes. This methodology involves matching DMV registration information to Smog Check data from the Bureau of Automotive Repair and using two different vehicle identification number (VIN) decoders to interpret registration data. The more extensive use of VIN decoders led to a marked improvement in the classification of vehicles among the light-duty truck categories, which are for the most part sport utility vehicles and lighter pick-up trucks. These improvements are apparent in the model from calendar year 2009 and into the future.

Staff retained the historical fleet mix and population estimates embedded in the model for calendar year 2000 and into the past. For calendar year 2000 through 2008, staff connected the historical population estimates based on the previous DMV processing methodology with the new population estimates developed using the updated DMV processing methodology. The impact of this methodology on emissions is not significant when viewed across all vehicle categories. However for a few categories, most notably light-heavy duty vehicles, the difference in fleet mix is significant between before and after calendar year 2009.

3.3.2 VMT AND SPEED DISTRIBUTIONS

For air quality and transportation planning purposes, EMFAC2011-LDV uses the VMT provided by regional transportation planning agencies (RTPA). For EMFAC 2011, ARB received VMT and speed submittals from the Southern California Association of Governments (SCAG), Bay Area Metropolitan Transportation Commission (MTC), San Diego Association of Governments (SANDAG), and San Joaquin Valley Councils of Government. In the absence of recent RTPA data, the model contains default speed distributions and estimated VMT as a function of vehicle population (from DMV) and mileage accrual rates (from the Bureau of Automotive Repair SmogCheck program).

In general regional transportation planning agencies did not reflect the impact of the economic cycle on VMT, and instead dampened future VMT forecasts in light of the slower economic recovery from the recent economic recession. As a result the recession is generally handled through long term rather than short-term forecasts in EMFAC2011-LDV.

Unlike previous versions of EMFAC, VMT estimates from medium and heavy heavy-duty diesel trucks and diesel bus categories are estimated directly from EMFAC2011-HD. When developing these VMT estimates staff accounted for both the short and long term impacts of the economic recession.

3.3.3 EMISSION RATES

3.3.3.1 CARBON DIOXIDE

EMFAC2007 outputs carbon dioxide (CO2) emissions and estimated fuel use using results from chassis dynamometer exhaust tests on the federal test procedure (FTP) assuming fuels oxygenated with MtBE. To update these assumptions for EMFAC2011-LDV, staff analyzed test data from the Unified Cycle (UC) using modern ethanol/gasoline blends consistent with RFG3. The update results in a small change in predicted CO2 emission rates. Details are available in Section 8.

3.3.3.2 CARBON DIOXIDE DISBENEFIT WITH AIR CONDITIONING USE

EMFAC2011-LDV estimates carbon dioxide emissions as a function of the use of air conditioning systems among other factors. The assumed use of vehicle air conditioning is a function of the temperature and relative humidity in the geographical area being modeled. In developing EMFAC2011, staff re-evaluated the algorithm to correctly reference the factors in the calculation.

EMFAC2011-LDV uses a correlation between emissions results on the FTP with 10% additional aerodynamic load and the UC with air conditioning turned on. The correlation has a slope and an intercept term. The intercept (as the ratio of intercept to slope) is added to the BER (basic emission rate) in the model. For LDVs, and MDVs, UC bag 2 (hot, stabilized) emission factors are used as BERs. For all other vehicle types, the average of the FTP Bag 2 (hot, stabilized) and Bag 3 (warm start) results is used. The program was applying the BER twice for LDVs and MDVs, rather than only applying the UC Bag 2 BER. This change led to a decrease of 30,000 tons/day CO2 statewide which is a 5% decrease in emissions from what was previously estimated.

3.3.3.3 EVAPORATIVE DIURNAL BASIC EMISSION RATE COEFFICIENTS

In EMFAC2011-LDV, hydrocarbon evaporative diurnal emission rates are modeled as polynomial equations dependent on ambient temperature and other factors. The polynomial equations generate a conversion factor that converts an evaporative base emission rate to an emission rate at a given temperature. In updating EMFAC2011-LDV, staff determined that several polynomial coefficients in the model were very small and the EMFAC model was not carrying sufficient significant figures in the coefficients. EMFAC2011 now uses additional significant figures in this calculation, which results in an increase of ROG emissions of 0.3 tpd statewide in 2007. The effect is larger in later years (because more zero evap vehicles are present), but it is still a very small effect.

3.3.3.4 EVAPORATIVE RESTING TIME IN HOT SOAK ALGORITHM

In EMFAC, hydrocarbon evaporative emissions generated through the hot soak process are calculated for both full hot soak events (the engine fully cools back to ambient) and partial hot soaks (the engine partially cools until the next start event) using a matrix of resting time values. During evaluation of the evaporative emissions processes for development of EMFAC2011, staff identified that hot soak emissions in EMFAC had been programmed using the time-off matrix of soak times before a cold start. This matrix represented the pre-start soak time as a function of time of day. EMFAC should have been programmed with the time-rest matrix of soak times after a key-off event by time of day. This correction results in an increase of ROG emissions of 8.5 tons/day statewide, which is a 1% increase in emissions.

3.3.3.5 BRAKE WEAR PM

EMFAC2007 estimated total particulate matter for brake wear using an emission factor of 12.8 mg/mile. This emission factor was applied to all vehicle classes on a per mile basis, and reflected emissions testing of asbestos friction-materials from automobile disc brakes. To update EMFAC2011-LDV, staff used two more recent studies performed on new brake materials that replaced the older asbestos based materials. Results from these studies were correlated with wheel load, braking speed, material, and number of brakes per vehicle.

In addition to updating the emission rate during braking events, staff used the same driving cycles upon which exhaust emission rates are derived to estimate the frequency and severity of braking events during driving in California. Staff used the unified cycle for light-duty vehicles, and the transient and cruise cycles for heavy-duty vehicles. Overall, brake wear emissions increase substantially in EMFAC2011-LDV as a result of this update. Details are available in Section 9.

3.3.3.6 GASOLINE EXHAUST PARTICULATE MATTER

Basic emission rates for light duty gasoline vehicles have been updated using data from EPA's project titled 'The Kansas City PM Characterization Study'. These vehicles were certified to Federal Standards. Emissions data was collected under conditions prescribed in the Federal Test Procedures, and vehicles were operated over the LA92 Unified Driving Cycle. Data for newer model years was supplemented by testing conducted at ARB's Haagen Smit Laboratory.

The current LEV II standard for PM is 10 mg/mi and will be in effect until 2016. As these standards are further reduced, we expect various engine technologies to penetrate in the fleet. For example, the percentage of gasoline direct injection (GDI) engines is expected to increase from current levels in order to meet the GHG emission targets. These effects and other results are detailed in Section 10.

3.3.4 OTHER IMPROVEMENTS

3.3.4.1 ELIMINATION OF TRUCK AND BUS CATEGORIES

The vehicle classes in EMFAC2011-LDV that are covered by EMFAC2011-HD are depopulated and effectively turned off in EMFAC2011-LDV since the official inventory for truck and bus categories will be generated by EMFAC2011-HD.

3.3.4.2 VEHICLE SURVIVAL

In the process of developing revised assumptions, staff evaluated vehicle survival curves in EMFAC2007 against curves that were derived using more recent DMV data. In most cases the survival curves derived from the new DMV data were similar to those in EMFAC2007 and as a result no changes were required. However, in two categories (light duty automobiles and light duty trucks 2) survival curves developed using 2005-2009 data were different than those included in EMFAC2007. Survival curves for those two categories were updated in EMFAC2011-LDV as a result. A comparison of old and new survival curves for these categories are shown in Figure 3-1 and Figure 3-2 below.

Figure 3-1. Light Duty Automobile Vehicle Survival Curves: EMFAC2007 vs. EMFAC2011-LDV

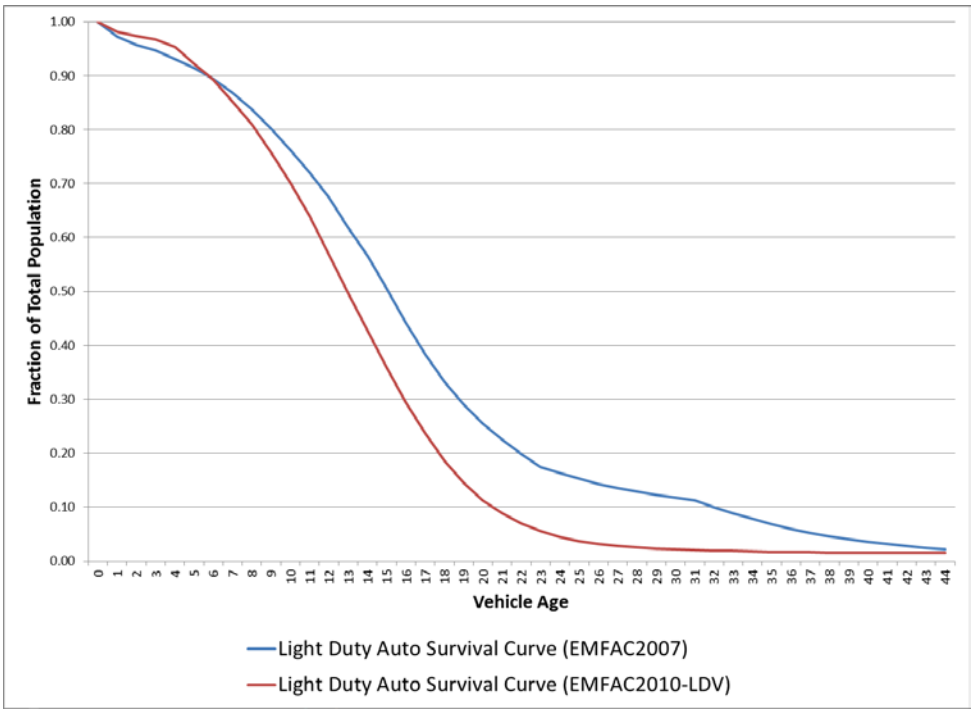
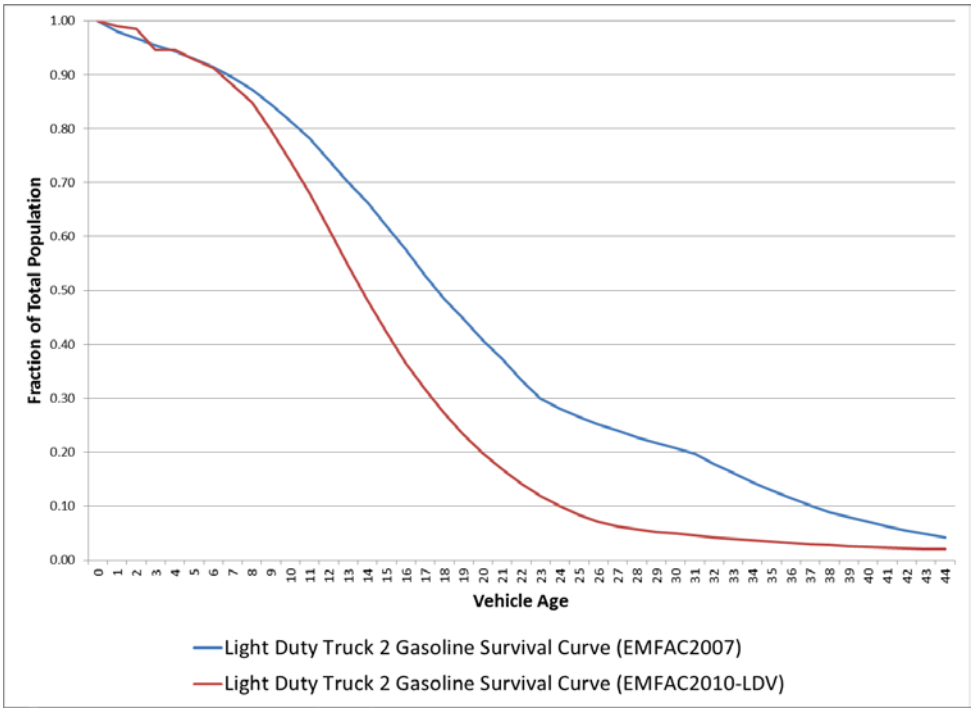


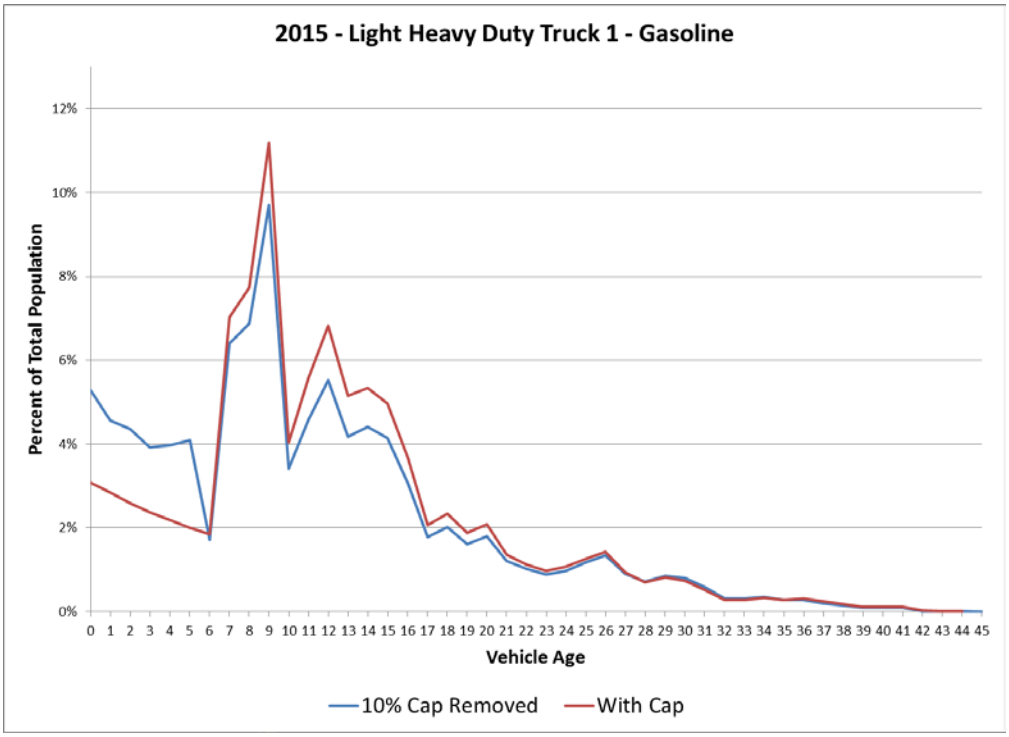
Figure 3-2. Light Duty Truck 2 Vehicle Survival Curves: EMFAC2007 vs. EMFAC2011-LDV



In EMFAC2007 and prior versions of the EMFAC model, new vehicle sales in a given calendar year and vehicle class were a function of the overall population growth rate, the total population in the vehicle class and calendar year, the survival curve for the vehicle class, and a 10% limit on increase in year to year new vehicle sales in any vehicle class. If the 10% cap were calculated to be exceeded, the algorithm capped the increase in new vehicle sales at 10% and allocated the remaining new vehicles to older ages.

One of the impacts of the 2007-2009 economic recession was a major decrease in new vehicle sales with the lowest level of vehicle sales occurring in calendar year 2009. New vehicle sales decreased 20% in light duty automobiles and 75% in light heavy duty trucks relative to 2007. When evaluating EMFAC2011-LDV, it became clear that the 10% cap on increase in new vehicle sales was artificially depressing new vehicle sales in the future and as a result artificially aging the fleet in an unrealistic manner. As a result, that cap was removed. This is illustrated in Figure 3-3. Without the cap, new vehicle sales cannot rise above 10% resulting in a slow smooth increase from 2009, and enhanced survival in older ages. With the cap removed, the survival curve is impacting the population as it should in older ages, and new vehicle sales are more in-line with historical trends.

Figure 3-3. 2015 Light Heavy Duty Truck 1 – Gasoline: Comparison Using EMFAC2007 Approach (10% Cap in Place) Against EMFAC2011-LDV Approach (Cap Removed)



3.3.4.3 LIFETIME MILEAGE

In the EMFAC model, emission rates are a function of the cumulative mileage on the vehicle through an emissions process called deterioration. Cumulative mileage was calculated in EMFAC2007 assuming that a vehicle's cumulative lifetime mileage, which can generally be measured using an odometer, is the sum of the average vehicle's mileage accrual schedule over time. Analysis of available odometer data and academic studies[1] for cars and trucks demonstrated that this assumption is incorrect. The reason for this difference is that when a vehicle is young, the average odometer of a group of similarly aged vehicles in a class is the average of some high accruing and some low accruing vehicles. As vehicles age, higher mileage vehicles are scrapped more quickly than lower mileage vehicles. So, surviving vehicles are more likely to have accrued fewer miles than average when those vehicles were younger. The net result is that the modeled average odometer at a given age is a function of both accrual rates up to that age, and survival rates up to that age for the vehicle class. Observed odometer readings in practice tend to level out to a constant level for older vehicles.

To make this change in the model, staff updated the way in which odometer is calculated in EMFAC. In the new method integrated into EMFAC2011, modeled odometer or lifetime mileage is a function of both mileage accrual rates and vehicle survival rates in the following equation:

$$\text{ODOMETER (10)} = \sum_{i=1}^{10} accrual(i) * survival(i)$$

This change leads to a small change in emissions in any calendar year, but effectively places a cap on deteriorated emission factors in older vehicles. Details on the emissions impact are available in Section 12.16.

3.3.4.4 TEMPERATURE AND RELATIVE HUMIDITY PROFILES

Due to the importance of environmental conditions on emissions, correction factors are used in EMFAC to adjust the laboratory-based emissions rates when actual ambient environmental conditions differ from the standard laboratory conditions under which emissions testing is conducted. More specifically, correction factors for ambient temperature and relative humidity are used to adjust base-rate exhaust emissions (running and starts, especially starts) and evaporative emissions (i.e., diurnal, hot-soak, running loss and resting loss) for non-standard conditions. Emission rates are adjusted for ambient temperature conditions that vary from 75 degrees Fahrenheit or absolute humidity that varies from 75 grains of water per pound of dry air (gr/lb). The default profiles are based on region-specific meteorological conditions that have occurred on days that ambient pollutant concentrations challenge attainment and maintenance of air

[1] Greenspan, A., Cohen, D, (1996). Motor Vehicle Stocks, Scrappage, and Sales. October 30, 1996. Available at: http://www.federalreserve.gov/pubs/feds/1996/199640/199640pap.pdf.

quality standards in a region, specifically the new federal 8-hour standard and the federal 24-hour $PM_{2.5}$ standards.

For each region, approximately 24 total days over 12 years (1996-2007) were selected for analysis. Key differences between this update and prior updates to the default temperature and relative humidity profiles in the models are as follows:

Profile Scenarios. The current update addresses four scenarios of environmental conditions:

- **ozone** exceedance conditions based on the new federal 8hr ozone standard;
- **PM** exceedance conditions based on the new federal 24-hour $PM_{2.5}$ standard;
- **monthly** average ambient environmental conditions; and
- **annual** average (based on monthly average data).

The last update to default temperature and relative humidity profiles occurred in late 2006, which only addressed summertime conditions based on data analyses spanning March through October.

Spatial-Weighting Schemes. In the last update, EMFAC summer profiles were developed with VMT weighting and simple averaging. In this current update, the spatial weighting by VMT is still used for EMFAC profile development.

Relative Humidity Calculation. Previously, temperature (T) and relative humidity (RH) profiles were developed independent from each other using the same selected days of simultaneously-collected T and RH data. Thus, these matched pairs of T and RH were dissociated and then averaged independently. A deficiency with this approach is that it allows the known, dependent relationship between temperature and pressure to be broken. To fix this methodological deficiency and better maintain the site- and day-specific relationship between temperature and relative humidity in this update, a different methodology was used. With this new method and for a specific monitoring location, hourly temperature averages are calculated first from simultaneously-collected pairs of T and RH values. Next, a relative humidity value for each of the resulting averaged temperature values is selected or interpolated from the original, matched pairs of T and RH via cross-referencing on temperature values. Because NOx emissions are sensitive to humidity, this change in methodology affects NOx emission estimates.

Data Period of Record and Screening Criterion. The Federal 8-hour ozone standard and 24-hour $PM_{2.5}$ standard have been revised since the previous update of temperature and relative humidity profiles. This update utilizes the present Federal 8-hour ozone and 24-hour $PM_{2.5}$ standards.

Use of Imputed Data. Missing data at one site are imputed if the data at that site are systematically related to the data measured for at least one other site. Using imputed data helped raise the number of sites with “complete” data from 1996 to 2007 from less than 100 to 156 sites.

Overall, the updated temperature and relative humidity profiles lead to +/- 5% change for ROG and NOx for ozone (summer) and PM (winter) profiles relative to previous estimates in EMFAC2007.

3.3.4.5 UPDATE TO NUMBER OF LIGHT-HEAVY DUTY VEHICLE STARTS

Emissions from gasoline powered vehicles during starts are higher than during normal vehicle operation because control equipment does not function optimally when a vehicle is cold. EMFAC2007 assumed 33 trips per day for gasoline powered light-heavy duty trucks, which are defined as having a gross vehicle weight rating between 8500 and 10,000 pounds. Staff reviewed this assumption, and found that given the uses for these vehicles the number of starts was excessive, leaving roughly 2 miles of driving per start and a mean time between trips of 15 minutes or less.

Staff re-evaluated the original data that were used to develop vehicle start estimates and found that Federal Express and United Parcel Service trucks seemed to be over-represented in the gasoline vehicle sample. When data were evaluated across both diesel and gasoline vehicles, staff estimated 15 starts per day. That value was used to update EMFAC2011-LDV.

3.3.4.6 UPDATE TO ASSUMED TO COMPLIANCE WITH THE LEV2 REGULATION

In November 1998 staff adopted new light duty vehicle emission standards (LEV2) that began implementation in 2004. In EMFAC2000 staff integrated the compliance pathways into EMFAC based on assumptions developed during rulemaking. The LEV2 standards set an average emissions target across vehicle classes, which allowed vehicle manufacturers some room to manufacturer different combination of vehicle technologies to meet the standard. In developing EMFAC2011 staff reviewed these assumptions.

Three updates were made. First, staff identified that when compared to certification data EMFAC assumed that by 2010 about 70% of new passenger cars would be certified to the partial zero emission vehicle (PZEV) standard, and be partially offset by a small fraction of dirtier vehicles. In reality, vehicle manufacturers sold 20% PZEV and 70% ultra-low emission vehicles or ULEV. In essence vehicle manufacturers sold a different mix of cars than assumed in EMFAC2007, but that met the same LEV2 emissions standards. Second, staff updated light-duty diesel sales fractions based on actual sales data. Since diesel cars sold after 2004 had to meet gasoline vehicle emissions standards, this had no impact on emissions. Finally, staff integrated new assumptions about penetration of zero-emission vehicles. EMFAC2007 had assumed a small fraction of ZEVs would be sold in the previous decade. Those sales were overestimated, and staff updated ZEV sales with actual sales data. Staff also updated ZEV forecasts between 2010 and 2017. Some electric vehicles are expected to be available because of the advent of plug-in hybrids that provide a relatively long all-electric range. Details are available in Sections 12.12 and 12.18.

3.3.4.7 JULY 2012 UPDATE TO SANTA CLARA COUNTY

In July 2012 corrected the number of starts in the LDV module code for gasoline urban bus, school bus, other bus, motorcycle, and motorhome vehicle categories in Santa Clara County. The number of starts in Santa Clara County had been incorrectly entered into the model code, which led to an overestimate of ROG emissions in Santa Clara County. On a statewide basis, fixing the error decreases ROG emissions by roughly 2%. The following table highlights the updated assumptions.

Santa Clara Summer Episodic On-Road Motor Vehicle Trips (Gas Fuel) (Calculated Using EMFAC2011 SG ver 1.0)									
Cal. Year	**1990**	**2000**	**2005**	**2007**	**2010**	**2014**	**2017**	**2020**	**2023**
LDA - GAS	3,790,078	4,530,263	4,195,684	4,362,438	4,553,513	4,708,469	4,834,715	4,975,065	5,127,224
LDT1 - GAS	889,237	1,046,089	432,348	448,152	468,092	477,033	488,910	504,987	519,104
LDT2 - GAS	827,975	1,229,457	1,347,456	1,398,502	1,452,201	1,488,956	1,525,961	1,563,504	1,605,381
LHD1 - GAS	40,422	39,533	347,799	364,489	382,184	391,548	400,853	410,416	419,958
LHD2 - GAS	78,479	63,500	35,727	34,535	34,643	35,437	36,255	37,106	37,850
MCY - GAS	1,746,805	1,078,474	1,267,800	1,316,968	1,376,328	1,427,789	1,484,933	1,550,751	1,611,305
MDV - GAS	119,039	263,342	898,861	932,395	965,064	984,292	1,001,985	1,020,300	1,036,812
MH - GAS	393,704	493,406	240,025	240,169	243,206	251,399	261,594	266,353	272,109
OBUS - GAS	10,780	9,920	26,459	27,659	28,816	29,624	30,311	30,894	31,351
SBUS - GAS	5,116	6,029	5,764	6,242	6,651	7,224	7,403	7,304	7,226
T6TS - GAS	67,295	62,531	30,459	31,669	33,223	34,407	35,335	36,275	37,076
T7IS - GAS	9,569	8,918	2,244	2,333	2,434	2,435	2,402	2,373	2,326
UBUS - GAS	4,053	2,356	1,701	1,810	1,891	1,839	1,936	1,979	2,028

Santa Clara Summer Episodic On-Road Motor Vehicle Trips (Gas Fuel) (Calculated Using EMFAC2011 SG ver 1.1)									
Cal. Year	**1990**	**2000**	**2005**	**2007**	**2010**	**2014**	**2017**	**2020**	**2023**
LDA - GAS	3,790,078	4,530,263	4,195,684	4,362,438	4,553,513	4,708,469	4,834,715	4,975,065	5,127,224
LDT1 - GAS	889,237	1,046,089	432,348	448,152	468,092	477,033	488,910	504,987	519,104
LDT2 - GAS	827,975	1,229,457	1,347,456	1,398,502	1,452,201	1,488,956	1,525,961	1,563,504	1,605,381
LHD1 - GAS	40,422	39,533	347,799	364,489	382,184	391,548	400,853	410,416	419,958
LHD2 - GAS	78,479	63,500	35,727	34,535	34,643	35,437	36,255	37,106	37,850
MCY - GAS	76,556	47,335	55,977	58,200	60,818	62,870	65,258	68,064	70,669
MDV - GAS	119,039	263,342	898,861	932,395	965,064	984,292	1,001,985	1,020,300	1,036,812
MH - GAS	863	1,092	543	550	562	574	587	601	613
OBUS - GAS	10,791	10,027	26,868	27,935	29,041	29,755	30,361	30,951	31,413
SBUS - GAS	568	674	611	635	656	662	677	695	709
T6TS - GAS	67,295	62,531	30,459	31,669	33,223	34,407	35,335	36,275	37,076
T7IS - GAS	9,569	8,918	2,244	2,333	2,434	2,435	2,402	2,373	2,326
UBUS - GAS	365	212	156	168	175	177	180	185	189

4 EMFAC2011-HD

4.1 INTRODUCTION AND DESIGN

Commercial heavy-duty diesel trucks and buses are defined as commercial diesel buses and trucks exceeding 14,000 pounds gross vehicle weight rating (GVWR). Combined these categories are the single largest source of NOx and diesel PM2.5 in California.

Beginning in 2007, staff began the process of redesigning the truck and bus emissions inventory in order to support development of the Drayage Truck and Statewide Truck and Bus Rules. These rules were ultimately adopted and amended between 2008 and 2010. In developing this new analysis, staff integrated new data and assumptions into an expanded methodology that builds upon modeling techniques in embedded EMFAC2007. The expanded approach accounts for the differences in trucking and busing operations which are a function of vehicle registration type, body type, and vocation.

EMFAC2011-HD is written in two parts. The activity portion of the inventory, which includes current, back-casted, and forecasted population / VMT is written in Microsoft Access. These activity estimates are the same as those assumed for the Truck and Bus Rule Amendments adopted in December 2010. The emission factor portion of EMFAC2011-HD is written in Visual Basic and MySQL. Whereas the December 2010 regulatory inventory relied upon statewide annual average emission rates adjusted for several updates, EMFAC2011-HD incorporates the same updates (as well as additional updates to temperature, relative humidity profiles, and regional speed distributions) into the full programming breadth that was previously included in the EMFAC model.

4.2 BASIC METHODOLOGY

Emissions are calculated as the product of a population of vehicles, the number of miles each vehicle travels, and emission rates per mile as shown below. Beneath this simple equation lies a series of data and assumptions about the population, miles traveled, and emission rates per vehicle model year in a given calendar year, growth and attrition estimates, deterioration, and other factors that affect emissions estimates, all of which is described in documentation developed for the Truck and Bus Rule or in this document. We applied the concept separately for each analysis category of trucks that shares similar travel, service, size, age or other characteristics.

$$\sum_{MY,\,C} \left(POP_{MY,\,C} \times AC_{MY,\,C} \times ER_{MY,\,C,\,R}\right) = EMS_{CY},$$

where: $POP_{MY,\,C}$ is the population of trucks for model year MY within each analysis category C for a given calendar year;

$AC_{MY,\,C}$ is the accrual rate (miles traveled per year) per truck by model year MY and analysis category C in a given calendar year;

$ER_{MY,\,C}$ is the calculated emission rate, in grams pollutant per mile driven, assuming regional speed distributions, regional temperature and relative humidity profiles by hour of day, and category specific cumulative mileage accrual over the life of the truck, by model year MY, analysis category C, and region R;

EMS_{CY} is the emissions calculated in tons per day for a given calendar year.

4.3 WHEN THE MODULE SHOULD BE USED

EMFAC2011-HD estimates emissions for commercial vehicles equipped with medium heavy or heavy-heavy duty diesel engines. The module outputs emissions from these categories assuming the benefits of recently adopted Rules. A user can run EMFAC2011-HD separately, or to obtain emissions estimates across all vehicle categories within a region, a user can use the new on-line database of EMFAC2011 output available through our website, may use the EMFAC2011-SG, or may run EMFAC2011-LDV and EMFAC2011-HD separately. In most cases, data served through the internet should be sufficient to answer many user questions without running the model.

4.4 RELATIONSHIP TO REGULATORY INVENTORIES

Population and activity estimates for years covered by the regulatory inventory are identical to those assumed for the Truck and Bus Rule amendments that were adopted by the Board in December 2010. Additional activity estimates have been made for the prior to 2000 and after 2025 so that emissions can be provided from the model for the full intended range of EMFAC2011: 1990-2035. EMFAC2011-HD generates emission rates using the same algorithm as EMFAC2007, but incorporates all of the emission factor related updates made as part of the regulatory inventory (chip reflash, updated medium heavy-duty base emission factors, etc.), and includes updated speed, temperature, and relative humidity profiles developed for EMFAC2011. More information on the truck and bus rules is available at: http://www.arb.ca.gov/msprog/onrdiesel/onrdiesel.htm.

4.5 ACTIVITY

Activity estimates are identical to those assumed for the Statewide Truck and Bus Rule Amendments in 2010 and cover the years 2000, 2002, 2004, 2005-2025. Detailed documentation on the methods and assumptions used are available at: **http://www.arb.ca.gov/regact/2010/truckbus10/truckbusappg.pdf;** **http://www.arb.ca.gov/msprog/onrdiesel/ab1085compliance.htm;** and **http://www.arb.ca.gov/regact/2008/truckbus08/appg.pdf.**

4.5.1 FORECAST/BACKCAST OUTSIDE OF REGULATORY INVENTORY

For forecasting activity beyond 2025, the methodology for growth trend follows the same approach as the regulatory inventory. For most of the medium and heavy-heavy duty diesel truck inventory, we assumed the same year to year growth rates as in EMFAC2007. Growth of Statewide construction truck fleets activity is based on the construction activity growth and the activity distributions to air basins are based on population growth. For activity prior to 2000, we used the trend of California diesel fuel sales to on-highway consumers between 1990 and 2000 from Energy Information Administration (EIA, California No 2 Diesel Adjusted Sales/Deliveries to On-Highway Consumers). The back-casted statewide activities for construction truck fleets followed the EIA fuel sales trends as most other truck fleets and distributed to air basin based on population growth. The activities outside of regulatory inventory for buses, drayage trucks, agricultural trucks, public and utility fleets and solid collection vehicles are assumed to follow their respective long term growth trends. As in the regulatory inventory, sales adjustment factors developed based on new truck sales were used to adjust the average age distribution in the baseline. We assume sales adjustment factors are 1 for all pre-1985 models and used the average of the newest five model year sales adjustments in the regulatory inventory for all additional forecast model years.

4.6 EMISSION FACTORS

In developing the Statewide Truck and Bus Rule emissions inventory, staff revised the emission factors for medium heavy-duty diesel trucks (MHDDT), made adjustment to the NOx emission rates of 2006-2011 model year heavy heavy-duty diesel trucks (HHDDT) and MHDDT, and updated the HHDDT and MHDDT CO2 emission rates as well as the HHDDT NOx idle emission rates. These adjustments were made outside of EMFAC2007. In developing EMFAC2011-HD, staff integrated these adjustments that were not included in EMFAC2007 but were included in the Statewide Truck and Bus Rule emissions inventory. Details are available in Section 11, and the following describes at a high level the revision and update of truck emission factors in EMFAC2011 relative to EMFAC2007.

4.6.1 MEDIUM HEAVY-DUTY TRUCK EMISSION RATES

In EMFAC2007, staff did not update the emission factors for medium heavy-duty trucks (MHDDT). Since the release of EMFAC2007, emission test data for MHDDT were made available by the Coordinating Research Council (CRC) through its E55/59 project final report. As a result, staff revised the MHDDT emission factors using the new test data in EMFAC2011.

The method of calculating MHDDT ZMR and DR is the same as that used for deriving emission factors of heavy heavy-duty trucks (HHDDT) in EMFAC2007. The test data were divided into a number of model year groups based on the general emission trends as well as changes in heavy-duty diesel engine emission standard and emission control technology. For each model year group, the zero-mile emission rates and deterioration rates for HC, CO, NOx, and PM were calculated.

4.6.2 2006-2011 MODEL YEAR NOX EMISSION RATE ADJUSTMENT

A review of the certification data shows that some engine manufacturers introduced 1.2 g/bhp-hr NOx heavy-duty engines one year earlier than required and one manufacturer chooses to meet USEPA 2010 NOx standard without using selective catalytic reduction. Staff updated the 2006-2011 model year HHDDT and MHDDT emission factors in EMFAC2011 to reflect how engine manufacturers complied with the 2007 engine standards and how they comply with the 2010 engine standards between 2007 and 2011.

4.6.3 HHDDT AND MHDDT CO2 EMISSION RATES

In EMFAC2007, CO2 emission rates of diesel trucks were assumed to remain constant regardless of model year, technology, activity, and other factors. However, with the emphasis on greenhouse gas emissions control over the past few years, staff decided to update CO2 emission rates for individual model years in EMFAC2011. During the Board's In-Use On-Road Diesel Vehicle rulemaking, staff evaluated available data to determine how improvements in engine technology and increasingly stringent criteria pollutant emission control requirements have affected the fuel economy of trucks. Staff reviewed multiple data sources to characterize the variations in the fuel economy values of trucks in California. After fuel economy was estimated, fuel usage was then converted to CO2 emissions.

4.6.4 MHDDT IDLE EMISSION RATES

As with the revision of MHDDT running exhaust emission rates, the available CRC E55/59 idle emission test data allowed staff to revise the MHDDT idle emission rates in EMFAC2011. The method of analysis is the same as that used for calculating HHDDT idle emission rates in EMFAC2007. The high idle correction factors for HHDDT in EMFAC2007 are also applicable to MHDDT.

4.6.5 HHDDT NOX AND CO2 IDLE EMISSION RATES

The EMFAC2007 NOx idle emission rates of HHDDT for 2007 and subsequent model years were projected based on the then prevailing assumption that engine manufacturers would adopt a 5-minute shut-off mechanism to comply with ARB's anti-idle rules. However, the certification data show that all engine manufacturers have instead chosen to meet the ARB requirements with the 30 g/hour NOx idle emission rate option. As a result, staff revised the NOx idle emission rates of 2008+ model year trucks in EMFAC2011 to reflect the engine certification data.

The CO2 idle emission rates of HHDDT were also updated in EMFAC2011. In EMFAC2007, an average CO2 idle emission rate was used for all model years. To be consistent with the revision to the CO2 exhaust emission rates of HHDDT, in EMFAC2011 idle emission rates of CO2 were calculated for individual model year groups.

5 EMFAC2011-SG

5.1 BACKGROUND ON CONFORMITY

State and federal law require regional planning agencies prepare both a transportation plan to benefit public mobility and an air quality plan to benefit public health. Under the federal Clean Air Act, transportation activities that receive federal funding or approval must be found to be fully consistent with the plan developed to meet federal clean air standards, known as the State Implementation Plan, or SIP. The requirement that federal activities--especially transportation plans and projects--be shown to help communities attain federal air quality standards is known as conformity.

Conformity applies to federal transportation decisions in all areas that are designated "nonattainment" for specific pollutants (ozone, carbon monoxide, particulate matter) by the U.S. EPA. These are areas that have recorded violations of the National Ambient Air Quality Standards. "Attainment" areas that have adopted air quality maintenance plans are also subject to conformity. Areas that have exceeded the more stringent California air quality standards but are within national standards are not subject to conformity (however, the California Environmental Quality Act applies to all projects in the state).

Adoption by a metropolitan planning organization (MPO) of a 20-year regional transportation plan (RTP), or a short-term federal transportation improvement program (TIP), must include a conformity analysis prepared by the MPO. In addition, sponsors of transportation projects that require a federal approval are responsible for assessing project conformity. Final determinations of conformity for RTPs, TIPs and projects are made by the Federal Highway Administration and the Federal Transit Administration.

Conformity assessments are part of a broader regional transportation planning process carried out by the MPO, or by another transportation agency in less urbanized areas. Because joint transportation and air quality planning assists both conformity assessments and air pollution reduction efforts, local air districts and transportation planning agencies regularly consult with each other and with involved state and federal agencies. Local transportation and air quality planning processes are also open to interested organizations and members of the public.

For RTP and TIP demonstrations, conformity first involves an emissions test. The air quality plan (SIP) forecasts levels of pollutant emissions that will enable steady progress toward attainment of air quality standards by Clean Air Act deadlines, backed up by control strategies that will enable these levels to be reached. Such forecasts are divided by emissions source. The on-road mobile source portion of the forecast is known as a motor vehicle emissions budget. To be found in conformity with the SIP, a

region's transportation plan and program must be found to result in emissions that are within each emissions budget.

In July 2012 staff identified a typographical error in the EMFAC2011-LDV module code that incorrectly assigned trips in gasoline powered school buses, urbant transit buses, other buses, motocycles, and motorhomes in Santa Clara County. These trips were overestimated as a result, which led to an overestimate of ROG emissions in the Bay Area and for the Statewide total. The EMFAC2011-LDV module has been corrected and re-released. New input files to SG were generated for Santa Clara County. The module code and algorithms in the EMFAC2011-SG module were not otherwise affected by this change.

5.2 THE EMFAC2011-SG TOOL

The EMFAC2011 modeling package contains a suite of modules for modeling different vehicle categories including:

- EMFAC2011-LDV, a Fortran-based module mainly representing light-duty fleet (an update to the original EMFAC model), and
- EMFAC2011-HD, a Visual Basic/SQL-based module representing the heavy-duty diesel trucks/vehicles.

Since the two modules are written on two different software platforms, and each contains a different portion of the motor vehicle fleet, outputs from both modules are needed to be processed externally to create the entire motor vehicle fleet inventory. Therefore, the EMFAC WIS processor (in the EMFAC LDV model) is no longer adequate to perform a complete regional transportation conformity analyses.

Staff developed a new module called EMFAC2011-SG. EMFAC2011-SG is developed in Microsoft® Access platform, and replaces the functionalities of the WIS module in EMFAC for transportation conformity purposes. EMFAC2011-SG is an external module that uses the inventory from EMFAC2011-LDV and EMFAC2011-HD modules and scales the emissions based on changes in total VMT, VMT distribution by vehicle class, and speed distribution. EMFAC2011-SG processes data only at a sub-area level. A sub-area is defined as a county that is split by county, air basin, and air district boundaries. Each sub-area within the county is a GAI. In order to process regional scenarios (State, Air Basin, Air District, MPO, County), the module needs to process all sub-Areas in its domain and aggregate the results. Therefore, for a single “Statewide” regional modeling scenario, the module will need to run and aggregate the results of all 69 sub-areas (GAIs). Regional-level outputs are aggregated as part of module output.

Therefore, the module will produce individual outputs for each GAI, but also produce the aggregated region-level output (Statewide output). EMFAC2011-SG uses the VMT-weighted speed distribution at the daily level, and allows users to generate MPO-level outputs, in addition to the Statewide, Air Basin, Air District, County, and Sub-Area level outputs. EMFAC2011-SG inputs and outputs are generated in Microsoft Excel format (*.xls).

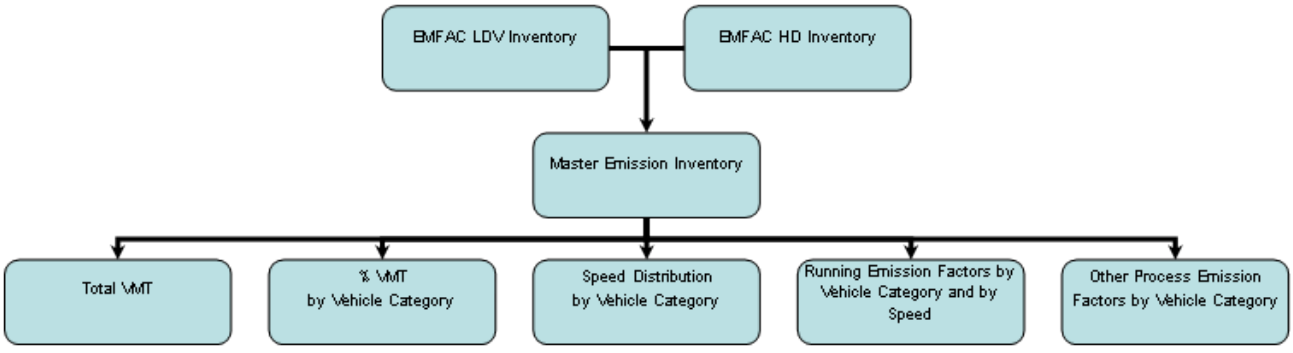
5.3 MODULE LOGIC

Since transportation plans and policies are created by the local governments, the vehicle usage and speed profiles vary greatly at the sub-area level. Therefore, the EMFAC2011-SG module was also developed to process speed data at the sub-area level.

EMFAC2011-SG uses the combined Burden-level outputs from the two modules (EMFAC2011-LDV and EMFAC2011-HD) disaggregated by Model-year and Speed (5-MPH increments) as the base inventory. The base inventories are pre-processed by sub-area, calendar year and season, and are available to download along with the SG package. Users are required to download the inventories for the regions of interest to their computers.

Each sub-area modeling case is called a 'Scenario' (defined by sub-area, calendar year, and season). When a user models a scenario, the EMFAC2011-SG module imports the data from the corresponding base inventory file, and disaggregates the data into different parameters as shown in Figure 5-1.

Figure 5-1. Schematic of EMFAC2011-SG Logic



Based on the modeling parameters, SG uses the following equations to calculate the emissions by Vehicle Category (VC):

$$\text{Running Emissions}_{\text{VC}} = \text{Total VMT} \times \text{VMT Fraction}_{\text{VC}} \times \left[\sum_{\text{Speed}=5}^{70} \text{Speed Fraction}_{\text{Speed,VC}} \times \text{Running Emission Factor}_{\text{Speed,VC}}\right]$$

$$\text{Other Process Emissions}_{\text{VC}} = \text{Total VMT} \times \text{VMT Fraction}_{\text{VC}} \times \text{Process Emission Factors}_{\text{VC}}$$

Where:

$$VMT\ Fraction_{VC} = \frac{VMT_{VC}}{Total\ VMT}$$

$$Speed\ Fraction\ (\%\ VMT_{Speed}) = \frac{VMT_{Speed,\ VC}}{Total\ VMT_{VC}}$$

$$Running\ Emission\ Factor\ (g/mile) = \frac{Default\ Running\ Emissions_{Speed,\ VC}}{Default\ VMT_{Speed,\ VC}}$$

$$Process\ Emission\ Factor\ (g/mile) = \frac{Default\ Process\ Emissions_{VC}}{Default\ Total\ VMT_{VC}}$$

5.4 MODULE OVERVIEW

The EMFAC2011-SG module has three main inputs, and one optional input:

- Base Inputs
- VMT Distribution by Vehicle Category
- Speed Profiles
- Regional Scenario (optional)

The modeling parameters in all three inputs are defined at the sub-area level. In order to correlate the data in the three input sheets, the module identifies each case as a "Scenario". In order to process regional scenarios, users are required to classify all the "scenarios" into groups; the module processes each scenario individually and aggregates data by groups at the end of module execution.

User Input #1: Base Inputs

- This is the most important input for the module. It contains scenario definitions that include sub-area, calendar year and season, and selections for VMT profile (module defaults or user defined), VMT by Vehicle Category (module defaults or user defined), and Speed distribution type (module defaults or user defined) for each scenario.
    - If the "VMT profile" entry for the scenario is *User* defined, then the user also needs to enter the "New Total VMT" data (miles/day)
- *Input Variables:*
    a. Group (Integer) – Identifier for grouping/aggregating the scenarios
    b. Area (Text) – Denotes the Area/Region name for each group
    c. Scenario (Integer) – Index representing order of modeling scenario
    d. Sub-Area (Text)
        - Options: Selected from a list of 69 sub-areas (CoABDis or GAI)
    e. CalYr (Calendar year)
        - Options: Any calendar year between 1990 and 2035
    f. Season (Text)
        - Options: "Annual", "Summer", or "Winter"
    g. Title (Text)
    h. VMT Profile (Text)
        - Options: "Default" (module default) or "User"(user-defined)
    i. VMT by Vehicle Category (Text)
        - Options: "Default" (module default) or "User"(user-defined)
    j. Speed Profile (Text)
        - Options: "Default" (module default) or "User"(user-defined)

k. New Total VMT (Numeric) – Represents user-defined total daily VMT (miles/day)
    - If the scenario VMT Profile is designated as “Default”, then the “New Total VMT” cell can be empty (user entry for “New Total VMT” will be ignored)
    - If the scenario ‘VMT Profile’ and the ‘VMT by Vehicle Category’ are designated as “User” (user-defined), then the “New Total VMT” cell can be empty (the VMT from the “VMT by Vehicle Category” table will be used in the calculations; user entry for “New Total VMT” will be ignored)
    - If the scenario VMT Profile is designated as “User” (user-defined), and the ‘VMT by Vehicle Category’ is designated as “Default” (module default), only then the “New Total VMT” cell needs to be populated (here, the Default VMT fractions by Vehicle Class are used to apportion the User provided “New Total VMT”)

User Input #2: VMT by Vehicle Category

- Contains daily VMT data by Vehicle Category, and is a required input if the “VMT by Vehicle Category” entry for the scenario is *User* defined,
    - Data entered in the “VMT_by_VehCat” worksheet (in miles/day)
- *Input Variables:*
    a. Group
    b. Area
    c. Scenario
    d. Sub-Area
    e. CalYr
    f. Season
    g. Title
    h. Veh & Tech: EMFAC2011 Vehicle Category
    i. New VMT: Daily VMT by Vehicle Category

User Input #3: Speed Profile

- Contains the speed distribution by vehicle category (represented by % VMT in each speed bin for an average day), and is a required input if the “Speed Profile” entry for the scenario is *User* defined
    - Data entered in the “Speed_Profile” worksheet
- *Input Variables:*
    a. Group
    b. Area
    c. Scenario

d. Sub-Area
e. CalYr
f. Season
g. Title
h. Veh & Tech: EMFAC2011 Vehicle Category
i. EMFAC2007 Veh & Tech: EMFAC2007 Vehicle Category
j-w. 5MPH-70MPH: Speed Distribution (% VMT at each speed bin) for Speeds 5 - 70 MPH @ 5 MPH increments

Optional User Input: Regional Scenarios

- This is an optional input for modeling
  - Allows users to create Regional (multi-GAI) scenarios outside the module, and import them into the module
- *Input Variables:*
  a. Group
  b. Group Type
  c. Area
  d. Calendar Year
  e. Season
- *Regional Scenario Groups*
  - All scenarios that need to be aggregated together should be classified in the same "Group" in the Scenario_Base_Inputs table
    - So if the user needs to run "Statewide 2008 Annual" and "Statewide 2010 Annual", then user should create two "Groups"
      - All scenarios related to Statewide Sub-Areas that relate to 2008 Annual period in one group
      - All scenarios related to Statewide Sub-Areas that relate to 2010 Annual period in another group
    - All input tables (Scenario_Base_Inputs, Scenario_VMT_by_VehCat, and Scenario_Speed_Profiles) need to identify the Group, Area name, Scenario, Sub-Area, CalYr, Season, and Title for each Scenario
  - If users only need Sub-Area (GAI) runs, then
    - All scenarios should be grouped in the 'Default' Group # (1)
    - Area name category should be "-" [Area name "-" denotes that it is not part of any "regional" run, and these results will not be aggregated by Group (results will still be generated))
  - For any scenarios where Area name <> "-", outputs will be aggregated based on Group ID
  - Module allows Regional Scenario table to be imported using the "Load/Run Regional Scenarios (External Files)" option

5.5 ASSESSING CO2 BENEFITS FOR THE PAVLEY RULE AND LOW CARBON FUEL STANDARD

EMFAC2011-SG outputs carbon dioxide (CO2) emissions both with and without the Pavley Rule and Low Carbon Fuel Standard. The method for calculating benefits is consistent with the Pavley-I and LCFS post-processor used for analysis for SB375, and estimates benefits only for CO2, not criteria pollutants. The method requires applying correction factors to by model year emissions inventory output. Because EMFAC2011-SG runs on emissions inventories summed across model years, the correction was applied to CO2 emissions estimates when developing the input inventory files for SG. The correction is not embedded in the EMFAC2011-SG module code; it is embedded in the emissions data that are input to EMFAC2011-SG.

The calculation used to adjust CO2 emissions is very simple, and derived from assessments developed during staff regulatory support efforts for the two programs.

- Pavley-I: A clean-car standard to reduce greenhouse gas (GHG) emissions from new passenger vehicles (LDA-MDV) from 2009 through 2016 (reduction factors are presented in Table 5-1).
- Low Carbon Fuel Standard: A fuel standard that requires a reduction of at least 10 percent in the carbon intensity of California's transportation fuels by 2020 (reduction factors are presented in Table 5-2).

Table 5-1. Pavely I Reduction Factors

Model Year	LDA/LDT1	LDT2/MDV
2008 and older	0.00%	0.00%
2009	0.00%	0.90%
2010	3.50%	5.20%
2011	14.40%	12.00%
2012	25.30%	18.50%
2013	27.20%	19.90%
2014	28.80%	21.00%
2015	31.70%	23.00%
2016 +	34.30%	25.10%

Table 5-2. LCFS Reduction Factors

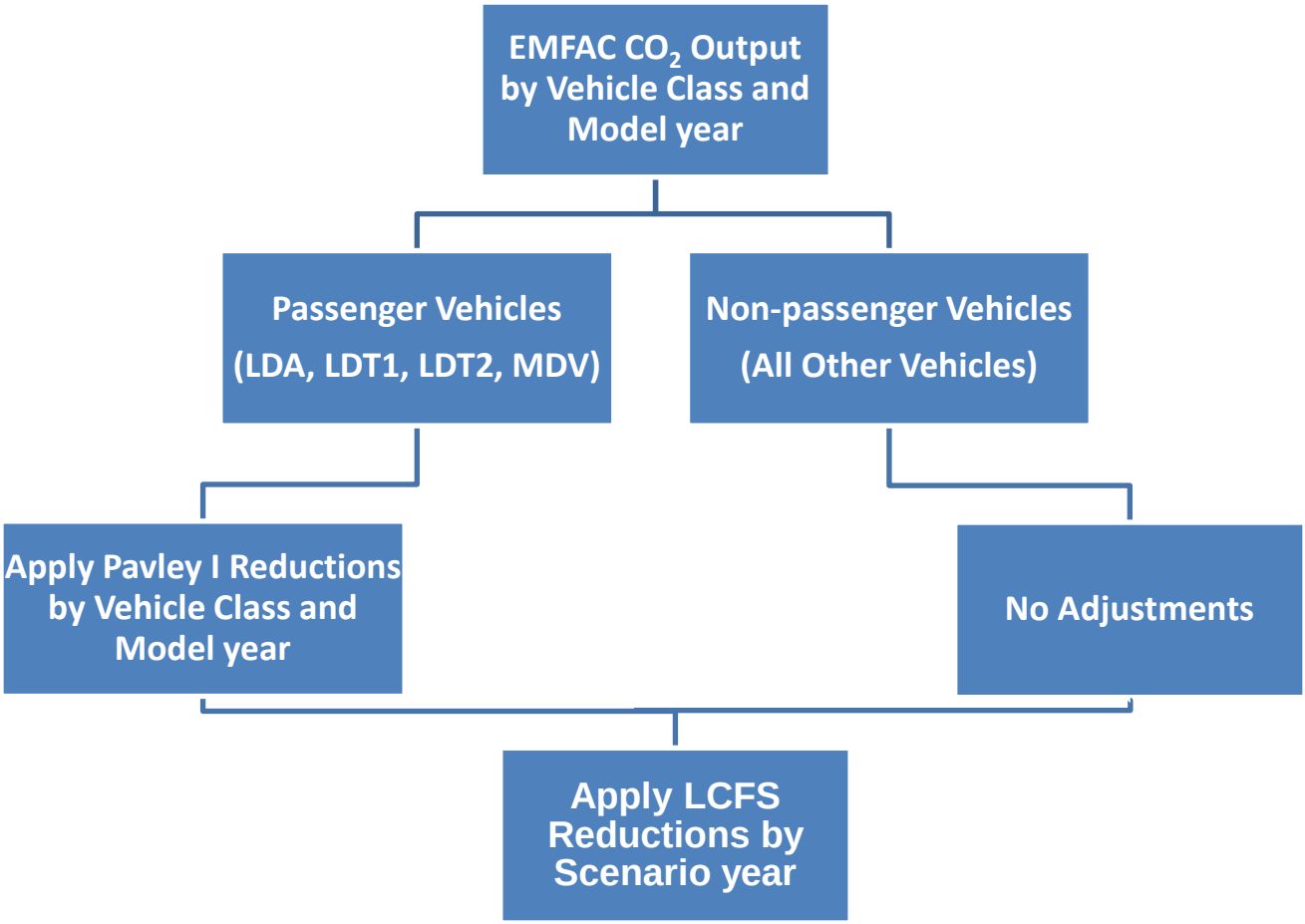
Calendar Year	Reduction Factor
2010	Reporting Only
2011	0.25%
2012	0.50%
2013	1.00%
2014	1.50%
2015	2.50%
2016	3.50%
2017	5.00%
2018	6.50%
2019	8.00%
2020 +	10.00%

LCFS reduction factors are based on the carbon intensity reductions required by the LCFS Regulation for transportation fuels used in California as provided from the Staff Report titled "California's Low Carbon Fuel Standard" dated October 2009.

Measured on a lifecycle basis, the carbon intensity represents the equivalent amount of carbon dioxide (CO_2e) emitted from each stage of producing, transporting, and using the fuel in a motor vehicle.

In order to calculate the CO2 emissions reductions from the two regulations, the following five-step process was used:

1. Extract CO_2 emission data from EMFAC by Vehicle Class and Model Year
2. Disaggregate the data into two groups:
    a. Passenger vehicles (LDA, LDT1, LDT2, MDV)
    b. Non-passenger vehicles (All other vehicles)
3. Apply the Pavley I reduction factors to the Passenger vehicles CO_2 emissions by vehicle class and model year
4. Aggregate the total CO_2 emissions for each vehicle class
5. Apply the Low Carbon Fuel Standard reduction factors to all vehicles based on the calendar year


EMFAC CO_2 Output by Vehicle Class and Model year
Passenger Vehicles (LDA, LDT1, LDT2, MDV)
Non-passenger Vehicles (All Other Vehicles)
Apply Pavley I Reductions by Vehicle Class and Model year
No Adjustments
Apply LCFS Reductions by Scenario year

5.6 MODULE OUTPUTS

The model outputs are denoted by “EMFAC2011-SG Output” and contain the Input Filename (e.g. if the Input Filename is “Sample”, the Output file will be called “EMFAC2011-SG Output - Sample”). The output files are created in Microsoft® Excel format (.xls), and contain the following variables:

- Group
- Area
- Scenario
- Sub-Area
- Calendar Year
- Season
- Title
- Veh & Tech
- EMFAC2007 Category
- Vehicle Population
- VMT (miles/average weekday)
- Trips (trips/average weekday)
- TOG Emissions (tons/average weekday)
- ROG Emissions (tons/average weekday)
- CO Emissions (tons/average weekday)
- NO_x Emissions (tons/average weekday)
- CO2 Emissions (tons/average weekday)
- CO2 Emissions (including Pavley I and LCFS adjustments) (tons/average weekday)
- PM_{10} Emissions (tons/average weekday)
- $PM_{2.5}$ Emissions (tons/average weekday)
- SO_x Emissions (tons/average weekday)
- Fuel Consumption – Gas and Diesel (1,000 gallons/average weekday; not adjusted for Pavley-I or LCFS)

The following process emissions are generated for each pollutant:

- All Pollutants - Running, Idling, Starting, and Total Exhaust
- ROG and TOG - Diurnal, Hot-Soak; Running and Resting Losses; Total
- PM10 and PM2.5 - Break wear, Tire wear, Total

The model appends the outputs for all scenarios defined in an input file in the same output file. The model also aggregates the results of all the scenarios that are classified in the same group, and appends the aggregated output for the group in the same file (along with the outputs for all sub-areas).

- For a single statewide regional case, the model will create a single Excel output table containing 70 sets of outputs (69 sub-area/GAI results, and 1 Statewide-totals result). The number of data rows will depend on number of Vehicle Categories applicable to each sub-area.

Table 5-3 provides sub-area classifications that can be run in EMFAC2011-SG. Table 5-4 provides vehicle categories classified by several vehicle category classification schemes that can be summarized using EMFAC2011.

Table 5-3. Sub-Area Classifications

Sub-Area	County	Air Basin	Air District	MPO
Alameda (SF)	Alameda	San Francisco Bay Area	Bay Area AQMD	MTC
Alpine (GBV)	Alpine	Great Basin Valley	Great Basin Unified APCD	
Amador (MC)	Amador	Mountain Counties	Amador County APCD	
Butte (SV)	Butte	Sacramento Valley	Butte County AQMD	BCAG
Calaveras (MC)	Calaveras	Mountain Counties	Calaveras County APCD	
Colusa (SV)	Colusa	Sacramento Valley	Colusa County APCD	
Contra Costa (SF)	Contra Costa	San Francisco Bay Area	Bay Area AQMD	MTC
Del Norte (NC)	Del Norte	North Coast	North Coast Unified AQMD	
El Dorado (LT)	El Dorado	Lake Tahoe	El Dorado County APCD	TMPO
El Dorado (MC)	El Dorado	Mountain Counties	El Dorado County APCD	SACOG
Fresno (SJV)	Fresno	San Joaquin Valley	San Joaquin Valley Unified APCD	COFCG
Glenn (SV)	Glenn	Sacramento Valley	Glenn County APCD	
Humboldt (NC)	Humboldt	North Coast	North Coast Unified AQMD	
Imperial (SS)	Imperial	Salton Sea	Imperial County APCD	SCAG
Inyo (GBV)	Inyo	Great Basin Valley	Great Basin Unified APCD	
Kern (MD)	Kern	Mojave Desert	Kern County APCD	KCOG
Kern (SJV)	Kern	San Joaquin Valley	San Joaquin Valley Unified APCD	KCOG
Kings (SJV)	Kings	San Joaquin Valley	San Joaquin Valley Unified APCD	KCAG
Lake (LC)	Lake	Lake County	Lake County APCD	
Lassen (NEP)	Lassen	Northeast Plateau	Lassen County APCD	
Los Angeles (MD)	Los Angeles	Mojave Desert	Antelope Valley APCD	SCAG
Los Angeles (SC)	Los Angeles	South Coast	South Coast AQMD	SCAG
Madera (SJV)	Madera	San Joaquin Valley	San Joaquin Valley Unified APCD	MCTC
Marin (SF)	Marin	San Francisco Bay Area	Bay Area AQMD	MTC
Mariposa (MC)	Mariposa	Mountain Counties	Mariposa County APCD	
Mendocino (NC)	Mendocino	North Coast	Mendocino County APCD	
Merced (SJV)	Merced	San Joaquin Valley	San Joaquin Valley Unified APCD	MCAG

Sub-Area	County	Air Basin	Air District	MPO
Modoc (NEP)	Modoc	Northeast Plateau	Modoc County APCD	
Mono (GBV)	Mono	Great Basin Valley	Great Basin Unified APCD	
Monterey (NCC)	Monterey	North Central Coast	Monterey Bay Unified APCD	AMBAG
Napa (SF)	Napa	San Francisco Bay Area	Bay Area AQMD	MTC
Nevada (MC)	Nevada	Mountain Counties	Northern Sierra AQMD	
Orange (SC)	Orange	South Coast	South Coast AQMD	SCAG
Placer (LT)	Placer	Lake Tahoe	Placer County APCD	TMPO
Placer (MC)	Placer	Mountain Counties	Placer County APCD	SACOG
Placer (SV)	Placer	Sacramento Valley	Placer County APCD	SACOG
Plumas (MC)	Plumas	Mountain Counties	Northern Sierra AQMD	
Riverside (MD/MDAQMD)	Riverside	Mojave Desert	Mojave Desert AQMD	SCAG
Riverside (MD/SCAQMD)	Riverside	Mojave Desert	South Coast AQMD	SCAG
Riverside (SC)	Riverside	South Coast	South Coast AQMD	SCAG
Riverside (SS)	Riverside	Salton Sea	South Coast AQMD	SCAG
Sacramento (SV)	Sacramento	Sacramento Valley	Sacramento Metropolitan AQMD	SACOG
San Benito (NCC)	San Benito	North Central Coast	Monterey Bay Unified APCD	AMBAG
San Bernardino (MD)	San Bernardino	Mojave Desert	Mojave Desert AQMD	SCAG
San Bernardino (SC)	San Bernardino	South Coast	South Coast AQMD	SCAG
San Diego (SD)	San Diego	San Diego	San Diego County APCD	SANDAG
San Francisco (SF)	San Francisco	San Francisco Bay Area	Bay Area AQMD	MTC
San Joaquin (SJV)	San Joaquin	San Joaquin Valley	San Joaquin Valley Unified APCD	SJCOG
San Luis Obispo (SCC)	San Luis Obispo	South Central Coast	San Luis Obispo County APCD	SLOCOG
San Mateo (SF)	San Mateo	San Francisco Bay Area	Bay Area AQMD	MTC
Santa Barbara (SCC)	Santa Barbara	South Central Coast	Santa Barbara County APCD	SBCAG
Santa Clara (SF)	Santa Clara	San Francisco Bay Area	Bay Area AQMD	MTC
Santa Cruz (NCC)	Santa Cruz	North Central Coast	Monterey Bay Unified APCD	AMBAG
Shasta (SV)	Shasta	Sacramento Valley	Shasta County AQMD	SCRTPA
Sierra (MC)	Sierra	Mountain Counties	Northern Sierra AQMD	

Sub-Area	County	Air Basin	Air District	MPO
Siskiyou (NEP)	Siskiyou	Northeast Plateau	Siskiyou County APCD	
Solano (SF)	Solano	San Francisco Bay Area	Bay Area AQMD	MTC
Solano (SV)	Solano	Sacramento Valley	Yolo/Solano AQMD	MTC
Sonoma (NC)	Sonoma	North Coast	Northern Sonoma County APCD	MTC
Sonoma (SF)	Sonoma	San Francisco Bay Area	Bay Area AQMD	MTC
Stanislaus (SJV)	Stanislaus	San Joaquin Valley	San Joaquin Valley Unified APCD	StanCOG
Sutter (SV)	Sutter	Sacramento Valley	Feather River AQMD	SACOG
Tehama (SV)	Tehama	Sacramento Valley	Tehama County APCD	
Trinity (NC)	Trinity	North Coast	North Coast Unified AQMD	
Tulare (SJV)	Tulare	San Joaquin Valley	San Joaquin Valley Unified APCD	TCAG
Tuolumne (MC)	Tuolumne	Mountain Counties	Tuolumne County APCD	
Ventura (SCC)	Ventura	South Central Coast	Ventura County APCD	SCAG
Yolo (SV)	Yolo	Sacramento Valley	Yolo/Solano AQMD	SACOG
Yuba (SV)	Yuba	Sacramento Valley	Feather River AQMD	SACOG

Table 5-4. EMFAC2011 Vehicle Category Classifications

Index	EMFAC2011 Veh & Tech	EMFAC2011 Vehicle	Source	EMFAC2007 Vehicle	EMFAC2007 Veh & Tech	CTEMFAC Vehicle
1	LDA - DSL	LDA	EMFAC2011-LDV	LDA	LDA - DSL	Non-Trucks
2	LDA - GAS	LDA	EMFAC2011-LDV	LDA	LDA - GAS	Non-Trucks
3	LDT1 - DSL	LDT1	EMFAC2011-LDV	LDT1	LDT1 - DSL	Non-Trucks
4	LDT1 - GAS	LDT1	EMFAC2011-LDV	LDT1	LDT1 - GAS	Non-Trucks
5	LDT2 - DSL	LDT2	EMFAC2011-LDV	LDT2	LDT2 - DSL	Non-Trucks
6	LDT2 - GAS	LDT2	EMFAC2011-LDV	LDT2	LDT2 - GAS	Non-Trucks
7	LHD1 - DSL	LHD1	EMFAC2011-LDV	LHDT1	LHDT1 - DSL	Trucks
8	LHD1 - GAS	LHD1	EMFAC2011-LDV	LHDT1	LHDT1 - GAS	Trucks
9	LHD2 - DSL	LHD2	EMFAC2011-LDV	LHDT2	LHDT2 - DSL	Trucks
10	LHD2 - GAS	LHD2	EMFAC2011-LDV	LHDT2	LHDT2 - GAS	Trucks
11	MCY - GAS	MCY	EMFAC2011-LDV	MCY	MCY - GAS	Non-Trucks
12	MDV - DSL	MDV	EMFAC2011-LDV	MDV	MDV - DSL	Trucks
13	MDV - GAS	MDV	EMFAC2011-LDV	MDV	MDV - GAS	Trucks
14	MH - DSL	MH	EMFAC2011-LDV	MH	MH - DSL	Non-Trucks
15	MH - GAS	MH	EMFAC2011-LDV	MH	MH - GAS	Non-Trucks
16	T6 Ag - DSL	T6 Ag	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
17	T6 CAIRP heavy - DSL	T6 CAIRP heavy	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks

Index	EMFAC2011 Veh & Tech	EMFAC2011 Vehicle	Source	EMFAC2007 Vehicle	EMFAC2007 Veh & Tech	CTEMFAC Vehicle
18	T6 CAIRP small - DSL	T6 CAIRP small	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
19	T6 instate construction heavy - DSL	T6 instate construction heavy	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
20	T6 instate construction small - DSL	T6 instate construction small	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
21	T6 instate heavy - DSL	T6 instate heavy	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
22	T6 instate small - DSL	T6 instate small	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
23	T6 OOS heavy - DSL	T6 OOS heavy	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
24	T6 OOS small - DSL	T6 OOS small	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
25	T6 Public - DSL	T6 Public	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
26	T6 utility - DSL	T6 utility	EMFAC2011-HDV	MHDT	MHDT - DSL	Trucks
27	T6TS - GAS	T6TS	EMFAC2011-LDV	MHDT	MHDT - GAS	Trucks
28	T7 Ag - DSL	T7 Ag	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
29	T7 CAIRP - DSL	T7 CAIRP	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
30	T7 CAIRP construction - DSL	T7 CAIRP construction	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
31	T7 NNOOS - DSL	T7 NNOOS	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
32	T7 NOOS - DSL	T7 NOOS	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
33	T7 other port - DSL	T7 other port	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
34	T7 POAK - DSL	T7 POAK	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks

Index	EMFAC2011 Veh & Tech	EMFAC2011 Vehicle	Source	EMFAC2007 Vehicle	EMFAC2007 Veh & Tech	CTEMFAC Vehicle
35	T7 POLA - DSL	T7 POLA	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
36	T7 Public - DSL	T7 Public	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
37	T7 Single - DSL	T7 Single	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
38	T7 single construction - DSL	T7 single construction	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
39	T7 SWCV - DSL	T7 SWCV	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
40	T7 tractor - DSL	T7 tractor	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
41	T7 tractor construction - DSL	T7 tractor construction	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
42	T7 utility - DSL	T7 utility	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
43	T7IS - GAS	T7IS	EMFAC2011-LDV	HHDT	HHDT - GAS	Trucks
44	PTO - DSL	PTO	EMFAC2011-HDV	HHDT	HHDT - DSL	Trucks
45	SBUS - DSL	SBUS	EMFAC2011-HDV	SBUS	SBUS - DSL	Non-Trucks
46	SBUS - GAS	SBUS	EMFAC2011-LDV	SBUS	SBUS - GAS	Non-Trucks
47	UBUS - DSL	UBUS	EMFAC2011-LDV	UBUS	UBUS - DSL	Non-Trucks
48	UBUS - GAS	UBUS	EMFAC2011-LDV	UBUS	UBUS - GAS	Non-Trucks
49	Motor Coach - DSL	Motor Coach	EMFAC2011-HDV	OBUS	OBUS - DSL	Non-Trucks
50	OBUS - GAS	OBUS	EMFAC2011-LDV	OBUS	OBUS - GAS	Non-Trucks
51	All Other Buses - DSL	All Other Buses	EMFAC2011-HDV	OBUS	OBUS - DSL	Non-Trucks

6 OVERVIEW OF INCREMENTAL CHANGES

EMFAC2011 emissions estimates are the sum of emissions estimated using the EMFAC2011-LDV and EMFAC2011-HD modules. While there have been many improvements to the EMFAC modeling system for EMFAC2011, overall activity and emissions are generally similar to those estimated using EMFAC2007.

6.1 POPULATION

To understand the differences in estimated population between EMFAC2007 and EMFAC2011, it is useful to recall differences between the population data in the two models. EMFAC2011 was updated with 2009 DMV data and 2010 commercial diesel truck population estimates. Both of these population data sources are impacted by the 2007-2009 economic recession, which substantially reduced new vehicle sales, and to a limited degree, vehicle populations. In contrast, EMFAC2007 contained 2005 population data; 2009 population estimates were forecasted using VMT forecasts provided by regional transportation agencies prior to the EMFAC2007 release.

EMFAC2007 contained 2000-2005 DMV data and backcast populations prior to 2000. The DMV data input to EMFAC contained anomalies in the 2004 and 2005 data that led the 2004 population to be underestimated and the 2005 population to be overestimated, primarily for heavy-duty trucks. The inconsistency in population and activity data in EMFAC2007 led to an increase in estimated population in 2005, coupled with an estimated decrease in population between 2005 and 2006 in most areas, primarily but not exclusively for heavy-duty trucks.

EMFAC2011 contains 2009 DMV data in EMFAC2011-LDV, and 2010 estimated truck populations developed from multiple data sources in EMFAC2011-HD. The process of using of multiple data sources to model populations in the HD module started with the Statewide Truck and Bus Rule inventory, and was developed specifically to correct for anomalies in truck data provided by DMV in their registration database. 1990-2008 calendar year populations are backcast using historical regional VMT trends in EMFAC2011. The anomaly present in EMFAC2007 has been removed for EMFAC2011.

Population comparisons are shown in Figure 6-1, Figure 6-2, and Figure 6-3 for Statewide, South Coast, and San Joaquin Valley respectively.

Figure 6-1. Statewide Population: EMFAC2011 vs EMFAC2007

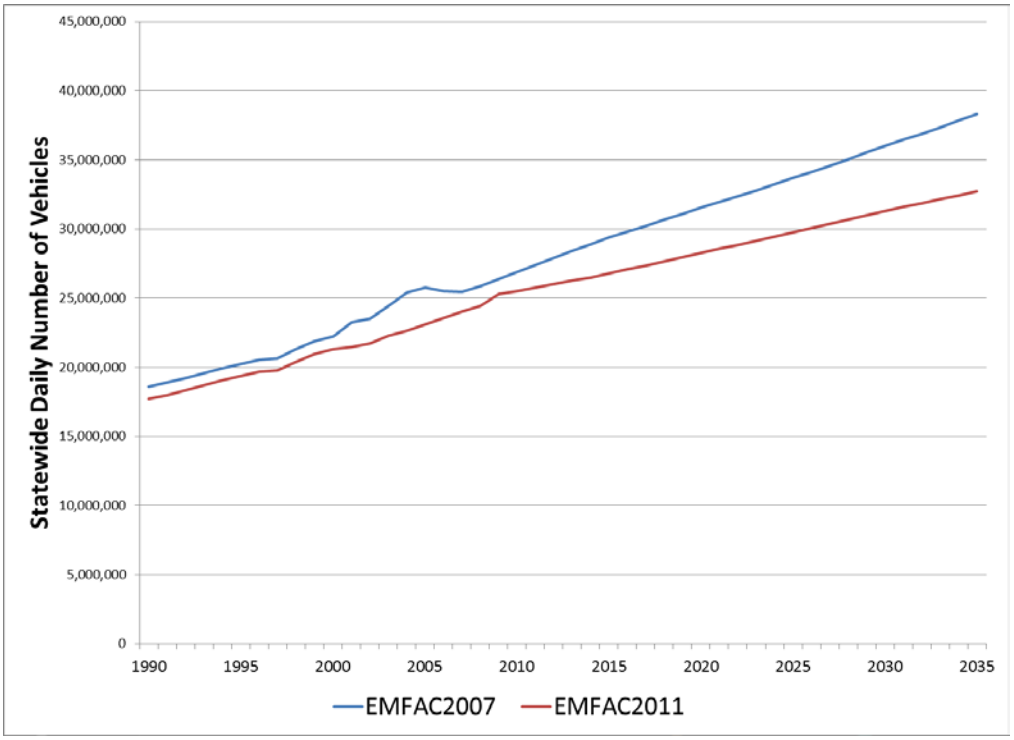


Figure 6-2. South Coast Population: EMFAC2011 vs EMFAC2007

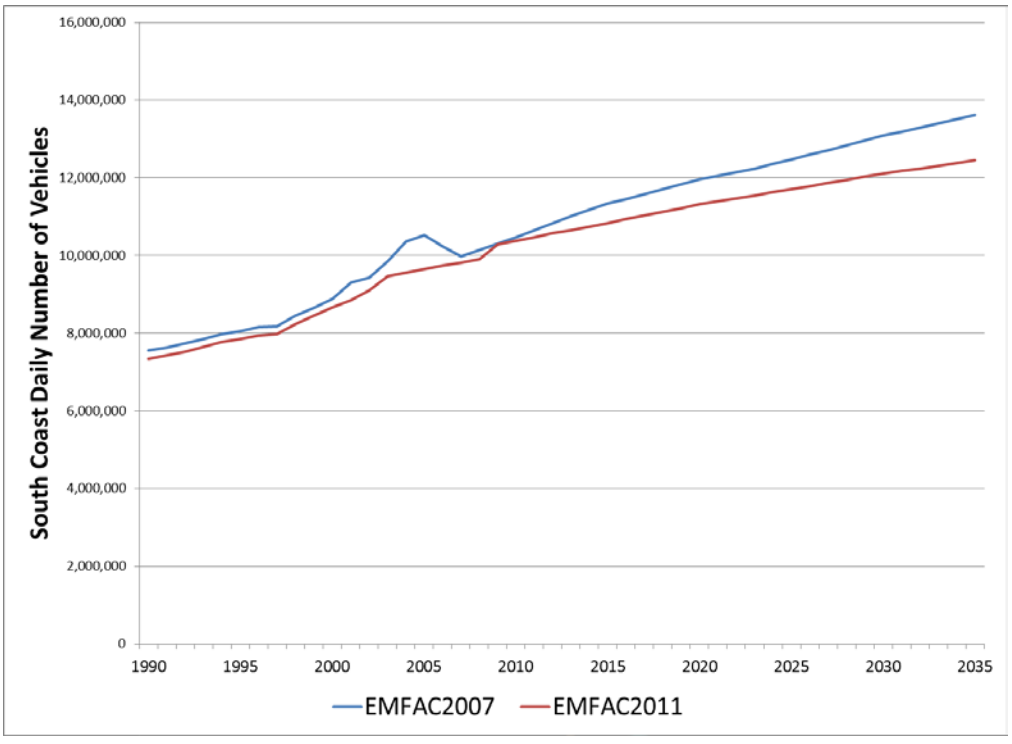
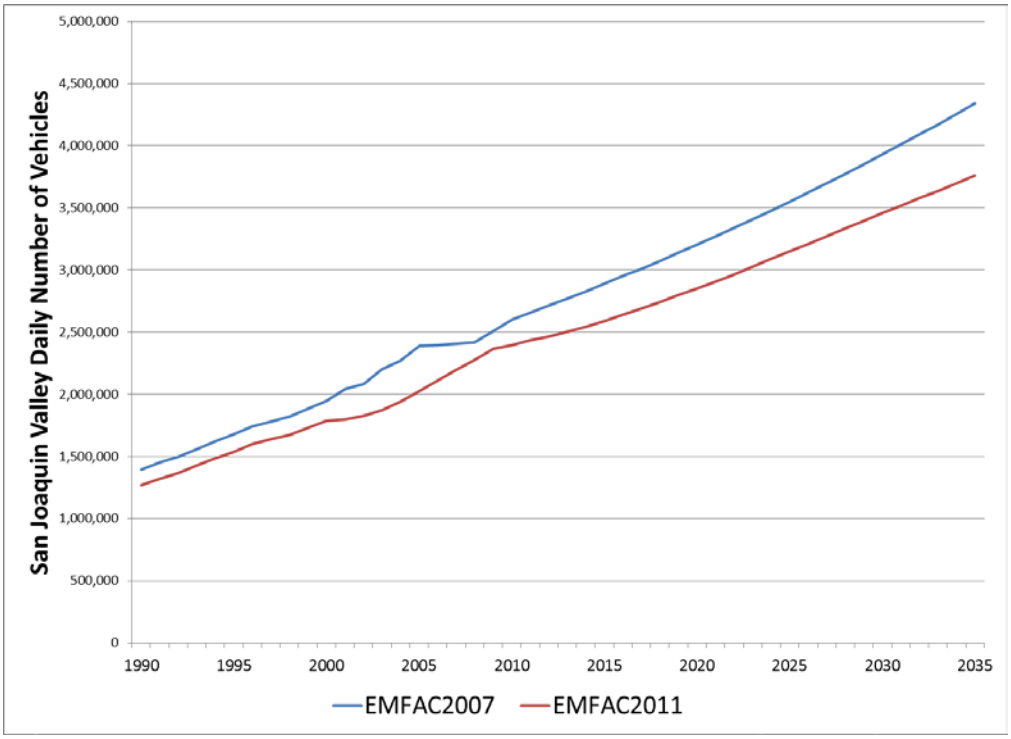


Figure 6-3 San Joaquin Valley Population: EMFAC2011 vs EMFAC2007



6.2 ACTIVITY

EMFAC2011 contains VMT estimates that have been updated by regional transportation planning agencies relative to what was submitted for EMFAC2007. In evaluating the data, long term forecasted VMT is lower in the new data than the submittals for EMFAC2007, which is reflective of the impact of the economic recession. However the full immediate impact of the recession is not generally reflected in the submitted activity data, and not all agencies provided updated data. As a result, on a statewide basis, VMT estimates from 1990-2008 are very similar between EMFAC2011 and EMFAC2007, with the exception of the population anomaly in EMFAC2007 discussed above. This is shown in Figure 6-4. Both South Coast and San Joaquin Valley VMT estimates are slightly lower in 2009, which results in a slightly lower VMT backcast between 1990 and 2008. The EMFAC2007 anomaly in VMT in both South Coast and the San Joaquin Valley has been corrected in EMFAC2011. Forecast VMT is slightly lower due to the recession. This is shown in Figure 6-5 and Figure 6-6.

Figure 6-4. Statewide VMT Estimates: EMFAC2011 vs. EMFAC2007

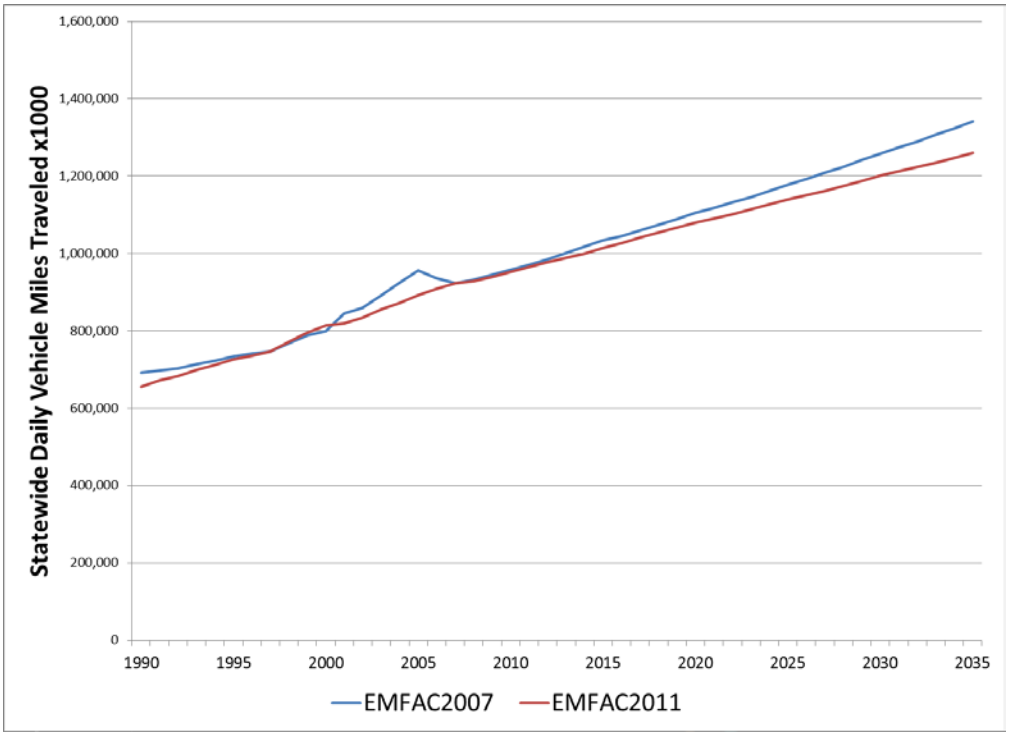


Figure 6-5. South Coast VMT Estimates: EMFAC2011 vs. EMFAC2007

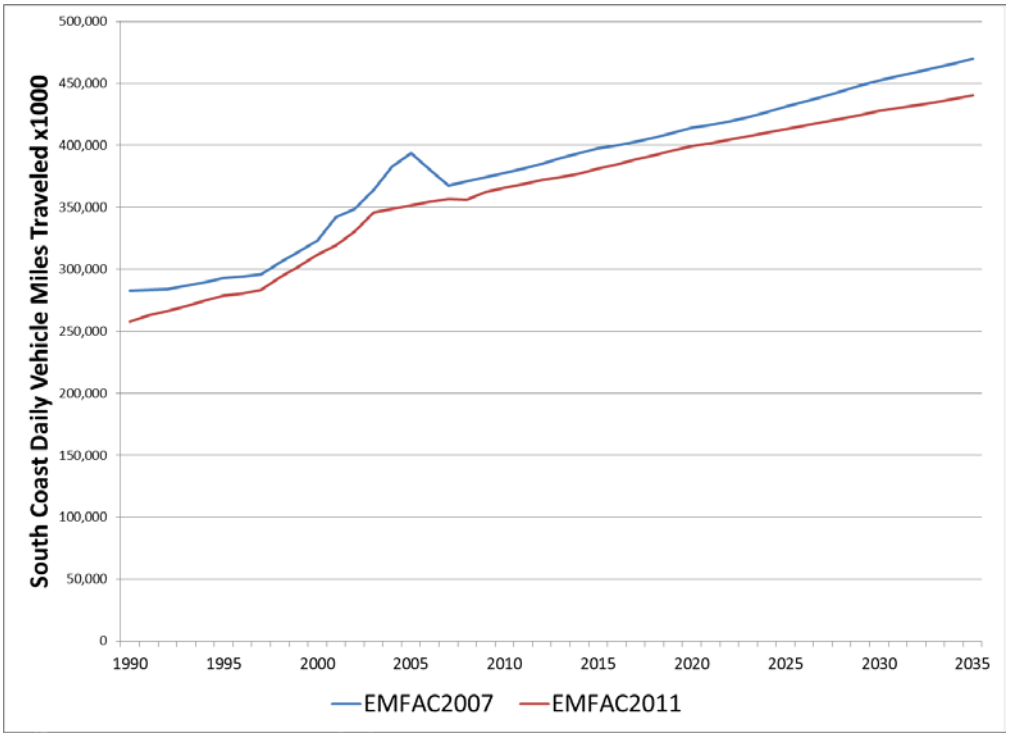
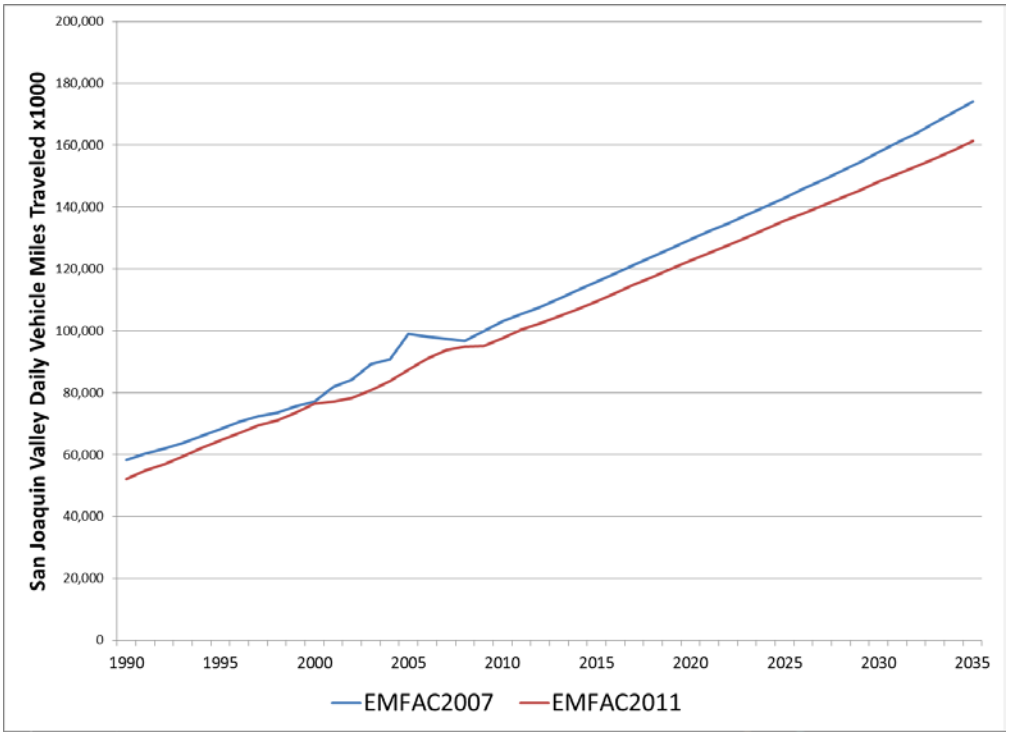


Figure 6-6. San Joaquin Valley VMT Estimates: EMFAC2011 vs. EMFAC2007



6.3 CARBON DIOXIDE EMISSIONS

Carbon dioxide emissions estimates are reduced by reductions in VMT that occur regionally as a result of updated VMT estimates, and as a result of minor improvements to CO2 emission rates described in Sections 3.3.3.1, 3.3.3.2 and 4.6.3. Estimated CO2 emissions are shown in Figure 6-7, Figure 6-8, and Figure 6-9 for Statewide, South Coast, and the San Joaquin Valley respectively.

All three charts show the significant impact that the Pavley-I Clean Car Standards and the Low Carbon Fuel Standard are projected to have in reducing fossil carbon dioxide emissions in the future. All three charts also show that the population anomaly in EMFAC2007 led to an anomaly in CO2 emissions which has been corrected in EMFAC2011.

In the San Joaquin Valley, the estimated heavy-duty truck VMT fraction relative to VMT from all vehicles decreased between EMFAC2007 and EMFAC2011. As a result, the projected light duty vehicle VMT increased between EMFAC2007 and EMFAC2011. Because light duty vehicles are more fuel efficient than heavy-duty vehicles, this led to an incremental decrease in projected CO2 emissions in the San Joaquin Valley per mile driven.

Figure 6-7. Statewide CO2 Emissions: EMFAC2011 vs. EMFAC2007

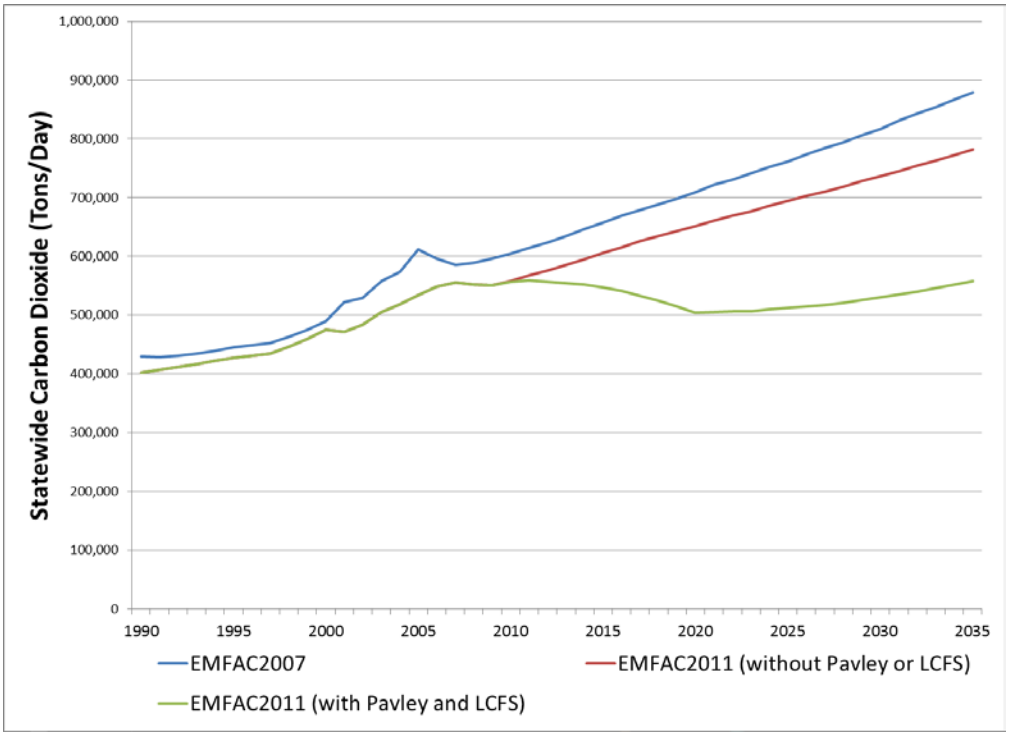


Figure 6-8. South Coast CO2 Emissions: EMFAC2011 vs EMFAC2007

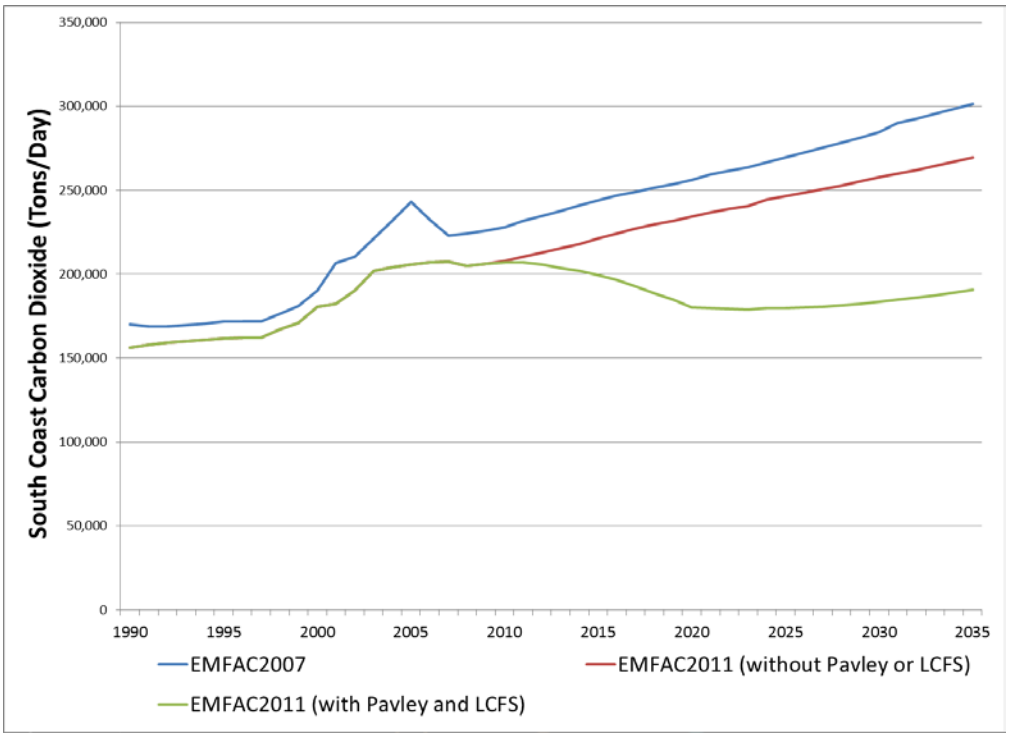
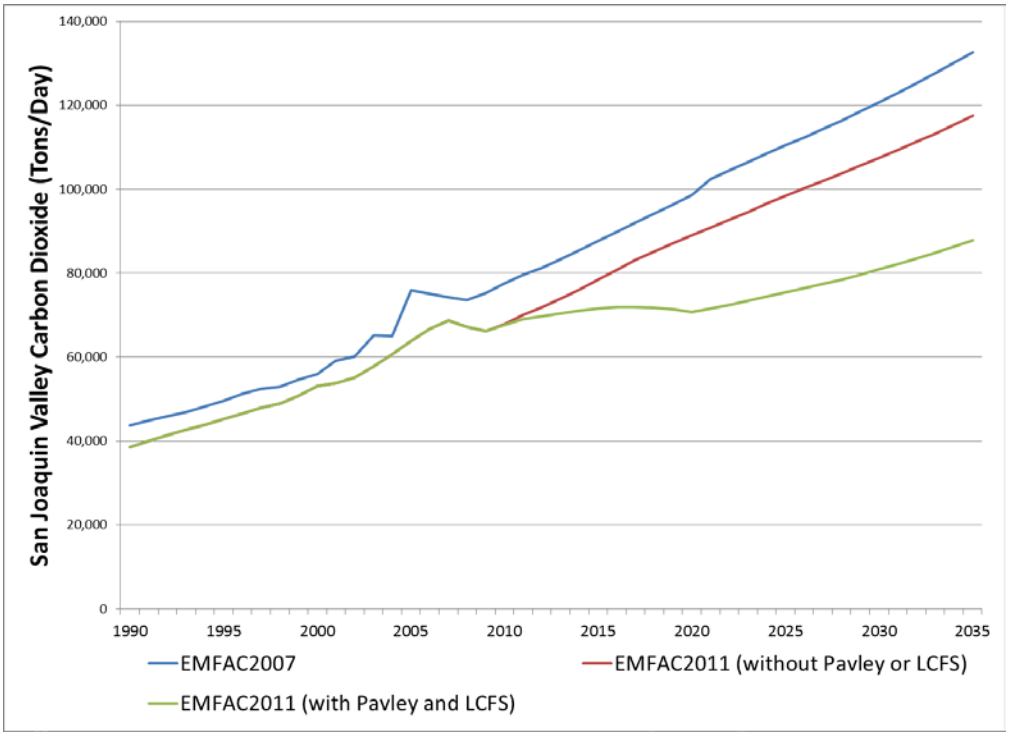


Figure 6-9. San Joaquin Valley CO2 Emissions: EMFAC2011 vs. EMFAC2007



6.4 NOX AND ROG EMISSIONS

NOx and ROG emissions show the continuing downward trend caused by decades of ARB rules designed to reduce emissions from cars and trucks. Statewide results are shown in Figure 6-10 and Figure 6-11 below. Several trends are apparent. The NOx anomaly caused by heavy-duty truck population estimates in EMFAC2007 is corrected for EMFAC2011. NOx emissions are driven by trucks and are lower due in part to the impact of the recession on heavy-duty truck emissions, the impact of ARB rules designed to reduce NOx emissions from diesel trucks and buses, and the impact of reduced out-year forecasts for truck VMT. ROG emissions are driven by gasoline vehicles, and are marginally lower due to improvements in emission rates, updates to vehicle survival algorithms that have the impact of slightly increasing vehicle turnover, and updated temperature and relative humidity estimates. These changes are discussed in Sections 3.3.3.3, 3.3.3.4, 3.3.4.2, and 3.3.4.4.

Figure 6-10. Statewide NOx Emissions: EMFAC2011 vs. EMFAC2007

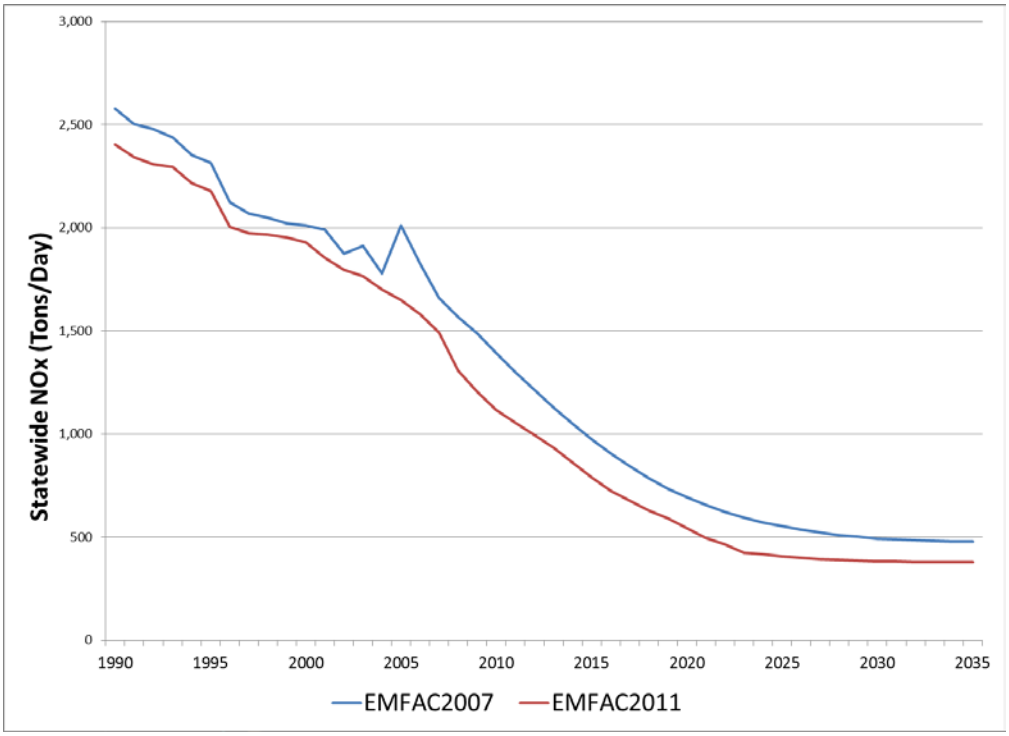
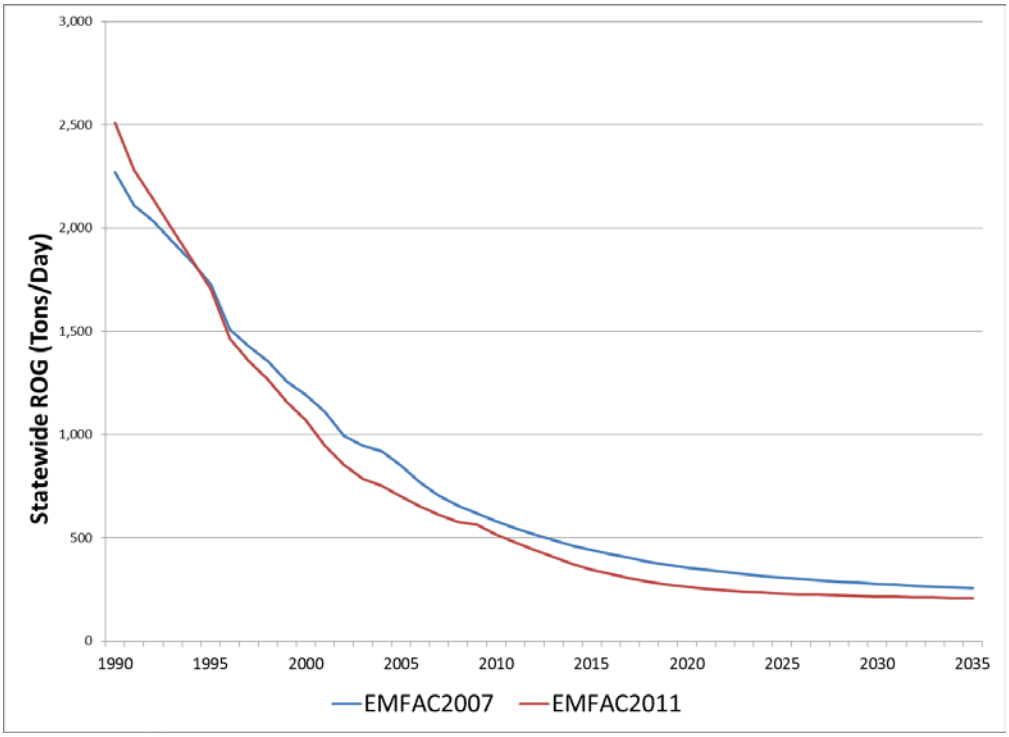


Figure 6-11. Statewide ROG Emissions: EMFAC2011 vs EMFAC2007



Results for the South Coast Air Basin are shown in Figure 6-12 and Figure 6-13. Results are very similar in the South Coast as on a Statewide basis, and differences between EMFAC2011 and EMFAC2007 are caused by the same factors discussed above.

Results for the San Joaquin Valley are shown in Figure 6-14 for NOx. In the figure NOx emissions are flat between 1990 and 2005, which is different than the trend seen Statewide or in the South Coast. These trends differ because the San Joaquin Valley is less densely populated and so historically cars, which used to have higher NOx emission rates but are today well controlled, played less of a role in overall San Joaquin Valley emissions than they did in South Coast or Statewide. NOx emissions are reduced in the San Joaquin Valley due to the penetration of newer trucks with cleaner technologies that is caused both by natural turnover and the Statewide Truck and Bus rule. Figure 6-15 shows ROG emissions in the San Joaquin Valley. ROG emissions are dominated by cars and so emissions in the San Joaquin Valley are much lower in total than in the South Coast. The reduction in emissions between EMFAC2011 and EMFAC2007 is caused by the same factors affecting differences in statewide ROG emissions.

Figure 6-12. South Coast NOx Emissions: EMFAC2011 vs EMFAC2007

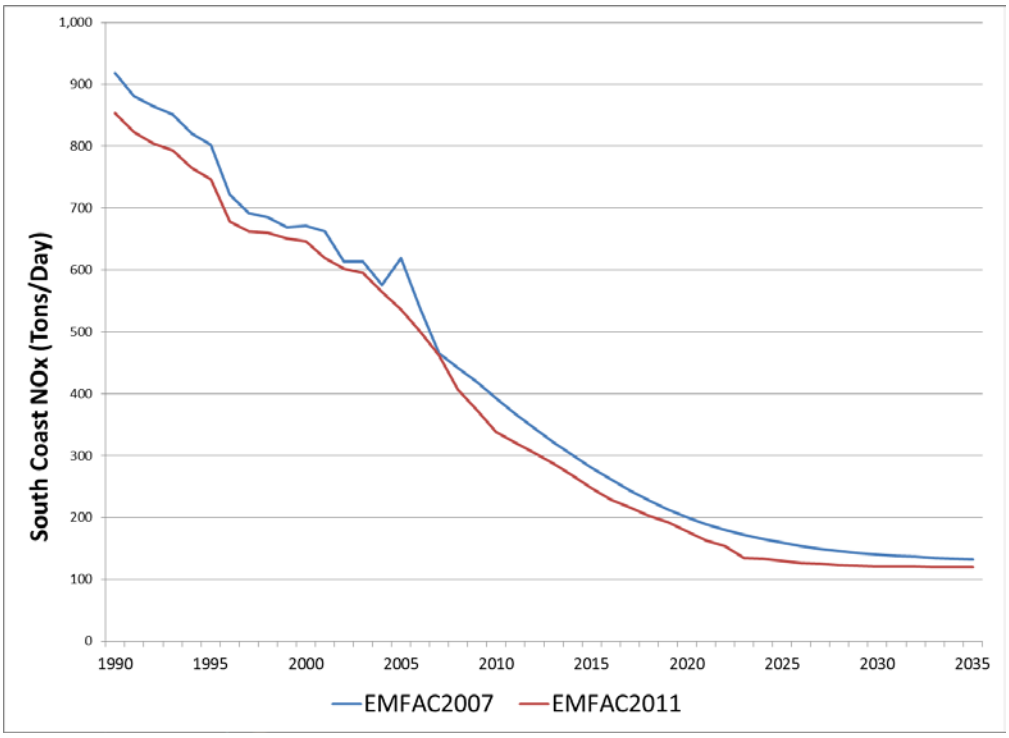


Figure 6-13. South Coast ROG Emissions: EMFAC2011 vs. EMFAC2007

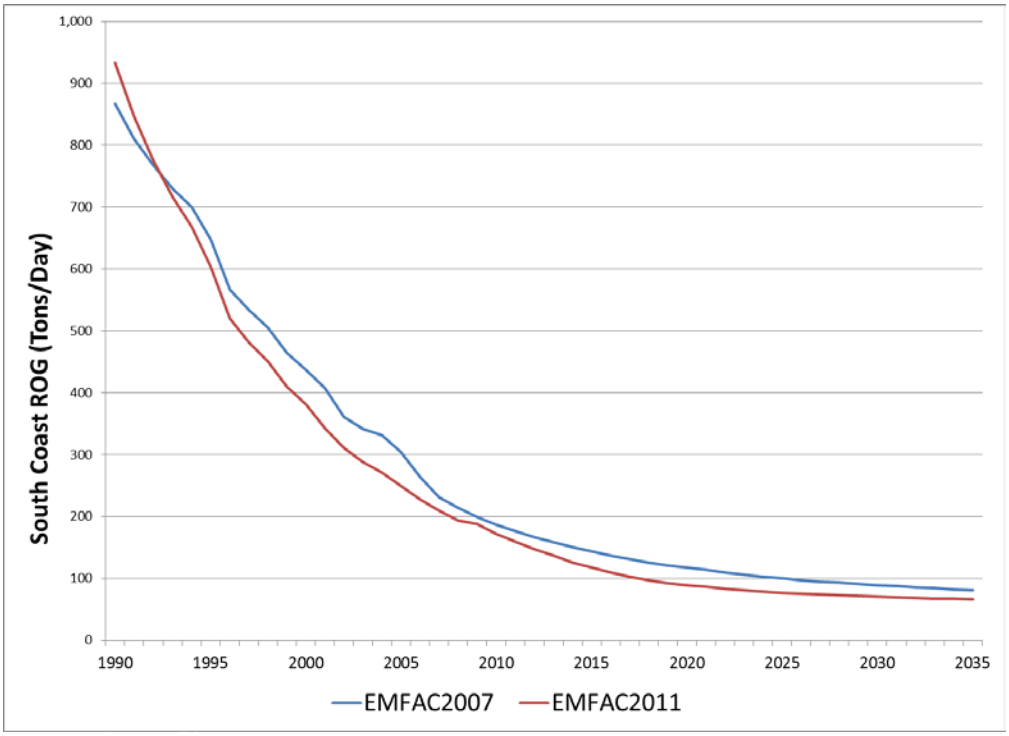


Figure 6-14. San Joaquin Valley NOx Emissions: EMFAC2011 vs. EMFAC2007

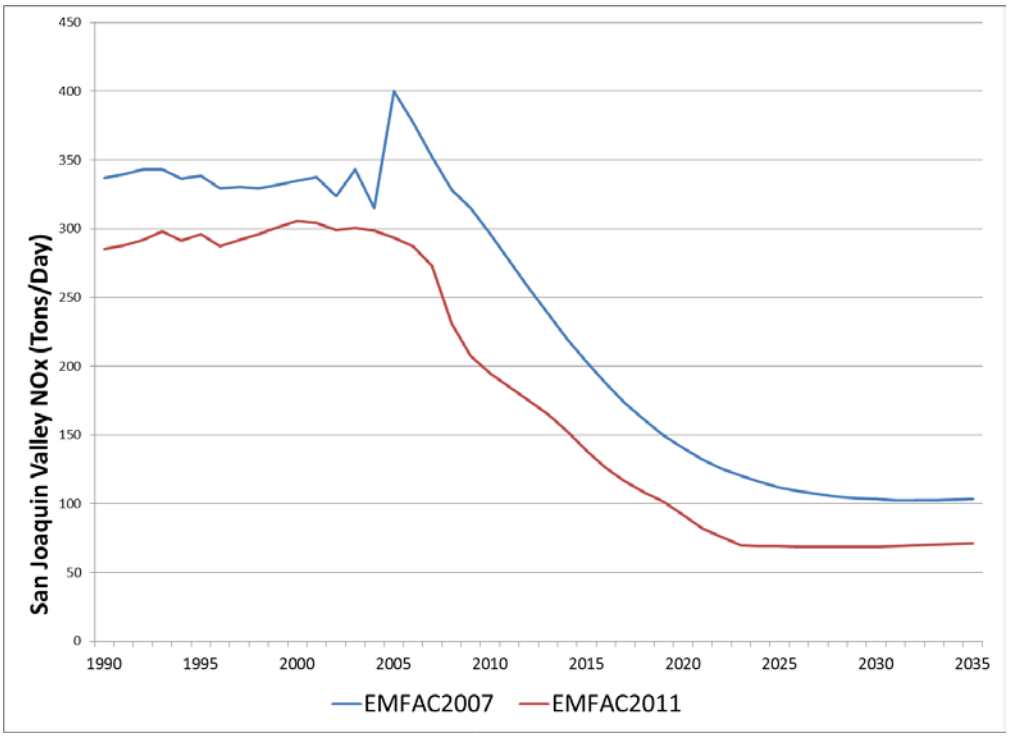
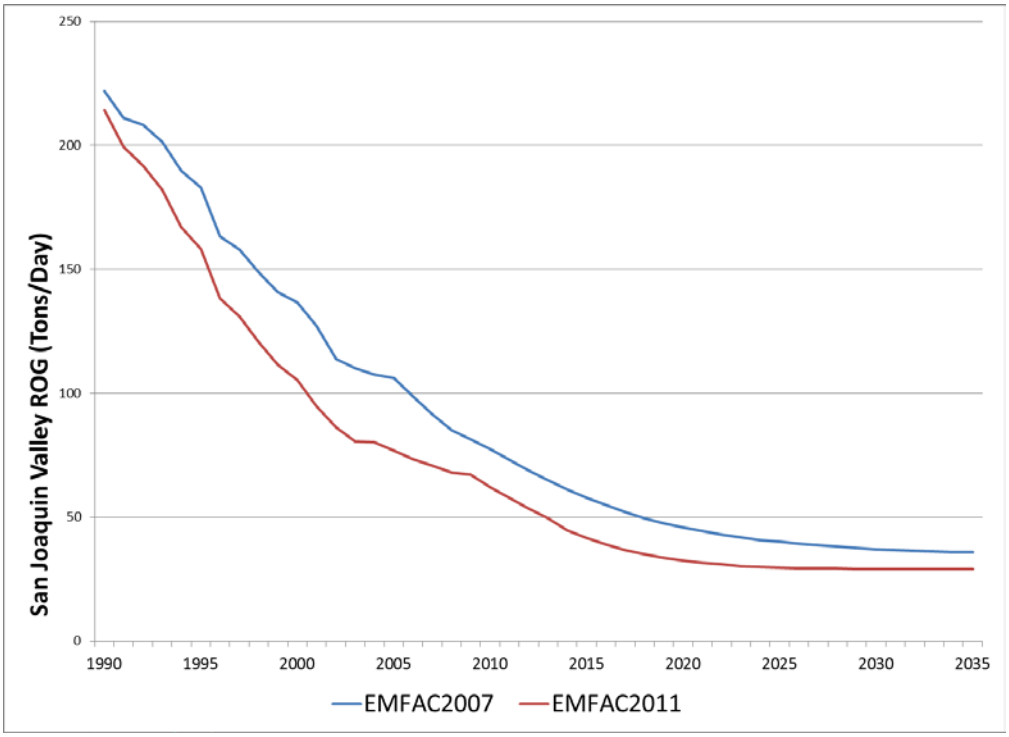


Figure 6-15. San Joaquin Valley ROG Emissions: EMFAC2011 vs. EMFAC2007



6.5 PM2.5 EMISSIONS

Statewide PM2.5 emissions are shown in Figure 6-16. Backcast emissions are higher because of new test data suggesting exhaust PM2.5 emission factors were higher for older vehicles historically than thought in EMFAC2007, which is discussed in Section 3.3.3.6. The anomaly in DMV population data led to an artificial increase in PM2.5 emissions in 2005 in EMFAC2007, which has been corrected in EMFAC2011. Forecast PM2.5 emissions are lower in EMFAC2011 than in EMFAC2007 because of the Statewide Truck and Bus Rule that drastically reduces diesel PM2.5 emissions from diesel trucks,and updates to future gasoline vehicle PM emission rates. Those decreases are partially offset by increases to brake wear PM emission rates.

Trends in South Coast, shown in Figure 6-17 are similar to those seen Statewide and caused by the same factors. Forecasted trends in the San Joaquin Valley, shown in Figure 6-18, are similar, however backcast emissions are lower in EMFAC2011 than in EMFAC2007 because EMFAC2011 assumes less total truck VMT than assumed in EMFAC2007, which has the effect of reducing PM2.5 emissions historically in the region.

Figure 6-16. Statewide PM2.5 Emissions: EMFAC2011 vs. EMFAC2007

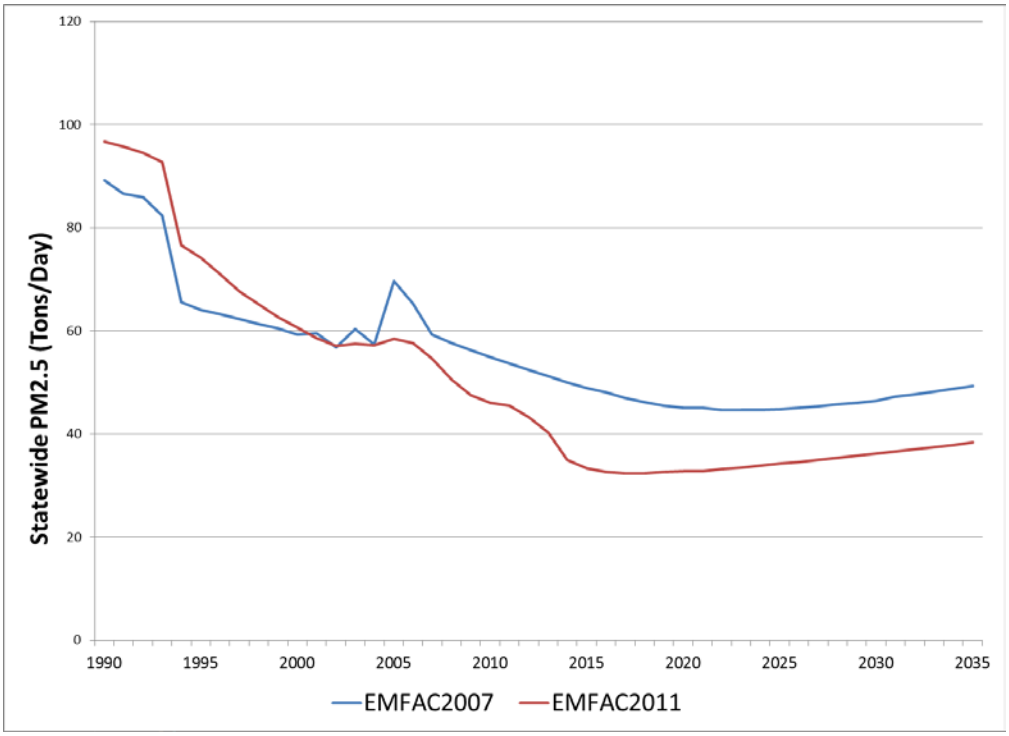


Figure 6-17. South Coast PM2.5 Emissions: EMFAC2011 vs. EMFAC2007

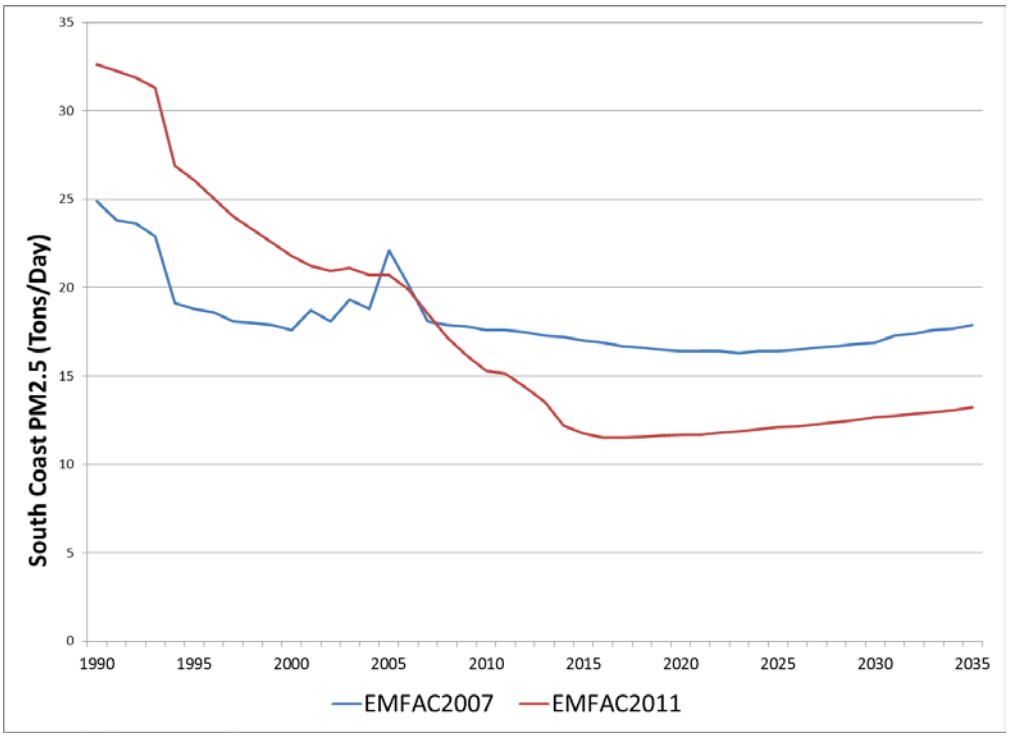
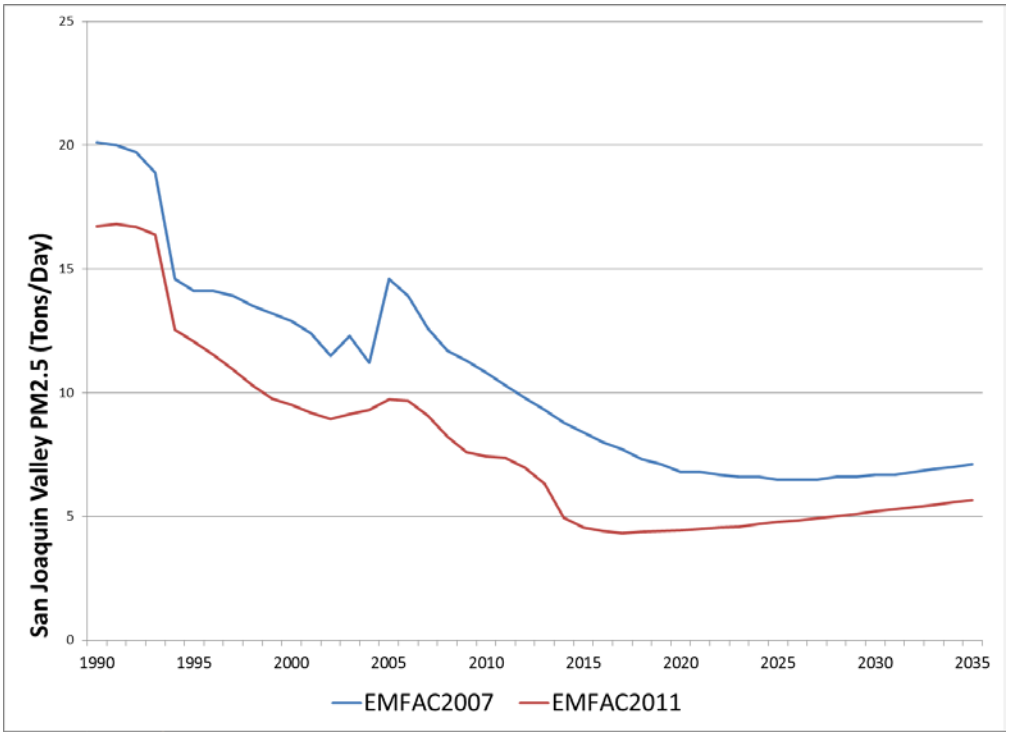


Figure 6-18. San Joaquin Valley PM2.5 Emissions: EMFAC2011 vs. EMFAC2007.



For detailed emissions impacts of each incremental change on selected air basins and calendar years of the State, see Section 12.

7 APPENDIX: UPDATING VEHICLE MILES TRAVELED AND SPEED DISTRIBUTIONS IN EMFAC2011

This section documents the final revisions to the motor vehicle activity data used EMFAC 2011. Vehicle miles traveled (VMT) and speed distributions were submitted by regional transportation planning agencies (RTPAs) in calendar year 2010 and early 2011 for five regions: South Coast region, the San Francisco Bay Area region, San Diego County, San Joaquin Valley region and Sacramento region. In addition to discussing the most recent activity data submitted by the TPAs, the memo recaps key information and issues from staff's review of TPA activity data submitted in 2010 and early 2011. Emissions impacts of the updated data are available in Sections 12.9 and 12.13.

ARB maintains EMFAC and periodically updates the model with new data. RTPAs are generally responsible for estimating vehicle miles and speed of travel in their respective regions. ARB relies on RTPAs, as well as the Department of Motor Vehicles (DMV), and the Bureau of Automotive Repair (BAR), for vehicle activity data in EMFAC. ARB solicited the data most recently adopted or proposed for adoption, prior to developing EMFAC 2011 model updates. In the absence of recent RTPA data, the model contains default speed distributions and estimated VMT as a function of vehicle population (from DMV) and mileage accrual rates (from BAR). Typically, for heavy-duty vehicles default speed distributions are based on instrumented vehicle studies. In some areas of the state, the VMT and speed distributions contained in EMFAC 2011 were carried forward from EMFAC 2007.

The years of VMT data provided by local RTPAs are summarized in Table 7-1. ARB processed base year vehicle population data from the California Department of Motor Vehicles (DMV) for year 2009. The model matched for 2009 population and VMT by allowing the accrual rate to float. The base years provided by RTPAs are also listed in Table 7-1.

Table 7-1. VMT and Speed Updated by Area

Agency	County	GAI	Base Year	Calendar Years	Received
SCAG	Ventura	58	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	Oct 2010
	Los Angeles	59	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
	Orange	60	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
	Riverside	61	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
	San Bernardino	62	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
	Imperial	63	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
	Riverside	64	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
	Riverside	66	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
	Riverside	67	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
	Los Angeles	68	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
	San Bernardino	69	2003	2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035	
Fresno COG	Fresno	48	2003	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035	Oct 2010
Kern COG	Kern (SJV)	49	2006	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035	
Kern COG	Kern (MD)	65	2006	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035	
Kings COG	Kings	50	2005	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035	
Madera COG	Madera	51	2000	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035	
Merced COG	Merced	52	2000	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035	
San Joaquin COG	San Joaquin	53	2005	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035	
Stanislaus COG	Stanislaus	54	2006	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035	
Tulare COG	Tulare	55	2007	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035	
SANDAG	San Diego	38	2008	2008, 2010, 2020, 2030	Oct 2010
SACOG	El Dorado (MC)	9	2005	2005, 2018, 2035	Feb 2011
	Placer (MC)	12	2005	2005, 2018, 2035	
	Placer (SV)	30	2005	2005, 2018, 2035	
	Sacramento	31	2005	2005, 2018, 2035	
	Sutter	34	2005	2005, 2018, 2035	
	Yolo	36	2005	2005, 2018, 2035	
	Yuba	37	2005	2005, 2018, 2035	
MTC	Sonoma (NC)	22	2005	2005, 2035	Jan 2011
	Solano (SV)	33	2005	2005, 2035	
	Alameda	39	2005	2005, 2035	
	Contra Costa	40	2005	2005, 2035	
	Marin	41	2005	2005, 2035	
	Napa	42	2005	2005, 2035	
	San Francisco	43	2005	2005, 2035	
	San Mateo	44	2005	2005, 2035	
	Santa Clara	45	2005	2005, 2035	
	Solano	46	2005	2005, 2035	
	Sonoma	47	2005	2005, 2035	

The RTPAs submitted VMT by speed bin by period of the day for selected calendar years. There are 14 speed bins from 5 to 70 miles per hour in 5 miles increment. Each RTPA may have different definitions of period ranging from daily, where the same speed distribution applies throughout the day, to hourly where distributions vary. However, most RTPAs submitted VMT by speed bin for morning peak (AMPK), afternoon peak (PMPK) and one or two off-peak periods. Table 7-2 summarizes the period definitions by area and calendar years provided by RTPAs. EMFAC coding requires six time periods: AMPK, Noon, Mid-day, PMPK and 2 Night periods, and staff matched the time periods provided by RTPAs to one of the six time periods in EMFAC as described in the regional submittal tables. For each region and time period, ARB staff developed the speed distribution using the VMT in the speed bins and the total VMT from all speed bins. ARB did not interpolate or extrapolate speed distributions, but applied the speed distribution for the next future year to interim years. The Southern California Association of Governments (SCAG) submitted separate VMTs for light-duty vehicles (LDV) and heavy-duty trucks (HDT). For all other regions, ARB default HDT speeds were used. In the absence of recent RTPA data, submittals for EMFAC2007 development were carried forward.

Staff evaluated speed data submitted by each RTPA, looking to make sure data were consistent and that generally in most cases peak speeds in urban areas declined with time, that peak speeds in growing rural areas were declining with time, that nighttime speeds were flat, and that peak speeds in rural non-growing areas were relatively high and flat over time. Speed data appeared reasonable in all cases and so were input to the model. Table 7-2 summarizes the period definitions by area and shows some differences from ARB's period definitions.

Table 7-2. Time Period Definitions for EMFAC 2011

GAI	AIR BASIN	COUNTY	COID	CALENDAR YEARS SUBMIITED	PERIODS SUBMITTED
9	MC	El Dorado County (portion)	9	**2005, 2018, 2035**	AMPK, NOON, PMPK, NIGHT
12	MC	Placer County (portion)	31	**2005, 2018, 2035**	AMPK, NOON, PMPK, NIGHT
30	SV	Placer County (portion)	31	**2005, 2018, 2035**	AMPK, NOON, PMPK, NIGHT
31	SV	Sacramento County	34	**2005, 2018, 2035**	AMPK, NOON, PMPK, NIGHT
34	SV	Sutter County	51	**2005, 2018, 2035**	AMPK, NOON, PMPK, NIGHT
36	SV	Yolo County	57	**2005, 2018, 2035**	AMPK, NOON, PMPK, NIGHT
37	SV	Yuba County	58	**2005, 2018, 2035**	AMPK, NOON, PMPK, NIGHT
38	SD	San Diego County	37	**2008, 2010, 2020, 2030**	Hourly converted to 6 periods
39	SF	Alameda County	1	**2000, 2005, 2035**	2, 5, 9, 11, 14, 18, 23
40	SF	Contra Costa County	7	**2000, 2005, 2035**	2, 5, 9, 11, 14, 18, 23
41	SF	Marin County	21	**2000, 2005, 2035**	2, 5, 9, 11, 14, 18, 23
42	SF	Napa County	28	**2000, 2005, 2035**	2, 5, 9, 11, 14, 18, 23
43	SF	San Francisco County	38	**2000, 2005, 2035**	2, 5, 9, 11, 14, 18, 23
44	SF	San Mateo County	41	**2000, 2005, 2035**	2, 5, 9, 11, 14, 18, 23
45	SF	Santa Clara County	43	**2000, 2005, 2035**	2, 5, 9, 11, 14, 18, 23
46	SF	Solano County (portion)	48	**2000, 2005, 2035**	2, 5, 9, 11, 14, 18, 23
47	SF	Sonoma County (portion)	49	**2000, 2005, 2035**	2, 5, 9, 11, 14, 18, 23
48	SJV	Fresno County	10	**2003, 2011, 2012, 2014, 2017**	AMPK, PMPK, OFFPK
				2020, 2023, 2025, 2035	
49	SJV	Kern County (portion)	15	**2006, 2011, 2012, 2014, 2017**	AMPK, PMPK, OFFPK
				2020, 2023, 2025, 2035	
50	SJV	Kings County	16	**2005, 2011, 2012, 2014, 2017**	DAILY
				2020, 2023, 2025, 2035	
51	SJV	Madera County	20	**2000, 2011, 2012, 2014, 2017**	DAILY
				2020, 2023, 2025, 2035	
52	SJV	Merced County	24	**2000, 2011, 2012, 2014, 2017**	DAILY
				2020, 2023, 2025, 2035	
53	SJV	San Joaquin County	39	**2005, 2011, 2012, 2014, 2017**	AMPK, PMPK, OFFPK
				2020, 2023, 2025, 2035	
54	SJV	Stanislaus County	50	**2006, 2011, 2012, 2014, 2017**	AMPK, PMPK, OFFPK
				2020, 2023, 2025, 2035	
55	SJV	Tulare County	54	**2007, 2011, 2012, 2014, 2017**	AMPK, PMPK, OFFPK
				2020, 2023, 2025, 2035	
58	SCC	Ventura County	56	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	
59	SC	Los Angeles County (portion)	19	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	
60	SC	Orange County	30	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	
61	SC	Riverside County (portion)	33	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	
62	SC	San Bernardino County (portion)	36	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	
63	SS	Imperial County	13	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	
64	SS	Riverside County (portion)	33	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	
66		Los Angeles County (portion)	19	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	
67/68	MD	Riverside County (portion)	33	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	
69	MD	San Bernardino County (portion)	36	**2003, 2008, 2009, 2010, 2012,**	AMPK, NOON, PMPK, NIGHT
				2014, 2018, 2020, 2023, 2030, 2035	

SCAG Region

The Southern California Association of Governments (SCAG) has submitted the latest motor vehicle activity for light-duty vehicles (LDV) and heavy-duty trucks (HDT). The submittal includes both vehicle miles traveled (VMT) and speed distributions for the CYs 2003, 2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035. ARB staff reviewed the data for anomalies and compared the VMT and speed trends to SCAG 2009 submittals, EMFAC 2007, MVSTAFF and human population. Table 7-3 describes the VMT and speed distribution data submitted by SCAG. Table 7-4 and Table 7-5 summarize the latest SCAG submittal for LDV+MDV and HDT respectively.

Table 7-3. SCAG Submittal

Traffic Model Citation:	The adopted 2008 RTP					
Date of Submittal:	October 12, 2010					
Years Provided:	2003, 2008, 2009, 2010, 2012, 2014, 2018, 2020, 2023, 2030, 2035.					
VMT Vehicle Classes:	SCAG provided LDV VMT totals for light- and medium-duty vehicles plus motorcycles combined and HDT VMT totals for heavy-duty trucks combined. (No data for buses or motor homes.) ARB will match SCAG total VMT for all vehicle classes except buses and motorhomes by sub-area and distribute VMT by vehicle class based on updated DMV/Smog Check data. The HD diesel truck, other buses (diesel) and school buses (diesel) VMT by vehicle class are from ARB's EMFAC2011-HD truck model and updated DMV and mileage accrual rate data. ARB will add VMT for remaining classes bus and motor homes using DMV data.					
Speed Vehicle Classes:	Speeds were provided for light- and medium-duty vehicles combined and separate speeds for heavy-duty trucks derived from SCAG's truck model.					
Speed Distributions by Periods:	SCAG provided speed distributions for AM Peak (period 2), PM Peak (period 5), Mid-day (periods 3 and 4), and Night (periods 1 and 6)					
Period Definitions	**Period 1**	**Period 2**	**Period 3**	**Period 4**	**Period 5**	**Period 6**
	Hours 0-5	6-8	9-11	12-14	15-18	19-23

Table 7-4. SCAG Daily VMT for Light & Medium-Duty Vehicles

	2003	2008	2009	2010	2012	2014	2018	2020	2023	2030	2035
SCCAB (VENTURA COUNTY)	17,413,687	18,133,177	18,589,246	18,916,106	19,194,130	19,455,513	20,098,341	20,339,177	20,670,693	21,572,162	21,929,329
SCAB (LOS ANGELES COUNTY)	197,363,189	197,642,045	199,885,430	200,687,898	202,609,690	203,912,836	208,530,138	210,610,605	212,542,579	218,591,292	221,559,676
SCAB (ORANGE COUNTY)	66,508,789	69,424,103	70,415,994	70,919,803	71,866,039	73,021,835	75,709,228	76,326,599	77,106,333	80,031,953	81,490,036
SCAB (RIVERSIDE COUNTY)	28,577,374	32,934,988	34,003,198	34,690,277	36,068,252	37,480,079	40,953,166	43,207,006	45,406,357	51,019,470	54,692,082
SCAB (SAN BERNARDINO CO)	31,191,348	33,365,272	34,530,061	34,943,836	36,096,490	36,776,758	39,260,208	40,857,102	42,363,075	46,072,018	48,789,598
MDAB (ANTELOPE VALLEY)	7,267,648	11,991,960	13,009,220	13,640,897	14,202,335	14,840,779	16,133,352	16,775,809	17,957,168	21,316,360	23,386,688
MDAB (SBD/VICTOR VALLEY)	12,025,375	14,440,422	15,007,736	15,470,876	16,151,550	16,526,470	17,699,114	18,586,270	19,801,108	22,989,905	25,324,447
MDAB (SBD/SEARLES VALLEY)	146,642	135,674	135,311	135,296	146,870	146,630	135,264	134,625	135,954	137,391	138,637
MDAB (SBD DESERT AREA)	3,991,069	4,503,771	4,610,436	4,693,348	4,841,857	5,018,906	5,450,014	5,696,524	6,096,714	7,175,768	8,081,644
MDAB (RIV/SCAQMD)	825,631	818,005	829,997	844,803	887,292	915,182	949,590	970,357	1,047,742	1,234,069	1,412,958
MDAB (RIV/MDAQMD)	658,434	747,333	774,647	780,944	898,733	1,071,474	1,458,543	1,614,711	1,722,600	2,011,027	2,207,366
SSAB (RIV/COACHELLA VALLEY)	9,287,682	11,169,419	11,538,819	11,840,696	12,530,631	13,304,204	15,134,322	16,254,281	17,768,683	21,415,665	24,083,905
SSAB (IMPERIAL)	4,131,747	5,596,791	5,811,566	6,017,818	6,437,638	7,191,621	8,709,248	9,522,149	10,207,464	11,981,615	12,999,895
SSAB (IMP MOUNTAIN AREA)	203,114	259,625	265,705	269,396	331,975	432,095	689,459	804,617	868,129	1,049,859	1,168,751
	==========	==========	==========	==========	==========	==========	==========	==========	==========	==========	==========
	379,591,729	401,162,584	409,407,365	413,851,992	422,263,482	430,094,381	450,909,986	461,699,834	473,694,598	506,598,554	527,265,011

Table 7-5. SCAG Daily VMT for Heavy-duty Trucks (T5, T6, T7, & T8)

	2003	2008	2009	2010	2012	2014	2018	2020	2023	2030	2035
SCCAB (VENTURA COUNTY)	1,213,762	1,290,481	1,315,000	1,339,826	1,379,706	1,417,515	1,503,290	1,542,867	1,598,207	1,723,514	1,797,606
SCAB (LOS ANGELES COUNTY)	11,656,122	12,315,589	12,578,242	12,851,058	13,172,808	13,448,619	14,173,911	14,618,063	15,075,112	16,072,957	16,717,001
SCAB (ORANGE COUNTY)	3,773,633	3,886,545	3,954,093	4,014,088	4,115,012	4,208,992	4,439,819	4,502,056	4,610,851	4,833,335	4,984,754
SCAB (RIVERSIDE COUNTY)	2,516,083	3,023,172	3,119,933	3,229,899	3,410,416	3,583,539	3,948,767	4,112,515	4,342,359	4,859,805	5,188,530
SCAB (SAN BERNARDINO CO)	3,159,385	3,790,804	3,977,303	4,122,268	4,351,627	4,555,756	4,939,192	5,165,764	5,445,746	6,049,384	6,536,718
MDAB (ANTELOPE VALLEY)	388,404	411,613	427,810	433,235	451,530	475,465	532,126	560,102	611,275	721,880	790,658
MDAB (SBD/VICTOR VALLEY)	2,481,485	3,195,594	3,346,011	3,491,079	3,726,227	3,953,330	4,426,983	4,660,331	4,974,511	5,691,089	6,210,855
MDAB (SBD/SEARLES VALLEY)	26,355	26,978	27,020	27,185	29,538	29,808	28,140	28,256	28,603	29,181	29,683
MDAB (SBD DESERT AREA)	1,231,852	1,588,309	1,660,019	1,730,995	1,836,579	1,945,551	2,175,887	2,284,020	2,435,401	2,770,730	2,999,864
MDAB (RIV/SCAQMD)	290,291	340,765	351,715	362,660	377,141	393,957	428,659	443,524	465,888	516,265	553,567
MDAB (RIV/MDAQMD)	229,359	265,311	272,629	280,147	292,489	304,615	328,230	339,799	356,486	392,647	417,454
SSAB (RIV/COACHELLA VALLEY)	1,469,194	1,701,337	1,754,251	1,806,866	1,908,317	2,003,180	2,173,760	2,259,484	2,381,607	2,647,268	2,824,574
SSAB (IMPERIAL)	568,065	644,239	664,411	683,215	727,919	774,339	844,158	874,288	914,830	999,912	1,058,210
SSAB (IMP MOUNTAIN AREA)	38,519	41,896	42,832	43,825	45,534	47,104	50,228	51,654	53,673	57,994	61,128
	==========	==========	==========	==========	==========	==========	==========	==========	==========	==========	==========
	29,042,509	32,522,632	33,491,270	34,416,346	35,824,843	37,141,772	39,993,152	41,442,724	43,294,547	47,365,963	50,170,601

Staff reviewed VMT and speed data submitted by SCAG for the South Coast region, comparing growth rates to human population growth trends, and comparing submitted data to the following data sources:

- SCAG 2010 submittal (October 2010).
- EMFAC 2007, Version 2.6 (Nov 06).
- SCAG 2009 submittal (April 2009).
- California Motor Vehicle Stock, Travel and Fuel Forecast, May 2008 (MVSTAFF)
- Human Population, State of California, Department of Finance, Population Projections for California and Its Counties 2000-2050, Sacramento, California, July 2007.

Figure 7-1 compares the SCAG region VMT from the different data sources. The October 2010 submittal follows a similar trend to SCAG 2009 and EMFAC2007. Figure 7-2 illustrates the growth rate of VMT and human population from the different sources. The split between the light-duty and heavy-duty vehicles and the growth rate of these vehicle classes are shown in Figure 7-3. Both SCAG 2010 VMT and human population grow at a similar rate of 1.29% and 1.25% per annum respectively. SCAG's 2010 LDV VMT growth rate at 1.22% and HDT growth rate at 2.27% per annum are similar to the EMFAC2007 LDV VMT growth rate of 1.01% and HDT growth rate of 2.43%. While VMT and human population growth rates are much lower in the SCAG data than MVSTAFF, staff places little weight on MVSTAFF estimates in this area. The SCAG model does not estimate VMT for buses or motor homes. ARB provides the estimates for these vehicle classes based on updated DMV data and mileage accrual rates. In EMFAC2011, other buses (diesel) and school buses (diesel) are based on the ARB's EMFAC2011-HD truck model. Long term heavy duty vehicle VMT forecasts developed by SCAG are similar to those developed for the Statewide Truck and Bus Rule; however short term estimates do not appear to reflect the downturn in the economic cycle.

Figure 7-1. VMT in the SCAG Region

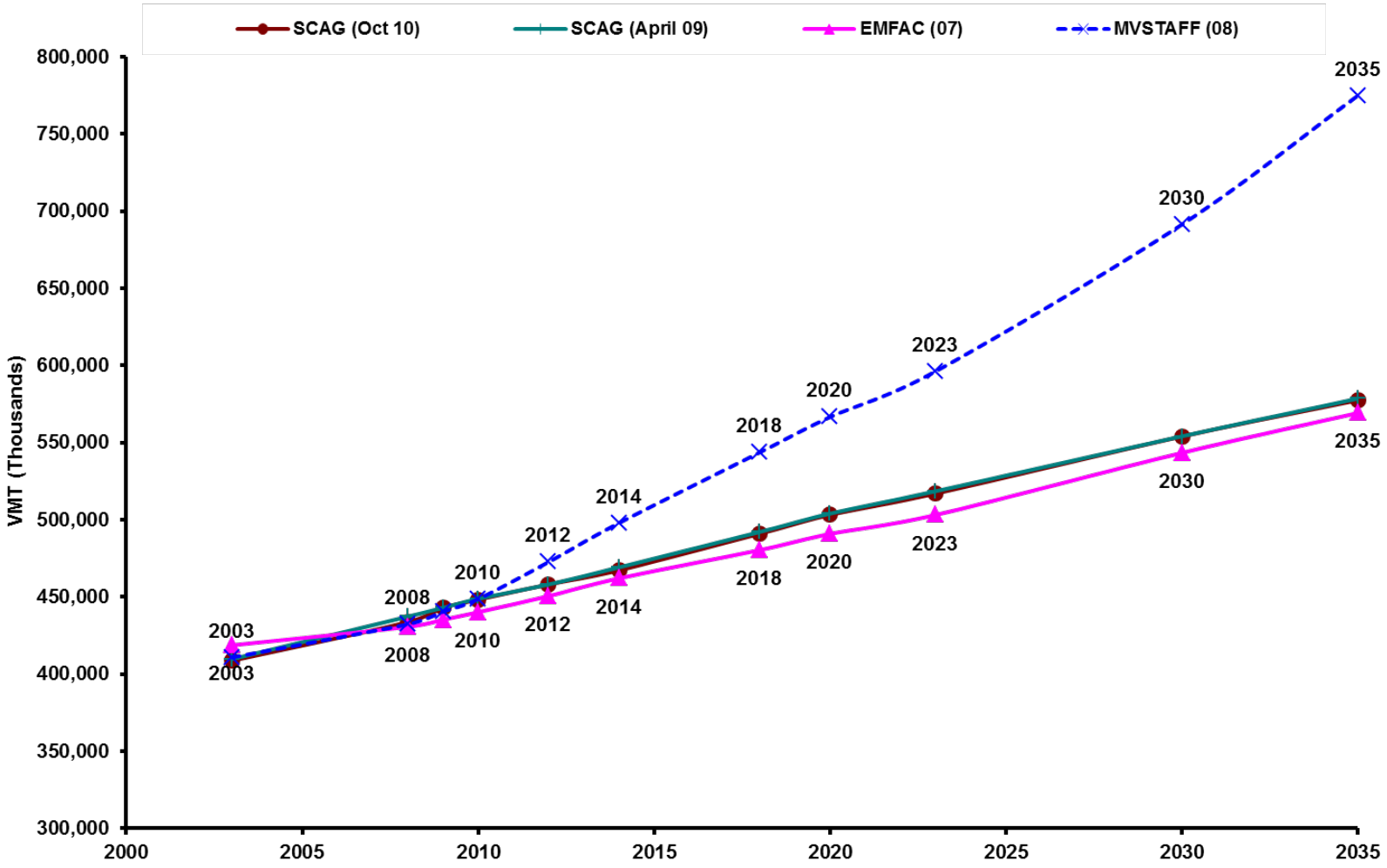


Figure 7-2. VMT and Human Population Growth in the SCAG Region

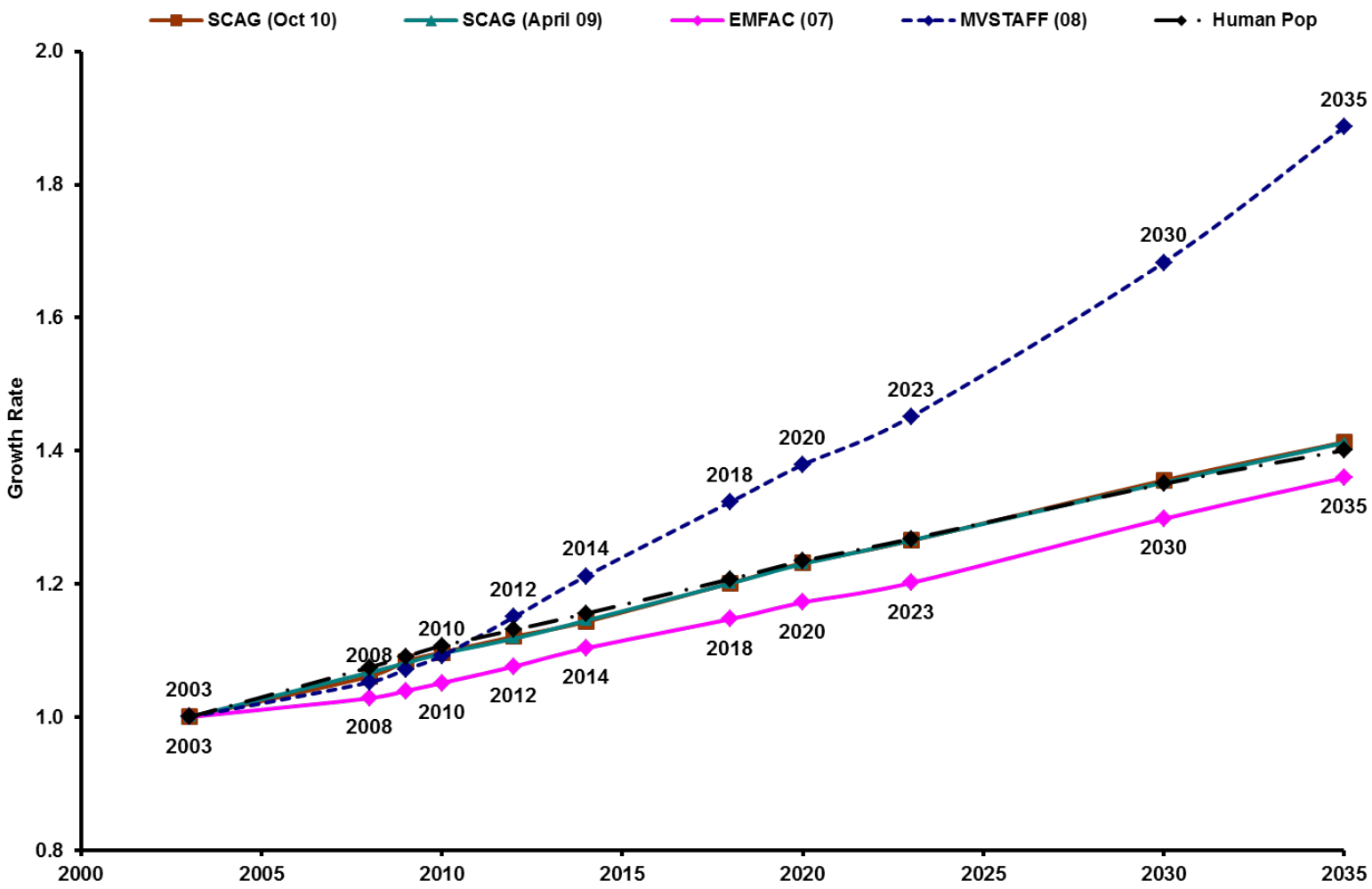
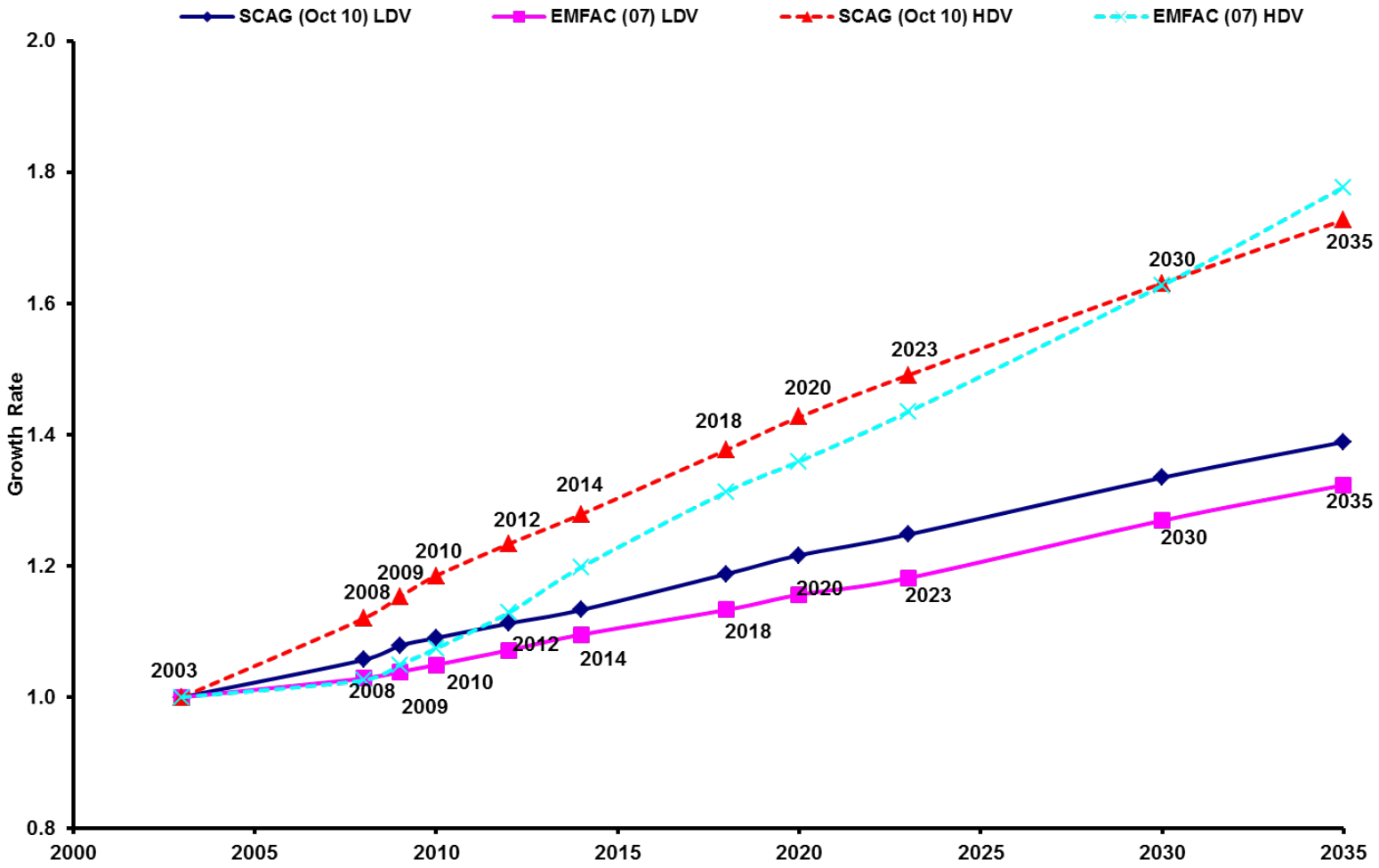


Figure 7-3. Light-Duty vs Heavy-Duty VMT Growth Rates in the SCAG Region



SAN DIEGO REGION

The San Diego Association of Governments (SANDAG) submitted vehicle activity data in October 2011. Table 7-6 provides details on the SANDAG submittal.

Table 7-6. SANDAG Data Submittal Summary

Traffic Model Citation:	**The adopted 2007 RTP.**					
Date of Submittal:	October 2010					
Years Provided:	2008, 2010, 2020, and 2030					
VMT Vehicle Classes:	VMT totals represent all vehicle classes. SANDAG provided VMT by vehicle class and by period; however, EMFAC 2011 reflects VMT distribution over the vehicle classes based on DMV data and ARB EMFAC2011-HD truck model.					
Speed Vehicle Classes:	Speed distributions were provided by vehicle class; however, only distribution for light duty vehicles was used because SANDAG has no heavy duty truck model.					
Speed Distributions by Periods:	Speed distributions were provided hourly and converted to AM Peak (period 2), PM Peak (period 5), Mid-day (periods 3 and 4), and Night (periods 1 and 6).					
Period Definitions	**Period 1**	**Period 2**	**Period 3**	**Period 4**	**Period 5**	**Period 6**
	Hours 0-5	6-8	9-11	12-14	15-17	18-23

Similar to the South Coast data review, ARB staff reviewed the activity data received from SANDAG and compared VMT trends for CYs 2008 through 2030 to other data sources including:

- SANDAG Activity Data Submitted to ARB in October 2010
- EMFAC 2007, Version 2.3 (Nov 2006)
- SANDAG Activity Data Submitted to ARB in 2008
- California Motor Vehicle Stock, Travel and Fuel Forecast, May 2008 (MVSTAFF)
- Human Population, State of California, Department of Finance, Population Projections for California and Its Counties 2000-2050, Sacramento, California, July 2007.

SANDAG provided VMT and speeds by vehicle class; however, because of ARB's recent efforts to update vehicle population and redistribute statewide VMT for heavy-duty trucks, ARB preferred to use SANDAG total VMT for the fleet as a whole but with ARB VMT distributions by vehicle class. The heavy heavy-duty truck VMT was updated to agree with ARB's EMFAC2011-HD truck model redistribution. DMV data was used to distribute the light-duty vehicles. Similarly, SANDAG agreed to use ARB default speed distributions for heavy-duty vehicles. EMFAC 2011 reflects SANDAG speed distributions for light-duty cars and trucks plus motorcycles. Figure 7-4 compares the SANDAG region VMT from different data sources. Figure 7-5 illustrates the growth rate of VMT and human population from the different sources. The October 2010 submittal follows a similar trend to SANDAG 2008 and EMFAC2007. However, it is about 7-10% lower than their previous submittal. Thought the total EMFAC2007 VMT is higher than the SANDAG 2010 submittal VMT, the annual growth rate is lower.

Figure 7-4. VMT in the San Diego Region

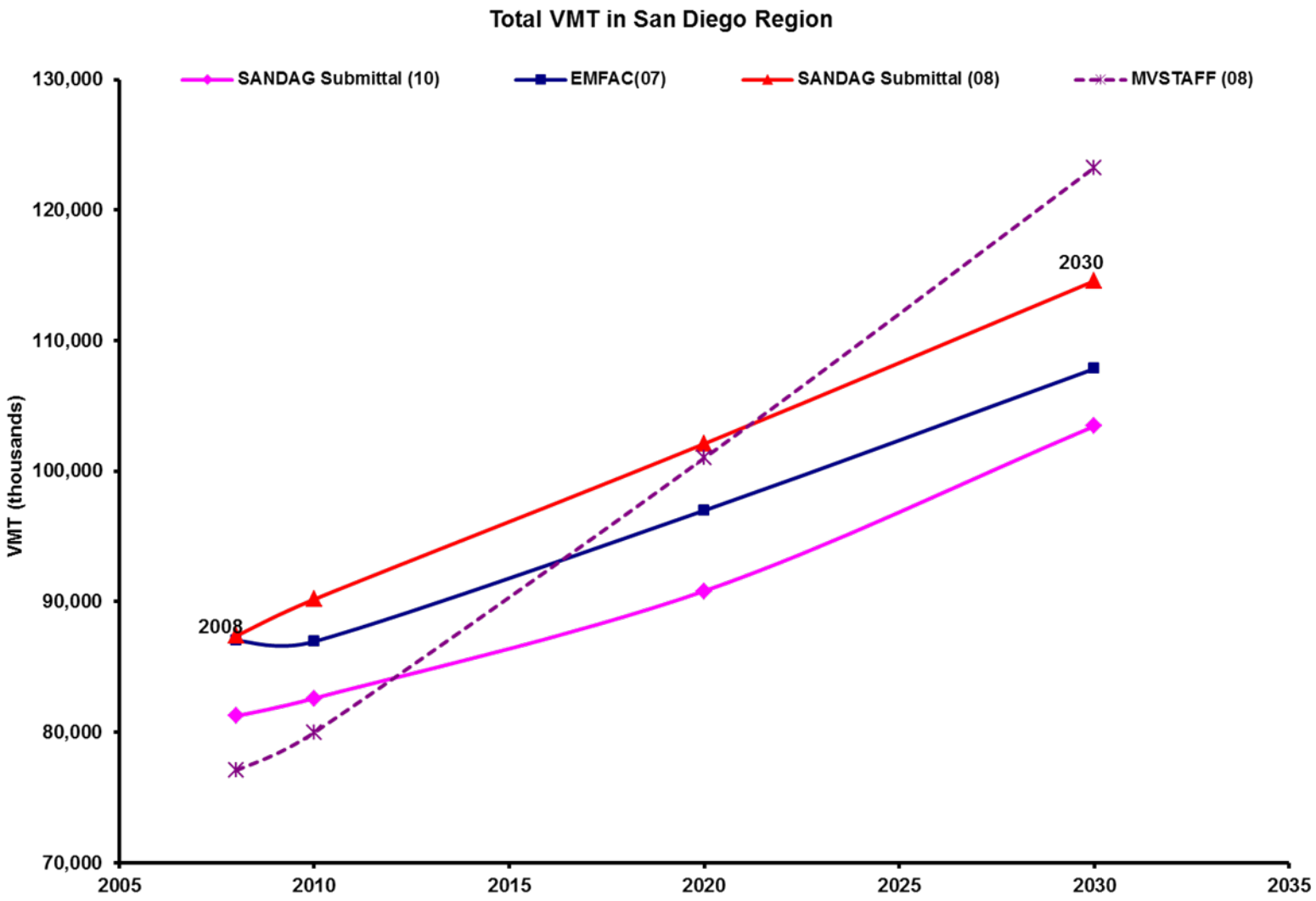
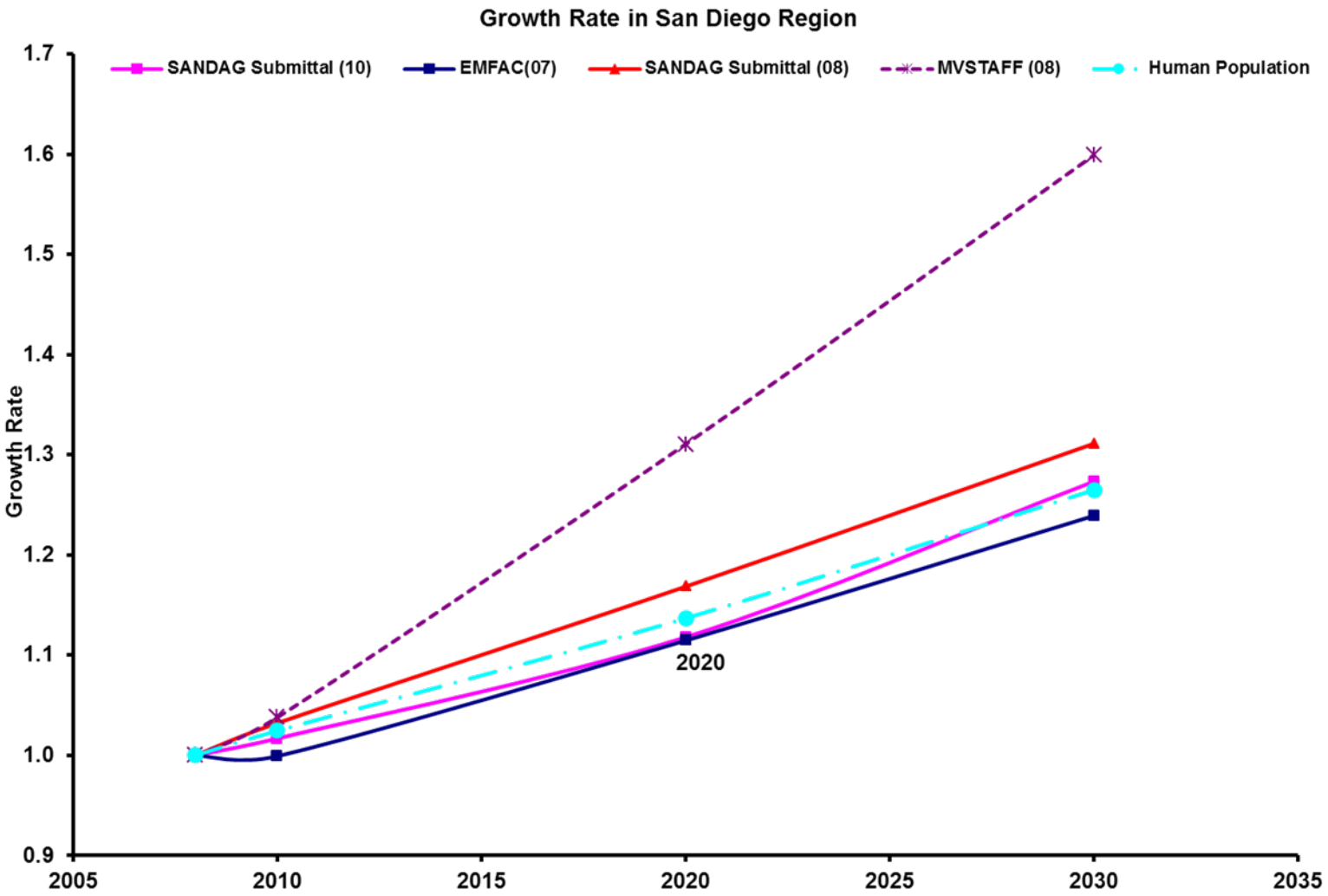


Figure 7-5. VMT and Human Population Growth in the San Diego Region



SAN FRANCISCO BAY AREA

The Metropolitan Transportation Commission (MTC) provided updated activity data in January 2011. Table 7-7 describes the VMT and speed distribution data submitted by MTC.

Table 7-7. Data Submittal from MTC

Traffic Model Citation:	Travel Model One (activity based model) using ABAG's Projections 2009 demographic/socio-economic/land forecast developed and adopted by ABAG in March 2009.					
Date of Submittal:	January 2011					
Years Provided:	2000, 2005, 2035					
VMT Vehicle Classes:	VMT totals represent all vehicle classes. EMFAC 2011 reflects VMT distribution over the vehicle classes based on DMV data and ARB EMFAC2011-HD truck model.					
Speed Vehicle Classes:	Apply to light-duty cars and trucks, medium-duty vehicles, and motorcycles. ARB default speeds to be used for all heavy-duty vehicles.					
Speed Distributions and Periods:	MTC submitted their speed distribution in 7 periods, which staff converted into 6 periods: AM Peak (period 2), PM Peak (period 5), Midday (periods 3 and 4), and Night (periods 1 and 6).					
Period Definitions	**Period 1**	**Period 2**	**Period 3**	**Period 4**	**Period 5**	**Period 6**
	Hours 3-5	6-9	10-11	12-14	15-18	19-2

Similar to the South Coast QA process, ARB staff reviewed MTC's VMT and speed distributions. ARB compared MTC's VMT and growth rates by county, CYs 2005 and 2035 to the following data sources:

- MTC Activity Data Submitted to ARB (Jan 2011)
- EMFAC 2007, Version 2.3 (Nov 2006)
- EMFAC 2011 (applied MTC growth rate for future years)
- California Motor Vehicle Stock, Travel and Fuel Forecast, May 2008 (MVSTAFF)
- Human Population, State of California, Department of Finance, Population Projections for California and Its Counties 2000-2050, Sacramento, California, July 2007.

Table 7-8 shows Daily VMT by County in the MTC region by calendar year.

Table 7-8. MTC Daily VMT by County

MTC	2000	2005	2035
Alameda	36,924,303	36,991,842	46,805,997
Contra Costa	19,947,507	21,437,898	26,777,372
Marin	6,064,389	6,186,610	6,962,727
Napa	2,611,517	3,045,574	3,513,033
San Francisco	8,724,235	8,699,284	10,568,527
San Mateo	15,543,143	14,841,629	18,518,543
Santa Clara	38,572,183	37,645,288	49,766,067
So. Solano	6,291,308	6,923,862	8,190,946
So. Sonoma	7,934,749	8,242,240	9,664,187
No. Solano	4,276,033	4,502,751	5,326,765
No. Sonoma	1,854,636	2,118,420	2,483,889
Total	148,744,003	150,635,398	188,578,053

For EMFAC2011, MTC VMT growth rates were applied to the default CY 2000 base year VMT in EMFAC 2011 to estimate the MTC CY 2005 and 2035 VMT. VMT comparison and growth rates for the San Francisco Air Basin are shown in Figure 7-6 and Figure 7-7. The EMFAC 2011 VMT is about 11 – 25% higher than the MTC 2011 VMT. Both MTC 2011 VMT and human population grow at a similar rate of 0.8% and 0.9% per annum respectively.

One important issue discussed during the QA process on MTC's data was VMT estimates for base year 2000. Prior investigations as well as the current analysis by ARB showed that MTC VMT estimates were significantly lower than those based on the DMV vehicle populations plus mileage accrual rates determined from California's Smog Check Program. In an effort to be fully protective of air quality, ARB, MTC, and the Bay Area Air Quality Management District (BAAQMD) determined that it would be better to have VMT forecasts that err on the side of being too high rather than too low. Therefore, these agencies agreed to adjust Bay Area VMT before using it in air quality plans or in the EMFAC model pending future efforts to determine the reasons for VMT discrepancies among independent sources of data.

Figure 7-6. VMT in the San Francisco Bay Area

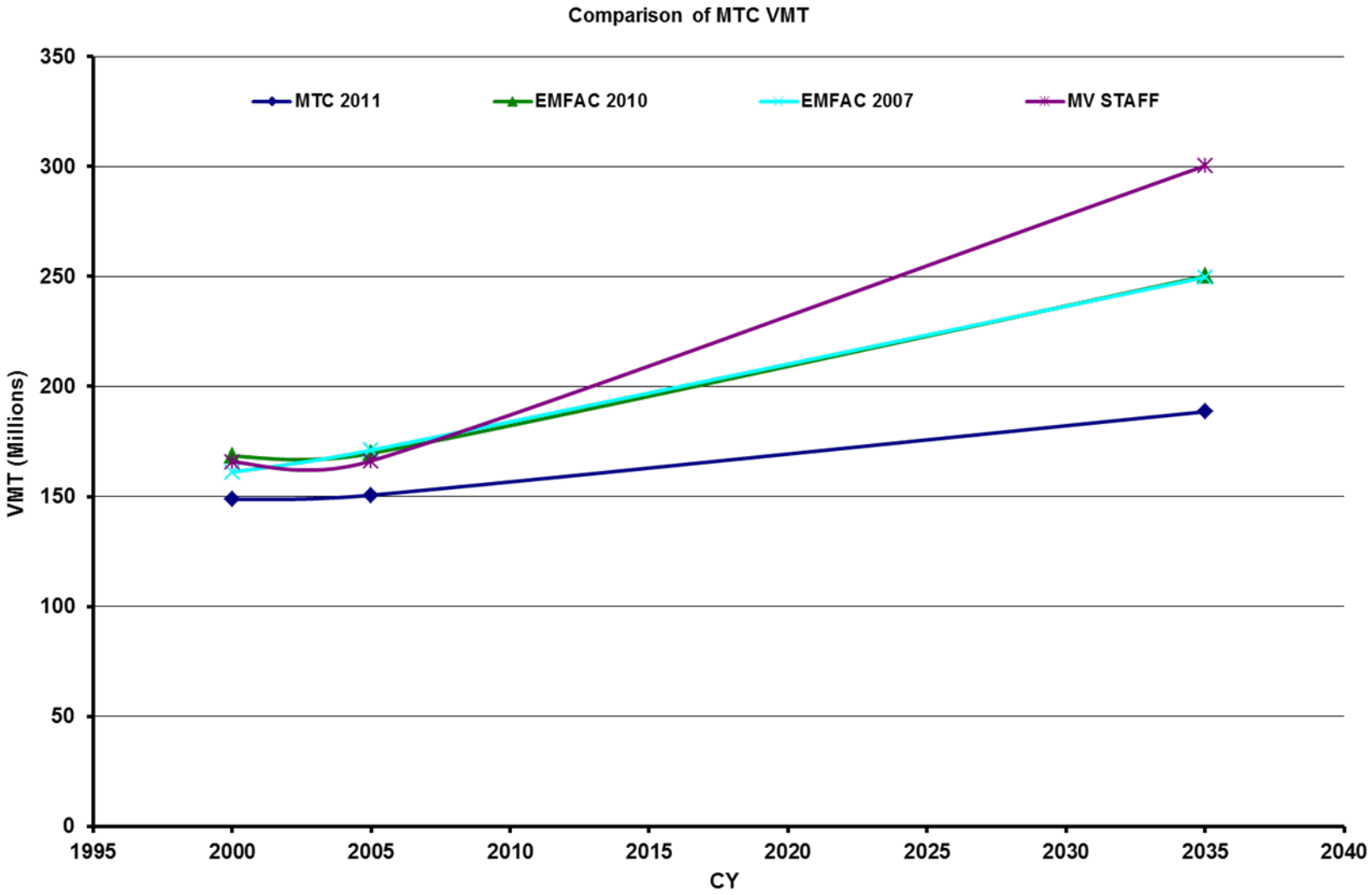
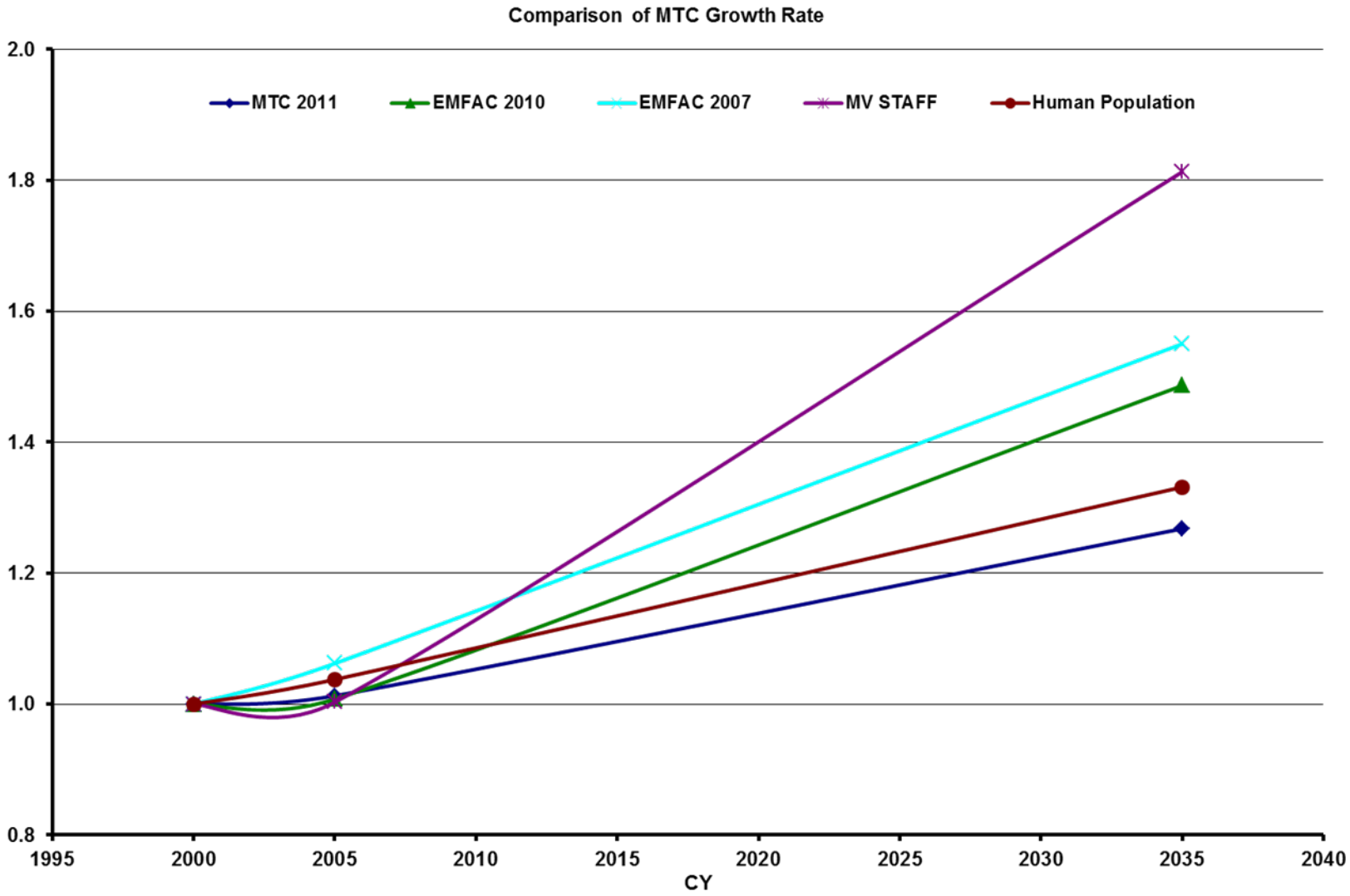


Figure 7-7. VMT and Human Population Growth in the San Francisco Bay Area



SACRAMENTO METROPOLITAN AREA

Sacramento Area Council of Governments (SACOG) provided ARB with VMT and speed distributions in February 2011. Table 7-9 illustrates the VMT and speed distribution data submitted by SACOG.

Table 7-9. SACOG Submittal

Traffic Model Citation:	The adopted Metropolitan Transportation Plan 2035.					
Date of Submittal:	February 2011					
Years Provided:	2005, 2018, and 2035					
VMT Vehicle Classes:	VMT totals represent all vehicle classes. EMFAC 2011 reflects VMT distribution over the vehicle classes based on DMV data and ARB truck model.					
Speed Vehicle Classes:	Apply to light-duty cars and trucks, medium-duty vehicles, and motorcycles. ARB default speeds to be used for all heavy-duty vehicles.					
Speed Distributions and Periods:	SACOG provided four speed distributions -- AM peak (period 2), PM peak (period 5), Noon (periods 3 and 4) and Night (periods 1 and 6)					
Period Definitions	**Period 1**	**Period 2**	**Period 3**	**Period 4**	**Period 5**	**Period 6**
	Hours 0-5	6-8	9-11	12-14	15-17	18-23

ARB staff reviewed SACOG's VMT and speed distributions. Table 9 shows the daily VMT provided by SACOG. ARB compared SACOG's VMT and growth rates by county to the following data sources:

- SACOG Activity Data Submitted to ARB (Feb 2011)
- EMFAC 2007, Version 2.3 (Nov 2006)
- SACOG Activity Data Submitted to ARB (2007)
- California Motor Vehicle Stock, Travel and Fuel Forecast, May 2008 (MVSTAFF)
- Human Population, State of California, Department of Finance, Population Projections for California and Its Counties 2000-2050, Sacramento, California, July 2007.

Table 7-10. SACOG Daily VMT by County

SACOG	Area	2005 (EMFAC)	2005 (SACOG07)	2005 (SACOG11)	2018 (EMFAC)	2018 (SACOG07)	2018 (SACOG11)	2035 (EMFAC)	2035 (SACOG07)	2035 (SACOG11)
El Dorado	9	3,706,000	4,157,854	4,155,441	4,305,000	5,045,473	4,948,882	5,241,000	5,819,807	5,361,995
Placer (MC)	12	988,000	1,600,847	1,474,339	1,220,000	1,853,406	1,658,850	1,464,000	2,306,092	1,664,866
Placer (SV)	30	8,947,000	7,504,939	7,505,269	9,773,000	10,427,060	10,358,839	11,699,000	13,667,576	13,194,072
Sacramento	31	32,513,000	33,376,527	33,392,044	36,614,000	41,019,449	40,824,102	42,026,000	49,397,697	48,856,257
Sutter	34	2,443,000	1,962,622	1,954,199	3,925,000	2,575,221	2,575,060	6,907,000	3,232,017	3,162,046
Yolo	36	5,733,000	5,421,497	5,372,892	6,806,000	6,593,345	6,583,507	7,907,000	7,906,712	7,785,032
Yuba	37	1,606,000	0	1,785,066	2,358,000	0	2,487,589	6,907,000	0	3,064,986
South Sutter		633,852	506,013	507,029	1,151,251	751,214	755,297	2,425,032	1,112,803	1,110,187
Total NAA		54,963,852	54,530,299	54,361,213	63,794,251	68,265,168	67,704,537	77,669,032	83,442,703	81,134,456
Total Region		55,936,000	54,024,286	55,639,250	65,001,000	67,513,953	69,436,829	82,151,000	82,329,900	83,089,255

SACOG provided VMT and speeds by vehicle class and by county. ARB used DMV population and EMFAC2011-HD truck model redistribution to update vehicle population. SACOG agreed to use ARB default speed distributions for heavy-duty vehicles. EMFAC 2011 reflects SACOG's speed distributions for light-duty cars and trucks plus motorcycles. The Sacramento Metropolitan Area VMT comparison and growth rates are shown in Figure 7-8 and Figure 7-9. SACOG 2011 has same VMT as in their previous submittal except less than 1% difference in their base year.

Figure 7-8. Sacramento Region VMT

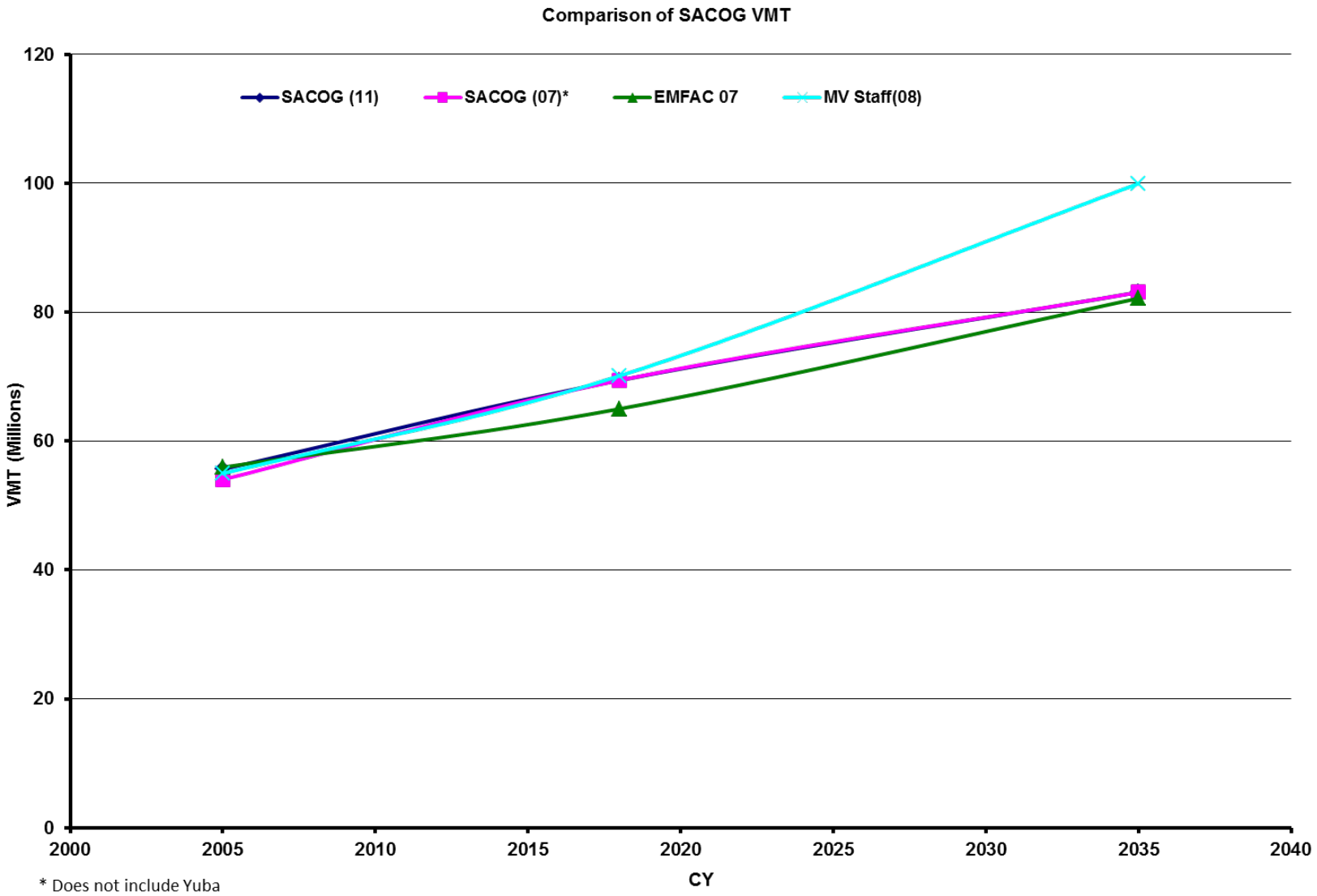
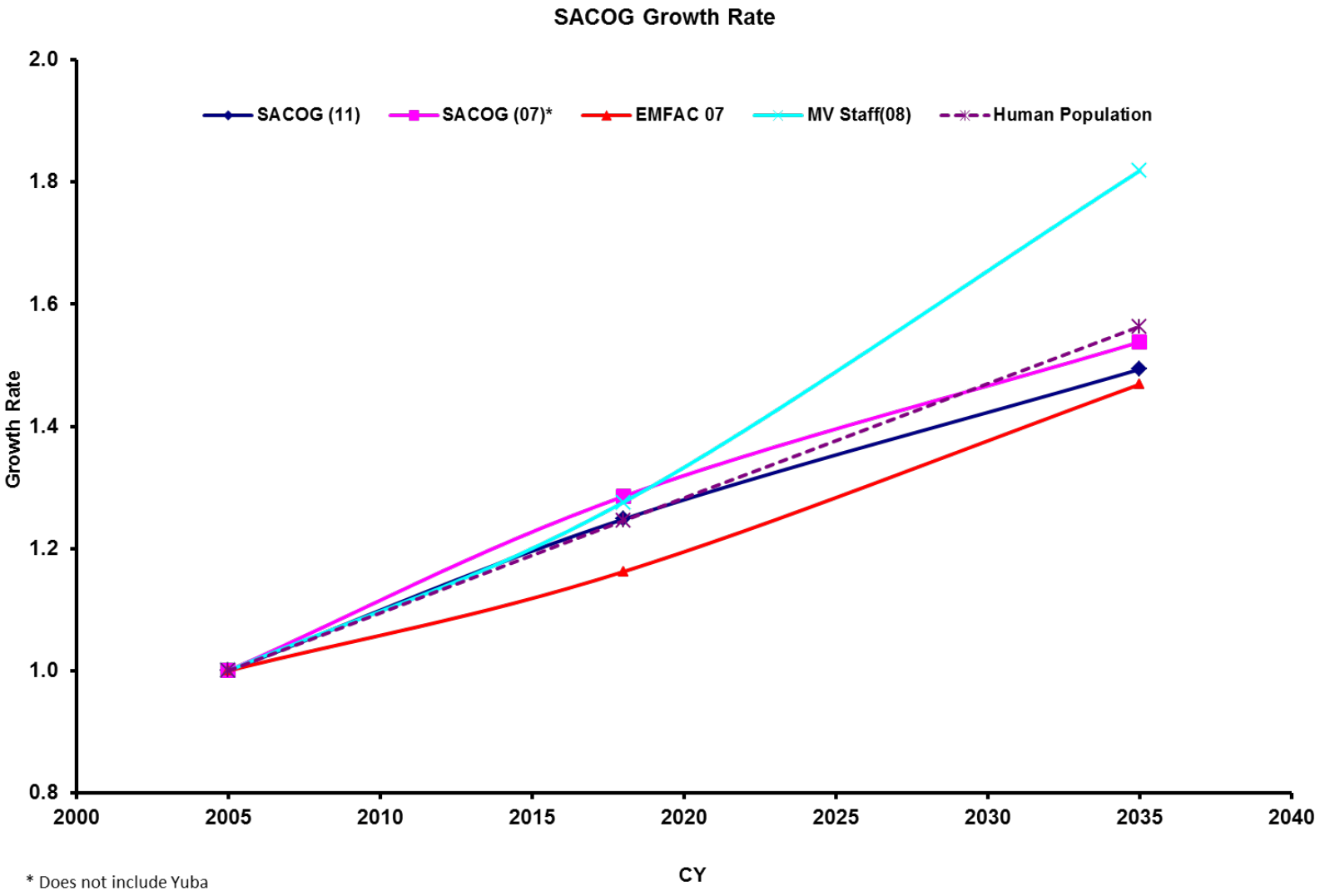


Figure 7-9. VMT and Human Population Growth in the Sacramento Region



SAN JOAQUIN VALLEY

There are eight COGs (Council of Governments) in the San Joaquin Valley (SJV) including Fresno, Kern, Kings, Madera, Merced, San Joaquin, Stanislaus, and Tulare. All the COGs in SJV region have submitted vehicle activity data in October 2011. Table 7-11 provides details on the SJV submittal.

Table 7-11. San Joaquin Valley Submittal

Traffic Model Citation:	Activity data is based 2011 TIP and RTP currently pending state and federal approval					
Date of Submittal:	October 2010					
Base Years Provided:	Fresno - 2003, Kern - 2006, Kings - 2005, Madera - 2000, Merced - 2000, San Joaquin - 2005, Stanislaus - 2006, and Tulare - 2007.					
Future Years Provided:	2011, 2012, 2014, 2017, 2020, 2023, 2025, 2035					
VMT Vehicle Classes:	VMT totals represent all vehicle classes. EMFAC 2011 reflects VMT distribution over the vehicle classes based on DMV data and ARB truck model.					
Speed Vehicle Classes:	Apply to light-duty cars and trucks, medium-duty vehicles, and motorcycles. ARB default speeds to be used for all heavy-duty vehicles.					
Speed Distributions and Periods:	Fresno, Kern, San Joaquin, Stanislaus and Tulare COGs provided speed distribution for three periods: AM peak (period 2); PM peak (period 5); and off-peak (periods 1, 3, 4, and 6). Kings, Madera and Merced COGs provided daily speed distribution.					
Period Definitions	**Period 1**	**Period 2**	**Period 3**	**Period 4**	**Period 5**	**Period 6**
	Hours 0-5	6-8	9-11	12-14	15-17	18-23
Period Definitions	Daily, same distribution for all periods.					

ARB staff reviewed the activity data received from SJV COGs and compared VMT trends for CYs 2000 through 2035 to other data sources:

- SJV Activity Data Submitted to ARB in October 2010
- EMFAC 2007, Version 2.3 (Nov 2006)
- SJV Activity Data Submitted to ARB in 2009
- California Motor Vehicle Stock, Travel and Fuel Forecast, May 2008 (MVSTAFF)
- Human Population, State of California, Department of Finance, Population Projections for California and Its Counties 2000-2050, Sacramento, California, July 2007.

SJV COGs provided total VMT and one speed distribution for all vehicle classes. The heavy heavy-duty truck VMT was updated to agree with ARB's EMFAC2011-HD truck model redistribution. Similarly, SJV COGs agreed to use ARB default speed distributions for heavy-duty vehicles. EMFAC2011 reflects SJV COGs speed distributions for light-duty cars and trucks plus motorcycles.

The SJV COGs total VMT comparison and growth rates (excluding the portion of Kern County located in the Mojave Desert Air Basin) are shown in Figure 7-10 and Figure 7-11. Figure 7-12 and Figure 7-13 show the VMT and growth rate comparisons for Kern County in the Mojave Desert Air Basin.

Figure 7-10. VMT in the San Joaquin Valley (Excluding Portions of Kern County Located in the Mojave Desert Air Basin)

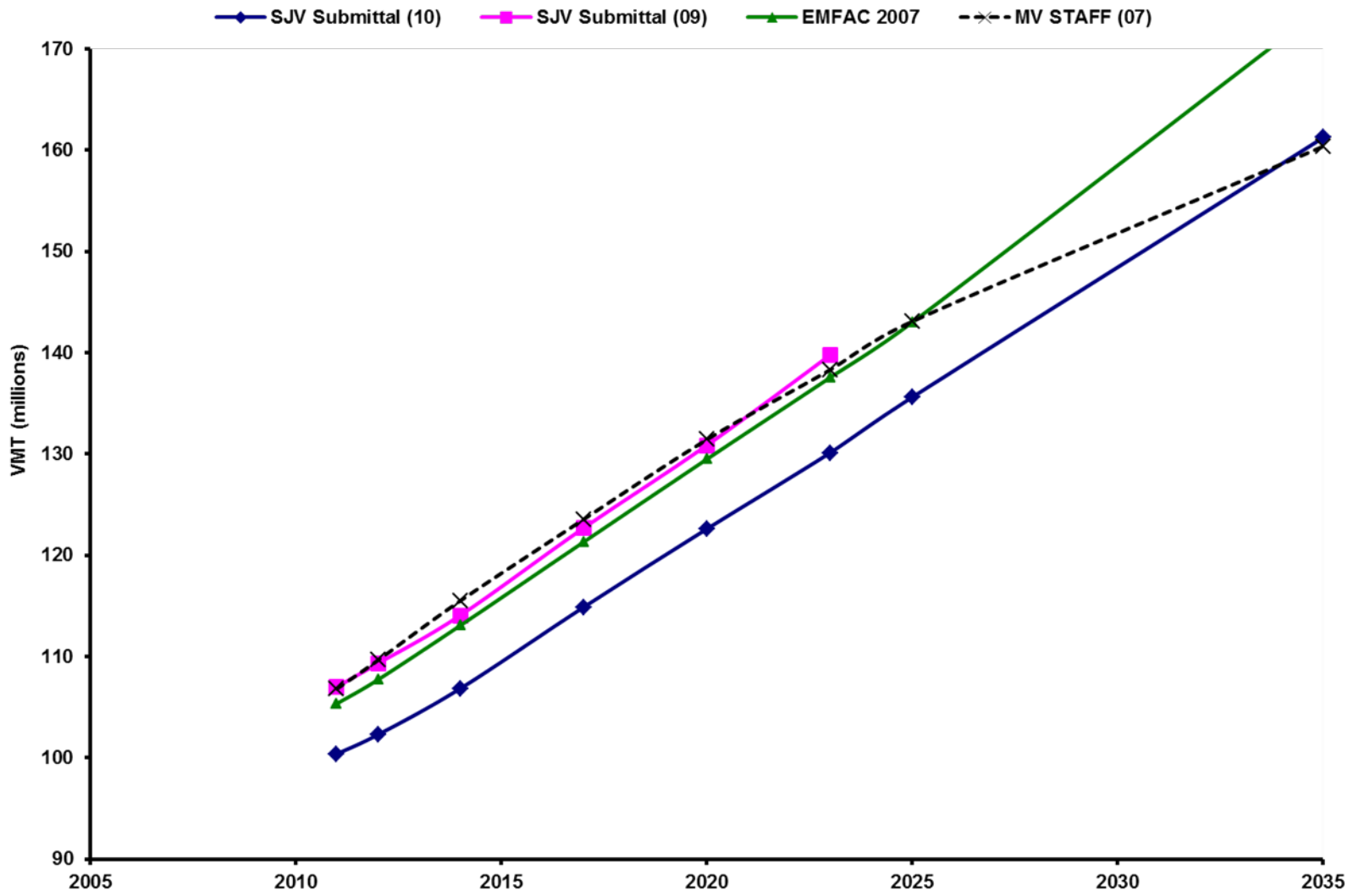


Figure 7-11. VMT and Human Population Growth in the San Joaquin Valley

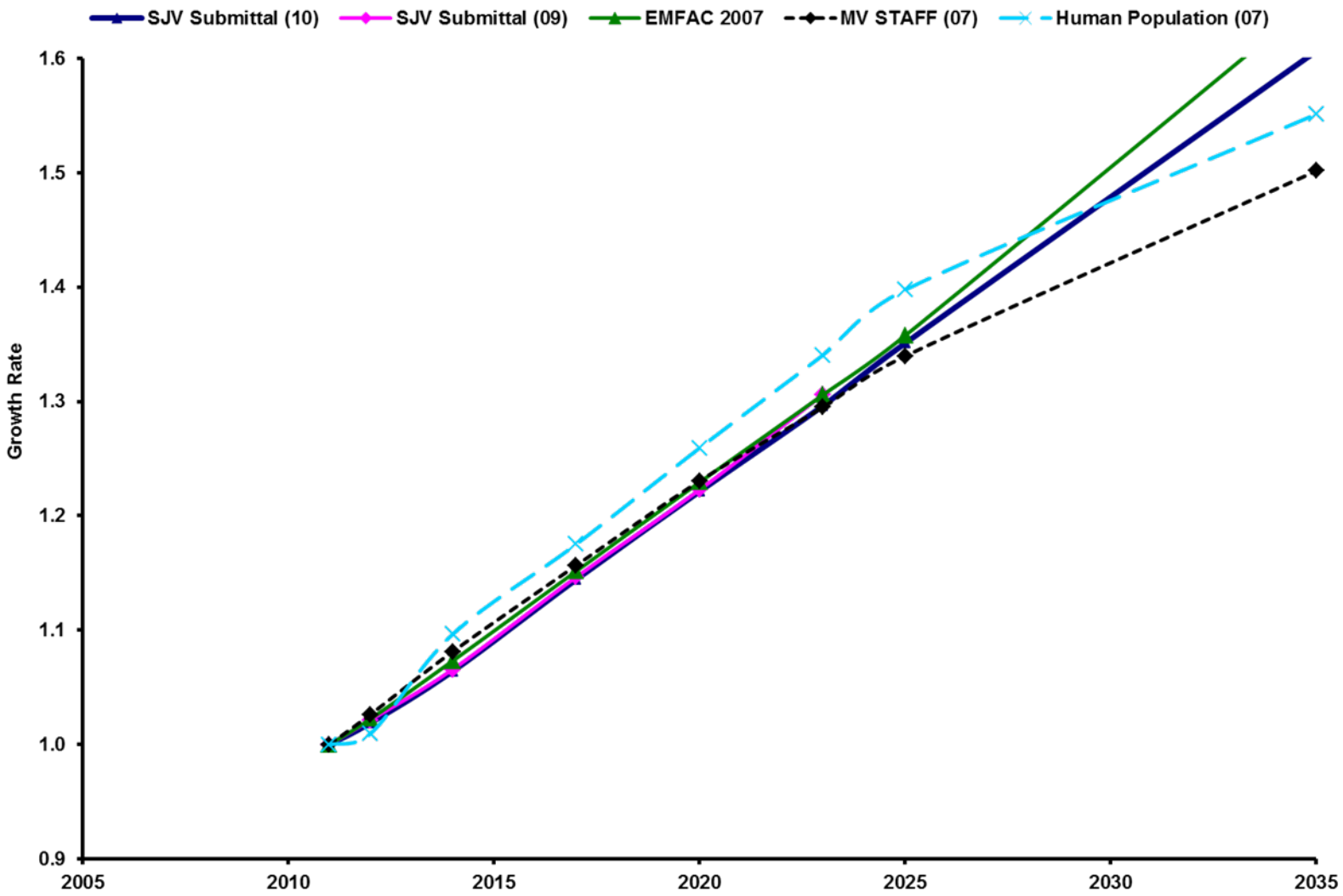


Figure 7-12. VMT in Kern County, Mojave Desert Air Basin

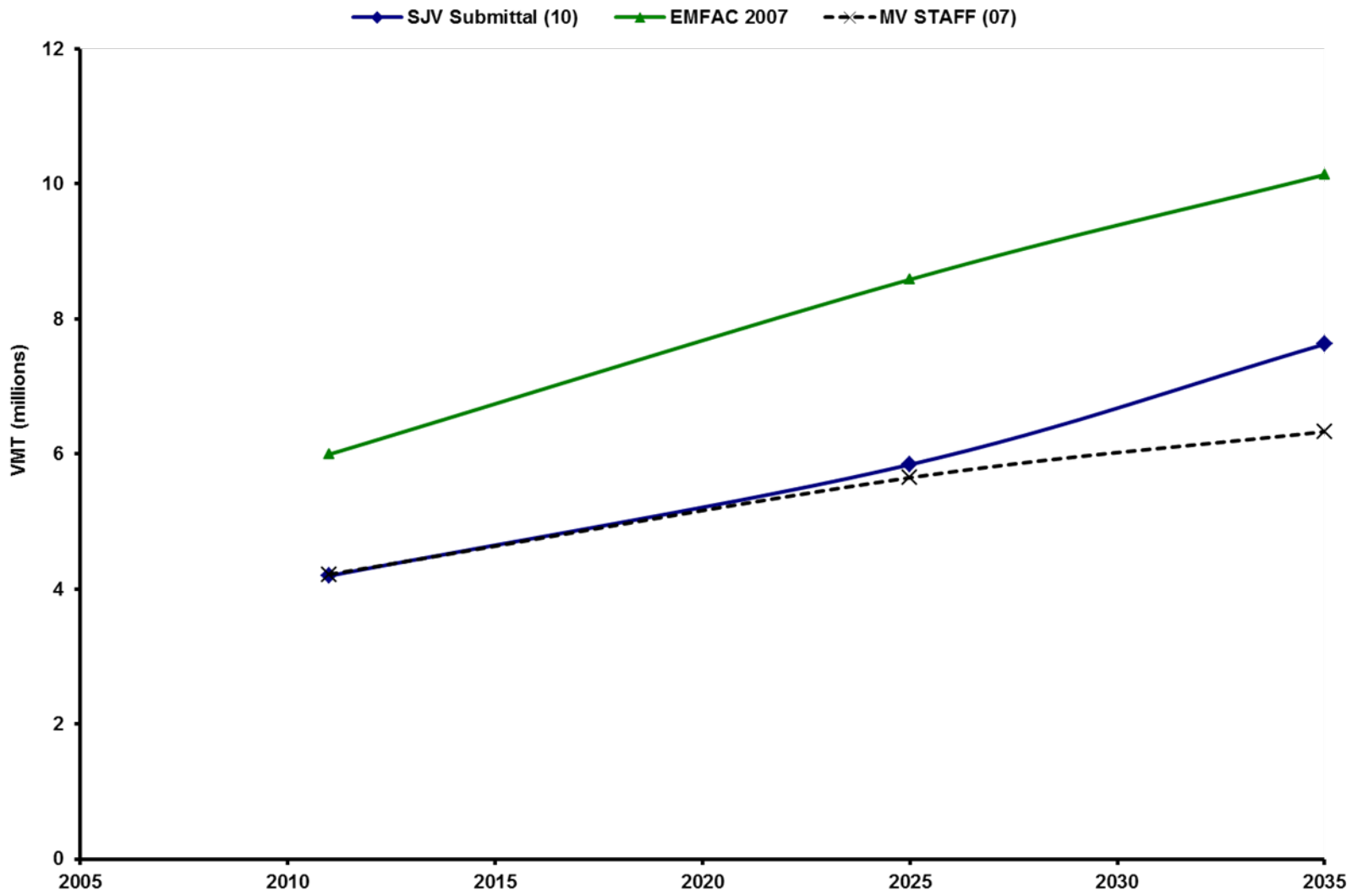
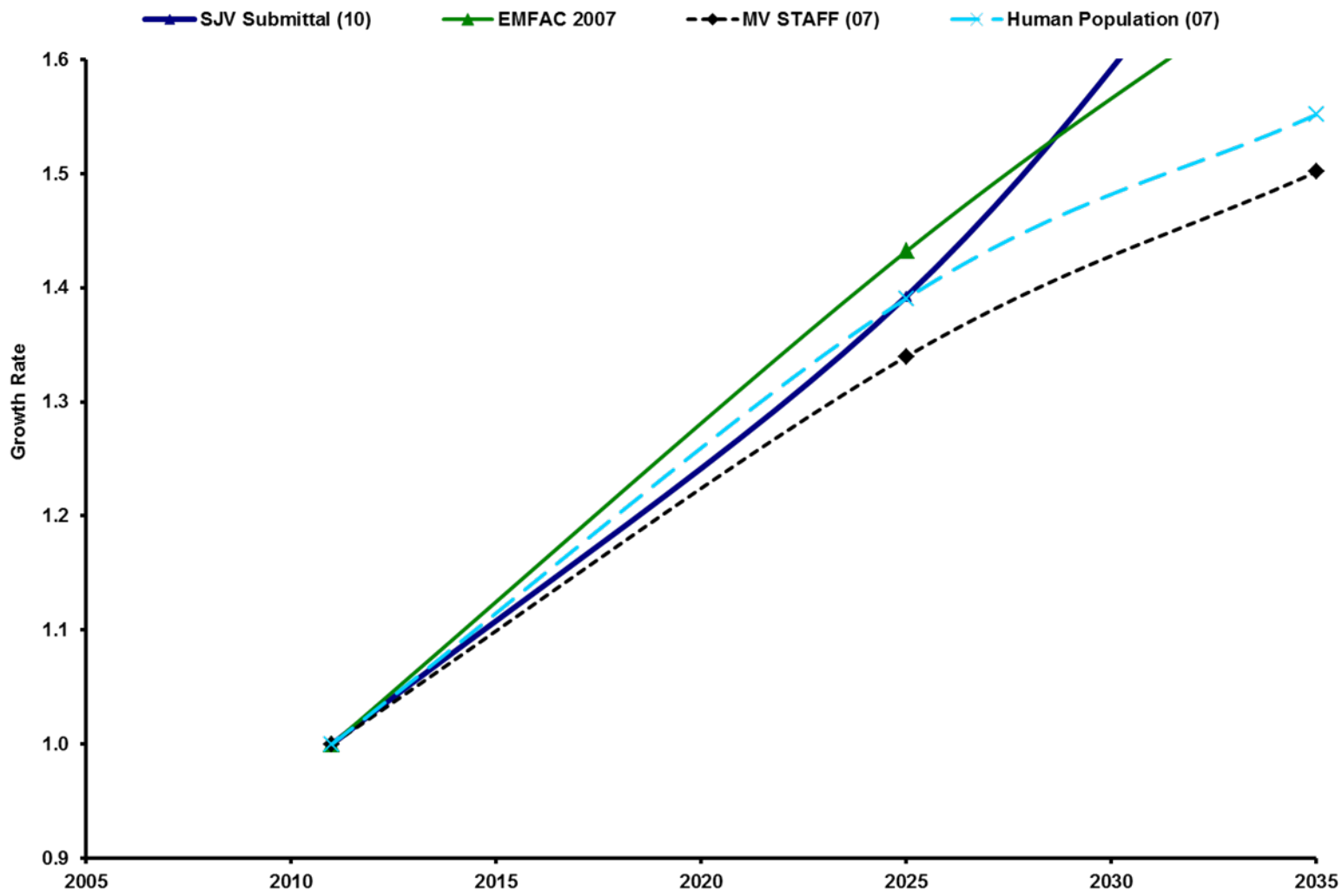


Figure 7-13. VMT and Human Population in Kern County, Mojave Desert Air Basin



8 APPENDIX: CARBON DIOXIDE EMISSION RATE UPDATES

The official greenhouse gas emissions inventory representing the transportation economic sector is developed using top-down methods based upon statewide fuel sales data and published at **http://www.arb.ca.gov/cc/inventory/inventory.htm**.

EMFAC2011 outputs carbon dioxide emissions and fuel consumption through the EMFAC2011-LDV and EMFAC2011-HD modules using a bottom-up emissions inventory calculation based on population, activity, and emission rates. The two approaches provide marginally different results. The top-down method is considered a more accurate assessment of fuel use and carbon dioxide emissions because fuel use is measured through tax receipts by the California State Board of Equalization. The bottom-up method used by EMFAC provides information the top-down method cannot provide, such as a breakdown of estimated fuel use and emissions by vehicle class, for different engine technologies, different model years of vehicles and other factors.

Since EMFAC2000 the model has output carbon dioxide emissions and estimated fuel consumption based on carbon balance. The estimates of carbon dioxide (CO_2) emissions in the EMFAC model come from direct chassis dynamometer exhaust tests. This section analyzes the effect of introduction of oxygenated fuels, and updates the previous (2000) analysis with recent dynamometer test data collected on the Unified Cycle. The methods of estimating fuel consumption from CO_2 emissions and carbon balance are explored as a function of oxygenate content. The forecasted CO_2 emissions estimates provided in EMFAC2011 assume implementation of the Pavley (AB1432) greenhouse gas emission limits; however these benefits are calculated in EMFAC2011-SG, and not in EMFAC2011-LDV; as a result the changes discussed here only reflect changes based on driving cycle and oxygenate content.

A summary of the emissions effects of these changes for various areas in the State is shown in Section 12. Table 8-1 shows the effect on criteria pollutants and CO2 for the year 2007. The proposed changes lower the CO2 emissions by about 2% in 2007, mostly due to slightly different results from the vehicle surveillance programs.

Table 8-1. Summary of Emissions Changes due to Revised Greenhouse Gas Emission Factors, Calendar Year 2007

Air Basin	CO2 Emission Changes (tons per day)
Statewide	-6,446
South Coast	-2,092
San Joaquin Valley	-596
Sacramento Valley	-476
San Diego	-502
San Francisco Bay Area	-1,673

We determine carbon dioxide emissions from vehicles by measuring the exhaust concentration of carbon dioxide while running a specified driving cycle with the car situated on a dynamometer. In this case we evaluated two driving cycles: the Federal Test Procedure (FTP) and Unified Cycle (UC). The FTP is used for emission certification of new vehicles, and is modeled after suburban driving around Los Angeles in 1977. The FTP is not considered to be representative of actual driving conditions as it reflects 30-year old traffic conditions, has no or little freeway driving, and measured acceleration rates were reduced in the development of the cycle to accommodate older technology dynamometers. In the EMFAC model, base emissions are estimated using the UC driving this cycle because it is intended to be representative of average driving. It was developed from California-specific chase-car studies completed in the early 90's. It reflects driving on freeways; therefore, has higher speeds and acceleration rates than the FTP cycle.

The CO_2 emissions data in the EMFAC2007 model date from EMFAC2000, and are based on testing of cars during the 1990s. Table 8-2 shows the sources of the FTP data analyzed for EMFAC2000 and for this update.

Table 8-3 shows the sources of UC data. The projects with "S" in the name are surveillances. The projects with "R" in the name are research projects. The middle digits (positions 3 and 4 from the left) are the year that the project was begun. The three surveillances listed in the last three lines of the table are new (since the EMFAC2000 work), and are the basis for this model revision.

Table 8-2. CO_2 FTP Emissions Projects

Project	Number of Passenger Cars	Number of Light Duty Trucks (Class-1)^	Number of Light Duty Trucks (Class-2)**	Number of Medium Duty Trucks^^
FTP Phase 1 Gasoline				
2S93C1	65		13*	
2R9312	74			
FTP Phase 2 Gasoline				
2S95C1	321		62*	16
2S97C1	217		64*	22
2R9312	39		6*	1
2R9513	89		6*	
2R9811	7		1*	
2S00C1	226	21	40	13
2S03C1	212	29	50	34
2S06C1	41		11	16

^ < 6000 pounds Gross Vehicle Weight Rating, < 3450 pounds Curb Weight
** < 6000 pounds Gross Vehicle Weight Rating, > 3450 pounds Curb Weight
^^ 6000 - 8500 pounds Gross Vehicle Weight Rating

Table 8-3. CO2 UC Emissions Projects

Project	Number of Passenger Cars	Number of Light Duty Trucks (Class-1)^	Number of Light Duty Trucks (Class-2)*	Number of Medium Duty Trucks^^
2R9312	39	6*		1
2R9513	89	6*		
2R9811	7	1*		
2S00C1	226	21	40	13
2S03C1	212	29	50	34
2S06C1	41		11	16

^ < 6000 pounds Gross Vehicle Weight Rating, < 3450 pounds Curb Weight
** < 6000 pounds Gross Vehicle Weight Rating, > 3450 pounds Curb Weight
^^ 6000 - 8500 pounds Gross Vehicle Weight Rating
* In these projects light duty trucks were not separated by class I and 2. They are combined.

The term “Phase 2” in Table 2 refers to Phase 2 Reformulated Gasoline. In 1995 vehicles being tested at the ARB Haagen-Smit Laboratory in El Monte started to be performed with commercial Phase 2 reformulated gasoline (RFG), which contained 10% MtBE (methyl t-butyl ether) at the time. We segregated the 1990’s data into Phase 1 and Phase 2 RFG. We combined the recent surveillance data (from 2000 to 2007). We compiled the results for the three bags (test phases) of the FTP, as well as new UC results. The results of the data compilation for passenger cars are shown in Table 8-4 through Table 8-7 for the FTP and Table 8-8 through Table 8-11 for the UC. The new composite results are remarkably close to the EMFAC 2000 results. Segregating the light trucks into the separate LT1 and LT2 categories shows that the LT1s and PCs have similar CO_2 emission rates.

In EMFAC2007, fuel usage estimates are determined by carbon balance, which means that the emissions results for carbon containing species (carbon dioxide, carbon monoxide, and total hydrocarbons or volatile organic compounds) are used to determine the amount of fuel combusted. For EMFAC2011 the carbon balance methodology has been improved by accounting for oxygenated reformulated gasolines.

The carbon balance equation (for gasoline) as described in the EMFAC2000 Technical Support Document is as follows:

$$\text{gal/d} = 375 \text{ gal/ton C} * (0.273*CO_2 \text{ tpd} + 0.429*\text{CO tpd} + 0.866*\text{HC tpd})$$

The quantity in parentheses is the mass of carbon in the emission gases. The coefficients of the CO_2, CO and HC terms (0.273, 0.429, 0,866) are the weight fractions of carbon in those quantities. The first factor in the equation (375 gal/ton C) is the only one that would be affected by the presence of oxygen in gasoline. It is calculated from:

$$\text{gal/ton C} = 2000 \text{ lb/ton} / \text{wt frac C} / \rho_{gaso} \text{ lb/gal}$$

where ρ_{gaso} is the density of liquid gasoline, lb/gal

Properties of gasoline as well as MtBE and Ethanol gasoline blends are provided in Table 8-12.

Table 8-4. Passenger Car FTP CO_2 Results

	FTP Bag 1			FTP Bag 2			FTP Bag 3		
	CO_2			CO_2			CO_2		
MY and Technology	Number of Observations	mean	Standard Deviation	Number of Observations	mean	Standard Deviation	Number of Observations	mean	Standard Deviation
		g/mi	g/mi		g/mi	g/mi		g/mi	g/mi
75-80 Carb	69	565	176	74	556	163	73	494	148
81-85 Carb	73	399	89	73	407	104	73	351	48
86-92 Carb	49	344	74	49	343	74	49	302	58
81-85 FI	29	413	63	29	410	53	29	353	46
86-92 FI	283	393	66	283	401	70	282	349	52
93-99 FI	315	368	68	315	369	74	314	315	57
00+ FI	96	380	61	96	371	60	96	318	50
81-85 TBI	30	473	115	30	481	116	30	408	99
86-92 TBI	58	394	82	58	397	97	59	341	73

Carbureted (Carb); FI (Fuel-Injected); TBI (Throttle-Body Single-Point Fuel Injection)

Table 8-5. Light Duty Truck Class I FTP CO2 Results

	FTP Bag 1			FTP Bag 2			FTP Bag 3		
	CO_2			CO_2			CO_2		
MY and Technology	**Number of Observations**	**mean**	**Standard Deviation**	**Number of Observations**	**mean**	**Standard Deviation**	**Number of Observations**	**mean**	**Standard Deviation**
		g/mi	**g/mi**		**g/mi**	**g/mi**		**g/mi**	**g/mi**
75-80 Carb				1	514		1	456	
81-85 Carb	2	438	56	2	446	36	2	377	58
86-92 Carb	4	456	47	4	434	68	4	395	37
81-85 FI									
86-92 FI	7	432	56	7	425	49	7	372	44
93-99 FI	28	400	51	29	385	49	28	345	38
00+ FI	6	452	58	6	420	71	6	384	50
81-85 TBI									
86-92 TBI	2	414	4	2	397	10	2	352	33

Carbureted (Carb); FI (Fuel-Injected); TBI (Throttle-Body Single-Point Fuel Injection)

Table 8-6. Light Duty Truck Class 2 FTP CO2 Results

	FTP Bag 1			FTP Bag 2			FTP Bag 3		
	CO_2			CO_2			CO_2		
MY and Technology	**Number of Observations**	**mean**	**Standard Deviation**	**Number of Observations**	**mean**	**Standard Deviation**	**Number of Observations**	**mean**	**Standard Deviation**
		g/mi	**g/mi**		**g/mi**	**g/mi**		**g/mi**	**g/mi**
75-80 Carb									
81-85 Carb	2	596	84	2	576	81	2	533	65
86-92 Carb									
81-85 FI									
86-92 FI	14	553	69	14	532	67	14	474	54
93-99 FI	57	494	41	56	482	42	56	426	34
00+ FI	25	515	72	25	483	74	26	433	63
81-85 TBI									
86-92 TBI	5	518	38	5	507	34	5	461	44

Carbureted (Carb); FI (Fuel-Injected); TBI (Throttle-Body Single-Point Fuel Injection)

Table 8-7. Medium-Duty Vehicle FTP CO2 Results

	FTP Bag 1			FTP Bag 2			FTP Bag 3		
	CO_2			CO_2			CO_2		
MY and Technology	**Number of Observations**	**mean**	**Standard Deviation**	**Number of Observations**	**mean**	**Standard Deviation**	**Number of Observations**	**mean**	**Standard Deviation**
		g/mi	**g/mi**		**g/mi**	**g/mi**		**g/mi**	**g/mi**
75-80 Carb	15	747	108	15	738	115	15	679	112
81-85 Carb	7	707	69	7	666	48	7	604	47
86-92 Carb									
81-85 FI									
86-92 FI	2	630	73				2	597	123
93-99 FI	29	585	87	31	579	86	29	510	76
00+ FI	21	641	112	21	634	126	21	563	102
81-85 TBI									
86-92 TBI	10	745	200	10	703	184	10	849	715

Carbureted (Carb); FI (Fuel-Injected); TBI (Throttle-Body Single-Point Fuel Injection)

Table 8-8. Passenger Vehicle Unified Cycle CO2 Results

	UC Bag 1			UC Bag 2		
		CO_2		CO_2		
MY Fuel Delivery	Number of Observations	mean	Standard Deviation	Number of Observations	mean	Standard Deviation
		g/mi	g/mi		g/mi	g/mi
75-80 Carb	9	878	315	10	535	141
81-85 Carb	24	651	188	24	390	90
86-92 Carb	19	485	109	19	315	54
81-85 FI	9	656	95	10	397	52
86-92 FI	153	610	114	86	368	58
93-99 FI	247	591	124	248	343	61
00+ FI	96	640	120	96	353	59
81-85 TBI	4	672	88			
86-92 TBI	31	612	182	31	366	89

Carbureted (Carb); FI (Fuel-Injected); TBI (Throttle-Body Single-Point Fuel Injection)

Table 8-9. Light Duty Truck Class I Unified Cycle CO2 Results

	UC Bag 1			UC Bag 2		
		CO_2		CO_2		
MY Fuel Delivery	Number of Observations	mean	Standard Deviation	Number of Observations	mean	Standard Deviation
		g/mi	g/mi		g/mi	g/mi
75-80 Carb	1	703		1	516	
81-85 Carb	2	697	135	2	411	54
86-92 Carb	4	675	114	4	425	38
81-85 FI						
86-92 FI	7	641	94	7	407	48
93-99 FI	28	619	93	28	379	39
00+ FI	6	720	120	6	411	42
81-85 TBI						
86-92 TBI	2	598	35	2	391	11

Carbureted (Carb); FI (Fuel-Injected); TBI (Throttle-Body Single-Point Fuel Injection)

Table 8-10. Light Duty Truck Class 2 Unified Cycle CO2 Results

	UC Bag 1			UC Bag 2		
		CO_2		CO_2		
MY Fuel Delivery	Number of Observations	mean	Standard Deviation	Number of Observations	mean	Standard Deviation
		g/mi	g/mi		g/mi	g/mi
75-80 Carb						
81-85 Carb	2	925	175	2	595	94
86-92 Carb						
81-85 FI						
86-92 FI	14	828	100	16	524	57
93-99 FI	53	777	75	55	474	39
00+ FI	25	824	122	25	480	72
81-85 TBI						
86-92 TBI	5	802	60	5	509	77

Carbureted (Carb); FI (Fuel-Injected); TBI (Throttle-Body Single-Point Fuel Injection)

Table 8-11. Medium Duty Truck Unified Cycle CO2 Results

	UC Bag 1			UC Bag 2		
		CO_2		CO_2		
MY Fuel Delivery	Number of Observations	mean	Standard Deviation	Number of Observations	mean	Standard Deviation
		g/mi	g/mi		g/mi	g/mi
75-80 Carb	2	1168	149	1	783	
81-85 Carb	3	1112	205	3	698	61
86-92 Carb						
81-85 FI						
86-92 FI	2	1042	300	2	643	119
93-99 FI	26	970	171	25	550	81
00+ FI	21	1044	177	21	616	103
81-85 TBI						
86-92 TBI	1	611		1	406	

Carbureted (Carb); FI (Fuel-Injected); TBI (Throttle-Body Single-Point Fuel Injection)

Table 8-12. Properties of Oxygenated and Non-Oxygenated Gasolines

	Weight Percent Carbon	Weight Percent Hydrogen	Weight Percent Oxygen	Density (lbs/gallon)	Gallons per Ton Carbon
Non-oxy gasoline	85-88	12-15	0-0.2	6.0-6.5	350-392
Indolene, 9 psi	85.4	13.4	0.2	6.2	378
M10 (10M-90I)	83.6	13.4	2.0	6.2	386
E6 (5.2E 94.8I)	83.6	13.4	2.0	6.22	385
E10 (10E 90I)	82.1	13.4	3.7	6.24	390
E85 (85E 15I)	57.0	13.1	29.8	6.54	537

(M10 is 10% MtBE and 90% Gasoline; E10 is 10% Ethanol, 90% Gasoline)

Prior to 1994, gasoline sold in California was non-oxygenated, with the exception of gasoline sold in the South Coast Air Quality Management District. Beginning in 1996, California required reformulated gasoline, which was formulated using 10% MtBE. In 2004, California banned the use of MtBE as a fuel oxygenate, requiring instead that ethanol be used at 6% by weight (E6). Beginning in 2010, petroleum marketers began selling a 10% ethanol gasoline blend (E10). Gasoline exceeding 10% ethanol can be sold in California but may only be used in flex-fuel vehicles, which is currently a very small proportion of the vehicle fleet in California.

The impact of this change on the emissions inventory is relatively minor, and caused by both the updated base emission rates using updated information and the UC, and the impact of the inclusion of oxygenated fuels. Table 8-13 compares running CO2 emission rates by model year between EMFAC2007 and EMFAC2011. Passenger car emission rates are about 5% lower than in EMFAC2007 than in EMFAC2011, and Light Duty Trucks (Class I) are 6% lower in EMFAC2011 than EMFAC2007. Light Duty Trucks (Class 2) and Medium Duty Trucks are about 6% higher in EMFAC2011 than assumed in EMFAC2007. The effect on CO2 emissions statewide is shown in Table 8-14. Overall the emission rate update had a minor effect on emissions which differs by calendar year and is about a 2% reduction in estimated emissions into the future.

The calculation of CO2 emissions is dependent on fuel carbon content. Table 8-15 compares estimated fuel use using the EMFAC2011 (accounting for fuel oxygenate content) and EMFAC2007 (no accounting for fuel oxygenates) approaches. Results show estimated fuel use is 3%-4% higher in EMFAC2011 than in EMFAC2007.

Table 8-13. CO2 Running Emission Rates: EMFAC2011 vs EMFAC2007.

	Running CO2							
	LDA		LT1		LT2		MT3	
Model Year	EMFAC 2010	EMFAC 2007	EMFAC 2010	EMFAC 2007	EMFAC 2010	EMFAC 2007	EMFAC 2010	EMFAC 2007
	g/mi	g/mi	g/mi	g/mi	g/mi	g/mi	g/mi	g/mi
1976	570	585	554	550	558	571	773	838
1977	569	584	554	550	559	572	774	839
1978	569	583	554	550	559	572	769	838
1979	566	584	554	549	559	572	769	839
1980	540	583	545	550	550	563	749	840
1981	489	430	548	436	553	637	761	748
1982	503	430	542	436	547	638	757	747
1983	498	430	490	441	495	644	769	745
1984	488	432	487	441	492	645	774	756
1985	422	433	479	443	484	648	754	758
1986	415	409	471	439	476	571	723	698
1987	415	409	467	439	473	572	685	699
1988	417	409	465	440	470	573	708	701
1989	440	409	467	442	472	575	712	704
1990	458	411	463	443	468	576	692	705
1991	458	411	456	443	462	577	697	706
1992	458	411	455	444	460	578	685	707
1993	438	385	473	414	479	524	665	606
1994	414	385	493	415	498	526	659	606
1995	405	386	500	416	506	527	660	607
1996	405	385	498	417	504	528	663	606
1997	405	385	499	418	505	529	702	606
1998	405	382	499	418	505	529	697	608
1999	404	379	498	418	504	529	690	610
2000	404	387	498	453	504	536	685	684
2001	403	387	498	453	504	536	683	685
2002	403	387	497	453	504	536	682	685
2003	403	387	497	453	503	535	682	685
2004	403	390	497	452	503	535	682	684
2005	402	394	496	452	503	534	681	683
2006	400	394	494	449	502	534	681	683
2007	399	394	493	448	502	534	681	682
2008	399	393	493	448	502	533	680	682

Table 8-14. CO2 Emissions by Calendar Year Resulting from CO2 Emission Rate Updates for Selected Calendar Years

	EMFAC 2007	EMFAC 2010	Difference	Percent Increase
Calendar Year	CO2 (Tons per Day)			
1990	409,201	411,454	2,253	0.6%
2000	463,492	453,755	-9,737	-2.1%
2005	575,802	562,711	-13,091	-2.3%
2007	552,745	540,824	-11,921	-2.2%
2010	574,626	563,701	-10,925	-1.9%
2014	620,393	610,742	-9,651	-1.6%
2017	654,218	645,355	-8,863	-1.4%
2020	683,822	675,460	-8,361	-1.2%
2023	714,979	706,729	-8,250	-1.2%

Table 8-15. Comparison of CO2 Emissions Calculated With and Without Accounting for Fuel Oxygenate Content

	1000 Gallons Gasoline per Day		
Calendar Year	Fuel Use Calculated Accounting for Oxygenated Fuel (EMFAC2011 Approach)	Fuel Use Calculated Without Accounting for Oxygenated Fuel (EMFAC2007 Approach)	Ratio EMFAC2011 Approach to EMFAC2007 Approach
1990	39,198	39,198	1.00
2000	41,781	40,695	1.03
2005	48,507	47,247	1.03
2007	47,230	46,003	1.03
2010	49,793	47,878	1.04
2014	53,533	51,474	1.04
2017	56,196	54,035	1.04
2020	58,564	56,312	1.04
2023	61,122	58,771	1.04

9 APPENDIX: BRAKE WEAR PARTICULATE MATTER EMISSIONS UPDATE

EMFAC2011 estimates the directly emitted emissions of total particulate matter (PM) for exhaust, tire wear, and brake wear. EMFAC2007 used an emission factor of 12.8 milligrams per mile (12.8 mg/mi PM) to estimate the amount of airborne dust attributable to brake wear. This emission factor, based on twenty-year-old tests of asbestos friction-materials from automobile disc brakes, was applied to all vehicles. Two studies were performed on modern asbestos-replacement friction materials over the past 10 years. Staff correlated results from these studies with wheel load, braking speed and material. The results were applied to the braking events of the Unified Cycle (UC), and the Transient and Cruise cycles for medium heavy-duty and heavy heavy-duty trucks, taking into account that heavy heavy-duty trucks have eight rear and two front brakes compared to four brakes on cars. This information was used to update brake wear emissions estimates in EMFAC2011.

Updated per-mile braking emission rates are, in general, about 2.5 times greater in EMFAC2011 than in EMFAC2007. This is increase is caused by modern brake pad materials that are more friable but less toxic than older asbestos based technologies. Staff analysis also found that the speed at which braking occurs, the weight of the vehicle, and, most importantly, the fraction of stop-and-go driving affects the per-mile airborne brake wear rate. A summary of the increases for various areas in the State is shown in Table 9-1 below. Implementation of the proposed modification results in an increase in the PM inventory of about 30 tons per day statewide in 2007. This is a more than tripling of the brakewear emissions and a 50% increase in total PM emissions. The increase in PM2.5 is less than for PM due to the relatively small fraction of brake wear PM in the PM2.5 size fraction.

Table 9-1. PM10 Emissions Increase Due to Brake Wear Method Improvement

Air Basin	PM10 Emissions Increase (Tons per Day)	
	2007	2023
Statewide	29.95	37.34
South Coast	11.65	13.55
San Joaquin Valley	3.32	4.69
Sacramento Valley	2.25	3.09
San Diego	2.77	3.17
San Francisco Bay Area	5.5	6.68

EMFAC2007 brake wear emission factors were, carried over from earlier versions of the U.S. EPA Mobile model. The U.S. EPA in turn derived the 13 mg/vehicle-mile estimate from a 1983 paper by Cha et al.[2] (U.S. EPA and Northrop). That work collected particles generated from operating a disc brake dynamometer with asbestos friction

[2] Cha, S, P. Carter, and R. Bradow. 1983. Simulation of Automobile Brake Wear Dynamics and Estimation of Emissions. SAE Paper 831036. Society of Automotive Engineers, Warrendale, PA.

materials on a schedule of braking events typical of Raleigh City driving. The original result of 12.8 mg/mile was calculated from the following equation:

(2 x 2.43 mg/application front disc + 2 x 1.68 mg/application rear) x 31% airborne x 5.1 applications/mi.

Cha et al estimated 0.75 mg PM / application airborne (out of a total brake pad weight loss of 2.43 mg/application) for one disc brake. So, when calculating brake wear emissions there are a number of variables of interest including (a) the number of brakes per vehicle; (b) dust emissions per brake application; (c) the fraction of dust that becomes airborne in various size fractions; and (d) the number of brake applications per mile. The amount of dust generated per brake application is a function of the type of friction material, the type of brake, the deceleration rate, the vehicle weight, and the brake pad size. In many studies, the experimental method used provides only the airborne dust per application (the product of the gross dust emissions per application and the fraction of dust airborne), so the variables of dust emission per application and fraction of dust airborne are in some cases not reported separately.

Two new studies over the past 10 years have characterized particulate matter emission from braking events using modern materials: Garg et al[34] from General Motors and Sanders et al[56] from Ford. Garg gave the breakdown of the sales of friction materials in 1998, shown below in Table 9-2. The trucks in this table are pickups and full-size sport utility vehicles. All vehicles were equipped with disc brakes unless otherwise noted. 1998 was the last year for asbestos-containing materials in OEM brake pads.

Table 9-2. Sales of Brake Pad Materials in 1998 (Garg et al., 2000a)

		Semi-Metallic	Low-Metallic	Non-Asbestos Organic	Asbestos Organic
Front Brakes	**Automobile**	70%	15%	15%	
	Truck	75%	25%		
Rear Brakes	**Automobile**	50%	12%	16%	22% (drum)
	Truck	75% (drum)	15%	10%	

[3] Garg, B., S. Cadle, P. Groblicki, P. Mulawa, C. Laroo, and G. Parr. 2000a. Brake-wear Particulate Matter Emissions. Report R&D 9033. General Motors Research and Development Center

[4] Garg, B., S. Cadle, P. Mulawa, P. Groblicki, C. Laroo, and G. Parr. 2000b. Brake Wear Particulate Emissions. *Env Sci & Tech* 34:4463-4469.

[5] Sanders, P., T. Dalka, N. Xu, M. Maricq, and R. Basch. 2002. Brake Dynamometer Measurement of Airborne Brake Wear Debris. SAE Paper 2002-01-1280. Society of Automotive Engineers, Warrendale, PA.

[6] Sanders, P., N. Xu, T. Dalka, and M. Maricq. 2003. Airborne Brake Wear Debris: Size Distributions, Composition, and a Comparison of Dynamometer and Vehicle Tests. *Env. Sci. & Tech.* 37:4060-4069.

In the Garg and Sanders studies, researchers tested brake pads with different friction materials on a shrouded brake dynamometer, sampled dusts generated by each braking event, and measured the breakdown of emissions by particle size. Results are shown in Table 9-3.

Table 9-3. Airborne Dust Emissions per Brake Application

Material	Source	Average Brake Speed (mph)	Dust (mg/stop)	Wheel Load (pounds)	Wind Speed (mph)	Brake Drag (pounds)
Low-Metallic	Sanders	15	6	1070	8	120
		15	4	1070	8	120
		31	21.5	1070	8	856
		15	9	1070	7	120
		30	24.5	1070	7	196
		30	21	1070	40	196
Semi-Metallic	Sanders	15	1.5	1447	8	160
		31	20	1447	8	1160
		15	1.5	1447	7	160
	Garg	16	3.9	1984	2-13	586
		16	1.7	1616	2-13	469
Non Asbestos Organic	Sanders	15	0.9	1070	8	120
		31	11.5	1070	8	856
		15	1.4	1076	7	120
	Garg	16	4.6	1376	2-13	407
		16	1.9	1671	2-13	500
		16	3	1577	2-13	469
		16	2.1	592	2-13	167
		16	6.5	1434	2-13	430

Results shown in Table 9-3 are corrected to a wheel-installed basis and 50% airborne sampling efficiency. Other characteristics of the tests varied. For example, while both studies measured emissions from full stops, Garg et al. used repeated stops from 32 miles per hour at a 6.5 miles/hour-sec deceleration rate. In contrast, Sanders et al., used several braking programs including a series of 24 stops from various speeds (22-55 mph) at variable deceleration rates (1.3-3.4 mph/second); a series of 10 stops from 62 miles per hour at 17.7 mph/second; and a series of stops from 60 miles per hour at 4 mph/second. Sanders et al found a 50% airborne fraction using a wind tunnel at 40 miles per hour. While this is higher than the 31% previously assumed, the Sanders study is more representative of real-world driving and as a result the 50% factor is used in this analysis.

The frequency and intensity of braking events is depending on real-world driving conditions. EMFAC2007 and previous versions of EMFAC assumed a PM emissions factor that was based on a statistical analysis of Raleigh city driving (Cha, 1983) which assumed 5.1 brake applications per mile, 69% on average of a full stop with an initial braking even velocity of 22 mph. The fraction of a full stop was defined as the initial velocity squared minus final velocity squared all divided by initial velocity squared - this is the ratio of the actual kinetic energy change to the absolute kinetic energy.

In this analysis we updated our assumptions about the frequency and intensity of braking events. We used the Unified Cycle (LA 92) for passenger cars and SUVs, which is the same driving cycle upon which emission factors are based for all pollutants in EMFAC. For medium heavy-duty trucks, we used the ARB MHD cycle developed for the CRC E55/59 testing. The cruise, low-speed transient, and high-speed transient cycles were weighted according to their activity. For heavy heavy-duty trucks, the ARB HHD cycles were used, and weighted according to their activity. Heavy-duty truck activity is dominated by high speed cruising (interstate travel). Thus the braking cycle for them has few applications per mile and the events are shallow (low-energy). For urban buses we used the OCTA (Orange County Transit Authority) cycle. This was developed from actual tracking of buses in Orange County, and has more 40 mi/h cruising than the New York City Cycle or the SAE Central Business District cycle. The severity, frequency and average speed of braking for each vehicle type is shown in Table 9-4, and is compared to the assumptions in EMFAC2007 based on Cha et al.

Table 9-4. Assumed Braking Attributes by Vehicle Type

Vehicle Type	Braking Cycle	Braking Frequency (Applications/mile)	Initial Velocity (mph)	Fraction Full Stop
Passenger Cars and SUVs	UC	4.0	27.1	61%
Medium-Heavy Duty Trucks	ARB-MHD	3.0	29.1	49%
Heavy Heavy-Duty Trucks	ARB-HD	1.2	31.5	8%
Urban Transit Buses	OCTA	8.2	21.2	73%
School Buses	MHD-LS Transient	14	12.7	79%
All (EMFAC2007) from Cha et al. (1983)	Raleigh City	5.1	22.1	69%

To develop a model of brake wear emissions, staff combined data by material and evaluated the results. Figure 9-1 and Figure 9-2 show modeled results for non-asbestos organic materials and semi-metallic materials respectively. The slope of the emissions vs wheel load was taken in both cases from the 15 and 16 mph results. The same slope was assumed for the 31 mph results. These slopes were used to scale the automotive data to truck applications. Representative loads are shown in Table 9-5.

Figure 9-1. Modeled Brake Dust Emissions: Non-Asbestos Organic Materials

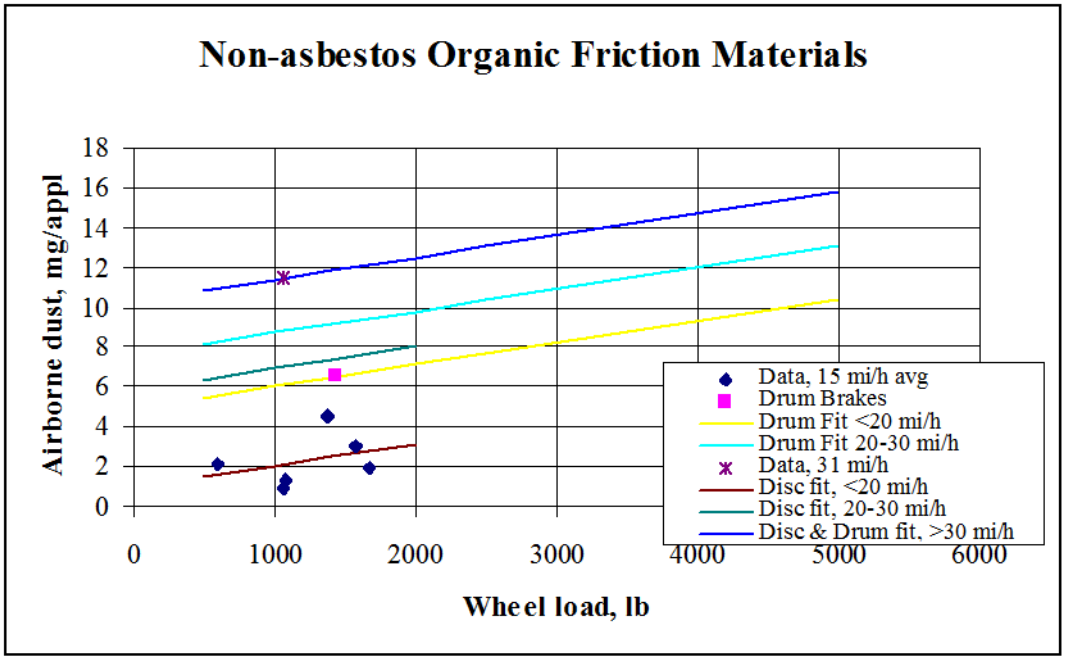


Figure 9-2. Modeled Brake Dust Emissions: Semi-Metallic Materials

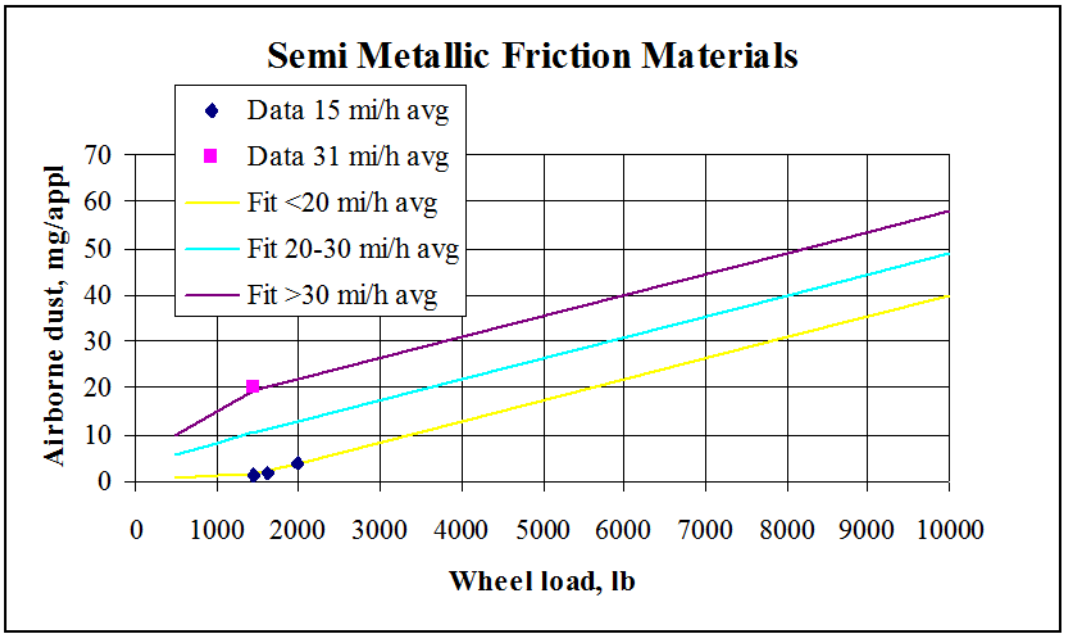


Table 9-5. Assumed Wheel Braking Loads

	Assumed Gross Vehicle Weight (pounds)	Front (pounds/wheel)	Rear (pounds/wheel)	Rear Trailer (pounds/wheel)
Car	3100	1100	450	
SUV	6000	2100	900	
Medium Heavy Truck	20000	7000	3000	
Heavy Heavy-Duty Truck	52000	9000	4600	3900
Urban Bus	42000	14700	6300	

The figures show a correlation between average brake event speed (average of beginning and ending speed in the braking event) and emissions. In general, emissions are higher as a function of wheel load (brake inertia weight). The newer asbestos-free braking materials were found to have much higher emissions than asbestos friction materials. The (Cha,1983) result was about 0.75 mg airborne dust per application for an automotive front brake.

Brakes with the wheel loads in Table 9-5 were modeled as being operated on braking cycles derived from typical driving based on the test cycles described in Table 9-4. Emission factors were estimated by friction material and scaled by wheel load by average braking speed (average of initial and final speeds). Braking event emissions for partial stops were scaled by the kinetic energy change (difference of final speed squared and initial speed squared).

Results of this calculation for several vehicle types are shown in Table 9-6 and Table 9-7. The predicted value for passenger cars is about 34 mg/mi, about two and a half times the value of Cha et al (1983). This is largely due to the fact that the new asbestos-free friction materials give off more dust during braking than did the old asbestos-containing materials.

Table 9-6. Calculated Brake Wear Emission Rates.

	EMFAC2011			
Vehicle Category	**Passenger Car**	**Light Duty Truck 1**	**Light Duty Truck 2**	**Medium Duty Vehicle**
Assumed GVWR (pounds)	3100	3700	5000	7000
Brake Description	4 discs	4 discs	4 discs	4 discs
Friction Material Front	semi-metallic	semi-metallic	semi-metallic	semi-metallic
Friction Material Rear	semi-metallic / non asbestos organic	semi-metallic	semi-metallic	semi-metallic
Emissions per Application (Front)	4.9	5.2	6.2	10
Emissions per Application (Rear)	3.6	3.1	3.5	4.4
Fraction Airborne	50%	50%	50%	50%
Applications per Mile	4	4	4	4
Initial Velocity (mph)	27.1	27.1	27.1	27.1
Fraction Full Stop	61%	59%	60%	60%
Total Airborne Dust (mg/mile)	34	33	38.6	57.4

	EMFAC2011			
Vehicle Category	**Light Heavy Truck 1**	**Light Heavy Truck 2**	**Medium Heavy Truck**	**Heavy Heavy Truck**
Assumed GVWR (pounds)	9500	12500	20000	52000
Brake Description	2 front discs and 2 rear drums	2 front discs and 2 rear drums	2 front discs and 2 rear drums	2 front discs, 4 tractor drums, 4 trailer drums
Friction Material Front	semi-metallic	semi-metallic	semi-metallic	semi-metallic
Friction Material Rear	non-asbestos organic	non-asbestos organic	non-asbestos organic	non-asbestos organic
Friction Material Trailer				non-asbestos organic
Emissions per Application (Front)	17.2	19.6	32.4	21.6
Emissions per Application (Rear)	7.4	8.4	11	6.2
Emissions per Application (Trailer)				5.8
Fraction Airborne	50%	50%	50%	50%
Applications per Mile	3.2	3.3	3	1.2
Initial Velocity (mph)	29.1	29.1	29.1	31.5
Fraction Full Stop	49%	49%	49%	8%
Total Airborne Dust (mg/mile)	78.3	91.2	132.9	62.9

	EMFAC2011			EMFAC2007
Vehicle Category	**Urban Bus**	**Motorcycle**	**School Bus**	**All Vehicles**
Assumed GVWR (pounds)	42000	650	20000	4300
Brake Description	2 front discs and 2 rear drums	2 discs	2 front discs and 2 rear drums	2 discs and 2 drums
Friction Material Front	semi-metallic	semi-metallic	semi-metallic	Asbestos-Organic
Friction Material Rear	non-asbestos organic	semi-metallic / non asbestos organic	non-asbestos organic	
Emissions per Application (Front)	88.8	2.9	40	2.4
Emissions per Application (Rear)	17.2	3.2	12	1.7
Fraction Airborne	50%	50%	50%	31%
Applications per Mile	8.2	4	14	5.1
Initial Velocity (mph)	21.2	27.8	12.7	22.1
Fraction Full Stop	73%	60%	79%	69%
Total Airborne Dust (mg/mile)	859.4	12.4	760	12.8

Table 9-7 Calculated Brake Wear Emission Rates by Technology Group.

Technology Group	Description	Present	Proposed
		g/mi	g/mi
1-37	PCs, LDTs, MDVs Gas	0.0128	0.0375
40-43	PCs (Mexican)	0.0128	0.0375
46-57	M4 Truck Gas	0.0128	0.078
60-71	M4 Truck Diesel	0.0128	0.078
76-87	M5 Truck Gas	0.0128	0.091
90-101	M5 Truck Diesel	0.0128	0.091
106-114	HDL&M Truck Gas	0.0128	0.133
120-131	HDL&M Truck MH OB Dsl	0.0128	0.133
136-144	HDH Truck Gas	0.0288	0.063
150-161	HDH Truck Diesel	0.0288	0.063
170-177	PCs Diesel	0.0128	0.034
178-185	LDTs Diesel	0.0128	0.036
186-194	MDVs Diesel	0.0128	0.057
200-211	HDH Truck Diesel Federal	0.0288	0.063
216-225	Urban Buses Diesel	0.0128	0.859
228-237	School Buses Gas	0.0128	0.76
240-251	School Buses Diesel	0.0128	0.76
260-277	Motorcycles	0.0064	0.012

10 APPENDIX: GASOLINE PM EMISSION FACTOR UPDATES

UPDATE

The current exhaust emissions standard for PM from automobiles is 10 mg/mile. As shown in Table 9-6 above, passenger vehicles emit between 30 and 60 mg PM / mile from brake wear alone. Passenger vehicles manufactured since 1996 have met and exceeded the current exhaust emissions standard, and today 85 – 90% of particulate matter emitted from passenger vehicles is dust from tire and break wear emissions.

Gasoline PM emission factor updates in EMFAC2011 for 2004 and newer port fuel injected (PFI) and gasoline direct injected (GDI) engines were developed using tests results from 40 vehicles on the Federal Test Procedure (FTP) driving cycle, and 10 vehicles on the Unified Cycle (UC). FTP results appeared to show a statistically significant difference between newer technology PFI engines and newer technology GDI engines. This difference is described in this section and integrated into EMFAC2011.

In August 2011 staff received new testing data from U.S. EPA on 15 PFI and 2 GDI vehicles. These vehicles were tested by USEPA on the FTP and the US06 which is an aggressive driving cycle designed to identify off FTP cycle emissions. These data cast the assumptions developed for EMFAC2011 into question. Results from analysis of the USEPA data suggested that under FTP driving conditions emission rates for most PFI vehicles are very clean, and potentially cleaner than GDI vehicles. However, some newer PFI vehicles may burn oil through engine deterioration, and that could lead to higher emissions that could meet or exceed PM emission rates from current GDI technologies. At the same time testing on the US06 driving cycle was suggestive, although not conclusive, that PFI engines may have less precise engine control under aggressive conditions than GDI vehicles, leading to potential off-cycle PM emissions in excess of those expected under the FTP.

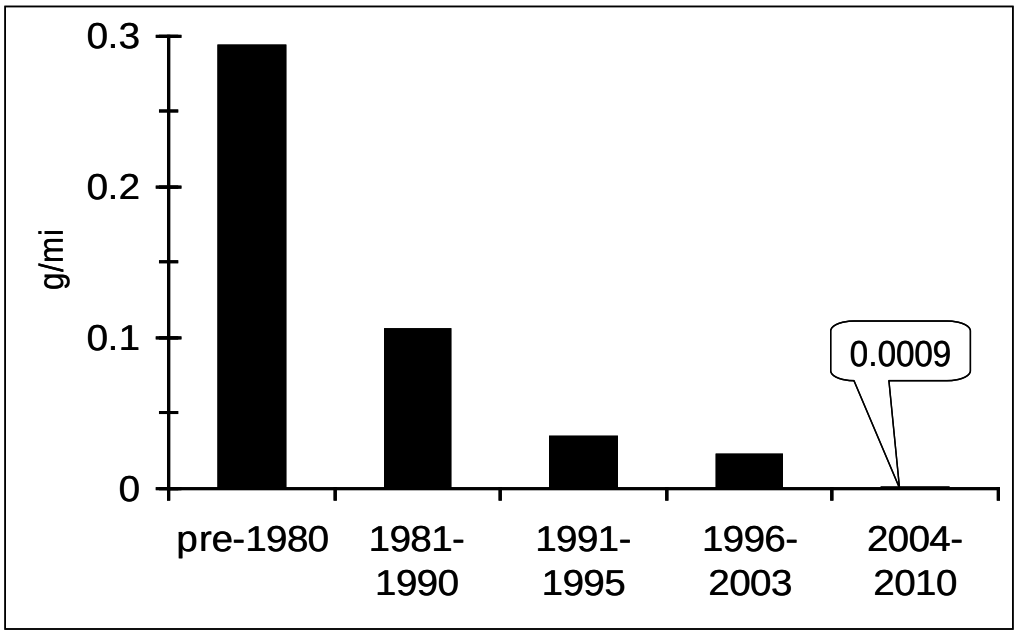
An overall analysis of the available data at this time suggests that while it is clear both GDI and PFI technologies emit at levels far below the current emissions standard of 10 mg/mile, it is not clear that 2004 and newer GDI technologies are cleaner than 2004 and newer GDI technologies.

The net impact on the inventory of this uncertainty is minimal, because the vast majority of fine particulate matter emitted by passenger vehicles is generated by tire and break wear. Statewide, gasoline vehicle particulate matter emissions from exhaust, tire wear, and brake wear represent around 3% of all PM2.5 emitted in California. Of that, only about 10-15% of that 3%, or 0.5% of the total is emitted from passenger vehicle exhaust.

These assumptions are continuing to be refined and will be discussed in greater detail through the Advanced Clean Car Rulemaking currently scheduled for December, 2011.

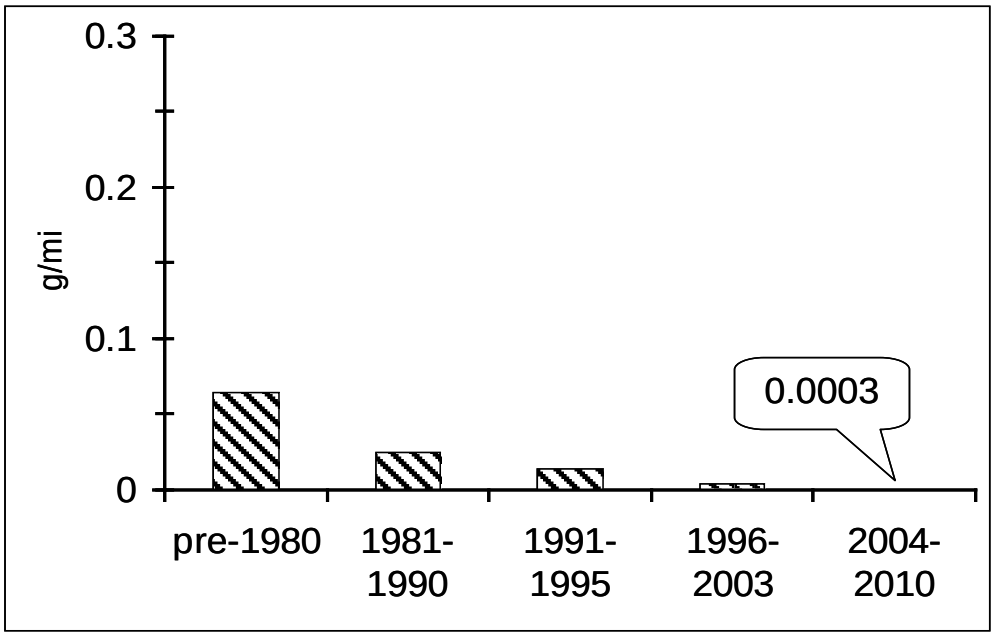
Basic emission rates for light duty gasoline vehicles have been updated using data from the Kansas City PM Characterization Study[7] (E-69). Those vehicles were certified to Federal Standards. Emissions data were collected under conditions prescribed in the Federal Test Procedures, and vehicles were operated over the LA92 Unified Driving Cycle. Figure 10-1 shows Kansas City Study $PM_{2.5}$ emission factors for cold start (Bag 1), and Figure 10-2 shows $PM_{2.5}$ hot running exhaust (Bag 2) by model year group through 2003. Measurements for newer vehicles (2004-2010) are from tests of in-use PFI vehicles over the same cycles and conditions but conducted at ARB's Haagen Smit Laboratory.

Figure 10-1. PM2.5 Emission Factor: Cold Start Exhaust (Bag 1)



[7] Kansas City PM Characterization Study, EPA420-R-08-009, April 2008

Figure 10-2. PM2.5 Emission Factor: Hot Stabilized Exhaust (Bag2)



We expect the percentage of gasoline direct injection (GDI) engines to increase from current levels in order to meet the GHG emission targets. We assumed that current wall-guided GDI engines will penetrate the fleet first, followed by cleaner center-walled technology in the future. The anticipated fleet penetration of GDI technology and its impact on $PM_{2.5}$ emission for both Bag 1 and Bag 2 are shown in Figure 10-3 and Figure 10-4, based on ARB testing of wall-guided technology GDI vehicles.

Figure 10-3. GDI Fleet Penetration and Cold Start Exhaust (grams per mile)

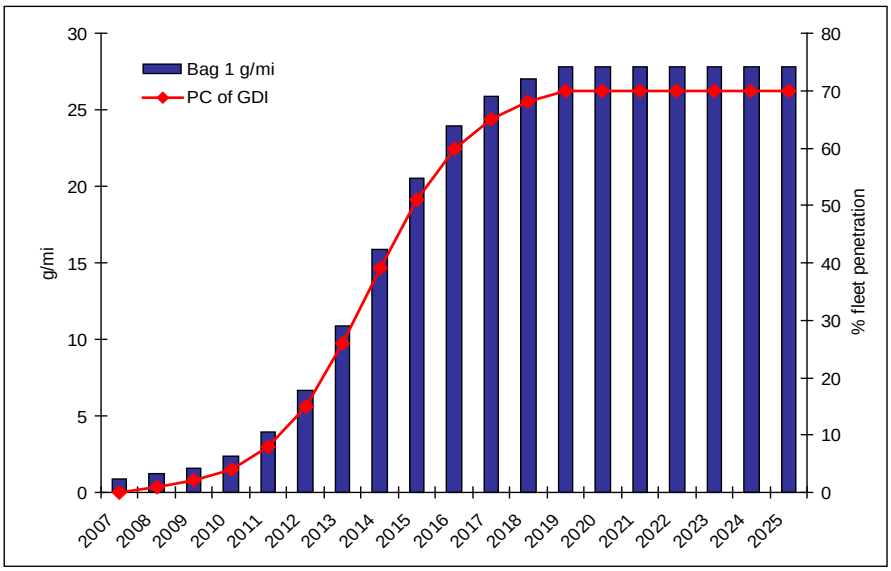
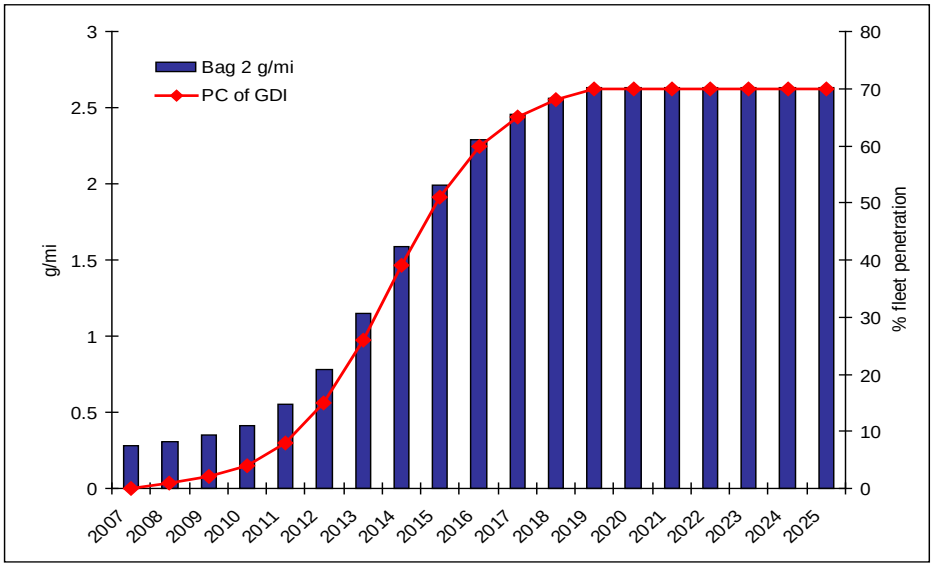


Figure 10-4. GDI Fleet Penetration and Hot Stabilized Exhaust (grams per mile)



The current LEV II standard for PM is 10 mg/mi and will be in effect until 2016. As these standards are further reduced we expect technological improvements in GDI engines will result in substantial PM reductions over the present GDI. Our test results of one center-guided GDI engine showed about ⅓ of the PM emissions from wall-guided GDI engines. We assume that in the future these low PM emitting center-guided GDI engines will be the dominant technology. Figure 10-5 shows how technology is expected to change and the expected emissions.

Figure 10-5. Forecasted Light Duty Vehicle Technology Mix: 2007-2025.

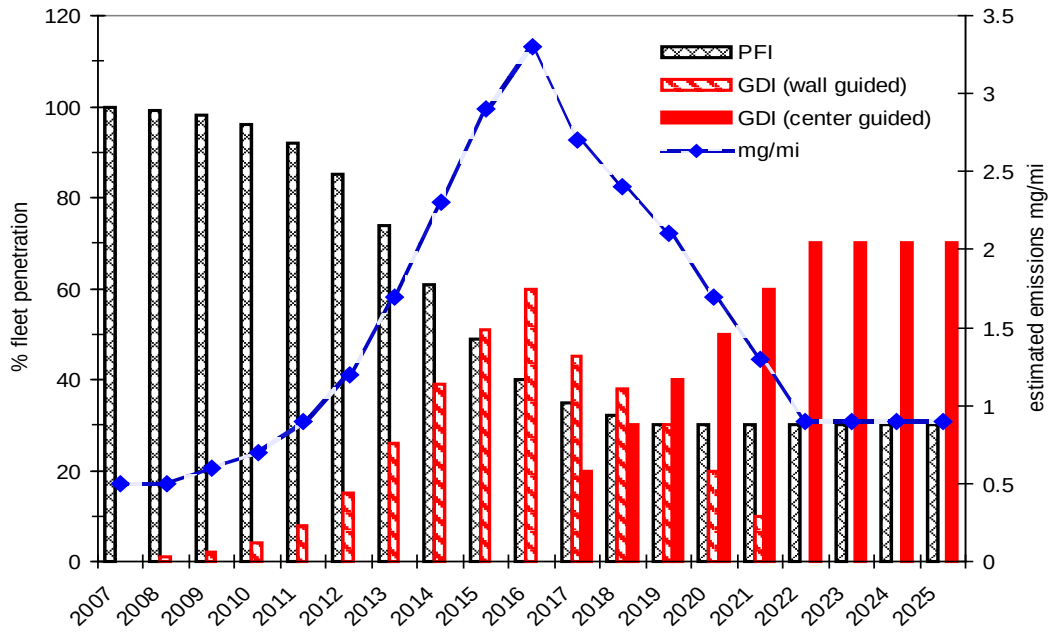


Table 10-1 below shows the reductions in emissions of PM from light duty gasoline vehicles with improved technology. We estimate there will be an approximately two ton per day reduction in PM emissions from light duty gasoline vehicles in 2040 caused by the penetration of GDI technology.

Table 10-1. Statewide Light Duty Gasoline PM Emissions (Tons per Day)

Year	Baseline (tons/day)	With Improved Technology (tons/day)
2014	3.6	3.6
2017	3.3	3.3
2018	3.3	3.2
2020	3.3	3.0
2022	3.4	2.8
2025	3.6	2.6
2035	4.2	2.0
2040	4.5	1.9

11 APPENDIX: HEAVY DUTY TRUCK EMISSION RATE UPDATES

The updates to heavy duty truck emission rates described in this section reflect updates to emission rates that were first integrated into the Statewide Truck and Bus Rule emissions inventory as external adjustments to emission rates developed using EMFAC2007. Those external adjustments in the rulemaking inventory have now been integrated into the EMFAC2011-HD calculation methodology and made consistent with EMFAC modeling methods on a regional basis.

There are four major updates that are covered in this document. The first is an update to medium-heavy duty diesel truck (MHDDT) running emission rates based on new test data. The second is improved assumptions about how manufacturers are complying with 2004, 2007, and 2010 emissions standards, and how that affects MHDDT and heavy heavy-duty diesel truck (HHDDT) emission rates. The third is carbon dioxide emission rate improvements using assumptions developed for the ARB's Statewide Truck and Bus Rule. Finally, idling emission rates are updated for both MHDDT and HHDDT.

In EMFAC2007 staff updated emission factors for heavy-heavy duty trucks based on emissions test data from the Coordinating Research Council (CRC) E55/59 project. The E55/59 project included testing on both heavy-heavy (HHDDT) and medium-heavy duty diesel trucks (MHDDT). After the release of EMFAC2007 the medium-heavy duty truck test data became available. Those data are used to update emission factors for medium-heavy duty trucks in EMFAC2011.

To revise the MHDDT emission factors, staff combined the E55/59 data into the data set that was used to develop MHDDT emission factors in EMFAC2002. The EMFAC2002 data set included emission test data from the New York State Department of Environmental Conservation and Energy, West Virginia University, and the Colorado Institute for Fuels, and High Altitude Engine Research. From the combined data set, staff then calculated zero-mile rates (ZMR) and deterioration rates (DR) of pollutants HC, CO, NOx, and PM.

The method of calculating MHDDT ZMR and DR is the same as that used for deriving emission factors of heavy heavy-duty trucks (HHDDT) in EMFAC2007. The method has been described in detail in an EMFAC2007 technical memo (ARB, 2006). Basically, the merged data set was divided into a number of model year groups by considering the general emission trends as well as changes in heavy-duty diesel engine emission standard and emission control technology. For each model year group, an average emission rate was calculated from the test data or projected based future emission standards, and a tampering and mal-maintenance (T&M) impact rate was also calculated. From the average emission rates and T&M impact rates, the ZMRs and DRs for HC, CO, NOx, and PM were then calculated for all model year groups using the Radian model. The revised MHDDT emission factors are given in Table 11-1. Note that in the table the NOx emission factors for 2006-2011 model years reflect the adjustments based on the latest engine certification data, and this will be discussed in the next section.

Table 11-1. Revised MHDDT ZMR (g/mi) and DR (g/mi/10,000mi) for MHDDT

Model Year Group	HC		CO		NOx		PM	
	ZMR	DR	ZMR	DR	ZMR	DR	ZMR	DR
Pre 1987	0.83	0.047	2.79	0.159	15.6	0.033	0.97	0.038
1987-90	0.65	0.056	2.19	0.189	15.4	0.044	1.05	0.034
1991-93	0.29	0.025	1.12	0.095	11.5	0.053	0.57	0.026
1994-97	0.21	0.028	0.83	0.109	11.3	0.068	0.31	0.017
1998-02	0.22	0.028	0.84	0.108	11.1	0.078	0.35	0.015
2003-06	0.14	0.013	0.37	0.033	7.35	0.077	0.22	0.008
2007	0.12	0.008	0.31	0.020	4.78	0.065	0.022	0.001
2008	0.12	0.008	0.31	0.020	4.39	0.064	0.022	0.001
2009	0.12	0.008	0.31	0.020	3.78	0.062	0.022	0.001
2010	0.10	0.002	0.26	0.005	1.01	0.054	0.022	0.001
2011	0.10	0.002	0.26	0.005	0.86	0.054	0.022	0.001
2012	0.10	0.002	0.26	0.005	0.67	0.041	0.022	0.001
2013+	0.10	0.002	0.26	0.005	0.67	0.041	0.022	0.001

Staff updated the 2006-2011 model year HHDDT and MHDDT emission factors in EMFAC2011 to reflect how engine manufacturers complied with the 2007 engine standards and how they comply with the 2010 engine standards between 2007 and 2011. A review of the certification data shows that some engine manufacturers introduced 1.2 g/bhp-hr NOx heavy-duty engines one year earlier than required and one manufacturer chooses to meet USEPA 2010 NOx standard without using selective catalytic reduction. For the earlier introduction of engines meeting 1.2 g/bhp-hr NOx and 0.01 g/bhp-hr PM standards, the current ZMR and DR for 2003-06 and 2007-09 model year groups were weighted by the sales fractions of 2007 standard compliant engines in 2006 model year.

For the introduction of 0.5 g/bhp-hr NOx engines in 2009 model year, staff estimated the ZMR by adjusting the current ZMR for 2006-2009 model year group (1.2 g/bhp-hr NOx engines) but assumed that the DR for 2006-09 model year would still apply to the 0.5 g/bhp-hr NOx engines. The assumption of unchanged DR is largely based on the fact that the engine manufacturers will achieve a 0.5 g/bhp-hr NOx level with an integrated technology solution based on their 2006-09 technologies.

Staff estimated the 2005-2008 model year sales fractions of heavy and medium heavy-duty diesel engines based on the sales fraction data reported by the manufacturers and projected the 2009-2012 model year sales fractions from the available information. The penetration rates for 2005 to 2012 model years are summarized in Table 11-2.

Table 11-2. Penetration Rates of 2005-2012 Model Year Engines

Model Year	Certified NOx (g/bhp-hr)				Certified PM (g/bhp-hr)	
	2.2	1.2	0.5	0.25/0.2*	0.1	0.01
HHDDT						
2005	100%					
2006	99%	1%			98%	2%
2007	14%	86%			1%	99%
2008	7%	93%				100%
2009		90%	10%			100%
2010			10%	90%		100%
2011			10%	95%		100%
2012				100%		100%
MHDDT						
2005	100%					
2006	100%				98%	2%
2007	23%	77%				100%
2008	12%	88%				100%
2009		90%	10%			100%
2010			10%	90%		100%
2011			10%	90%		100%
2012				100%		100%

* 0.25 g/bhp-hour applies to 2010 model year only.

Using the sales fractions in Table 11-2, staff calculated the NOx ZMR and DR of both HHDDT and MHDDT for 2006-2011 model years by weighting the ZMR and DR of the corresponding model year groups. Staff did not revise the PM emission factors for HHDDT and MHDDT. As seen in Table 11-2, the sales of PM filter-equipped engines in 2006 account for only about 2% for both HHDDT and MHDDT categories, and therefore the impact of DPF engines on the PM emission rates for the 2006 model year should be negligible. Table 11-3 shows the current and adjusted NOx ZMR and DR for 2006-2011 model year HHDDT and MHDDT.

Table 11-3. Before- and After-Adjustment NOx ZMR (g/mi) and DR (g/mi/10,000 mi) for 2006-2011 MY Heavy-Duty Diesel Trucks

Model Year	HHDDT				MHDDT			
	Before		After		Before		After*	
	ZMR	DR	ZMR	DR	ZMR	DR	ZMR	DR
2006	12.5	0.052	12.5	0.052	7.35	0.077	7.35	0.077
2007	6.84	0.047	7.66	0.057	4.01	0.062	4.78	0.065
2008			7.25	0.047			4.39	0.064
2009			6.44	0.046			3.78	0.062
2010	1.14	0.041	1.72	0.041	0.669	0.053	1.01	0.054
2011			1.46	0.041			0.859	0.054

*The adjusted ZMR and DR have been reflected in Table 1 (Revised MHDDT Emission Factors).

In addition to updating MHDDT emission factors for E55/59 test data, staff also updated carbon dioxide (CO2) emission rates using a similar methodology to that described in ARB (2008). In EMFAC2007, CO2 emission rates of diesel trucks were assumed to remain constant regardless of model year, technology, activity, and other factors. However, with the emphasis on greenhouse gas emissions control over the past few years, staff decided to update CO2 emission rates for individual model years in EMFAC2011. During the Board's In-Use On-Road Diesel Vehicle rulemaking, staff evaluated available data to determine how improvements in engine technology and increasingly stringent criteria pollutant emission control requirements have affected the fuel economy of trucks. Staff reviewed multiple data sources to characterize the variations in the fuel economy values of trucks in California. After fuel economy was estimated, fuel usage was then converted to CO2 emissions. The data and the method used for the CO2 emission rate update have been described in detail in Appendix G of the In-Use On-Road Diesel Vehicle rulemaking staff report (ARB, 2008). Table 11-4 gives the CO2 emission rates for both HHDDT and MHDDT.

Table 11-4. HHDDT and MHDDT CO2 Emission Rates (g/mi)

MY Group	HHDDT	MHDDT
Pre-1987	2,335	1,511
1987-1990	2,262	1,464
1991-1993	2,176	1,408
1994-1997	2,086	1,350
1998-2002	2,137	1,383
2003-2006	2,112	1,367
2007-2009	2,171	1,405
2010+	2,099	1,358
2010+/OBD	2,094	1,355

Staff also updated MHDDT idle emission rates. As with the revision of MHDDT running exhaust emission rates, the available CRC E55/59 idle emission test data allowed staff to revise the MHDDT idle emission rates in EMFAC2011. The method of analysis is the same as that used for calculating HHDDT idle emission rates in EMFAC2007 (see ARB, 2006). The high idle correction factors for HHDDT in EMFAC2007 are also applicable to MHDDT. Table 11-5 shows the revised MHDDT idle emission rates.

Table 11-5. Revised MHDDT Idle Emission Rates (g/hour)

	HC	CO	NOx	PM	CO2
Low Idle					
Pre-1987	20.0	38.7	59.4	5.65	3,802
1987-1990	8.08	23.7	64.6	2.47	4,029
1991-1993	5.56	19.3	71.9	1.75	4,246
1994-1997	3.86	15.7	82.1	1.25	4,532
1998-2002	2.41	12.1	99.3	0.810	4,995
2003-2006	1.73	10.1	113	0.599	5,357
2007-2009	1.73	10.1	30.0	0.0599	5,357
EPA2007	1.73	10.1	30.0	0.0599	5,357
High Idle for Summer					
Pre-1987	33.9	120	125	14.1	8,744
1987-1990	13.7	73.6	136	6.17	9,266
1991-1993	9.46	59.8	151	4.38	9,766
1994-1997	6.56	48.7	172	3.13	10,424
1998-2002	4.09	37.5	208	2.02	11,489
2003-2006	2.95	31.3	237	1.50	12,322
2007-2009	2.95	31.3	63.0	0.150	12,322
EPA2007	2.95	31.3	63.0	0.150	12,322
High Idle for Winter					
1987-1990	17.8	173	116	10.6	7,252
1991-1993	12.2	141	129	7.53	7,643
1994-1997	8.49	115	148	5.38	8,158
1998-2002	5.30	88.2	179	3.48	8,991
2003-2006	3.81	73.7	203	2.58	9,643
2007-2009	3.81	73.7	54.0	0.258	9,643
EPA2007	3.81	73.7	54.0	0.258	9,643

Finally, staff updated HHDDT idle emission rates for NOx and CO2. The EMFAC2007 NOx idle emission rates for HHDDT for 2007 and subsequent model years were projected based on the then prevailing assumption that engine manufacturers would adopt a 5-minute shut-off mechanism to comply with ARB's anti-idle rules. However, ARB certification data demonstrate that all engine manufacturers have instead chosen to meet the ARB requirements with the 30 g/hour NOx idle emission rate option. As a result, staff revised the NOx idle emission rates of 2008+ model year trucks in EMFAC2011 to reflect the engine certification data.

The HHDDT CO2 idle emission rates were also updated in EMFAC2011. In EMFAC2007, an average CO2 idle emission rate was used for all model years. To be consistent with the revision to the CO2 exhaust emission rates of HHDDT, in EMFAC2011 idle emission rates of CO2 were calculated for individual model year groups. Table 11-6 gives the updated NOx and CO2 idle emission rates of HHDDT.

Table 11-6. Updated HHDDT NOx and CO2 Idle Emission Rates (g/hour)

Model Year Group	NOx			CO2		
	Low Idle	High Idle for Summer	High Idle for Winter	Low Idle	High Idle for Summer	High Idle for Winter
Pre-1987	45.7	95.9	82.2	4,271	9,822	7,687
1987-1990	70.2	147	126	4,507	10,365	8,112
1991-1993	78.4	165	141	4,610	10,603	8,298
1994-1997	85.3	179	153	4,713	10,840	8,483
1998-2002	92.1	193	166	4,846	11,145	8,722
2003-2006	95.5	201	172	4,934	11,349	8,882
2007-2009	30.0	63.0	54.0	4,934	11,349	8,882
EPA2007	30.0	63.0	54.0	4,934	11,349	8,882

References

ARB, 2006. California Air Resources Board. *EMFAC Modeling Change Technical Memo: Revision of Heavy Heavy Duty Diesel Truck Emission Factors and Speed Correction Factors*. October 2006. Available at: http://www.arb.ca.gov/msei/onroad/techmemo/revised_hhddt_emission_factors_and_speed_corr_factors.pdf

ARB, 2008. California Air Resources Board. *Staff Report: Proposed Regulation for In-Use On-Road Diesel Vehicles, Appendix G*: *Emissions Inventory Method and Results.* October 2008. Available at: http://www.arb.ca.gov/regact/2008/truckbus08/appg.pdf

12 APPENDIX: DETAILED INCREMENTAL CHANGES

This section documents incremental changes made from EMFAC2007 to the final version of EMFAC2011-SG. Each major change is referenced in each section, and incremental changes are provided for each change for Statewide emissions as well as for the Sacramento Valley, San Francisco Bay Area, San Diego, San Joaquin Valley, and South Coast air basins. Model version names and version numbers are listed in each change.

12.1 REVISE CO_2 EMISSIONS DUE TO AIR CONDITIONING

The following pages give a breakdown and discussion of the emissions changes of the items discussed in Section 3.3.3.2. This update had the effect of reducing CO2 emissions by approximately 5%. This will vary by region depending upon temperature and humidity and age of the fleet.

Table 12-1. Impact on Statewide Inventory of Change 12.1

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.500)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,314	691,047,620	2,239	23,348	2,550	430,950	107.7
2000	22,238,266	799,847,680	1,181	11,621	1,990	491,504	76.4
2005	25,779,210	955,232,640	831	7,688	1,977	614,490	90.4
2007	25,431,494	923,187,330	686	6,327	1,628	587,279	78.5
2010	26,905,676	958,078,400	566	5,086	1,365	606,130	74.1
2014	28,886,190	1,016,924,100	453	3,865	1,026	648,503	69.8
2017	30,223,024	1,059,723,600	394	3,194	824	681,124	67.3
2020	31,543,326	1,104,522,800	350	2,702	676	711,057	65.8
2023	32,780,300	1,145,924,000	320	2,359	581	743,154	66.0

Statewide Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.501)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,314	691,047,620	2,239	23,348	2,550	416,606	107.7
2000	22,238,266	799,847,680	1,181	11,621	1,990	468,243	76.4
2005	25,779,210	955,232,640	831	7,688	1,977	583,869	90.4
2007	25,431,494	923,187,330	686	6,327	1,628	557,267	78.5
2010	26,905,676	958,078,400	566	5,086	1,365	574,695	74.1
2014	28,886,190	1,016,924,100	453	3,865	1,026	614,607	69.8
2017	30,223,024	1,059,723,600	394	3,194	824	645,454	67.3
2020	31,543,326	1,104,522,800	350	2,702	676	673,688	65.8
2023	32,780,300	1,145,924,000	320	2,359	581	704,024	66.0

Difference (Ver. 2.501 - Ver. 2.500) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT(mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0	0	0	-14,344	0.0
2000	0	0	0	0	0	-23,261	0.0
2005	0	0	0	0	0	-30,621	0.0
2007	0	0	0	0	0	-30,012	0.0
2010	0	0	0	0	0	-31,435	0.0
2014	0	0	0	0	0	-33,896	0.0
2017	0	0	0	0	0	-35,670	0.0
2020	0	0	0	0	0	-37,369	0.0
2023	0	0	0	0	0	-39,130	0.0

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.500)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	-3.33%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	-4.73%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	-4.98%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	-5.11%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	-5.19%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	-5.23%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	-5.24%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	-5.26%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	-5.27%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-2. Impact on Sacramento Valley Air Basin Inventory of Change 12.1

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.500)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,223	50,471,700	184.3	1924.2	204.1	32,015	9.13
2000	1,640,558	57,266,592	97.3	940.5	161.2	35,900	6.19
2005	1,987,329	71,431,680	71.2	624.5	166.7	46,619	7.19
2007	1,965,062	68,937,520	60.7	524.4	143.4	45,228	6.39
2010	2,128,722	73,600,320	51.2	427.3	121.0	47,687	5.95
2014	2,322,142	80,409,344	40.8	320.8	90.3	52,260	5.51
2017	2,460,579	85,238,032	35.3	262.0	72.0	55,648	5.27
2020	2,597,977	89,913,456	31.4	220.2	58.7	58,795	5.15
2023	2,742,230	94,758,616	28.8	192.8	50.1	62,091	5.18

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.501)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,223	50,471,700	184.3	1924.2	204.1	30,680	9.13
2000	1,640,558	57,266,592	97.3	940.5	161.2	33,798	6.19
2005	1,987,329	71,431,680	71.2	624.5	166.7	43,732	7.19
2007	1,965,062	68,937,520	60.7	524.4	143.4	42,404	6.39
2010	2,128,722	73,600,320	51.2	427.3	121.0	44,657	5.95
2014	2,322,142	80,409,344	40.8	320.8	90.3	48,892	5.51
2017	2,460,579	85,238,032	35.3	262.0	72.0	52,055	5.27
2020	2,597,977	89,913,456	31.4	220.2	58.7	54,994	5.15
2023	2,742,230	94,758,616	28.8	192.8	50.1	58,071	5.18

Difference (Ver. 2.501 - Ver. 2.500) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT(mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	-1,335	0.00
2000	0	0	0.0	0.0	0.0	-2,102	0.00
2005	0	0	0.0	0.0	0.0	-2,886	0.00
2007	0	0	0.0	0.0	0.0	-2,824	0.00
2010	0	0	0.0	0.0	0.0	-3,030	0.00
2014	0	0	0.0	0.0	0.0	-3,368	0.00
2017	0	0	0.0	0.0	0.0	-3,592	0.00
2020	0	0	0.0	0.0	0.0	-3,800	0.00
2023	0	0	0.0	0.0	0.0	-4,020	0.00

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.500)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	-4.17%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	-5.86%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	-6.19%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	-6.24%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	-6.35%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	-6.44%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	-6.46%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	-6.46%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	-6.47%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-3. Impact on San Diego Air Basin Inventory of Change 12.1

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.500)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,500	65,249,684	196.5	2152.8	206.0	40,979	6.40
2000	1,930,482	74,567,344	95.5	1006.5	147.6	43,250	5.20
2005	2,229,141	87,943,560	63.5	655.9	122.9	52,596	5.90
2007	2,268,770	87,223,808	55.0	568.6	108.9	52,077	5.67
2010	2,329,638	86,947,928	45.0	452.8	89.3	51,691	5.46
2014	2,454,378	90,207,104	36.6	345.7	68.9	53,616	5.41
2017	2,552,300	93,440,232	32.4	289.6	57.3	55,457	5.42
2020	2,654,134	96,987,496	29.6	249.4	48.8	57,432	5.48
2023	2,749,919	100,077,140	27.5	222.6	42.7	59,551	5.61

San Diego Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.501)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,500	65,249,684	196	2153	206	39,690	6.40
2000	1,930,482	74,567,344	96	1006	148	41,425	5.20
2005	2,229,141	87,943,560	64	656	123	50,220	5.90
2007	2,268,770	87,223,808	55	569	109	49,718	5.67
2010	2,329,638	86,947,928	45	453	89	49,336	5.46
2014	2,454,378	90,207,104	37	346	69	51,143	5.41
2017	2,552,300	93,440,232	32	290	57	52,883	5.42
2020	2,654,134	96,987,496	30	249	49	54,753	5.48
2023	2,749,919	100,077,140	28	223	43	56,764	5.61

Difference (Ver. 2.501 - Ver. 2.500) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT(mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	-1,289	0.00
2000	0	0	0.0	0.0	0.0	-1,825	0.00
2005	0	0	0.0	0.0	0.0	-2,376	0.00
2007	0	0	0.0	0.0	0.0	-2,359	0.00
2010	0	0	0.0	0.0	0.0	-2,355	0.00
2014	0	0	0.0	0.0	0.0	-2,473	0.00
2017	0	0	0.0	0.0	0.0	-2,574	0.00
2020	0	0	0.0	0.0	0.0	-2,679	0.00
2023	0	0	0.0	0.0	0.0	-2,786	0.00

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.500)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	-3.14%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	-4.22%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	-4.52%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	-4.53%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	-4.56%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	-4.61%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	-4.64%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	-4.66%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	-4.68%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-4. Impact on San Francisco Bay Air Basin Inventory of Change 12.1

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.500)

Cal. Year	Population	VMT mi/d	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,765	132,557,320	424.5	4409.2	436.5	76,279	12.40
2000	4,710,429	154,959,220	225.9	2183.4	303.5	92,229	9.82
2005	4,931,736	163,790,940	145.9	1322.3	244.2	99,546	10.45
2007	5,036,947	164,750,270	126.1	1126.9	216.0	99,894	10.16
2010	5,292,331	170,504,190	103.3	896.9	177.8	103,271	9.97
2014	5,653,666	180,540,940	81.2	665.5	131.1	108,683	9.79
2017	5,903,500	187,522,050	70.3	545.5	105.6	113,700	9.85
2020	6,144,994	194,476,420	62.3	455.5	86.9	117,593	9.91
2023	6,396,558	201,902,660	56.5	393.5	74.1	121,733	10.08

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.501)

Cal. Year	Population	VMT mi/d	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,765	132,557,320	424.5	4409.2	436.5	73,379	12.40
2000	4,710,429	154,959,220	225.9	2183.4	303.5	87,397	9.82
2005	4,931,736	163,790,940	145.9	1322.3	244.2	93,989	10.45
2007	5,036,947	164,750,270	126.1	1126.9	216.0	94,268	10.16
2010	5,292,331	170,504,190	103.3	896.9	177.8	97,377	9.97
2014	5,653,666	180,540,940	81.2	665.5	131.1	102,380	9.79
2017	5,903,500	187,522,050	70.3	545.5	105.6	107,074	9.85
2020	6,144,994	194,476,420	62.3	455.5	86.9	110,707	9.91
2023	6,396,558	201,902,660	56.5	393.5	74.1	114,570	10.08

Difference (Ver. 2.501 - Ver. 2.500) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	-2,900	0.00
2000	0	0	0.0	0.0	0.0	-4,831	0.00
2005	0	0	0.0	0.0	0.0	-5,557	0.00
2007	0	0	0.0	0.0	0.0	-5,626	0.00
2010	0	0	0.0	0.0	0.0	-5,894	0.00
2014	0	0	0.0	0.0	0.0	-6,303	0.00
2017	0	0	0.0	0.0	0.0	-6,625	0.00
2020	0	0	0.0	0.0	0.0	-6,886	0.00
2023	0	0	0.0	0.0	0.0	-7,163	0.00

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.500)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	-3.80%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	-5.24%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	-5.58%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	-5.63%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	-5.71%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	-5.80%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	-5.83%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	-5.86%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	-5.88%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-5. Impact on San Joaquin Valley Air Basin Inventory of Change 12.1

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.500)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,276	58,324,144	217.2	2295.9	336.1	43,727	22.84
2000	1,943,266	77,177,384	136.0	1329.0	334.8	56,057	15.36
2005	2,389,187	98,949,360	105.3	911.9	398.6	75,951	17.65
2007	2,407,269	97,362,664	91.1	779.7	351.6	74,238	15.39
2010	2,600,194	103,175,620	76.9	637.4	295.4	77,581	13.55
2014	2,830,486	113,056,740	60.8	480.9	219.2	85,499	11.62
2017	3,012,235	121,296,220	51.8	394.2	172.8	92,103	10.47
2020	3,203,412	129,483,290	45.5	334.8	139.7	98,656	9.75
2023	3,406,506	137,546,020	41.8	300.0	119.7	106,532	9.63

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.501)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,276	58,324,144	217.2	2295.9	336.1	42,187	22.84
2000	1,943,266	77,177,384	136.0	1329.0	334.8	53,215	15.36
2005	2,389,187	98,949,360	105.3	911.9	398.6	71,973	17.65
2007	2,407,269	97,362,664	91.1	779.7	351.6	70,282	15.39
2010	2,600,194	103,175,620	76.9	637.4	295.4	73,320	13.55
2014	2,830,486	113,056,740	60.8	480.9	219.2	80,735	11.62
2017	3,012,235	121,296,220	51.8	394.2	172.8	86,967	10.47
2020	3,203,412	129,483,290	45.5	334.8	139.7	93,172	9.75
2023	3,406,506	137,546,020	41.8	300.0	119.7	100,580	9.63

Difference (Ver. 2.501 - Ver. 2.500) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT(mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	-1,540	0.00
2000	0	0	0.0	0.0	0.0	-2,841	0.00
2005	0	0	0.0	0.0	0.0	-3,978	0.00
2007	0	0	0.0	0.0	0.0	-3,956	0.00
2010	0	0	0.0	0.0	0.0	-4,261	0.00
2014	0	0	0.0	0.0	0.0	-4,765	0.00
2017	0	0	0.0	0.0	0.0	-5,136	0.00
2020	0	0	0.0	0.0	0.0	-5,484	0.00
2023	0	0	0.0	0.0	0.0	-5,953	0.00

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.500)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	-3.52%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	-5.07%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	-5.24%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	-5.33%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	-5.49%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	-5.57%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	-5.58%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	-5.56%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	-5.59%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-6. Impact on South Coast Air Basin Inventory of Change 12.1

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.500)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,354	282,561,700	864.7	8974.6	915.0	170,319	31.42
2000	8,884,512	323,009,820	435.5	4364.1	668.4	190,653	23.83
2005	10,519,533	393,767,140	302.4	2952.0	617.2	243,338	29.71
2007	9,971,546	367,745,790	229.9	2264.3	463.3	223,457	24.95
2010	10,473,329	377,734,460	185.2	1793.4	391.1	228,520	24.61
2014	11,179,470	393,460,000	149.3	1377.6	298.8	241,333	24.36
2017	11,582,715	403,019,170	130.6	1143.5	242.4	249,438	24.05
2020	11,961,100	414,266,140	116.7	966.4	199.3	256,666	23.82
2023	12,242,332	422,567,460	105.9	835.0	170.9	264,171	23.92

South Coast Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.501)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,354	282,561,700	864.7	8974.6	915.0	165,412	31.42
2000	8,884,512	323,009,820	435.5	4364.1	668.4	182,610	23.83
2005	10,519,533	393,767,140	302.4	2952.0	617.2	232,449	29.71
2007	9,971,546	367,745,790	229.9	2264.3	463.3	213,120	24.95
2010	10,473,329	377,734,460	185.2	1793.4	391.1	217,868	24.61
2014	11,179,470	393,460,000	149.3	1377.6	298.8	230,137	24.36
2017	11,582,715	403,019,170	130.6	1143.5	242.4	237,892	24.05
2020	11,961,100	414,266,140	116.7	966.4	199.3	244,741	23.82
2023	12,242,332	422,567,460	105.9	835.0	170.9	251,932	23.92

Difference (Ver. 2.501 - Ver. 2.500) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	-4,907	0.00
2000	0	0	0.0	0.0	0.0	-8,043	0.00
2005	0	0	0.0	0.0	0.0	-10,889	0.00
2007	0	0	0.0	0.0	0.0	-10,338	0.00
2010	0	0	0.0	0.0	0.0	-10,652	0.00
2014	0	0	0.0	0.0	0.0	-11,196	0.00
2017	0	0	0.0	0.0	0.0	-11,546	0.00
2020	0	0	0.0	0.0	0.0	-11,925	0.00
2023	0	0	0.0	0.0	0.0	-12,239	0.00

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.500)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	-2.88%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	-4.22%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	-4.47%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	-4.63%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	-4.66%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	-4.64%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	-4.63%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	-4.65%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	-4.63%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type Using Populations

12.2 REVISE EVAPORATIVE DIURNAL BASIC EMISSION RATE COEFFICIENTS

Please refer to Section 3.3.3.3. This change only affects evaporative diurnal emissions, and has less than a 1% effect on emissions. It will vary by area based upon temperature, but for most areas this effect is insignificant.

Table 12-7. Impact on Statewide Inventory of Change 12.2

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.501a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,372	691,048,900	2,239	23,348	2,550	416,607	107.71
2000	22,238,334	799,849,410	1,181	11,621	1,990	468,244	76.42
2005	25,779,274	955,234,500	831	7,688	1,977	583,870	90.36
2007	25,431,560	923,189,120	686	6,327	1,628	557,268	78.52
2010	26,905,742	958,080,320	566	5,086	1,365	574,696	74.11
2014	28,886,264	1,016,926,600	453	3,865	1,026	614,608	69.81
2017	30,223,104	1,059,726,400	394	3,194	824	645,455	67.30
2020	31,543,406	1,104,525,600	350	2,702	676	673,689	65.83
2023	32,780,384	1,145,927,000	320	2,359	581	704,026	66.04

Statewide Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,372	691,048,900	2,239	23,348	2,550	416,607	107.71
2000	22,238,334	799,849,410	1,181	11,621	1,990	468,244	76.42
2005	25,779,274	955,234,500	831	7,688	1,977	583,870	90.36
2007	25,431,560	923,189,120	686	6,327	1,628	557,268	78.52
2010	26,905,742	958,080,320	567	5,086	1,365	574,696	74.11
2014	28,886,264	1,016,926,600	455	3,865	1,026	614,608	69.81
2017	30,223,104	1,059,726,400	396	3,194	824	645,455	67.30
2020	31,543,406	1,104,525,600	353	2,702	676	673,689	65.83
2023	32,780,384	1,145,927,000	323	2,359	581	704,026	66.04

Difference (Ver. 2.503a - Ver. 2.501a) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.1	0.0	0.0	0	0.00
2007	0	0	0.3	0.0	0.0	0	0.00
2010	0	0	0.6	0.0	0.0	0	0.00
2014	0	0	1.3	0.0	0.0	0	0.00
2017	0	0	1.9	0.0	0.0	0	0.00
2020	0	0	2.5	0.0	0.0	0	0.00
2023	0	0	3.1	0.0	0.0	0	0.00

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.501a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.02%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.05%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.11%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.29%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.48%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.70%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.97%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-8. Impact on Sacramento Valley Air Basin Inventory of Change 12.2

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.501a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,228	50,471,784	184.3	1924.2	204.1	30,680	9.13
2000	1,640,564	57,266,724	97.3	940.5	161.2	33,798	6.19
2005	1,987,335	71,431,800	71.2	624.5	166.7	43,732	7.19
2007	1,965,067	68,937,656	60.7	524.4	143.4	42,404	6.39
2010	2,128,728	73,600,472	51.2	427.3	121.0	44,657	5.95
2014	2,322,148	80,409,520	40.8	320.8	90.3	48,892	5.51
2017	2,460,585	85,238,240	35.3	262.0	72.0	52,055	5.27
2020	2,597,983	89,913,688	31.4	220.2	58.7	54,995	5.15
2023	2,742,237	94,758,856	28.8	192.8	50.1	58,071	5.18

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,228	50,471,784	184.3	1924.2	204.1	30,680	9.13
2000	1,640,564	57,266,724	97.3	940.5	161.2	33,798	6.19
2005	1,987,335	71,431,800	71.2	624.5	166.7	43,732	7.19
2007	1,965,067	68,937,656	60.7	524.4	143.4	42,404	6.39
2010	2,128,728	73,600,472	51.2	427.3	121.0	44,657	5.95
2014	2,322,148	80,409,520	41.0	320.8	90.3	48,892	5.51
2017	2,460,585	85,238,240	35.5	262.0	72.0	52,055	5.27
2020	2,597,983	89,913,688	31.7	220.2	58.7	54,995	5.15
2023	2,742,237	94,758,856	29.1	192.8	50.1	58,071	5.18

Difference (Ver. 2.503a - Ver. 2.501a) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.1	0.0	0.0	0	0.00
2014	0	0	0.1	0.0	0.0	0	0.00
2017	0	0	0.2	0.0	0.0	0	0.00
2020	0	0	0.2	0.0	0.0	0	0.00
2023	0	0	0.3	0.0	0.0	0	0.00

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.501a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.02%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.04%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.11%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.29%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.50%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.78%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.10%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-9. Impact on San Diego Air Basin Inventory of Change 12.2

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.501a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,505	65,249,804	196.5	2152.8	206.0	39,690	6.40
2000	1,930,488	74,567,480	95.5	1006.5	147.6	41,425	5.20
2005	2,229,147	87,943,704	63.5	655.9	122.9	50,220	5.90
2007	2,268,776	87,223,976	55.0	568.6	108.9	49,718	5.67
2010	2,329,644	86,948,128	45.0	452.8	89.3	49,336	5.46
2014	2,454,384	90,207,336	36.5	345.7	68.9	51,143	5.41
2017	2,552,306	93,440,464	32.4	289.6	57.3	52,884	5.42
2020	2,654,140	96,987,712	29.5	249.3	48.8	54,753	5.48
2023	2,749,925	100,077,350	27.5	222.6	42.7	56,764	5.61

San Diego Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,505	65,249,804	196	2153	206	39,690	6.40
2000	1,930,488	74,567,480	96	1006	148	41,425	5.20
2005	2,229,147	87,943,704	64	656	123	50,220	5.90
2007	2,268,776	87,223,976	55	569	109	49,718	5.67
2010	2,329,644	86,948,128	45	453	89	49,336	5.46
2014	2,454,384	90,207,336	37	346	69	51,143	5.41
2017	2,552,306	93,440,464	33	290	57	52,884	5.42
2020	2,654,140	96,987,712	30	249	49	54,753	5.48
2023	2,749,925	100,077,350	28	223	43	56,764	5.61

Difference (Ver. 2.503a - Ver. 2.501a) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.1	0.0	0.0	0	0.00
2017	0	0	0.1	0.0	0.0	0	0.00
2020	0	0	0.1	0.0	0.0	0	0.00
2023	0	0	0.2	0.0	0.0	0	0.00

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.501a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.01%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.03%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.08%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.19%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.31%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.47%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.63%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-10. Impact on San Francisco Bay Air Basin Inventory of Change 12.2

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.501a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,775	132,557,540	424.5	4409.2	436.5	73,379	12.40
2000	4,710,441	154,959,550	226.0	2183.4	303.5	87,398	9.82
2005	4,931,748	163,791,250	145.9	1322.3	244.2	93,989	10.45
2007	5,036,958	164,750,560	126.1	1126.9	216.0	94,268	10.16
2010	5,292,343	170,504,560	103.3	896.9	177.8	97,377	9.97
2014	5,653,678	180,541,380	81.2	665.5	131.1	102,380	9.79
2017	5,903,513	187,522,510	70.3	545.5	105.6	107,075	9.85
2020	6,145,010	194,476,900	62.3	455.5	86.9	110,708	9.91
2023	6,396,572	201,903,140	56.5	393.5	74.1	114,571	10.08

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,775	132,557,540	424.5	4409.2	436.5	73,379	12.40
2000	4,710,441	154,959,550	226.0	2183.4	303.5	87,398	9.82
2005	4,931,748	163,791,250	145.9	1322.3	244.2	93,989	10.45
2007	5,036,958	164,750,560	126.1	1126.9	216.0	94,268	10.16
2010	5,292,343	170,504,560	103.4	896.9	177.8	97,377	9.97
2014	5,653,678	180,541,380	81.4	665.5	131.1	102,380	9.79
2017	5,903,513	187,522,510	70.7	545.5	105.6	107,075	9.85
2020	6,145,010	194,476,900	62.8	455.5	86.9	110,708	9.91
2023	6,396,572	201,903,140	57.1	393.5	74.1	114,571	10.08

Difference (Ver. 2.503a - Ver. 2.501a) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.1	0.0	0.0	0	0.00
2010	0	0	0.1	0.0	0.0	0	0.00
2014	0	0	0.2	0.0	0.0	0	0.00
2017	0	0	0.4	0.0	0.0	0	0.00
2020	0	0	0.5	0.0	0.0	0	0.00
2023	0	0	0.6	0.0	0.0	0	0.00

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.501a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.02%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.05%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.12%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.30%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.52%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.80%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.14%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-11. Impact on San Joaquin Valley Air Basin Inventory of Change 12.2

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.501a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,280	58,324,240	217.2	2295.9	336.1	42,187	22.85
2000	1,943,272	77,177,536	136.0	1329.0	334.8	53,215	15.36
2005	2,389,193	98,949,512	105.3	911.9	398.6	71,973	17.65
2007	2,407,275	97,362,808	91.1	779.7	351.6	70,282	15.39
2010	2,600,200	103,175,860	76.9	637.4	295.4	73,320	13.55
2014	2,830,493	113,057,020	60.8	480.9	219.2	80,735	11.62
2017	3,012,243	121,296,510	51.8	394.2	172.8	86,967	10.47
2020	3,203,419	129,483,600	45.5	334.8	139.7	93,173	9.75
2023	3,406,514	137,546,340	41.8	300.0	119.7	100,580	9.63

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,280	58,324,240	217.2	2295.9	336.1	42,187	22.85
2000	1,943,272	77,177,536	136.0	1329.0	334.8	53,215	15.36
2005	2,389,193	98,949,512	105.4	911.9	398.6	71,973	17.65
2007	2,407,275	97,362,808	91.1	779.7	351.6	70,282	15.39
2010	2,600,200	103,175,860	77.0	637.4	295.4	73,320	13.55
2014	2,830,493	113,057,020	60.9	480.9	219.2	80,735	11.62
2017	3,012,243	121,296,510	52.0	394.2	172.8	86,967	10.47
2020	3,203,419	129,483,600	45.9	334.8	139.7	93,173	9.75
2023	3,406,514	137,546,340	42.2	300.0	119.7	100,580	9.63

Difference (Ver. 2.503a - Ver. 2.501a) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.1	0.0	0.0	0	0.00
2014	0	0	0.1	0.0	0.0	0	0.00
2017	0	0	0.2	0.0	0.0	0	0.00
2020	0	0	0.3	0.0	0.0	0	0.00
2023	0	0	0.4	0.0	0.0	0	0.00

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.501a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.01%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.04%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.09%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.25%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.43%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.69%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.97%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-12. Impact on South Coast Air Basin Inventory of Change 12.2

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2009 ver 2.501a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,380	282,562,270	864.7	8974.7	915.0	165,412	31.42
2000	8,884,540	323,010,620	435.5	4364.2	668.4	182,611	23.83
2005	10,519,562	393,767,940	302.4	2952.0	617.2	232,450	29.71
2007	9,971,575	367,746,560	229.9	2264.3	463.3	213,120	24.95
2010	10,473,359	377,735,330	185.2	1793.4	391.1	217,869	24.61
2014	11,179,501	393,461,060	149.3	1377.6	298.8	230,138	24.36
2017	11,582,747	403,020,260	130.6	1143.5	242.4	237,893	24.05
2020	11,961,135	414,267,300	116.7	966.4	199.3	244,742	23.82
2023	12,242,366	422,568,670	105.9	835.0	170.9	251,932	23.92

South Coast Summer Episodic On-Road Motor Vehicle Inventories With CO2 Bug Fix
(Calculated Using EMFAC2009 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,380	282,562,270	864.7	8974.7	915.0	165,412	31.42
2000	8,884,540	323,010,620	435.5	4364.2	668.4	182,611	23.83
2005	10,519,562	393,767,940	302.4	2952.0	617.2	232,450	29.71
2007	9,971,575	367,746,560	230.1	2264.3	463.3	213,120	24.95
2010	10,473,359	377,735,330	185.4	1793.4	391.1	217,869	24.61
2014	11,179,501	393,461,060	149.7	1377.6	298.8	230,138	24.36
2017	11,582,747	403,020,260	131.2	1143.5	242.4	237,893	24.05
2020	11,961,135	414,267,300	117.4	966.4	199.3	244,742	23.82
2023	12,242,366	422,568,670	106.8	835.0	170.9	251,932	23.92

Difference (Ver. 2.503a - Ver. 2.501a) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.1	0.0	0.0	0	0.00
2007	0	0	0.1	0.0	0.0	0	0.00
2010	0	0	0.2	0.0	0.0	0	0.00
2014	0	0	0.4	0.0	0.0	0	0.00
2017	0	0	0.5	0.0	0.0	0	0.00
2020	0	0	0.7	0.0	0.0	0	0.00
2023	0	0	0.9	0.0	0.0	0	0.00

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.501a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.02%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.05%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.12%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.26%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.42%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.62%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.84%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.3 FIX RESTING TIME IN HOT SOAK ALGORITHM

Please refer to Section 3.3.3.4. This change resulted in a general increase in hot soak evaporative emissions by less than 2%. This factor does not very significantly by area.

Table 12-13. Impact on Statewide Inventory of Change 12.3

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,372	691,048,900	2,239	23,348	2,550	416,607	130.23
2000	22,238,334	799,849,410	1,181	11,621	1,990	468,244	102.21
2005	25,779,274	955,234,500	831	7,688	1,977	583,870	121.44
2007	25,431,560	923,189,120	686	6,327	1,628	557,268	108.46
2010	26,905,742	958,080,320	567	5,086	1,365	574,696	105.25
2014	28,886,264	1,016,926,600	455	3,865	1,026	614,608	102.87
2017	30,223,104	1,059,726,400	396	3,194	824	645,455	101.78
2020	31,543,406	1,104,525,600	353	2,702	676	673,689	101.76
2023	32,780,384	1,145,927,000	323	2,359	581	704,026	103.38

Statewide Summer Episodic On-Road Motor Vehicle Inventories With HS Resting time Bug Fix
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,372	691,048,900	2,269	23,348	2,550	416,607	130.23
2000	22,238,334	799,849,410	1,195	11,621	1,990	468,244	102.21
2005	25,779,274	955,234,500	841	7,688	1,977	583,870	121.44
2007	25,431,560	923,189,120	695	6,327	1,628	557,268	108.46
2010	26,905,742	958,080,320	574	5,086	1,365	574,696	105.25
2014	28,886,264	1,016,926,600	462	3,865	1,026	614,608	102.87
2017	30,223,104	1,059,726,400	403	3,194	824	645,455	101.78
2020	31,543,406	1,104,525,600	360	2,702	676	673,689	101.76
2023	32,780,384	1,145,927,000	329	2,359	581	704,026	103.38

Difference (Ver. 2.504a - Ver. 2.504) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	30.1	0.0	0.0	0	0.00
2000	0	0	13.6	0.0	0.0	0	0.00
2005	0	0	9.7	0.0	0.0	0	0.00
2007	0	0	8.5	0.0	0.0	0	0.00
2010	0	0	7.9	0.0	0.0	0	0.00
2014	0	0	7.4	0.0	0.0	0	0.00
2017	0	0	7.1	0.0	0.0	0	0.00
2020	0	0	6.7	0.0	0.0	0	0.00
2023	0	0	6.3	0.0	0.0	0	0.00

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.504)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.34%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.15%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	1.16%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	1.24%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	1.40%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.64%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	1.78%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	1.89%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.94%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-14. Impact on Sacramento Valley Air Basin Inventory of Change 12.3

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,228	50,471,784	184.3	1924.2	204.1	30,680	10.78
2000	1,640,564	57,266,724	97.3	940.5	161.2	33,798	8.03
2005	1,987,335	71,431,800	71.2	624.5	166.7	43,732	9.52
2007	1,965,067	68,937,656	60.7	524.4	143.4	42,404	8.64
2010	2,128,728	73,600,472	51.2	427.3	121.0	44,657	8.35
2014	2,322,148	80,409,520	41.0	320.8	90.3	48,892	8.13
2017	2,460,585	85,238,240	35.5	262.0	72.0	52,055	8.05
2020	2,597,983	89,913,688	31.7	220.2	58.7	54,995	8.08
2023	2,742,237	94,758,856	29.1	192.8	50.1	58,071	8.27

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With HS Resting time Bug Fix
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,228	50,471,784	187.0	1924.2	204.1	30,680	10.78
2000	1,640,564	57,266,724	98.6	940.5	161.2	33,798	8.03
2005	1,987,335	71,431,800	72.1	624.5	166.7	43,732	9.52
2007	1,965,067	68,937,656	61.6	524.4	143.4	42,404	8.64
2010	2,128,728	73,600,472	52.0	427.3	121.0	44,657	8.35
2014	2,322,148	80,409,520	41.7	320.8	90.3	48,892	8.13
2017	2,460,585	85,238,240	36.2	262.0	72.0	52,055	8.05
2020	2,597,983	89,913,688	32.3	220.2	58.7	54,995	8.08
2023	2,742,237	94,758,856	29.7	192.8	50.1	58,071	8.27

Difference (Ver. 2.504a - Ver. 2.504) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	2.7	0.0	0.0	0	0.00
2000	0	0	1.3	0.0	0.0	0	0.00
2005	0	0	1.0	0.0	0.0	0	0.00
2007	0	0	0.9	0.0	0.0	0	0.00
2010	0	0	0.8	0.0	0.0	0	0.00
2014	0	0	0.7	0.0	0.0	0	0.00
2017	0	0	0.7	0.0	0.0	0	0.00
2020	0	0	0.6	0.0	0.0	0	0.00
2023	0	0	0.6	0.0	0.0	0	0.00

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.504)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.47%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.32%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	1.34%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	1.40%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	1.55%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.74%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	1.86%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	1.93%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.96%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-15. Impact on San Diego Air Basin Inventory of Change 12.3

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,505	65,249,804	196.5	2152.8	206.0	39,690	8.43
2000	1,930,488	74,567,480	95.5	1006.5	147.6	41,425	7.52
2005	2,229,147	87,943,704	63.5	655.9	122.9	50,220	8.69
2007	2,268,776	87,223,976	55.0	568.6	108.9	49,718	8.44
2010	2,329,644	86,948,128	45.0	452.8	89.3	49,336	8.23
2014	2,454,384	90,207,336	36.6	345.7	68.9	51,143	8.28
2017	2,552,306	93,440,464	32.5	289.6	57.3	52,884	8.38
2020	2,654,140	96,987,712	29.7	249.3	48.8	54,753	8.55
2023	2,749,925	100,077,350	27.7	222.6	42.7	56,764	8.78

San Diego Summer Episodic On-Road Motor Vehicle Inventories With HS Resting time Bug Fix
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,505	65,249,804	199	2153	206	39,690	8.43
2000	1,930,488	74,567,480	97	1006	148	41,425	7.52
2005	2,229,147	87,943,704	64	656	123	50,220	8.69
2007	2,268,776	87,223,976	56	569	109	49,718	8.44
2010	2,329,644	86,948,128	46	453	89	49,336	8.23
2014	2,454,384	90,207,336	37	346	69	51,143	8.28
2017	2,552,306	93,440,464	33	290	57	52,884	8.38
2020	2,654,140	96,987,712	30	249	49	54,753	8.55
2023	2,749,925	100,077,350	28	223	43	56,764	8.78

Difference (Ver. 2.504a - Ver. 2.504) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	2.3	0.0	0.0	0	0.00
2000	0	0	1.0	0.0	0.0	0	0.00
2005	0	0	0.7	0.0	0.0	0	0.00
2007	0	0	0.6	0.0	0.0	0	0.00
2010	0	0	0.6	0.0	0.0	0	0.00
2014	0	0	0.6	0.0	0.0	0	0.00
2017	0	0	0.6	0.0	0.0	0	0.00
2020	0	0	0.5	0.0	0.0	0	0.00
2023	0	0	0.5	0.0	0.0	0	0.00

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.504)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.17%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.04%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	1.07%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	1.14%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	1.31%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.56%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	1.70%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	1.79%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.83%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-16. Impact on San Francisco Bay Air Basin Inventory of Change 12.3

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,775	132,557,540	424.5	4409.2	436.5	73,379	16.74
2000	4,710,441	154,959,550	226.0	2183.4	303.5	87,398	14.92
2005	4,931,748	163,791,250	145.9	1322.3	244.2	93,989	15.92
2007	5,036,958	164,750,560	126.1	1126.9	216.0	94,268	15.66
2010	5,292,343	170,504,560	103.4	896.9	177.8	97,377	15.66
2014	5,653,678	180,541,380	81.4	665.5	131.1	102,380	15.79
2017	5,903,513	187,522,510	70.7	545.5	105.6	107,075	16.07
2020	6,145,010	194,476,900	62.8	455.5	86.9	110,708	16.36
2023	6,396,572	201,903,140	57.1	393.5	74.1	114,571	16.76

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With HS Resting time Bug Fix
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,775	132,557,540	430.7	4409.2	436.5	73,379	16.74
2000	4,710,441	154,959,550	228.9	2183.4	303.5	87,398	14.92
2005	4,931,748	163,791,250	147.9	1322.3	244.2	93,989	15.92
2007	5,036,958	164,750,560	128.0	1126.9	216.0	94,268	15.66
2010	5,292,343	170,504,560	105.1	896.9	177.8	97,377	15.66
2014	5,653,678	180,541,380	83.0	665.5	131.1	102,380	15.79
2017	5,903,513	187,522,510	72.2	545.5	105.6	107,075	16.07
2020	6,145,010	194,476,900	64.1	455.5	86.9	110,708	16.36
2023	6,396,572	201,903,140	58.4	393.5	74.1	114,571	16.76

Difference (Ver. 2.504a - Ver. 2.504) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	6.3	0.0	0.0	0	0.00
2000	0	0	3.0	0.0	0.0	0	0.00
2005	0	0	2.0	0.0	0.0	0	0.00
2007	0	0	1.9	0.0	0.0	0	0.00
2010	0	0	1.7	0.0	0.0	0	0.00
2014	0	0	1.6	0.0	0.0	0	0.00
2017	0	0	1.5	0.0	0.0	0	0.00
2020	0	0	1.4	0.0	0.0	0	0.00
2023	0	0	1.3	0.0	0.0	0	0.00

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.504)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.48%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.31%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	1.38%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	1.47%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	1.67%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.94%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	2.09%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	2.19%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	2.22%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-17. Impact on San Joaquin Valley Air Basin Inventory of Change 12.3

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,280	58,324,240	217.2	2295.9	336.1	42,187	24.89
2000	1,943,272	77,177,536	136.0	1329.0	334.8	53,215	17.96
2005	2,389,193	98,949,512	105.4	911.9	398.6	71,973	21.01
2007	2,407,275	97,362,808	91.1	779.7	351.6	70,282	18.71
2010	2,600,200	103,175,860	77.0	637.4	295.4	73,320	17.07
2014	2,830,493	113,057,020	60.9	480.9	219.2	80,735	15.47
2017	3,012,243	121,296,510	52.0	394.2	172.8	86,967	14.60
2020	3,203,419	129,483,600	45.9	334.8	139.7	93,173	14.16
2023	3,406,514	137,546,340	42.2	300.0	119.7	100,580	14.32

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With HS Resting time Bug Fix
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,280	58,324,240	220.0	2295.9	336.1	42,187	24.89
2000	1,943,272	77,177,536	137.6	1329.0	334.8	53,215	17.96
2005	2,389,193	98,949,512	106.5	911.9	398.6	71,973	21.01
2007	2,407,275	97,362,808	92.1	779.7	351.6	70,282	18.71
2010	2,600,200	103,175,860	78.0	637.4	295.4	73,320	17.07
2014	2,830,493	113,057,020	61.8	480.9	219.2	80,735	15.47
2017	3,012,243	121,296,510	52.8	394.2	172.8	86,967	14.60
2020	3,203,419	129,483,600	46.6	334.8	139.7	93,173	14.16
2023	3,406,514	137,546,340	42.9	300.0	119.7	100,580	14.32

Difference (Ver. 2.504a - Ver. 2.504) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	2.8	0.0	0.0	0	0.00
2000	0	0	1.5	0.0	0.0	0	0.00
2005	0	0	1.2	0.0	0.0	0	0.00
2007	0	0	1.0	0.0	0.0	0	0.00
2010	0	0	1.0	0.0	0.0	0	0.00
2014	0	0	0.9	0.0	0.0	0	0.00
2017	0	0	0.8	0.0	0.0	0	0.00
2020	0	0	0.7	0.0	0.0	0	0.00
2023	0	0	0.7	0.0	0.0	0	0.00

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.504)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.30%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.13%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	1.10%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	1.15%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	1.27%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.43%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	1.53%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	1.60%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.62%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-18. Impact on South Coast Air Basin Inventory of Change 12.3

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,380	282,562,270	864.7	8974.7	915.0	165,412	40.60
2000	8,884,540	323,010,620	435.5	4364.2	668.4	182,611	34.19
2005	10,519,562	393,767,940	302.4	2952.0	617.2	232,450	42.30
2007	9,971,575	367,746,560	230.1	2264.3	463.3	213,120	36.60
2010	10,473,359	377,735,330	185.4	1793.4	391.1	217,869	36.63
2014	11,179,501	393,461,060	149.7	1377.6	298.8	230,138	36.90
2017	11,582,747	403,020,260	131.2	1143.5	242.4	237,893	36.93
2020	11,961,135	414,267,300	117.4	966.4	199.3	244,742	37.06
2023	12,242,366	422,568,670	106.8	835.0	170.9	251,932	37.47

South Coast Summer Episodic On-Road Motor Vehicle Inventories With HS Resting time Bug Fix
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,380	282,562,270	876.2	8974.7	915.0	165,412	40.60
2000	8,884,540	323,010,620	440.3	4364.2	668.4	182,611	34.19
2005	10,519,562	393,767,940	305.9	2952.0	617.2	232,450	42.30
2007	9,971,575	367,746,560	232.9	2264.3	463.3	213,120	36.60
2010	10,473,359	377,735,330	188.1	1793.4	391.1	217,869	36.63
2014	11,179,501	393,461,060	152.2	1377.6	298.8	230,138	36.90
2017	11,582,747	403,020,260	133.6	1143.5	242.4	237,893	36.93
2020	11,961,135	414,267,300	119.7	966.4	199.3	244,742	37.06
2023	12,242,366	422,568,670	109.0	835.0	170.9	251,932	37.47

Difference (Ver. 2.504a - Ver. 2.504) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	11.5	0.0	0.0	0	0.00
2000	0	0	4.8	0.0	0.0	0	0.00
2005	0	0	3.4	0.0	0.0	0	0.00
2007	0	0	2.8	0.0	0.0	0	0.00
2010	0	0	2.6	0.0	0.0	0	0.00
2014	0	0	2.5	0.0	0.0	0	0.00
2017	0	0	2.4	0.0	0.0	0	0.00
2020	0	0	2.3	0.0	0.0	0	0.00
2023	0	0	2.2	0.0	0.0	0	0.00

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.504)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.33%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.11%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	1.14%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	1.23%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	1.41%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.68%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	1.86%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	1.99%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	2.06%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type Using Populations

12.4 REVISE BRAKE WEAR PM EMISSION FACTOR

Please refer to Section 3.3.3.5. This update significantly increases PM10 emissions by 40 to 50%. The largest part of the change is due to moving the fleet away asbestos brakes which is independent of area. There is some regional variation largely because of fleet mix differences in heavy-duty trucks. It should be noted that brake ware tends to be larger particles, so the PM 2.5 disbenefit would be approximately 60% less.

Table 12-19. Impact on Statewide Inventory of Change 12.4

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,372	691,048,900	2,239	23,348	2,550	416,607	107.71
2000	22,238,334	799,849,410	1,181	11,621	1,990	468,244	76.42
2005	25,779,274	955,234,500	831	7,688	1,977	583,870	90.36
2007	25,431,560	923,189,120	686	6,327	1,628	557,268	78.52
2010	26,905,742	958,080,320	567	5,086	1,365	574,696	74.11
2014	28,886,264	1,016,926,600	455	3,865	1,026	614,608	69.81
2017	30,223,104	1,059,726,400	396	3,194	824	645,455	67.30
2020	31,543,406	1,104,525,600	353	2,702	676	673,689	65.83
2023	32,780,384	1,145,927,000	323	2,359	581	704,026	66.04

Statewide Summer Episodic On-Road Motor Vehicle Inventories With Programming Change
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,372	691,048,900	2,239	23,348	2,550	416,607	130.23
2000	22,238,334	799,849,410	1,181	11,621	1,990	468,244	102.21
2005	25,779,274	955,234,500	831	7,688	1,977	583,870	121.44
2007	25,431,560	923,189,120	686	6,327	1,628	557,268	108.46
2010	26,905,742	958,080,320	567	5,086	1,365	574,696	105.25
2014	28,886,264	1,016,926,600	455	3,865	1,026	614,608	102.87
2017	30,223,104	1,059,726,400	396	3,194	824	645,455	101.78
2020	31,543,406	1,104,525,600	353	2,702	676	673,689	101.76
2023	32,780,384	1,145,927,000	323	2,359	581	704,026	103.38

Difference (Ver. 2.504 - Ver. 2.503a) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	22.52
2000	0	0	0.0	0.0	0.0	0	25.80
2005	0	0	0.0	0.0	0.0	0	31.08
2007	0	0	0.0	0.0	0.0	0	29.95
2010	0	0	0.0	0.0	0.0	0	31.14
2014	0	0	0.0	0.0	0.0	0	33.06
2017	0	0	0.0	0.0	0.0	0	34.48
2020	0	0	0.0	0.0	0.0	0	35.94
2023	0	0	0.0	0.0	0.0	0	37.34

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.503a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	20.91%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	33.76%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	34.40%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	38.14%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	42.02%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	47.36%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	51.23%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	54.60%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	56.54%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-20. Impact on Sacramento Valley Air Basin Inventory of Change 12.4

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,228	50,471,784	184.3	1924.2	204.1	30,680	9.13
2000	1,640,564	57,266,724	97.3	940.5	161.2	33,798	6.19
2005	1,987,335	71,431,800	71.2	624.5	166.7	43,732	7.19
2007	1,965,067	68,937,656	60.7	524.4	143.4	42,404	6.39
2010	2,128,728	73,600,472	51.2	427.3	121.0	44,657	5.95
2014	2,322,148	80,409,520	41.0	320.8	90.3	48,892	5.51
2017	2,460,585	85,238,240	35.5	262.0	72.0	52,055	5.27
2020	2,597,983	89,913,688	31.7	220.2	58.7	54,995	5.15
2023	2,742,237	94,758,856	29.1	192.8	50.1	58,071	5.18

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Programming Change
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,228	50,471,784	184.3	1924.2	204.1	30,680	10.78
2000	1,640,564	57,266,724	97.3	940.5	161.2	33,798	8.03
2005	1,987,335	71,431,800	71.2	624.5	166.7	43,732	9.52
2007	1,965,067	68,937,656	60.7	524.4	143.4	42,404	8.64
2010	2,128,728	73,600,472	51.2	427.3	121.0	44,657	8.35
2014	2,322,148	80,409,520	41.0	320.8	90.3	48,892	8.13
2017	2,460,585	85,238,240	35.5	262.0	72.0	52,055	8.05
2020	2,597,983	89,913,688	31.7	220.2	58.7	54,995	8.08
2023	2,742,237	94,758,856	29.1	192.8	50.1	58,071	8.27

Difference (Ver. 2.504 - Ver. 2.503a) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	1.65
2000	0	0	0.0	0.0	0.0	0	1.84
2005	0	0	0.0	0.0	0.0	0	2.33
2007	0	0	0.0	0.0	0.0	0	2.25
2010	0	0	0.0	0.0	0.0	0	2.41
2014	0	0	0.0	0.0	0.0	0	2.62
2017	0	0	0.0	0.0	0.0	0	2.78
2020	0	0	0.0	0.0	0.0	0	2.93
2023	0	0	0.0	0.0	0.0	0	3.09

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.503a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	18.10%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	29.75%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	32.42%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	35.30%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	40.47%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	47.60%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	52.70%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	56.94%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	59.67%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-21. Impact on San Diego Air Basin Inventory of Change 12.4

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,505	65,249,804	196.5	2152.8	206.0	39,690	6.40
2000	1,930,488	74,567,480	95.5	1006.5	147.6	41,425	5.20
2005	2,229,147	87,943,704	63.5	655.9	122.9	50,220	5.90
2007	2,268,776	87,223,976	55.0	568.6	108.9	49,718	5.67
2010	2,329,644	86,948,128	45.0	452.8	89.3	49,336	5.46
2014	2,454,384	90,207,336	36.6	345.7	68.9	51,143	5.41
2017	2,552,306	93,440,464	32.5	289.6	57.3	52,884	5.42
2020	2,654,140	96,987,712	29.7	249.3	48.8	54,753	5.48
2023	2,749,925	100,077,350	27.7	222.6	42.7	56,764	5.61

San Diego Summer Episodic On-Road Motor Vehicle Inventories With Programming Change
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,505	65,249,804	196	2153	206	39,690	8.43
2000	1,930,488	74,567,480	96	1006	148	41,425	7.52
2005	2,229,147	87,943,704	64	656	123	50,220	8.69
2007	2,268,776	87,223,976	55	569	109	49,718	8.44
2010	2,329,644	86,948,128	45	453	89	49,336	8.23
2014	2,454,384	90,207,336	37	346	69	51,143	8.28
2017	2,552,306	93,440,464	33	290	57	52,884	8.38
2020	2,654,140	96,987,712	30	249	49	54,753	8.55
2023	2,749,925	100,077,350	28	223	43	56,764	8.78

Difference (Ver. 2.504 - Ver. 2.503a) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	2.03
2000	0	0	0.0	0.0	0.0	0	2.32
2005	0	0	0.0	0.0	0.0	0	2.79
2007	0	0	0.0	0.0	0.0	0	2.77
2010	0	0	0.0	0.0	0.0	0	2.77
2014	0	0	0.0	0.0	0.0	0	2.87
2017	0	0	0.0	0.0	0.0	0	2.96
2020	0	0	0.0	0.0	0.0	0	3.07
2023	0	0	0.0	0.0	0.0	0	3.17

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.503a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	31.75%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	44.54%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	47.31%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	48.95%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	50.61%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	52.95%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	54.69%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	56.08%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	56.43%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-22. Impact on San Francisco Bay Air Basin Inventory of Change 12.4

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,775	132,557,540	424.5	4409.2	436.5	73,379	12.40
2000	4,710,441	154,959,550	226.0	2183.4	303.5	87,398	9.82
2005	4,931,748	163,791,250	145.9	1322.3	244.2	93,989	10.45
2007	5,036,958	164,750,560	126.1	1126.9	216.0	94,268	10.16
2010	5,292,343	170,504,560	103.4	896.9	177.8	97,377	9.97
2014	5,653,678	180,541,380	81.4	665.5	131.1	102,380	9.79
2017	5,903,513	187,522,510	70.7	545.5	105.6	107,075	9.85
2020	6,145,010	194,476,900	62.8	455.5	86.9	110,708	9.91
2023	6,396,572	201,903,140	57.1	393.5	74.1	114,571	10.08

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Programming Change
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,775	132,557,540	424.5	4409.2	436.5	73,379	16.74
2000	4,710,441	154,959,550	226.0	2183.4	303.5	87,398	14.92
2005	4,931,748	163,791,250	145.9	1322.3	244.2	93,989	15.92
2007	5,036,958	164,750,560	126.1	1126.9	216.0	94,268	15.66
2010	5,292,343	170,504,560	103.4	896.9	177.8	97,377	15.66
2014	5,653,678	180,541,380	81.4	665.5	131.1	102,380	15.79
2017	5,903,513	187,522,510	70.7	545.5	105.6	107,075	16.07
2020	6,145,010	194,476,900	62.8	455.5	86.9	110,708	16.36
2023	6,396,572	201,903,140	57.1	393.5	74.1	114,571	16.76

Difference (Ver. 2.504 - Ver. 2.503a) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	4.34
2000	0	0	0.0	0.0	0.0	0	5.09
2005	0	0	0.0	0.0	0.0	0	5.46
2007	0	0	0.0	0.0	0.0	0	5.50
2010	0	0	0.0	0.0	0.0	0	5.68
2014	0	0	0.0	0.0	0.0	0	6.00
2017	0	0	0.0	0.0	0.0	0	6.22
2020	0	0	0.0	0.0	0.0	0	6.45
2023	0	0	0.0	0.0	0.0	0	6.68

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.503a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	35.01%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	51.82%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	52.27%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	54.11%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	57.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	61.31%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	63.18%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	65.04%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	66.31%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-23. Impact on San Joaquin Valley Air Basin Inventory of Change 12.4

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,280	58,324,240	217.2	2295.9	336.1	42,187	22.85
2000	1,943,272	77,177,536	136.0	1329.0	334.8	53,215	15.36
2005	2,389,193	98,949,512	105.4	911.9	398.6	71,973	17.65
2007	2,407,275	97,362,808	91.1	779.7	351.6	70,282	15.39
2010	2,600,200	103,175,860	77.0	637.4	295.4	73,320	13.55
2014	2,830,493	113,057,020	60.9	480.9	219.2	80,735	11.62
2017	3,012,243	121,296,510	52.0	394.2	172.8	86,967	10.47
2020	3,203,419	129,483,600	45.9	334.8	139.7	93,173	9.75
2023	3,406,514	137,546,340	42.2	300.0	119.7	100,580	9.63

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Programming Change
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,280	58,324,240	217.2	2295.9	336.1	42,187	24.89
2000	1,943,272	77,177,536	136.0	1329.0	334.8	53,215	17.96
2005	2,389,193	98,949,512	105.4	911.9	398.6	71,973	21.01
2007	2,407,275	97,362,808	91.1	779.7	351.6	70,282	18.71
2010	2,600,200	103,175,860	77.0	637.4	295.4	73,320	17.07
2014	2,830,493	113,057,020	60.9	480.9	219.2	80,735	15.47
2017	3,012,243	121,296,510	52.0	394.2	172.8	86,967	14.60
2020	3,203,419	129,483,600	45.9	334.8	139.7	93,173	14.16
2023	3,406,514	137,546,340	42.2	300.0	119.7	100,580	14.32

Difference (Ver. 2.504 - Ver. 2.503a) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	2.04
2000	0	0	0.0	0.0	0.0	0	2.60
2005	0	0	0.0	0.0	0.0	0	3.36
2007	0	0	0.0	0.0	0.0	0	3.32
2010	0	0	0.0	0.0	0.0	0	3.52
2014	0	0	0.0	0.0	0.0	0	3.85
2017	0	0	0.0	0.0	0.0	0	4.13
2020	0	0	0.0	0.0	0.0	0	4.41
2023	0	0	0.0	0.0	0.0	0	4.69

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.503a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	8.93%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	16.91%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	19.06%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	21.55%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	25.96%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	33.16%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	39.47%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	45.30%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	48.73%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-24. Impact on South Coast Air Basin Inventory of Change 12.4

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.503a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,380	282,562,270	864.7	8974.7	915.0	165,412	31.42
2000	8,884,540	323,010,620	435.5	4364.2	668.4	182,611	23.83
2005	10,519,562	393,767,940	302.4	2952.0	617.2	232,450	29.71
2007	9,971,575	367,746,560	230.1	2264.3	463.3	213,120	24.95
2010	10,473,359	377,735,330	185.4	1793.4	391.1	217,869	24.61
2014	11,179,501	393,461,060	149.7	1377.6	298.8	230,138	24.36
2017	11,582,747	403,020,260	131.2	1143.5	242.4	237,893	24.05
2020	11,961,135	414,267,300	117.4	966.4	199.3	244,742	23.82
2023	12,242,366	422,568,670	106.8	835.0	170.9	251,932	23.92

South Coast Summer Episodic On-Road Motor Vehicle Inventories With Programming Change
(Calculated Using EMFAC2011 ver 2.504)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,380	282,562,270	864.7	8974.7	915.0	165,412	40.60
2000	8,884,540	323,010,620	435.5	4364.2	668.4	182,611	34.19
2005	10,519,562	393,767,940	302.4	2952.0	617.2	232,450	42.30
2007	9,971,575	367,746,560	230.1	2264.3	463.3	213,120	36.60
2010	10,473,359	377,735,330	185.4	1793.4	391.1	217,869	36.63
2014	11,179,501	393,461,060	149.7	1377.6	298.8	230,138	36.90
2017	11,582,747	403,020,260	131.2	1143.5	242.4	237,893	36.93
2020	11,961,135	414,267,300	117.4	966.4	199.3	244,742	37.06
2023	12,242,366	422,568,670	106.8	835.0	170.9	251,932	37.47

Difference (Ver. 2.504 - Ver. 2.503a) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	9.18
2000	0	0	0.0	0.0	0.0	0	10.35
2005	0	0	0.0	0.0	0.0	0	12.59
2007	0	0	0.0	0.0	0.0	0	11.65
2010	0	0	0.0	0.0	0.0	0	12.02
2014	0	0	0.0	0.0	0.0	0	12.54
2017	0	0	0.0	0.0	0.0	0	12.87
2020	0	0	0.0	0.0	0.0	0	13.24
2023	0	0	0.0	0.0	0.0	0	13.55

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.503a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	29.23%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	43.45%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	42.36%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	46.70%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	48.82%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	51.48%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	53.52%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	55.58%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	56.67%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type Using Populations

12.5 HEAVY DUTY DIESEL TRUCK EMISSION FACTOR UPDATE

As described in Section 4.6, heavy duty diesel truck emission factors have been updated in accordance with new assumptions developed through the Statewide Truck and Bus Rule development process.

Table 12-25. Impact on Statewide Inventory of Change 12.5

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,372	691,048,900	2,269	23,348	2,550	416,607	130.23
2000	22,238,334	799,849,410	1,195	11,621	1,990	468,244	102.21
2005	25,779,274	955,234,500	841	7,688	1,977	583,870	121.44
2007	25,431,560	923,189,120	695	6,327	1,628	557,268	108.46
2010	26,905,742	958,080,320	574	5,086	1,365	574,696	105.25
2014	28,886,264	1,016,926,600	462	3,865	1,026	614,608	102.87
2017	30,223,104	1,059,726,400	403	3,194	824	645,455	101.78
2020	31,543,406	1,104,525,600	360	2,702	676	673,689	101.76
2023	32,780,384	1,145,927,000	329	2,359	581	704,026	103.38

Statewide Summer Episodic On-Road Motor Vehicle Inventories With MHDT BER update
(Calculated Using EMFAC2011 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,372	691,048,900	2,280	23,350	2,506	416,499	133.76
2000	22,238,334	799,849,410	1,200	11,620	1,968	463,492	104.43
2005	25,779,274	955,234,500	847	7,684	1,961	575,802	123.84
2007	25,431,560	923,189,120	699	6,323	1,621	549,751	110.52
2010	26,905,742	958,080,320	579	5,081	1,359	566,707	107.09
2014	28,886,264	1,016,926,700	466	3,859	1,018	605,152	104.12
2017	30,223,104	1,059,726,400	406	3,186	808	634,826	102.45
2020	31,543,406	1,104,525,600	362	2,692	652	662,065	101.80
2023	32,780,384	1,145,927,000	331	2,346	549	691,551	102.86

Difference (Ver. 2.505 - Ver. 2.504a) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	10.6	1.5	-43.5	-108	3.53
2000	0	0	5.5	-1.5	-22.8	-4,753	2.22
2005	0	0	5.7	-4.0	-15.4	-8,067	2.40
2007	0	0	4.9	-4.2	-7.2	-7,517	2.06
2010	0	0	4.5	-4.9	-5.7	-7,989	1.84
2014	0	100	3.7	-5.9	-7.9	-9,456	1.25
2017	0	0	2.9	-7.8	-15.4	-10,629	0.67
2020	0	0	2.1	-10.4	-23.7	-11,624	0.04
2023	0	0	1.4	-13.2	-31.7	-12,475	-0.53

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.504a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.47%	0.01%	-1.70%	-0.03%	2.71%
2000	0.00%	0.00%	0.46%	-0.01%	-1.15%	-1.02%	2.17%
2005	0.00%	0.00%	0.68%	-0.05%	-0.78%	-1.38%	1.97%
2007	0.00%	0.00%	0.70%	-0.07%	-0.44%	-1.35%	1.90%
2010	0.00%	0.00%	0.78%	-0.10%	-0.42%	-1.39%	1.75%
2014	0.00%	0.00%	0.80%	-0.15%	-0.77%	-1.54%	1.22%
2017	0.00%	0.00%	0.72%	-0.25%	-1.87%	-1.65%	0.66%
2020	0.00%	0.00%	0.58%	-0.39%	-3.50%	-1.73%	0.04%
2023	0.00%	0.00%	0.42%	-0.56%	-5.46%	-1.77%	-0.51%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-26. Impact on Sacramento Valley Air Basin Inventory of Change 12.5

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,228	50,471,784	187.0	1924.2	204.1	30,680	10.78
2000	1,640,564	57,266,724	98.6	940.5	161.2	33,798	8.03
2005	1,987,335	71,431,800	72.1	624.5	166.7	43,732	9.52
2007	1,965,067	68,937,656	61.6	524.4	143.4	42,404	8.64
2010	2,128,728	73,600,472	52.0	427.3	121.0	44,657	8.35
2014	2,322,148	80,409,520	41.7	320.8	90.3	48,892	8.13
2017	2,460,585	85,238,240	36.2	262.0	72.0	52,055	8.05
2020	2,597,983	89,913,688	32.3	220.2	58.7	54,995	8.08
2023	2,742,237	94,758,856	29.7	192.8	50.1	58,071	8.27

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With New MHDT BERs and Phase-in
(Calculated Using EMFAC2011 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,228	50,471,784	187.9	1924.4	200.3	30,680	11.07
2000	1,640,564	57,266,724	99.1	940.4	159.8	33,423	8.23
2005	1,987,335	71,431,800	72.7	624.2	166.5	43,053	9.71
2007	1,965,067	68,937,656	62.0	524.1	143.7	41,730	8.82
2010	2,128,728	73,600,472	52.5	427.0	121.2	43,939	8.51
2014	2,322,148	80,409,528	42.1	320.3	90.0	48,029	8.24
2017	2,460,585	85,238,248	36.5	261.3	70.8	51,076	8.10
2020	2,597,983	89,913,688	32.5	219.3	56.6	53,913	8.07
2023	2,742,237	94,758,856	29.8	191.7	47.1	56,904	8.21

Difference (Ver. 2.505 - Ver. 2.504a) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.9	0.2	-3.8	-1	0.29
2000	0	0	0.5	-0.1	-1.3	-375	0.19
2005	0	0	0.5	-0.3	-0.2	-680	0.19
2007	0	0	0.5	-0.3	0.2	-674	0.18
2010	0	0	0.5	-0.4	0.2	-717	0.15
2014	0	8	0.4	-0.5	-0.3	-864	0.10
2017	0	8	0.3	-0.7	-1.2	-979	0.05
2020	0	0	0.2	-0.9	-2.1	-1,082	-0.01
2023	0	0	0.2	-1.1	-3.0	-1,167	-0.05

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.504a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.47%	0.01%	-1.86%	0.00%	2.67%
2000	0.00%	0.00%	0.55%	-0.01%	-0.84%	-1.11%	2.42%
2005	0.00%	0.00%	0.74%	-0.05%	-0.09%	-1.55%	2.01%
2007	0.00%	0.00%	0.80%	-0.06%	0.15%	-1.59%	2.03%
2010	0.00%	0.00%	0.89%	-0.09%	0.20%	-1.61%	1.83%
2014	0.00%	0.00%	0.93%	-0.15%	-0.36%	-1.77%	1.26%
2017	0.00%	0.00%	0.86%	-0.25%	-1.64%	-1.88%	0.62%
2020	0.00%	0.00%	0.72%	-0.40%	-3.63%	-1.97%	-0.06%
2023	0.00%	0.00%	0.55%	-0.59%	-6.00%	-2.01%	-0.64%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-27. Impact on San Diego Air Basin Inventory of Change 12.5

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,505	65,249,804	198.8	2152.8	206.0	39,690	8.43
2000	1,930,488	74,567,480	96.5	1006.5	147.6	41,425	7.52
2005	2,229,147	87,943,704	64.2	655.9	122.9	50,220	8.69
2007	2,268,776	87,223,976	55.6	568.6	108.9	49,718	8.44
2010	2,329,644	86,948,128	45.6	452.8	89.3	49,336	8.23
2014	2,454,384	90,207,336	37.2	345.7	68.9	51,143	8.28
2017	2,552,306	93,440,464	33.1	289.6	57.3	52,884	8.38
2020	2,654,140	96,987,712	30.2	249.3	48.8	54,753	8.55
2023	2,749,925	100,077,350	28.2	222.6	42.7	56,764	8.78

San Diego Summer Episodic On-Road Motor Vehicle Inventories With New MHDT BERs and Phase-in
(Calculated Using EMFAC2011 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,505	65,249,804	199	2153	203	39,643	8.67
2000	1,930,488	74,567,480	97	1006	147	41,158	7.68
2005	2,229,147	87,943,704	65	656	123	49,749	8.87
2007	2,268,776	87,223,976	56	568	110	49,233	8.60
2010	2,329,644	86,948,128	46	452	90	48,846	8.36
2014	2,454,384	90,207,336	38	345	70	50,603	8.36
2017	2,552,306	93,440,464	33	289	58	52,306	8.42
2020	2,654,140	96,987,712	30	248	49	54,142	8.54
2023	2,749,925	100,077,350	28	222	43	56,132	8.74

Difference (Ver. 2.505 - Ver. 2.504a) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.7	0.1	-2.8	-47	0.24
2000	0	0	0.5	-0.1	-0.9	-267	0.17
2005	0	0	0.5	-0.3	0.2	-471	0.18
2007	0	0	0.5	-0.4	0.6	-485	0.16
2010	0	0	0.4	-0.4	0.8	-490	0.13
2014	0	0	0.4	-0.5	0.8	-540	0.08
2017	0	0	0.3	-0.6	0.5	-578	0.04
2020	0	0	0.2	-0.8	0.2	-611	-0.01
2023	0	0	0.2	-1.0	0.0	-632	-0.04

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.504a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.35%	0.00%	-1.36%	-0.12%	2.85%
2000	0.00%	0.00%	0.49%	-0.01%	-0.58%	-0.65%	2.21%
2005	0.00%	0.00%	0.79%	-0.05%	0.19%	-0.94%	2.06%
2007	0.00%	0.00%	0.87%	-0.06%	0.53%	-0.98%	1.93%
2010	0.00%	0.00%	0.96%	-0.09%	0.91%	-0.99%	1.62%
2014	0.00%	0.00%	0.96%	-0.15%	1.11%	-1.06%	1.02%
2017	0.00%	0.00%	0.87%	-0.22%	0.91%	-1.09%	0.46%
2020	0.00%	0.00%	0.72%	-0.32%	0.48%	-1.12%	-0.07%
2023	0.00%	0.00%	0.58%	-0.43%	-0.02%	-1.11%	-0.50%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-28. Impact on San Francisco Bay Air Basin Inventory of Change 12.5

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,775	132,557,540	430.7	4409.2	436.5	73,379	16.74
2000	4,710,441	154,959,550	228.9	2183.4	303.5	87,398	14.92
2005	4,931,748	163,791,250	147.9	1322.3	244.2	93,989	15.92
2007	5,036,958	164,750,560	128.0	1126.9	216.0	94,268	15.66
2010	5,292,343	170,504,560	105.1	896.9	177.8	97,377	15.66
2014	5,653,678	180,541,380	83.0	665.5	131.1	102,380	15.79
2017	5,903,513	187,522,510	72.2	545.5	105.6	107,075	16.07
2020	6,145,010	194,476,900	64.1	455.5	86.9	110,708	16.36
2023	6,396,572	201,903,140	58.4	393.5	74.1	114,571	16.76

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated MHDT BERs and Phase-in
(Calculated Using EMFAC2011 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,775	132,557,540	432.7	4409.5	428.0	73,171	17.40
2000	4,710,441	154,959,550	230.1	2183.1	301.0	86,687	15.35
2005	4,931,748	163,791,250	149.0	1321.8	244.5	93,002	16.28
2007	5,036,958	164,750,560	129.0	1126.2	217.0	93,255	15.99
2010	5,292,343	170,504,580	106.1	896.1	179.1	96,332	15.92
2014	5,653,678	180,541,380	83.8	664.5	132.2	101,220	15.95
2017	5,903,513	187,522,510	72.8	544.2	106.1	105,834	16.13
2020	6,145,010	194,476,900	64.6	453.9	86.7	109,394	16.33
2023	6,396,572	201,903,140	58.7	391.6	73.3	113,194	16.66

Difference (Ver. 2.505 - Ver. 2.504a) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	1.9	0.3	-8.4	-208	0.66
2000	0	0	1.2	-0.3	-2.5	-711	0.43
2005	0	0	1.0	-0.6	0.3	-987	0.36
2007	0	0	1.0	-0.7	1.0	-1,013	0.33
2010	0	20	0.9	-0.8	1.4	-1,045	0.27
2014	0	0	0.7	-1.0	1.1	-1,160	0.16
2017	0	0	0.6	-1.3	0.5	-1,241	0.06
2020	0	0	0.5	-1.6	-0.2	-1,314	-0.03
2023	0	0	0.4	-2.0	-0.8	-1,377	-0.10

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.504a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.45%	0.01%	-1.94%	-0.28%	3.96%
2000	0.00%	0.00%	0.52%	-0.01%	-0.81%	-0.81%	2.88%
2005	0.00%	0.00%	0.70%	-0.04%	0.13%	-1.05%	2.28%
2007	0.00%	0.00%	0.77%	-0.06%	0.46%	-1.07%	2.12%
2010	0.00%	0.00%	0.86%	-0.09%	0.76%	-1.07%	1.70%
2014	0.00%	0.00%	0.90%	-0.15%	0.84%	-1.13%	1.00%
2017	0.00%	0.00%	0.83%	-0.24%	0.49%	-1.16%	0.40%
2020	0.00%	0.00%	0.72%	-0.36%	-0.20%	-1.19%	-0.15%
2023	0.00%	0.00%	0.60%	-0.50%	-1.07%	-1.20%	-0.59%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-29. Impact on San Joaquin Valley Air Basin Inventory of Change 12.5

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,280	58,324,240	220.0	2295.9	336.1	42,187	24.89
2000	1,943,272	77,177,536	137.6	1329.0	334.8	53,215	17.96
2005	2,389,193	98,949,512	106.5	911.9	398.6	71,973	21.01
2007	2,407,275	97,362,808	92.1	779.7	351.6	70,282	18.71
2010	2,600,200	103,175,860	78.0	637.4	295.4	73,320	17.07
2014	2,830,493	113,057,020	61.8	480.9	219.2	80,735	15.47
2017	3,012,243	121,296,510	52.8	394.2	172.8	86,967	14.60
2020	3,203,419	129,483,600	46.6	334.8	139.7	93,173	14.16
2023	3,406,514	137,546,340	42.9	300.0	119.7	100,580	14.32

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With new MHDT BERs and Phase-in
(Calculated Using EMFAC2011 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,280	58,324,240	221.5	2296.1	330.5	42,412	25.36
2000	1,943,272	77,177,536	138.5	1328.8	332.5	52,477	18.31
2005	2,389,193	98,949,512	107.5	911.4	398.1	70,456	21.34
2007	2,407,275	97,362,808	93.1	779.1	351.8	68,755	19.03
2010	2,600,200	103,175,860	78.8	636.7	295.4	71,728	17.38
2014	2,830,493	113,057,020	62.5	480.0	217.2	78,777	15.69
2017	3,012,243	121,296,520	53.4	393.0	168.1	84,689	14.71
2020	3,203,419	129,483,600	46.9	332.9	132.0	90,593	14.15
2023	3,406,514	137,546,340	43.1	297.5	109.4	97,767	14.20

Difference (Ver. 2.505 - Ver. 2.504a) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	1.5	0.2	-5.6	224	0.48
2000	0	0	1.0	-0.2	-2.3	-738	0.35
2005	0	0	1.0	-0.5	-0.5	-1,517	0.33
2007	0	0	0.9	-0.6	0.2	-1,527	0.32
2010	0	0	0.9	-0.7	-0.1	-1,592	0.31
2014	0	0	0.7	-0.8	-2.0	-1,958	0.22
2017	0	10	0.5	-1.2	-4.7	-2,278	0.11
2020	0	0	0.4	-1.8	-7.7	-2,579	-0.01
2023	0	0	0.2	-2.5	-10.3	-2,813	-0.12

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.504a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.66%	0.01%	-1.66%	0.53%	1.91%
2000	0.00%	0.00%	0.71%	-0.02%	-0.69%	-1.39%	1.92%
2005	0.00%	0.00%	0.92%	-0.06%	-0.12%	-2.11%	1.58%
2007	0.00%	0.00%	0.99%	-0.08%	0.06%	-2.17%	1.73%
2010	0.00%	0.00%	1.10%	-0.11%	-0.03%	-2.17%	1.80%
2014	0.00%	0.00%	1.14%	-0.17%	-0.93%	-2.43%	1.41%
2017	0.00%	0.00%	1.01%	-0.31%	-2.73%	-2.62%	0.75%
2020	0.00%	0.00%	0.76%	-0.55%	-5.49%	-2.77%	-0.09%
2023	0.00%	0.00%	0.45%	-0.83%	-8.61%	-2.80%	-0.85%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-30. Impact on South Coast Air Basin Inventory of Change 12.5

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.504a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,380	282,562,270	876.2	8974.7	915.0	165,412	40.60
2000	8,884,540	323,010,620	440.3	4364.2	668.4	182,611	34.19
2005	10,519,562	393,767,940	305.9	2952.0	617.2	232,450	42.30
2007	9,971,575	367,746,560	232.9	2264.3	463.3	213,120	36.60
2010	10,473,359	377,735,330	188.1	1793.4	391.1	217,869	36.63
2014	11,179,501	393,461,060	152.2	1377.6	298.8	230,138	36.90
2017	11,582,747	403,020,260	133.6	1143.5	242.4	237,893	36.93
2020	11,961,135	414,267,300	119.7	966.4	199.3	244,742	37.06
2023	12,242,366	422,568,670	109.0	835.0	170.9	251,932	37.47

South Coast Summer Episodic On-Road Motor Vehicle Inventories With new MHDT BERs and Phase-in
(Calculated Using EMFAC2011 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,380	282,562,270	880.6	8975.3	897.2	165,057	42.04
2000	8,884,540	323,010,620	442.0	4363.5	656.0	180,651	34.99
2005	10,519,562	393,767,940	307.8	2950.2	605.5	229,571	43.29
2007	9,971,575	367,746,560	234.2	2262.6	456.5	210,709	37.35
2010	10,473,359	377,735,330	189.3	1791.5	385.8	215,245	37.30
2014	11,179,501	393,461,060	153.2	1375.3	295.3	227,114	37.35
2017	11,582,747	403,020,290	134.4	1140.5	237.8	234,559	37.20
2020	11,961,135	414,267,300	120.3	962.6	193.6	241,178	37.12
2023	12,242,366	422,568,670	109.3	830.5	163.9	248,143	37.34

Difference (Ver. 2.505 - Ver. 2.504a) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	4.4	0.6	-17.8	-355	1.44
2000	0	0	1.6	-0.7	-12.4	-1,960	0.80
2005	0	0	1.9	-1.8	-11.7	-2,879	1.00
2007	0	0	1.4	-1.7	-6.8	-2,411	0.75
2010	0	0	1.2	-1.9	-5.3	-2,624	0.67
2014	0	0	1.0	-2.3	-3.5	-3,024	0.45
2017	0	30	0.8	-3.0	-4.6	-3,334	0.27
2020	0	0	0.5	-3.7	-5.7	-3,564	0.06
2023	0	0	0.3	-4.6	-7.1	-3,789	-0.13

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.504a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.50%	0.01%	-1.95%	-0.21%	3.56%
2000	0.00%	0.00%	0.37%	-0.02%	-1.85%	-1.07%	2.34%
2005	0.00%	0.00%	0.62%	-0.06%	-1.90%	-1.24%	2.36%
2007	0.00%	0.00%	0.58%	-0.07%	-1.47%	-1.13%	2.04%
2010	0.00%	0.00%	0.64%	-0.11%	-1.36%	-1.20%	1.82%
2014	0.00%	0.00%	0.65%	-0.17%	-1.16%	-1.31%	1.22%
2017	0.00%	0.00%	0.58%	-0.26%	-1.91%	-1.40%	0.73%
2020	0.00%	0.00%	0.45%	-0.39%	-2.87%	-1.46%	0.15%
2023	0.00%	0.00%	0.31%	-0.55%	-4.14%	-1.50%	-0.36%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.6 HEAVY DUTY T6/T7 EXPANSION

This update was made to EMFAC but ultimately not used in EMFAC2011-LDV because MHDDT and HHDDT emissions are generated from EMFAC2011-HD.

Table 12-31. Impact on Statewide Inventory of Change 12.6

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,589,834	657,667,390	2,630	26,244	2,657	406,902	134.46
2000	22,238,334	799,849,410	1,199	11,613	1,960	464,900	104.06
2005	25,779,274	955,234,500	845	7,675	1,947	579,553	123.44
2007	25,431,556	930,013,120	697	6,291	1,596	556,330	110.11
2010	26,905,730	976,336,700	567	5,013	1,321	578,421	106.84
2014	28,886,172	1,052,095,200	423	3,647	968	625,339	104.28
2017	30,222,932	1,103,817,200	341	2,852	754	660,104	102.76
2020	31,543,150	1,153,718,100	283	2,294	600	690,508	102.15
2023	32,780,034	1,198,555,400	251	1,929	502	722,345	103.21

Statewide Summer Episodic On-Road Motor Vehicle Inventories With New T6/T7 Categories/Pops/Accruals
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,665,808	693,786,560	2,317	23,358	2,559	422,428	125.28
2000	22,316,218	804,194,820	1,201	11,513	2,061	471,882	92.63
2005	25,801,910	950,032,770	829	7,541	1,717	566,290	106.55
2007	25,453,716	919,583,940	684	6,209	1,375	543,289	97.23
2010	26,930,604	953,876,160	564	4,985	1,111	558,556	95.50
2014	28,913,014	1,008,486,200	458	3,800	862	589,623	96.10
2017	30,251,150	1,047,308,500	405	3,153	731	612,246	97.06
2020	31,572,478	1,088,821,100	366	2,678	633	633,705	98.15
2023	32,810,362	1,128,081,400	338	2,345	573	659,453	100.23

Difference (Ver. 2.507e - Ver. 2.506) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	75,974	36,119,170	-312.5	-2886.3	-98.3	15,526	-9.18
2000	77,884	4,345,410	2.0	-100.4	101.5	6,982	-11.43
2005	22,636	-5,201,730	-16.3	-134.7	-230.3	-13,263	-16.89
2007	22,160	-10,429,180	-13.7	-81.9	-220.1	-13,041	-12.88
2010	24,874	-22,460,540	-2.4	-27.9	-210.7	-19,865	-11.34
2014	26,842	-43,609,000	35.0	152.5	-106.7	-35,716	-8.18
2017	28,218	-56,508,700	63.4	301.3	-23.3	-47,857	-5.70
2020	29,328	-64,897,000	82.5	383.6	33.2	-56,804	-4.00
2023	30,328	-70,474,000	86.9	416.7	71.1	-62,891	-2.99

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.506)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.41%	5.49%	-11.88%	-11.00%	-3.70%	3.82%	-6.83%
2000	0.35%	0.54%	0.17%	-0.86%	5.18%	1.50%	-10.98%
2005	0.09%	-0.54%	-1.92%	-1.75%	-11.83%	-2.29%	-13.68%
2007	0.09%	-1.12%	-1.96%	-1.30%	-13.79%	-2.34%	-11.70%
2010	0.09%	-2.30%	-0.43%	-0.56%	-15.94%	-3.43%	-10.61%
2014	0.09%	-4.14%	8.27%	4.18%	-11.02%	-5.71%	-7.84%
2017	0.09%	-5.12%	18.58%	10.57%	-3.08%	-7.25%	-5.54%
2020	0.09%	-5.63%	29.13%	16.72%	5.53%	-8.23%	-3.92%
2023	0.09%	-5.88%	34.66%	21.61%	14.17%	-8.71%	-2.89%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-32. Impact on Sacramento Valley Air Basin Inventory of Change 12.6

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,117	47,687,332	214.5	2140.3	211.1	29,814	11.17
2000	1,640,564	57,266,724	99.0	939.7	159.1	33,549	8.19
2005	1,987,335	71,431,800	72.5	623.3	165.1	43,386	9.67
2007	1,965,067	69,355,912	62.2	525.0	141.5	42,248	8.77
2010	2,128,723	74,675,936	52.4	431.6	118.2	44,753	8.47
2014	2,322,124	82,988,688	39.5	316.7	86.3	49,564	8.26
2017	2,460,544	88,857,680	31.7	245.5	66.8	53,133	8.16
2020	2,597,919	94,183,600	26.1	194.6	52.7	56,346	8.16
2023	2,742,155	99,436,240	23.1	162.2	43.5	59,591	8.32

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With New T6/T7 Categories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,394,479	50,672,340	187.1	1916.5	203.6	31,075	10.27
2000	1,649,156	57,586,440	97.5	926.4	165.8	33,955	7.03
2005	1,990,785	70,935,288	69.8	608.7	142.8	42,016	8.20
2007	1,968,651	68,579,312	59.4	510.9	119.1	40,956	7.62
2010	2,132,849	73,205,224	50.1	415.2	96.8	43,030	7.49
2014	2,326,865	79,631,664	40.4	312.2	74.1	46,439	7.53
2017	2,465,686	84,103,120	35.4	255.7	62.2	48,852	7.60
2020	2,603,439	88,483,016	31.8	215.7	53.4	51,171	7.70
2023	2,748,041	93,168,168	29.4	189.3	47.6	53,890	7.90

Difference (Ver. 2.507e - Ver. 2.506) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	8362	2985008	-27.5	-223.7	-7.5	1,262	-0.90
2000	8592	319716	-1.5	-13.3	6.7	406	-1.16
2005	3450	-496512	-2.7	-14.6	-22.3	-1,370	-1.47
2007	3584	-776600	-2.8	-14.2	-22.3	-1,291	-1.15
2010	4126	-1470712	-2.3	-16.4	-21.4	-1,723	-0.97
2014	4741	-3357024	0.9	-4.5	-12.2	-3,124	-0.73
2017	5142	-4754560	3.7	10.2	-4.6	-4,281	-0.57
2020	5520	-5700584	5.7	21.1	0.8	-5,176	-0.47
2023	5886	-6268072	6.3	27.1	4.1	-5,701	-0.41

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.506)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.60%	6.26%	-12.80%	-10.45%	-3.57%	4.23%	-8.03%
2000	0.52%	0.56%	-1.55%	-1.41%	4.24%	1.21%	-14.14%
2005	0.17%	-0.70%	-3.72%	-2.34%	-13.52%	-3.16%	-15.24%
2007	0.18%	-1.12%	-4.48%	-2.70%	-15.79%	-3.06%	-13.08%
2010	0.19%	-1.97%	-4.38%	-3.81%	-18.11%	-3.85%	-11.48%
2014	0.20%	-4.05%	2.26%	-1.44%	-14.11%	-6.30%	-8.83%
2017	0.21%	-5.35%	11.72%	4.17%	-6.84%	-8.06%	-6.93%
2020	0.21%	-6.05%	21.75%	10.84%	1.48%	-9.19%	-5.72%
2023	0.21%	-6.30%	27.07%	16.73%	9.32%	-9.57%	-4.96%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-33. Impact on San Diego Air Basin Inventory of Change 12.6

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,475	61,840,940	234.4	2457.4	216.3	38,565	8.63
2000	1,930,488	74,567,480	96.9	1006.1	146.4	41,243	7.67
2005	2,229,147	87,943,704	64.7	655.2	122.6	49,951	8.85
2007	2,268,776	88,395,248	55.5	560.6	107.5	49,950	8.60
2010	2,329,644	89,562,128	44.2	436.1	86.5	50,210	8.39
2014	2,454,383	94,253,912	33.0	314.8	64.4	52,660	8.42
2017	2,552,306	98,096,680	27.1	248.4	51.9	54,685	8.48
2020	2,654,140	101,946,790	23.1	203.8	43.1	56,708	8.60
2023	2,749,926	105,317,070	20.8	175.4	37.2	58,877	8.80

San Diego Summer Episodic On-Road Motor Vehicle Inventories With New T6/T7 Categories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,624,564	65,528,924	200	2158	209	40,173	8.55
2000	1,938,446	74,991,368	96	1001	156	41,974	7.32
2005	2,234,307	87,936,400	64	649	119	49,825	8.31
2007	2,273,919	87,207,744	55	563	103	49,287	8.14
2010	2,334,764	86,859,984	45	448	82	48,730	7.94
2014	2,459,716	89,958,456	37	343	64	50,200	8.01
2017	2,557,805	93,097,080	33	287	54	51,737	8.11
2020	2,659,812	96,587,824	30	247	47	53,482	8.24
2023	2,755,727	99,663,704	28	221	41	55,458	8.44

Difference (Ver. 2.507e - Ver. 2.506) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,089	3,687,984	-34.6	-299.8	-7.7	1,608	-0.08
2000	7,958	423,888	-0.5	-5.3	9.6	731	-0.35
2005	5,160	-7,304	-1.1	-5.9	-3.7	-126	-0.54
2007	5,143	-1,187,504	-0.4	2.3	-4.3	-663	-0.47
2010	5,120	-2,702,144	1.0	12.0	-4.1	-1,480	-0.45
2014	5,333	-4,295,456	3.9	27.7	-0.6	-2,460	-0.41
2017	5,499	-4,999,600	5.9	38.8	2.0	-2,948	-0.37
2020	5,672	-5,358,966	7.1	43.5	3.4	-3,225	-0.36
2023	5,801	-5,653,366	7.4	45.5	4.1	-3,420	-0.37

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.506)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.44%	5.96%	-14.76%	-12.20%	-3.57%	4.17%	-0.87%
2000	0.41%	0.57%	-0.54%	-0.52%	6.53%	1.77%	-4.53%
2005	0.23%	-0.01%	-1.63%	-0.90%	-3.03%	-0.25%	-6.10%
2007	0.23%	-1.34%	-0.76%	0.40%	-4.01%	-1.33%	-5.44%
2010	0.22%	-3.02%	2.37%	2.76%	-4.69%	-2.95%	-5.36%
2014	0.22%	-4.56%	11.95%	8.81%	-0.98%	-4.67%	-4.85%
2017	0.22%	-5.10%	21.85%	15.61%	3.87%	-5.39%	-4.39%
2020	0.21%	-5.26%	30.73%	21.34%	7.86%	-5.69%	-4.22%
2023	0.21%	-5.37%	35.60%	25.97%	11.01%	-5.81%	-4.16%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-34. Impact on San Francisco Bay Air Basin Inventory of Change 12.6

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,750	126,570,600	508.8	5091.7	463.8	71,905	17.51
2000	4,710,441	154,959,550	230.0	2182.5	300.3	86,913	15.31
2005	4,931,748	163,791,250	148.8	1321.0	243.3	93,415	16.25
2007	5,036,958	165,888,140	128.3	1120.2	213.4	94,212	15.94
2010	5,292,342	173,551,680	102.8	881.3	173.2	98,153	15.90
2014	5,653,678	186,284,220	73.7	615.5	123.7	104,269	15.96
2017	5,903,514	194,840,240	57.9	466.3	96.2	109,720	16.14
2020	6,145,010	202,564,850	47.3	364.0	76.9	113,701	16.32
2023	6,396,570	210,436,450	42.0	306.0	64.7	117,744	16.63

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With New T6/T7 Categories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,948,878	133,723,100	479.8	4458.4	461.3	76,222	19.70
2000	4,724,726	156,271,180	248.3	2208.1	339.1	89,810	15.82
2005	4,936,224	164,056,700	163.5	1338.5	249.1	94,241	15.98
2007	5,041,512	165,108,000	141.1	1142.2	217.1	94,645	15.76
2010	5,296,710	170,781,230	116.2	912.3	174.2	97,406	15.72
2014	5,657,796	180,488,980	93.2	684.1	134.4	101,805	15.91
2017	5,907,515	187,260,050	82.0	565.5	113.4	106,078	16.19
2020	6,148,934	194,075,760	73.7	476.6	97.7	109,433	16.42
2023	6,400,411	201,447,360	67.8	415.5	87.0	113,179	16.76

Difference (Ver. 2.507e - Ver. 2.506) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	14,128	7,152,500	-29.0	-633.3	-2.5	4,317	2.19
2000	14,285	1,311,630	18.3	25.6	38.7	2,897	0.50
2005	4,476	265,450	14.7	17.5	5.8	826	-0.27
2007	4,554	-780,140	12.8	21.9	3.7	433	-0.19
2010	4,368	-2,770,450	13.3	31.0	1.0	-748	-0.17
2014	4,118	-5,795,240	19.5	68.6	10.7	-2,464	-0.05
2017	4,001	-7,580,190	24.2	99.2	17.3	-3,642	0.05
2020	3,924	-8,489,090	26.4	112.6	20.8	-4,268	0.10
2023	3,841	-8,989,090	25.8	109.5	22.3	-4,565	0.13

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.506)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.36%	5.65%	-5.70%	-12.44%	-0.55%	6.00%	12.48%
2000	0.30%	0.85%	7.95%	1.17%	12.90%	3.33%	3.28%
2005	0.09%	0.16%	9.85%	1.33%	2.39%	0.88%	-1.64%
2007	0.09%	-0.47%	9.97%	1.96%	1.72%	0.46%	-1.18%
2010	0.08%	-1.60%	12.97%	3.52%	0.58%	-0.76%	-1.10%
2014	0.07%	-3.11%	26.44%	11.15%	8.63%	-2.36%	-0.34%
2017	0.07%	-3.89%	41.77%	21.27%	17.97%	-3.32%	0.30%
2020	0.06%	-4.19%	55.88%	30.93%	27.06%	-3.75%	0.64%
2023	0.06%	-4.27%	61.35%	35.80%	34.42%	-3.88%	0.75%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-35. Impact on San Joaquin Valley Air Basin Inventory of Change 12.6

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,175	55,468,272	247.8	2537.4	341.2	41,131	25.39
2000	1,943,272	77,177,536	138.2	1327.0	330.1	52,748	18.20
2005	2,389,193	98,949,512	107.1	909.0	394.0	71,319	21.23
2007	2,407,275	98,375,648	92.5	774.0	346.4	70,076	18.94
2010	2,600,200	105,634,770	77.2	629.3	288.9	73,651	17.34
2014	2,830,493	117,297,570	57.9	462.1	210.1	81,724	15.74
2017	3,012,242	126,354,730	46.4	363.2	160.8	88,275	14.79
2020	3,203,419	134,943,280	38.6	295.9	125.1	94,639	14.25
2023	3,406,514	143,350,660	34.3	254.6	103.1	102,241	14.30

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With New T6/T7 Categories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,405,114	58,580,652	219.0	2299.7	331.9	42,756	22.06
2000	1,953,670	77,852,040	133.2	1295.6	344.3	53,500	14.06
2005	2,389,626	97,397,040	98.3	869.2	320.3	67,037	16.20
2007	2,408,237	96,145,336	84.4	741.6	268.3	66,019	14.80
2010	2,601,884	101,880,540	71.1	604.4	212.5	68,742	13.76
2014	2,832,668	110,655,900	57.2	456.9	160.1	73,869	13.15
2017	3,014,826	117,719,150	49.7	376.5	133.8	77,712	12.92
2020	3,206,450	124,790,200	44.6	321.3	114.8	81,640	12.84
2023	3,410,038	132,194,620	41.5	288.6	103.3	87,649	13.15

Difference (Ver. 2.507e - Ver. 2.506) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	9,939	3,112,380	-28.9	-237.8	-9.3	1,626	-3.33
2000	10,398	674,504	-5.0	-31.5	14.1	752	-4.14
2005	433	-1,552,472	-8.8	-39.8	-73.7	-4,282	-5.03
2007	962	-2,230,312	-8.1	-32.4	-78.0	-4,057	-4.14
2010	1,684	-3,754,230	-6.0	-24.9	-76.4	-4,909	-3.58
2014	2,175	-6,641,670	-0.7	-5.2	-50.0	-7,855	-2.59
2017	2,584	-8,635,580	3.3	13.2	-27.0	-10,564	-1.87
2020	3,031	-10,153,080	6.1	25.4	-10.4	-12,999	-1.41
2023	3,524	-11,156,040	7.2	34.0	0.1	-14,591	-1.15

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.506)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.71%	5.61%	-11.65%	-9.37%	-2.72%	3.95%	-13.10%
2000	0.54%	0.87%	-3.64%	-2.37%	4.28%	1.42%	-22.76%
2005	0.02%	-1.57%	-8.22%	-4.38%	-18.71%	-6.00%	-23.70%
2007	0.04%	-2.27%	-8.77%	-4.18%	-22.53%	-5.79%	-21.87%
2010	0.06%	-3.55%	-7.79%	-3.95%	-26.45%	-6.67%	-20.66%
2014	0.08%	-5.66%	-1.27%	-1.13%	-23.79%	-9.61%	-16.45%
2017	0.09%	-6.83%	7.13%	3.64%	-16.79%	-11.97%	-12.66%
2020	0.09%	-7.52%	15.71%	8.57%	-8.29%	-13.73%	-9.86%
2023	0.10%	-7.78%	21.01%	13.37%	0.14%	-14.27%	-8.04%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-36. Impact on South Coast Air Basin Inventory of Change 12.6

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,190	269,279,940	1017.9	10012.5	956.3	161,692	42.44
2000	8,884,540	323,010,620	441.7	4362.0	654.3	181,129	34.91
2005	10,519,562	393,767,940	307.5	2948.3	602.5	230,707	43.21
2007	9,971,575	369,926,690	233.5	2250.4	449.6	212,629	37.23
2010	10,473,358	384,491,940	184.3	1757.5	373.5	219,250	37.19
2014	11,179,501	407,362,660	137.7	1281.0	278.4	234,650	37.37
2017	11,582,748	420,267,420	112.2	1009.6	220.1	243,918	37.24
2020	11,961,132	433,515,070	94.1	817.5	176.9	251,624	37.16
2023	12,242,366	443,030,180	82.8	683.1	148.9	259,363	37.37

South Coast Summer Episodic On-Road Motor Vehicle Inventories With New T6/T7 Categories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,587,541	284,337,980	882.2	8983.9	935.1	168,928	41.96
2000	8,926,638	325,417,540	439.9	4337.8	708.1	185,478	34.06
2005	10,551,385	393,939,550	301.0	2913.0	583.1	230,533	40.55
2007	9,997,372	368,084,260	228.8	2235.2	430.8	211,870	35.37
2010	10,501,053	378,033,630	184.3	1766.7	356.3	216,185	35.53
2014	11,209,772	392,913,630	150.6	1360.9	285.1	226,596	36.46
2017	11,614,586	401,614,560	133.7	1134.1	246.0	232,508	37.06
2020	11,994,158	412,199,680	121.0	962.3	215.6	237,963	37.53
2023	12,277,238	420,057,220	111.2	835.0	199.5	244,167	38.17

Difference (Ver. 2.507e - Ver. 2.506) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	40,351	15,058,040	-135.6	-1028.5	-21.3	7,236	-0.49
2000	42,098	2,406,920	-1.8	-24.2	53.8	4,349	-0.85
2005	31,823	171,610	-6.4	-35.3	-19.4	-174	-2.66
2007	25,797	-1,842,430	-4.6	-15.2	-18.8	-760	-1.86
2010	27,695	-6,458,310	0.0	9.1	-17.3	-3,065	-1.66
2014	30,271	-14,449,030	12.9	79.9	6.7	-8,053	-0.91
2017	31,838	-18,652,860	21.5	124.5	25.9	-11,410	-0.19
2020	33,026	-21,315,390	26.9	144.8	38.7	-13,661	0.38
2023	34,872	-22,972,960	28.4	151.9	50.6	-15,195	0.80

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.506)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.53%	5.59%	-13.33%	-10.27%	-2.22%	4.48%	-1.15%
2000	0.47%	0.75%	-0.40%	-0.56%	8.22%	2.40%	-2.43%
2005	0.30%	0.04%	-2.08%	-1.20%	-3.23%	-0.08%	-6.16%
2007	0.26%	-0.50%	-1.98%	-0.68%	-4.18%	-0.36%	-4.99%
2010	0.26%	-1.68%	0.01%	0.52%	-4.62%	-1.40%	-4.47%
2014	0.27%	-3.55%	9.35%	6.24%	2.39%	-3.43%	-2.43%
2017	0.27%	-4.44%	19.18%	12.33%	11.77%	-4.68%	-0.51%
2020	0.28%	-4.92%	28.56%	17.71%	21.90%	-5.43%	1.01%
2023	0.28%	-5.19%	34.24%	22.24%	33.97%	-5.86%	2.14%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.7 NEW SURVIVAL RATES AND LIFETIME MILEAGE CALCULATIONS FOR MOTOR VEHICLES

Please refer to Section 3.3.4.3. This update includes two distinct changes, only one of which made its way into the final model. There are two separate effects. First, originally staff had planned to update survival rates for all vehicle categories, and this change was made here. The survival rates tend to make the fleet younger into the future. Ultimately, these survival rates were removed and only two were updated as described in Section 3.3.4.2. That change is discussed later in this section. Second, the new lifetime mileage calculation tends to reduce the odometer and therefore reduces the emissions. The first effect can be seen in the VMT. There is a slight increase because newer vehicles travel more. CO2 follows the VMT because there is not a strong age dependency, and there are no deterioration rates. The second effect is much more difficult to observe. The lower odometer values will reduce emissions, but the amount of production is dependent upon the smog check program in place and the area-specific accrual rates.

Table 12-37. Impact on Statewide Inventory of Change 12.7

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,590,372	691,048,900	2,278	23,342	2,501	414,325	133.12
2000	22,238,334	799,849,410	1,199	11,613	1,960	464,900	104.06
2005	25,779,274	955,234,500	845	7,675	1,947	579,553	123.44
2007	25,431,560	923,189,120	698	6,315	1,607	553,343	110.21
2010	26,905,742	958,080,320	578	5,074	1,347	570,532	106.85
2014	28,886,264	1,016,926,700	465	3,851	1,006	610,197	103.95
2017	30,223,104	1,059,726,400	405	3,179	797	640,847	102.33
2020	31,543,406	1,104,525,600	361	2,684	643	668,884	101.71
2023	32,780,384	1,145,927,000	330	2,338	540	699,032	102.79

Statewide Summer Episodic On-Road Motor Vehicle Inventories With New Survival Rates
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,589,834	657,667,390	2,630	26,244	2,657	406,902	134.46
2000	22,238,334	799,849,410	1,199	11,613	1,960	464,900	104.06
2005	25,779,274	955,234,500	845	7,675	1,947	579,553	123.44
2007	25,431,556	930,013,120	697	6,291	1,596	556,330	110.11
2010	26,905,730	976,336,700	567	5,013	1,321	578,421	106.84
2014	28,886,172	1,052,095,200	423	3,647	968	625,339	104.28
2017	30,222,932	1,103,817,200	341	2,852	754	660,104	102.76
2020	31,543,150	1,153,718,100	283	2,294	600	690,508	102.15
2023	32,780,034	1,198,555,400	251	1,929	502	722,345	103.21

Difference (Ver. 2.506 - Ver. 2.505) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-538	-33,381,510	352.2	2902.2	156.6	-7,423	1.34
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	-4	6,824,000	-0.8	-24.1	-11.9	2,987	-0.10
2010	-12	18,256,380	-11.2	-61.1	-25.2	7,889	-0.01
2014	-92	35,168,500	-41.4	-204.1	-37.8	15,142	0.33
2017	-172	44,090,800	-64.0	-327.2	-43.2	19,257	0.43
2020	-256	49,192,500	-77.4	-389.9	-43.0	21,624	0.44
2023	-350	52,628,400	-79.0	-409.7	-38.8	23,312	0.42

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.505)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	-4.83%	15.46%	12.43%	6.26%	-1.79%	1.01%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.74%	-0.12%	-0.38%	-0.74%	0.54%	-0.09%
2010	0.00%	1.91%	-1.93%	-1.20%	-1.87%	1.38%	-0.01%
2014	0.00%	3.46%	-8.91%	-5.30%	-3.76%	2.48%	0.32%
2017	0.00%	4.16%	-15.79%	-10.29%	-5.41%	3.00%	0.42%
2020	0.00%	4.45%	-21.45%	-14.53%	-6.68%	3.23%	0.43%
2023	0.00%	4.59%	-23.95%	-17.52%	-7.17%	3.33%	0.41%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-38. Impact on Sacramento Valley Air Basin Inventory of Change 12.7

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,228	50,471,784	187.7	1923.6	199.8	30,480	11.00
2000	1,640,564	57,266,724	99.0	939.7	159.1	33,549	8.19
2005	1,987,335	71,431,800	72.5	623.3	165.1	43,386	9.67
2007	1,965,067	68,937,656	61.9	523.3	142.3	42,067	8.79
2010	2,128,728	73,600,472	52.4	426.2	120.0	44,300	8.48
2014	2,322,148	80,409,528	42.0	319.6	88.8	48,508	8.22
2017	2,460,585	85,238,248	36.4	260.5	69.7	51,650	8.09
2020	2,597,983	89,913,688	32.4	218.5	55.6	54,567	8.07
2023	2,742,237	94,758,856	29.7	190.9	46.2	57,622	8.21

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With New Survival Rates
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,386,117	47,687,332	214.5	2140.3	211.1	29,814	11.17
2000	1,640,564	57,266,724	99.0	939.7	159.1	33,549	8.19
2005	1,987,335	71,431,800	72.5	623.3	165.1	43,386	9.67
2007	1,965,067	69,355,912	62.2	525.0	141.5	42,248	8.77
2010	2,128,723	74,675,936	52.4	431.6	118.2	44,753	8.47
2014	2,322,124	82,988,688	39.5	316.7	86.3	49,564	8.26
2017	2,460,544	88,857,680	31.7	245.5	66.8	53,133	8.16
2020	2,597,919	94,183,600	26.1	194.6	52.7	56,346	8.16
2023	2,742,155	99,436,240	23.1	162.2	43.5	59,591	8.32

Difference (Ver. 2.506 - Ver. 2.505) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-111	-2784452	26.9	216.6	11.3	-666	0.17
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	418256	0.3	1.7	-0.8	181	-0.01
2010	-5	1075464	0.0	5.4	-1.8	453	-0.02
2014	-24	2579160	-2.5	-2.8	-2.5	1,056	0.04
2017	-41	3619432	-4.7	-15.1	-2.9	1,483	0.07
2020	-64	4269912	-6.3	-23.9	-3.0	1,779	0.10
2023	-82	4677384	-6.6	-28.7	-2.7	1,969	0.11

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.505)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-0.01%	-5.52%	14.31%	11.26%	5.67%	-2.19%	1.51%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.61%	0.49%	0.33%	-0.59%	0.43%	-0.17%
2010	0.00%	1.46%	0.02%	1.27%	-1.46%	1.02%	-0.20%
2014	0.00%	3.21%	-5.88%	-0.89%	-2.77%	2.18%	0.46%
2017	0.00%	4.25%	-13.02%	-5.79%	-4.16%	2.87%	0.92%
2020	0.00%	4.75%	-19.40%	-10.95%	-5.34%	3.26%	1.19%
2023	0.00%	4.94%	-22.22%	-15.05%	-5.93%	3.42%	1.31%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-39. Impact on San Diego Air Basin Inventory of Change 12.7

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,505	65,249,804	199.4	2152.6	203.0	39,560	8.64
2000	1,930,488	74,567,480	96.9	1006.1	146.4	41,243	7.67
2005	2,229,147	87,943,704	64.7	655.2	122.6	49,951	8.85
2007	2,268,776	87,223,976	56.1	567.9	109.0	49,448	8.59
2010	2,329,644	86,948,128	46.0	452.1	89.6	49,068	8.35
2014	2,454,384	90,207,336	37.5	344.9	69.1	50,869	8.35
2017	2,552,306	93,440,464	33.3	288.7	57.3	52,603	8.42
2020	2,654,140	96,987,712	30.4	248.2	48.6	54,464	8.54
2023	2,749,925	100,077,350	28.3	221.3	42.3	56,466	8.73

San Diego Summer Episodic On-Road Motor Vehicle Inventories With New Survival Rates
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,617,475	61,840,940	234	2457	216	38,565	8.63
2000	1,930,488	74,567,480	97	1006	146	41,243	7.67
2005	2,229,147	87,943,704	65	655	123	49,951	8.85
2007	2,268,776	88,395,248	56	561	107	49,950	8.60
2010	2,329,644	89,562,128	44	436	86	50,210	8.39
2014	2,454,383	94,253,912	33	315	64	52,660	8.42
2017	2,552,306	98,096,680	27	248	52	54,685	8.48
2020	2,654,140	101,946,790	23	204	43	56,708	8.60
2023	2,749,926	105,317,070	21	175	37	58,877	8.80

Difference (Ver. 2.506 - Ver. 2.505) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-30	-3,408,864	35.0	304.8	13.3	-995	-0.02
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	1,171,272	-0.6	-7.3	-1.5	502	0.02
2010	0	2,614,000	-1.9	-16.0	-3.2	1,142	0.04
2014	-1	4,046,576	-4.5	-30.1	-4.7	1,791	0.06
2017	0	4,656,216	-6.3	-40.3	-5.4	2,082	0.07
2020	0	4,959,078	-7.3	-44.3	-5.5	2,244	0.07
2023	1	5,239,720	-7.5	-46.0	-5.1	2,411	0.07

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.505)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	-5.22%	17.56%	14.16%	6.58%	-2.52%	-0.19%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	1.34%	-0.99%	-1.28%	-1.37%	1.02%	0.18%
2010	0.00%	3.01%	-4.08%	-3.54%	-3.54%	2.33%	0.48%
2014	0.00%	4.49%	-11.94%	-8.74%	-6.83%	3.52%	0.77%
2017	0.00%	4.98%	-18.82%	-13.96%	-9.42%	3.96%	0.79%
2020	0.00%	5.11%	-24.02%	-17.86%	-11.28%	4.12%	0.77%
2023	0.00%	5.24%	-26.57%	-20.76%	-12.08%	4.27%	0.78%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-40. Impact on San Francisco Bay Air Basin Inventory of Change 12.7

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,775	132,557,540	432.5	4408.7	427.5	73,005	17.34
2000	4,710,441	154,959,550	230.0	2182.5	300.3	86,913	15.31
2005	4,931,748	163,791,250	148.8	1321.0	243.3	93,415	16.25
2007	5,036,958	164,750,560	128.9	1125.4	215.8	93,687	15.96
2010	5,292,343	170,504,580	106.0	895.4	178.0	96,780	15.90
2014	5,653,678	180,541,380	83.7	663.8	131.2	101,758	15.93
2017	5,903,513	187,522,510	72.7	543.5	105.2	106,432	16.12
2020	6,145,010	194,476,900	64.5	453.2	85.9	110,043	16.32
2023	6,396,572	201,903,140	58.6	390.9	72.6	113,881	16.65

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With New Survival Rates
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,934,750	126,570,600	508.8	5091.7	463.8	71,905	17.51
2000	4,710,441	154,959,550	230.0	2182.5	300.3	86,913	15.31
2005	4,931,748	163,791,250	148.8	1321.0	243.3	93,415	16.25
2007	5,036,958	165,888,140	128.3	1120.2	213.4	94,212	15.94
2010	5,292,342	173,551,680	102.8	881.3	173.2	98,153	15.90
2014	5,653,678	186,284,220	73.7	615.5	123.7	104,269	15.96
2017	5,903,514	194,840,240	57.9	466.3	96.2	109,720	16.14
2020	6,145,010	202,564,850	47.3	364.0	76.9	113,701	16.32
2023	6,396,570	210,436,450	42.0	306.0	64.7	117,744	16.63

Difference (Ver. 2.506 - Ver. 2.505) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-25	-5,986,940	76.3	683.0	36.3	-1,100	0.17
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	1,137,580	-0.6	-5.2	-2.4	525	-0.02
2010	-1	3,047,100	-3.1	-14.1	-4.8	1,373	-0.01
2014	0	5,742,840	-10.0	-48.4	-7.5	2,511	0.03
2017	1	7,317,730	-14.8	-77.2	-9.0	3,288	0.02
2020	0	8,087,950	-17.2	-89.2	-9.1	3,658	0.00
2023	-2	8,533,310	-16.6	-84.9	-7.9	3,863	-0.02

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.505)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	-4.52%	17.65%	15.49%	8.48%	-1.51%	0.98%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.69%	-0.43%	-0.46%	-1.09%	0.56%	-0.12%
2010	0.00%	1.79%	-2.95%	-1.57%	-2.69%	1.42%	-0.03%
2014	0.00%	3.18%	-11.95%	-7.28%	-5.70%	2.47%	0.20%
2017	0.00%	3.90%	-20.42%	-14.20%	-8.58%	3.09%	0.12%
2020	0.00%	4.16%	-26.70%	-19.68%	-10.57%	3.32%	-0.02%
2023	0.00%	4.23%	-28.32%	-21.72%	-10.85%	3.39%	-0.12%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-41. Impact on San Joaquin Valley Air Basin Inventory of Change 12.7

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,280	58,324,240	220.9	2294.3	329.0	41,812	25.19
2000	1,943,272	77,177,536	138.2	1327.0	330.1	52,748	18.20
2005	2,389,193	98,949,512	107.1	909.0	394.0	71,319	21.23
2007	2,407,275	97,362,808	92.7	776.9	347.7	69,641	18.94
2010	2,600,200	103,175,860	78.5	634.6	291.5	72,654	17.30
2014	2,830,493	113,057,020	62.2	477.9	213.6	80,019	15.63
2017	3,012,243	121,296,520	53.1	390.9	164.8	86,207	14.67
2020	3,203,419	129,483,600	46.7	330.7	129.1	92,367	14.12
2023	3,406,514	137,546,340	42.8	295.2	106.8	99,728	14.18

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With New Survival Rates
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,395,175	55,468,272	247.8	2537.4	341.2	41,131	25.39
2000	1,943,272	77,177,536	138.2	1327.0	330.1	52,748	18.20
2005	2,389,193	98,949,512	107.1	909.0	394.0	71,319	21.23
2007	2,407,275	98,375,648	92.5	774.0	346.4	70,076	18.94
2010	2,600,200	105,634,770	77.2	629.3	288.9	73,651	17.34
2014	2,830,493	117,297,570	57.9	462.1	210.1	81,724	15.74
2017	3,012,242	126,354,730	46.4	363.2	160.8	88,275	14.79
2020	3,203,419	134,943,280	38.6	295.9	125.1	94,639	14.25
2023	3,406,514	143,350,660	34.3	254.6	103.1	102,241	14.30

Difference (Ver. 2.506 - Ver. 2.505) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-105	-2,855,968	26.9	243.1	12.1	-681	0.20
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	1,012,840	-0.2	-2.8	-1.3	435	0.00
2010	0	2,458,910	-1.3	-5.4	-2.5	997	0.04
2014	0	4,240,550	-4.3	-15.8	-3.5	1,706	0.10
2017	-1	5,058,210	-6.7	-27.6	-4.0	2,068	0.12
2020	0	5,459,680	-8.1	-34.8	-4.0	2,272	0.13
2023	0	5,804,320	-8.5	-40.6	-3.7	2,513	0.12

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.505)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-0.01%	-4.90%	12.19%	10.60%	3.69%	-1.63%	0.79%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	1.04%	-0.18%	-0.36%	-0.38%	0.62%	0.02%
2010	0.00%	2.38%	-1.68%	-0.84%	-0.87%	1.37%	0.24%
2014	0.00%	3.75%	-6.96%	-3.30%	-1.65%	2.13%	0.65%
2017	0.00%	4.17%	-12.54%	-7.06%	-2.44%	2.40%	0.82%
2020	0.00%	4.22%	-17.40%	-10.53%	-3.09%	2.46%	0.90%
2023	0.00%	4.22%	-19.91%	-13.76%	-3.46%	2.52%	0.87%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-42. Impact on South Coast Air Basin Inventory of Change 12.7

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.505)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,380	282,562,270	880.1	8973.4	895.9	164,587	41.91
2000	8,884,540	323,010,620	441.7	4362.0	654.3	181,129	34.91
2005	10,519,562	393,767,940	307.5	2948.3	602.5	230,707	43.21
2007	9,971,575	367,746,560	234.0	2261.2	454.0	211,679	37.30
2010	10,473,359	377,735,330	189.1	1790.0	383.3	216,301	37.25
2014	11,179,501	393,461,060	153.0	1373.7	292.9	228,495	37.32
2017	11,582,747	403,020,290	134.2	1138.9	235.6	236,187	37.17
2020	11,961,135	414,267,300	120.1	961.0	191.6	242,982	37.10
2023	12,242,366	422,568,670	109.1	828.7	162.0	250,127	37.33

South Coast Summer Episodic On-Road Motor Vehicle Inventories With New Survival Rates
(Calculated Using EMFAC2010 ver 2.506)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,547,190	269,279,940	1017.9	10012.5	956.3	161,692	42.44
2000	8,884,540	323,010,620	441.7	4362.0	654.3	181,129	34.91
2005	10,519,562	393,767,940	307.5	2948.3	602.5	230,707	43.21
2007	9,971,575	369,926,690	233.5	2250.4	449.6	212,629	37.23
2010	10,473,358	384,491,940	184.3	1757.5	373.5	219,250	37.19
2014	11,179,501	407,362,660	137.7	1281.0	278.4	234,650	37.37
2017	11,582,748	420,267,420	112.2	1009.6	220.1	243,918	37.24
2020	11,961,132	433,515,070	94.1	817.5	176.9	251,624	37.16
2023	12,242,366	443,030,180	82.8	683.1	148.9	259,363	37.37

Difference (Ver. 2.506 - Ver. 2.505) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-190	-13,282,330	137.8	1039.1	60.4	-2,896	0.54
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	2,180,130	-0.6	-10.8	-4.4	950	-0.06
2010	-1	6,756,610	-4.8	-32.4	-9.8	2,949	-0.06
2014	0	13,901,600	-15.3	-92.7	-14.5	6,155	0.05
2017	1	17,247,130	-22.0	-129.3	-15.5	7,732	0.07
2020	-3	19,247,770	-25.9	-143.4	-14.7	8,642	0.06
2023	0	20,461,510	-26.3	-145.6	-13.1	9,236	0.04

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.505)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	-4.70%	15.65%	11.58%	6.74%	-1.76%	1.28%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.59%	-0.24%	-0.48%	-0.98%	0.45%	-0.17%
2010	0.00%	1.79%	-2.52%	-1.81%	-2.55%	1.36%	-0.15%
2014	0.00%	3.53%	-10.00%	-6.75%	-4.94%	2.69%	0.13%
2017	0.00%	4.28%	-16.43%	-11.35%	-6.57%	3.27%	0.19%
2020	0.00%	4.65%	-21.60%	-14.93%	-7.69%	3.56%	0.16%
2023	0.00%	4.84%	-24.10%	-17.57%	-8.09%	3.69%	0.10%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.8 REVISE GREENHOUSE GAS EMISSION FACTORS AND GASOLINE PM

Please refer to Section 3.3.3.1. and Section 3.3.3.6. These components were added to the model and the incremental emissions are discussed here together. The change in CO2 emissions are small, generally reducing by 0 to 2% independent of region. PM generally went down significantly, as much as 20% in the future. This is due to lower emission factors for newer vehicles and LEV. There was some increase in the past as older vehicles were found to be higher emitting in the Kansas City study.

Table 12-43. Impact on Statewide Inventory of Change 12.8

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.50.7e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,665,808	693,786,560	2,317	23,358	2,559	422,428	125.28
2000	22,316,218	804,194,820	1,201	11,513	2,061	471,882	92.63
2005	25,801,910	950,032,770	829	7,541	1,717	566,290	106.55
2007	25,453,716	919,583,940	684	6,209	1,375	543,289	97.23
2010	26,930,604	953,876,160	564	4,985	1,111	558,556	95.50
2014	28,913,014	1,008,486,200	458	3,800	862	589,623	96.10
2017	30,251,150	1,047,308,500	405	3,153	731	612,246	97.06
2020	31,572,478	1,088,821,100	366	2,678	633	633,705	98.15
2023	32,810,362	1,128,081,400	338	2,345	573	659,453	100.23

Statewide Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs/Gasoline PM BERs
(Calculated Using EMFAC2010 ver 2.509e))

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,665,456	660,180,670	2,621	26,029	2,692	416,365	138.90
2000	22,316,218	804,194,820	1,169	11,274	2,033	461,826	91.01
2005	25,801,910	950,032,770	809	7,394	1,704	558,598	96.96
2007	25,453,712	926,450,180	667	6,047	1,355	540,049	85.64
2010	26,930,592	972,253,500	544	4,814	1,082	563,855	80.79
2014	28,912,956	1,043,885,300	416	3,565	828	612,723	78.31
2017	30,251,038	1,091,683,800	349	2,888	699	649,197	77.83
2020	31,572,304	1,138,232,200	305	2,451	610	682,234	77.99
2023	32,810,114	1,180,801,200	283	2,192	562	717,559	78.96

Difference (Ver. 2.509e - Ver. 2.507e) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-352	-33,605,890	303.6	2670.9	133.0	-6,063	13.62
2000	0	0	-32.4	-238.8	-28.0	-10,056	-1.62
2005	0	0	-20.4	-146.7	-13.0	-7,692	-9.60
2007	-4	6,866,240	-16.7	-161.8	-20.7	-3,241	-11.59
2010	-12	18,377,340	-20.6	-171.0	-28.6	5,299	-14.71
2014	-58	35,399,100	-42.1	-234.3	-33.3	23,101	-17.79
2017	-112	44,375,300	-56.2	-264.8	-31.6	36,951	-19.23
2020	-174	49,411,100	-60.9	-226.5	-23.1	48,529	-20.16
2023	-248	52,719,800	-54.8	-152.7	-10.2	58,105	-21.27

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.507e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	-4.84%	13.10%	11.43%	5.20%	-1.44%	10.87%
2000	0.00%	0.00%	-2.70%	-2.07%	-1.36%	-2.13%	-1.75%
2005	0.00%	0.00%	-2.46%	-1.95%	-0.76%	-1.36%	-9.01%
2007	0.00%	0.75%	-2.45%	-2.61%	-1.51%	-0.60%	-11.92%
2010	0.00%	1.93%	-3.64%	-3.43%	-2.57%	0.95%	-15.40%
2014	0.00%	3.51%	-9.19%	-6.17%	-3.87%	3.92%	-18.51%
2017	0.00%	4.24%	-13.88%	-8.40%	-4.32%	6.04%	-19.81%
2020	0.00%	4.54%	-16.63%	-8.46%	-3.65%	7.66%	-20.54%
2023	0.00%	4.67%	-16.23%	-6.51%	-1.79%	8.81%	-21.22%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-44. Impact on Sacramento Valley Air Basin Inventory of Change 12.8

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,394,479	50,672,340	187.1	1916.5	203.6	31,075	10.27
2000	1,649,156	57,586,440	97.5	926.4	165.8	33,955	7.03
2005	1,990,785	70,935,288	69.8	608.7	142.8	42,016	8.20
2007	1,968,651	68,579,312	59.4	510.9	119.1	40,956	7.62
2010	2,132,849	73,205,224	50.1	415.2	96.8	43,030	7.49
2014	2,326,865	79,631,664	40.4	312.2	74.1	46,439	7.53
2017	2,465,686	84,103,120	35.4	255.7	62.2	48,852	7.60
2020	2,603,439	88,483,016	31.8	215.7	53.4	51,171	7.70
2023	2,748,041	93,168,168	29.4	189.3	47.6	53,890	7.90

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,394,423	47,858,188	210.8	2121.6	212.9	30,547	11.17
2000	1,649,156	57,586,440	95.1	908.5	163.8	33,351	6.90
2005	1,990,785	70,935,288	68.3	597.7	141.9	41,473	7.53
2007	1,968,651	69,001,744	58.4	501.5	117.8	40,685	6.80
2010	2,132,846	74,276,584	49.2	411.4	95.3	43,265	6.46
2014	2,326,848	82,219,424	37.6	307.0	72.6	48,105	6.30
2017	2,465,655	87,740,744	31.0	246.6	60.9	51,749	6.30
2020	2,603,390	92,759,936	26.7	207.1	52.9	55,106	6.34
2023	2,747,977	97,836,016	24.6	184.2	48.2	58,681	6.47

Difference (Ver. 2.509e - Ver. 2.507e) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-56	-2814152	23.8	205.0	9.3	-528	0.89
2000	0	0	-2.3	-17.9	-2.0	-605	-0.13
2005	0	0	-1.5	-11.0	-0.9	-543	-0.66
2007	0	422432	-1.0	-9.4	-1.4	-271	-0.83
2010	-3	1071360	-0.9	-3.8	-1.5	235	-1.04
2014	-17	2587760	-2.8	-5.2	-1.6	1,665	-1.23
2017	-31	3637624	-4.4	-9.1	-1.3	2,896	-1.30
2020	-49	4276920	-5.2	-8.6	-0.5	3,936	-1.36
2023	-64	4667848	-4.8	-5.2	0.6	4,791	-1.44

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.507e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	-5.55%	12.70%	10.70%	4.55%	-1.70%	8.68%
2000	0.00%	0.00%	-2.37%	-1.93%	-1.20%	-1.78%	-1.88%
2005	0.00%	0.00%	-2.14%	-1.81%	-0.65%	-1.29%	-8.10%
2007	0.00%	0.62%	-1.68%	-1.83%	-1.16%	-0.66%	-10.83%
2010	0.00%	1.46%	-1.80%	-0.92%	-1.59%	0.55%	-13.83%
2014	0.00%	3.25%	-6.87%	-1.66%	-2.10%	3.59%	-16.28%
2017	0.00%	4.33%	-12.36%	-3.55%	-2.13%	5.93%	-17.12%
2020	0.00%	4.83%	-16.19%	-4.00%	-1.01%	7.69%	-17.65%
2023	0.00%	5.01%	-16.39%	-2.72%	1.36%	8.89%	-18.17%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-45. Impact on San Diego Air Basin Inventory of Change 12.8

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,624,564	65,528,924	199.8	2157.6	208.6	40,173	8.55
2000	1,938,446	74,991,368	96.4	1000.8	156.0	41,974	7.32
2005	2,234,307	87,936,400	63.6	649.3	118.8	49,825	8.31
2007	2,273,919	87,207,744	55.1	562.9	103.2	49,287	8.14
2010	2,334,764	86,859,984	45.2	448.1	82.4	48,730	7.94
2014	2,459,716	89,958,456	37.0	342.5	63.8	50,200	8.01
2017	2,557,805	93,097,080	33.0	287.1	54.0	51,737	8.11
2020	2,659,812	96,587,824	30.2	247.4	46.5	53,482	8.24
2023	2,755,727	99,663,704	28.2	220.9	41.3	55,458	8.44

San Diego Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,624,538	62,118,796	230	2436	220	39,680	9.92
2000	1,938,446	74,991,368	94	981	153	41,159	7.03
2005	2,234,307	87,936,400	62	637	118	49,217	7.27
2007	2,273,918	88,381,360	53	543	101	49,303	6.87
2010	2,334,764	89,490,896	42	422	79	49,730	6.42
2014	2,459,717	94,034,376	32	309	59	52,762	6.24
2017	2,557,806	97,781,896	27	252	49	55,419	6.25
2020	2,659,812	101,558,500	24	216	43	58,097	6.32
2023	2,755,726	104,891,340	22	197	39	60,886	6.41

Difference (Ver. 2.509e - Ver. 2.507e) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-26	-3,410,128	30.5	278.2	11.6	-493	1.37
2000	0	0	-2.6	-19.9	-2.6	-815	-0.29
2005	0	0	-1.6	-12.8	-1.1	-608	-1.04
2007	-1	1,173,616	-1.9	-19.7	-2.4	16	-1.26
2010	0	2,630,912	-2.8	-25.7	-3.8	1,000	-1.53
2014	1	4,075,920	-4.8	-33.1	-4.7	2,563	-1.77
2017	1	4,684,816	-6.0	-35.5	-4.7	3,682	-1.86
2020	0	4,970,676	-6.3	-31.1	-3.9	4,614	-1.93
2023	-1	5,227,636	-5.9	-24.3	-2.8	5,429	-2.02

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.507e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	-5.20%	15.26%	12.89%	5.54%	-1.23%	16.03%
2000	0.00%	0.00%	-2.67%	-1.99%	-1.66%	-1.94%	-3.89%
2005	0.00%	0.00%	-2.53%	-1.96%	-0.96%	-1.22%	-12.48%
2007	0.00%	1.35%	-3.52%	-3.51%	-2.35%	0.03%	-15.52%
2010	0.00%	3.03%	-6.30%	-5.73%	-4.61%	2.05%	-19.20%
2014	0.00%	4.53%	-13.06%	-9.66%	-7.33%	5.10%	-22.10%
2017	0.00%	5.03%	-18.11%	-12.37%	-8.75%	7.12%	-22.95%
2020	0.00%	5.15%	-20.95%	-12.56%	-8.47%	8.63%	-23.36%
2023	0.00%	5.25%	-20.87%	-10.98%	-6.69%	9.79%	-24.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-46. Impact on San Francisco Bay Air Basin Inventory of Change 12.8

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,948,878	133,723,100	479.8	4458.4	461.3	76,222	19.70
2000	4,724,726	156,271,180	248.3	2208.1	339.1	89,810	15.82
2005	4,936,224	164,056,700	163.5	1338.5	249.1	94,241	15.98
2007	5,041,512	165,108,000	141.1	1142.2	217.1	94,645	15.76
2010	5,296,710	170,781,230	116.2	912.3	174.2	97,406	15.72
2014	5,657,796	180,488,980	93.2	684.1	134.4	101,805	15.91
2017	5,907,515	187,260,050	82.0	565.5	113.4	106,078	16.19
2020	6,148,934	194,075,760	73.7	476.6	97.7	109,433	16.42
2023	6,400,411	201,447,360	67.8	415.5	87.0	113,179	16.76

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,948,864	127,727,790	542.7	5081.8	493.4	74,729	22.73
2000	4,724,726	156,271,180	241.5	2157.6	333.5	87,079	16.01
2005	4,936,224	164,056,700	159.3	1305.9	246.1	92,360	14.76
2007	5,041,512	166,244,540	138.0	1108.1	212.8	93,523	14.07
2010	5,296,710	173,828,480	113.3	877.6	168.5	97,828	13.45
2014	5,657,793	186,259,340	86.7	633.1	127.1	105,475	13.12
2017	5,907,514	194,613,550	73.1	505.1	105.9	112,442	13.12
2020	6,148,931	202,176,860	64.9	425.7	91.6	117,930	13.21
2023	6,400,408	209,965,740	61.6	389.5	83.8	123,397	13.44

Difference (Ver. 2.509e - Ver. 2.507e) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-14	-5,995,310	62.9	623.4	32.2	-1,494	3.03
2000	0	0	-6.8	-50.5	-5.5	-2,731	0.19
2005	0	0	-4.2	-32.6	-3.0	-1,880	-1.22
2007	0	1,136,540	-3.1	-34.1	-4.3	-1,122	-1.68
2010	0	3,047,250	-2.9	-34.7	-5.7	422	-2.27
2014	-3	5,770,360	-6.5	-51.0	-7.3	3,670	-2.79
2017	-1	7,353,500	-8.9	-60.3	-7.5	6,364	-3.07
2020	-3	8,101,100	-8.8	-50.9	-6.0	8,497	-3.21
2023	-3	8,518,380	-6.2	-26.1	-3.2	10,218	-3.32

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.507e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	-4.48%	13.11%	13.98%	6.97%	-1.96%	15.39%
2000	0.00%	0.00%	-2.75%	-2.29%	-1.64%	-3.04%	1.20%
2005	0.00%	0.00%	-2.56%	-2.44%	-1.21%	-2.00%	-7.66%
2007	0.00%	0.69%	-2.18%	-2.99%	-1.97%	-1.19%	-10.68%
2010	0.00%	1.78%	-2.49%	-3.81%	-3.27%	0.43%	-14.47%
2014	0.00%	3.20%	-6.95%	-7.46%	-5.44%	3.61%	-17.55%
2017	0.00%	3.93%	-10.83%	-10.67%	-6.65%	6.00%	-18.97%
2020	0.00%	4.17%	-11.97%	-10.68%	-6.18%	7.76%	-19.55%
2023	0.00%	4.23%	-9.14%	-6.27%	-3.69%	9.03%	-19.81%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-47. Impact on San Joaquin Valley Air Basin Inventory of Change 12.8

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,405,114	58,580,652	219.0	2299.7	331.9	42,756	22.06
2000	1,953,670	77,852,040	133.2	1295.6	344.3	53,500	14.06
2005	2,389,626	97,397,040	98.3	869.2	320.3	67,037	16.20
2007	2,408,237	96,145,336	84.4	741.6	268.3	66,019	14.80
2010	2,601,884	101,880,540	71.1	604.4	212.5	68,742	13.76
2014	2,832,668	110,655,900	57.2	456.9	160.1	73,869	13.15
2017	3,014,826	117,719,150	49.7	376.5	133.8	77,712	12.92
2020	3,206,450	124,790,200	44.6	321.3	114.8	81,640	12.84
2023	3,410,038	132,194,620	41.5	288.6	103.3	87,649	13.15

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,405,043	55,697,916	242.3	2525.9	341.7	42,250	22.79
2000	1,953,670	77,852,040	129.9	1271.8	341.4	52,789	13.67
2005	2,389,626	97,397,040	96.0	853.7	318.6	66,368	15.07
2007	2,408,237	97,169,640	82.2	723.3	265.6	65,892	13.49
2010	2,601,884	104,381,480	68.2	586.3	208.9	69,519	12.16
2014	2,832,668	114,960,370	51.9	437.1	156.4	76,429	11.27
2017	3,014,826	122,836,810	43.1	355.9	130.6	81,641	10.92
2020	3,206,450	130,277,340	37.5	305.7	112.7	86,705	10.72
2023	3,410,038	137,987,070	34.8	278.2	102.7	93,782	10.76

Difference (Ver. 2.509e - Ver. 2.507e) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-71	-2,882,736	23.3	226.3	9.8	-506	0.73
2000	0	0	-3.2	-23.8	-2.9	-711	-0.39
2005	0	0	-2.3	-15.5	-1.7	-669	-1.13
2007	0	1,024,304	-2.2	-18.4	-2.7	-127	-1.31
2010	0	2,500,940	-2.9	-18.1	-3.6	777	-1.60
2014	0	4,304,470	-5.2	-19.8	-3.7	2,560	-1.88
2017	0	5,117,660	-6.7	-20.6	-3.3	3,930	-2.00
2020	0	5,487,140	-7.1	-15.6	-2.1	5,064	-2.13
2023	0	5,792,450	-6.6	-10.3	-0.6	6,133	-2.39

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.507e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-0.01%	-4.92%	10.65%	9.84%	2.94%	-1.18%	3.29%
2000	0.00%	0.00%	-2.42%	-1.84%	-0.83%	-1.33%	-2.75%
2005	0.00%	0.00%	-2.34%	-1.79%	-0.52%	-1.00%	-6.95%
2007	0.00%	1.07%	-2.59%	-2.48%	-1.01%	-0.19%	-8.88%
2010	0.00%	2.45%	-4.13%	-3.00%	-1.68%	1.13%	-11.63%
2014	0.00%	3.89%	-9.14%	-4.34%	-2.32%	3.47%	-14.28%
2017	0.00%	4.35%	-13.42%	-5.47%	-2.45%	5.06%	-15.50%
2020	0.00%	4.40%	-16.02%	-4.85%	-1.80%	6.20%	-16.56%
2023	0.00%	4.38%	-16.03%	-3.58%	-0.55%	7.00%	-18.17%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-48. Impact on South Coast Air Basin Inventory of Change 12.8

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.507e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,587,541	284,337,980	882.2	8983.9	935.1	168,928	41.96
2000	8,926,638	325,417,540	439.9	4337.8	708.1	185,478	34.06
2005	10,551,385	393,939,550	301.0	2913.0	583.1	230,533	40.55
2007	9,997,372	368,084,260	228.8	2235.2	430.8	211,870	35.37
2010	10,501,053	378,033,630	184.3	1766.7	356.3	216,185	35.53
2014	11,209,772	392,913,630	150.6	1360.9	285.1	226,596	36.46
2017	11,614,586	401,614,560	133.7	1134.1	246.0	232,508	37.06
2020	11,994,158	412,199,680	121.0	962.3	215.6	237,963	37.53
2023	12,277,238	420,057,220	111.2	835.0	199.5	244,167	38.17

South Coast Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,587,395	270,934,940	1001.9	9919.6	985.7	167,087	47.67
2000	8,926,638	325,417,540	427.4	4245.3	697.2	181,740	33.25
2005	10,551,385	393,939,550	293.2	2855.1	578.0	227,723	36.28
2007	9,997,371	370,286,240	222.4	2174.5	423.4	210,810	30.47
2010	10,501,055	384,846,080	175.5	1694.8	345.8	218,693	29.31
2014	11,209,772	406,880,000	133.8	1257.2	272.8	236,382	28.81
2017	11,614,590	418,954,500	112.6	1025.6	234.9	247,561	28.73
2020	11,994,159	431,569,890	98.7	873.9	207.8	257,501	28.81
2023	12,277,239	440,648,480	90.5	775.7	195.9	267,283	29.02

Difference (Ver. 2.509e - Ver. 2.507e) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-146	-13,403,040	119.6	935.7	50.6	-1,841	5.72
2000	0	0	-12.6	-92.4	-10.9	-3,738	-0.82
2005	0	0	-7.8	-58.0	-5.0	-2,810	-4.27
2007	-1	2,201,980	-6.4	-60.6	-7.4	-1,060	-4.91
2010	2	6,812,450	-8.8	-71.9	-10.5	2,508	-6.22
2014	0	13,966,370	-16.8	-103.7	-12.3	9,786	-7.66
2017	4	17,339,940	-21.1	-108.5	-11.1	15,053	-8.33
2020	1	19,370,210	-22.4	-88.4	-7.8	19,538	-8.73
2023	1	20,591,260	-20.6	-59.3	-3.6	23,116	-9.14

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.507e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	-4.71%	13.56%	10.42%	5.41%	-1.09%	13.62%
2000	0.00%	0.00%	-2.85%	-2.13%	-1.54%	-2.02%	-2.40%
2005	0.00%	0.00%	-2.60%	-1.99%	-0.86%	-1.22%	-10.52%
2007	0.00%	0.60%	-2.81%	-2.71%	-1.71%	-0.50%	-13.87%
2010	0.00%	1.80%	-4.75%	-4.07%	-2.93%	1.16%	-17.50%
2014	0.00%	3.55%	-11.18%	-7.62%	-4.32%	4.32%	-21.00%
2017	0.00%	4.32%	-15.77%	-9.57%	-4.50%	6.47%	-22.47%
2020	0.00%	4.70%	-18.48%	-9.18%	-3.62%	8.21%	-23.25%
2023	0.00%	4.90%	-18.55%	-7.10%	-1.82%	9.47%	-23.95%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type Using Populations

12.9 VMT AND SPEED DISTRIBUTIONS (FIRST ROUND)

Please refer to Section 3.3.2. This change is complex and highly regional specific. In general, the future VMT projections are lower. Intuitively, one would expect the future emissions to be lower. However, the slowdown in growth means fewer new vehicle sales, so the fleet actually gets older and new technologies are not introduced as quickly.

Table 12-49. Impact on Statewide Inventory of Change 12.9

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.50.09e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,665,456	660,180,670	2,621	26,029	2,692	416,365	138.90
2000	22,316,218	804,194,820	1,169	11,274	2,033	461,826	91.01
2005	25,801,910	950,032,770	809	7,394	1,704	558,598	96.96
2007	25,453,712	926,450,180	667	6,047	1,355	540,049	85.64
2010	26,930,592	972,253,500	544	4,814	1,082	563,855	80.79
2014	28,912,956	1,043,885,300	416	3,565	828	612,723	78.31
2017	30,251,038	1,091,683,800	349	2,888	699	649,197	77.83
2020	31,572,304	1,138,232,200	305	2,451	610	682,234	77.99
2023	32,810,114	1,180,801,200	283	2,192	562	717,559	78.96

Statewide Summer Episodic On-Road Motor Vehicle Inventories With 09 Populations VMT matched
(Calculated Using EMFAC2010 ver 2.50.20a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,378,444	689,422,780	2,574	25,824	2,725	430,674	145.38
2000	22,181,680	839,893,380	1,102	10,866	2,158	490,598	94.16
2005	23,631,408	895,163,970	734	6,764	1,631	531,065	92.19
2007	24,447,824	923,793,790	643	5,871	1,469	552,134	89.30
2010	26,118,692	946,227,260	573	5,083	1,287	568,335	84.68
2014	28,247,534	999,628,350	487	4,142	1,033	609,254	80.78
2017	29,519,084	1,044,801,900	429	3,486	886	646,130	77.98
2020	30,517,106	1,089,757,700	383	2,980	777	680,375	77.12
2023	31,298,652	1,130,831,900	351	2,573	702	716,532	77.51

Difference (Ver. 2.5020a - Ver. 2.509e) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-287,012	29,242,110	-46.9	-205.0	33.3	14,309	6.48
2000	-134,538	35,698,560	-66.6	-407.9	124.8	28,772	3.15
2005	-2,170,502	-54,868,800	-75.1	-630.3	-72.6	-27,533	-4.76
2007	-1,005,888	-2,656,390	-24.5	-176.4	114.6	12,086	3.66
2010	-811,900	-26,026,240	29.0	269.2	204.9	4,480	3.89
2014	-665,422	-44,256,950	70.3	576.7	205.1	-3,470	2.47
2017	-731,954	-46,881,900	80.1	598.0	187.0	-3,067	0.15
2020	-1,055,198	-48,474,500	78.0	529.0	167.6	-1,859	-0.87
2023	-1,511,462	-49,969,300	67.9	380.9	139.4	-1,027	-1.45

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.509e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-1.54%	4.43%	-1.79%	-0.79%	1.24%	3.44%	4.67%
2000	-0.60%	4.44%	-5.70%	-3.62%	6.14%	6.23%	3.46%
2005	-8.41%	-5.78%	-9.29%	-8.52%	-4.26%	-4.93%	-4.91%
2007	-3.95%	-0.29%	-3.67%	-2.92%	8.46%	2.24%	4.27%
2010	-3.01%	-2.68%	5.33%	5.59%	18.93%	0.79%	4.82%
2014	-2.30%	-4.24%	16.88%	16.18%	24.76%	-0.57%	3.15%
2017	-2.42%	-4.29%	22.98%	20.71%	26.73%	-0.47%	0.19%
2020	-3.34%	-4.26%	25.58%	21.58%	27.48%	-0.27%	-1.12%
2023	-4.61%	-4.23%	24.01%	17.38%	24.78%	-0.14%	-1.84%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-50. Impact on Sacramento Valley Air Basin Inventory of Change 12.9

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,394,423	47,858,188	210.8	2121.6	212.9	30,547	11.17
2000	1,649,156	57,586,440	95.1	908.5	163.8	33,351	6.90
2005	1,990,785	70,935,288	68.3	597.7	141.9	41,473	7.53
2007	1,968,651	69,001,744	58.4	501.5	117.8	40,685	6.80
2010	2,132,846	74,276,584	49.2	411.4	95.3	43,265	6.46
2014	2,326,848	82,219,424	37.6	307.0	72.6	48,105	6.30
2017	2,465,655	87,740,744	31.0	246.6	60.9	51,749	6.30
2020	2,603,390	92,759,936	26.7	207.1	52.9	55,106	6.34
2023	2,747,977	97,836,016	24.6	184.2	48.2	58,681	6.47

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With 09 Populations VMT matched
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,362,128	51,431,120	210.9	2186.2	218.7	32,506	11.56
2000	1,634,783	63,336,324	87.9	877.5	179.3	37,337	7.40
2005	1,780,274	68,268,976	60.6	548.7	136.5	40,405	7.19
2007	1,860,962	70,353,144	54.6	486.4	123.9	42,177	6.97
2010	2,030,439	72,283,584	50.8	436.4	109.5	43,271	6.61
2014	2,285,447	78,601,864	45.8	376.9	91.0	47,483	6.44
2017	2,424,266	82,960,408	41.4	323.9	79.5	50,604	6.27
2020	2,522,620	87,144,888	37.3	277.8	70.7	53,555	6.25
2023	2,604,611	91,737,016	34.2	234.4	64.2	56,833	6.35

Difference (Ver. 2.5020a - Ver. 2.509e) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-32295	3572932	0.1	64.7	5.8	1,959	0.40
2000	-14373	5749884	-7.2	-31.0	15.4	3,987	0.50
2005	-210511	-2666312	-7.8	-49.0	-5.4	-1,068	-0.34
2007	-107689	1351400	-3.9	-15.2	6.1	1,492	0.17
2010	-102407	-1993000	1.6	25.0	14.2	6	0.15
2014	-41401	-3617560	8.2	69.9	18.5	-622	0.14
2017	-41389	-4780336	10.4	77.3	18.6	-1,145	-0.03
2020	-80770	-5615048	10.7	70.8	17.8	-1,552	-0.09
2023	-143366	-6099000	9.6	50.2	16.0	-1,848	-0.11

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.509e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-2.32%	7.47%	0.05%	3.05%	2.74%	6.41%	3.54%
2000	-0.87%	9.98%	-7.59%	-3.41%	9.42%	11.95%	7.31%
2005	-10.57%	-3.76%	-11.34%	-8.20%	-3.81%	-2.57%	-4.51%
2007	-5.47%	1.96%	-6.61%	-3.02%	5.17%	3.67%	2.52%
2010	-4.80%	-2.68%	3.17%	6.08%	14.95%	0.01%	2.38%
2014	-1.78%	-4.40%	21.84%	22.76%	25.42%	-1.29%	2.15%
2017	-1.68%	-5.45%	33.54%	31.33%	30.54%	-2.21%	-0.50%
2020	-3.10%	-6.05%	39.97%	34.17%	33.69%	-2.82%	-1.36%
2023	-5.22%	-6.23%	39.01%	27.28%	33.11%	-3.15%	-1.78%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-51. Impact on San Diego Air Basin Inventory of Change 12.9

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,624,538	62,118,796	230.3	2435.8	220.2	39,680	9.92
2000	1,938,446	74,991,368	93.8	980.9	153.4	41,159	7.03
2005	2,234,307	87,936,400	62.0	636.6	117.7	49,217	7.27
2007	2,273,918	88,381,360	53.2	543.2	100.7	49,303	6.87
2010	2,334,764	89,490,896	42.4	422.4	78.6	49,730	6.42
2014	2,459,717	94,034,376	32.1	309.4	59.1	52,762	6.24
2017	2,557,806	97,781,896	27.0	251.6	49.2	55,419	6.25
2020	2,659,812	101,558,500	23.9	216.3	42.6	58,097	6.32
2023	2,755,726	104,891,340	22.3	196.7	38.5	60,886	6.41

San Diego Summer Episodic On-Road Motor Vehicle Inventories With 09 Populations VMT matched
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,615,290	60,116,672	226	2341	210	37,836	9.61
2000	1,930,842	73,980,216	86	888	145	40,705	6.82
2005	2,066,461	79,199,328	55	542	104	44,397	6.56
2007	2,138,676	81,365,584	48	471	94	46,080	6.40
2010	2,277,702	82,533,536	43	408	83	47,215	6.16
2014	2,432,722	85,930,752	36	325	66	49,539	5.94
2017	2,480,927	88,470,176	30	263	55	51,374	5.80
2020	2,523,581	90,974,512	26	221	47	53,291	5.77
2023	2,599,894	94,694,160	24	195	43	56,424	5.90

Difference (Ver. 2.5020a - Ver. 2.509e) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-9,248	-2,002,124	-4.2	-94.5	-9.7	-1,844	-0.32
2000	-7,604	-1,011,152	-7.9	-93.0	-8.6	-454	-0.21
2005	-167,846	-8,737,072	-7.4	-94.5	-13.4	-4,820	-0.72
2007	-135,242	-7,015,776	-5.4	-71.7	-6.6	-3,224	-0.47
2010	-57,062	-6,957,360	0.3	-14.5	4.2	-2,514	-0.26
2014	-26,995	-8,103,624	3.6	15.5	6.9	-3,223	-0.30
2017	-76,879	-9,311,720	3.3	11.6	5.9	-4,045	-0.45
2020	-136,231	-10,583,988	2.5	4.3	4.8	-4,806	-0.55
2023	-155,832	-10,197,180	1.7	-2.0	4.2	-4,462	-0.51

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.509e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-0.57%	-3.22%	-1.80%	-3.88%	-4.41%	-4.65%	-3.20%
2000	-0.39%	-1.35%	-8.47%	-9.48%	-5.63%	-1.10%	-2.99%
2005	-7.51%	-9.94%	-11.94%	-14.85%	-11.35%	-9.79%	-9.83%
2007	-5.95%	-7.94%	-10.13%	-13.20%	-6.58%	-6.54%	-6.87%
2010	-2.44%	-7.77%	0.78%	-3.44%	5.32%	-5.06%	-4.03%
2014	-1.10%	-8.62%	11.13%	5.01%	11.64%	-6.11%	-4.77%
2017	-3.01%	-9.52%	12.22%	4.59%	11.98%	-7.30%	-7.21%
2020	-5.12%	-10.42%	10.42%	1.97%	11.35%	-8.27%	-8.67%
2023	-5.65%	-9.72%	7.74%	-1.01%	10.95%	-7.33%	-8.03%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-52. Impact on San Francisco Bay Air Basin Inventory of Change 12.9

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,948,864	127,727,790	542.7	5081.8	493.4	74,729	22.73
2000	4,724,726	156,271,180	241.5	2157.6	333.5	87,079	16.01
2005	4,936,224	164,056,700	159.3	1305.9	246.1	92,360	14.76
2007	5,041,512	166,244,540	138.0	1108.1	212.8	93,523	14.07
2010	5,296,710	173,828,480	113.3	877.6	168.5	97,828	13.45
2014	5,657,793	186,259,340	86.7	633.1	127.1	105,475	13.12
2017	5,907,514	194,613,550	73.1	505.1	105.9	112,442	13.12
2020	6,148,931	202,176,860	64.9	425.7	91.6	117,930	13.21
2023	6,400,408	209,965,740	61.6	389.5	83.8	123,397	13.44

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With 09 Populations VMT matched
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,877,920	137,914,140	533.6	5124.9	515.7	80,787	24.46
2000	4,700,962	168,181,650	233.8	2179.6	364.4	95,856	17.05
2005	4,757,890	169,061,820	154.3	1321.5	264.7	96,931	15.47
2007	4,815,908	170,597,250	132.7	1115.4	233.8	98,995	14.79
2010	5,024,152	171,597,970	116.6	943.9	200.2	100,898	13.99
2014	5,426,317	181,193,380	98.7	752.5	157.9	106,970	13.43
2017	5,621,512	188,394,240	86.0	617.8	132.7	113,179	13.07
2020	5,754,138	195,584,700	76.0	514.4	114.4	118,446	12.99
2023	5,893,056	202,578,720	69.8	444.3	102.4	123,719	13.11

Difference (Ver. 2.5020a - Ver. 2.509e) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-70,944	10,186,350	-9.1	43.1	22.3	6,058	1.73
2000	-23,764	11,910,470	-7.6	22.0	30.8	8,776	1.05
2005	-178,334	5,005,120	-5.0	15.5	18.6	4,571	0.72
2007	-225,604	4,352,710	-5.3	7.3	20.9	5,473	0.71
2010	-272,558	-2,230,510	3.4	66.3	31.7	3,070	0.55
2014	-231,476	-5,065,960	12.0	119.5	30.8	1,495	0.31
2017	-286,002	-6,219,310	12.9	112.6	26.8	737	-0.05
2020	-394,793	-6,592,160	11.1	88.7	22.8	517	-0.23
2023	-507,352	-7,387,020	8.2	54.8	18.6	322	-0.33

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.509e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-1.80%	7.98%	-1.67%	0.85%	4.51%	8.11%	7.61%
2000	-0.50%	7.62%	-3.16%	1.02%	9.25%	10.08%	6.54%
2005	-3.61%	3.05%	-3.17%	1.19%	7.56%	4.95%	4.86%
2007	-4.47%	2.62%	-3.86%	0.66%	9.84%	5.85%	5.06%
2010	-5.15%	-1.28%	2.96%	7.56%	18.82%	3.14%	4.06%
2014	-4.09%	-2.72%	13.79%	18.87%	24.23%	1.42%	2.36%
2017	-4.84%	-3.20%	17.61%	22.30%	25.28%	0.66%	-0.35%
2020	-6.42%	-3.26%	17.11%	20.83%	24.86%	0.44%	-1.72%
2023	-7.93%	-3.52%	13.26%	14.07%	22.26%	0.26%	-2.48%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-53 Impact on San Joaquin Valley Air Basin Inventory of Change 12.9

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,405,043	55,697,916	242.3	2525.9	341.7	42,250	22.79
2000	1,953,670	77,852,040	129.9	1271.8	341.4	52,789	13.67
2005	2,389,626	97,397,040	96.0	853.7	318.6	66,368	15.07
2007	2,408,237	97,169,640	82.2	723.3	265.6	65,892	13.49
2010	2,601,884	104,381,480	68.2	586.3	208.9	69,519	12.16
2014	2,832,668	114,960,370	51.9	437.1	156.4	76,429	11.27
2017	3,014,826	122,836,810	43.1	355.9	130.6	81,641	10.92
2020	3,206,450	130,277,340	37.5	305.7	112.7	86,705	10.72
2023	3,410,038	137,987,070	34.8	278.2	102.7	93,782	10.76

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With 09 Populations VMT matched
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,371,368	58,661,744	230.8	2441.8	342.4	43,703	23.49
2000	1,936,457	83,696,632	112.4	1130.8	356.7	57,176	13.86
2005	2,141,954	91,088,576	81.5	736.0	284.7	61,976	13.66
2007	2,254,768	94,929,168	73.8	657.2	258.4	64,939	13.13
2010	2,493,321	98,407,960	70.3	604.7	226.2	67,570	12.17
2014	2,852,481	107,711,930	65.4	539.9	181.0	74,969	11.52
2017	3,115,979	116,216,320	60.8	478.8	155.1	81,282	10.89
2020	3,309,582	124,193,760	55.7	419.0	134.7	87,075	10.64
2023	3,430,952	131,662,430	51.1	360.2	119.6	93,918	10.55

Difference (Ver. 2.5020a - Ver. 2.509e) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-33,675	2,963,828	-11.5	-84.1	0.7	1,453	0.70
2000	-17,213	5,844,592	-17.5	-141.0	15.3	4,387	0.19
2005	-247,672	-6,308,464	-14.5	-117.6	-33.9	-4,393	-1.41
2007	-153,469	-2,240,472	-8.4	-66.1	-7.2	-953	-0.36
2010	-108,563	-5,973,520	2.1	18.4	17.2	-1,949	0.01
2014	19,813	-7,248,440	13.5	102.8	24.6	-1,460	0.25
2017	101,153	-6,620,490	17.7	123.0	24.6	-360	-0.03
2020	103,132	-6,083,580	18.2	113.4	22.0	370	-0.08
2023	20,914	-6,324,640	16.3	82.0	16.9	136	-0.22

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.509e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-2.40%	5.32%	-4.76%	-3.33%	0.22%	3.44%	3.09%
2000	-0.88%	7.51%	-13.48%	-11.09%	4.49%	8.31%	1.40%
2005	-10.36%	-6.48%	-15.10%	-13.78%	-10.64%	-6.62%	-9.37%
2007	-6.37%	-2.31%	-10.24%	-9.14%	-2.72%	-1.45%	-2.66%
2010	-4.17%	-5.72%	3.05%	3.14%	8.25%	-2.80%	0.05%
2014	0.70%	-6.31%	25.99%	23.52%	15.72%	-1.91%	2.23%
2017	3.36%	-5.39%	41.12%	34.55%	18.81%	-0.44%	-0.31%
2020	3.22%	-4.67%	48.63%	37.08%	19.52%	0.43%	-0.74%
2023	0.61%	-4.58%	46.73%	29.45%	16.47%	0.15%	-2.03%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-54. Impact on South Coast Air Basin Inventory of Change 12.9

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.509e)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,587,395	270,934,940	1001.9	9919.6	985.7	167,087	47.67
2000	8,926,638	325,417,540	427.4	4245.3	697.2	181,740	33.25
2005	10,551,385	393,939,550	293.2	2855.1	578.0	227,723	36.28
2007	9,997,371	370,286,240	222.4	2174.5	423.4	210,810	30.47
2010	10,501,055	384,846,080	175.5	1694.8	345.8	218,693	29.31
2014	11,209,772	406,880,000	133.8	1257.2	272.8	236,382	28.81
2017	11,614,590	418,954,500	112.6	1025.6	234.9	247,561	28.73
2020	11,994,159	431,569,890	98.7	873.9	207.8	257,501	28.81
2023	12,277,239	440,648,480	90.5	775.7	195.9	267,283	29.02

South Coast Summer Episodic On-Road Motor Vehicle Inventories With 09 Populations VMT matched
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,534,778	267,446,580	970.4	9422.9	943.0	162,610	46.35
2000	8,888,948	316,402,500	396.8	3896.1	677.3	177,802	31.97
2005	9,572,533	342,575,040	259.4	2439.1	510.6	199,026	31.73
2007	9,927,914	355,912,900	222.9	2082.6	457.8	207,711	30.87
2010	10,537,437	365,239,650	190.0	1734.1	400.3	213,641	29.58
2014	11,072,442	377,542,370	150.3	1321.6	318.4	225,504	28.07
2017	11,422,403	389,182,050	128.2	1080.8	272.4	236,806	27.30
2020	11,709,373	400,561,250	112.6	914.0	238.4	246,987	26.88
2023	11,886,469	407,786,110	102.2	797.5	216.3	256,607	26.77

Difference (Ver. 2.5020a - Ver. 2.509e) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-52,617	-3,488,360	-31.5	-496.7	-42.7	-4,477	-1.32
2000	-37,690	-9,015,040	-30.6	-349.3	-19.9	-3,937	-1.27
2005	-978,852	-51,364,510	-33.8	-416.0	-67.4	-28,697	-4.55
2007	-69,457	-14,373,340	0.5	-91.9	34.5	-3,098	0.41
2010	36,382	-19,606,430	14.4	39.4	54.5	-5,051	0.27
2014	-137,330	-29,337,630	16.6	64.4	45.6	-10,878	-0.73
2017	-192,187	-29,772,450	15.6	55.2	37.5	-10,755	-1.43
2020	-284,786	-31,008,640	14.0	40.0	30.6	-10,514	-1.93
2023	-390,770	-32,862,370	11.7	21.8	20.5	-10,676	-2.25

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.509e)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-0.69%	-1.29%	-3.14%	-5.01%	-4.33%	-2.68%	-2.77%
2000	-0.42%	-2.77%	-7.16%	-8.23%	-2.86%	-2.17%	-3.83%
2005	-9.28%	-13.04%	-11.53%	-14.57%	-11.67%	-12.60%	-12.55%
2007	-0.69%	-3.88%	0.23%	-4.23%	8.14%	-1.47%	1.33%
2010	0.35%	-5.09%	8.23%	2.32%	15.75%	-2.31%	0.93%
2014	-1.23%	-7.21%	12.40%	5.12%	16.71%	-4.60%	-2.55%
2017	-1.65%	-7.11%	13.90%	5.38%	15.96%	-4.34%	-4.97%
2020	-2.37%	-7.19%	14.14%	4.58%	14.73%	-4.08%	-6.69%
2023	-3.18%	-7.46%	12.89%	2.81%	10.44%	-3.99%	-7.76%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.10 TEMPERATURE AND RELATIVE HUMIDITY PROFILES

Please refer to Section 3.3.4.4. These changes are regionally dependent as well as seasonal. The incremental changes shown below are for summer. There would be other differences for winter or monthly. In general, where emissions increase the temperatures have gotten warmer and/or more humid. This results in more air conditioning usage. NOx is more complex in that higher humidity lowers NOx formation, but simultaneously increases air conditioning usage. This update also includes a change to the fleet mix update for 2000-2008 relative to connecting current and historical fleet mix estimates using multiple DMV analysis methods as described in Section 3.3.1**.**

Table 12-55. Impact on Statewide Inventory of Change 12.10

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,378,444	689,422,780	2,574	25,824	2,725	430,674	145.38
2000	22,181,680	839,893,380	1,102	10,866	2,158	490,598	94.16
2005	23,631,408	895,163,970	734	6,764	1,631	531,065	92.19
2007	24,447,824	923,793,790	643	5,871	1,469	552,134	89.30
2010	26,118,692	946,227,260	573	5,083	1,287	568,335	84.68
2014	28,247,534	999,628,350	487	4,142	1,033	609,254	80.78
2017	29,519,084	1,044,801,900	429	3,486	886	646,130	77.98
2020	30,517,106	1,089,757,700	383	2,980	777	680,375	77.12
2023	31,298,652	1,130,831,900	351	2,573	702	716,532	77.51

Statewide Summer Episodic On-Road Motor Vehicle Inventories With Updated Temp/RH Profiles
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,378,444	689,422,780	2,527	24,167	2,594	424,355	145.38
2000	22,181,680	839,893,380	1,079	10,004	2,066	477,035	94.16
2005	23,631,408	895,163,970	720	6,222	1,571	514,640	92.19
2007	24,447,824	923,793,790	631	5,402	1,417	534,851	89.30
2010	26,118,692	946,227,260	564	4,682	1,243	550,560	84.68
2014	28,247,534	999,628,350	480	3,816	999	590,254	80.78
2017	29,519,084	1,044,801,900	423	3,214	858	625,966	77.98
2020	30,517,106	1,089,757,700	379	2,752	754	659,117	77.12
2023	31,298,652	1,130,831,900	347	2,378	682	694,108	77.51

Difference (Ver. 2.5021a - Ver. 2.5020a) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	-47.1	-1656.9	-131.0	-6,319	0.00
2000	0	0	-23.2	-861.3	-92.3	-13,563	0.00
2005	0	0	-13.5	-542.2	-60.1	-16,425	0.00
2007	0	0	-11.2	-468.5	-52.1	-17,283	0.00
2010	0	0	-9.0	-401.1	-44.5	-17,774	0.00
2014	0	0	-6.8	-326.7	-34.4	-19,000	0.00
2017	0	0	-5.4	-272.5	-28.3	-20,164	0.00
2020	0	0	-4.4	-228.4	-23.6	-21,258	0.00
2023	0	0	-3.7	-195.5	-20.2	-22,423	0.00

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5020a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	-1.83%	-6.42%	-4.81%	-1.47%	0.00%
2000	0.00%	0.00%	-2.11%	-7.93%	-4.28%	-2.76%	0.00%
2005	0.00%	0.00%	-1.84%	-8.02%	-3.69%	-3.09%	0.00%
2007	0.00%	0.00%	-1.74%	-7.98%	-3.55%	-3.13%	0.00%
2010	0.00%	0.00%	-1.58%	-7.89%	-3.46%	-3.13%	0.00%
2014	0.00%	0.00%	-1.39%	-7.89%	-3.33%	-3.12%	0.00%
2017	0.00%	0.00%	-1.26%	-7.82%	-3.19%	-3.12%	0.00%
2020	0.00%	0.00%	-1.14%	-7.66%	-3.03%	-3.12%	0.00%
2023	0.00%	0.00%	-1.06%	-7.60%	-2.87%	-3.13%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-56. Impact on Sacramento Valley Air Basin Inventory of Change 12.10

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,362,128	51,431,120	210.9	2186.2	218.7	32,506	11.56
2000	1,634,783	63,336,324	87.9	877.5	179.3	37,337	7.40
2005	1,780,274	68,268,976	60.6	548.7	136.5	40,405	7.19
2007	1,860,962	70,353,144	54.6	486.4	123.9	42,177	6.97
2010	2,030,439	72,283,584	50.8	436.4	109.5	43,271	6.61
2014	2,285,447	78,601,864	45.8	376.9	91.0	47,483	6.44
2017	2,424,266	82,960,408	41.4	323.9	79.5	50,604	6.27
2020	2,522,620	87,144,888	37.3	277.8	70.7	53,555	6.25
2023	2,604,611	91,737,016	34.2	234.4	64.2	56,833	6.35

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Updated Temp/RH Profiles
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,362,128	51,431,120	212.0	2058.3	205.6	31,912	11.56
2000	1,634,783	63,336,324	88.2	807.1	170.3	36,070	7.40
2005	1,780,274	68,268,976	61.5	503.3	130.6	38,864	7.19
2007	1,860,962	70,353,144	55.4	446.0	118.7	40,550	6.97
2010	2,030,439	72,283,584	51.6	401.0	105.0	41,620	6.61
2014	2,285,447	78,601,864	46.6	346.2	87.4	45,687	6.44
2017	2,424,266	82,960,408	42.1	297.5	76.4	48,693	6.27
2020	2,522,620	87,144,888	38.0	255.4	68.1	51,535	6.25
2023	2,604,611	91,737,016	34.9	215.3	62.0	54,684	6.35

Difference (Ver. 2.5021a - Ver. 2.5020a) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	1.1	-127.9	-13.0	-594	0.00
2000	0	0	0.3	-70.4	-9.0	-1,267	0.00
2005	0	0	0.9	-45.4	-5.9	-1,542	0.00
2007	0	0	0.8	-40.3	-5.2	-1,627	0.00
2010	0	0	0.8	-35.4	-4.5	-1,651	0.00
2014	0	0	0.8	-30.7	-3.7	-1,796	0.00
2017	0	0	0.8	-26.3	-3.1	-1,911	0.00
2020	0	0	0.7	-22.4	-2.6	-2,020	0.00
2023	0	0	0.7	-19.1	-2.2	-2,149	0.00

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.5020a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.52%	-5.85%	-5.97%	-1.83%	0.00%
2000	0.00%	0.00%	0.36%	-8.02%	-5.01%	-3.39%	0.00%
2005	0.00%	0.00%	1.47%	-8.27%	-4.34%	-3.82%	0.00%
2007	0.00%	0.00%	1.50%	-8.29%	-4.20%	-3.86%	0.00%
2010	0.00%	0.00%	1.62%	-8.12%	-4.13%	-3.81%	0.00%
2014	0.00%	0.00%	1.71%	-8.15%	-4.01%	-3.78%	0.00%
2017	0.00%	0.00%	1.81%	-8.13%	-3.84%	-3.78%	0.00%
2020	0.00%	0.00%	1.94%	-8.06%	-3.64%	-3.77%	0.00%
2023	0.00%	0.00%	2.05%	-8.15%	-3.43%	-3.78%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-57. Impact on San Diego Air Basin Inventory of Change 12.10

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,615,290	60,116,672	226.1	2341.3	210.5	37,836	9.61
2000	1,930,842	73,980,216	85.9	887.9	144.7	40,705	6.82
2005	2,066,461	79,199,328	54.6	542.0	104.3	44,397	6.56
2007	2,138,676	81,365,584	47.8	471.4	94.1	46,080	6.40
2010	2,277,702	82,533,536	42.7	407.9	82.8	47,215	6.16
2014	2,432,722	85,930,752	35.7	324.9	66.0	49,539	5.94
2017	2,480,927	88,470,176	30.3	263.2	55.1	51,374	5.80
2020	2,523,581	90,974,512	26.3	220.5	47.4	53,291	5.77
2023	2,599,894	94,694,160	24.0	194.7	42.8	56,424	5.90

San Diego Summer Episodic On-Road Motor Vehicle Inventories With Updated Temp/RH Profiles
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,615,290	60,116,672	220	2196	201	37,319	9.61
2000	1,930,842	73,980,216	84	828	139	39,689	6.82
2005	2,066,461	79,199,328	53	505	101	43,181	6.56
2007	2,138,676	81,365,584	47	439	91	44,817	6.40
2010	2,277,702	82,533,536	42	381	80	45,936	6.16
2014	2,432,722	85,930,752	35	303	64	48,205	5.94
2017	2,480,927	88,470,176	30	246	53	49,992	5.80
2020	2,523,581	90,974,512	26	207	46	51,862	5.77
2023	2,599,894	94,694,160	24	183	42	54,913	5.90

Difference (Ver. 2.5021a - Ver. 2.5020a) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	-5.7	-145.8	-9.3	-517	0.00
2000	0	0	-2.3	-60.1	-6.0	-1,016	0.00
2005	0	0	-1.5	-37.2	-3.7	-1,216	0.00
2007	0	0	-1.3	-32.0	-3.2	-1,263	0.00
2010	0	0	-1.0	-27.3	-2.7	-1,279	0.00
2014	0	0	-0.8	-21.5	-2.1	-1,334	0.00
2017	0	0	-0.7	-17.0	-1.6	-1,382	0.00
2020	0	0	-0.5	-13.7	-1.3	-1,429	0.00
2023	0	0	-0.5	-11.8	-1.1	-1,511	0.00

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.5020a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	-2.51%	-6.23%	-4.43%	-1.37%	0.00%
2000	0.00%	0.00%	-2.63%	-6.77%	-4.11%	-2.50%	0.00%
2005	0.00%	0.00%	-2.71%	-6.85%	-3.56%	-2.74%	0.00%
2007	0.00%	0.00%	-2.62%	-6.78%	-3.41%	-2.74%	0.00%
2010	0.00%	0.00%	-2.46%	-6.69%	-3.28%	-2.71%	0.00%
2014	0.00%	0.00%	-2.29%	-6.60%	-3.14%	-2.69%	0.00%
2017	0.00%	0.00%	-2.17%	-6.45%	-2.99%	-2.69%	0.00%
2020	0.00%	0.00%	-2.09%	-6.22%	-2.83%	-2.68%	0.00%
2023	0.00%	0.00%	-2.03%	-6.08%	-2.68%	-2.68%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-58. Impact on San Francisco Bay Air Basin Inventory of Change 12.10

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,877,920	137,914,140	533.6	5124.9	515.7	80,787	24.46
2000	4,700,962	168,181,650	233.8	2179.6	364.4	95,856	17.05
2005	4,757,890	169,061,820	154.3	1321.5	264.7	96,931	15.47
2007	4,815,908	170,597,250	132.7	1115.4	233.8	98,995	14.79
2010	5,024,152	171,597,970	116.6	943.9	200.2	100,898	13.99
2014	5,426,317	181,193,380	98.7	752.5	157.9	106,970	13.43
2017	5,621,512	188,394,240	86.0	617.8	132.7	113,179	13.07
2020	5,754,138	195,584,700	76.0	514.4	114.4	118,446	12.99
2023	5,893,056	202,578,720	69.8	444.3	102.4	123,719	13.11

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated Temp/RH Profiles
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,877,920	137,914,140	519.4	4752.4	488.0	79,483	24.46
2000	4,700,962	168,181,650	226.9	1997.8	346.3	92,987	17.05
2005	4,757,890	169,061,820	149.5	1211.0	253.5	93,745	15.47
2007	4,815,908	170,597,250	128.7	1023.1	224.4	95,720	14.79
2010	5,024,152	171,597,970	113.4	866.8	192.4	97,561	13.99
2014	5,426,317	181,193,380	96.1	692.4	152.0	103,433	13.43
2017	5,621,512	188,394,240	84.0	569.9	128.0	109,430	13.07
2020	5,754,138	195,584,700	74.3	476.7	110.7	114,527	12.99
2023	5,893,056	202,578,720	68.3	413.6	99.3	119,639	13.11

Difference (Ver. 2.5021a - Ver. 2.5020a) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	-14.2	-372.5	-27.6	-1,304	0.00
2000	0	0	-7.0	-181.8	-18.0	-2,868	0.00
2005	0	0	-4.8	-110.5	-11.3	-3,186	0.00
2007	0	0	-4.0	-92.3	-9.4	-3,276	0.00
2010	0	0	-3.2	-77.1	-7.8	-3,337	0.00
2014	0	0	-2.5	-60.1	-5.9	-3,537	0.00
2017	0	0	-2.1	-47.9	-4.6	-3,749	0.00
2020	0	0	-1.7	-37.7	-3.7	-3,919	0.00
2023	0	0	-1.5	-30.7	-3.1	-4,080	0.00

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.5020a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	-2.66%	-7.27%	-5.36%	-1.61%	0.00%
2000	0.00%	0.00%	-2.98%	-8.34%	-4.95%	-2.99%	0.00%
2005	0.00%	0.00%	-3.10%	-8.36%	-4.26%	-3.29%	0.00%
2007	0.00%	0.00%	-2.98%	-8.28%	-4.03%	-3.31%	0.00%
2010	0.00%	0.00%	-2.79%	-8.17%	-3.91%	-3.31%	0.00%
2014	0.00%	0.00%	-2.55%	-7.99%	-3.71%	-3.31%	0.00%
2017	0.00%	0.00%	-2.39%	-7.75%	-3.49%	-3.31%	0.00%
2020	0.00%	0.00%	-2.23%	-7.32%	-3.25%	-3.31%	0.00%
2023	0.00%	0.00%	-2.10%	-6.90%	-3.03%	-3.30%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-59. Impact on San Joaquin Valley Air Basin Inventory of Change 12.10

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,371,368	58,661,744	230.8	2441.8	342.4	43,703	23.49
2000	1,936,457	83,696,632	112.4	1130.8	356.7	57,176	13.86
2005	2,141,954	91,088,576	81.5	736.0	284.7	61,976	13.66
2007	2,254,768	94,929,168	73.8	657.2	258.4	64,939	13.13
2010	2,493,321	98,407,960	70.3	604.7	226.2	67,570	12.17
2014	2,852,481	107,711,930	65.4	539.9	181.0	74,969	11.52
2017	3,115,979	116,216,320	60.8	478.8	155.1	81,282	10.89
2020	3,309,582	124,193,760	55.7	419.0	134.7	87,075	10.64
2023	3,430,952	131,662,430	51.1	360.2	119.6	93,918	10.55

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Updated Temp/RH Profiles
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,371,368	58,661,744	229.3	2274.8	329.8	43,044	23.49
2000	1,936,457	83,696,632	111.3	1027.2	345.6	55,489	13.86
2005	2,141,954	91,088,576	81.3	668.2	277.1	59,889	13.66
2007	2,254,768	94,929,168	73.7	597.0	251.6	62,723	13.13
2010	2,493,321	98,407,960	70.3	549.8	220.2	65,272	12.17
2014	2,852,481	107,711,930	65.5	490.4	176.2	72,448	11.52
2017	3,115,979	116,216,320	60.9	435.1	151.1	78,545	10.89
2020	3,309,582	124,193,760	55.9	381.0	131.3	84,127	10.64
2023	3,430,952	131,662,430	51.3	327.0	116.7	90,667	10.55

Difference (Ver. 2.5021a - Ver. 2.5020a) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	-1.5	-167.0	-12.6	-659	0.00
2000	0	0	-1.1	-103.5	-11.2	-1,686	0.00
2005	0	0	-0.1	-67.9	-7.7	-2,087	0.00
2007	0	0	-0.1	-60.2	-6.8	-2,216	0.00
2010	0	0	0.0	-54.9	-6.0	-2,298	0.00
2014	0	0	0.1	-49.5	-4.8	-2,521	0.00
2017	0	0	0.2	-43.8	-4.0	-2,736	0.00
2020	0	0	0.2	-38.0	-3.4	-2,948	0.00
2023	0	0	0.2	-33.1	-2.9	-3,251	0.00

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.5020a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	-0.63%	-6.84%	-3.69%	-1.51%	0.00%
2000	0.00%	0.00%	-0.97%	-9.16%	-3.13%	-2.95%	0.00%
2005	0.00%	0.00%	-0.18%	-9.22%	-2.70%	-3.37%	0.00%
2007	0.00%	0.00%	-0.10%	-9.15%	-2.62%	-3.41%	0.00%
2010	0.00%	0.00%	0.01%	-9.08%	-2.64%	-3.40%	0.00%
2014	0.00%	0.00%	0.14%	-9.16%	-2.64%	-3.36%	0.00%
2017	0.00%	0.00%	0.26%	-9.14%	-2.59%	-3.37%	0.00%
2020	0.00%	0.00%	0.38%	-9.08%	-2.50%	-3.39%	0.00%
2023	0.00%	0.00%	0.44%	-9.20%	-2.40%	-3.46%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-60. Impact on South Coast Air Basin Inventory of Change 12.10

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5020a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,534,778	267,446,580	970.4	9422.9	943.0	162,610	46.35
2000	8,888,948	316,402,500	396.8	3896.1	677.3	177,802	31.97
2005	9,572,533	342,575,040	259.4	2439.1	510.6	199,026	31.73
2007	9,927,914	355,912,900	222.9	2082.6	457.8	207,711	30.87
2010	10,537,437	365,239,650	190.0	1734.1	400.3	213,641	29.58
2014	11,072,442	377,542,370	150.3	1321.6	318.4	225,504	28.07
2017	11,422,403	389,182,050	128.2	1080.8	272.4	236,806	27.30
2020	11,709,373	400,561,250	112.6	914.0	238.4	246,987	26.88
2023	11,886,469	407,786,110	102.2	797.5	216.3	256,607	26.77

South Coast Summer Episodic On-Road Motor Vehicle Inventories With Updated Temp/RH Profiles
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,534,778	267,446,580	957.7	8953.6	902.1	160,572	46.35
2000	8,888,948	316,402,500	390.4	3640.6	650.2	173,500	31.97
2005	9,572,533	342,575,040	255.8	2275.2	493.0	193,670	31.73
2007	9,927,914	355,912,900	220.0	1942.5	442.7	202,045	30.87
2010	10,537,437	365,239,650	187.8	1618.8	387.7	207,811	29.58
2014	11,072,442	377,542,370	148.8	1233.9	309.0	219,395	28.07
2017	11,422,403	389,182,050	127.1	1010.1	265.0	230,433	27.30
2020	11,709,373	400,561,250	111.7	855.6	232.3	240,364	26.88
2023	11,886,469	407,786,110	101.5	747.3	211.2	249,784	26.77

Difference (Ver. 2.5021a - Ver. 2.5020a8) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	-12.6	-469.3	-40.8	-2,038	0.00
2000	0	0	-6.4	-255.4	-27.1	-4,302	0.00
2005	0	0	-3.6	-163.8	-17.6	-5,356	0.00
2007	0	0	-2.9	-140.1	-15.1	-5,666	0.00
2010	0	0	-2.2	-115.3	-12.6	-5,830	0.00
2014	0	0	-1.5	-87.7	-9.3	-6,109	0.00
2017	0	0	-1.2	-70.7	-7.5	-6,374	0.00
2020	0	0	-0.9	-58.3	-6.1	-6,622	0.00
2023	0	0	-0.7	-50.1	-5.2	-6,823	0.00

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.5020a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	-1.30%	-4.98%	-4.33%	-1.25%	0.00%
2000	0.00%	0.00%	-1.61%	-6.56%	-3.99%	-2.42%	0.00%
2005	0.00%	0.00%	-1.39%	-6.72%	-3.45%	-2.69%	0.00%
2007	0.00%	0.00%	-1.31%	-6.73%	-3.30%	-2.73%	0.00%
2010	0.00%	0.00%	-1.17%	-6.65%	-3.15%	-2.73%	0.00%
2014	0.00%	0.00%	-1.02%	-6.63%	-2.93%	-2.71%	0.00%
2017	0.00%	0.00%	-0.90%	-6.54%	-2.75%	-2.69%	0.00%
2020	0.00%	0.00%	-0.79%	-6.38%	-2.56%	-2.68%	0.00%
2023	0.00%	0.00%	-0.72%	-6.28%	-2.39%	-2.66%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.11 UPDATES TO SPEED DISTRIBUTIONS

Please refer to Section 3.3.2. This updated the latest speed profiles provided by the local transportation planning agencies. The incremental changes vary by region but tend to be small.

Table 12-61. Impact on Statewide Inventory of Change 12.11

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.50.21a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,378,444	689,422,780	2,527	24,167	2,594	424,355	145.38
2000	22,181,680	839,893,380	1,079	10,004	2,066	477,035	94.16
2005	23,631,408	895,163,970	720	6,222	1,571	514,640	92.19
2007	24,447,824	923,793,790	631	5,402	1,417	534,851	89.30
2010	26,118,692	946,227,260	564	4,682	1,243	550,560	84.68
2014	28,247,534	999,628,350	480	3,816	999	590,254	80.78
2017	29,519,084	1,044,801,900	423	3,214	858	625,966	77.98
2020	30,517,106	1,089,757,700	379	2,752	754	659,117	77.12
2023	31,298,652	1,130,831,900	347	2,378	682	694,108	77.51

Statewide Summer Episodic On-Road Motor Vehicle Inventories With Updated Speed Distributions
(Calculated Using EMFAC2010 ver 2.50.22)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,378,444	689,422,780	2,561	25,718	2,646	430,610	145.38
2000	22,181,680	839,893,380	1,097	10,839	2,102	490,506	94.16
2005	23,631,408	895,163,970	729	6,751	1,592	531,002	92.19
2007	24,447,824	923,793,790	639	5,861	1,435	552,087	89.30
2010	26,118,692	946,227,260	570	5,075	1,257	568,306	84.69
2014	28,247,534	999,628,350	484	4,137	1,010	609,254	80.83
2017	29,519,084	1,044,801,900	427	3,483	867	646,157	78.08
2020	30,517,106	1,089,757,700	381	2,978	762	680,428	77.26
2023	31,298,652	1,130,831,900	349	2,573	688	716,609	77.67

Difference (Ver. 2.5022 - Ver. 2.5021a) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	34.0	1550.9	52.1	6,255	0.00
2000	0	0	17.7	835.0	36.3	13,471	0.00
2005	0	0	9.2	528.9	21.2	16,362	0.00
2007	0	0	7.9	458.5	18.2	17,236	0.00
2010	0	0	5.6	393.1	14.4	17,746	0.01
2014	0	0	4.2	320.8	11.3	19,000	0.05
2017	0	0	3.8	268.8	9.4	20,191	0.10
2020	0	0	2.4	226.3	7.5	21,311	0.14
2023	0	0	2.2	194.7	6.2	22,501	0.16

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5021a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.35%	6.42%	2.01%	1.47%	0.00%
2000	0.00%	0.00%	1.64%	8.35%	1.76%	2.82%	0.00%
2005	0.00%	0.00%	1.28%	8.50%	1.35%	3.18%	0.00%
2007	0.00%	0.00%	1.26%	8.49%	1.28%	3.22%	0.00%
2010	0.00%	0.00%	1.00%	8.40%	1.16%	3.22%	0.01%
2014	0.00%	0.00%	0.87%	8.41%	1.13%	3.22%	0.07%
2017	0.00%	0.00%	0.90%	8.36%	1.10%	3.23%	0.12%
2020	0.00%	0.00%	0.63%	8.22%	1.00%	3.23%	0.18%
2023	0.00%	0.00%	0.64%	8.19%	0.91%	3.24%	0.21%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-62. Impact on Sacramento Valley Air Basin Inventory of Change 12.11

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,362,128	51,431,120	212.0	2058.3	205.6	31,912	11.56
2000	1,634,783	63,336,324	88.2	807.1	170.3	36,070	7.40
2005	1,780,274	68,268,976	61.5	503.3	130.6	38,864	7.19
2007	1,860,962	70,353,144	55.4	446.0	118.7	40,550	6.97
2010	2,030,439	72,283,584	51.6	401.0	105.0	41,620	6.61
2014	2,285,447	78,601,864	46.6	346.2	87.4	45,687	6.44
2017	2,424,266	82,960,408	42.1	297.5	76.4	48,693	6.27
2020	2,522,620	87,144,888	38.0	255.4	68.1	51,535	6.25
2023	2,604,611	91,737,016	34.9	215.3	62.0	54,684	6.35

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Updated Speed Distributions
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,362,128	51,431,120	216.2	2252.9	211.8	32,597	11.56
2000	1,634,783	63,336,324	90.2	903.2	174.6	37,528	7.40
2005	1,780,274	68,268,976	62.5	564.3	133.1	40,636	7.19
2007	1,860,962	70,353,144	56.2	500.1	120.8	42,420	6.97
2010	2,030,439	72,283,584	52.3	448.6	106.8	43,516	6.61
2014	2,285,447	78,601,864	47.1	387.2	88.9	47,749	6.44
2017	2,424,266	82,960,408	42.6	332.6	77.7	50,887	6.27
2020	2,522,620	87,144,888	38.4	285.0	69.2	53,854	6.26
2023	2,604,611	91,737,016	35.1	240.2	62.8	57,152	6.37

Difference (Ver. 2.5022 - Ver. 2.5021a) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	4.2	194.6	6.2	685	0.00
2000	0	0	2.0	96.1	4.3	1,458	0.00
2005	0	0	1.0	61.0	2.5	1,772	0.00
2007	0	0	0.8	54.1	2.1	1,870	0.00
2010	0	0	0.7	47.6	1.8	1,896	0.00
2014	0	0	0.5	41.0	1.5	2,062	0.00
2017	0	0	0.5	35.1	1.3	2,194	0.00
2020	0	0	0.4	29.6	1.1	2,319	0.01
2023	0	0	0.2	24.9	0.8	2,468	0.02

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.5021a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.96%	9.45%	3.00%	2.15%	0.01%
2000	0.00%	0.00%	2.23%	11.91%	2.50%	4.04%	0.06%
2005	0.00%	0.00%	1.57%	12.13%	1.88%	4.56%	0.03%
2007	0.00%	0.00%	1.51%	12.14%	1.77%	4.61%	-0.01%
2010	0.00%	0.00%	1.28%	11.86%	1.73%	4.56%	0.03%
2014	0.00%	0.00%	1.17%	11.84%	1.69%	4.51%	0.04%
2017	0.00%	0.00%	1.10%	11.78%	1.68%	4.51%	0.07%
2020	0.00%	0.00%	0.96%	11.60%	1.56%	4.50%	0.20%
2023	0.00%	0.00%	0.66%	11.57%	1.33%	4.51%	0.26%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-63. Impact on San Diego Air Basin Inventory of Change 12.11

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,615,290	60,116,672	220.0	2196.0	201.0	37,319	9.61
2000	1,930,842	73,980,216	84.0	828.0	139.0	39,689	6.82
2005	2,066,461	79,199,328	53.0	505.0	101.0	43,181	6.56
2007	2,138,676	81,365,584	47.0	439.0	91.0	44,817	6.40
2010	2,277,702	82,533,536	42.0	381.0	80.0	45,936	6.16
2014	2,432,722	85,930,752	35.0	303.0	64.0	48,205	5.94
2017	2,480,927	88,470,176	30.0	246.0	53.0	49,992	5.80
2020	2,523,581	90,974,512	26.0	207.0	46.0	51,862	5.77
2023	2,599,894	94,694,160	24.0	183.0	42.0	54,913	5.90

San Diego Summer Episodic On-Road Motor Vehicle Inventories With Updated Speed Distributions
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,615,290	60,116,672	223	2306	205	37,786	9.61
2000	1,930,842	73,980,216	85	878	141	40,606	6.82
2005	2,066,461	79,199,328	54	536	102	44,278	6.56
2007	2,138,676	81,365,584	47	467	92	45,956	6.40
2010	2,277,702	82,533,536	42	404	81	47,090	6.16
2014	2,432,722	85,930,752	35	322	65	49,408	5.95
2017	2,480,927	88,470,176	30	261	54	51,238	5.80
2020	2,523,581	90,974,512	26	219	47	53,150	5.78
2023	2,599,894	94,694,160	24	193	42	56,274	5.91

Difference (Ver. 2.5022 - Ver. 2.5021a) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	3.1	110.5	3.9	467	0.00
2000	0	0	0.7	50.3	2.2	917	0.00
2005	0	0	0.7	31.4	1.0	1,097	0.00
2007	0	0	0.0	27.6	1.0	1,139	0.00
2010	0	0	0.0	22.8	1.0	1,154	0.00
2014	0	0	0.2	18.8	0.6	1,203	0.01
2017	0	0	-0.1	14.9	1.1	1,246	0.00
2020	0	0	0.0	11.9	0.5	1,288	0.01
2023	0	0	-0.3	10.4	0.0	1,361	0.01

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.5021a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.40%	5.03%	1.92%	1.25%	-0.05%
2000	0.00%	0.00%	0.84%	6.08%	1.58%	2.31%	0.05%
2005	0.00%	0.00%	1.23%	6.22%	0.97%	2.54%	-0.03%
2007	0.00%	0.00%	-0.07%	6.28%	1.14%	2.54%	0.02%
2010	0.00%	0.00%	-0.01%	5.99%	1.27%	2.51%	0.02%
2014	0.00%	0.00%	0.49%	6.22%	0.98%	2.50%	0.09%
2017	0.00%	0.00%	-0.48%	6.07%	1.98%	2.49%	0.07%
2020	0.00%	0.00%	-0.16%	5.75%	1.14%	2.48%	0.10%
2023	0.00%	0.00%	-1.27%	5.71%	0.00%	2.48%	0.09%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-64. Impact on San Francisco Bay Air Basin Inventory of Change 12.11

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,877,920	137,914,140	519.4	4752.4	488.0	79,483	24.46
2000	4,700,962	168,181,650	226.9	1997.8	346.3	92,987	17.05
2005	4,757,890	169,061,820	149.5	1211.0	253.5	93,745	15.47
2007	4,815,908	170,597,250	128.7	1023.1	224.4	95,720	14.79
2010	5,024,152	171,597,970	113.4	866.8	192.4	97,561	13.99
2014	5,426,317	181,193,380	96.1	692.4	152.0	103,433	13.43
2017	5,621,512	188,394,240	84.0	569.9	128.0	109,430	13.07
2020	5,754,138	195,584,700	74.3	476.7	110.7	114,527	12.99
2023	5,893,056	202,578,720	68.3	413.6	99.3	119,639	13.11

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated Speed Distributions
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,877,920	137,914,140	525.7	5054.4	498.4	80,677	24.46
2000	4,700,962	168,181,650	230.3	2155.2	353.3	95,617	17.05
2005	4,757,890	169,061,820	151.3	1307.5	257.5	96,667	15.47
2007	4,815,908	170,597,250	130.1	1103.8	227.6	98,725	14.79
2010	5,024,152	171,597,970	114.4	934.3	195.0	100,622	13.99
2014	5,426,317	181,193,380	96.9	745.4	154.0	106,678	13.44
2017	5,621,512	188,394,240	84.5	612.5	129.5	112,871	13.09
2020	5,754,138	195,584,700	74.7	510.7	111.9	118,124	13.01
2023	5,893,056	202,578,720	68.6	441.8	100.3	123,383	13.13

Difference (Ver. 2.5022 - Ver. 2.5021a) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	6.3	302.0	10.4	1,194	0.00
2000	0	0	3.4	157.4	7.0	2,630	0.00
2005	0	0	1.8	96.5	4.0	2,922	0.00
2007	0	0	1.4	80.7	3.2	3,005	0.00
2010	0	0	1.0	67.5	2.6	3,061	0.00
2014	0	0	0.8	53.0	2.0	3,245	0.01
2017	0	0	0.5	42.6	1.5	3,441	0.02
2020	0	0	0.4	34.0	1.2	3,597	0.02
2023	0	0	0.3	28.2	1.0	3,744	0.02

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.5021a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.21%	6.35%	2.14%	1.50%	0.00%
2000	0.00%	0.00%	1.48%	7.88%	2.03%	2.83%	0.02%
2005	0.00%	0.00%	1.17%	7.97%	1.57%	3.12%	0.02%
2007	0.00%	0.00%	1.10%	7.88%	1.43%	3.14%	-0.02%
2010	0.00%	0.00%	0.92%	7.79%	1.34%	3.14%	0.03%
2014	0.00%	0.00%	0.80%	7.66%	1.30%	3.14%	0.05%
2017	0.00%	0.00%	0.60%	7.48%	1.21%	3.14%	0.15%
2020	0.00%	0.00%	0.55%	7.13%	1.05%	3.14%	0.15%
2023	0.00%	0.00%	0.49%	6.81%	0.97%	3.13%	0.19%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-65. Impact on San Joaquin Valley Air Basin Inventory of Change 12.11

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,371,368	58,661,744	229.3	2274.8	329.8	43,044	23.49
2000	1,936,457	83,696,632	111.3	1027.2	345.6	55,489	13.86
2005	2,141,954	91,088,576	81.3	668.2	277.1	59,889	13.66
2007	2,254,768	94,929,168	73.7	597.0	251.6	62,723	13.13
2010	2,493,321	98,407,960	70.3	549.8	220.2	65,272	12.17
2014	2,852,481	107,711,930	65.5	490.4	176.2	72,448	11.52
2017	3,115,979	116,216,320	60.9	435.1	151.1	78,545	10.89
2020	3,309,582	124,193,760	55.9	381.0	131.3	84,127	10.64
2023	3,430,952	131,662,430	51.3	327.0	116.7	90,667	10.55

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Updated Speed Distributions
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,371,368	58,661,744	233.6	2481.1	336.5	43,742	23.49
2000	1,936,457	83,696,632	113.8	1148.0	351.3	57,276	13.86
2005	2,141,954	91,088,576	82.6	746.8	280.5	62,098	13.66
2007	2,254,768	94,929,168	74.8	666.7	254.6	65,069	13.13
2010	2,493,321	98,407,960	71.2	613.4	222.8	67,704	12.17
2014	2,852,481	107,711,930	66.3	547.7	178.4	75,116	11.53
2017	3,115,979	116,216,320	61.5	485.7	153.0	81,442	10.90
2020	3,309,582	124,193,760	56.4	424.9	132.9	87,246	10.65
2023	3,430,952	131,662,430	51.7	365.1	118.1	94,108	10.57

Difference (Ver. 2.5022 - Ver. 2.5021a) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	4.3	206.3	6.7	698	0.00
2000	0	0	2.5	120.8	5.7	1,787	0.00
2005	0	0	1.3	78.6	3.4	2,209	0.00
2007	0	0	1.1	69.7	3.0	2,346	0.00
2010	0	0	0.9	63.6	2.6	2,432	0.00
2014	0	0	0.8	57.3	2.2	2,668	0.01
2017	0	0	0.6	50.6	1.9	2,897	0.01
2020	0	0	0.5	43.9	1.6	3,119	0.01
2023	0	0	0.4	38.1	1.4	3,441	0.02

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.5021a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.86%	9.07%	2.04%	1.62%	0.01%
2000	0.00%	0.00%	2.22%	11.76%	1.64%	3.22%	0.02%
2005	0.00%	0.00%	1.61%	11.77%	1.21%	3.69%	-0.01%
2007	0.00%	0.00%	1.50%	11.68%	1.18%	3.74%	-0.02%
2010	0.00%	0.00%	1.31%	11.57%	1.17%	3.73%	-0.01%
2014	0.00%	0.00%	1.20%	11.68%	1.23%	3.68%	0.06%
2017	0.00%	0.00%	1.05%	11.62%	1.23%	3.69%	0.06%
2020	0.00%	0.00%	0.87%	11.52%	1.24%	3.71%	0.13%
2023	0.00%	0.00%	0.85%	11.65%	1.19%	3.80%	0.17%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-66. Impact on South Coast Air Basin Inventory of Change 12.11

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5021a)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,534,778	267,446,580	957.7	8953.6	902.1	160,572	46.35
2000	8,888,948	316,402,500	390.4	3640.6	650.2	173,500	31.97
2005	9,572,533	342,575,040	255.8	2275.2	493.0	193,670	31.73
2007	9,927,914	355,912,900	220.0	1942.5	442.7	202,045	30.87
2010	10,537,437	365,239,650	187.8	1618.8	387.7	207,811	29.58
2014	11,072,442	377,542,370	148.8	1233.9	309.0	219,395	28.07
2017	11,422,403	389,182,050	127.1	1010.1	265.0	230,433	27.30
2020	11,709,373	400,561,250	111.7	855.6	232.3	240,364	26.88
2023	11,886,469	407,786,110	101.5	747.3	211.2	249,784	26.77

South Coast Summer Episodic On-Road Motor Vehicle Inventories With Updated Speed Distributions
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,534,778	267,446,580	968.1	9412.1	917.6	162,586	46.35
2000	8,888,948	316,402,500	395.9	3894.1	661.0	177,774	31.97
2005	9,572,533	342,575,040	258.7	2438.3	499.3	198,998	31.73
2007	9,927,914	355,912,900	222.3	2082.0	448.0	207,685	30.88
2010	10,537,437	365,239,650	189.5	1733.7	391.8	213,617	29.59
2014	11,072,442	377,542,370	150.0	1321.4	312.0	225,484	28.09
2017	11,422,403	389,182,050	127.9	1080.8	267.2	236,789	27.34
2020	11,709,373	400,561,250	112.4	914.2	234.1	246,976	26.93
2023	11,886,469	407,786,110	102.0	797.8	212.7	256,602	26.84

Difference (Ver. 2.5022 - Ver. 2.5021a) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	10.4	458.5	15.5	2,014	0.00
2000	0	0	5.5	253.5	10.8	4,274	0.00
2005	0	0	2.9	163.1	6.3	5,328	0.00
2007	0	0	2.3	139.5	5.3	5,640	0.01
2010	0	0	1.7	114.9	4.1	5,806	0.01
2014	0	0	1.2	87.5	3.0	6,089	0.02
2017	0	0	0.8	70.7	2.2	6,356	0.04
2020	0	0	0.7	58.6	1.8	6,612	0.05
2023	0	0	0.5	50.5	1.5	6,818	0.07

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.5021a)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	1.08%	5.12%	1.72%	1.25%	0.00%
2000	0.00%	0.00%	1.41%	6.96%	1.66%	2.46%	0.01%
2005	0.00%	0.00%	1.12%	7.17%	1.27%	2.75%	0.00%
2007	0.00%	0.00%	1.04%	7.18%	1.19%	2.79%	0.02%
2010	0.00%	0.00%	0.89%	7.10%	1.06%	2.79%	0.03%
2014	0.00%	0.00%	0.78%	7.09%	0.97%	2.78%	0.08%
2017	0.00%	0.00%	0.66%	7.00%	0.85%	2.76%	0.14%
2020	0.00%	0.00%	0.59%	6.85%	0.76%	2.75%	0.20%
2023	0.00%	0.00%	0.49%	6.76%	0.69%	2.73%	0.25%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.12 2004 - 2010 LEV 2 AND DIESEL BASELINE UPDATES

During the development of the LEV3 regulation it became apparent that many of the assumptions regarding the LEV2 program were obsolete and should be adjusted. Additionally, new registration data pointed out that many of our assumptions regarding light duty diesel technology were also obsolete. We updated EMFAC for what was actually sold. This generally reduced emissions, regardless of region.

Table 12-67. Impact on Statewide Inventory of Change 12.12

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.50.22)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,378,444	689,422,780	2,561	25,718	2,646	430,610	145.38
2000	22,181,680	839,893,380	1,097	10,839	2,102	490,506	94.16
2005	23,631,408	895,163,970	729	6,751	1,592	531,002	92.19
2007	24,447,824	923,793,790	639	5,861	1,435	552,087	89.30
2010	26,118,692	946,227,260	570	5,075	1,257	568,306	84.69
2014	28,247,534	999,628,350	484	4,137	1,010	609,254	80.83
2017	29,519,084	1,044,801,900	427	3,483	867	646,157	78.08
2020	30,517,106	1,089,757,700	381	2,978	762	680,428	77.26
2023	31,298,652	1,130,831,900	349	2,573	688	716,609	77.67

Statewide Summer Episodic On-Road Motor Vehicle Inventories With Revised Speed Profiles
(Calculated Using EMFAC2010 ver 2.50.23)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,502,400	683,627,650	2,555	25,299	2,596	428,274	144.37
2000	22,167,354	832,353,470	1,094	10,806	2,060	494,731	93.59
2005	23,289,752	876,678,210	706	6,519	1,515	518,485	89.72
2007	24,181,280	910,324,160	621	5,688	1,379	544,418	87.50
2010	26,118,692	946,227,200	563	4,998	1,241	572,977	84.05
2014	28,247,532	999,628,290	478	4,069	996	612,245	80.43
2017	29,519,082	1,044,801,800	422	3,426	854	648,525	77.69
2020	30,517,098	1,089,757,600	377	2,932	749	682,840	76.97
2023	31,298,648	1,130,831,700	345	2,532	676	716,693	77.42

Difference (Ver. 2.5023 - Ver. 2.5022) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	123,956	-5,795,130	-6.5	-419.5	-50.1	-2,336	-1.01
2000	-14,326	-7,539,910	-3.3	-32.5	-42.0	4,225	-0.57
2005	-341,656	-18,485,760	-22.8	-231.6	-76.8	-12,517	-2.47
2007	-266,544	-13,469,630	-17.6	-172.8	-55.5	-7,669	-1.80
2010	0	-60	-7.5	-76.8	-15.7	4,671	-0.64
2014	-2	-60	-6.1	-68.2	-14.2	2,991	-0.40
2017	-2	-100	-5.3	-57.1	-13.5	2,368	-0.39
2020	-8	-100	-3.8	-45.5	-13.0	2,412	-0.29
2023	-4	-200	-3.6	-41.3	-11.6	84	-0.25

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5022)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.67%	-0.84%	-0.25%	-1.63%	-1.89%	-0.54%	-0.70%
2000	-0.06%	-0.90%	-0.30%	-0.30%	-2.00%	0.86%	-0.60%
2005	-1.45%	-2.07%	-3.13%	-3.43%	-4.82%	-2.36%	-2.68%
2007	-1.09%	-1.46%	-2.75%	-2.95%	-3.87%	-1.39%	-2.02%
2010	0.00%	0.00%	-1.31%	-1.51%	-1.25%	0.82%	-0.76%
2014	0.00%	0.00%	-1.25%	-1.65%	-1.41%	0.49%	-0.49%
2017	0.00%	0.00%	-1.25%	-1.64%	-1.55%	0.37%	-0.50%
2020	0.00%	0.00%	-1.00%	-1.53%	-1.71%	0.35%	-0.38%
2023	0.00%	0.00%	-1.02%	-1.61%	-1.69%	0.01%	-0.32%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-68. Impact on Sacramento Valley Air Basin Inventory of Change 12.12

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,362,128	51,431,120	216.2	2252.9	211.8	32,597	11.56
2000	1,634,783	63,336,324	90.2	903.2	174.6	37,528	7.40
2005	1,780,274	68,268,976	62.5	564.3	133.1	40,636	7.19
2007	1,860,962	70,353,144	56.2	500.1	120.8	42,420	6.97
2010	2,030,439	72,283,584	52.3	448.6	106.8	43,516	6.61
2014	2,285,447	78,601,864	47.1	387.2	88.9	47,749	6.44
2017	2,424,266	82,960,408	42.6	332.6	77.7	50,887	6.27
2020	2,522,620	87,144,888	38.4	285.0	69.2	53,854	6.26
2023	2,604,611	91,737,016	35.1	240.2	62.8	57,152	6.37

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,373,306	50,465,080	217.3	2221.3	204.7	32,071	11.36
2000	1,628,305	62,080,196	90.9	907.3	167.8	37,499	7.27
2005	1,731,112	65,796,664	60.4	545.3	124.0	39,864	6.94
2007	1,823,069	68,580,760	54.5	485.5	114.7	42,217	6.79
2010	2,030,438	72,283,584	52.1	447.0	106.6	45,182	6.61
2014	2,285,447	78,601,864	47.0	385.8	88.7	49,473	6.44
2017	2,424,266	82,960,408	42.4	331.8	77.6	52,740	6.27
2020	2,522,620	87,144,880	38.2	285.1	69.1	55,776	6.26
2023	2,604,610	91,737,016	35.0	240.5	62.7	59,082	6.36

Difference (Ver. 2.5023 - Ver. 2.5022) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	11178	-966040	1.1	-31.6	-7.1	-526	-0.20
2000	-6478	-1256128	0.7	4.1	-6.8	-29	-0.13
2005	-49162	-2472312	-2.1	-19.0	-9.1	-772	-0.25
2007	-37893	-1772384	-1.7	-14.6	-6.1	-203	-0.18
2010	-1	0	-0.2	-1.6	-0.2	1,666	0.00
2014	0	0	-0.1	-1.4	-0.2	1,724	0.00
2017	0	0	-0.2	-0.8	-0.1	1,853	0.00
2020	0	-8	-0.2	0.1	-0.1	1,922	0.00
2023	-1	0	-0.1	0.3	-0.1	1,930	-0.01

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.5022)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.82%	-1.88%	0.51%	-1.40%	-3.37%	-1.61%	-1.74%
2000	-0.40%	-1.98%	0.72%	0.46%	-3.88%	-0.08%	-1.74%
2005	-2.76%	-3.62%	-3.36%	-3.37%	-6.87%	-1.90%	-3.46%
2007	-2.04%	-2.52%	-2.99%	-2.92%	-5.04%	-0.48%	-2.57%
2010	0.00%	0.00%	-0.39%	-0.37%	-0.19%	3.83%	0.03%
2014	0.00%	0.00%	-0.29%	-0.37%	-0.21%	3.61%	0.00%
2017	0.00%	0.00%	-0.43%	-0.23%	-0.18%	3.64%	0.04%
2020	0.00%	0.00%	-0.44%	0.02%	-0.22%	3.57%	0.01%
2023	0.00%	0.00%	-0.26%	0.12%	-0.12%	3.38%	-0.12%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-69. Impact on San Diego Air Basin Inventory of Change 12.12

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,615,290	60,116,672	223.0	2306.0	205.0	37,786	9.61
2000	1,930,842	73,980,216	85.0	878.0	141.0	40,606	6.82
2005	2,066,461	79,199,328	54.0	536.0	102.0	44,278	6.56
2007	2,138,676	81,365,584	47.0	467.0	92.0	45,956	6.40
2010	2,277,702	82,533,536	42.0	404.0	81.0	47,090	6.16
2014	2,432,722	85,930,752	35.0	322.0	65.0	49,408	5.95
2017	2,480,927	88,470,176	30.0	261.0	54.0	51,238	5.80
2020	2,523,581	90,974,512	26.0	219.0	47.0	53,150	5.78
2023	2,599,894	94,694,160	24.0	193.0	42.0	56,274	5.91

San Diego Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,609,040	59,230,324	222	2268	201	37,710	9.50
2000	1,925,116	73,764,616	83	860	138	41,257	6.77
2005	2,054,304	78,582,088	52	523	98	44,004	6.46
2007	2,129,484	80,890,632	46	458	90	45,845	6.33
2010	2,277,701	82,533,528	41	397	81	46,939	6.14
2014	2,432,723	85,930,784	35	317	64	49,290	5.93
2017	2,480,928	88,470,224	30	257	54	51,132	5.79
2020	2,523,582	90,974,536	26	216	46	53,051	5.76
2023	2,599,896	94,694,192	23	190	42	55,836	5.89

Difference (Ver. 2.5023 - Ver. 2.5022) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-6,250	-886,348	-1.4	-38.0	-4.4	-76	-0.11
2000	-5,726	-215,600	-2.3	-18.5	-2.9	651	-0.05
2005	-12,157	-617,240	-2.0	-13.2	-3.5	-274	-0.10
2007	-9,192	-474,952	-1.1	-9.1	-2.2	-111	-0.07
2010	-1	-8	-0.6	-6.9	-0.3	-151	-0.02
2014	1	32	-0.3	-5.3	-0.6	-118	-0.02
2017	1	48	-0.5	-4.0	-0.2	-106	-0.01
2020	1	24	-0.3	-3.4	-0.6	-99	-0.02
2023	2	32	-0.6	-2.9	-0.2	-438	-0.02

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.5022)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-0.39%	-1.47%	-0.64%	-1.65%	-2.14%	-0.20%	-1.13%
2000	-0.30%	-0.29%	-2.68%	-2.11%	-2.02%	1.60%	-0.74%
2005	-0.59%	-0.78%	-3.70%	-2.46%	-3.46%	-0.62%	-1.54%
2007	-0.43%	-0.58%	-2.32%	-1.95%	-2.36%	-0.24%	-1.04%
2010	0.00%	0.00%	-1.46%	-1.71%	-0.40%	-0.32%	-0.35%
2014	0.00%	0.00%	-0.76%	-1.64%	-0.95%	-0.24%	-0.35%
2017	0.00%	0.00%	-1.58%	-1.55%	-0.29%	-0.21%	-0.18%
2020	0.00%	0.00%	-1.16%	-1.55%	-1.38%	-0.19%	-0.33%
2023	0.00%	0.00%	-2.38%	-1.49%	-0.46%	-0.78%	-0.41%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-70. Impact on San Francisco Bay Air Basin Inventory of Change 12.12

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,877,920	137,914,140	525.7	5054.4	498.4	80,677	24.46
2000	4,700,962	168,181,650	230.3	2155.2	353.3	95,617	17.05
2005	4,757,890	169,061,820	151.3	1307.5	257.5	96,667	15.47
2007	4,815,908	170,597,250	130.1	1103.8	227.6	98,725	14.79
2010	5,024,152	171,597,970	114.4	934.3	195.0	100,622	13.99
2014	5,426,317	181,193,380	96.9	745.4	154.0	106,678	13.44
2017	5,621,512	188,394,240	84.5	612.5	129.5	112,871	13.09
2020	5,754,138	195,584,700	74.7	510.7	111.9	118,124	13.01
2023	5,893,056	202,578,720	68.6	441.8	100.3	123,383	13.13

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,932,100	138,236,160	531.2	4998.5	490.3	80,545	24.45
2000	4,709,472	166,475,900	234.1	2178.4	347.5	95,294	16.97
2005	4,663,679	164,225,200	149.6	1284.4	246.8	95,231	15.13
2007	4,742,242	167,109,980	128.6	1087.4	220.6	98,753	14.54
2010	5,024,152	171,597,970	114.4	934.3	195.0	103,886	13.99
2014	5,426,317	181,193,380	96.9	745.4	154.0	110,150	13.44
2017	5,621,512	188,394,240	84.5	612.5	129.5	116,571	13.09
2020	5,754,138	195,584,690	74.7	510.7	111.9	122,008	13.01
2023	5,893,057	202,578,720	68.6	441.8	100.3	127,438	13.13

Difference (Ver. 2.5023 - Ver. 2.5022) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	54,180	322,020	5.5	-55.9	-8.1	-132	-0.01
2000	8,510	-1,705,750	3.8	23.2	-5.8	-323	-0.08
2005	-94,211	-4,836,620	-1.7	-23.1	-10.7	-1,436	-0.34
2007	-73,666	-3,487,270	-1.5	-16.4	-7.0	28	-0.25
2010	0	0	0.0	0.0	0.0	3,264	0.00
2014	0	0	0.0	0.0	0.0	3,472	0.00
2017	0	0	0.0	0.0	0.0	3,700	0.00
2020	0	-10	0.0	0.0	0.0	3,884	0.00
2023	1	0	0.0	0.0	0.0	4,055	0.00

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.5022)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1.40%	0.23%	1.04%	-1.11%	-1.62%	-0.16%	-0.04%
2000	0.18%	-1.01%	1.64%	1.08%	-1.64%	-0.34%	-0.46%
2005	-1.98%	-2.86%	-1.11%	-1.77%	-4.17%	-1.49%	-2.22%
2007	-1.53%	-2.04%	-1.13%	-1.49%	-3.09%	0.03%	-1.67%
2010	0.00%	0.00%	0.04%	0.00%	-0.01%	3.24%	0.03%
2014	0.00%	0.00%	-0.03%	0.00%	-0.02%	3.25%	-0.03%
2017	0.00%	0.00%	0.01%	0.00%	0.04%	3.28%	-0.01%
2020	0.00%	0.00%	0.01%	0.00%	-0.04%	3.29%	-0.01%
2023	0.00%	0.00%	0.05%	0.00%	-0.04%	3.29%	0.04%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-71. Impact on San Joaquin Valley Air Basin Inventory of Change 12.12

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,371,368	58,661,744	233.6	2481.1	336.5	43,742	23.49
2000	1,936,457	83,696,632	113.8	1148.0	351.3	57,276	13.86
2005	2,141,954	91,088,576	82.6	746.8	280.5	62,098	13.66
2007	2,254,768	94,929,168	74.8	666.7	254.6	65,069	13.13
2010	2,493,321	98,407,960	71.2	613.4	222.8	67,704	12.17
2014	2,852,481	107,711,930	66.3	547.7	178.4	75,116	11.53
2017	3,115,979	116,216,320	61.5	485.7	153.0	81,442	10.90
2020	3,309,582	124,193,760	56.4	424.9	132.9	87,246	10.65
2023	3,430,952	131,662,430	51.7	365.1	118.1	94,108	10.57

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,378,730	58,052,672	229.7	2405.6	329.7	43,532	23.36
2000	1,925,682	82,513,360	112.3	1134.9	345.2	57,681	13.74
2005	2,101,642	88,853,616	80.5	728.9	273.4	62,164	13.43
2007	2,223,821	93,314,824	73.5	656.7	250.2	65,958	12.97
2010	2,493,321	98,407,960	71.1	612.7	223.0	70,222	12.16
2014	2,852,481	107,711,920	66.1	544.1	178.4	77,480	11.51
2017	3,115,978	116,216,310	61.4	482.0	153.0	84,089	10.88
2020	3,309,581	124,193,740	56.3	421.5	133.0	90,244	10.64
2023	3,430,952	131,662,410	51.4	355.9	117.9	95,735	10.54

Difference (Ver. 2.5023 - Ver. 2.5022) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,362	-609,072	-3.9	-75.5	-6.8	-210	-0.13
2000	-10,775	-1,183,272	-1.5	-13.1	-6.1	405	-0.12
2005	-40,312	-2,234,960	-2.1	-17.9	-7.1	66	-0.23
2007	-30,947	-1,614,344	-1.3	-10.0	-4.4	889	-0.16
2010	0	0	-0.1	-0.7	0.2	2,518	-0.01
2014	0	-10	-0.2	-3.6	0.0	2,364	-0.02
2017	-1	-10	-0.1	-3.7	0.0	2,647	-0.02
2020	-1	-20	-0.1	-3.4	0.1	2,998	-0.01
2023	0	-20	-0.3	-9.2	-0.2	1,627	-0.03

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.5022)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.54%	-1.04%	-1.68%	-3.04%	-2.01%	-0.48%	-0.57%
2000	-0.56%	-1.41%	-1.35%	-1.15%	-1.74%	0.71%	-0.86%
2005	-1.88%	-2.45%	-2.50%	-2.40%	-2.54%	0.11%	-1.67%
2007	-1.37%	-1.70%	-1.69%	-1.50%	-1.71%	1.37%	-1.20%
2010	0.00%	0.00%	-0.12%	-0.11%	0.08%	3.72%	-0.11%
2014	0.00%	0.00%	-0.36%	-0.65%	0.03%	3.15%	-0.16%
2017	0.00%	0.00%	-0.17%	-0.76%	0.01%	3.25%	-0.15%
2020	0.00%	0.00%	-0.15%	-0.80%	0.05%	3.44%	-0.07%
2023	0.00%	0.00%	-0.51%	-2.51%	-0.20%	1.73%	-0.29%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-72. Impact on South Coast Air Basin Inventory of Change 12.12

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5022)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,534,778	267,446,580	968.1	9412.1	917.6	162,586	46.35
2000	8,888,948	316,402,500	395.9	3894.1	661.0	177,774	31.97
2005	9,572,533	342,575,040	258.7	2438.3	499.3	198,998	31.73
2007	9,927,914	355,912,900	222.3	2082.0	448.0	207,685	30.88
2010	10,537,437	365,239,650	189.5	1733.7	391.8	213,617	29.59
2014	11,072,442	377,542,370	150.0	1321.4	312.0	225,484	28.09
2017	11,422,403	389,182,050	127.9	1080.8	267.2	236,789	27.34
2020	11,709,373	400,561,250	112.4	914.2	234.1	246,976	26.93
2023	11,886,469	407,786,110	102.0	797.8	212.7	256,602	26.84

South Coast Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,537,252	264,378,460	958.8	9212.1	902.3	162,405	46.15
2000	8,885,995	315,646,460	390.5	3837.3	651.1	182,423	31.90
2005	9,547,117	340,782,370	249.1	2345.5	475.7	193,041	30.44
2007	9,907,570	354,557,890	215.1	2009.8	429.3	201,968	29.78
2010	10,537,439	365,239,680	185.0	1685.7	379.6	209,404	28.71
2014	11,072,440	377,542,340	146.4	1281.8	301.7	219,670	27.45
2017	11,422,400	389,182,020	125.2	1049.2	257.7	230,012	26.80
2020	11,709,370	400,561,150	110.1	888.9	225.5	239,553	26.52
2023	11,886,466	407,786,080	100.1	778.1	204.9	248,366	26.50

Difference (Ver. 2.5023 - Ver. 2.5022) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	2,474	-3,068,120	-9.3	-200.0	-15.3	-181	-0.20
2000	-2,953	-756,040	-5.4	-56.8	-9.9	4,649	-0.07
2005	-25,416	-1,792,670	-9.6	-92.8	-23.6	-5,957	-1.29
2007	-20,344	-1,355,010	-7.2	-72.2	-18.7	-5,717	-1.10
2010	2	30	-4.5	-48.0	-12.2	-4,213	-0.88
2014	-2	-30	-3.6	-39.6	-10.3	-5,814	-0.64
2017	-3	-30	-2.7	-31.6	-9.5	-6,777	-0.54
2020	-3	-100	-2.3	-25.3	-8.6	-7,423	-0.41
2023	-3	-30	-1.9	-19.7	-7.8	-8,236	-0.34

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.5022)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.03%	-1.15%	-0.96%	-2.12%	-1.67%	-0.11%	-0.42%
2000	-0.03%	-0.24%	-1.37%	-1.46%	-1.49%	2.62%	-0.20%
2005	-0.27%	-0.52%	-3.70%	-3.80%	-4.73%	-2.99%	-4.08%
2007	-0.20%	-0.38%	-3.23%	-3.47%	-4.18%	-2.75%	-3.56%
2010	0.00%	0.00%	-2.39%	-2.77%	-3.11%	-1.97%	-2.96%
2014	0.00%	0.00%	-2.42%	-3.00%	-3.29%	-2.58%	-2.28%
2017	0.00%	0.00%	-2.14%	-2.92%	-3.57%	-2.86%	-1.99%
2020	0.00%	0.00%	-2.03%	-2.77%	-3.69%	-3.01%	-1.54%
2023	0.00%	0.00%	-1.82%	-2.47%	-3.65%	-3.21%	-1.28%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.13 REGIONAL VMT AND SPEED DISTRIBUTIONS (SECOND ROUND)

Please refer to Section 3.3.2. This change is complex and highly regional specific. In general, the future VMT projections are lower.

Table 12-73. Impact on Statewide Inventory of Change 12.13

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,502,400	683,627,650	2,555	25,299	2,596	428,274	144.37
2000	22,167,354	832,353,470	1,094	10,806	2,060	494,731	93.59
2005	23,289,752	876,678,210	706	6,519	1,515	518,485	89.72
2007	24,181,280	910,324,160	621	5,688	1,379	544,418	87.50
2010	26,118,692	946,227,200	563	4,998	1,241	572,977	84.05
2014	28,247,532	999,628,290	478	4,069	996	612,245	80.43
2017	29,519,082	1,044,801,800	422	3,426	854	648,525	77.69
2020	30,517,098	1,089,757,600	377	2,932	749	682,840	76.97
2023	31,298,648	1,130,831,700	345	2,532	676	716,693	77.42

Statewide Summer Episodic On-Road Motor Vehicle Inventories Re VMT Matched FB LD HD
(Calculated Using EMFAC2010 ver 2.5041)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,391,380	686,190,590	2,568	25,774	2,554	427,867	133.40
2000	22,080,430	838,198,140	1,088	10,883	1,983	491,115	89.99
2005	23,867,536	916,943,680	719	6,696	1,584	546,922	90.61
2007	24,564,360	939,992,190	628	5,788	1,409	562,064	87.50
2010	26,094,882	954,860,220	554	4,942	1,177	564,727	81.02
2014	27,924,486	999,837,570	462	3,923	941	596,149	78.67
2017	28,952,354	1,039,317,600	400	3,212	796	624,114	76.80
2020	29,729,388	1,078,534,500	349	2,658	677	649,418	76.30
2023	30,293,924	1,113,356,000	310	2,192	586	673,595	76.58

Difference (Ver. 2.5041 - Ver. 2.5023) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-111,020	2,562,940	12.9	474.5	-41.6	-407	-10.97
2000	-86,924	5,844,670	-6.3	76.7	-77.1	-3,616	-3.60
2005	577,784	40,265,470	13.1	176.9	68.9	28,437	0.89
2007	383,080	29,668,030	7.2	100.1	30.0	17,646	0.00
2010	-23,810	8,633,020	-8.9	-55.9	-64.4	-8,250	-3.03
2014	-323,046	209,280	-15.9	-145.9	-55.0	-16,096	-1.76
2017	-566,728	-5,484,200	-22.4	-213.9	-58.4	-24,411	-0.89
2020	-787,710	-11,223,100	-27.9	-274.1	-71.9	-33,422	-0.67
2023	-1,004,724	-17,475,700	-34.6	-340.2	-90.2	-43,098	-0.84

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5023)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-0.60%	0.37%	0.51%	1.88%	-1.60%	-0.10%	-7.60%
2000	-0.39%	0.70%	-0.58%	0.71%	-3.74%	-0.73%	-3.85%
2005	2.48%	4.59%	1.86%	2.71%	4.55%	5.48%	0.99%
2007	1.58%	3.26%	1.15%	1.76%	2.17%	3.24%	0.00%
2010	-0.09%	0.91%	-1.58%	-1.12%	-5.19%	-1.44%	-3.61%
2014	-1.14%	0.02%	-3.33%	-3.59%	-5.52%	-2.63%	-2.19%
2017	-1.92%	-0.52%	-5.31%	-6.24%	-6.84%	-3.76%	-1.14%
2020	-2.58%	-1.03%	-7.41%	-9.35%	-9.60%	-4.89%	-0.87%
2023	-3.21%	-1.55%	-10.02%	-13.44%	-13.35%	-6.01%	-1.08%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-74. Impact on Sacramento Valley Air Basin Inventory of Change 12.13

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,373,306	50,465,080	217.3	2221.3	204.7	32,071	11.36
2000	1,628,305	62,080,196	90.9	907.3	167.8	37,499	7.27
2005	1,731,112	65,796,664	60.4	545.3	124.0	39,864	6.94
2007	1,823,069	68,580,760	54.5	485.5	114.7	42,217	6.79
2010	2,030,438	72,283,584	52.1	447.0	106.6	45,182	6.61
2014	2,285,447	78,601,864	47.0	385.8	88.7	49,473	6.44
2017	2,424,266	82,960,408	42.4	331.8	77.6	52,740	6.27
2020	2,522,620	87,144,880	38.2	285.1	69.1	55,776	6.26
2023	2,604,610	91,737,016	35.0	240.5	62.7	59,082	6.36

Sacramento Summer Episodic On-Road Motor Vehicle Inventories Re VMT Matched FB LD HD
(Calculated Using EMFAC2010 ver 2.5041)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,343,634	51,658,632	219.5	2298.9	208.1	32,831	11.02
2000	1,600,770	64,021,716	90.8	928.9	172.4	38,760	7.44
2005	1,786,511	71,607,608	62.7	582.5	136.0	44,312	7.42
2007	1,863,163	73,436,584	56.2	513.3	123.2	45,662	7.21
2010	2,023,808	74,876,264	52.2	455.5	108.1	46,490	6.83
2014	2,241,985	79,746,664	45.9	379.1	85.7	49,472	6.52
2017	2,367,428	83,731,984	40.6	317.2	72.2	52,008	6.32
2020	2,446,532	87,438,952	35.6	262.5	61.8	54,193	6.29
2023	2,482,411	90,641,440	31.3	209.7	53.3	56,204	6.33

Difference (Ver. 2.5041 - Ver. 2.5023) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-29672	1193552	2.2	77.6	3.4	760	-0.34
2000	-27535	1941520	-0.1	21.6	4.6	1,261	0.17
2005	55399	5810944	2.3	37.2	12.0	4,448	0.48
2007	40094	4855824	1.7	27.8	8.5	3,445	0.42
2010	-6630	2592680	0.1	8.5	1.5	1,308	0.22
2014	-43462	1144800	-1.1	-6.7	-3.0	-1	0.08
2017	-56838	771576	-1.8	-14.6	-5.4	-732	0.05
2020	-76088	294072	-2.6	-22.6	-7.3	-1,583	0.03
2023	-122199	-1095576	-3.7	-30.8	-9.4	-2,878	-0.03

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.5023)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-2.16%	2.37%	0.99%	3.50%	1.64%	2.37%	-2.96%
2000	-1.69%	3.13%	-0.09%	2.38%	2.76%	3.36%	2.28%
2005	3.20%	8.83%	3.84%	6.82%	9.68%	11.16%	6.93%
2007	2.20%	7.08%	3.19%	5.72%	7.38%	8.16%	6.14%
2010	-0.33%	3.59%	0.23%	1.90%	1.42%	2.90%	3.29%
2014	-1.90%	1.46%	-2.44%	-1.74%	-3.43%	0.00%	1.17%
2017	-2.34%	0.93%	-4.26%	-4.41%	-6.95%	-1.39%	0.75%
2020	-3.02%	0.34%	-6.72%	-7.92%	-10.56%	-2.84%	0.54%
2023	-4.69%	-1.19%	-10.51%	-12.80%	-14.91%	-4.87%	-0.49%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-75. Impact on San Diego Air Basin Inventory of Change 12.13

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,609,040	59,230,324	222.0	2268.0	201.0	37,710	9.50
2000	1,925,116	73,764,616	83.0	860.0	138.0	41,257	6.77
2005	2,054,304	78,582,088	52.0	523.0	98.0	44,004	6.46
2007	2,129,484	80,890,632	46.0	458.0	90.0	45,845	6.33
2010	2,277,701	82,533,528	41.0	397.0	81.0	46,939	6.14
2014	2,432,723	85,930,784	35.0	317.0	64.0	49,290	5.93
2017	2,480,928	88,470,224	30.0	257.0	54.0	51,132	5.79
2020	2,523,582	90,974,536	26.0	216.0	46.0	53,051	5.76
2023	2,599,896	94,694,192	23.0	190.0	42.0	55,836	5.89

San Diego Summer Episodic On-Road Motor Vehicle Inventories Re VMT Matched FB LD HD
(Calculated Using EMFAC2010 ver 2.5041)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,604,708	59,061,544	221	2266	200	37,630	9.42
2000	1,919,750	73,521,104	83	858	137	41,092	6.76
2005	2,061,644	79,028,376	52	522	101	44,606	6.59
2007	2,149,164	81,902,376	47	460	93	46,975	6.54
2010	2,278,918	82,599,080	41	390	79	46,826	6.11
2014	2,426,912	85,887,064	34	306	63	48,832	5.98
2017	2,470,420	88,353,024	29	243	52	50,247	5.88
2020	2,509,912	90,818,672	25	199	44	51,727	5.89
2023	2,586,244	94,608,376	22	168	38	54,041	6.03

Difference (Ver. 2.5041 - Ver. 2.5023) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-4,332	-168,780	-0.7	-1.7	-0.8	-80	-0.08
2000	-5,366	-243,512	-0.4	-1.6	-1.1	-165	-0.01
2005	7,340	446,288	0.4	-1.0	2.6	602	0.13
2007	19,680	1,011,744	0.6	1.7	3.5	1,130	0.21
2010	1,217	65,552	0.2	-7.0	-2.4	-113	-0.03
2014	-5,811	-43,720	-0.8	-11.0	-1.5	-458	0.05
2017	-10,508	-117,200	-1.3	-13.5	-2.2	-885	0.09
2020	-13,670	-155,864	-1.4	-17.4	-2.2	-1,324	0.13
2023	-13,652	-85,816	-1.1	-22.2	-3.8	-1,795	0.14

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.5023)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-0.27%	-0.28%	-0.32%	-0.08%	-0.38%	-0.21%	-0.83%
2000	-0.28%	-0.33%	-0.47%	-0.18%	-0.82%	-0.40%	-0.19%
2005	0.36%	0.57%	0.72%	-0.20%	2.62%	1.37%	2.06%
2007	0.92%	1.25%	1.37%	0.37%	3.87%	2.47%	3.27%
2010	0.05%	0.08%	0.41%	-1.76%	-2.95%	-0.24%	-0.43%
2014	-0.24%	-0.05%	-2.35%	-3.48%	-2.34%	-0.93%	0.76%
2017	-0.42%	-0.13%	-4.33%	-5.27%	-4.05%	-1.73%	1.55%
2020	-0.54%	-0.17%	-5.49%	-8.04%	-4.76%	-2.50%	2.24%
2023	-0.53%	-0.09%	-4.87%	-11.68%	-9.07%	-3.22%	2.36%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-76. Impact on San Francisco Bay Air Basin Inventory of Change 12.13

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,932,100	138,236,160	531.2	4998.5	490.3	80,545	24.45
2000	4,709,472	166,475,900	234.1	2178.4	347.5	95,294	16.97
2005	4,663,679	164,225,200	149.6	1284.4	246.8	95,231	15.13
2007	4,742,242	167,109,980	128.6	1087.4	220.6	98,753	14.54
2010	5,024,152	171,597,970	114.4	934.3	195.0	103,886	13.99
2014	5,426,317	181,193,380	96.9	745.4	154.0	110,150	13.44
2017	5,621,512	188,394,240	84.5	612.5	129.5	116,571	13.09
2020	5,754,138	195,584,690	74.7	510.7	111.9	122,008	13.01
2023	5,893,057	202,578,720	68.6	441.8	100.3	127,438	13.13

San Francisco Summer Episodic On-Road Motor Vehicle Inventories Re VMT Matched FB LD HD
(Calculated Using EMFAC2010 ver 2.5041)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,926,764	141,436,530	538.5	5140.5	498.3	82,069	24.26
2000	4,702,158	170,357,330	230.5	2186.2	347.5	93,112	17.02
2005	4,740,305	174,199,680	147.2	1277.6	260.0	97,337	15.54
2007	4,816,329	175,699,760	127.7	1090.1	230.4	99,628	15.02
2010	4,992,611	175,105,090	110.8	905.8	195.3	98,995	14.21
2014	5,256,748	180,757,220	90.5	692.3	149.8	102,102	13.56
2017	5,345,382	184,993,810	76.2	542.9	121.9	104,552	13.05
2020	5,392,182	189,232,540	65.0	432.6	101.2	107,003	12.86
2023	5,463,724	193,470,030	57.7	355.3	86.9	109,632	12.86

Difference (Ver. 2.5041 - Ver. 2.5023) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-5,336	3,200,370	7.3	142.0	8.0	1,524	-0.19
2000	-7,314	3,881,430	-3.6	7.8	0.0	-2,182	0.05
2005	76,626	9,974,480	-2.4	-6.8	13.2	2,106	0.41
2007	74,087	8,589,780	-0.9	2.7	9.8	875	0.48
2010	-31,541	3,507,120	-3.6	-28.5	0.3	-4,891	0.22
2014	-169,569	-436,160	-6.4	-53.1	-4.2	-8,048	0.12
2017	-276,130	-3,400,430	-8.3	-69.6	-7.6	-12,019	-0.04
2020	-361,956	-6,352,150	-9.7	-78.1	-10.7	-15,006	-0.15
2023	-429,333	-9,108,690	-10.9	-86.5	-13.4	-17,806	-0.27

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.5023)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-0.14%	2.32%	1.37%	2.84%	1.64%	1.89%	-0.77%
2000	-0.16%	2.33%	-1.53%	0.36%	-0.01%	-2.29%	0.32%
2005	1.64%	6.07%	-1.61%	-0.53%	5.33%	2.21%	2.71%
2007	1.56%	5.14%	-0.73%	0.25%	4.42%	0.89%	3.32%
2010	-0.63%	2.04%	-3.19%	-3.05%	0.13%	-4.71%	1.55%
2014	-3.12%	-0.24%	-6.57%	-7.12%	-2.71%	-7.31%	0.86%
2017	-4.91%	-1.80%	-9.88%	-11.36%	-5.87%	-10.31%	-0.28%
2020	-6.29%	-3.25%	-12.98%	-15.30%	-9.58%	-12.30%	-1.15%
2023	-7.29%	-4.50%	-15.94%	-19.57%	-13.39%	-13.97%	-2.08%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-77. Impact on San Joaquin Valley Air Basin Inventory of Change 12.13

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,378,730	58,052,672	229.7	2405.6	329.7	43,532	23.36
2000	1,925,682	82,513,360	112.3	1134.9	345.2	57,681	13.74
2005	2,101,642	88,853,616	80.5	728.9	273.4	62,164	13.43
2007	2,223,821	93,314,824	73.5	656.7	250.2	65,958	12.97
2010	2,493,321	98,407,960	71.1	612.7	223.0	70,222	12.16
2014	2,852,481	107,711,920	66.1	544.1	178.4	77,480	11.51
2017	3,115,978	116,216,310	61.4	482.0	153.0	84,089	10.88
2020	3,309,581	124,193,740	56.3	421.5	133.0	90,244	10.64
2023	3,430,952	131,662,410	51.4	355.9	117.9	95,735	10.54

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories Re VMT Matched FB LD HD
(Calculated Using EMFAC2010 ver 2.5041)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,360,512	57,189,940	230.2	2430.5	327.0	43,383	22.43
2000	1,904,122	81,665,680	111.7	1129.0	337.2	57,322	13.50
2005	2,133,798	91,497,808	82.1	743.5	289.0	65,930	13.88
2007	2,267,481	96,045,520	74.9	668.9	260.2	69,091	13.28
2010	2,493,775	98,289,208	70.3	605.1	211.8	69,123	11.71
2014	2,824,995	106,825,710	64.4	527.0	168.6	75,788	11.20
2017	3,054,319	114,866,860	59.2	456.1	145.7	82,593	10.88
2020	3,226,845	122,597,250	53.1	389.0	123.5	88,297	10.67
2023	3,337,928	130,114,810	47.7	318.0	106.3	93,507	10.61

Difference (Ver. 2.5041 - Ver. 2.5023) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-18,218	-862,732	0.5	24.9	-2.7	-149	-0.93
2000	-21,560	-847,680	-0.6	-5.9	-8.0	-359	-0.24
2005	32,156	2,644,192	1.6	14.6	15.6	3,766	0.45
2007	43,660	2,730,696	1.4	12.2	10.0	3,133	0.31
2010	454	-118,752	-0.8	-7.6	-11.2	-1,099	-0.45
2014	-27,486	-886,210	-1.7	-17.1	-9.8	-1,692	-0.31
2017	-61,659	-1,349,450	-2.2	-25.9	-7.3	-1,496	0.00
2020	-82,736	-1,596,490	-3.2	-32.5	-9.5	-1,947	0.03
2023	-93,024	-1,547,600	-3.7	-37.9	-11.6	-2,228	0.07

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.5023)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-1.32%	-1.49%	0.24%	1.03%	-0.82%	-0.34%	-3.97%
2000	-1.12%	-1.03%	-0.52%	-0.52%	-2.32%	-0.62%	-1.74%
2005	1.53%	2.98%	2.01%	2.00%	5.71%	6.06%	3.37%
2007	1.96%	2.93%	1.97%	1.85%	3.98%	4.75%	2.37%
2010	0.02%	-0.12%	-1.15%	-1.24%	-5.03%	-1.56%	-3.69%
2014	-0.96%	-0.82%	-2.55%	-3.15%	-5.49%	-2.18%	-2.67%
2017	-1.98%	-1.16%	-3.59%	-5.37%	-4.76%	-1.78%	-0.03%
2020	-2.50%	-1.29%	-5.68%	-7.71%	-7.16%	-2.16%	0.24%
2023	-2.71%	-1.18%	-7.16%	-10.65%	-9.85%	-2.33%	0.66%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-78. Impact on South Coast Air Basin Inventory of Change 12.13

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2010 ver 2.5023)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,537,252	264,378,460	958.8	9212.1	902.3	162,405	46.15
2000	8,885,995	315,646,460	390.5	3837.3	651.1	182,423	31.90
2005	9,547,117	340,782,370	249.1	2345.5	475.7	193,041	30.44
2007	9,907,570	354,557,890	215.1	2009.8	429.3	201,968	29.78
2010	10,537,439	365,239,680	185.0	1685.7	379.6	209,404	28.71
2014	11,072,440	377,542,340	146.4	1281.8	301.7	219,670	27.45
2017	11,422,400	389,182,020	125.2	1049.2	257.7	230,012	26.80
2020	11,709,370	400,561,150	110.1	888.9	225.5	239,553	26.52
2023	11,886,466	407,786,080	100.1	778.1	204.9	248,366	26.50

South Coast Summer Episodic On-Road Motor Vehicle Inventories Re VMT Matched FB LD HD
(Calculated Using EMFAC2010 ver 2.5041)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,571,598	265,998,580	968.7	9386.1	888.2	162,224	42.97
2000	8,920,368	316,923,010	392.8	3886.7	624.0	181,241	30.54
2005	9,929,820	358,652,290	261.0	2460.2	514.8	207,806	31.67
2007	10,070,007	362,891,810	219.0	2042.4	442.1	209,078	30.06
2010	10,528,747	365,448,830	182.6	1655.3	355.6	207,479	27.64
2014	11,020,486	376,985,500	143.0	1234.5	287.9	216,555	27.21
2017	11,338,318	388,208,510	120.5	985.6	243.3	225,020	26.89
2020	11,605,638	399,393,310	104.0	806.1	206.4	232,307	26.81
2023	11,783,338	406,889,700	92.6	672.9	178.5	238,864	26.82

Difference (Ver. 2.5041 - Ver. 2.5023) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	34,346	1,620,120	9.9	174.0	-14.1	-181	-3.18
2000	34,373	1,276,550	2.3	49.4	-27.1	-1,182	-1.36
2005	382,703	17,869,920	11.9	114.7	39.1	14,765	1.23
2007	162,437	8,333,920	3.9	32.6	12.8	7,110	0.28
2010	-8,692	209,150	-2.4	-30.4	-24.0	-1,925	-1.07
2014	-51,954	-556,840	-3.4	-47.3	-13.8	-3,116	-0.24
2017	-84,082	-973,510	-4.7	-63.6	-14.4	-4,992	0.09
2020	-103,732	-1,167,840	-6.1	-82.8	-19.1	-7,246	0.29
2023	-103,128	-896,380	-7.5	-105.2	-26.4	-9,503	0.32

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.5023)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.46%	0.61%	1.03%	1.89%	-1.56%	-0.11%	-6.89%
2000	0.39%	0.40%	0.59%	1.29%	-4.16%	-0.65%	-4.27%
2005	4.01%	5.24%	4.76%	4.89%	8.22%	7.65%	4.05%
2007	1.64%	2.35%	1.80%	1.62%	2.98%	3.52%	0.93%
2010	-0.08%	0.06%	-1.31%	-1.80%	-6.31%	-0.92%	-3.74%
2014	-0.47%	-0.15%	-2.31%	-3.69%	-4.58%	-1.42%	-0.87%
2017	-0.74%	-0.25%	-3.72%	-6.06%	-5.59%	-2.17%	0.35%
2020	-0.89%	-0.29%	-5.53%	-9.32%	-8.46%	-3.02%	1.08%
2023	-0.87%	-0.22%	-7.54%	-13.52%	-12.90%	-3.83%	1.21%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.14 MISCELLANEOUS SURVIVAL RATE AND NEW VEHICLE SALES ADJUSTMENT

Please refer to Section 3.3.2. In addition to the survival rate adjustments, a cap was removed limiting the rate of new vehicle sales. That is, new vehicle sales were limited to no more than a 10% increase from the previous year. Because the base year, 2009 was a very low sales year, this limit was causing anomalies in the age distributions. This generally reduces emissions by allowing more new vehicles to enter the fleet, which generally have lower emission rates.

Table 12-79. Impact on Statewide Inventory of Change 12.14

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.41)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,391,380	686,190,590	2,568	25,774	2,554	427,867	133.40
2000	22,080,430	838,198,140	1,088	10,883	1,983	491,115	89.99
2005	23,867,536	916,943,680	719	6,696	1,584	546,922	90.61
2007	24,564,360	939,992,190	628	5,788	1,409	562,064	87.50
2010	26,094,882	954,860,220	554	4,942	1,177	564,727	81.02
2014	27,924,486	999,837,570	462	3,923	941	596,149	78.67
2017	28,952,354	1,039,317,600	400	3,212	796	624,114	76.80
2020	29,729,388	1,078,534,500	349	2,658	677	649,418	76.30
2023	30,293,924	1,113,356,000	310	2,192	586	673,595	76.58

Statewide Summer Episodic On-Road Motor Vehicle Inventories w Dynamic Population/07 Survivals exc PC/LT2
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	17,772,142	660,221,950	2,556	25,044	2,452	411,831	127.17
2000	21,310,336	812,222,140	1,069	10,437	1,879	475,745	86.41
2005	23,126,388	889,263,170	699	6,458	1,522	530,563	87.46
2007	24,037,784	920,125,310	615	5,639	1,373	550,409	85.48
2010	25,632,786	954,863,490	527	4,698	1,147	563,878	80.53
2014	26,602,556	1,000,107,900	390	3,311	861	595,399	77.66
2017	27,483,390	1,039,511,200	321	2,604	702	624,383	75.82
2020	28,419,452	1,078,516,100	277	2,146	581	650,674	75.42
2023	29,314,482	1,113,351,200	253	1,879	501	675,619	75.85

Difference (Ver. 2.5044 - Ver. 2.5041) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-619,238	-25,968,640	-12.1	-729.7	-102.0	-16,036	-6.23
2000	-770,094	-25,976,000	-18.4	-446.0	-103.6	-15,370	-3.58
2005	-741,148	-27,680,510	-19.8	-238.1	-61.6	-16,359	-3.15
2007	-526,576	-19,866,880	-13.5	-149.0	-36.0	-11,655	-2.02
2010	-462,096	3,270	-27.1	-244.3	-29.1	-848	-0.49
2014	-1,321,930	270,330	-72.5	-611.8	-80.3	-750	-1.00
2017	-1,468,964	193,600	-78.5	-607.7	-93.6	268	-0.98
2020	-1,309,936	-18,400	-71.9	-511.6	-95.7	1,256	-0.88
2023	-979,442	-4,800	-57.8	-313.1	-84.7	2,025	-0.73

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5041)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-3.37%	-3.78%	-0.47%	-2.83%	-3.99%	-3.75%	-4.67%
2000	-3.49%	-3.10%	-1.70%	-4.10%	-5.22%	-3.13%	-3.97%
2005	-3.11%	-3.02%	-2.75%	-3.56%	-3.89%	-2.99%	-3.48%
2007	-2.14%	-2.11%	-2.16%	-2.57%	-2.55%	-2.07%	-2.31%
2010	-1.77%	0.00%	-4.89%	-4.94%	-2.47%	-0.15%	-0.60%
2014	-4.73%	0.03%	-15.70%	-15.60%	-8.53%	-0.13%	-1.28%
2017	-5.07%	0.02%	-19.65%	-18.92%	-11.76%	0.04%	-1.28%
2020	-4.41%	0.00%	-20.61%	-19.25%	-14.13%	0.19%	-1.16%
2023	-3.23%	0.00%	-18.62%	-14.29%	-14.47%	0.30%	-0.96%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-80. Impact on Sacramento Valley Air Basin Inventory of Change12.14

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.41)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,343,634	51,658,632	219.5	2298.9	208.1	32,831	11.02
2000	1,600,770	64,021,716	90.8	928.9	172.4	38,760	7.44
2005	1,786,511	71,607,608	62.7	582.5	136.0	44,312	7.42
2007	1,863,163	73,436,584	56.2	513.3	123.2	45,662	7.21
2010	2,023,808	74,876,264	52.2	455.5	108.1	46,490	6.83
2014	2,241,985	79,746,664	45.9	379.1	85.7	49,472	6.52
2017	2,367,428	83,731,984	40.6	317.2	72.2	52,008	6.32
2020	2,446,532	87,438,952	35.6	262.5	61.8	54,193	6.29
2023	2,482,411	90,641,440	31.3	209.7	53.3	56,204	6.33

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,274,816	48,658,028	215.4	2204.0	197.0	31,033	10.42
2000	1,518,151	60,879,456	89.7	892.2	163.1	37,013	7.07
2005	1,698,989	68,124,624	60.4	555.6	129.9	42,295	7.08
2007	1,817,064	71,630,616	55.0	499.7	120.2	44,610	7.03
2010	1,969,133	74,875,336	48.8	425.4	104.7	46,392	6.77
2014	2,065,937	80,023,600	35.6	295.2	75.7	49,471	6.40
2017	2,154,850	83,938,640	28.9	230.0	60.5	52,067	6.20
2020	2,246,959	87,433,544	24.8	188.7	50.1	54,284	6.18
2023	2,339,770	90,662,608	22.6	165.1	43.4	56,460	6.24

Difference (Ver. 2.5044 - Ver. 2.5041) in Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-68818	-3000604	-4.1	-95.0	-11.0	-1,798	-0.61
2000	-82619	-3142260	-1.1	-36.7	-9.3	-1,748	-0.37
2005	-87522	-3482984	-2.3	-26.9	-6.1	-2,017	-0.34
2007	-46099	-1805968	-1.2	-13.5	-3.0	-1,053	-0.17
2010	-54675	-928	-3.4	-30.1	-3.4	-98	-0.06
2014	-176048	276936	-10.3	-83.8	-9.9	-1	-0.12
2017	-212578	206656	-11.7	-87.2	-11.7	59	-0.12
2020	-199573	-5408	-10.9	-73.8	-11.7	91	-0.11
2023	-142641	21168	-8.7	-44.6	-10.0	257	-0.09

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.5041)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-5.12%	-5.81%	-1.86%	-4.13%	-5.30%	-5.48%	-5.51%
2000	-5.16%	-4.91%	-1.19%	-3.95%	-5.39%	-4.51%	-4.92%
2005	-4.90%	-4.86%	-3.73%	-4.63%	-4.51%	-4.55%	-4.56%
2007	-2.47%	-2.46%	-2.17%	-2.64%	-2.41%	-2.31%	-2.41%
2010	-2.70%	0.00%	-6.56%	-6.61%	-3.11%	-0.21%	-0.88%
2014	-7.85%	0.35%	-22.44%	-22.12%	-11.60%	0.00%	-1.77%
2017	-8.98%	0.25%	-28.73%	-27.49%	-16.18%	0.11%	-1.87%
2020	-8.16%	-0.01%	-30.46%	-28.12%	-18.96%	0.17%	-1.81%
2023	-5.75%	0.02%	-27.77%	-21.29%	-18.66%	0.46%	-1.42%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-81. Impact on San Diego Air Basin Inventory of Change 12.14

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.41)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,604,708	59,061,544	221.3	2266.3	200.2	37,630	9.42
2000	1,919,750	73,521,104	82.6	858.4	136.9	41,092	6.76
2005	2,061,644	79,028,376	52.4	522.0	100.6	44,606	6.59
2007	2,149,164	81,902,376	46.6	459.7	93.5	46,975	6.54
2010	2,278,918	82,599,080	41.2	390.0	78.6	46,826	6.11
2014	2,426,912	85,887,064	34.2	306.0	62.5	48,832	5.98
2017	2,470,420	88,353,024	28.7	243.5	51.8	50,247	5.88
2020	2,509,912	90,818,672	24.6	198.6	43.8	51,727	5.89
2023	2,586,244	94,608,376	21.9	167.8	38.2	54,041	6.03

San Diego Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,567,046	57,477,284	221	2217	195	36,660	9.16
2000	1,874,501	72,232,144	81	829	132	40,390	6.59
2005	2,013,087	77,339,664	51	507	98	43,693	6.44
2007	2,098,513	80,059,032	45	447	91	45,950	6.38
2010	2,240,420	82,599,296	39	373	77	46,753	6.08
2014	2,327,605	85,886,136	30	266	58	48,809	5.91
2017	2,391,866	88,352,776	25	211	47	50,393	5.83
2020	2,457,305	90,816,720	22	175	39	51,933	5.84
2023	2,551,118	94,602,256	20	156	34	54,250	5.99

Difference (Ver. 2.5044 - Ver. 2.5041) in Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-37,662	-1,584,260	0.2	-49.0	-5.4	-969	-0.27
2000	-45,249	-1,288,960	-1.4	-29.3	-5.3	-702	-0.16
2005	-48,557	-1,688,712	-1.2	-15.1	-2.9	-913	-0.16
2007	-50,651	-1,843,344	-1.2	-13.0	-2.5	-1,025	-0.15
2010	-38,498	216	-1.8	-17.0	-1.9	-73	-0.03
2014	-99,307	-928	-4.3	-39.5	-5.0	-23	-0.06
2017	-78,554	-248	-3.6	-32.2	-4.9	145	-0.05
2020	-52,607	-1,952	-2.6	-23.4	-4.6	206	-0.04
2023	-35,126	-6,120	-1.7	-11.6	-4.0	209	-0.04

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.5041)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-2.35%	-2.68%	0.08%	-2.16%	-2.70%	-2.58%	-2.82%
2000	-2.36%	-1.75%	-1.74%	-3.42%	-3.88%	-1.71%	-2.44%
2005	-2.36%	-2.14%	-2.29%	-2.89%	-2.88%	-2.05%	-2.36%
2007	-2.36%	-2.25%	-2.59%	-2.82%	-2.63%	-2.18%	-2.33%
2010	-1.69%	0.00%	-4.34%	-4.37%	-2.44%	-0.16%	-0.53%
2014	-4.09%	0.00%	-12.45%	-12.91%	-8.00%	-0.05%	-1.06%
2017	-3.18%	0.00%	-12.51%	-13.22%	-9.50%	0.29%	-0.89%
2020	-2.10%	0.00%	-10.67%	-11.79%	-10.42%	0.40%	-0.75%
2023	-1.36%	-0.01%	-7.64%	-6.93%	-10.38%	0.39%	-0.64%

ROG_Tot[1] - This includes running, starting, idling and evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-82. Impact on San Francisco Bay Air Basin Inventory of Change 12.14

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.41)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,926,764	141,436,530	538.5	5140.5	498.3	82,069	24.26
2000	4,702,158	170,357,330	230.5	2186.2	347.5	93,112	17.02
2005	4,740,305	174,199,680	147.2	1277.6	260.0	97,337	15.54
2007	4,816,329	175,699,760	127.7	1090.1	230.4	99,628	15.02
2010	4,992,611	175,105,090	110.8	905.8	195.3	98,995	14.21
2014	5,256,748	180,757,220	90.5	692.3	149.8	102,102	13.56
2017	5,345,382	184,993,810	76.2	542.9	121.9	104,552	13.05
2020	5,392,182	189,232,540	65.0	432.6	101.2	107,003	12.86
2023	5,463,724	193,470,030	57.7	355.3	86.9	109,632	12.86

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,791,482	136,096,320	544.2	5014.7	478.6	79,054	23.12
2000	4,539,164	165,382,590	230.0	2094.3	327.7	90,395	16.35
2005	4,573,979	168,301,140	143.7	1224.3	247.8	94,019	14.91
2007	4,731,008	172,683,570	125.9	1065.4	224.5	97,907	14.71
2010	4,915,308	175,104,750	105.6	867.8	191.0	98,854	14.11
2014	5,039,932	180,753,710	77.1	602.0	138.6	101,887	13.37
2017	5,147,419	184,990,540	63.1	466.6	110.5	104,573	12.90
2020	5,264,958	189,228,260	54.4	379.9	90.9	107,236	12.73
2023	5,390,386	193,460,990	49.6	330.4	78.4	109,886	12.75

Difference (Ver. 2.5044 - Ver. 2.5041) in Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-135,282	-5,340,210	5.7	-125.8	-19.8	-3,016	-1.14
2000	-162,994	-4,974,740	-0.5	-91.8	-19.8	-2,717	-0.67
2005	-166,326	-5,898,540	-3.5	-53.3	-12.1	-3,318	-0.63
2007	-85,321	-3,016,190	-1.8	-24.8	-5.8	-1,722	-0.32
2010	-77,303	-340	-5.1	-38.0	-4.3	-142	-0.09
2014	-216,816	-3,510	-13.5	-90.3	-11.2	-215	-0.18
2017	-197,963	-3,270	-13.0	-76.4	-11.4	21	-0.16
2020	-127,224	-4,280	-10.6	-52.7	-10.2	233	-0.13
2023	-73,338	-9,040	-8.1	-24.9	-8.5	253	-0.11

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.5041)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-3.45%	-3.78%	1.05%	-2.45%	-3.96%	-3.67%	-4.69%
2000	-3.47%	-2.92%	-0.24%	-4.20%	-5.70%	-2.92%	-3.95%
2005	-3.51%	-3.39%	-2.38%	-4.17%	-4.67%	-3.41%	-4.02%
2007	-1.77%	-1.72%	-1.39%	-2.27%	-2.52%	-1.73%	-2.11%
2010	-1.55%	0.00%	-4.62%	-4.20%	-2.20%	-0.14%	-0.66%
2014	-4.12%	0.00%	-14.88%	-13.05%	-7.47%	-0.21%	-1.34%
2017	-3.70%	0.00%	-17.08%	-14.06%	-9.32%	0.02%	-1.21%
2020	-2.36%	0.00%	-16.27%	-12.18%	-10.12%	0.22%	-1.00%
2023	-1.34%	0.00%	-14.02%	-7.01%	-9.76%	0.23%	-0.82%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear

Fuel[3] - VMT Matching by Fuel Type

Table 12-83. Impact on San Joaquin Valley Air Basin Inventory of Change 12.14

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.41)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,360,512	57,189,940	230.2	2430.5	327.0	43,383	22.43
2000	1,904,122	81,665,680	111.7	1129.0	337.2	57,322	13.50
2005	2,133,798	91,497,808	82.1	743.5	289.0	65,930	13.88
2007	2,267,481	96,045,520	74.9	668.9	260.2	69,091	13.28
2010	2,493,775	98,289,208	70.3	605.1	211.8	69,123	11.71
2014	2,824,995	106,825,710	64.4	527.0	168.6	75,788	11.20
2017	3,054,319	114,866,860	59.2	456.1	145.7	82,593	10.88
2020	3,226,845	122,597,250	53.1	389.0	123.5	88,297	10.67
2023	3,337,928	130,114,810	47.7	318.0	106.3	93,507	10.61

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,279,267	53,535,128	222.6	2304.9	306.8	40,717	21.02
2000	1,789,931	77,188,680	106.5	1056.8	315.0	54,226	12.70
2005	2,026,420	87,160,304	78.2	705.6	274.8	62,912	13.22
2007	2,197,510	93,262,648	72.5	647.0	252.3	67,147	12.88
2010	2,412,169	98,288,880	65.3	559.8	206.7	68,940	11.63
2014	2,552,338	106,823,070	49.1	395.7	153.4	75,435	11.00
2017	2,704,630	114,862,440	41.0	314.0	127.4	82,350	10.66
2020	2,872,522	122,591,290	35.6	262.4	104.7	88,293	10.47
2023	3,047,654	130,107,720	32.6	233.6	89.7	93,786	10.45

Difference (Ver. 2.5044 - Ver. 2.5041) in Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-81,245	-3,654,812	-7.6	-125.6	-20.2	-2,665	-1.41
2000	-114,191	-4,477,000	-5.2	-72.2	-22.2	-3,096	-0.81
2005	-107,378	-4,337,504	-3.9	-37.8	-14.2	-3,018	-0.67
2007	-69,971	-2,782,872	-2.4	-21.9	-7.9	-1,944	-0.39
2010	-81,606	-328	-5.0	-45.3	-5.1	-183	-0.08
2014	-272,657	-2,640	-15.3	-131.3	-15.2	-353	-0.20
2017	-349,689	-4,420	-18.2	-142.1	-18.3	-244	-0.21
2020	-354,323	-5,960	-17.5	-126.7	-18.8	-5	-0.19
2023	-290,274	-7,090	-15.1	-84.4	-16.6	279	-0.16

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.5041)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-5.97%	-6.39%	-3.32%	-5.17%	-6.17%	-6.14%	-6.30%
2000	-6.00%	-5.48%	-4.64%	-6.39%	-6.59%	-5.40%	-5.97%
2005	-5.03%	-4.74%	-4.77%	-5.09%	-4.91%	-4.58%	-4.80%
2007	-3.09%	-2.90%	-3.20%	-3.27%	-3.03%	-2.81%	-2.96%
2010	-3.27%	0.00%	-7.12%	-7.49%	-2.39%	-0.27%	-0.71%
2014	-9.65%	0.00%	-23.79%	-24.91%	-9.00%	-0.47%	-1.80%
2017	-11.45%	0.00%	-30.76%	-31.16%	-12.56%	-0.30%	-1.97%
2020	-10.98%	0.00%	-32.92%	-32.56%	-15.23%	-0.01%	-1.83%
2023	-8.70%	-0.01%	-31.74%	-26.54%	-15.61%	0.30%	-1.54%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-84. Impact on South Coast Air Basin Inventory of Change 12.14

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.41)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,571,598	265,998,580	968.7	9386.1	888.2	162,224	42.97
2000	8,920,368	316,923,010	392.8	3886.7	624.0	181,241	30.54
2005	9,929,820	358,652,290	261.0	2460.2	514.8	207,806	31.67
2007	10,070,007	362,891,810	219.0	2042.4	442.1	209,078	30.06
2010	10,528,747	365,448,830	182.6	1655.3	355.6	207,479	27.64
2014	11,020,486	376,985,500	143.0	1234.5	287.9	216,555	27.21
2017	11,338,318	388,208,510	120.5	985.6	243.3	225,020	26.89
2020	11,605,638	399,393,310	104.0	806.1	206.4	232,307	26.81
2023	11,783,338	406,889,700	92.6	672.9	178.5	238,864	26.82

South Coast Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,369,264	258,397,090	964.2	9205.9	865.1	157,885	41.65
2000	8,667,431	309,433,410	383.4	3740.7	601.1	177,195	29.73
2005	9,686,220	350,403,970	254.2	2385.4	501.1	203,355	30.93
2007	9,860,916	355,615,360	213.9	1989.7	432.7	205,147	29.48
2010	10,426,589	365,454,110	177.3	1606.0	349.2	207,360	27.54
2014	10,797,710	376,983,170	132.5	1143.0	272.8	216,712	27.06
2017	11,105,086	388,201,180	109.9	902.5	225.1	225,360	26.75
2020	11,412,823	399,391,550	94.9	740.7	186.7	232,701	26.67
2023	11,644,727	406,887,300	85.9	641.8	160.3	239,156	26.70

Difference (Ver. 2.5044 - Ver. 2.5041) in Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-202,334	-7,601,490	-4.6	-180.2	-23.1	-4,339	-1.32
2000	-252,937	-7,489,600	-9.5	-146.0	-22.9	-4,046	-0.81
2005	-243,600	-8,248,320	-6.8	-74.9	-13.7	-4,450	-0.74
2007	-209,091	-7,276,450	-5.1	-52.6	-9.4	-3,931	-0.58
2010	-102,158	5,280	-5.3	-49.4	-6.4	-119	-0.10
2014	-222,776	-2,330	-10.5	-91.5	-15.0	158	-0.15
2017	-233,232	-7,330	-10.6	-83.1	-18.2	340	-0.15
2020	-192,815	-1,760	-9.2	-65.3	-19.7	394	-0.14
2023	-138,611	-2,400	-6.7	-31.1	-18.2	292	-0.12

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.5041)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-2.67%	-2.86%	-0.47%	-1.92%	-2.60%	-2.67%	-3.08%
2000	-2.84%	-2.36%	-2.41%	-3.76%	-3.67%	-2.23%	-2.66%
2005	-2.45%	-2.30%	-2.59%	-3.04%	-2.67%	-2.14%	-2.33%
2007	-2.08%	-2.01%	-2.32%	-2.58%	-2.12%	-1.88%	-1.93%
2010	-0.97%	0.00%	-2.90%	-2.98%	-1.81%	-0.06%	-0.36%
2014	-2.02%	0.00%	-7.34%	-7.41%	-5.23%	0.07%	-0.57%
2017	-2.06%	0.00%	-8.82%	-8.43%	-7.49%	0.15%	-0.54%
2020	-1.66%	0.00%	-8.80%	-8.10%	-9.55%	0.17%	-0.51%
2023	-1.18%	0.00%	-7.22%	-4.62%	-10.19%	0.12%	-0.45%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.15 REMOVAL OF HEAVY DUTY TRUCKS

Please refer to Section 4. These incremental emissions document how the EMFAC model responds as the heavy duty fleet is removed from EMFAC to the truck model. As expected, this significantly reduces the NOx and PM inventories. Areas with higher truck VMT are disproportionately affected.

Table 12-85. Impact on Statewide Inventory of Change 12.15

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	17,772,142	660,221,950	2,556	25,044	2,452	411,831	127.17
2000	21,310,336	812,222,140	1,069	10,437	1,879	475,745	86.41
2005	23,126,388	889,263,170	699	6,458	1,522	530,563	87.46
2007	24,037,784	920,125,310	615	5,639	1,373	550,409	85.48
2010	25,632,786	954,863,490	527	4,698	1,147	563,878	80.53
2014	26,602,556	1,000,107,900	390	3,311	861	595,399	77.66
2017	27,483,390	1,039,511,200	321	2,604	702	624,383	75.82
2020	28,419,452	1,078,516,100	277	2,146	581	650,674	75.42
2023	29,314,482	1,113,351,200	253	1,879	501	675,619	75.85

Statewide Summer Episodic On-Road Motor Vehicle Inventories Remove HD Diesel Populations
(Calculated Using EMFAC2011 ver 2.50.50)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	17,402,036	627,215,300	2,432	24,325	1,788	349,011	62.53
2000	20,918,632	772,769,920	1,031	10,380	1,061	403,712	54.83
2005	22,628,922	844,379,140	653	6,267	753	450,852	53.63
2007	23,529,334	874,683,840	573	5,475	679	469,678	53.71
2010	25,124,892	911,216,640	493	4,573	598	486,812	54.08
2014	26,007,884	949,234,750	361	3,199	450	505,994	54.14
2017	26,820,658	982,844,990	295	2,499	370	524,749	55.42
2020	27,702,826	1,017,386,400	254	2,049	312	543,466	57.12
2023	28,553,862	1,048,442,400	231	1,786	273	561,739	58.83

Difference (Ver. 2.5050 - Ver. 2.5045) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-370,106	-33,006,650	-124.2	-718.5	-664.5	-62,820	-64.64
2000	-391,704	-39,452,220	-38.2	-56.2	-818.3	-72,033	-31.59
2005	-497,466	-44,884,030	-46.7	-191.0	-769.4	-79,711	-33.83
2007	-508,450	-45,441,470	-41.9	-163.6	-694.1	-80,732	-31.76
2010	-507,894	-43,646,850	-34.1	-124.6	-549.7	-77,067	-26.45
2014	-594,672	-50,873,150	-28.7	-112.3	-410.9	-89,405	-23.53
2017	-662,732	-56,666,210	-25.9	-104.9	-332.1	-99,634	-20.40
2020	-716,626	-61,129,700	-23.6	-97.6	-269.3	-107,208	-18.30
2023	-760,620	-64,908,800	-21.9	-92.6	-227.6	-113,881	-17.02

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5045)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-2.08%	-5.00%	-4.86%	-2.87%	-27.10%	-15.25%	-50.83%
2000	-1.84%	-4.86%	-3.58%	-0.54%	-43.54%	-15.14%	-36.55%
2005	-2.15%	-5.05%	-6.68%	-2.96%	-50.54%	-15.02%	-38.68%
2007	-2.12%	-4.94%	-6.81%	-2.90%	-50.55%	-14.67%	-37.16%
2010	-1.98%	-4.57%	-6.46%	-2.65%	-47.90%	-13.67%	-32.85%
2014	-2.24%	-5.09%	-7.38%	-3.39%	-47.74%	-15.02%	-30.29%
2017	-2.41%	-5.45%	-8.08%	-4.03%	-47.31%	-15.96%	-26.91%
2020	-2.52%	-5.67%	-8.51%	-4.55%	-46.31%	-16.48%	-24.27%
2023	-2.59%	-5.83%	-8.68%	-4.93%	-45.42%	-16.86%	-22.44%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from evaporative processes.
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.
Fuel[3] - VMT Matching by Fuel Type

Table 12-86. Impact on Sacramento Valley Air Basin Inventory of Change 12.15

Sacramento Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,274,816	48,658,028	215.4	2204.0	197.0	31,033	10.42
2000	1,518,151	60,879,456	89.7	892.2	163.1	37,013	7.07
2005	1,698,989	68,124,624	60.4	555.6	129.9	42,295	7.08
2007	1,817,064	71,630,616	55.0	499.7	120.2	44,610	7.03
2010	1,969,133	74,875,336	48.8	425.4	104.7	46,392	6.77
2014	2,065,937	80,023,600	35.6	295.2	75.7	49,471	6.40
2017	2,154,850	83,938,640	28.9	230.0	60.5	52,067	6.20
2020	2,246,959	87,433,544	24.8	188.7	50.1	54,284	6.18
2023	2,339,770	90,662,608	22.6	165.1	43.4	56,460	6.24

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.50)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,237,256	45,631,808	201.9	2095.9	137.2	25,413	4.49
2000	1,479,047	56,912,088	85.6	879.2	82.0	29,786	3.93
2005	1,656,074	64,188,484	56.3	537.5	63.2	35,259	4.11
2007	1,772,208	67,571,552	51.4	485.1	59.1	37,373	4.20
2010	1,921,162	70,735,288	45.6	413.2	53.6	39,064	4.28
2014	2,013,171	75,488,296	33.0	285.1	40.1	41,476	4.36
2017	2,098,532	79,107,024	26.7	221.1	32.9	43,552	4.51
2020	2,186,689	82,271,032	22.8	180.5	27.6	45,201	4.66
2023	2,275,619	85,172,008	20.8	157.4	24.0	46,802	4.81

Difference (Ver. 2.5050 - Ver. 2.5045) in Sacramento Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-37560	-3026220	-13.5	-108.1	-59.9	-5,620	-5.93
2000	-39104	-3967368	-4.2	-13.0	-81.1	-7,226	-3.14
2005	-42915	-3936140	-4.1	-18.1	-66.7	-7,035	-2.97
2007	-44856	-4059064	-3.7	-14.6	-61.1	-7,237	-2.83
2010	-47971	-4140048	-3.2	-12.2	-51.2	-7,329	-2.49
2014	-52766	-4535304	-2.6	-10.1	-35.6	-7,996	-2.04
2017	-56318	-4831616	-2.2	-8.9	-27.7	-8,516	-1.69
2020	-60270	-5162512	-2.0	-8.1	-22.5	-9,084	-1.52
2023	-64151	-5490600	-1.9	-7.7	-19.4	-9,658	-1.43

Percentage Change in Sacramento Emission Inventories (relative to Ver. 2.5045)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-2.95%	-6.22%	-6.25%	-4.90%	-30.39%	-18.11%	-56.90%
2000	-2.58%	-6.52%	-4.62%	-1.45%	-49.73%	-19.52%	-44.36%
2005	-2.53%	-5.78%	-6.83%	-3.25%	-51.33%	-16.63%	-41.97%
2007	-2.47%	-5.67%	-6.66%	-2.93%	-50.85%	-16.22%	-40.24%
2010	-2.44%	-5.53%	-6.55%	-2.86%	-48.87%	-15.80%	-36.79%
2014	-2.55%	-5.67%	-7.20%	-3.42%	-47.05%	-16.16%	-31.86%
2017	-2.61%	-5.76%	-7.65%	-3.87%	-45.71%	-16.35%	-27.33%
2020	-2.68%	-5.90%	-8.03%	-4.31%	-44.87%	-16.73%	-24.67%
2023	-2.74%	-6.06%	-8.21%	-4.67%	-44.81%	-17.11%	-22.94%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from evaporative processes

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear

Fuel[3] - VMT Matching by Fuel Type

Table 12-87. Impact on San Diego Air Basin Inventory of Change 12.15

San Diego Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,567,046	57,477,284	221.5	2217.3	194.8	36,660	9.16
2000	1,874,501	72,232,144	81.2	829.1	131.6	40,390	6.59
2005	2,013,087	77,339,664	51.2	506.9	97.7	43,693	6.44
2007	2,098,513	80,059,032	45.4	446.7	91.0	45,950	6.38
2010	2,240,420	82,599,296	39.4	373.0	76.7	46,753	6.08
2014	2,327,605	85,886,136	29.9	266.5	57.5	48,809	5.91
2017	2,391,866	88,352,776	25.1	211.3	46.9	50,393	5.83
2020	2,457,305	90,816,720	21.9	175.2	39.2	51,933	5.84
2023	2,551,118	94,602,256	20.2	156.2	34.2	54,250	5.99

San Diego Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.50)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,545,039	55,695,720	215	2179	160	33,411	5.85
2000	1,849,785	70,049,760	79	827	88	36,489	4.79
2005	1,984,103	74,943,112	49	499	58	39,523	4.65
2007	2,067,644	77,550,736	43	439	53	41,609	4.68
2010	2,210,591	80,286,288	38	367	48	42,784	4.70
2014	2,293,489	83,253,688	29	261	36	44,319	4.70
2017	2,354,498	85,480,472	24	206	30	45,502	4.78
2020	2,416,712	87,704,976	21	171	25	46,648	4.89
2023	2,507,908	91,308,168	19	152	23	48,674	5.09

Difference (Ver. 2.5050 - Ver. 2.5045) in San Diego Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-22,007	-1,781,564	-6.6	-38.6	-34.6	-3,250	-3.31
2000	-24,716	-2,182,384	-2.0	-2.5	-43.4	-3,901	-1.80
2005	-28,984	-2,396,552	-2.1	-7.4	-40.1	-4,170	-1.78
2007	-30,869	-2,508,296	-2.1	-7.9	-37.5	-4,341	-1.70
2010	-29,829	-2,313,008	-1.7	-6.2	-29.0	-3,969	-1.38
2014	-34,116	-2,632,448	-1.4	-5.4	-21.3	-4,490	-1.21
2017	-37,368	-2,872,304	-1.2	-5.0	-16.9	-4,891	-1.04
2020	-40,593	-3,111,744	-1.1	-4.6	-13.8	-5,285	-0.95
2023	-43,210	-3,294,088	-1.0	-4.4	-11.7	-5,576	-0.90

Percentage Change in San Diego Emission Inventories (relative to Ver. 2.5045)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-1.40%	-3.10%	-2.97%	-1.74%	-17.75%	-8.86%	-36.15%
2000	-1.32%	-3.02%	-2.41%	-0.30%	-33.01%	-9.66%	-27.28%
2005	-1.44%	-3.10%	-4.13%	-1.46%	-41.11%	-9.54%	-27.71%
2007	-1.47%	-3.13%	-4.57%	-1.78%	-41.23%	-9.45%	-26.67%
2010	-1.33%	-2.80%	-4.22%	-1.66%	-37.76%	-8.49%	-22.72%
2014	-1.47%	-3.07%	-4.67%	-2.04%	-37.06%	-9.20%	-20.45%
2017	-1.56%	-3.25%	-4.95%	-2.35%	-36.09%	-9.71%	-17.92%
2020	-1.65%	-3.43%	-5.15%	-2.61%	-35.21%	-10.18%	-16.31%
2023	-1.69%	-3.48%	-5.19%	-2.80%	-34.11%	-10.28%	-15.01%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-88. Impact on San Francisco Bay Air Basin Inventory of Change 12.15

San Francisco Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,791,482	136,096,320	544.2	5014.7	478.6	79,054	23.12
2000	4,539,164	165,382,590	230.0	2094.3	327.7	90,395	16.35
2005	4,573,979	168,301,140	143.7	1224.3	247.8	94,019	14.91
2007	4,731,008	172,683,570	125.9	1065.4	224.5	97,907	14.71
2010	4,915,308	175,104,750	105.6	867.8	191.0	98,854	14.11
2014	5,039,932	180,753,710	77.1	602.0	138.6	101,887	13.37
2017	5,147,419	184,990,540	63.1	466.6	110.5	104,573	12.90
2020	5,264,958	189,228,260	54.4	379.9	90.9	107,236	12.73
2023	5,390,386	193,460,990	49.6	330.4	78.4	109,886	12.75

San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.50)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,736,332	131,079,910	527.4	4949.8	375.7	69,459	12.99
2000	4,482,899	159,858,610	225.6	2101.8	212.8	80,240	11.50
2005	4,508,814	162,538,700	137.6	1200.9	146.2	83,877	10.50
2007	4,661,293	166,557,410	120.1	1044.1	127.7	87,125	10.43
2010	4,838,772	168,633,490	100.4	849.0	107.5	87,619	10.20
2014	4,955,524	173,712,480	72.8	586.1	78.9	89,754	10.08
2017	5,057,204	177,521,460	59.4	452.3	64.0	91,712	10.17
2020	5,169,034	181,332,000	51.1	366.8	53.6	93,674	10.34
2023	5,288,756	185,137,150	46.5	318.1	47.0	95,625	10.54

Difference (Ver. 2.5050 - Ver. 2.5045) in San Francisco Bay Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-55,150	-5,016,410	-16.7	-64.9	-102.9	-9,595	-10.14
2000	-56,265	-5,523,980	-4.4	7.4	-114.9	-10,155	-4.85
2005	-65,165	-5,762,440	-6.1	-23.3	-101.6	-10,142	-4.42
2007	-69,715	-6,126,160	-5.8	-21.3	-96.8	-10,782	-4.27
2010	-76,536	-6,471,260	-5.2	-18.7	-83.5	-11,235	-3.91
2014	-84,408	-7,041,230	-4.3	-15.9	-59.7	-12,133	-3.30
2017	-90,215	-7,469,080	-3.7	-14.2	-46.6	-12,861	-2.73
2020	-95,924	-7,896,260	-3.4	-13.1	-37.3	-13,562	-2.40
2023	-101,630	-8,323,840	-3.1	-12.3	-31.4	-14,261	-2.21

Percentage Change in San Francisco Bay Emission Inventories (relative to Ver. 2.5045)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-1.45%	-3.69%	-3.07%	-1.29%	-21.51%	-12.14%	-43.83%
2000	-1.24%	-3.34%	-1.91%	0.35%	-35.05%	-11.23%	-29.64%
2005	-1.42%	-3.42%	-4.24%	-1.91%	-40.99%	-10.79%	-29.61%
2007	-1.47%	-3.55%	-4.59%	-2.00%	-43.11%	-11.01%	-29.06%
2010	-1.56%	-3.70%	-4.96%	-2.16%	-43.73%	-11.37%	-27.74%
2014	-1.67%	-3.90%	-5.56%	-2.64%	-43.06%	-11.91%	-24.66%
2017	-1.75%	-4.04%	-5.90%	-3.05%	-42.12%	-12.30%	-21.15%
2020	-1.82%	-4.17%	-6.17%	-3.45%	-41.03%	-12.65%	-18.83%
2023	-1.89%	-4.30%	-6.21%	-3.72%	-40.02%	-12.98%	-17.37%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-89. Impact on San Joaquin Valley Air Basin Inventory of Change 12.15

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,279,267	53,535,128	222.6	2304.9	306.8	40,717	21.02
2000	1,789,931	77,188,680	106.5	1056.8	315.0	54,226	12.70
2005	2,026,420	87,160,304	78.2	705.6	274.8	62,912	13.22
2007	2,197,510	93,262,648	72.5	647.0	252.3	67,147	12.88
2010	2,412,169	98,288,880	65.3	559.8	206.7	68,940	11.63
2014	2,552,338	106,823,070	49.1	395.7	153.4	75,435	11.00
2017	2,704,630	114,862,440	41.0	314.0	127.4	82,350	10.66
2020	2,872,522	122,591,290	35.6	262.4	104.7	88,293	10.47
2023	3,047,654	130,107,720	32.6	233.6	89.7	93,786	10.45

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.50)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,216,824	45,962,660	197.2	2146.8	145.9	25,728	4.49
2000	1,718,132	67,534,608	96.5	1024.9	102.4	35,812	4.67
2005	1,939,609	76,538,128	66.8	657.3	79.3	42,956	4.90
2007	2,107,607	82,480,304	62.1	603.8	76.0	46,899	5.12
2010	2,322,059	87,957,792	56.6	526.0	68.3	49,650	5.29
2014	2,447,596	94,888,144	41.7	365.9	51.4	53,258	5.46
2017	2,583,723	101,152,060	34.1	285.7	42.5	56,891	5.73
2020	2,741,452	107,784,100	29.4	236.4	36.2	60,850	6.07
2023	2,907,917	114,356,040	26.8	209.3	32.0	64,612	6.43

Difference (Ver. 2.5050 - Ver. 2.5045) in San Joaquin Valley Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-62,443	-7,572,468	-25.4	-158.1	-161.0	-14,989	-16.53
2000	-71,799	-9,654,072	-10.0	-31.9	-212.6	-18,414	-8.02
2005	-86,811	-10,622,176	-11.4	-48.4	-195.5	-19,956	-8.32
2007	-89,903	-10,782,344	-10.5	-43.2	-176.3	-20,248	-7.76
2010	-90,110	-10,331,088	-8.7	-33.8	-138.5	-19,290	-6.34
2014	-104,742	-11,934,926	-7.4	-29.8	-102.0	-22,177	-5.55
2017	-120,907	-13,710,380	-6.9	-28.3	-84.9	-25,459	-4.93
2020	-131,070	-14,807,190	-6.2	-26.0	-68.5	-27,443	-4.40
2023	-139,737	-15,751,680	-5.7	-24.3	-57.7	-29,174	-4.02

Percentage Change in San Joaquin Valley Emission Inventories (relative to Ver. 2.5045)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-4.88%	-14.14%	-11.42%	-6.86%	-52.46%	-36.81%	-78.66%
2000	-4.01%	-12.51%	-9.42%	-3.02%	-67.50%	-33.96%	-63.18%
2005	-4.28%	-12.19%	-14.55%	-6.85%	-71.14%	-31.72%	-62.95%
2007	-4.09%	-11.56%	-14.45%	-6.68%	-69.86%	-30.15%	-60.23%
2010	-3.74%	-10.51%	-13.26%	-6.03%	-66.98%	-27.98%	-54.52%
2014	-4.10%	-11.17%	-15.11%	-7.54%	-66.48%	-29.40%	-50.41%
2017	-4.47%	-11.94%	-16.82%	-9.01%	-66.61%	-30.92%	-46.22%
2020	-4.56%	-12.08%	-17.48%	-9.91%	-65.40%	-31.08%	-41.99%
2023	-4.59%	-12.11%	-17.61%	-10.41%	-64.38%	-31.11%	-38.46%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-90. Impact on South Coast Air Basin Inventory of Change 12.15

South Coast Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.45)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,369,264	258,397,090	964.2	9205.9	865.1	157,885	41.65
2000	8,667,431	309,433,410	383.4	3740.7	601.1	177,195	29.73
2005	9,686,220	350,403,970	254.2	2385.4	501.1	203,355	30.93
2007	9,860,916	355,615,360	213.9	1989.7	432.7	205,147	29.48
2010	10,426,589	365,454,110	177.3	1606.0	349.2	207,360	27.54
2014	10,797,710	376,983,170	132.5	1143.0	272.8	216,712	27.06
2017	11,105,086	388,201,180	109.9	902.5	225.1	225,360	26.75
2020	11,412,823	399,391,550	94.9	740.7	186.7	232,701	26.67
2023	11,644,727	406,887,300	85.9	641.8	160.3	239,156	26.70

South Coast Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs
(Calculated Using EMFAC2011 ver 2.50.50)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,232,260	248,651,890	926.6	8963.6	678.7	140,024	25.09
2000	8,526,977	298,739,360	376.0	3739.5	394.8	158,722	21.53
2005	9,485,378	336,339,490	241.8	2323.9	275.8	179,695	21.23
2007	9,664,210	341,888,290	203.7	1943.6	236.6	182,018	20.73
2010	10,242,853	353,053,950	170.0	1577.3	201.1	186,573	20.61
2014	10,573,581	361,797,660	125.7	1115.0	152.9	191,281	20.40
2017	10,852,563	371,091,390	103.5	875.4	126.6	196,648	20.75
2020	11,138,933	380,855,680	88.9	715.0	106.9	201,716	21.24
2023	11,354,450	387,191,140	80.3	616.9	94.0	206,143	21.64

Difference (Ver. 2.5050 - Ver. 2.5045) in South Coast Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-137,004	-9,745,200	-37.6	-242.3	-186.4	-17,861	-16.56
2000	-140,454	-10,694,050	-7.3	-1.2	-206.3	-18,473	-8.20
2005	-200,842	-14,064,480	-12.4	-61.4	-225.3	-23,660	-9.71
2007	-196,706	-13,727,070	-10.2	-46.1	-196.1	-23,130	-8.74
2010	-183,736	-12,400,160	-7.3	-28.7	-148.1	-20,787	-6.93
2014	-224,129	-15,185,510	-6.8	-28.0	-119.9	-25,431	-6.65
2017	-252,523	-17,109,790	-6.4	-27.1	-98.5	-28,712	-6.00
2020	-273,890	-18,535,870	-5.9	-25.8	-79.8	-30,985	-5.43
2023	-290,277	-19,696,160	-5.6	-24.9	-66.2	-33,013	-5.07

Percentage Change in South Coast Emission Inventories (relative to Ver. 2.5045)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	-1.86%	-3.77%	-3.90%	-2.63%	-21.55%	-11.31%	-39.77%
2000	-1.62%	-3.46%	-1.91%	-0.03%	-34.31%	-10.43%	-27.58%
2005	-2.07%	-4.01%	-4.86%	-2.58%	-44.97%	-11.63%	-31.38%
2007	-1.99%	-3.86%	-4.76%	-2.32%	-45.32%	-11.27%	-29.66%
2010	-1.76%	-3.39%	-4.13%	-1.78%	-42.42%	-10.02%	-25.16%
2014	-2.08%	-4.03%	-5.16%	-2.45%	-43.95%	-11.73%	-24.60%
2017	-2.27%	-4.41%	-5.83%	-3.00%	-43.76%	-12.74%	-22.43%
2020	-2.40%	-4.64%	-6.25%	-3.48%	-42.72%	-13.32%	-20.36%
2023	-2.49%	-4.84%	-6.51%	-3.87%	-41.33%	-13.80%	-18.97%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type Using Populations

12.16 LIFETIME MILEAGE CALCULATIONS AND EVAPORATIVE I/M

Please refer to Section 3.3.4.3. Staff inadvertently left out the adjustment for evaporative I/M. This had the effect of overestimating the benefit for smog check areas. In general this change increases ROG emissions on the order of a couple percent.

Table 12-91. Impact on Statewide Inventory of Change 12.16

Statewide Summer Episodic On-Road Motor Vehicle Inventories
(Calculated Using EMFAC2011 ver 2.50.50)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	17,402,036	627,215,300	2,432	24,325	1,788	349,011	62.53
2000	20,918,632	772,769,920	1,031	10,380	1,061	403,712	54.83
2005	22,628,922	844,379,140	653	6,267	753	450,852	53.63
2007	23,529,334	874,683,840	573	5,475	679	469,678	53.71
2010	25,124,892	911,216,640	493	4,573	598	486,812	54.08
2014	26,007,884	949,234,750	361	3,199	450	505,994	54.14
2017	26,820,658	982,844,990	295	2,499	370	524,749	55.42
2020	27,702,826	1,017,386,400	254	2,049	312	543,466	57.12
2023	28,553,862	1,048,442,400	231	1,786	273	561,739	58.83

Statewide Summer Episodic On-Road LD Motor Vehicle Inventories with revised ODO/Fuel
(Calculated Using EMFAC2011 ver 2.50.51)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	17,402,036	627,215,300	2,432	24,325	1,788	349,011	62.53
2000	20,918,632	772,769,920	1,052	10,380	1,061	403,712	54.83
2005	22,628,922	844,379,140	669	6,267	753	450,852	53.63
2007	23,529,334	874,683,840	585	5,475	679	469,678	53.71
2010	25,124,892	911,216,640	503	4,573	598	486,812	54.08
2014	26,007,884	949,234,750	368	3,199	450	505,994	54.14
2017	26,820,658	982,844,990	301	2,499	370	524,749	55.42
2020	27,702,826	1,017,386,400	259	2,049	312	543,466	57.12
2023	28,553,862	1,048,442,400	235	1,786	273	561,739	58.83

Difference (Ver. 2.5051 - Ver. 2.5050) in Statewide Emission Inventories (tons per day)

Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	20.9	0.0	0.0	0	0.00
2005	0	0	16.1	0.0	0.0	0	0.00
2007	0	0	12.8	0.0	0.0	0	0.00
2010	0	0	10.2	0.0	0.0	0	0.00
2014	0	0	7.1	0.0	0.0	0	0.00
2017	0	0	6.2	0.0	0.0	0	0.00
2020	0	0	5.1	0.0	0.0	0	0.00
2023	0	0	4.2	0.0	0.0	0	0.00

Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5050)

Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	2.02%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	2.47%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	2.23%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	2.07%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.96%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	2.09%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	2.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.82%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-92. Impact on Sacramento Valley Air Basin Inventory of Change 12.16

Sacramento Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.50)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,237,256	45,631,808	201.9	2095.9	137.2	25,413	4.49
2000	1,479,047	56,912,088	85.6	879.2	82.0	29,786	3.93
2005	1,656,074	64,188,484	56.3	537.5	63.2	35,259	4.11
2007	1,772,208	67,571,552	51.4	485.1	59.1	37,373	4.20
2010	1,921,162	70,735,288	45.6	413.2	53.6	39,064	4.28
2014	2,013,171	75,488,296	33.0	285.1	40.1	41,476	4.36
2017	2,098,532	79,107,024	26.7	221.1	32.9	43,552	4.51
2020	2,186,689	82,271,032	22.8	180.5	27.6	45,201	4.66
2023	2,275,619	85,172,008	20.8	157.4	24.0	46,802	4.81
Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,237,256	45,631,808	201.9	2095.9	137.2	25,413	4.49
2000	1,479,047	56,912,088	87.0	879.2	82.0	29,786	3.93
2005	1,656,074	64,188,484	57.3	537.5	63.2	35,259	4.11
2007	1,772,208	67,571,552	52.2	485.1	59.1	37,373	4.20
2010	1,921,162	70,735,288	46.3	413.2	53.6	39,064	4.28
2014	2,013,171	75,488,296	33.5	285.1	40.1	41,476	4.36
2017	2,098,532	79,107,024	27.1	221.1	32.9	43,552	4.51
2020	2,186,689	82,271,032	23.1	180.5	27.6	45,201	4.66
2023	2,275,619	85,172,008	21.1	157.4	24.0	46,802	4.81
Difference (Ver. 2.5051 - Ver. 2.5050) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	1.4	0.0	0.0	0	0.00
2005	0	0	1.1	0.0	0.0	0	0.00
2007	0	0	0.9	0.0	0.0	0	0.00
2010	0	0	0.7	0.0	0.0	0	0.00
2014	0	0	0.5	0.0	0.0	0	0.00
2017	0	0	0.4	0.0	0.0	0	0.00
2020	0	0	0.3	0.0	0.0	0	0.00
2023	0	0	0.3	0.0	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5050)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.65%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	1.89%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	1.70%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	1.50%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.43%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	1.50%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	1.53%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.44%	0.00%	0.00%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-93. Impact on San Diego Air Basin Inventory of Change 12.16

San Diego Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.50)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,545,039	55,695,720	214.9	2178.7	160.3	33,411	5.85
2000	1,849,785	70,049,760	79.2	826.7	88.1	36,489	4.79
2005	1,984,103	74,943,112	49.1	499.5	57.5	39,523	4.65
2007	2,067,644	77,550,736	43.3	438.8	53.5	41,609	4.68
2010	2,210,591	80,286,288	37.7	366.8	47.7	42,784	4.70
2014	2,293,489	83,253,688	28.5	261.0	36.2	44,319	4.70
2017	2,354,498	85,480,472	23.9	206.3	30.0	45,502	4.78
2020	2,416,712	87,704,976	20.8	170.6	25.4	46,648	4.89
2023	2,507,908	91,308,168	19.2	151.8	22.6	48,674	5.09
San Diego Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,545,039	55,695,720	215	2179	160	33,411	5.85
2000	1,849,785	70,049,760	81	827	88	36,489	4.79
2005	1,984,103	74,943,112	50	499	58	39,523	4.65
2007	2,067,644	77,550,736	44	439	53	41,609	4.68
2010	2,210,591	80,286,288	38	367	48	42,784	4.70
2014	2,293,489	83,253,688	29	261	36	44,319	4.70
2017	2,354,498	85,480,472	24	206	30	45,502	4.78
2020	2,416,712	87,704,976	21	171	25	46,648	4.89
2023	2,507,908	91,308,168	19	152	23	48,674	5.09
Difference (Ver. 2.5051 - Ver. 2.5050) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	1.6	0.0	0.0	0	0.00
2005	0	0	1.1	0.0	0.0	0	0.00
2007	0	0	0.9	0.0	0.0	0	0.00
2010	0	0	0.7	0.0	0.0	0	0.00
2014	0	0	0.5	0.0	0.0	0	0.00
2017	0	0	0.4	0.0	0.0	0	0.00
2020	0	0	0.4	0.0	0.0	0	0.00
2023	0	0	0.3	0.0	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5050)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.98%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	2.29%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	2.01%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	1.80%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.73%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	1.81%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	1.73%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.56%	0.00%	0.00%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-94. Impact on San Francisco Bay Air Basin Inventory of Change 12.16

San Francisco Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.50)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	3,736,332	131,079,910	527.4	4949.8	375.7	69,459	12.99
2000	4,482,899	159,858,610	225.6	2101.8	212.8	80,240	11.50
2005	4,508,814	162,538,700	137.6	1200.9	146.2	83,877	10.50
2007	4,661,293	166,557,410	120.1	1044.1	127.7	87,125	10.43
2010	4,838,772	168,633,490	100.4	849.0	107.5	87,619	10.20
2014	4,955,524	173,712,480	72.8	586.1	78.9	89,754	10.08
2017	5,057,204	177,521,460	59.4	452.3	64.0	91,712	10.17
2020	5,169,034	181,332,000	51.1	366.8	53.6	93,674	10.34
2023	5,288,756	185,137,150	46.5	318.1	47.0	95,625	10.54
San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	3,736,332	131,079,910	527.4	4949.8	375.7	69,459	12.99
2000	4,482,899	159,858,610	230.0	2101.8	212.8	80,240	11.50
2005	4,508,814	162,538,700	140.8	1200.9	146.2	83,877	10.50
2007	4,661,293	166,557,410	122.8	1044.1	127.7	87,125	10.43
2010	4,838,772	168,633,490	102.5	849.0	107.5	87,619	10.20
2014	4,955,524	173,712,480	74.4	586.1	78.9	89,754	10.08
2017	5,057,204	177,521,460	60.9	452.3	64.0	91,712	10.17
2020	5,169,034	181,332,000	52.3	366.8	53.6	93,674	10.34
2023	5,288,756	185,137,150	47.4	318.1	47.0	95,625	10.54
Difference (Ver. 2.5051 - Ver. 2.5050) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	4.4	0.0	0.0	0	0.00
2005	0	0	3.2	0.0	0.0	0	0.00
2007	0	0	2.7	0.0	0.0	0	0.00
2010	0	0	2.1	0.0	0.0	0	0.00
2014	0	0	1.6	0.0	0.0	0	0.00
2017	0	0	1.4	0.0	0.0	0	0.00
2020	0	0	1.2	0.0	0.0	0	0.00
2023	0	0	0.9	0.0	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5050)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.96%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	2.35%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	2.26%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	2.13%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	2.22%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	2.43%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	2.35%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	1.91%	0.00%	0.00%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-95. Impact on San Joaquin Valley Air Basin Inventory of Change 12.16

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.50)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,216,824	45,962,660	197.2	2146.8	145.9	25,728	4.49
2000	1,718,132	67,534,608	96.5	1024.9	102.4	35,812	4.67
2005	1,939,609	76,538,128	66.8	657.3	79.3	42,956	4.90
2007	2,107,607	82,480,304	62.1	603.8	76.0	46,899	5.12
2010	2,322,059	87,957,792	56.6	526.0	68.3	49,650	5.29
2014	2,447,596	94,888,144	41.7	365.9	51.4	53,258	5.46
2017	2,583,723	101,152,060	34.1	285.7	42.5	56,891	5.73
2020	2,741,452	107,784,100	29.4	236.4	36.2	60,850	6.07
2023	2,907,917	114,356,040	26.8	209.3	32.0	64,612	6.43
San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,216,824	45,962,660	197.2	2146.8	145.9	25,728	4.49
2000	1,718,132	67,534,608	98.1	1024.9	102.4	35,812	4.67
2005	1,939,609	76,538,128	68.0	657.3	79.3	42,956	4.90
2007	2,107,607	82,480,304	63.0	603.8	76.0	46,899	5.12
2010	2,322,059	87,957,792	57.4	526.0	68.3	49,650	5.29
2014	2,447,596	94,888,144	42.2	365.9	51.4	53,258	5.46
2017	2,583,723	101,152,060	34.6	285.7	42.5	56,891	5.73
2020	2,741,452	107,784,100	29.9	236.4	36.2	60,850	6.07
2023	2,907,917	114,356,040	27.5	209.3	32.0	64,612	6.43
Difference (Ver. 2.5051 - Ver. 2.5050) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	1.6	0.0	0.0	0	0.00
2005	0	0	1.2	0.0	0.0	0	0.00
2007	0	0	0.9	0.0	0.0	0	0.00
2010	0	0	0.8	0.0	0.0	0	0.00
2014	0	0	0.6	0.0	0.0	0	0.00
2017	0	0	0.5	0.0	0.0	0	0.00
2020	0	0	0.5	0.0	0.0	0	0.00
2023	0	0	0.7	0.0	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5050)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	1.64%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	1.81%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	1.51%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	1.35%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	1.36%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	1.48%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	1.68%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	2.45%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-96. Impact on South Coast Air Basin Inventory of Change 12.16

South Coast Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.50)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	7,232,260	248,651,890	926.6	8963.6	678.7	140,024	25.09
2000	8,526,977	298,739,360	376.0	3739.5	394.8	158,722	21.53
2005	9,485,378	336,339,490	241.8	2323.9	275.8	179,695	21.23
2007	9,664,210	341,888,290	203.7	1943.6	236.6	182,018	20.73
2010	10,242,853	353,053,950	170.0	1577.3	201.1	186,573	20.61
2014	10,573,581	361,797,660	125.7	1115.0	152.9	191,281	20.40
2017	10,852,563	371,091,390	103.5	875.4	126.6	196,648	20.75
2020	11,138,933	380,855,680	88.9	715.0	106.9	201,716	21.24
2023	11,354,450	387,191,140	80.3	616.9	94.0	206,143	21.64
South Coast Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	7,232,260	248,651,890	926.6	8963.6	678.7	140,024	25.09
2000	8,526,977	298,739,360	384.2	3739.5	394.8	158,722	21.53
2005	9,485,378	336,339,490	248.5	2323.9	275.8	179,695	21.23
2007	9,664,210	341,888,290	209.1	1943.6	236.6	182,018	20.73
2010	10,242,853	353,053,950	174.2	1577.3	201.1	186,573	20.61
2014	10,573,581	361,797,660	129.0	1115.0	152.9	191,281	20.40
2017	10,852,563	371,091,390	106.4	875.4	126.6	196,648	20.75
2020	11,138,933	380,855,680	91.3	715.0	106.9	201,716	21.24
2023	11,354,450	387,191,140	82.0	616.9	94.0	206,143	21.64
Difference (Ver. 2.5051 - Ver. 2.5050) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	8.2	0.0	0.0	0	0.00
2005	0	0	6.7	0.0	0.0	0	0.00
2007	0	0	5.4	0.0	0.0	0	0.00
2010	0	0	4.3	0.0	0.0	0	0.00
2014	0	0	3.3	0.0	0.0	0	0.00
2017	0	0	2.9	0.0	0.0	0	0.00
2020	0	0	2.4	0.0	0.0	0	0.00
2023	0	0	1.7	0.0	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5050)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	2.19%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	2.75%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	2.63%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	2.50%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	2.65%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	2.84%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	2.65%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	2.10%	0.00%	0.00%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type Using Populations							

12.17 REDUCTION IN LIGHT-HEAVY TRUCK STARTS

Please refer to Section 3.3.4.5. Reducing the number of starts reduces ROG and NOx, especially in the future. This effect is more pronounced in future years because of the large contribution of light heavy trucks to the hot soak and starts inventory in future calendar years.

Table 12-97. Impact on Statewide Inventory of Change 12.17

Statewide Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	17,402,036	627,215,300	2,432	24,325	1,788	349,011	62.53
2000	20,918,632	772,769,920	1,052	10,380	1,061	403,712	54.83
2005	22,628,922	844,379,140	669	6,267	753	450,852	53.63
2007	23,529,334	874,683,840	585	5,475	679	469,678	53.71
2010	25,124,892	911,216,640	503	4,573	598	486,812	54.08
2014	26,007,884	949,234,750	368	3,199	450	505,994	54.14
2017	26,820,658	982,844,990	301	2,499	370	524,749	55.42
2020	27,702,826	1,017,386,400	259	2,049	312	543,466	57.12
2023	28,553,862	1,048,442,400	235	1,786	273	561,739	58.83
Statewide Summer Episodic On-Road LD Motor Vehicle Inventories with reduced LHD1 Starts (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	17,402,036	627,215,300	2,380	23,929	1,768	348,170	62.33
2000	20,918,632	772,769,920	1,006	10,192	1,047	403,240	54.73
2005	22,628,922	844,379,140	630	6,135	733	450,340	53.57
2007	23,529,334	874,683,840	552	5,358	656	469,129	53.66
2010	25,124,892	911,216,640	472	4,469	572	486,216	54.04
2014	26,007,884	949,234,750	343	3,123	423	505,382	54.11
2017	26,820,658	982,844,990	278	2,437	344	524,108	55.40
2020	27,702,826	1,017,386,400	238	1,997	286	542,793	57.11
2023	28,553,862	1,048,442,400	216	1,741	249	561,027	58.82
Difference (Ver. 2.5052 - Ver. 2.5051) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	-51.6	-396.7	-19.6	-841	-0.20
2000	0	0	-45.6	-188.3	-13.9	-472	-0.09
2005	0	0	-38.3	-131.9	-19.9	-512	-0.06
2007	0	0	-33.5	-117.3	-22.5	-549	-0.05
2010	0	0	-31.1	-104.7	-25.8	-596	-0.04
2014	0	0	-25.3	-76.4	-26.5	-612	-0.03
2017	0	0	-22.9	-61.9	-26.3	-641	-0.02
2020	0	0	-20.5	-51.7	-25.8	-673	-0.01
2023	0	0	-18.7	-45.1	-24.8	-712	-0.01
Percentage Change in Emission Inventories (relative to Ver. 2.5051)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	-2.12%	-1.63%	-1.10%	-0.24%	-0.32%
2000	0.00%	0.00%	-4.33%	-1.81%	-1.31%	-0.12%	-0.17%
2005	0.00%	0.00%	-5.73%	-2.10%	-2.64%	-0.11%	-0.11%
2007	0.00%	0.00%	-5.73%	-2.14%	-3.32%	-0.12%	-0.09%
2010	0.00%	0.00%	-6.18%	-2.29%	-4.32%	-0.12%	-0.08%
2014	0.00%	0.00%	-6.88%	-2.39%	-5.89%	-0.12%	-0.05%
2017	0.00%	0.00%	-7.59%	-2.48%	-7.11%	-0.12%	-0.03%
2020	0.00%	0.00%	-7.94%	-2.52%	-8.25%	-0.12%	-0.02%
2023	0.00%	0.00%	-7.97%	-2.53%	-9.08%	-0.13%	-0.01%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-98. Impact on Sacramento Valley Air Basin Inventory of Change 12.17

Sacramento Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,237,256	45,631,808	201.9	2095.9	137.2	25,413	4.49
2000	1,479,047	56,912,088	87.0	879.2	82.0	29,786	3.93
2005	1,656,074	64,188,484	57.3	537.5	63.2	35,259	4.11
2007	1,772,208	67,571,552	52.2	485.1	59.1	37,373	4.20
2010	1,921,162	70,735,288	46.3	413.2	53.6	39,064	4.28
2014	2,013,171	75,488,296	33.5	285.1	40.1	41,476	4.36
2017	2,098,532	79,107,024	27.1	221.1	32.9	43,552	4.51
2020	2,186,689	82,271,032	23.1	180.5	27.6	45,201	4.66
2023	2,275,619	85,172,008	21.1	157.4	24.0	46,802	4.81
Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,237,256	45,631,808	196.2	2052.9	135.6	25,321	4.47
2000	1,479,047	56,912,088	82.9	857.8	80.9	29,735	3.92
2005	1,656,074	64,188,484	54.1	524.2	61.6	35,212	4.10
2007	1,772,208	67,571,552	49.2	472.8	57.2	37,322	4.20
2010	1,921,162	70,735,288	43.4	402.2	51.4	39,009	4.27
2014	2,013,171	75,488,296	31.1	277.5	37.9	41,421	4.36
2017	2,098,532	79,107,024	25.0	215.2	30.7	43,495	4.50
2020	2,186,689	82,271,032	21.2	176.0	25.5	45,142	4.65
2023	2,275,619	85,172,008	19.3	153.4	21.9	46,740	4.81
Difference (Ver. 2.5052 - Ver. 2.5051) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	-5.7	-43.0	-1.6	-92	-0.02
2000	0	0	-4.1	-21.4	-1.1	-51	-0.01
2005	0	0	-3.2	-13.3	-1.6	-47	-0.01
2007	0	0	-3.0	-12.3	-1.9	-50	-0.01
2010	0	0	-2.9	-11.1	-2.2	-55	0.00
2014	0	0	-2.4	-7.7	-2.2	-55	0.00
2017	0	0	-2.1	-5.9	-2.2	-56	0.00
2020	0	0	-1.9	-4.6	-2.1	-58	0.00
2023	0	0	-1.8	-4.0	-2.1	-62	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5051)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	-2.81%	-2.05%	-1.16%	-0.36%	-0.50%
2000	0.00%	0.00%	-4.71%	-2.43%	-1.36%	-0.17%	-0.28%
2005	0.00%	0.00%	-5.63%	-2.47%	-2.56%	-0.13%	-0.15%
2007	0.00%	0.00%	-5.76%	-2.54%	-3.16%	-0.13%	-0.13%
2010	0.00%	0.00%	-6.30%	-2.67%	-4.10%	-0.14%	-0.11%
2014	0.00%	0.00%	-7.14%	-2.69%	-5.54%	-0.13%	-0.06%
2017	0.00%	0.00%	-7.87%	-2.65%	-6.67%	-0.13%	-0.04%
2020	0.00%	0.00%	-8.34%	-2.54%	-7.76%	-0.13%	-0.02%
2023	0.00%	0.00%	-8.45%	-2.52%	-8.63%	-0.13%	-0.02%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-99. Impact on San Diego Air Basin Inventory of Change 12.17

San Diego Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,545,039	55,695,720	214.9	2178.7	160.3	33,411	5.85
2000	1,849,785	70,049,760	80.8	826.7	88.1	36,489	4.79
2005	1,984,103	74,943,112	50.2	499.5	57.5	39,523	4.65
2007	2,067,644	77,550,736	44.2	438.8	53.5	41,609	4.68
2010	2,210,591	80,286,288	38.4	366.8	47.7	42,784	4.70
2014	2,293,489	83,253,688	29.0	261.0	36.2	44,319	4.70
2017	2,354,498	85,480,472	24.3	206.3	30.0	45,502	4.78
2020	2,416,712	87,704,976	21.2	170.6	25.4	46,648	4.89
2023	2,507,908	91,308,168	19.5	151.8	22.6	48,674	5.09
San Diego Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,545,039	55,695,720	212	2157	159	33,364	5.83
2000	1,849,785	70,049,760	78	817	87	36,463	4.79
2005	1,984,103	74,943,112	48	492	56	39,496	4.65
2007	2,067,644	77,550,736	42	432	52	41,574	4.68
2010	2,210,591	80,286,288	36	360	46	42,742	4.70
2014	2,293,489	83,253,688	27	256	34	44,276	4.70
2017	2,354,498	85,480,472	23	202	28	45,456	4.78
2020	2,416,712	87,704,976	20	167	24	46,601	4.89
2023	2,507,908	91,308,168	18	149	21	48,624	5.09
Difference (Ver. 2.5052 - Ver. 2.5051) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	-2.8	-21.3	-1.1	-47	-0.01
2000	0	0	-2.9	-10.0	-0.8	-26	0.00
2005	0	0	-2.3	-7.1	-1.0	-27	0.00
2007	0	0	-2.2	-7.0	-1.4	-35	0.00
2010	0	0	-2.1	-6.6	-1.8	-42	0.00
2014	0	0	-1.8	-5.0	-1.9	-44	0.00
2017	0	0	-1.7	-4.2	-1.9	-45	0.00
2020	0	0	-1.6	-3.5	-1.8	-47	0.00
2023	0	0	-1.4	-3.1	-1.7	-49	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5051)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	-1.32%	-0.98%	-0.67%	-0.14%	-0.19%
2000	0.00%	0.00%	-3.60%	-1.22%	-0.91%	-0.07%	-0.10%
2005	0.00%	0.00%	-4.56%	-1.43%	-1.81%	-0.07%	-0.07%
2007	0.00%	0.00%	-4.89%	-1.59%	-2.70%	-0.08%	-0.06%
2010	0.00%	0.00%	-5.57%	-1.81%	-3.87%	-0.10%	-0.05%
2014	0.00%	0.00%	-6.32%	-1.93%	-5.23%	-0.10%	-0.03%
2017	0.00%	0.00%	-6.98%	-2.02%	-6.21%	-0.10%	-0.02%
2020	0.00%	0.00%	-7.32%	-2.08%	-7.09%	-0.10%	-0.02%
2023	0.00%	0.00%	-7.27%	-2.05%	-7.71%	-0.10%	-0.01%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-100. Impact on San Francisco Bay Air Basin Inventory of Change 12.17

San Francisco Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	3,736,332	131,079,910	527.4	4949.8	375.7	69,459	12.99
2000	4,482,899	159,858,610	230.0	2101.8	212.8	80,240	11.50
2005	4,508,814	162,538,700	140.8	1200.9	146.2	83,877	10.50
2007	4,661,293	166,557,410	122.8	1044.1	127.7	87,125	10.43
2010	4,838,772	168,633,490	102.5	849.0	107.5	87,619	10.20
2014	4,955,524	173,712,480	74.4	586.1	78.9	89,754	10.08
2017	5,057,204	177,521,460	60.9	452.3	64.0	91,712	10.17
2020	5,169,034	181,332,000	52.3	366.8	53.6	93,674	10.34
2023	5,288,756	185,137,150	47.4	318.1	47.0	95,625	10.54
San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	3,736,332	131,079,910	519.3	4888.3	373.1	69,325	12.95
2000	4,482,899	159,858,610	221.7	2071.9	210.9	80,166	11.49
2005	4,508,814	162,538,700	133.4	1180.1	142.9	83,789	10.49
2007	4,661,293	166,557,410	116.1	1025.5	124.0	87,032	10.42
2010	4,838,772	168,633,490	96.5	832.8	103.4	87,523	10.19
2014	4,955,524	173,712,480	69.4	574.2	74.9	89,660	10.07
2017	5,057,204	177,521,460	56.4	442.8	60.0	91,615	10.17
2020	5,169,034	181,332,000	48.2	358.7	49.8	93,575	10.33
2023	5,288,756	185,137,150	43.8	311.2	43.4	95,521	10.54
Difference (Ver. 2.5052 - Ver. 2.5051) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	-8.1	-61.5	-2.5	-134	-0.03
2000	0	0	-8.3	-29.8	-1.9	-74	-0.01
2005	0	0	-7.4	-20.8	-3.4	-88	-0.01
2007	0	0	-6.7	-18.6	-3.7	-92	-0.01
2010	0	0	-6.1	-16.2	-4.0	-95	-0.01
2014	0	0	-5.0	-11.9	-4.1	-94	0.00
2017	0	0	-4.5	-9.6	-4.0	-97	0.00
2020	0	0	-4.1	-8.1	-3.8	-99	0.00
2023	0	0	-3.6	-6.9	-3.6	-103	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5051)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	-1.54%	-1.24%	-0.67%	-0.19%	-0.25%
2000	0.00%	0.00%	-3.60%	-1.42%	-0.91%	-0.09%	-0.13%
2005	0.00%	0.00%	-5.26%	-1.73%	-2.31%	-0.10%	-0.10%
2007	0.00%	0.00%	-5.44%	-1.78%	-2.91%	-0.11%	-0.08%
2010	0.00%	0.00%	-5.91%	-1.91%	-3.74%	-0.11%	-0.07%
2014	0.00%	0.00%	-6.69%	-2.04%	-5.15%	-0.11%	-0.04%
2017	0.00%	0.00%	-7.38%	-2.12%	-6.21%	-0.11%	-0.03%
2020	0.00%	0.00%	-7.77%	-2.20%	-7.17%	-0.11%	-0.02%
2023	0.00%	0.00%	-7.50%	-2.16%	-7.76%	-0.11%	-0.01%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-101. Impact on San Joaquin Valley Air Basin Inventory of Change 12.17

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,216,824	45,962,660	197.2	2146.8	145.9	25,728	4.49
2000	1,718,132	67,534,608	98.1	1024.9	102.4	35,812	4.67
2005	1,939,609	76,538,128	68.0	657.3	79.3	42,956	4.90
2007	2,107,607	82,480,304	63.0	603.8	76.0	46,899	5.12
2010	2,322,059	87,957,792	57.4	526.0	68.3	49,650	5.29
2014	2,447,596	94,888,144	42.2	365.9	51.4	53,258	5.46
2017	2,583,723	101,152,060	34.6	285.7	42.5	56,891	5.73
2020	2,741,452	107,784,100	29.9	236.4	36.2	60,850	6.07
2023	2,907,917	114,356,040	27.5	209.3	32.0	64,612	6.43
San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,216,824	45,962,660	191.4	2103.3	144.0	25,635	4.46
2000	1,718,132	67,534,608	93.6	1002.9	101.0	35,758	4.66
2005	1,939,609	76,538,128	64.3	642.8	77.4	42,902	4.89
2007	2,107,607	82,480,304	59.5	590.4	73.6	46,836	5.12
2010	2,322,059	87,957,792	54.1	514.1	65.5	49,582	5.28
2014	2,447,596	94,888,144	39.5	357.3	48.6	53,188	5.45
2017	2,583,723	101,152,060	32.1	278.9	39.7	56,818	5.73
2020	2,741,452	107,784,100	27.5	230.9	33.3	60,773	6.07
2023	2,907,917	114,356,040	25.2	204.4	29.1	64,529	6.43
Difference (Ver. 2.5052 - Ver. 2.5051) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	-5.8	-43.6	-1.9	-93	-0.02
2000	0	0	-4.5	-22.0	-1.4	-54	-0.01
2005	0	0	-3.7	-14.5	-1.9	-54	-0.01
2007	0	0	-3.5	-13.4	-2.4	-63	-0.01
2010	0	0	-3.3	-11.9	-2.8	-68	-0.01
2014	0	0	-2.8	-8.6	-2.9	-69	0.00
2017	0	0	-2.5	-6.7	-2.9	-73	0.00
2020	0	0	-2.3	-5.5	-2.9	-77	0.00
2023	0	0	-2.3	-4.8	-2.8	-83	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5051)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	-2.96%	-2.03%	-1.29%	-0.36%	-0.50%
2000	0.00%	0.00%	-4.59%	-2.15%	-1.36%	-0.15%	-0.24%
2005	0.00%	0.00%	-5.51%	-2.21%	-2.45%	-0.13%	-0.15%
2007	0.00%	0.00%	-5.53%	-2.22%	-3.19%	-0.13%	-0.12%
2010	0.00%	0.00%	-5.76%	-2.27%	-4.07%	-0.14%	-0.10%
2014	0.00%	0.00%	-6.56%	-2.34%	-5.59%	-0.13%	-0.06%
2017	0.00%	0.00%	-7.29%	-2.36%	-6.79%	-0.13%	-0.04%
2020	0.00%	0.00%	-7.83%	-2.31%	-7.96%	-0.13%	-0.02%
2023	0.00%	0.00%	-8.25%	-2.31%	-8.86%	-0.13%	-0.01%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-102. Impact on South Coast Air Basin Inventory of Change 12.17

South Coast Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.51)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	7,232,260	248,651,890	926.6	8963.6	678.7	140,024	25.09
2000	8,526,977	298,739,360	384.2	3739.5	394.8	158,722	21.53
2005	9,485,378	336,339,490	248.5	2323.9	275.8	179,695	21.23
2007	9,664,210	341,888,290	209.1	1943.6	236.6	182,018	20.73
2010	10,242,853	353,053,950	174.2	1577.3	201.1	186,573	20.61
2014	10,573,581	361,797,660	129.0	1115.0	152.9	191,281	20.40
2017	10,852,563	371,091,390	106.4	875.4	126.6	196,648	20.75
2020	11,138,933	380,855,680	91.3	715.0	106.9	201,716	21.24
2023	11,354,450	387,191,140	82.0	616.9	94.0	206,143	21.64
South Coast Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	7,232,260	248,651,890	906.4	8803.5	669.6	139,697	25.01
2000	8,526,977	298,739,360	366.5	3666.8	388.6	158,538	21.49
2005	9,485,378	336,339,490	233.1	2271.2	267.1	179,481	21.20
2007	9,664,210	341,888,290	196.2	1899.3	227.4	181,800	20.72
2010	10,242,853	353,053,950	162.7	1539.1	190.9	186,342	20.60
2014	10,573,581	361,797,660	119.1	1086.4	142.3	191,037	20.39
2017	10,852,563	371,091,390	97.4	851.5	116.0	196,390	20.74
2020	11,138,933	380,855,680	83.2	694.5	96.5	201,442	21.24
2023	11,354,450	387,191,140	74.9	598.7	84.0	205,853	21.63
Difference (Ver. 2.5052 - Ver. 2.5051) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	-20.2	-160.1	-9.2	-327	-0.08
2000	0	0	-17.8	-72.7	-6.2	-184	-0.03
2005	0	0	-15.4	-52.8	-8.6	-214	-0.02
2007	0	0	-12.9	-44.3	-9.2	-218	-0.02
2010	0	0	-11.5	-38.2	-10.2	-231	-0.01
2014	0	0	-9.9	-28.6	-10.6	-244	-0.01
2017	0	0	-9.1	-23.9	-10.6	-259	-0.01
2020	0	0	-8.1	-20.5	-10.4	-274	0.00
2023	0	0	-7.1	-18.2	-10.0	-290	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5051)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	-2.18%	-1.79%	-1.35%	-0.23%	-0.31%
2000	0.00%	0.00%	-4.62%	-1.94%	-1.57%	-0.12%	-0.15%
2005	0.00%	0.00%	-6.18%	-2.27%	-3.14%	-0.12%	-0.11%
2007	0.00%	0.00%	-6.18%	-2.28%	-3.89%	-0.12%	-0.09%
2010	0.00%	0.00%	-6.62%	-2.42%	-5.07%	-0.12%	-0.07%
2014	0.00%	0.00%	-7.66%	-2.56%	-6.93%	-0.13%	-0.04%
2017	0.00%	0.00%	-8.52%	-2.73%	-8.36%	-0.13%	-0.03%
2020	0.00%	0.00%	-8.87%	-2.87%	-9.72%	-0.14%	-0.02%
2023	0.00%	0.00%	-8.67%	-2.95%	-10.68%	-0.14%	-0.02%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type Using Populations							

12.18 DEFAULT ZEV IMPLEMENTATION ASSUMPTIONS

Please refer to Section 3.3.4.6. The change in the number of ZEVs had no measurable impact on the emissions inventory. EMFAC2011-LDV was finalized based on the working inventory *EMFAC2010-2.50.53*.

Table 12-103. Impact on Statewide Inventory of Change 12.18

Statewide Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	17,402,036	627,215,300	2,380	23,929	1,768	348,170	62.33
2000	20,918,632	772,769,920	1,006	10,192	1,047	403,240	54.73
2005	22,628,922	844,379,140	630	6,135	733	450,340	53.57
2007	23,529,334	874,683,840	552	5,358	656	469,129	53.66
2010	25,124,892	911,216,640	472	4,469	572	486,216	54.04
2014	26,007,884	949,234,750	343	3,123	423	505,382	54.11
2017	26,820,658	982,844,990	278	2,437	344	524,108	55.40
2020	27,702,826	1,017,386,400	238	1,997	286	542,793	57.11
2023	28,553,862	1,048,442,400	216	1,741	249	561,027	58.82
Statewide Summer Episodic On-Road LD Motor Vehicle Inventories with increased ZEV implementation (Calculated Using EMFAC2010 ver 2.50.53)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	17,402,036	627,215,300	2,380	23,929	1,768	348,170	62.33
2000	20,918,632	772,769,920	1,006	10,192	1,047	403,240	54.73
2005	22,628,922	844,379,140	630	6,135	733	450,340	53.57
2007	23,529,334	874,683,840	552	5,358	656	469,129	53.66
2010	25,124,892	911,216,640	472	4,469	572	486,216	54.04
2014	26,007,884	949,234,750	343	3,123	423	505,382	54.11
2017	26,820,658	982,844,990	278	2,438	344	524,108	55.40
2020	27,702,826	1,017,386,400	238	1,997	286	542,793	57.11
2023	28,553,862	1,048,442,400	216	1,741	249	561,027	58.82
Difference (Ver. 2.5053 - Ver. 2.5052) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.1	0.0	0	0.00
2023	0	0	0.0	0.1	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5052)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-104. Impact on Sacramento Valley Air Basin Inventory of Change 12.18

Sacramento Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,237,256	45,631,808	196.2	2052.9	135.6	25,321	4.47
2000	1,479,047	56,912,088	82.9	857.8	80.9	29,735	3.92
2005	1,656,074	64,188,484	54.1	524.2	61.6	35,212	4.10
2007	1,772,208	67,571,552	49.2	472.8	57.2	37,322	4.20
2010	1,921,162	70,735,288	43.4	402.2	51.4	39,009	4.27
2014	2,013,171	75,488,296	31.1	277.5	37.9	41,421	4.36
2017	2,098,532	79,107,024	25.0	215.2	30.7	43,495	4.50
2020	2,186,689	82,271,032	21.2	176.0	25.5	45,142	4.65
2023	2,275,619	85,172,008	19.3	153.4	21.9	46,740	4.81

Sacramento Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.53)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,237,256	45,631,808	196.2	2052.9	135.6	25,321	4.47
2000	1,479,047	56,912,088	82.9	857.8	80.9	29,735	3.92
2005	1,656,074	64,188,484	54.1	524.2	61.6	35,212	4.10
2007	1,772,208	67,571,552	49.2	472.8	57.2	37,322	4.20
2010	1,921,162	70,735,288	43.4	402.2	51.4	39,009	4.27
2014	2,013,171	75,488,296	31.1	277.5	37.9	41,421	4.36
2017	2,098,532	79,107,024	25.0	215.2	30.7	43,495	4.50
2020	2,186,689	82,271,032	21.2	176.0	25.5	45,142	4.65
2023	2,275,619	85,172,008	19.3	153.4	21.9	46,740	4.81

Difference (Ver. 2.5053 - Ver. 2.5052) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.0	0.0	0	0.00
2023	0	0	0.0	0.0	0.0	0	0.00

Percentage Change in Emission Inventories (relative to Ver. 2.5052)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-105. Impact on San Diego Air Basin Inventory of Change 12.18

San Diego Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,545,039	55,695,720	212.1	2157.4	159.2	33,364	5.83
2000	1,849,785	70,049,760	77.9	816.6	87.3	36,463	4.79
2005	1,984,103	74,943,112	47.9	492.3	56.5	39,496	4.65
2007	2,067,644	77,550,736	42.1	431.8	52.0	41,574	4.68
2010	2,210,591	80,286,288	36.3	360.1	45.9	42,742	4.70
2014	2,293,489	83,253,688	27.2	256.0	34.3	44,276	4.70
2017	2,354,498	85,480,472	22.6	202.1	28.1	45,456	4.78
2020	2,416,712	87,704,976	19.6	167.1	23.6	46,601	4.89
2023	2,507,908	91,308,168	18.0	148.7	20.8	48,624	5.09
San Diego Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.53)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,545,039	55,695,720	212	2157	159	33,364	5.83
2000	1,849,785	70,049,760	78	817	87	36,463	4.79
2005	1,984,103	74,943,112	48	492	56	39,496	4.65
2007	2,067,644	77,550,736	42	432	52	41,574	4.68
2010	2,210,591	80,286,288	36	360	46	42,742	4.70
2014	2,293,489	83,253,688	27	256	34	44,276	4.70
2017	2,354,498	85,480,472	23	202	28	45,456	4.78
2020	2,416,712	87,704,976	20	167	24	46,601	4.89
2023	2,507,908	91,308,160	18	149	21	48,624	5.09
Difference (Ver. 2.5053 - Ver. 2.5052) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.0	0.0	0	0.00
2023	0	-8	0.0	0.0	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5052)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.01%	0.01%	0.00%	0.00%
2023	0.00%	0.00%	0.01%	0.01%	0.01%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-106. Impact on San Francisco Bay Air Basin Inventory of Change 12.18

San Francisco Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	3,736,332	131,079,910	519.3	4888.3	373.1	69,325	12.95
2000	4,482,899	159,858,610	221.7	2071.9	210.9	80,166	11.49
2005	4,508,814	162,538,700	133.4	1180.1	142.9	83,789	10.49
2007	4,661,293	166,557,410	116.1	1025.5	124.0	87,032	10.42
2010	4,838,772	168,633,490	96.5	832.8	103.4	87,523	10.19
2014	4,955,524	173,712,480	69.4	574.2	74.9	89,660	10.07
2017	5,057,204	177,521,460	56.4	442.8	60.0	91,615	10.17
2020	5,169,034	181,332,000	48.2	358.7	49.8	93,575	10.33
2023	5,288,756	185,137,150	43.8	311.2	43.4	95,521	10.54
San Francisco Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.53)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	3,736,332	131,079,910	519.3	4888.3	373.1	69,325	12.95
2000	4,482,899	159,858,610	221.7	2071.9	210.9	80,166	11.49
2005	4,508,814	162,538,700	133.4	1180.1	142.9	83,789	10.49
2007	4,661,293	166,557,410	116.1	1025.5	124.0	87,032	10.42
2010	4,838,772	168,633,490	96.5	832.8	103.4	87,523	10.19
2014	4,955,524	173,712,480	69.4	574.2	74.9	89,660	10.07
2017	5,057,204	177,521,460	56.4	442.8	60.0	91,615	10.17
2020	5,169,034	181,332,000	48.2	358.7	49.8	93,575	10.33
2023	5,288,756	185,137,170	43.8	311.2	43.4	95,521	10.54
Difference (Ver. 2.5053 - Ver. 2.5052) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.0	0.0	0	0.00
2023	0	20	0.0	0.0	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5052)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.

PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.

Fuel[3] - VMT Matching by Fuel Type

Table 12-107. Impact on San Joaquin Valley Air Basin Inventory of Change 12.18

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,216,824	45,962,660	191.4	2103.3	144.0	25,635	4.46
2000	1,718,132	67,534,608	93.6	1002.9	101.0	35,758	4.66
2005	1,939,609	76,538,128	64.3	642.8	77.4	42,902	4.89
2007	2,107,607	82,480,304	59.5	590.4	73.6	46,836	5.12
2010	2,322,059	87,957,792	54.1	514.1	65.5	49,582	5.28
2014	2,447,596	94,888,144	39.5	357.3	48.6	53,188	5.45
2017	2,583,723	101,152,060	32.1	278.9	39.7	56,818	5.73
2020	2,741,452	107,784,100	27.5	230.9	33.3	60,773	6.07
2023	2,907,917	114,356,040	25.2	204.4	29.1	64,529	6.43
San Joaquin Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.53)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,216,824	45,962,660	191.4	2103.3	144.0	25,635	4.46
2000	1,718,132	67,534,608	93.6	1002.9	101.0	35,758	4.66
2005	1,939,609	76,538,128	64.3	642.8	77.4	42,902	4.89
2007	2,107,607	82,480,304	59.5	590.4	73.6	46,836	5.12
2010	2,322,059	87,957,792	54.1	514.1	65.5	49,582	5.28
2014	2,447,596	94,888,152	39.5	357.3	48.6	53,188	5.45
2017	2,583,723	101,152,060	32.1	278.9	39.7	56,818	5.73
2020	2,741,452	107,784,100	27.5	230.9	33.3	60,773	6.07
2023	2,907,917	114,356,050	25.2	204.4	29.1	64,529	6.43
Difference (Ver. 2.5053 - Ver. 2.5052) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	8	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.0	0.0	0	0.00
2023	0	10	0.0	0.0	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5052)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-108. Impact on South Coast Air Basin Inventory of Change 12.18

South Coast Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.52)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	7,232,260	248,651,890	906.4	8803.5	669.6	139,697	25.01
2000	8,526,977	298,739,360	366.5	3666.8	388.6	158,538	21.49
2005	9,485,378	336,339,490	233.1	2271.2	267.1	179,481	21.20
2007	9,664,210	341,888,290	196.2	1899.3	227.4	181,800	20.72
2010	10,242,853	353,053,950	162.7	1539.1	190.9	186,342	20.60
2014	10,573,581	361,797,660	119.1	1086.4	142.3	191,037	20.39
2017	10,852,563	371,091,390	97.4	851.5	116.0	196,390	20.74
2020	11,138,933	380,855,680	83.2	694.5	96.5	201,442	21.24
2023	11,354,450	387,191,140	74.9	598.7	84.0	205,853	21.63
South Coast Summer Episodic On-Road Motor Vehicle Inventories With Updated CO2 BERs (Calculated Using EMFAC2010 ver 2.50.53)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	7,232,260	248,651,890	906.4	8803.5	669.6	139,697	25.01
2000	8,526,977	298,739,360	366.5	3666.8	388.6	158,538	21.49
2005	9,485,378	336,339,490	233.1	2271.2	267.1	179,481	21.20
2007	9,664,210	341,888,290	196.2	1899.3	227.4	181,800	20.72
2010	10,242,853	353,053,950	162.7	1539.1	190.9	186,342	20.60
2014	10,573,581	361,797,660	119.1	1086.4	142.3	191,037	20.39
2017	10,852,563	371,091,390	97.4	851.5	116.0	196,390	20.74
2020	11,138,933	380,855,680	83.2	694.5	96.5	201,442	21.24
2023	11,354,451	387,191,140	74.9	598.8	84.0	205,853	21.63
Difference (Ver. 2.5053 - Ver. 2.5052) in Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.0	0.0	0	0.00
2023	1	0	0.0	0.0	0.0	0	0.00
Percentage Change in Emission Inventories (relative to Ver. 2.5052)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type Using Populations							

12.19 FINAL EMFAC2011

The final change to the EMFAC2011 emissions inventory is the addition of the diesel fueled truck and bus categories calculated using EMFAC2011-HD to the emissions inventory generated using EMFAC2011-LDV. The combined total from the LDV and HD modules is used as input to EMFAC2011-SG. The impact on emissions is shown below.

Table 12-109. Impact on Statewide Inventory of Change 12.19

Statewide Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.53							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	17,402,036	627,215,300	2,380	23,929	1,768	348,170	62.33
2000	20,918,632	772,769,920	1,006	10,192	1,047	403,240	54.73
2005	22,628,922	844,379,140	630	6,135	733	450,340	53.57
2007	23,529,334	874,683,840	552	5,358	656	469,129	53.66
2010	25,124,892	911,216,640	472	4,469	572	486,216	54.04
2014	26,007,884	949,234,750	343	3,123	423	505,382	54.11
2017	26,820,658	982,844,990	278	2,438	344	524,108	55.40
2020	27,702,826	1,017,386,400	238	1,997	286	542,793	57.11
2023	28,553,862	1,048,442,400	216	1,741	249	561,027	58.82
Statewide Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	17,696,484	655,347,606	2,510	24,857	2,405	402,387	127.06
2000	21,316,586	813,291,572	1,069	10,580	1,930	475,088	92.00
2005	23,099,492	892,024,442	698	6,574	1,647	534,252	92.33
2007	23,992,395	923,818,396	613	5,747	1,492	555,905	89.04
2010	25,524,283	953,028,822	516	4,767	1,115	558,459	80.17
2014	26,496,651	1,000,024,272	372	3,322	861	594,951	69.90
2017	27,359,099	1,040,528,213	304	2,602	675	626,221	68.42
2020	28,261,858	1,079,010,873	263	2,149	543	651,853	70.11
2023	29,134,444	1,113,870,536	240	1,883	422	676,661	72.08
Difference (SG - Ver. 2.5053) in Statewide Emission Inventories (tons per day)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	294,448	28,132,306	130.4	928.0	636.2	54,216	64.74
2000	397,954	40,521,652	62.9	388.0	882.7	71,848	37.27
2005	470,570	47,645,302	67.9	438.7	914.4	83,912	38.76
2007	463,061	49,134,556	60.6	388.5	835.4	86,776	35.37
2010	399,391	41,812,182	43.9	298.5	543.5	72,243	26.13
2014	488,767	50,789,522	29.6	199.1	438.2	89,569	15.79
2017	538,441	57,683,223	25.3	164.4	331.7	102,113	13.01
2020	559,032	61,624,473	24.4	151.7	256.6	109,060	13.01
2023	580,582	65,428,136	23.6	141.7	173.7	115,633	13.26
Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5053)							
Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1.69%	4.49%	5.48%	3.88%	35.98%	15.57%	103.87%
2000	1.90%	5.24%	6.25%	3.81%	84.30%	17.82%	68.08%
2005	2.08%	5.64%	10.78%	7.15%	124.75%	18.63%	72.37%
2007	1.97%	5.62%	10.98%	7.25%	127.28%	18.50%	65.91%
2010	1.59%	4.59%	9.31%	6.68%	95.02%	14.86%	48.35%
2014	1.88%	5.35%	8.65%	6.37%	103.52%	17.72%	29.17%
2017	2.01%	5.87%	9.10%	6.75%	96.54%	19.48%	23.49%
2020	2.02%	6.06%	10.26%	7.59%	89.61%	20.09%	22.78%
2023	2.03%	6.24%	10.92%	8.14%	69.86%	20.61%	22.55%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-110. Impact on Sacramento Valley Air Basin Inventory of Change 12.19

Sacramento Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.53							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,237,256	45,631,808	196.2	2,052.9	135.6	25,321	4.47
2000	1,479,047	56,912,088	82.9	857.8	80.9	29,735	3.92
2005	1,656,074	64,188,484	54.1	524.2	61.6	35,212	4.10
2007	1,772,208	67,571,552	49.2	472.8	57.2	37,322	4.20
2010	1,921,162	70,735,288	43.4	402.2	51.4	39,009	4.27
2014	2,013,171	75,488,296	31.1	277.5	37.9	41,421	4.36
2017	2,098,532	79,107,024	25.0	215.2	30.7	43,495	4.50
2020	2,186,689	82,271,032	21.2	176.0	25.5	45,142	4.65
2023	2,275,619	85,172,008	19.3	153.4	21.9	46,740	4.81
Sacramento Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,266,806	48,108,360	207.7	2,112.8	189.3	30,144	10.11
2000	1,517,285	60,388,742	87.8	874.5	154.9	36,091	6.96
2005	1,702,055	68,337,230	60.0	558.3	139.3	42,720	7.28
2007	1,818,000	71,857,110	54.5	504.2	127.7	45,073	7.06
2010	1,960,623	74,371,974	47.3	426.4	98.3	45,466	6.45
2014	2,061,356	79,886,220	33.6	293.0	75.3	49,355	5.67
2017	2,151,771	84,095,784	27.0	227.9	58.3	52,508	5.60
2020	2,242,308	87,602,648	23.1	187.6	46.3	54,771	5.74
2023	2,333,593	90,811,038	21.2	164.1	36.8	56,920	5.89
Difference (SG - Ver. 2.5053) in Statewide Emission Inventories (tons per day)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	29,550	2,476,552	11.5	59.9	53.7	4,824	5.64
2000	38,238	3,476,654	4.9	16.7	74.0	6,356	3.04
2005	45,981	4,148,746	5.9	34.1	77.7	7,508	3.18
2007	45,792	4,285,558	5.3	31.5	70.5	7,751	2.86
2010	39,461	3,636,686	3.9	24.2	46.9	6,458	2.18
2014	48,185	4,397,924	2.5	15.5	37.4	7,934	1.32
2017	53,239	4,988,760	2.0	12.7	27.7	9,013	1.09
2020	55,619	5,331,616	1.9	11.6	20.9	9,629	1.08
2023	57,974	5,639,030	1.9	10.7	14.9	10,179	1.09
Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5053)							
Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	2.39%	5.43%	5.86%	2.92%	39.64%	19.05%	126.25%
2000	2.59%	6.11%	5.87%	1.95%	91.45%	21.38%	77.43%
2005	2.78%	6.46%	10.98%	6.51%	126.16%	21.32%	77.42%
2007	2.58%	6.34%	10.78%	6.66%	123.25%	20.77%	68.20%
2010	2.05%	5.14%	9.07%	6.02%	91.36%	16.55%	50.91%
2014	2.39%	5.83%	7.96%	5.59%	98.87%	19.15%	30.21%
2017	2.54%	6.31%	8.18%	5.90%	90.25%	20.72%	24.27%
2020	2.54%	6.48%	9.10%	6.60%	81.96%	21.33%	23.22%
2023	2.55%	6.62%	9.78%	6.96%	67.96%	21.78%	22.60%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-111. Impact on San Diego Air Basin Inventory of Change 12.19

San Diego Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.53							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,545,039	55,695,720	212.1	2,157.4	159.2	33,364	5.83
2000	1,849,785	70,049,760	77.9	816.6	87.3	36,463	4.79
2005	1,984,103	74,943,112	47.9	492.3	56.5	39,496	4.65
2007	2,067,644	77,550,736	42.1	431.8	52.0	41,574	4.68
2010	2,210,591	80,286,288	36.3	360.1	45.9	42,742	4.70
2014	2,293,489	83,253,688	27.2	256.0	34.3	44,276	4.70
2017	2,354,498	85,480,472	22.6	202.1	28.1	45,456	4.78
2020	2,416,712	87,704,976	19.6	167.1	23.6	46,601	4.89
2023	2,507,908	91,308,160	18.0	148.7	20.8	48,624	5.09
San Diego Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,563,181	57,264,281	216.1	2,169.3	192.0	36,353	9.34
2000	1,874,347	72,291,135	80.4	826.3	133.3	40,476	6.82
2005	2,012,525	77,498,458	50.4	502.0	102.0	44,007	6.64
2007	2,098,404	80,362,284	44.4	441.1	96.0	46,504	6.57
2010	2,236,543	82,630,299	37.9	366.5	75.2	46,833	6.08
2014	2,324,015	85,995,798	28.2	260.3	57.4	49,143	5.55
2017	2,387,137	88,513,963	23.5	205.8	45.0	50,859	5.45
2020	2,449,655	90,863,164	20.5	170.8	35.9	52,239	5.54
2023	2,542,202	94,653,759	18.9	152.6	28.8	54,588	5.75
Difference (SG - Ver. 2.5053) in Statewide Emission Inventories (tons per day)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	18,142	1,568,561	4.0	11.9	32.8	2,989	3.51
2000	24,562	2,241,375	2.5	9.7	46.0	4,013	2.03
2005	28,422	2,555,346	2.5	9.7	45.5	4,511	1.99
2007	30,760	2,811,548	2.4	9.3	43.9	4,930	1.89
2010	25,952	2,344,011	1.6	6.4	29.3	4,091	1.38
2014	30,526	2,742,110	1.1	4.3	23.1	4,868	0.85
2017	32,639	3,033,491	0.9	3.6	16.9	5,403	0.67
2020	32,943	3,158,188	0.9	3.7	12.3	5,638	0.65
2023	34,294	3,345,599	0.9	3.9	8.0	5,963	0.66
Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5053)							
Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1.17%	2.82%	1.88%	0.55%	20.61%	8.96%	60.10%
2000	1.33%	3.20%	3.27%	1.19%	52.67%	11.01%	42.43%
2005	1.43%	3.41%	5.18%	1.96%	80.50%	11.42%	42.69%
2007	1.49%	3.63%	5.62%	2.15%	84.43%	11.86%	40.47%
2010	1.17%	2.92%	4.50%	1.78%	63.81%	9.57%	29.41%
2014	1.33%	3.29%	3.90%	1.69%	67.42%	10.99%	18.14%
2017	1.39%	3.55%	3.82%	1.80%	60.14%	11.89%	14.06%
2020	1.36%	3.60%	4.39%	2.23%	52.16%	12.10%	13.24%
2023	1.37%	3.66%	4.92%	2.59%	38.30%	12.26%	12.93%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-112. Impact on San Francisco Bay Air Basin Inventory of Change 12.19

San Francisco Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.53							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,736,332	131,079,910	519.3	4,888.3	373.1	69,325	12.95
2000	4,482,899	159,858,610	221.7	2,071.9	210.9	80,166	11.49
2005	4,508,814	162,538,700	133.4	1,180.1	142.9	83,789	10.49
2007	4,661,293	166,557,410	116.1	1,025.5	124.0	87,032	10.42
2010	4,838,772	168,633,490	96.5	832.8	103.4	87,523	10.19
2014	4,955,524	173,712,480	69.4	574.2	74.9	89,660	10.07
2017	5,057,204	177,521,460	56.4	442.8	60.0	91,615	10.17
2020	5,169,034	181,332,000	48.2	358.7	49.8	93,575	10.33
2023	5,288,756	185,137,170	43.8	311.2	43.4	95,521	10.54
San Francisco Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,783,955	134,996,518	530.1	4,929.7	454.4	76,595	21.49
2000	4,547,994	165,491,199	228.3	2,100.5	323.7	89,996	16.60
2005	4,575,209	168,375,048	139.4	1,205.5	248.0	94,043	15.08
2007	4,736,313	173,274,236	122.0	1,050.5	229.5	98,711	14.96
2010	4,901,995	174,234,952	100.6	850.1	175.2	97,218	13.49
2014	5,030,221	180,290,209	72.1	586.2	131.0	101,222	12.17
2017	5,136,821	184,790,054	58.5	452.6	101.2	104,433	11.81
2020	5,249,149	188,906,075	50.3	368.4	80.1	106,962	11.91
2023	5,372,322	193,202,834	46.0	321.0	62.5	109,744	12.14
Difference (SG - Ver. 2.5053) in Statewide Emission Inventories (tons per day)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	47,623	3,916,608	10.8	41.4	81.2	7,270	8.53
2000	65,095	5,632,589	6.6	28.6	112.8	9,830	5.11
2005	66,395	5,836,348	6.0	25.4	105.1	10,254	4.59
2007	75,020	6,716,826	5.9	24.9	105.4	11,679	4.54
2010	63,223	5,601,462	4.1	17.3	71.8	9,695	3.30
2014	74,697	6,577,729	2.7	12.0	56.1	11,563	2.09
2017	79,617	7,268,594	2.2	9.8	41.2	12,817	1.64
2020	80,115	7,574,075	2.1	9.7	30.3	13,387	1.57
2023	83,566	8,065,664	2.2	9.8	19.1	14,223	1.60
Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5053)							
Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1.27%	2.99%	2.07%	0.85%	21.77%	10.49%	65.86%
2000	1.45%	3.52%	2.96%	1.38%	53.47%	12.26%	44.50%
2005	1.47%	3.59%	4.47%	2.15%	73.60%	12.24%	43.76%
2007	1.61%	4.03%	5.04%	2.43%	85.00%	13.42%	43.51%
2010	1.31%	3.32%	4.23%	2.08%	69.37%	11.08%	32.35%
2014	1.51%	3.79%	3.88%	2.09%	74.99%	12.90%	20.79%
2017	1.57%	4.09%	3.83%	2.22%	68.68%	13.99%	16.15%
2020	1.55%	4.18%	4.42%	2.71%	60.85%	14.31%	15.24%
2023	1.58%	4.36%	4.95%	3.14%	44.00%	14.89%	15.23%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-113. Impact on San Joaquin Valley Air Basin Inventory of Change 12.19

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.53							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,216,824	45,962,660	191.4	2,103.3	144.0	25,635	4.46
2000	1,718,132	67,534,608	93.6	1,002.9	101.0	35,758	4.66
2005	1,939,609	76,538,128	64.3	642.8	77.4	42,902	4.89
2007	2,107,607	82,480,304	59.5	590.4	73.6	46,836	5.12
2010	2,322,059	87,957,792	54.1	514.1	65.5	49,582	5.28
2014	2,447,596	94,888,152	39.5	357.3	48.6	53,188	5.45
2017	2,583,723	101,152,060	32.1	278.9	39.7	56,818	5.73
2020	2,741,452	107,784,100	27.5	230.9	33.3	60,773	6.07
2023	2,907,917	114,356,050	25.2	204.4	29.1	64,529	6.43
San Joaquin Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,269,165	52,198,974	214.1	2,204.0	285.3	38,521	20.07
2000	1,786,636	76,444,639	105.4	1,052.8	305.6	53,043	12.95
2005	2,026,950	87,485,059	76.8	697.9	293.5	63,828	13.65
2007	2,193,674	93,877,396	70.8	640.8	272.9	68,665	13.14
2010	2,396,408	97,700,102	62.0	549.5	194.8	67,933	11.39
2014	2,541,218	106,873,471	44.6	381.6	152.7	76,061	9.05
2017	2,689,189	114,919,482	36.7	300.9	116.4	83,058	8.67
2020	2,853,066	122,652,744	32.4	253.6	91.9	89,052	9.06
2023	3,024,536	130,172,967	30.3	227.8	69.6	94,551	9.47
Difference (SG - Ver. 2.5053) in Statewide Emission Inventories (tons per day)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	52,341	6,236,314	22.7	100.7	141.3	12,886	15.61
2000	68,504	8,910,031	11.8	49.9	204.7	17,286	8.29
2005	87,341	10,946,931	12.5	55.1	216.1	20,926	8.76
2007	86,067	11,397,092	11.3	50.4	199.3	21,829	8.03
2010	74,349	9,742,310	7.9	35.4	129.3	18,351	6.11
2014	93,622	11,985,319	5.2	24.3	104.2	22,873	3.60
2017	105,466	13,767,422	4.6	22.0	76.8	26,240	2.93
2020	111,614	14,868,644	4.8	22.7	58.6	28,279	2.99
2023	116,619	15,816,917	5.1	23.4	40.4	30,022	3.05
Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5053)							
Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	4.30%	13.57%	11.87%	4.79%	98.12%	50.27%	349.78%
2000	3.99%	13.19%	12.60%	4.98%	202.70%	48.34%	177.69%
2005	4.50%	14.30%	19.41%	8.58%	279.33%	48.78%	179.11%
2007	4.08%	13.82%	19.01%	8.53%	270.72%	46.61%	156.83%
2010	3.20%	11.08%	14.56%	6.89%	197.46%	37.01%	115.63%
2014	3.83%	12.63%	13.08%	6.79%	214.55%	43.00%	66.00%
2017	4.08%	13.61%	14.44%	7.89%	193.67%	46.18%	51.18%
2020	4.07%	13.79%	17.57%	9.84%	175.80%	46.53%	49.22%
2023	4.01%	13.83%	20.11%	11.43%	138.85%	46.52%	47.39%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-114 . Impact on South Coast Air Basin Inventory of Change 12.19

South Coast Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2010 ver 2.50.53							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,232,260	248,651,890	906.4	8,803.5	669.6	139,697	25.01
2000	8,526,977	298,739,360	366.5	3,666.8	388.6	158,538	21.49
2005	9,485,378	336,339,490	233.1	2,271.2	267.1	179,481	21.20
2007	9,664,210	341,888,290	196.2	1,899.3	227.4	181,800	20.72
2010	10,242,853	353,053,950	162.7	1,539.1	190.9	186,342	20.60
2014	10,573,581	361,797,660	119.1	1,086.4	142.3	191,037	20.39
2017	10,852,563	371,091,390	97.4	851.5	116.0	196,390	20.74
2020	11,138,933	380,855,680	83.2	694.5	96.5	201,442	21.24
2023	11,354,451	387,191,140	74.9	598.8	84.0	205,853	21.63
South Coast Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	7,334,677	257,490,447	933.1	8,922.0	853.6	156,396	44.03
2000	8,669,709	311,684,446	381.7	3,739.3	645.9	180,639	33.60
2005	9,655,054	351,679,769	248.8	2,342.8	535.9	205,753	33.67
2007	9,817,110	356,588,945	209.6	1,961.8	461.5	207,271	31.38
2010	10,375,851	365,619,731	171.4	1,581.1	337.4	207,790	28.03
2014	10,736,194	377,184,908	125.3	1,117.1	267.1	217,827	25.08
2017	11,031,711	388,633,894	103.0	879.7	216.2	227,094	24.74
2020	11,324,682	399,639,267	88.9	723.3	177.1	234,309	25.28
2023	11,545,941	407,150,864	80.4	626.8	134.8	240,730	25.79
Difference (SG - Ver. 2.5053) in Statewide Emission Inventories (tons per day)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	102,417	8,838,557	26.7	118.5	184.0	16,699	19.02
2000	142,732	12,945,086	15.2	72.5	257.3	22,101	12.10
2005	169,676	15,340,279	15.6	71.7	268.8	26,271	12.47
2007	152,900	14,700,655	13.5	62.5	234.1	25,471	10.66
2010	132,998	12,565,781	8.7	41.9	146.6	21,448	7.43
2014	162,613	15,387,248	6.2	30.7	124.8	26,790	4.69
2017	179,148	17,542,504	5.6	28.2	100.2	30,705	4.00
2020	185,749	18,783,587	5.7	28.9	80.6	32,867	4.04
2023	191,490	19,959,724	5.5	28.0	50.8	34,877	4.15
Percentage Change in Statewide Emission Inventories (relative to Ver. 2.5053)							
Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1.42%	3.55%	2.94%	1.35%	27.48%	11.95%	76.07%
2000	1.67%	4.33%	4.16%	1.98%	66.20%	13.94%	56.32%
2005	1.79%	4.56%	6.70%	3.16%	100.65%	14.64%	58.79%
2007	1.58%	4.30%	6.87%	3.29%	102.96%	14.01%	51.48%
2010	1.30%	3.56%	5.38%	2.72%	76.78%	11.51%	36.07%
2014	1.54%	4.25%	5.21%	2.83%	87.69%	14.02%	22.97%
2017	1.65%	4.73%	5.75%	3.31%	86.38%	15.63%	19.29%
2020	1.67%	4.93%	6.85%	4.16%	83.49%	16.32%	19.02%
2023	1.69%	5.16%	7.40%	4.68%	60.53%	16.94%	19.20%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes.							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear.							
Fuel[3] - VMT Matching by Fuel Type							

12.20 JULY 2012 UPDATE TO SANTA CLARA COUNTY VEHICLE STARTS

The impact on emissions of fixing the bug described in Section 3.3.4.7 is shown below.

Table 12-115 Impact on Statewide Inventory of Change 12.20

Statewide Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2011 SG ver 1.0)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	17,696,484	655,347,606	2,510	24,856	2,404	402,387	126.90
2000	21,316,586	813,291,572	1,069	10,580	1,930	475,087	91.84
2005	23,099,492	892,024,442	698	6,574	1,647	534,252	92.17
2007	23,992,395	923,818,396	612	5,746	1,492	555,905	88.88
2010	25,524,283	953,028,822	516	4,767	1,115	558,459	80.01
2014	26,496,651	1,000,024,272	372	3,321	861	594,951	69.75
2017	27,359,099	1,040,528,213	304	2,602	675	626,220	68.26
2020	28,261,858	1,079,010,873	262	2,149	543	651,852	69.96
2023	29,134,443	1,113,870,535	240	1,883	422	676,661	71.93
Statewide Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG ver 1.1)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	17,696,484	655,347,606	2,463	24,810	2,403	402,251	126.82
2000	21,316,586	813,291,572	1,048	10,552	1,928	474,984	91.80
2005	23,099,492	892,024,442	684	6,555	1,646	534,163	92.15
2007	23,992,395	923,818,396	601	5,729	1,491	555,814	88.86
2010	25,524,283	953,028,822	506	4,750	1,114	558,372	80.00
2014	26,496,651	1,000,024,272	365	3,305	860	594,869	69.74
2017	27,359,099	1,040,528,213	297	2,586	674	626,139	68.26
2020	28,261,858	1,079,010,873	256	2,132	542	651,771	69.96
2023	29,134,443	1,113,870,535	233	1,866	421	676,578	71.93
Difference (SG 1.1 - SG 1.0) in Statewide Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	-47.1	-46.4	-1.6	-136	-0.09
2000	0	0	-21.1	-28.0	-1.4	-103	-0.03
2005	0	0	-14.4	-18.1	-0.9	-89	-0.02
2007	0	0	-11.8	-17.4	-0.9	-91	-0.01
2010	0	0	-9.4	-16.6	-0.8	-87	-0.01
2014	0	0	-7.4	-16.0	-0.8	-82	0.00
2017	0	0	-6.8	-16.1	-0.8	-81	0.00
2020	0	0	-6.4	-16.2	-0.7	-82	0.00
2023	0	0	-6.3	-16.6	-0.7	-83	0.00
Percentage Change in Statewide Emission Inventories (relative to SG 1.0)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	-1.88%	-0.19%	-0.07%	-0.03%	-0.07%
2000	0.00%	0.00%	-1.97%	-0.26%	-0.07%	-0.02%	-0.04%
2005	0.00%	0.00%	-2.06%	-0.28%	-0.05%	-0.02%	-0.02%
2007	0.00%	0.00%	-1.93%	-0.30%	-0.06%	-0.02%	-0.01%
2010	0.00%	0.00%	-1.83%	-0.35%	-0.08%	-0.02%	-0.01%
2014	0.00%	0.00%	-1.98%	-0.48%	-0.09%	-0.01%	-0.01%
2017	0.00%	0.00%	-2.22%	-0.62%	-0.11%	-0.01%	0.00%
2020	0.00%	0.00%	-2.45%	-0.75%	-0.13%	-0.01%	0.00%
2023	0.00%	0.00%	-2.63%	-0.88%	-0.17%	-0.01%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-116 Impact on Sacramento Valley Air Basin Inventory of Change 12.20

Sacramento Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2011 SG ver 1.0)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,266,806	48,108,360	207.7	2,112.8	189.3	30,144	10.08
2000	1,517,285	60,388,741	87.7	874.5	154.8	36,091	6.93
2005	1,702,055	68,337,230	60.0	558.3	139.3	42,720	7.25
2007	1,818,000	71,857,110	54.5	504.2	127.7	45,073	7.03
2010	1,960,622	74,371,974	47.3	426.3	98.2	45,466	6.42
2014	2,061,356	79,886,220	33.5	292.9	75.3	49,355	5.65
2017	2,151,771	84,095,784	27.0	227.9	58.3	52,508	5.57
2020	2,242,308	87,602,648	23.1	187.6	46.3	54,771	5.71
2023	2,333,593	90,811,038	21.1	164.0	36.7	56,920	5.87
Sacramento Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG ver 1.1)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	1,266,806	48,108,360	207.7	2,112.8	189.3	30,144	10.08
2000	1,517,285	60,388,741	87.7	874.5	154.8	36,091	6.93
2005	1,702,055	68,337,230	60.0	558.3	139.3	42,720	7.25
2007	1,818,000	71,857,110	54.5	504.2	127.7	45,073	7.03
2010	1,960,622	74,371,974	47.3	426.3	98.2	45,466	6.42
2014	2,061,356	79,886,220	33.5	292.9	75.3	49,355	5.65
2017	2,151,771	84,095,784	27.0	227.9	58.3	52,508	5.57
2020	2,242,308	87,602,648	23.1	187.6	46.3	54,771	5.71
2023	2,333,593	90,811,038	21.1	164.0	36.7	56,920	5.87
Difference (SG 1.1 - SG 1.0) in Statewide Emission Inventories (tons per day)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.0	0.0	0	0.00
2023	0	0	0.0	0.0	0.0	0	0.00
Percentage Change in Statewide Emission Inventories (relative to SG 1.0)							
Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes							
PM10_Tot[2] - Total emissions from running, starting, idle processes, and from tire wear and brake wear							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-117 Impact on San Diego Air Basin Inventory of Change 12.20

San Diego Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2011 SG ver 1.0)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,563,181	57,264,281	216.1	2,169.3	192.0	36,353	9.34
2000	1,874,347	72,291,135	80.4	826.3	133.3	40,476	6.82
2005	2,012,525	77,498,458	50.4	502.0	101.9	44,007	6.63
2007	2,098,404	80,362,284	44.4	441.1	96.0	46,504	6.57
2010	2,236,543	82,630,299	37.9	366.5	75.2	46,833	6.08
2014	2,324,015	85,995,798	28.2	260.3	57.4	49,143	5.55
2017	2,387,137	88,513,963	23.5	205.8	45.0	50,859	5.45
2020	2,449,655	90,863,164	20.5	170.8	35.9	52,239	5.54
2023	2,542,202	94,653,759	18.9	152.6	28.8	54,588	5.75
San Diego Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG ver 1.1)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,563,181	57,264,281	216.1	2,169.3	192.0	36,353	9.34
2000	1,874,347	72,291,135	80.4	826.3	133.3	40,476	6.82
2005	2,012,525	77,498,458	50.4	502.0	101.9	44,007	6.63
2007	2,098,404	80,362,284	44.4	441.1	96.0	46,504	6.57
2010	2,236,543	82,630,299	37.9	366.5	75.2	46,833	6.08
2014	2,324,015	85,995,798	28.2	260.3	57.4	49,143	5.55
2017	2,387,137	88,513,963	23.5	205.8	45.0	50,859	5.45
2020	2,449,655	90,863,164	20.5	170.8	35.9	52,239	5.54
2023	2,542,202	94,653,759	18.9	152.6	28.8	54,588	5.75
Difference (SG 1.1 - SG 1.0) in Statewide Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.0	0.0	0	0.00
2023	0	0	0.0	0.0	0.0	0	0.00
Percentage Change in Statewide Emission Inventories (relative to SG 1.0)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%

ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes

PM10_Tot[2] evaporative processes.

Fuel[3] - VMT Matching by Fuel Type

Table 12-118 Impact on San Francisco Bay Air Basin Inventory of Change 12.20

San Francisco Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2011 SG ver 1.0)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,783,955	134,996,518	530.0	4,929.7	454.4	76,595	21.46
2000	4,547,994	165,491,199	228.3	2,100.5	323.6	89,996	16.58
2005	4,575,209	168,375,048	139.4	1,205.5	248.0	94,043	15.06
2007	4,736,313	173,274,236	122.0	1,050.4	229.4	98,711	14.94
2010	4,901,995	174,234,952	100.5	850.1	175.2	97,218	13.47
2014	5,030,221	180,290,209	72.1	586.1	131.0	101,222	12.14
2017	5,136,821	184,790,054	58.5	452.5	101.2	104,433	11.79
2020	5,249,149	188,906,075	50.3	368.4	80.1	106,962	11.89
2023	5,372,322	193,202,834	46.0	321.0	62.4	109,744	12.12
San Francisco Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG ver 1.1)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	3,783,955	134,996,518	482.9	4,883.3	452.7	76,459	21.38
2000	4,547,994	165,491,199	207.2	2,072.5	322.2	89,893	16.55
2005	4,575,209	168,375,048	125.0	1,187.4	247.1	93,955	15.04
2007	4,736,313	173,274,236	110.2	1,033.0	228.6	98,620	14.92
2010	4,901,995	174,234,952	91.1	833.4	174.3	97,131	13.46
2014	5,030,221	180,290,209	64.7	570.1	130.2	101,140	12.14
2017	5,136,821	184,790,054	51.8	436.4	100.4	104,352	11.78
2020	5,249,149	188,906,075	43.9	352.2	79.3	106,881	11.89
2023	5,372,322	193,202,834	39.7	304.3	61.7	109,662	12.12
Difference (SG 1.1 - SG 1.0) in Statewide Emission Inventories (tons per day)							
Cal. Year	Population	VMT (mi/d)	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0	0	-47.1	-46.4	-1.6	-136	-0.09
2000	0	0	-21.1	-28.0	-1.4	-103	-0.03
2005	0	0	-14.4	-18.1	-0.9	-89	-0.02
2007	0	0	-11.8	-17.4	-0.9	-91	-0.01
2010	0	0	-9.4	-16.6	-0.8	-87	-0.01
2014	0	0	-7.4	-16.0	-0.8	-82	0.00
2017	0	0	-6.8	-16.1	-0.8	-81	0.00
2020	0	0	-6.4	-16.2	-0.7	-82	0.00
2023	0	0	-6.3	-16.6	-0.7	-83	0.00
Percentage Change in Statewide Emission Inventories (relative to SG 1.0)							
Cal. Year	Population	VMT	ROG_Tot[1]	CO_Tot	NOx_Tot	CO2_Tot	PM10_Tot[2]
1990	0.00%	0.00%	-8.89%	-0.94%	-0.36%	-0.18%	-0.40%
2000	0.00%	0.00%	-9.22%	-1.33%	-0.43%	-0.11%	-0.21%
2005	0.00%	0.00%	-10.31%	-1.50%	-0.34%	-0.09%	-0.12%
2007	0.00%	0.00%	-9.67%	-1.66%	-0.37%	-0.09%	-0.09%
2010	0.00%	0.00%	-9.39%	-1.96%	-0.48%	-0.09%	-0.06%
2014	0.00%	0.00%	-10.23%	-2.73%	-0.59%	-0.08%	-0.03%
2017	0.00%	0.00%	-11.55%	-3.56%	-0.75%	-0.08%	-0.02%
2020	0.00%	0.00%	-12.75%	-4.40%	-0.91%	-0.08%	-0.01%
2023	0.00%	0.00%	-13.73%	-5.18%	-1.15%	-0.08%	-0.01%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes							
PM10_Tot[2] evaporative processes.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-119 Impact on San Joaquin Valley Air Basin Inventory of Change 12.20

San Joaquin Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2011 SG ver 1.0)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,269,165	52,198,974	214.1	2,204.0	285.3	38,521	20.05
2000	1,786,636	76,444,639	105.4	1,052.8	305.6	53,043	12.93
2005	2,026,949	87,485,059	76.7	697.9	293.4	63,828	13.63
2007	2,193,674	93,877,396	70.8	640.7	272.9	68,665	13.12
2010	2,396,408	97,700,102	61.9	549.5	194.7	67,933	11.37
2014	2,541,218	106,873,471	44.6	381.6	152.7	76,061	9.03
2017	2,689,189	114,919,482	36.7	300.9	116.4	83,058	8.64
2020	2,853,066	122,652,744	32.4	253.6	91.9	89,052	9.04
2023	3,024,536	130,172,967	30.3	227.8	69.5	94,551	9.45
San Joaquin Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG ver 1.1)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	1,269,165	52,198,974	214.1	2,204.0	285.3	38,521	20.05
2000	1,786,636	76,444,639	105.4	1,052.8	305.6	53,043	12.93
2005	2,026,949	87,485,059	76.7	697.9	293.4	63,828	13.63
2007	2,193,674	93,877,396	70.8	640.7	272.9	68,665	13.12
2010	2,396,408	97,700,102	61.9	549.5	194.7	67,933	11.37
2014	2,541,218	106,873,471	44.6	381.6	152.7	76,061	9.03
2017	2,689,189	114,919,482	36.7	300.9	116.4	83,058	8.64
2020	2,853,066	122,652,744	32.4	253.6	91.9	89,052	9.04
2023	3,024,536	130,172,967	30.3	227.8	69.5	94,551	9.45
Difference (SG 1.1 - SG 1.0) in Statewide Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.0	0.0	0	0.00
2023	0	0	0.0	0.0	0.0	0	0.00
Percentage Change in Statewide Emission Inventories (relative to SG 1.0)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes							
PM10_Tot[2] evaporative processes.							
Fuel[3] - VMT Matching by Fuel Type							

Table 12-120 Impact on South Coast Air Basin Inventory of Change 12.20

South Coast Summer Episodic On-Road Motor Vehicle Inventories (Calculated Using EMFAC2011 SG ver 1.0)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	7,334,677	257,490,447	933.1	8,922.0	853.6	156,396	44.02
2000	8,669,709	311,684,446	381.7	3,739.3	645.9	180,639	33.59
2005	9,655,054	351,679,769	248.8	2,342.8	535.9	205,753	33.66
2007	9,817,110	356,588,945	209.6	1,961.8	461.5	207,271	31.37
2010	10,375,851	365,619,731	171.4	1,581.1	337.4	207,790	28.02
2014	10,736,194	377,184,908	125.3	1,117.1	267.1	217,827	25.07
2017	11,031,711	388,633,894	103.0	879.7	216.2	227,094	24.73
2020	11,324,682	399,639,267	88.9	723.3	177.1	234,309	25.27
2023	11,545,941	407,150,864	80.4	626.8	134.8	240,730	25.78
South Coast Summer Episodic On-Road Motor Vehicle Inventories with EMFAC-HD (Calculated Using EMFAC2011 SG ver 1.1)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	7,334,677	257,490,447	933.10	8,921.97	853.56	156,395.69	44.02
2000	8,669,709	311,684,446	381.72	3,739.29	645.90	180,639.15	33.59
2005	9,655,054	351,679,769	248.76	2,342.83	535.94	205,752.73	33.66
2007	9,817,110	356,588,945	209.63	1,961.82	461.49	207,270.95	31.37
2010	10,375,851	365,619,731	171.43	1,581.08	337.42	207,789.91	28.02
2014	10,736,194	377,184,908	125.33	1,117.12	267.13	217,827.42	25.07
2017	11,031,711	388,633,894	102.96	879.65	216.17	227,094.23	24.73
2020	11,324,682	399,639,267	88.88	723.31	177.14	234,308.64	25.27
2023	11,545,941	407,150,864	80.39	626.77	134.82	240,729.76	25.78
Difference (SG 1.1 - SG 1.0) in Statewide Emission Inventories (tons per day)							
Cal. Year	**Population**	**VMT (mi/d)**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0	0	0.0	0.0	0.0	0	0.00
2000	0	0	0.0	0.0	0.0	0	0.00
2005	0	0	0.0	0.0	0.0	0	0.00
2007	0	0	0.0	0.0	0.0	0	0.00
2010	0	0	0.0	0.0	0.0	0	0.00
2014	0	0	0.0	0.0	0.0	0	0.00
2017	0	0	0.0	0.0	0.0	0	0.00
2020	0	0	0.0	0.0	0.0	0	0.00
2023	0	0	0.0	0.0	0.0	0	0.00
Percentage Change in Statewide Emission Inventories (relative to SG 1.0)							
Cal. Year	**Population**	**VMT**	**ROG_Tot[1]**	**CO_Tot**	**NOx_Tot**	**CO2_Tot**	**PM10_Tot[2]**
1990	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2000	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2005	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2007	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2010	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2014	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2017	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2020	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
2023	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
ROG_Tot[1] - This includes running, starting, idle exhaust emissions and emissions from all evaporative processes							
PM10_Tot[2] evaporative processes.							
Fuel[3] - VMT Matching by Fuel Type							

EXHIBIT 11

South Coast
Air Quality Management District
21865 Copley Drive, Diamond Bar, CA 91765-4182
(909) 396-2000 • www.aqmd.gov

SCAQMD Air Quality Significance Thresholds

Mass Daily Thresholds [a]		
Pollutant	**Construction [b]**	**Operation [c]**
NOx	100 lbs/day	55 lbs/day
VOC	75 lbs/day	55 lbs/day
PM10	150 lbs/day	150 lbs/day
PM2.5	55 lbs/day	55 lbs/day
SOx	150 lbs/day	150 lbs/day
CO	550 lbs/day	550 lbs/day
Lead	3 lbs/day	3 lbs/day
Toxic Air Contaminants (TACs), Odor, and GHG Thresholds		
TACs (including carcinogens and non-carcinogens)	Maximum Incremental Cancer Risk ≥ 10 in 1 million Cancer Burden > 0.5 excess cancer cases (in areas ≥ 1 in 1 million) Chronic & Acute Hazard Index ≥ 1.0 (project increment)	
Odor	Project creates an odor nuisance pursuant to SCAQMD Rule 402	
GHG	10,000 MT/yr CO2eq for industrial facilities	
Ambient Air Quality Standards for Criteria Pollutants [d]		
NO2 1-hour average annual arithmetic mean	SCAQMD is in attainment; project is significant if it causes or contributes to an exceedance of the following attainment standards: 0.18 ppm (state) 0.03 ppm (state) and 0.0534 ppm (federal)	
PM10 24-hour average annual average	10.4 μg/m^3 (construction)[e] & 2.5 μg/m^3 (operation) 1.0 μg/m^3	
PM2.5 24-hour average	10.4 μg/m^3 (construction)[e] & 2.5 μg/m^3 (operation)	
SO2 1-hour average 24-hour average	0.25 ppm (state) & 0.075 ppm (federal – 99th percentile) 0.04 ppm (state)	
Sulfate 24-hour average	25 μg/m^3 (state)	
CO 1-hour average 8-hour average	SCAQMD is in attainment; project is significant if it causes or contributes to an exceedance of the following attainment standards: 20 ppm (state) and 35 ppm (federal) 9.0 ppm (state/federal)	
Lead 30-day Average Rolling 3-month average	1.5 μg/m^3 (state) 0.15 μg/m^3 (federal)	

[a] Source: SCAQMD CEQA Handbook (SCAQMD, 1993)
[b] Construction thresholds apply to both the South Coast Air Basin and Coachella Valley (Salton Sea and Mojave Desert Air Basins).
[c] For Coachella Valley, the mass daily thresholds for operation are the same as the construction thresholds.
[d] Ambient air quality thresholds for criteria pollutants based on SCAQMD Rule 1303, Table A-2 unless otherwise stated.
[e] Ambient air quality threshold based on SCAQMD Rule 403.

KEY: lbs/day = pounds per day ppm = parts per million μg/m^3 = microgram per cubic meter ≥ = greater than or equal to
MT/yr CO2eq = metric tons per year of CO2 equivalents > = greater than

EXHIBIT 12

Regulation

- ▶ Mobile Source Regulatory Comparison: U.S. EPA/California vs. European Union
- ▶ U.S. EPA Tier 3 and California LEV III Rulemakings
- ▶ U.S. EPA Clean Air Nonroad Diesel Rule
- ▶ U.S. EPA 2007/2010 Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements
- ▶ U.S. EPA Light-Duty Tier 2 and Gasoline Sulfur Rulemaking
- ▶ The California Air Resources Board
- ▶ The U.S. Environmental Protection Agency's Motor Vehicle Compliance Program
- ▶ The Clean Air Act

U.S. EPA 2007/2010 Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements

In December 2000, EPA adopted a rulemaking to establish stringent standards designed to reduce emissions from on-road heavy-duty trucks and buses by up to 95 percent and to cut the allowable levels of sulfur in diesel fuel by 97 percent. The EPA rule is the most significant and far-reaching mobile source initiative since Congress adopted the 1970 Clean Air Act Amendments establishing the U.S. Mobile Source Emission Control Program.

Beginning with the 2007 model year, 100 percent of the on-road diesel HDEs will require the use of a diesel particulate filter and 50 percent of the engines will require NOx exhaust control technology; beginning with the 2010 model year, 100 percent of the on-road heavy-duty diesel engines will require NOx exhaust control technology.

Key Elements of the Regulation

The rule has several key elements:

- A sulfur cap of 15 ppm beginning June 1, 2006 for 80 percent of the diesel fuel sold by major refiners for use in highway vehicles, becoming 100 percent after December 31, 2009. (ULSD became available at retail outlets on October 15, 2006.) In addition, small refiners, who produce approximately five percent of the on-road diesel fuel, are eligible to sell diesel fuel with 500 ppm sulfur until 2010.
- 0.01 g/bhp-hr PM standard that takes effect with the 2007 model year for all on-road diesel HDEs
- 0.2 g/bhp-hr NOx standard, a 0.14 g/bhp-hr NMHC standard, and a 0.016 g/bhp-hr formaldehyde (HCHO) standard for HDEs to be phased in beginning with the 2007 model year (diesel HDEs -- 50% for MYs 2007-2009 and 100% beginning with the 2010 MY; gasoline HDEs – 100% in 2007)
- New requirements for crankcase emissions on turbocharged diesel engines
- Tighter standards for heavy-duty vehicles certified as complete vehicles
- Standards requiring reduced evaporative emissions

Emission Standards

The emission standards for HDEs are shown in Table 1 below. Under the final rule, 50% of the engines must meet the 0.2 NOx standard for model years 2007-2009.

Manufacturers who sell diesel HDEs meeting the 0.2 g/bhp-hr NOx and 0.01 g/bhp-hr PM standards before the 2007 MY deadline will receive a credit of 1.5 for each engine sold (i.e., for every two low emitting engines sold before 2007, a manufacture will receive a credit for three engines sold beginning in 2007).

Table 1. Full Useful Life Heavy-Duty Engine Emission Standards and Phase-Ins

Engine Type	Pollutant	Standard (g/bhp-hr)	2007	2008	2009	2010
Diesel	NOx	0.20	50%	50%	50%	100%
Diesel	NMHC	0.14	50%	50%	50%	100%
Diesel	HCHO	0.016	50%	50%	50%	100%
Gasoline	NOx	0.20	0%	50%	100%	100%
Gasoline	NMHC	0.14	0%	50%	100%	100%
Gasoline	HCHO	0.016	0%	50%	100%	100%
Diesel	PM	0.01	100%	100%	100%	100%
Gasoline	PM	0.01	0%	50%	100%	100%

For vehicles between 10,000 and 14,000 pounds, the standards are 0.4 g/mi for NOx, 0.02 g/mi for PM, and 0.230 g/mi for NMHC. EPA believes these standards are roughly comparable to the engine-based standards in these size ranges.

Note that these standards would not apply to vehicles above 8500 pounds that we classify as medium-duty passenger vehicles as part of our Tier 2 program because of their primary use as passenger vehicles (the final standards for these vehicles are in 65 FR 6698, February 10, 2000). The standards for HDVs are shown in Table 2.

Table 2. Emission Standards for HDVs

GVWR (lbs)	PM (g/mi)	NOx (g/mi)	NMHC (g/mi)
8,500-10,000	0.02	0.2	0.195
>10,000-14,000	0.02	0.4	0.230

EPA also revised the evaporative emissions standards for heavy-duty engines and vehicles, effective in the 2007 model year. The evaporative emission standards are shown in Table 3.

Table 3. Evaporative Emission Standards

GVWR (lbs)	3-Day Diurnal Test	Supplemental 2-Day Diurnal Test
8,500-14,000	1.4 g/test	1.75 g/test
>14,000	1.9 g/test	2.3 g/test

Diesel Sulfur Limits

EPA had originally proposed a 15 ppm sulfur cap for all diesel fuel sold for on-road vehicles beginning in 2006. To address concerns expressed by the U.S. Department of Energy regarding possible fuel shortages and price spikes, and in recognition of the special burden placed by the rule on small refiners, EPA in the final rule called for a phase-in of the 15 ppm low sulfur diesel fuel requirement. From June 1, 2006 to December 31, 2009, a minimum of 80% of the diesel fuel sold by large refiners (on a pad-by-pad) basis must meet the 15 ppm sulfur limit, with the remaining diesel fuel meeting a 500 ppm limit. Credit trading between refiners is allowed on a regional (i.e., pad-by-pad) basis. Small

refiners, who produce approximately five percent of the on-road diesel fuel, are given until 2010 to meet the 15 ppm sulfur limit.

Small refiners who elect to produce diesel fuel meeting the 15 ppm sulfur limit are also given the option of receiving an additional three years or until 2011 to meet the gasoline sulfur limit.

Less than 100 percent availability of 15 ppm sulfur diesel does raise the issues of a duel fuel distribution system and the possibility of fuel contamination, but EPA is confident these issues can be addressed. Also, EPA anticipates that the amount of low sulfur diesel fuel actually sold in the 2006-2009 timeframe will approach 90 percent.

Costs

EPA estimates that the proposed standards will add about $1200 to $1900 per new vehicle, depending on the vehicle size. To put these costs in perspective, a truck can costs as much as $150,000 and a bus can cost as much as $250,000.

The agency estimates that cutting sulfur levels from the current 500 ppm level to 15 ppm will add about 4-5 cents per gallon to produce and distribute diesel fuel, but there will be a cost off-set of 1 cent per gallon from vehicle maintenance savings resulting from the use of cleaner diesel fuel.

Air Quality Benefits

EPA estimates that the new standards will reduce smog-forming NOx emission by 2.6 million tons annually when the program is fully implemented in 2030. Emissions of PM will be reduced by 110,000 tons each year, hydrocarbons by 115,000 tons each year, and toxic air pollutants such as benzene by 17,000 tons annually. EPA estimates that the emission reduction benefits of the rule is equivalent to removing 13 million of today's trucks out of service.

For More Information

For more information on this rule, go to: **www.epa.gov/otaq/highway-diesel/index.htm**.

2200 Wilson Boulevard, Suite 310, Arlington, VA 22201 **202.296.4797**

Brake and Tire Wear Emissions from On-road Vehicles in MOVES2014

EPA United States Environmental Protection Agency

Brake and Tire Wear Emissions from On-road Vehicles in MOVES2014

Assessment and Standards Division
Office of Transportation and Air Quality
U.S. Environmental Protection Agency

NOTICE

This technical report does not necessarily represent final EPA decisions or positions. It is intended to present technical analysis of issues using data that are currently available. The purpose in the release of such reports is to facilitate the exchange of technical information and to inform the public of technical developments.

EPA United States Environmental Protection Agency

EPA-420-R-14-013
December 2014

Table of Contents

1 Introduction

The mobile source particulate matter inventory includes exhaust emissions and non-exhaust emissions. Exhaust emissions include particulate matter attributable to engine related processes such as fuel combustion, burnt oil, and other particles that exit the tailpipe. Non-exhaust processes include brake wear, tire wear, suspension or resuspension of road dust, and other sources. Particulate matter from brakes and tires is defined as the airborne portion of the "wear" that can be created by abrasion, corrosion, and turbulence. These wear processes can result in particles being suspended in the atmosphere. The size, chemical composition, and emission rate of particles arising from such sources contributes to atmospheric particle concentrations. However, these particles are composed of different species and size than exhaust particulate matter.[1]

The literature review for the development of the brake and tire wear emission models was conducted in 2006 and 2007, the models were developed for MOVES2010, and this report was written in 2008. However this documentation was not revised until the peer review complete in 2014 and no revisions to the model were made. As of 2007, the references in this report were recent, yet there were likely a few publications on particulate matter from brake and tire wear which were not included in the original literature review. In the sections below we present the studies from the literature conducted at the time, as well as the models that were developed based on the best data presented in the papers cited. A more recent literature search and a potential model update will be conducted in the future.

2 Brakewear

2.1 Literature Review

There are two main types of brakes used in conventional (or non-hybrid electric) vehicles: disc brakes and drum brakes. In a drum brake the components are housed in a round drum that rotates with the wheel. Inside the drum are shoes that, when the brake pedal is pressed, force the shoes against the drum and slow the wheel. By contrast, disc brakes use an external rotor and caliper to halt wheel movement. Within the caliper are brake pads on each side of the rotor that clamp together when the brake pedal is pressed.[2]

The definition of wear versus airborne PM seems to have slightly different definitions in the literature. In this paper it is generally the mass of material lost, whether in the brake pads or the tires. A fraction of that wear is airborne PM. Some studies look at both wear and airborne PM, others look at one or the other. In brakes, the composition of the brakeliner has an influence on the quantity and makeup of the released particles. Disc brakes are lined with brake pads while drum brakes use brake-shoes or friction linings. These materials differ in their rate of wear, their portion of wear particles that become airborne, and the size as well as composition of those particles. Both types of brakes use frictional processes to resist inertial vehicle motion. The action of braking results in wear and consequent release of a wide variety of materials (elemental, organic and inorganic compounds) into the environment.

The overall size or mass of the brake pads also varies with vehicle type. Typically trucks use larger brakes than passenger vehicles because the mass of vehicle that requires slowing down or stopping is greater. In 2004, most light duty vehicles used disc brakes in the front and drum

brakes in the rear. Disc brakes tend to have improved braking performance compared to drum brakes and have correspondingly higher cost. Disc brakes are sometimes used on rear wheels as well for higher performance (sportier) vehicles.

As a complicating issue, the particulate matter from brakes is dependent on the geometry of the brakes, wheels and rims. The air flow through the rims to cool the brakes and rotors play a key role in determining the wear characteristics. The emissions are also sensitive to driver activity patterns, where more aggressive stop and go driving will naturally cause greater wear and emissions.

There are only a very limited number of publications on brake wear PM emissions. There are even fewer publications discussing size distributions and speciation, and none quantifying emissions modally on which to directly base a model. This section summarizes the limited literature as of 2006. More details of the literature on brake and tire wear can be found in Appendix D. One of the earliest studies on brake wear emissions was done in 1983.[3] Particulate emissions from asbestos-based brakes from automobiles were measured under conditions simulating downtown city driving. The report presented a systematic approach to simulating brake applications and defining particulate emissions, and was used in the development of the EPA PART5 model.[4] For PART5, EPA calculated PM_{10} emission factors for light-duty gasoline vehicles of 12.5 mg/mi for brake wear. Since 1985, the asbestos in brakes has been replaced by other materials, and newer studies have been conducted. These factors suggest the need for this update of the emission factors applicable to more modern vehicles.

Garg et al. (2000) conducted a study in which a brake dynamometer was used to generate wear particles under four wear conditions (much of the background information provided in the previous paragraphs are from this paper).[5] The study was performed using seven brake pad formulations that were in high volume use in 1998. Measurements were taken on both front disc as well as rear drum brakes. The study measured mass, size distribution, elemental composition, as well as fiber concentration at four temperature intervals. The report also estimated $PM_{2.5}$ and PM_{10} emissions for light-duty vehicles of 3.4 and 4.6 mg/mile, respectively for small vehicles, and $PM_{2.5}$ and PM_{10} emissions of 8.9 and 12.1 mg/mile, respectively for pickup trucks.

Sanders et al (2003)[6] looked at three more current (as of ~2003) classes of lining materials: low metallic, semi-metallic and non-asbestos organic (NAO) representing about 90% of automotive brakes at that time. Three kinds of tests were conducted: a dynamometer test, a wind tunnel test and a track test at the Ford Dearborn proving grounds. Three sets of brake conditions were used: (a) the first set of tests evaluated all three materials on a brake dynamometer under mild and aggressive driving conditions, the urban driving program (UDP) with a set of 24 stops and a -7.9 m/s^2 deceleration called the Auto Motor und Sport magazine (AMS) test; (b) a series of high speed 1.8 m/s^2 stops of a mid-size sedan with low metallic brakes were conducted in a wind tunnel; and c) measurements of the same vehicle on a test track where collected where decelerations were made from 60 mph at 0.15, 0.25 and 0.35 g-forces, the latter corresponding to the AMS test to compare to the brake dynamometer. The latter test included low metallic as well as NAO materials. The authors found that the mean particle size and the shape of the mass distribution are very similar for each of the three linings, however they found that the low metallic linings generate 2-3 times the number of wear particles compared to semi-metallic and NAO linings. They also found that wear (and portion of wear that is airborne PM emissions) increased non-linearly with higher levels of deceleration. Wear debris composition was found to

have the most abundant elements consisting of Fe, Cu, Si, Ba, K and Ti, although the relative composition varied significantly by brake type. The authors further found that 50-70% of the total wear material was released in the form of airborne particles.

Table 2-1 contains the emission rates derived from the literature review conducted in support of MOVES2009. While there are emission rates presented from other papers, this paper largely relies on the Sanders et al. paper as it includes the widest array of materials currently in use, measurement techniques, and deceleration ranges in a scientifically designed study. It is the only paper from which modal rates can be derived. It is also the most recent of the papers listed and improves on the measurement methods introduced in its predecessors. The other papers results are provided as a source of comparison. Note that the range of rates from Sanders et al. (2003) largely covers the range presented in the other papers as well. When determining the rates below, the values from Garg et al. (2000), are also used.

Table 2-1 Non-Exhaust PM Emissions (per vehicle) from Mobile Sources Literature Values of emission factors from brake lining wear (largely cited in Luhana et al. (2004)'s literature review

Literature Source	Vehicle Type	$PM_{2.5}$ [mg/km]	PM_{10} [mg/km]
Luhana et al.(2004)	Light Duty		0-79
	Heavy Duty		0-610
Sanders et al. (2003)	Light Duty		1.5 -7.0
Abu- Allaban et al.(2003)	Light Duty	0 - 5	0-80
	Heavy Duty	0-15	0-610
Westurland (2001)	Light Duty		6.9
	Heavy Duty		41.2
Garg et al(2000)	Passenger Cars*	3.4	4.6
	Large Pickup Trucks	8.9	12.1
Rauterberg-Wulff (1999)	Passenger Cars		1.0
	Heavy Duty Vehicles		24.5
Carbotech (1999)	Light Duty		1.8-4.9
	Heavy Duty		3.5
Cha et al.(1983) used in PART5	Cars and Trucks		7.8

* In this table, "passenger cars" are equivalent to light duty cars. "Light Duty" on their own includes all Light-duty vehicles, including trucks though the studies are not all equivalent in their definitions.

2.2 Developing Rates for MOVES

2.2.1 Emissions during braking

The MOVES2009 braking emission rate is based on the average of:

(1) Composition of brake pad
(2) Number (and type) of brakes
(3) Front vs rear braking
(4) Airborne fraction

and explicitly accounts for:

(1) Particle mass size distribution ($PM_{2.5}$ vs PM_{10})
(2) Braking intensity
(3) Vehicle class: Light-Duty vs Heavy-Duty

As discussed in Sanders et al. (2003), most brake pads (at the time of the publication of that paper) are either low-metallic, semi-metallic (full-truck), or non-asbestos organic (full-size car). Using the results from this study, we make the following assumptions which are consistent with those used in the paper.

- equal mix of the three brake types,
- four brakes per light duty vehicle, including 2 front disc brakes, and 2 rear drum brakes
- 2/3 of braking power (and thus emissions) in front brakes (1/3 rear)[a]
- the fraction of total PM below 2.5um is ~ 10% (+/-5%)[b]
- 60% of brake wear is airborne PM (+/- 10%).

We also do not compensate for the different average weights of the vehicles (though the MOVES VSP bins scale emissions with mass). We assume there is an equal mix of the three brake types because the market share penetration is not known.

For each test cycle from Sanders et al. (2003) and Garg et al. (2000), the following figures show how we went from the measured results to emission rates of g/hour (for deceleration times only) at various deceleration speeds. Sanders et al. (2003) used three measurement techniques, a filter, an Electrical Low Pressure Impactor (ELPI), and a Micro-Orifice Uniform Deposition Impactor (MOUDI). While all three measurement techniques produced similar results, we show all here. Test results are shown for the UDP and wind tunnel tests from Sanders et al. (2003), as well as the Garg et al. (2000) analysis. The latter paper adds another deceleration point for comparison. The AMS results are not presented in the Sanders paper, however, the authors provided the data for the purposes of this study.

[a] Based on discussions with author of paper Matti Mariq at Ford Motor Company and consistent with the Garg et al. (2000) paper, who used 70%. Some of the other assumptions in this list is also from these discussions

[b] More will be discussed below.

Table 2-2 – UDP results[c]

Test	brake lining	PM_{10} emiss. (mg/stop/brake) filter	ELPI	
UDP				
	low metallic	6.9[d]	7.0	
	semi-metallic	1.7	1.7	
	Non-asbestos	1.1	1.5	
Average/stop/brake		3.2	3.4	
Avg. /veh		9.7	10.2	
deceleration =			**0.0012**	**km/s^2**
avg. brake time in secs =			13.5	secs
avg. emissions in mg/stop =			9.95	Mg/stop
emission rate for the UDP test =			**2.65**	**g/hr**

Table 2-3 – Wind Tunnel results

Test	brake lining	PM_{10} emiss. (mg/stop/brake) filter*	ELPI	MOUDI	
Tunnel					
	low metallic	44	45	40	
deceleration=			**0.0018**	**in km/s^2**	
Initial Velocity V(0) =			0.0267	in km/s	
avg. brake time in sec =V(0)/dec			14.8	secs	
avg. emissions in mg/stop =			129.0	mg/stop	
emision rate for the wind tunnel test=			**31.4**	**g/hr**	

[c] As these are intermediate values, the number of significant digits may exceed the precision known, however they are kept in this presentation, and rounded for the final results. The UDP decelerations are the average decelerations from those measured in the Sanders paper. The average brake times were determined with the assistance of one of the original authors of the paper (Matti Mariq) who supplied the second by second trace. The filter PM10 were determined by multiplying the total PM reported in Table 5 of the paper with the PM10 to total PM ratio determined from the ELPI measurement.

[d] Sanders et al, reports the total filter PM to be 8.2 mg/brake/stop. In order to get PM10 equivalent, we applied the ELPI ratio from table 5 in the reference. So 6.9 = 8.2* (7/8.3). The other numbers were calculated in a similar fashion. Also, the avg per vehicle emissions is the avg stop/veh/brake emissions multiplied by 3. This is based on the assumption made earlier that 2/3 of braking comes from the front brakes and 1/3 from the rear brakes.

Table 2-4 – AMS results

Test	brake lining	PM_{10} emiss.	(mg/stop/brake)
AMS		filter	ELPI
	low metallic	800	70
	semi-metallic	510	63
	Non-asbestos	550	92
	Average=	620	75
	Avg/veh rate =	1116	135

deceleration =	0.0079	in km/s^2
Initial Velocity V(0) =	0.0278	in km/s
avg. break time in sec =V(0)/dec	3.5	secs
avg. emissions in mg/stop for PM_{10}=	1116	mg/stop
emision rate for PM_{10} for the AMS test=	**1143**	**g/hr**
avg. emissions in mg/stop for $PM_{2.5}$=	135.0	mg/stop
emision rate for $PM_{2.5}$ for the AMS test=	**138.2**	**g/hr**

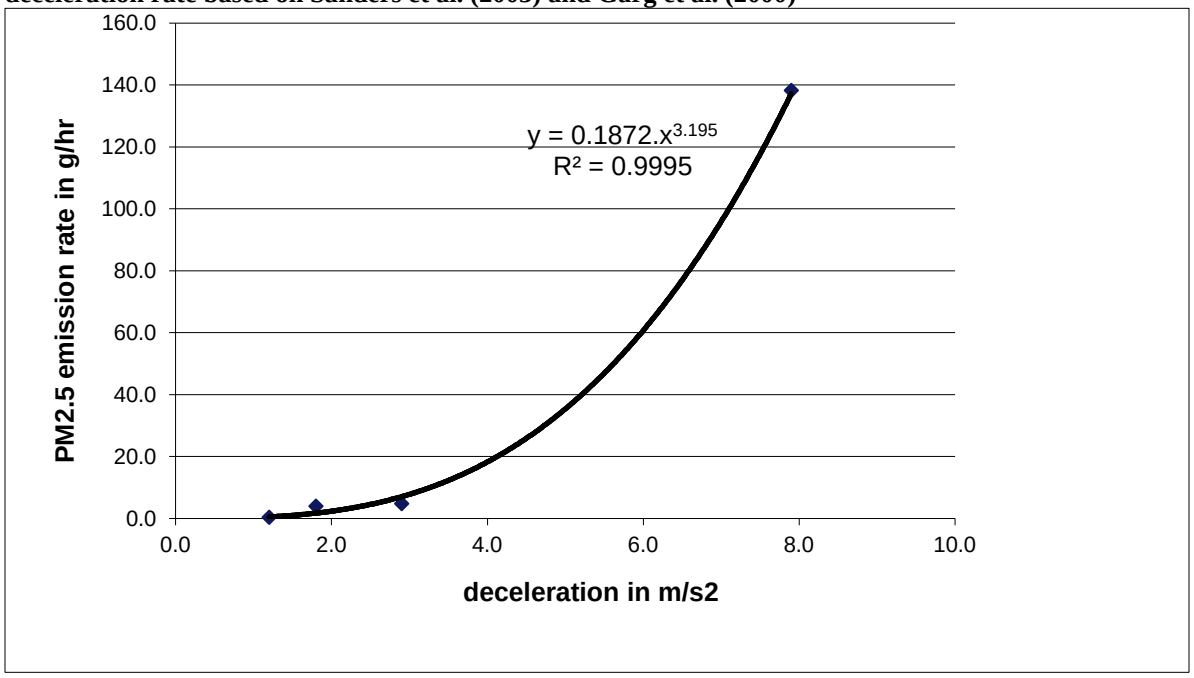
Table 2-5 – Garg et al. (2000) results

Test	brake lining	PM_{10} emiss.*	$PM_{2.5}$**	(mg/stop/brake)
avg. over all temp.	semi-metallic #1	1.85	1.35	
	semi-metallic #5	0.82	0.60	
	NAOS #2	2.14	1.57	
	NAOS #3	0.89	0.66	
	NAOS#7	1.41	1.03	
	Grand Avg. =	1.42	1.04	mg/stop

deceleration =	0.00294	in km/s^2
Initial Velocity V(0) =	0.0139	in km/s
avg. break time in sec =V(0)/dec	4.7	secs
avg. emissions in mg/stop for PM_{10} =	1.42	mg/stop
emision rate for PM_{10} for the GM test=	**1.08**	**g/hr**
avg. emissions in mg/stop for $PM_{2.5}$ =	1.04	mg/stop
emision rate for $PM_{2.5}$ for the test=	**0.79**	g/hr

We used these four data points to fit a power function to determine the emission rate at different deceleration levels shown in the following figure. The AMS test, at higher decelerations, clearly has a significant influence on results of the curve fit. Additional high speed tests could be used for future refinement of this data.

Figure 2-1- Brake wear $PM_{2.5}$ emission rates in units of grams per hour for light duty vehicles as a function of deceleration rate based on Sanders et al. (2003) and Garg et al. (2000)



2.2.2 Activity

In the previous section, we determined the rate of particulate matter emissions during braking in units of grams per hour (per vehicle) as a function of deceleration level for a light-duty vehicle. However, for MOVES, we also need to determine the frequency of different levels of braking. The MOVES vehicle specific power (VSP) bins are relatively coarse for braking.[e,7] There is a large braking bin (operating mode 0) that contains a large fraction of driving activity, however there are also a number of "coasting" bins that also contain braking events in each speed category (Table 2-6). Each of these deceleration operating modes include some braking as well as cruise and coasting operation (where the throttle is closed or nearly closed, but the brakes are not applied). Therefore, the emission rate assigned to these bins need to contain the appropriate average rates including the mix of driving and deceleration frequencies, and including decelerations that do not include braking.

[e] While this document does not provide a detailed discussion of vehicle specific power, the light duty emission rate report have an extensive discussion

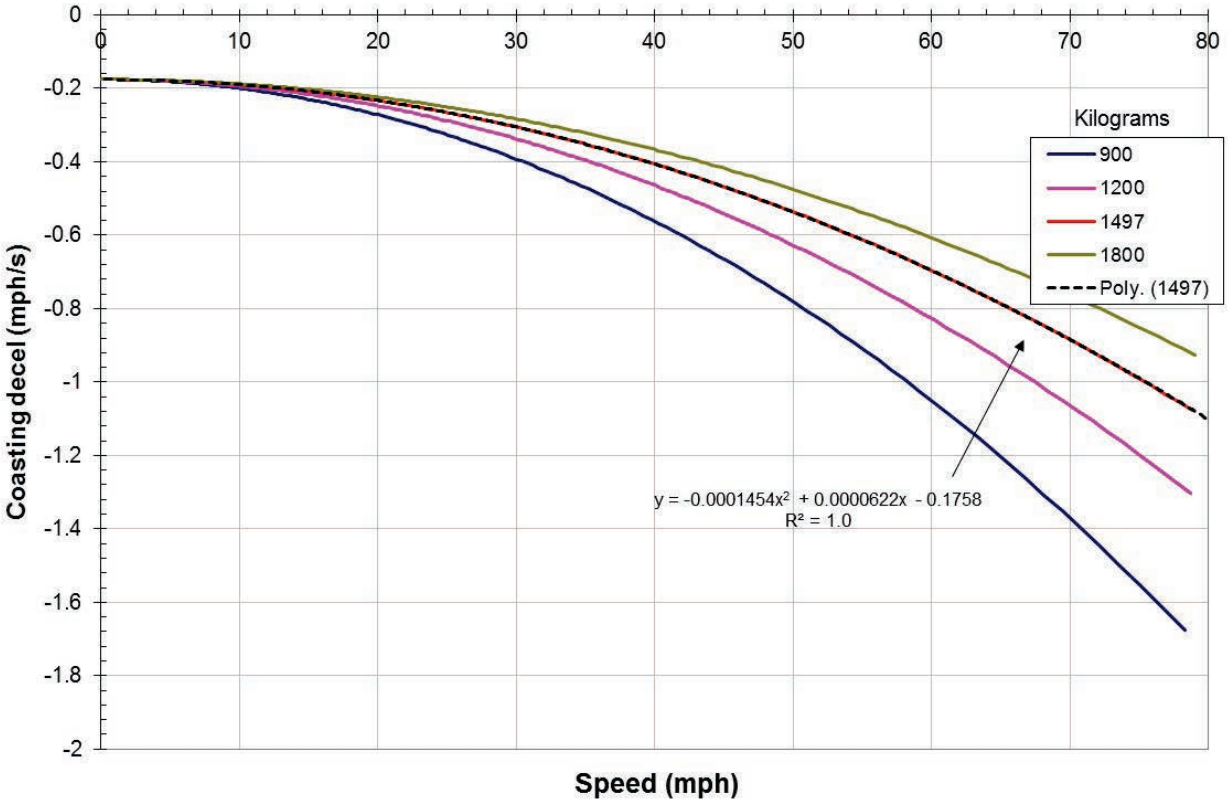
Table 2-6. VSP Operating Mode Bins by VSP and speed. Operating mode 0 and 1 (not listed) are braking and idle respectively

VSP Class (kW/tonne)	Speed Class (MPH)		
	1-25	25-50	50+
30+	16	30	40
27-30		29	39
24-27			
21-24		28	38
18-21			
15-18		27	37
12-15			
9-12	15	25	35
6-9	14	24	
3-6	13	23	33
0-3	12	22	
<0	11	21	
Operating mode where braking is assumed			

We estimated the fraction of activity that is braking within each of the "coasting" bins by first determining the coast down curve, then combining that with the activity fraction as seen in real-world driving surveys.

The coastdown curves were generated using the Physical Emission Rate Estimator (PERE).[8] This was done by using the coastdown equations from PERE, and calculating the deceleration at each speed when the forward tractive power is zero. We assumed all activity below coastdown is braking and all activity above the curve is low throttle deceleration. Figure 2-2 shows coastdown curves for cars of a variety of weights (and coastdown coefficients). The dotted curve is a typical coast down curve for this class of vehicle, where 1,497 kg is the typical mass of a light duty vehicle. The average weight for passenger cars used in MOVES is 1,497 kg.

Figure 2-2- Modeled Coastdown curves using the PERE model for a variety of light-duty vehicles masses



The deceleration activity was determined from two real world instrumented vehicle studies: one from Kansas City and the other in Los Angeles. The Kansas City study was conducted by EPA and Eastern Research Group (ERG) in 2005 to study real world driving activity and fuel economy on conventional as well as hybrid electric vehicles.[9] Over 200 vehicles were recruited, though for the current analysis, only the activity data from the conventional, or non-hybrid, population were examined. The Los Angeles activity data was conducted by Sierra Research for the California Department of Transportation with both instrumented vehicles as well as chase car data[10,11,12]. The deceleration data was analyzed for both of these studies.

Table 2-7 shows the distribution of braking activity across deceleration levels from both of these studies. As expected, the vast majority of braking occurs during mild decelerations rather than full (high decel) stops. More information about the PERE coastdown calculation process is described in Appendix A.

Table 2-7 – Activity Distribution of braking activity in the LA and Kansas City studies for each deceleration bin.

Decel (mph/s)	LA urban	LA rural	KC	AVG
1	37.1%	27.1%	54.5%	39.5%
2	26.3%	27.9%	26.3%	26.9%
3	17.9%	20.2%	12.8%	17.0%
4	10.2%	12.2%	4.6%	9.0%
5	5.6%	8.2%	1.3%	5.0%
6	1.6%	2.4%	0.30%	1.4%
7	0.64%	0.98%	0.07%	0.6%
8	0.28%	0.41%	0.02%	0.2%
9	0.17%	0.26%	0.02%	0.2%
10	0.10%	0.13%	0.01%	0.08%
11	0.05%	0.09%	0.01%	0.05%
12	0.03%	0.05%	0%	0.03%
13	0.01%	0.01%	0%	0.01%
14	0%	0.01%	0%	0%

2.2.3 Emission Rate for Light Duty vehicles

The emission rate curve from Figure 2-1 was combined with the average activity in Table 2-7 discussed above (using a sum of the product) to calculate MOVES rates for light duty vehicles. This gives an average $PM_{2.5}$ braking emission rate of 0.557 g/hr.

However, as mentioned earlier, MOVES has brake emissions in not only VSP op-mode bin 0 (defined as the braking bin), but also in modes 1,11,21,33. Idle (zero speed, op-mode bin 0) braking occurs in the transition (deceleration) from non-zero speed to zero speed which is a small amount of activity in this bin. Bins 12 and 22 also contain a very small amount of braking, which are ignored – i.e, the rates in these bins are set to zero. The brake emission rate in the other bins were reduced by the amount of braking activity in each bin.[f] These braking fractions were derived by combining the amount of average activity from Kansas City and LA above and the coast down curves from PERE discussed earlier. The results are shown in Table 2-8 below.

[f] For example, the $PM_{2.5}$ emission rate in VSP bin 11 for light-duty vehicles is 0.557 * 0.978 = 0.546 g/hr

Table 2-8 – Vehicle Specification (top) and Fraction of Activity in VSP bin that is braking (last 5 rows) for a variety of vehicle types (motorcycle and bus activity fractions were copied from Light-duty and heavy-duty trucks respectively).

	Mid-size car (LDV)	SUV (LDT)	LHDT (<=14k)	LHDT (>14k)	MHDT	HHDT
wgt (kg)	1497	1800	5602	9333	13517	22680
Cr0 (rolling resistance)	0.008	0.008	0.008	0.008	0.01	0.01
Cd (drag coeff)	0.32	0.36	0.37	0.44	0.44	0.44
A (frontal area m^2)	2.25	2.5	2.75	6.7	6.7	8.64
vsp bin						
0	1	1	1	1	1	1
1	0.0437	0.0437	0.0316	0.0316	0.0316	0.016
11	0.975	0.975	0.913	0.906	0.91	1
21	0.641	0.661	0.743	0.685	0.725	0.641
33	0.115	0.122	0.126	0.116	0.121	0.068

2.2.4 $PM_{10}/PM_{2.5}$ Brake Wear Ratio

MOVES stores $PM_{2.5}$ brake wear emission rates by operating mode bin, then estimates PM_{10} emission rates by applying a $PM_{10}/PM_{2.5}$ ratio. The $PM_{10}/PM_{2.5}$ ratio is based on the assumptions that the mass fraction of particles below PM_{10} is 0.8, and the mass fraction of particles below $PM_{2.5}$ is 0.1. More specifically, Sanders et al. (2003), report PM "fractions and cutoffs of 0.8 at 10 µm, 0.6 at 7 µm, 0.35 at 4.7 µm, 0.02 at 1.1 µm, and <0.01 at 0.43 µm for the UDP stops typical of urban driving". These assumptions result in a $PM_{10}/PM_{2.5}$ ratio of 8. Where no $PM_{2.5}$ values were reported, we calculated $PM_{2.5}$ from PM_{10} emission rates using this fraction. This estimate widely varies in the literature. Abu- Allaban et al. (2003) reports that only 5-17% of PM_{10} is $PM_{2.5}$, which is consistent with Sanders. Garg et al. (2000), report 72% of PM_{10} is $PM_{2.5}$, which is disputed by Sanders et al. (2003). The current study does use the $PM_{2.5}$ measurement reported by Garg et al. (2000), however in reality, this single value has little impact on the curve fit in Figure 2-1, which is dominated by the more recent data from Sanders et al. (2003).

The emission rates in g/hr $PM_{2.5}$ and PM_{10} by operating mode and regulatory class are included in Appendix B. The rates are calculated per the methodology described above and is independent of model year and environmental conditions. The average $PM_{2.5}$ and PM_{10} brake wear emission rates for passenger cars and trucks from three urban county inventories, using MOVES2014 are displayed in Table 2-9. MOVES brake wear emission rates by source type will vary according to the inputs of average speed, and VMT by road type, which impacts the distribution of operating modes within each source type in MOVES.

Table 2-9 Average PM2.5 and PM10 brake wear emission rates (mg/mile) for passenger cars and trucks from 3 urban county inventories using MOVES2014

	$PM_{2.5}$	PM_{10}
Passenger Cars (21)	3.7	29.8
Passenger Trucks (31)	6.2	49.8

The average passenger car MOVES PM_{10} emission rates of 29.8 mg/mi (output from the model) is compared to the previous studies (in the literature) in Table 2-1. Carbotech (1999), Sanders et al. (2003) , Garg et al. (2000), are all laboratory measurements and have significantly smaller reported emission rates than the present study. On the other hand Luhana et al. (2004), Abu-Allaban et al. (2003), Westurland (2001), and Rauteberg-Wulff (1999) are roadside measurement or tunnel measurements. These studies generally have higher emissions than laboratory measurements. The MOVES rates largely generated from Sanders et al. (2003), are also considerably larger than the publication cites. This is largely due to the fact that Sanders et al. (2003), cites results primarily from the UDP braking events which are significantly milder than the AMS decelerations. Through the modeling described in this paper, the AMS deceleration rates are weighted in to the milder deceleration emission rates to give higher rates comparable now to some of the results achieved from the tunnel and roadside studies. The light duty rates are thus calibrated to laboratory measurements adjusted to real-world factors, and "validated" to be within the range of roadside and tunnel measurements.

2.2.5 Brake Wear Emissions for Heavy-Duty Vehicles and Other Vehicle Types

There is very little literature on direct heavy-duty brake emissions measurements. To decelerate, heavy-duty vehicles employ technologies such as disc and drum as well as other braking methods including downshifting and engine (or "jake") braking. A scientific study comparing the emissions and relative activity of each of these methods of braking is beyond the scope of this report. In order to estimate brake wear emission factors for heavy-duty vehicles an engineering analysis was combined with results from a top-down study performed by Mahmoud Abu-Allaban et al. (2003).[13] The authors collected particulate matter on filters near roadways and apportioned them to sources utilizing Chemical Mass Balance, CMB, receptor modeling along with Scanning Electron Microscopy. The study was performed at roadside locations in Reno, Nevada and Durham, North Carolina where intensive mass and chemical measurements were taken. The authors of the paper attempted to collect and differentiate between PM measurements from tailpipe, tire, road dust, and brake from light- and heavy-duty vehicle types. Compared to the other papers described in the previous section (on light-duty braking) that include heavy-duty rates, the Abu-Allaban paper is one of the most recent studies of its kind performed at the time of the writing of this paper. The results are consistent with the heavy-duty rates measured from Luhana et al. (2004) as well as Westurland (2001), but is the only paper to measure $PM_{2.5}$. The paper's light-duty rates are also aligned with the rates determined above.

In this study, $PM_{2.5}$ brake wear emission rates for heavy duty vehicles ranged from 0 to 15 mg/km (0 to 24 mg/mi). For this analysis we have assumed the emission rate was the midpoint of the range of emission factors, or 12 mg/mi. For the purposes of populating MOVES rates, we

do not employ the measured emission rate directly from this study due to the extreme uncertainty and variability of measurement and locations selected. Rather, we rely on the paper's comparison of light-duty to heavy-duty emission factors. On table 5 of the paper, the emission rates for the exit ramps are reproduced below. Only the exit lanes were included of the many roads where measurements were collected. The remainder of the roads are represented by the average and the (min to max) range reported in the table.

Table 2-10 Brake Wear Emission Rates reproduced from Abu-Allaban et al. (2003)

Location	Vehicle Type	PM_{10} (mg/km)	$PM_{2.5}$ (mg/km)
J. Motley Exit	Heavy-Duty	610 ± 170	0 ± 0
	Light-Duty	79 ± 23	0 ± 0
Moana Lane Exit	Heavy-Duty	120 ± 33	0 ± 0
	Light-Duty	10 ± 3	0 ± 0
Average over all roads	Heavy-Duty	124 ± 71	2 ± 2
	Light-Duty	12 ± 8	1 ± 0
Range (min to max) of measurements on all roads	Heavy-Duty	0 to 610	0 to 15
	Light-Duty	0 to 80	0 to 5

Due to the difficulty of differentiating a small brake emissions signal from the much larger signal coming from tailpipe, tire wear and road dust combined, there is much uncertainty in these measurements – yet another reason why adjusted laboratory measurements were favored above. Clearly $PM_{2.5}$ was difficult to measure from most sites. Interestingly, the heavy-duty measurements were highest on the exit lanes for PM_{10}, however (rather inexplicably), the other road types had higher emissions than the exit lanes for $PM_{2.5}$. For these reasons, we rely more on averages to determine our ratio of heavy-duty to light-duty brake emission factors. From these measurements, we can determine that the average ratio of HD to LD brake emissions is 10 and 2 for PM_{10} and $PM_{2.5}$ respectively.[g] On average, based on Table 2-10, the ratio is 7.6 for PM10. The following table compares the ratio for the remaining studies for comparison.

Table 2-11- Ratio of Heavy-Duty to Light-Duty PM from the literature.

Study	**$PM_{2.5}$**	**PM10**
Luhana et al. (2004)		7.7
Abu-Allaban et al. (2003)	3	7.6
Westurland (2001)		6.0
Rauterburg-Wulff (1999)		24.5
Carbotech (1999)		0.7

For the purposes of MOVES, a simpler model requiring a single ratio of HD to LD brake emissions and another ratio of PM_{10} to $PM_{2.5}$ brake emissions is attractive – particularly since the data to populate the model is sparse. Also the broad range of uncertainties in the literature can support such simplification. Based on the range in the table, above, the value of the ratio chosen is 7.5, very close to the ratio as measured by Abu-Alaban et al. (2003), and consistent with the range of studies.

[g] Though it is not shown in the table here, according to Abu-Alaban, based on the highest sampling sites (maximum measurements from the table), the ratio of HD to LD brake emissions is 41 and 16 for PM_{10} and $PM_{2.5}$ respectively.

The estimated emission factors for all other categories of vehicles (between light and heavy-duty) were derived by linearly interpolating the rates between light-duty and combination heavy-duty vehicle classes by their respective weights as shown in the figure below. This is based on a rather simple engineering (and unproven in this study) hypothesis that the relative brake emissions is proportional to the weight of the vehicle classes relative to (and bounded by) light and heavy-duty vehicles. The hypothesis is based on the assumption that relative mass of the vehicles is proportional to the relative energy required to stop the vehicles. Figure 2-3 below shows the relative mass of light- and heavy-duty vehicles. The corresponding emission rates are in Table 2-12.

Figure 2-3 – Interpolated Brake $PM_{2.5}$ Emission Rates by Regulatory Class Weight. Passenger Cars and Combination Heavy duty Trucks Define the Slope.

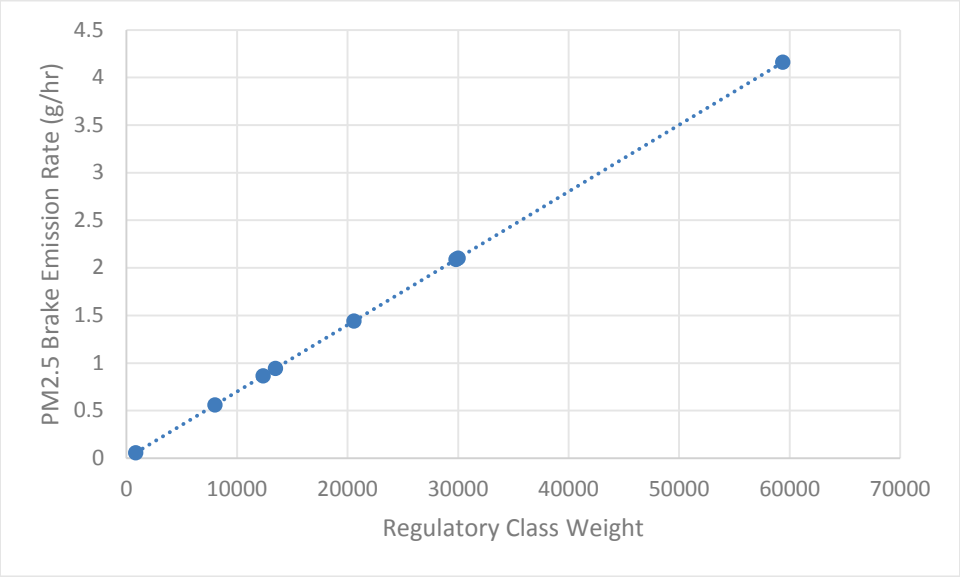


Table 2-12contains average brakewear PM emission rates from three MOVES runs for urban counties using MOVES2014, for each source types. As mentioned earlier, average emission rates by source type will vary for local users according to inputs that impact the operating mode distribution of vehicle operation

Table 2-12. Scaling Emission Rates to their vehicle class. regclass id is the MOVES identifier for regulatory class.

	regclasswt in lbs.	regclassid	g/hr
MC	830	10	0.056
LDGV	8000	20	0.56
LDT	13,474	30	0.94
HD<=14k	12,358	41	0.87
HD>14k	20,575	42	1.4
MHDD	29,808	46	2.1
HHDD	59,369	47	4.2
Urban Bus	30,000	48	2.1

Table 2-13 Average PM2.5 and PM10 brake wear PM emission rates (mg/veh-mile) for the MOVES regulatory classes from three urban county inventories using MOVES2014

sourceTypeID	Source Type	$PM_{2.5}$	PM_{10}
11	Motorcycle	0.3	2.6
21	Passenger Car	3.7	29.8
31	Passenger Truck	6.2	49.8
32	Light Commercial Truck	6.2	49.8
41	Intercity Bus	23.3	186.4
42	Transit Bus	12.6	100.9
43	School Bus	13.2	105.3
51	Refuse Truck	25.6	204.4
52	Single Unit Short-haul Truck	13.6	109.2
53	Single Unit Long-haul Truck	16.6	132.4
54	Motor Home	11.5	92.2
61	Combination Short-haul Truck	24.3	194.0
62	Combination Long-haul Truck	26.6	213.0

3 Tirewear

3.1 Introduction

Tires are an essential part of any vehicle and the number and size of tires increase with the size of the vehicle. Contact between tires and the road surface causes the tires to wear, with the rate dependent on a variety of factors.

EPA's previous estimates of tire wear are contained in the PART5 model, and are emission rates of 0.002 grams per mile per wheel. Two LDV studies from the 1970s are the basis for these emission rates. The PART5 emissions factors are based on tests of older bias-ply tires rather than more modern radial tire technologies The National Resource Council report on the MOBILE model, suggested that the PART5 rates may be out of date.[14]

Tire wear occurs through frictional contact between the tire and the road surface. Friction causes small and larger particles to wear from tire, which are then either released as airborne particulates, deposited onto the road surface or retained in the wheel hub temporarily or permanently until washed off. The road surface causes friction and abrasion and therefore the roughness of the surface affects the wear rate by a factor of 2-3.[15]

In addition to road surface roughness, tires wear is dependent upon a combination of activity factors such as route and style of driving, and seasonal influences. Heavy braking and accelerating (including turning and road grade) especially increases tire wear. The route and style of driving determine the amount of acceleration. Highway geometry is a key factor with rise and fall in roads also resulting in increased tread wear. The acceleration of the vehicle determines the forces applied to the tire, and includes turning. Tire wear due to tire/road interface is determined by and is directly proportional to these forces.[16] The season results in temperature, humidity and water contact variations. Wear rates are lower in wet compared to dry conditions.

Finally vehicle characteristics also influence tire wear. Key factors are the weight, suspension, steering geometry, and tire material and design. Axle geometry changes result in uneven wear across the tire width. The type of tire influences the wear significantly. In particular, the physical characteristics like the shape of the tire (determined by stiffness), the rubber volume (tread pattern), and the characteristic of the tire (rubber type etc.). As a consequence of different manufacturing specifications, different brands of tires wear at different rates. Retreads are also considered to wear more than new tires. Wear rate studies on tire fleets reported in Bennett & Greenwood (2001) also indicated that retreads had only about 75% of the tire tread volume that new tires had. Cenek et al. (1993) reported that 20% of New Zealand passenger tire sales were retreads and that retreads made up 75% of the tire tread in a sample of buses in the New Zealand fleet.[17] However, modeling emissions from retreads was deemed beyond the scope of the report

According to the literature, the most straightforward method for determining tire wear is the periodic measurement of tread depth. However, variations in the extent of wear across the tire and irregularities in tire shape could lead to inaccurate measurements. Determining tire weight loss is a more sensitive approach than the measurement of tire depth, though care must be taken to avoid errors due to damage to tires as a result of their removal from the vehicle and hubs, and material embedded in the tire. To minimize damage to the tire, Lowne (1970) weighed both the wheel and tire simultaneously after the wheel was brushed and stones embedded in the tire were

removed.[18] Table 3-1shows a summary of the literature search conducted as of 2006 on the mass of tire wear.

Wear rates for tires have typically been calculated based on tire lifetime (in kilometers traveled), initial weight and tread surface depth. Tire wear occurs constantly for moving vehicles, but may be significantly higher for cars which tend to brake suddenly or accelerate rapidly. Tire wear rates have been found to vary significantly between a wide range of studies.[19]

Speed variation is an important factor as well. Carpenter & Cenek (1999) have shown that the effect of speed variation is highest at low speeds as a result of inertial effects and effective mass.[20] They also examined lateral force effects on tires and assessed tire wear on routes of different amounts of horizontal curvature and found that there was little variation.

Tire abrasion is difficult to simulate in the laboratory, since the varied nature of the road and driving conditions influence wear rates in urban environments. Hildemann et al. (1991) determined the chemical composition of tire wear particles using a rolling resistance testing machine at a tire testing laboratory over a period of several days.[21] Rauterberg-Wulff (1999) determined particle emission factors for tire wear using modeling in combination with measurements conducted in the Berlin-Tegel tunnel.[22]

Tire wear rates have been measured and estimated for a range of vehicles from passenger cars to light and heavy duty trucks with results reported either as emission per tire or per vehicle. Most of the studies report only wear, not airborne PM. The wear rates found in the literature are summarized in Table 3-1 below and are converted to a per vehicle rate (units are in per vehicle kilometer). A range of light-duty tire wear rates from 64-360 mg/vehicle/km has been reported in the literature. Much of the variability in these wear rates can probably be explained by the factors mentioned above. These studies made no distinction between front and rear tires, even though they can wear at different rates.[23]

Table 3-1 - Tire wear rates found in the literature. Rates are per vehicle. Estimated number of tires is described later.

Source	Remarks	rate in mg/vkm
Kupiainen,K.J. et al(2005)[24]	Measured tire wear rate	9 mg/km - PM_{10} 2 mg/km -$PM_{2.5}$
Luhana et al (2003)	Measured tire wear rate	74
Councell,T.B. et al (2004) U.S. Geological Survey[25]	Calculated rate based on literature	200
Warner et al. (2002)[26]	Average tire wear for a vehicle	97
Kolioussis and Pouftis (2000)[27]	Average estimated tire wear	40
EMPA (2000)[28]	Light duty vehicle tire wear rate	53
	Heavy duty vehicle tire wear rate	798
SENCO (Sustainable Environment Consultants Ltd.) (1999)[29]	Light duty vehicle tire wear rate	53
	Wear rate for trucks	1403
	Estimated rate for light duty vehicles	68
Legret and Pagotto (1999a)	Estimated rate for heavy vehicles (>3.5t)	136
Baumann (1997)[30]	Passenger car tire wear rate	80
	Heavy duty vehicle tire wear rate	189
	Articulated lorry tire wear rate	234
	Bus tire wear rate	192
Garben (1997)[31]	Passenger car tire wear rate	64
	Light duty vehicle tire wear rate	112
	Heavy duty vehicle tire wear rate	768
	Motorbike tire wear rate	32
Gebbe (1997)[32]	Passenger car tire wear rate	53
	Light duty vehicle tire wear rate	110
	Heavy duty vehicle tire wear rate	539
	Motorbike tire wear rate	26.4
Lee et al (1997)[33]	Estimated tire wear rate	64
Sakai,H (1995)	Measured tire wear rate	184
Baekken (1993)[34]	Estimated tire wear rate	200
CARB (1993)	Passenger car tire wear rate	120
Muschack (1990)	Estimated tire wear rate	120
Schuring and Clark (1988)[35]	Estimated tire wear rate	240-360
Pierce,R.N. (1984)	Estimated tire wear rate	120
Malmqvist (1983)[36]	Estimated tire wear rate	120
Gottle (1979)[37]	Estimated tire wear rate	120
Cadle et al. (1978)[38]	Measured tire wear rate	4
Dannis (1974)[39]		90

While there is significant literature on tear wear, there is relatively little published on airborne particulate matter from tires. In this report, a model for tire wear rates are first determined, and then a discussion of the modeling of airborne $PM_{2.5}$ and PM_{10} follows building off the wear model.

3.2 Methodology

This report begins by estimating the tire wear from light-duty vehicles, then based on the per tire wear, extrapolates to other vehicle types. Then the emission rates are derived from the wear rates. The method primarily depends on the data from work published by Luhana et al. (2004) wherein wear loss rates for tires have been determined gravimetrically for in-service cars.[40] At the time of this analysis, this paper was both a recent and comprehensive study. The authors weighed car tires at two-month intervals, and asked drivers to note the details of each trip undertaken. Five test vehicles (labeled A-E) were selected for the tests. Of these vehicles A (1998 Audi A3), B (1994 Ford Mondeo), C (1990 Peugeot 205) and E (1992 Vauxhall Cavalier) were front-wheel drive vehicles (FWD). According to the driver surveys, the predominant road type used by vehicles A and B were motorways, for vehicle D (1990 Ford Sierra) it was rural roads and motorways for vehicle C it was suburban roads, and for vehicle E, it was rural roads. Vehicle D was excluded from this study since it was a rear-wheel drive (RWD) vehicle. RWD vehicles are relatively uncommon amongst passenger vehicles in the United States, and the wear from this particular vehicle was more than double the other FWD vehicles. It is uncertain whether the discrepancy from this vehicle was because it was a rear-wheel drive or for some other reason. The selection of vehicles was based primarily on driving conditions, as defined by the main type of road used by the owner and annual distance driven.

Results from the Luhana et al. (2004) study indicated that the lowest tire wear rates (56 mg/vkm and 67 mg/vkm respectively[h]) were for vehicles A and B that were driven predominantly on motorways. Vehicles C and E had very similar wear rates (around 85 mg/vkm) although these vehicles tended to be driven on different roads. Based on the wear rates from the four front-wheel drive cars alone, the study concluded that the average wear rate is around 74 mg/vkm. This value seems to lean towards the lower end of the range of wear rates reported in the literature.

The data presented in Table 3-2 includes calculations for the distances completed by each vehicle between successive tests, the estimated average trip speeds and predominant road types for the equivalent periods. It was assumed that the weight of the wheels remained constant during the tests, and any weight loss was due solely to the loss of tire rubber during driving.

[h] vkm is “vehicle kilometer” and assumes four times a per tire rate for light-duty vehicles.

Table 3-2: Data from Luhana et al. (2004) with measurements of tire wear for a variety of trips

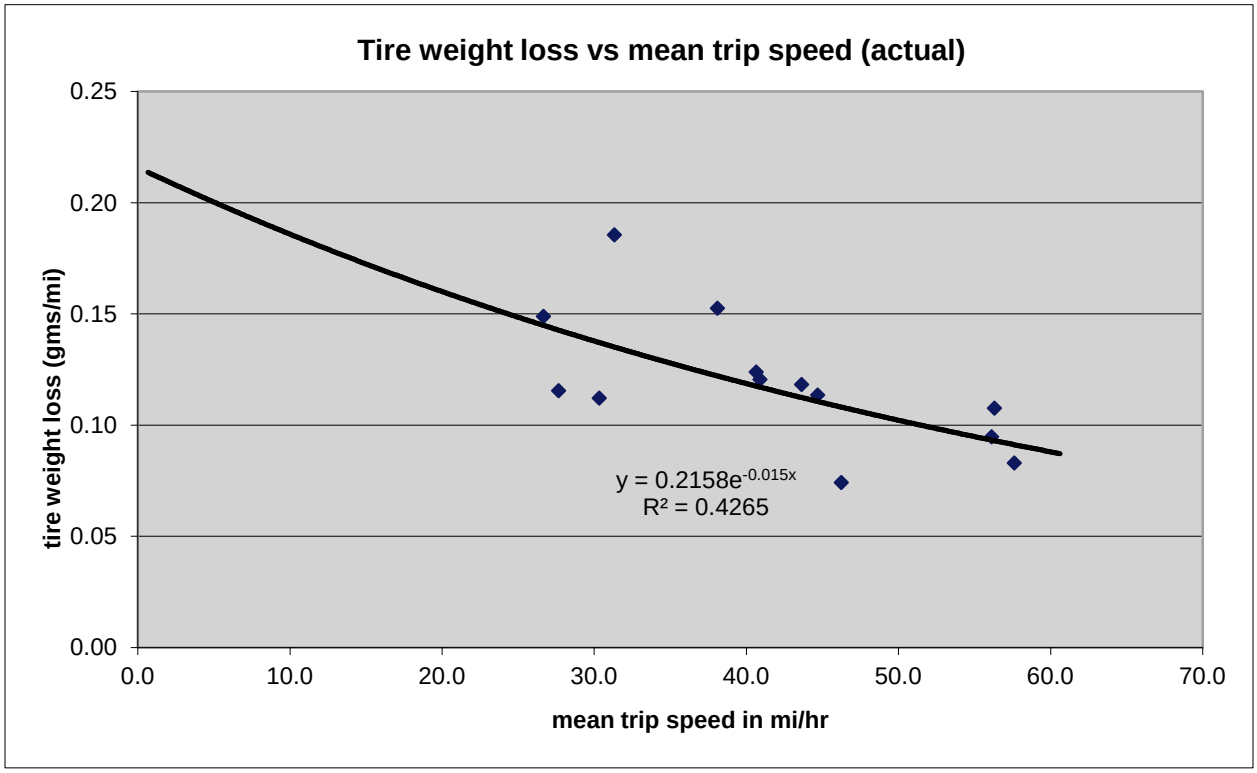
	Avg. trip speed	Tire Wt. Loss (per axle)		total wt. loss (per vehicle)	total wt. loss (per vehicle)	avg. speed
vehicle tests	km/hr	Front mean (g/km)	Rear Mean (g/km)	g/km	g/mi	mi/hr
test1-A	90.3	0.0202	0.0092	0.0589	0.0947	56.1
test2-A	90.6	0.0209	0.0126	0.0669	0.1076	56.3
test3-A	93.9	-	0.0069	-	-	58.4
test4-A	92.7	0.0172	0.0086	0.0516	0.083	57.6
test1-B	65.4	0.0298	0.0087	0.077	0.1239	40.6
test2-B	71.9	0.0262	0.0091	0.0705	0.1135	44.7
test3-B	74.4	0.019	0.004	0.0461	0.0742	46.2
test4-B	70.2	0.0297	0.007	0.0735	0.1183	43.6
test1-C	44.5	0.0312	0.0047	0.0718	0.1155	27.7
test2-C	42.9	0.0331	0.0132	0.0925	0.1489	26.7
test3-C	48.8	0.0284	0.0064	0.0697	0.1121	30.3
test4-C	50.4	0.0532	0.0045	0.1153	0.1855	31.3
test3-E	61.3	0.037	0.0104	0.0948	0.1525	38.1
test4-E	65.8	0.0265	0.0109	0.0749	0.1205	40.9

Note: Vehicles A and B were driven mainly on motorways (freeways)
Vehicle C was driven on Suburban Roads and
Vehicle E was driven mostly on Rural roads

Using the above data on average speed and total weight loss an exponential regression curve was fitted which was characterized by an R^2 value of 0.43. The actual and predicted values are presented in Figure 3-1.

A weak negative correlation is shown between tire wear and average trip speed, with wear being around 50% higher at an average speed of 40 km/h (dominated by urban driving) than at an average speed of 90 km/h (dominated by motorway driving).

Figure 3-1 Relationship between light-duty tire weight loss (per vehicle) and mean trip speed between tests



The shape of the curve in Figure 3-1 deserves some discussion. It can be seen from the curve that the wear is maximum at zero speed and goes down as the speed goes up. This is based on the extrapolation of the fitted curve. It may seem counter-intuitive that emissions is highest when speed equals zero, however, it is important to note that the relationship does not take accelerations (and turning) into account. Much of the tirewear occurs when the magnitude of a vehicle's acceleration/deceleration is at its greatest, e.g. at low speeds when the vehicle is accelerating from rest, or when the vehicle is braking hard to stop. A more improved relationship would be by VSP bin, however there is insufficient data to characterize tire wear on a second-by-second basis to enable binning by operating mode bins. The model has been simplified to be based on speed at this time. However, for MOVES, the emission rate at zero speed is set to zero to avoid anomalous results in project level analyses where increased idling would result in an over prediction of tire emissions.

The predicted values as determined above are for passenger cars (LDVs). To determine tire wear loss rates for other regulatory classes it was assumed that total tire wear per vehicle is dependent upon the number of tires on the vehicle which in turn is a function of the number of axles per vehicle by vehicle class. The latter data were found to be available in the Vehicle Inventory and Use Survey (VIUS 2002) data base. This data enabled the calculation of tires per vehicle for each of the six truck classes and thereby tire-wear losses for the different truck categories (regulatory classes) were determined. The average number of tires per truck is given in Table 3-3 below.

Table 3-3 - Average Number of Tires per Truck – Calculated from 2002 VIUS Survey of axle count.

RegClassID	RegClass name	Average Tires Per Vehicle
10	MC	2.0
20	LDV	4.0
30	LDT	4.0
41	LHD<=14K	5.5
42	LHD45	6.0
46	MHDD	7.0
47	HHDD	14.9
48	Urban Bus	8.0

* Note: Tires per vehicle for LDT is the same as that for LDV

In a future study, another literature search should be conducted to search for differences in (per tire) wear and emission rates from heavy-duty tires compared to those from the light-duty market. There is another assumption made for the sake of simplicity, which is to keep the emission rates of the tractive wheels identical to those of the wheels disconnected from the drivetrain axles. A more recent literature search may also help determine whether another approach is warranted.

Now that the average tire wear is quantified, it is critical to determine the fraction of that wear that becomes airborne PM. The literature indicates that probably less than 10% of car tire wear is emitted as PM_{10} under 'typical' driving conditions but the proportion could be as high as 30% (Boulter2005a). According to Luhana et al. (2004), PM_{10} appears to be released from (all 4) tires at a rate of between 4 and 6 mg/vkm for passenger cars. This suggests that generally between around 1% and 15% by mass of passenger car tire wear material is emitted as PM_{10} (though much higher proportions have been reported in some studies). For this study, it is assumed that 8% of tire wear is emitted as PM_{10} (average of 1% and 16%. According to Kupiainen et al (2005), $PM_{2.5}$ fractions were on average 15% of PM_{10}.[24] Based on this study, it is assumed that 1.2% of the total tire wear is emitted as $PM_{2.5}$ to develop our brakeweare emission rate. The 1.2% is derived from assuming that 8% of tire wear to be emitted as PM_{10} and 15% of PM_{10} is $PM_{2.5}$.

We then convert the g/vehicle/mile brakewear emission rates to g/hr by multiplying by the average speed of each MOVES speed bin. The g/hour brakewear emission rate for all regulatory classes used in MOVES can be found in Appendix B. MOVES applies the same brake wear emission rate for all vehicle fuel types (gasoline, diesel, flex-fuel, and CNG) within a MOVES regulatory class. The average $PM_{2.5}$ tire wear emission rates in (mg/mile) for each regulatory class, from three urban county inventories in MOVES2014 is shown in Table 3-4.

Table 3-4 Average $PM_{2.5}$ and PM_{10} tire wear PM emission rates (mg/veh-mile) for the MOVES regulatory classes from three urban county inventories using MOVES2014

sourceTypeID	sourcetypename	$PM_{2.5}$	PM_{10}
11	Motorcycle	0.7	4.9
21	Passenger Car	1.5	9.8
31	Passenger Truck	1.5	10.0
32	Light Commercial Truck	1.5	10.2
41	Intercity Bus	4.4	29.3
42	Transit Bus	2.9	19.7
43	School Bus	2.7	17.8
51	Refuse Truck	5.1	34.3
52	Single Unit Short-haul Truck	2.7	17.7
53	Single Unit Long-haul Truck	3.1	20.6
54	Motor Home	2.4	15.8
61	Combination Short-haul Truck	4.7	31.6
62	Combination Long-haul Truck	5.2	34.9

3.2.1 $PM_{10}/PM_{2.5}$ Tire Wear Ratio

MOVES stores $PM_{2.5}$ tire wear emission rates by operating mode bin (in this case, speed bins), then estimates PM_{10} emission rates by applying a $PM_{10}/PM_{2.5}$ ratio. Thus MOVES applies a $PM_{10}/PM_{2.5}$ ratio of 6.667, which is based on the particle size distribution of tire wear measured by Kupianen et al. (2005)[i]. The average PM_{10} emission rates from three urban county inventories using MOVES2014 are displayed in Table 3-4.

4 Next Steps

As mentioned in the earlier section, this report underwent revisions since the previous version, but these changes were largely editorial in nature in response to the peer review. There were no changes made to the model or the rates since MOVES2010. There are a number of updates that can be made to both this report and the model.

As a number of years have passed, it is possible that there are more publications in the literature or airborne brake and tire emissions from mobile sources. These papers may shed light on emission rates, size distributions, activity or speciation of PM. There is especially little information in the literature on the latter. These newer papers can either be used to modify the model, or validate the current rates.

The MOVES model has undergone changes since MOVES2010b. MOVES2014 includes some changes to the vehicle specifications described in this report. For example, the default

[i] The $PM_{10}/PM_{2.5}$ ratio is derived from dividing the PM_{10} fraction of total PM, by the $PM_{2.5}$ fraction of total PM, : .08/.012 = 6.667 from values reported by Kupianen et al. (2005).

assumptions regarding axle count (and thus number of wheels per vehicle), average weights, aerodynamics, and rolling resistance, of certain regulatory classes have changed. The weights will have a more significant impact on the brake rates (in particular) than the latter coefficients.

For brakes, the analysis from this study also only looked at front wheel drive brakes and primarily from vehicles equipped with disc brakes in the front and drum brakes in the rear (the most common light duty configuration). It was beyond the scope of this study to modify the rates the fraction of vehicles with four disc brakes, or to update the speciation profile for brake emissions, or to capture more advanced technology vehicles with electric regenerative braking. Vehicles with four disc brakes should presumably have higher, while hybrids and electric vehicles should have lower brake emissions. Moreover, the incident rate of other forms of decelerating a truck such as downshifting and engine (or jake) braking are also not considered in this study due to a lack of data.

Since the writing of this report, the only change that was made to the brake wear model in MOVES2014 was that for project level analysis, the emission rates in the idle bin was set to zero. This was done to avoid results where users may get increasing brake emissions in particular cases where idle rates are high. As mentioned above, the idle operating mode bin does contain a small amount of deceleration when a vehicle transitions from motion to non-motion (stop). However, if a user is increasing idle rates based on local knowledge compared to the MOVES default, it is logical to assume that they should not get higher brake emissions. Therefore, for inventory mode, the emission rates were maintained as described above in the idle bin and the change was made only to project level analysis.

The idling tire wear emission rate is set to zero in the default emission rate table (Appendix B). Thus, for idling tire wear emission rate is zero for both project level and inventory mode.

For tire emissions, it was beyond the scope of this study to quantify the differences in emissions (per tire) between light duty and heavy duty tires (and everything in between). It was also beyond the scope of this study to look at how trends in rolling resistance improvement may increase or decrease tire wear emissions. Finally a more complete model including speciation of tire and break PM, was beyond the scope of this study. Some of the references employed did include some of these measurements, however brake material has been known to evolve over time. These are all subjects for future study.

Appendix A Deceleration from PERE

This appendix briefly describes some of analytical methods used to determine the deceleration point at which coasting becomes braking. A full description of the PERE model is provided in a separate EPA report as cited earlier. This section, provides additional information beyond what can be found in the PERE documentation.

The basis for the tractive load equations in there PERE model are found in the A, B, C coastdown coefficients described in the report. The author of this report conducted coastdown testing on a ~2001 Nissan Altima on relatively "flat" roads in Southeast Michigan. The A, B, C coefficients for this vehicle can be found in the EPA database. The A,B,C tractive load equations in PERE were converted to a coastdown curve and plotted compared to the data below. The area above the curve is throttle and the area below the curve is braking. The curve itself is "coasting" on neutral gear.

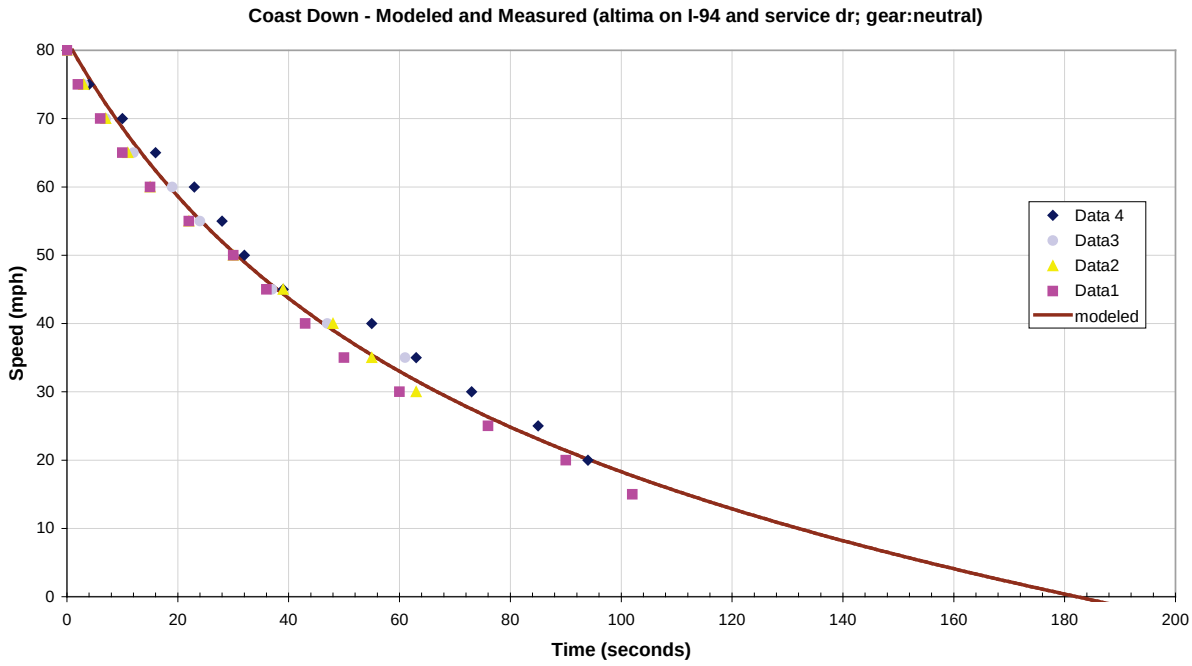


Figure A-1 Coast Down- Modeled and Measured (Altima on I-94 and Service Drive; Gear: neutral)

Based on these coastdown equations, a series of coastdown curves are generated as a function of vehicle mass. As in the previous plot, the area under the curve is braking and the area above the curve is throttling.

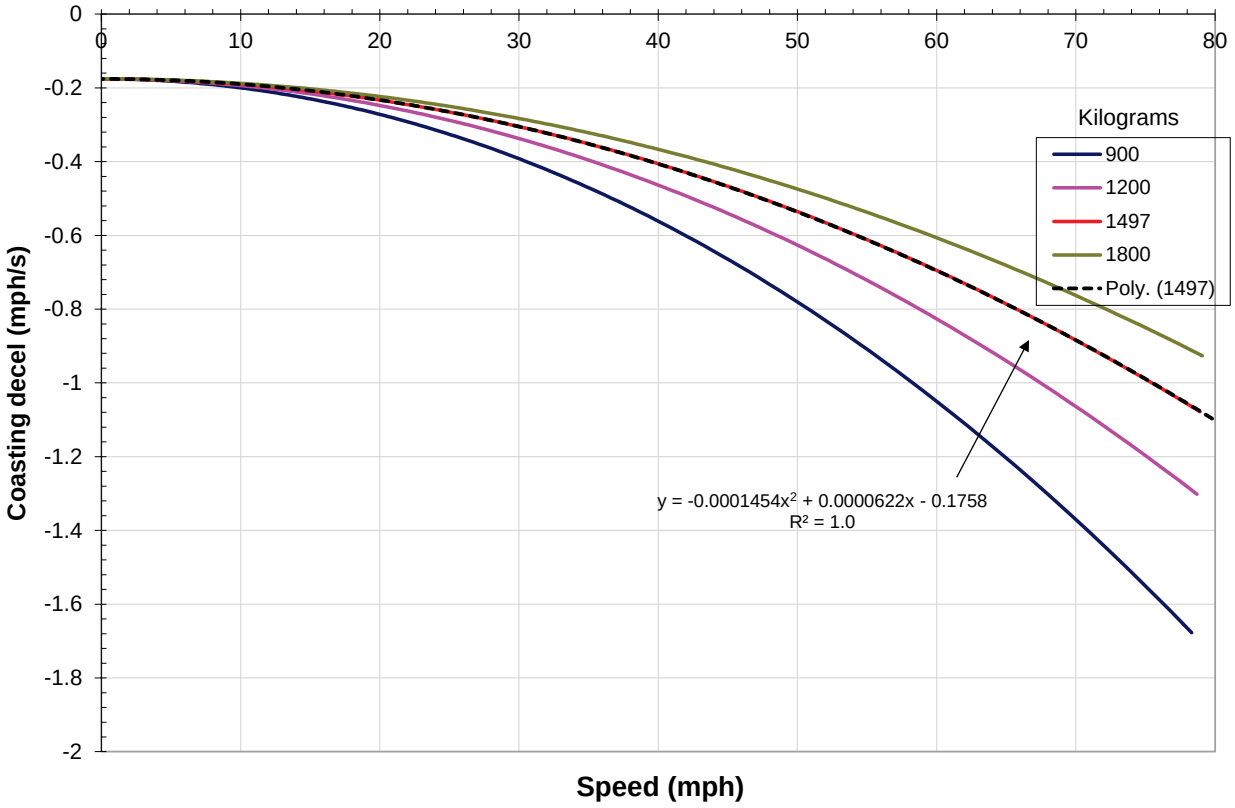


Figure A-2. Coast down Curves as a Function of Vehicle Mass

A PERE simulation is run on the FTP cycle and the braking episodes are flagged in the figure below (for a typical 1497kg LDV).

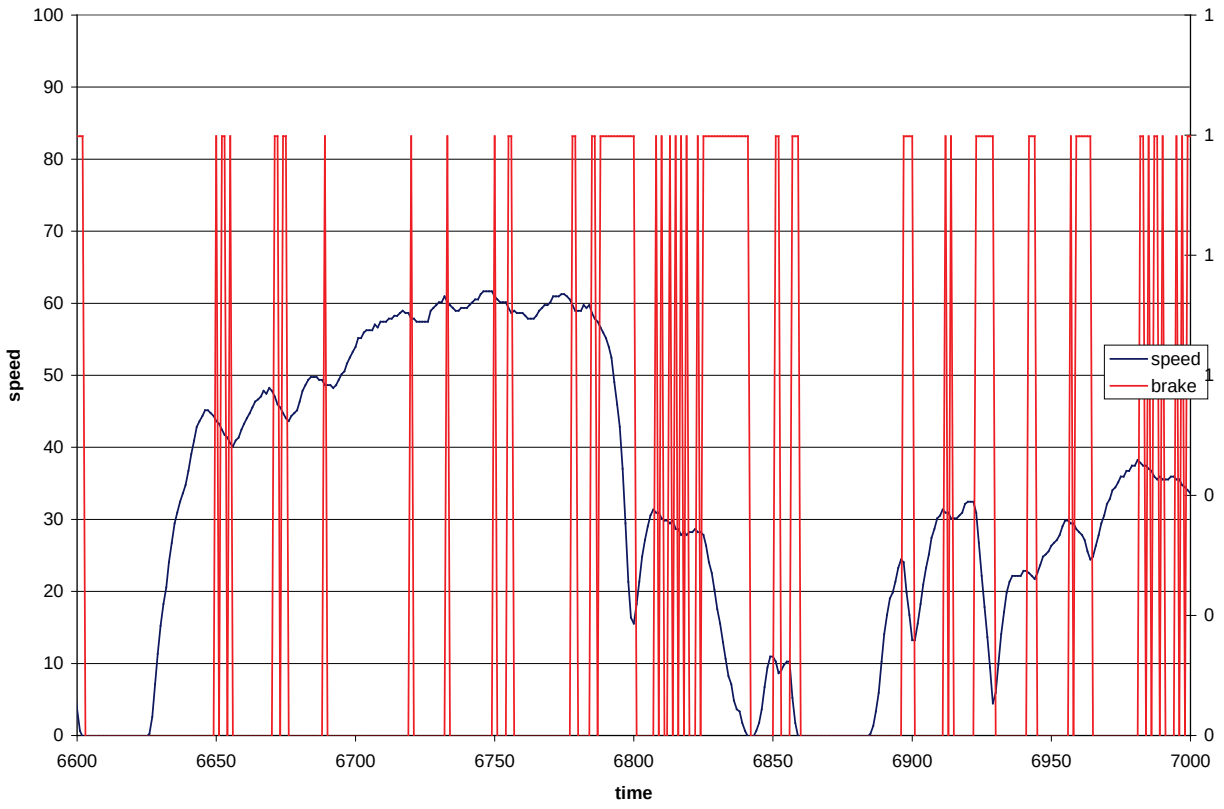


Figure A-3 Braking Episodes over the FTP cycle

Appendix B Brake and Tire Wear Emission Rates

This Appendix includes the brake and tire emission rates as a function of regulatory class and operating mode which are stored in the MOVES emissionrate table.

Table B-1 PM2.5 Brake Emission Rates by Regulatory Class and Operating Mode (g/hr)

regclassID	regClassName	opModeID	opModeName	MeanBaseRate (g/hr)
10	MC	0	Braking	0.055664
10	MC	1	Idling	0.0024472
10	MC	11	Low Speed Coasting; VSP< 0; 1<=Speed<25	0.054488
10	MC	21	Moderate Speed Coasting; VSP< 0; 25<=Speed<50	0.03584
10	MC	33	Cruise/Acceleration; VSP< 6; 50<=Speed	0.0056
20	LDV	0	Braking	0.55846
20	LDV	1	Idling	0.024472
20	LDV	11	Low Speed Coasting; VSP< 0; 1<=Speed<25	0.546
20	LDV	21	Moderate Speed Coasting; VSP< 0; 25<=Speed<50	0.35896
20	LDV	33	Cruise/Acceleration; VSP< 6; 50<=Speed	0.0644
30	LDT	0	Braking	0.940406
30	LDT	1	Idling	0.0412091
30	LDT	11	Low Speed Coasting; VSP< 0; 1<=Speed<25	0.919425
30	LDT	21	Moderate Speed Coasting; VSP< 0; 25<=Speed<50	0.623323
30	LDT	33	Cruise/Acceleration; VSP< 6; 50<=Speed	0.115046
40	LHD <= 10k	0	Braking	0.865
40	LHD <= 10k	1	Idling	0.027334
40	LHD <= 10k	11	Low Speed Coasting; VSP< 0; 1<=Speed<25	0.789745
40	LHD <= 10k	21	Moderate Speed Coasting; VSP< 0; 25<=Speed<50	0.642695
40	LHD <= 10k	33	Cruise/Acceleration; VSP< 6; 50<=Speed	0.10899
41	LHD <= 14k	0	Braking	0.865
41	LHD <= 14k	1	Idling	0.027334
41	LHD <= 14k	11	Low Speed Coasting; VSP< 0; 1<=Speed<25	0.789745
41	LHD <= 14k	21	Moderate Speed Coasting; VSP< 0; 25<=Speed<50	0.642695
41	LHD <= 14k	33	Cruise/Acceleration; VSP< 6; 50<=Speed	0.10899
42	LHD45	0	Braking	1.44
42	LHD45	1	Idling	0.045504
42	LHD45	11	Low Speed Coasting; VSP< 0; 1<=Speed<25	1.31472
42	LHD45	21	Moderate Speed Coasting; VSP< 0;	1.06848

			25<=Speed<50	
42	LHD45	33	Cruise/Acceleration; VSP< 6; 50<=Speed	0.18576
46	MHD67	0	Braking	2.09
46	MHD67	1	Idling	0.066044
46	MHD67	11	Low Speed Coasting; VSP< 0; 1<=Speed<25	1.9019
46	MHD67	21	Moderate Speed Coasting; VSP< 0; 25<=Speed<50	1.51525
46	MHD67	33	Cruise/Acceleration; VSP< 6; 50<=Speed	0.25289
47	HHD8	0	Braking	4.16
47	HHD8	1	Idling	0.06656
47	HHD8	11	Low Speed Coasting; VSP< 0; 1<=Speed<25	4.16
47	HHD8	21	Moderate Speed Coasting; VSP< 0; 25<=Speed<50	2.66656
47	HHD8	33	Cruise/Acceleration; VSP< 6; 50<=Speed	0.28288
48	Urban Bus	0	Braking	2.1
48	Urban Bus	1	Idling	0.0336
48	Urban Bus	11	Low Speed Coasting; VSP< 0; 1<=Speed<25	2.1
48	Urban Bus	21	Moderate Speed Coasting; VSP< 0; 25<=Speed<50	1.3461
48	Urban Bus	33	Cruise/Acceleration; VSP< 6; 50<=Speed	0.1428

Table B-2 PM2.5 Tire Emission Rates by Regulatory Class and Operating Mode (g/hr)

regclassID	regClassName	opModeID	opModeName	MeanBaseRate (g/hr)
10	MC	400	idle	0
10	MC	401	speed < 2.5mph	0.0031775
10	MC	402	2.5mph <= speed < 7.5mph	0.00601
10	MC	403	7.5mph <= speed < 12.5mph	0.01116
10	MC	404	12.5mph <= speed < 17.5mph	0.015525
10	MC	405	17.5mph <= speed <22.5mph	0.01922
10	MC	406	22.5mph <= speed < 27.5mph	0.0223
10	MC	407	27.5mph <= speed < 32.5mph	0.02484
10	MC	408	32.5mph <= speed < 37.5mph	0.026915
10	MC	409	37.5mph <= speed < 42.5mph	0.02852
10	MC	410	42.5mph <= speed < 47.5mph	0.02979
10	MC	411	47.5mph <= speed < 52.5mph	0.03075
10	MC	412	52.5mph <= speed < 57.5mph	0.031405
10	MC	413	57.5mph <= speed < 62.5mph	0.0318
10	MC	414	62.5mph <= speed < 67.5mph	0.03198
10	MC	415	67.5mph <= speed < 72.5mph	0.03192
10	MC	416	72.5mph <= speed	0.0318
20	LDV	400	idle	0
20	LDV	401	speed < 2.5mph	0.006355
20	LDV	402	2.5mph <= speed < 7.5mph	0.01202
20	LDV	403	7.5mph <= speed < 12.5mph	0.02231
20	LDV	404	12.5mph <= speed < 17.5mph	0.031065
20	LDV	405	17.5mph <= speed <22.5mph	0.03844
20	LDV	406	22.5mph <= speed < 27.5mph	0.0446
20	LDV	407	27.5mph <= speed < 32.5mph	0.04968
20	LDV	408	32.5mph <= speed < 37.5mph	0.053795
20	LDV	409	37.5mph <= speed < 42.5mph	0.05708
20	LDV	410	42.5mph <= speed < 47.5mph	0.05958
20	LDV	411	47.5mph <= speed < 52.5mph	0.06145
20	LDV	412	52.5mph <= speed < 57.5mph	0.062755
20	LDV	413	57.5mph <= speed < 62.5mph	0.06354
20	LDV	414	62.5mph <= speed < 67.5mph	0.063895
20	LDV	415	67.5mph <= speed < 72.5mph	0.06391
20	LDV	416	72.5mph <= speed	0.063525
30	LDT	400	idle	0
30	LDT	401	speed < 2.5mph	0.006355
30	LDT	402	2.5mph <= speed < 7.5mph	0.01202
30	LDT	403	7.5mph <= speed < 12.5mph	0.02231

30	LDT	404	12.5mph <= speed < 17.5mph	0.031065
30	LDT	405	17.5mph <= speed <22.5mph	0.03844
30	LDT	406	22.5mph <= speed < 27.5mph	0.0446
30	LDT	407	27.5mph <= speed < 32.5mph	0.04968
30	LDT	408	32.5mph <= speed < 37.5mph	0.053795
30	LDT	409	37.5mph <= speed < 42.5mph	0.05708
30	LDT	410	42.5mph <= speed < 47.5mph	0.05958
30	LDT	411	47.5mph <= speed < 52.5mph	0.06145
30	LDT	412	52.5mph <= speed < 57.5mph	0.062755
30	LDT	413	57.5mph <= speed < 62.5mph	0.06354
30	LDT	414	62.5mph <= speed < 67.5mph	0.063895
30	LDT	415	67.5mph <= speed < 72.5mph	0.06391
30	LDT	416	72.5mph <= speed	0.063525
40	LHD <= 10k	400	idle	0
40	LHD <= 10k	401	speed < 2.5mph	0.0087725
40	LHD <= 10k	402	2.5mph <= speed < 7.5mph	0.016595
40	LHD <= 10k	403	7.5mph <= speed < 12.5mph	0.0308
40	LHD <= 10k	404	12.5mph <= speed < 17.5mph	0.042885
40	LHD <= 10k	405	17.5mph <= speed <22.5mph	0.05308
40	LHD <= 10k	406	22.5mph <= speed < 27.5mph	0.061575
40	LHD <= 10k	407	27.5mph <= speed < 32.5mph	0.06861
40	LHD <= 10k	408	32.5mph <= speed < 37.5mph	0.07427
40	LHD <= 10k	409	37.5mph <= speed < 42.5mph	0.0788
40	LHD <= 10k	410	42.5mph <= speed < 47.5mph	0.082305
40	LHD <= 10k	411	47.5mph <= speed < 52.5mph	0.08485
40	LHD <= 10k	412	52.5mph <= speed < 57.5mph	0.086625
40	LHD <= 10k	413	57.5mph <= speed < 62.5mph	0.08772
40	LHD <= 10k	414	62.5mph <= speed < 67.5mph	0.088205
40	LHD <= 10k	415	67.5mph <= speed < 72.5mph	0.0882
40	LHD <= 10k	416	72.5mph <= speed	0.087675
41	LHD <= 14k	400	idle	0
41	LHD <= 14k	401	speed < 2.5mph	0.0087725
41	LHD <= 14k	402	2.5mph <= speed < 7.5mph	0.016595
41	LHD <= 14k	403	7.5mph <= speed < 12.5mph	0.0308
41	LHD <= 14k	404	12.5mph <= speed < 17.5mph	0.042885
41	LHD <= 14k	405	17.5mph <= speed <22.5mph	0.05308
41	LHD <= 14k	406	22.5mph <= speed < 27.5mph	0.061575
41	LHD <= 14k	407	27.5mph <= speed < 32.5mph	0.06861
41	LHD <= 14k	408	32.5mph <= speed < 37.5mph	0.07427
41	LHD <= 14k	409	37.5mph <= speed < 42.5mph	0.0788
41	LHD <= 14k	410	42.5mph <= speed < 47.5mph	0.082305

41	LHD <= 14k	411	47.5mph <= speed < 52.5mph	0.08485
41	LHD <= 14k	412	52.5mph <= speed < 57.5mph	0.086625
41	LHD <= 14k	413	57.5mph <= speed < 62.5mph	0.08772
41	LHD <= 14k	414	62.5mph <= speed < 67.5mph	0.088205
41	LHD <= 14k	415	67.5mph <= speed < 72.5mph	0.0882
41	LHD <= 14k	416	72.5mph <= speed	0.087675
42	LHD45	400	idle	0
42	LHD45	401	speed < 2.5mph	0.0095
42	LHD45	402	2.5mph <= speed < 7.5mph	0.017965
42	LHD45	403	7.5mph <= speed < 12.5mph	0.03335
42	LHD45	404	12.5mph <= speed < 17.5mph	0.04644
42	LHD45	405	17.5mph <= speed <22.5mph	0.05748
42	LHD45	406	22.5mph <= speed < 27.5mph	0.066675
42	LHD45	407	27.5mph <= speed < 32.5mph	0.07428
42	LHD45	408	32.5mph <= speed < 37.5mph	0.08043
42	LHD45	409	37.5mph <= speed < 42.5mph	0.08532
42	LHD45	410	42.5mph <= speed < 47.5mph	0.0891
42	LHD45	411	47.5mph <= speed < 52.5mph	0.0919
42	LHD45	412	52.5mph <= speed < 57.5mph	0.09383
42	LHD45	413	57.5mph <= speed < 62.5mph	0.09498
42	LHD45	414	62.5mph <= speed < 67.5mph	0.09555
42	LHD45	415	67.5mph <= speed < 72.5mph	0.09548
42	LHD45	416	72.5mph <= speed	0.09495
46	MHD67	400	idle	0
46	MHD67	401	speed < 2.5mph	0.011045
46	MHD67	402	2.5mph <= speed < 7.5mph	0.02089
46	MHD67	403	7.5mph <= speed < 12.5mph	0.03878
46	MHD67	404	12.5mph <= speed < 17.5mph	0.054
46	MHD67	405	17.5mph <= speed <22.5mph	0.06682
46	MHD67	406	22.5mph <= speed < 27.5mph	0.077525
46	MHD67	407	27.5mph <= speed < 32.5mph	0.08637
46	MHD67	408	32.5mph <= speed < 37.5mph	0.09352
46	MHD67	409	37.5mph <= speed < 42.5mph	0.0992
46	MHD67	410	42.5mph <= speed < 47.5mph	0.10359
46	MHD67	411	47.5mph <= speed < 52.5mph	0.10685
46	MHD67	412	52.5mph <= speed < 57.5mph	0.109065
46	MHD67	413	57.5mph <= speed < 62.5mph	0.11046
46	MHD67	414	62.5mph <= speed < 67.5mph	0.111085
46	MHD67	415	67.5mph <= speed < 72.5mph	0.11102
46	MHD67	416	72.5mph <= speed	0.1104
47	HHD8	400	idle	0

47	HHD8	401	speed < 2.5mph	0.023655
47	HHD8	402	2.5mph <= speed < 7.5mph	0.04474
47	HHD8	403	7.5mph <= speed < 12.5mph	0.08305
47	HHD8	404	12.5mph <= speed < 17.5mph	0.115635
47	HHD8	405	17.5mph <= speed <22.5mph	0.14312
47	HHD8	406	22.5mph <= speed < 27.5mph	0.16605
47	HHD8	407	27.5mph <= speed < 32.5mph	0.18495
47	HHD8	408	32.5mph <= speed < 37.5mph	0.200305
47	HHD8	409	37.5mph <= speed < 42.5mph	0.21248
47	HHD8	410	42.5mph <= speed < 47.5mph	0.22185
47	HHD8	411	47.5mph <= speed < 52.5mph	0.2288
47	HHD8	412	52.5mph <= speed < 57.5mph	0.23364
47	HHD8	413	57.5mph <= speed < 62.5mph	0.23658
47	HHD8	414	62.5mph <= speed < 67.5mph	0.2379
47	HHD8	415	67.5mph <= speed < 72.5mph	0.23779
47	HHD8	416	72.5mph <= speed	0.236475
48	Urban Bus	400	idle	0
48	Urban Bus	401	speed < 2.5mph	0.01271
48	Urban Bus	402	2.5mph <= speed < 7.5mph	0.024035
48	Urban Bus	403	7.5mph <= speed < 12.5mph	0.04462
48	Urban Bus	404	12.5mph <= speed < 17.5mph	0.06213
48	Urban Bus	405	17.5mph <= speed <22.5mph	0.0769
48	Urban Bus	406	22.5mph <= speed < 27.5mph	0.089225
48	Urban Bus	407	27.5mph <= speed < 32.5mph	0.09936
48	Urban Bus	408	32.5mph <= speed < 37.5mph	0.107625
48	Urban Bus	409	37.5mph <= speed < 42.5mph	0.11416
48	Urban Bus	410	42.5mph <= speed < 47.5mph	0.119205
48	Urban Bus	411	47.5mph <= speed < 52.5mph	0.12295
48	Urban Bus	412	52.5mph <= speed < 57.5mph	0.12551
48	Urban Bus	413	57.5mph <= speed < 62.5mph	0.12708
48	Urban Bus	414	62.5mph <= speed < 67.5mph	0.12779
48	Urban Bus	415	67.5mph <= speed < 72.5mph	0.12775
48	Urban Bus	416	72.5mph <= speed	0.12705

Appendix C Literature Review

Table C-1 Brief review of literature on brake and tire wear

Luhana,L.;Sokhi,R.;Warner,L.;Mao,H; Boulter,P;McCrae,I.S.;Wright,J and Osborn,D,"Non-exhaust particulate measurements:results," *Deliverable 8 of the European Commission DG TrEn, 5th Framework PARTICULATES project , Contract No.* 2000 -RD.11091, Version 2.0 , October 2004.	2004	Non-exhaust particle research was conducted in the Hatfield road tunnel. Combined tire and break wear emissions for PM_{10} from LDVs and HDVs in the tunnel were found to be 6.9mg/vkm and 49.7mg/vkm respectively. These emission factors from the Hatfield Tunnel Study appears to be at the lower end of the range of values reported elsewhere. The report also includes a literature review which examines the state of the art in the field. Tire wear and brake wear rates are listed below.
Sanders, Paul G.;Xu, Ning ;Dalka, Tom M.; and Maricq, M. Matti, "Airborne Brake Wear Debris: Size Distributions, Composition, and a Comparison of Dynamometer and Vehicle Tests",*Environ. Sci. Technol.*, 37,4060-4069,2003	2003	A brake wear study was performed using seven brake pad formulations that were in high volume use in 1998. Included were low-metallic,semi-metallic and non-asbestos organic (NAO) brakes.The quantity of airborne PM generated by automotive disk brakes was measured on a brake dynamometer that simulated : urban driving (low velocity,low g) and the Auto Motor und Sport (AMS,high velocity, high g). Airborne fractions from the low-metallic and semi-matallic linings were 5 and 1.5 times higher than the NAO lining.
L.R.Warner; R.S. Sokhi; L.Luhana ; P.G. Boulter; and I. McCrae,"Non-exhaust particle Emisions from Road Transport", *Proceedings of the 11th International Symposium on Transport and Air Pollution*, Graz, 2002.	2002	The paper presents preliminary results of gravimetric determination of tire and brake wear for cars, and chemical analysis of ambient particle samples for source identification using Inductively Coupled Plasma (ICP) spectrometry. Results suggest that the average loss rates of tire and brake material are 97 and 9 mg/vkm respectively. The ICP analysis shows a high relative abundance of Ba,Sb,Zr and Sr for brake and Zn for tire material. The chemical analysis also suggests that for tire wear it is much more difficult to use metal concentrations as tracers.
Abu-Allaban, M.;Gillies, J.A.;Gertler,A.W.;Clayton ,R.; and Proffitt,D., "Tailpipe, re-suspended road dust, and brake wear emission factors from on-road vehicles," *Atmospheric Environment*, 37(1),5283-5293,2002.	2002	Intensive mass and chemical measurements were performed at roadside locations to derive brake-wear emission factors from in-use vehicles. PM_{10} emission rates for LDSI vehicles ranged from 0 to 80 mg/vkm and for HDVs from 0 to 610 mg/vkm. The $PM_{2.5}$ emissions ranged from 0 to 5mg/vkm for LDSI vehicles and from 0 to 15mg/vkm for HDVs. Emissions from brake wear were highest near motorway exits.
Lukewille,A.;Bertok,I.;Amann, M., Cofala,J.;Gyarfas,F.;Heyes,C.;Karvosenoja,N.;Klimont		

Z.; and Schopp, W., " A framework to estimate the potential and costs for the control of fine particulate emissions in Europe",*IIASA Interim Report IR-01-023,*Laxenburg, Austria,2001.		
Westerlund ,K.G.," Metal emissions from Stockholm traffic –wear of brake linings ",*The Stockholm Environment and Health Protection Administration*, 100,64,Stockholm,Sweden,2001.	2001	Westerlund estimated the amount of material lost due to brake wear from passenger cars and heavy goods vehicles. The PM_{10} emission factors were determined to be 6.9 and 41.2mg/vkm for LDVs and HDVs respectively.
Garg, B.D.; Cadle, S.H.; Mulawa,P.A.; Groblicki, P.J.;Laroo,C.; and Parr,G.A., "Brake wear particulate matter emissions", *Environmental Science & Technology*, 34(21),4463,2000b.	2000	A brake wear study was performed using seven brake pad formulations (non-asbestos) that were in high volume use in 1998. Brakes were tested on a brake dynamometer under four wear conditions. The brake application was designed to simulate real world events by braking from 50km/h to 0km/h at a deceleration of 2.94 m/s^2. The estimated range of PM emission rates for small vehicles to large pickup trucks are 2.9 -7.5 mg/vkm and 2.1 – 5.5 mg/vkm for PM_{10} and $PM_{2.5}$ respectively.
Annette Rauterberg-Wulff , "Determination of emission factors for tire wear particles up to 10um by tunnel measurements", *Proceedings of 8th International Symposium on Transport and Air Pollution*, Graz, 1999.	1999	PM_{10} emission factors were determined for tire and brake wear using receptor modeling in combination with measurements conducted in the Berlin-Tegel tunnel. Tire wear emission factors for LDVs and HGVs in the tunnel was calculated to be 6.1 mg/vkm and 31 mg/vkm. For brake wear it was 1.0 and 24.5 mg/vkm respectively.
Carbotech, "PM_{10} Emissionsfaktoren:Mechanischer …….",*Arbeitsunterlage,* ,17,1999	1999	Cited in Lukewille et al.(2001). The PM_{10} brake wear emission factor for LDVs was determined to be 1.8 mg/km and for HDVs it was 3.5 mg/vkm.
Cha,S.; Carter,P.; and Bradow, R.L., "Simulation of automobile brake wear dynamics and estimation of emissions,"*SAE Transactions Paper,*831036, Society of Automotive Engineers, Warrendale, Pennsylvania,1983	1983	Particulate emissions from asbestos-based brakes from automobiles were measured under conditions simulating downtown city driving. The report presents a systematic approach to simulating brake applications and defining particulate emissions. Based on the 1.6:1.1 wear ratio between disc and drum brakes,the estimated airborne particulate (PM_{10}) emission rate was estimated to be 12.8mg/vmi or 7.9 mg/vkm.

Appendix D Responses to Peer-Review Comments

This section provides a verbatim list of peer reviewer comments submitted in response to the charge questions for the Brake and Tire Wear Emissions/Temperature Effects Report.

D.1 Adequacy of Selected Data Sources

Does the presentation give a description of selected data sources sufficient to allow the reader to form a general view of the quantity, quality and representativeness of data used in the development of emission rates? Are you able to recommend alternate data sources might better allow the model to estimate national or regional default values?

D.1.1 Dr. Chris Frey

Table 2-1 of the report is a helpful summary of data sources that were reviewed by EPA as a possible basis for estimating brake wear emission rates. EPA chose to base the brake wear emission rates for light duty vehicles mostly on a study by Sanders et al. (2003). The brake wear emission rates for heavy duty vehicles are based mostly on a study by Moahmoud Abu-Allabah et al. (2003). The tire wear emission rates are based mostly on a study by Lahuna et al. (2004).

There is a need for more critical discussion of the representativeness of the data from these studies for the U.S. onroad fleet. Since all three of these key studies are approximately a decade old, a question naturally emerges in the reader's mind as to whether the brake lining, brake shoe, or tire materials that were the basis of these studies are representative of materials currently in use. Furthermore, are the vehicles measured representative of vehicles currently in use in terms of the most relevant characteristics, such as vehicle weight, and factors such as the ratio of brake pad or brake shoe area to vehicle weight, and tire tread dimensions to vehicle weight, and so on. If these questions are not answerable, then explain why they cannot be answered. However, it is important to indicate that these issues were at least considered, even if there is not a quantitative basis upon which to make a judgment.

> *RESPONSE: An explanation was added to the paper, which describes that the paper was originally drafted in 2006, and the literature review was current at the time. The MOVES team had not the resources to update this paper in time for MOVES2014 due to the many other updates required. A Next Steps section was also added to the report describing what a future study could update. As to whether the papers are representative, this is a difficult question to answer, as these are the only papers that could be found at the time, there simply is not a large amount of research conducted on airborne tire and brake wear particulate matter emissions. The author of the report attempted to analytically adjust the data to be as representative as practicable.*

D.1.2 Dr. Joe Zietsman

The literature used as the basis for this work is sufficient. I am not aware of any other literature that has been overlooked. The only concern is that the literature is quite old (newest study is from 2004 for brake wear and 2005 for tire wear).

On Table 2-1, the vehicle type classification for the Warner et al study refers to both "passenger cars" as well as "light duty". I am not sure what the distinction is or whether it is a typo. In the Sanders work, UDS, UDP, and AMS should be defined. On looking at the source reference looks like UDS may be a typo. It seems as though the description of the Sanders study on page 3 needs to be corrected – for example, -7.9m/s should be -7.9m/s2; specify what g is in the context of the decelerations, etc.

RESPONSE: These issues have been addressed in the report. The table has also been shortened to only include papers with measurements; the papers with brake emissions estimates based on models and literature reviews have been omitted.

On page 7 – first sentence should refer to accelerations if referring to Figure 2.5.

RESPONSE: This has been clarified in the report.

On page 15, effect of horizontal curvature is discussed. It is assumed vertical curvature or grade could have an effect on tire wear, but it was not mentioned in this work.

RESPONSE: Agreed: thank you for the suggestion, this has been added to the report.

In Table 3-1 – there is no mention of the Luhana study

RESPONSE: We added the Luhana et al. (2003) study to Table 3-1.

In Table 3-3 and related text– it should be better clarified how total weight loss is calculated.

RESPONSE: the table description now includes the information that the tires were weighed.

On Page 20, Table 5 is referred to as "above", and it is actually below; Table 6 is referred to, but it doesn't exist.

RESPONSE: this has been corrected.

D.2 Clarity of Analytical Methods and Procedures

Is the description of analytic methods and procedures clear and detailed enough to allow the reader to develop an adequate understanding of the steps taken and assumptions made by EPA

to develop the model inputs? Are examples selected for tables and figures well chosen and designed to assist the reader in understanding approaches and methods?

D.2.1 Dr. Chris Frey

No.

Consider Figure 2-1. This is apparently an excel worksheet that was copied into the report. The figure is labeled as "UDP results." What is UDP? Spell it out. Results for what, exactly? The table reports "PM10 emiss." for "filter" and "ELPI" (again, always make tables and figures self-documenting – what is ELPI?). I looked in Sanders et al. (2003) to try to figure out where these reported numbers come from. For UDP, low metallic, I find in Table 5 that "filter" results are reported for "total mass" in mg/stop/brake. However, the numbers in the draft report are not the same as from Sanders et al. (2003). For example, the low metallic filter PM10 emiss. is reported as 6.9 (are the units mg/stop-brake? – not very clear given that units are not given for each column of data). Let's assume that this is 6.9 mg/stop-brake. In Table 5 of Sanders et al. (2003), I find that the individual tests range from 6.2 mg/stop-brake to 11.7 mg/stop-brake, with an average of 8.3 mg/stop-brake. Thus, as a reader, I cannot figure out either why did EPA choose one test from among the multiple reported by Sanders et al. (2003), or what adjustment did EPA make from the average of 8.3 mg/stop-brake to arrive at 6.9 mg/stop-brake? On the other hand, for the ELPI results, EPA seems to be reporting the same values as shown by Sanders et al. (2003) for the average of all reported tests for each of the three types of brake pad linings.

> *RESPONSE: All of these have been clarified in the section 2.1. For example, the acronyms have been spelled out and the units are more clear. Describing what exactly these instruments do is beyond the scope of this report. We also removed UDP from the heading of Figure 2-1. As for the reason why the paper's UDP number doesn't match Sanders et al.: We calculated 6.9 mg/stop/brake from the 8.2 mg/stop/brake. The filter number reported in Sanders is TOTAL PM Mass. In order to get the PM10 mass, the ELPI ratios were used, thus 8.2 * (7/8.3). Likewise, 1.7 was determined from 1.7 * (1.7/2) etc. A footnote was added with an example calculation.*

Also unclear: how does EPA go from average/stop/brake (what is this… isn't this average emission rate in mg/stop-brake) to "Avg./veh" (again, what is this – always report units). It would help to show an example calculation. The ratio of avg./veh to 'average/stop/brake" is approximately 3, which may be based on an assumption that the non-drive wheels have ½ the brake wear of the drive wheels. This could be more clear.

> *RESPONSE: The avg per vehicle emissions is the avg stop/veh/brake emissions multiplied by 3. This is based on the assumption made earlier that 2/3 of braking comes from the front brakes and 1/3 from the rear brakes. Footnote has been added.*

As far as the deceleration in Figure 2-1, where does this come from? Is this an average of all decelerations from all stops during the UDP? This could be more clear. Is the value of 0.0012 km/s2 reported by Sanders et al. (2003) or was this inferred by EPA. If the latter, how?Similarly, what is the basis for the "average brake time in secs" that is reported in Figure 2-1?

Similarly, what is the basis for the "avg . emissions in mg/ stop" that is reported in Figure 2-1?

> *RESPONSE: The following footnote was added below the table (f). The UDP decelerations are the average decelerations from those measured in the Sanders paper. The average brake times were determined with the assistance of one of the original authors of the paper (Matti Mariq) who supplied the second by second trace.*

The bottom of Figure 2-1 includes "emission rate for the UDP test" of 2.65 gms/hr. Since the previous lines indicate that time is for braking or per stop, clarification is needed as to the time basis for 2.65 g/hr. Is this based only on braking time? Is this based on total travel or trip time?

> RESPONSE: Yes, the emission rate is only during braking events and times as clarified in the text near the figure.

Similar comments apply to Figures 2-2, 2-3, and 2-4.

Without clear documentation of how the emission rates were estimated, it is not possible to comment on whether the curve fit in Figure 2-5 is reasonable. The fit shown is reasonable given the numbers used in fitting the curve, but the basis for, and meaning of, the numbers is unclear.

Additional comments regarding needs for improved communication of the data and methods are given in detail in a section on "Specific Comments"

D.2.2 Dr. Joe Zietsman

The methods and approaches are adequate. It is not clear in this report how the exact measurement (of the PM emissions) was conducted in the source studies, as well as the basis for assumptions/measurements regarding apportionment (of what gets emitted into the air as PM10) and what percentage of PM10 is PM2.5. For example, on Page 20 (last sentence) – 8% of tire wear as PM10 assumption is not referenced.

RESPONSE: these have been addressed throughout the document. For example the 8% text is made more clear on page 22.

D.3 Appropriateness of Technical Approach

Are the methods and procedures employed technically appropriate and reasonable, with respect to the relevant disciplines, including physics, chemistry, engineering, mathematics and statistics?

Are you able to suggest or recommend alternate approaches that might better achieve the goal of developing accurate and representative model inputs? In making recommendations please distinguish between cases involving reasonable disagreement in adoption of methods as opposed to cases where you conclude that current methods involve specific technical errors.

D.3.1 Dr. Chris Frey

EPA is making use of data collected by others and reported in the literature. The selected references generally seem to be reasonable.

The methods and procedures employed by EPA include judgments about the representativeness and appropriateness of the selected data sets for use as a basis for developing MOVES inputs, and regarding the statistical analyses conducted based on the selected data. Given lack of sufficient data from which to develop more detailed models, EPA has developed relatively simply models. The general approach is reasonable. However, the communication of what was done could and should be more clear and complete. Ideally, sufficient information should be communicated regarding the underlying data and inference approaches such that an independent investigator can reproduce the results and obtain the same answer. Many of the detailed comments given below under "specific comments" are aimed at this objective.

> *RESPONSE: These comments have been addressed throughout the report as described in greater detail in the more detailed comments.*

D.3.2 Dr. Joe Zietsman

No response.

D.4 Appropriateness of Assumptions

In areas where EPA has concluded that applicable data is meager or unavailable, and consequently has made assumptions to frame approaches and arrive at solutions, do you agree that the assumptions made are appropriate and reasonable? If not, and you are so able, please suggest alternative sets of assumptions that might lead to more reasonable or accurate model inputs while allowing a reasonable margin of environmental protection.

D.4.1 Dr. Chris Frey

In general, I agree that EPA has done a reasonable job with a very limited data set to make inferences and develop data and fitted models for use with MOVES.

D.4.2 Dr. Joe Zietsman

No response.

D.5 Consistency with Existing Body of Data and Literature

Are the resulting model inputs appropriate, and to the best of your knowledge and experience, reasonably consistent with physical and chemical processes involved in exhaust emissions

formation and control? Are the resulting model inputs empirically consistent with the body of data and literature that has come to your attention?

D.5.1 Dr. Chris Frey

Yes.

D.5.2 Dr. Joe Zietsman

With regard to the above three questions, based on the available data the methods are appropriate and reasonable. I concur with what is noted in the report regarding improvements with more data, for example allocating tire wear by VSP bin, etc. I am not aware of any current datasets or methods that can enhance this work.

D.6 Tire and Brakewear PM2.5 and PM10 Emission Rates and Speciation

MOVES2014 estimates total PM2.5 and PM10 emission rates from brake and tire wear. Additional PM2.5 speciation capabilities have been incorporated in MOVES2014 for exhaust emission processes. What recommendations do you have for EPA for incorporating the PM2.5 and PM10 speciation of tire and brakewear for future versions of MOVES?

D.6.1 Dr. Chris Frey

Clearly, it will be desirable to include speciation of PM10 and PM2.5 into future versions of MOVES for brake and tire where when sufficient data are available to support such estimates. In the current report, EPA could provide at least a paragraph summarizing what is known about the PM composition of brake wear debris, to expand upon some text on page 3. To the extent that there is or isn't information on PM composition of tire wear, EPA should add at least a paragraph to discuss this and, if applicable, summarize available information. Of particular interest is what are the key indicator species or components of brake wear and of tire wear, and are these sensitive to the materials used, or is there insufficient information to address these points? There is no information offered in the section on tire wear regarding the chemical composition of tear wear debris or emissions. Is this because no information is available?

> *RESPONSE: The literature had very limited discussion of PM speciation. There were some measurements of this, however, it was beyond the scope of this current modeling exercise. We added discussion in Section 4 Next Steps, that we would like to do more work on PM speciation of brake and tire wear.*

D.6.2 Dr. Joe Zietsman

While the additional PM2.5 speciation capabilities in MOVES 2013/2014 (in terms of added species, as relevant) will be useful, a first step for brake and tire wear emissions would be to better estimate and justify the following factors: a) PM-10 to PM2.5 ratio; b) percentage of brake and tire wear that is actually emitted as PM-10 and PM2.5.

RESPONSE: We hope to improve on these emission factors as we become aware of new literature and data. We have added a statement that we would like to improve the information on size distributions in the Next Steps Section.

D.7 General/Catch-All Review

Please provide any additional thoughts or review of the material you feel important to note that is not captured by the preceding questions.

D.7.1 Dr. Chris Frey

This is a significant report that documents an important part of the MOVES emission factor model, which is used nationally for a wide variety of regulatory and other analyses. As such, it is critically important that the report be well written and very clear. While the current draft of the report is good in many respects, it comes across as a draft and is not in final form in terms of the critical thinking needed to make sure that it clearly communicates information to the reader.

For each of the major sections on brake and tire wear, it will help the reader to have clearly labeled sections that deal with light duty vehicles and with all other vehicle source categories. It will also help to clearly define and consistently use terms and concepts. For example, it is critically important to have a clear quantitative definition of break wear and of tire wear, and to be clear as to whether these are rates per wheel, per vehicle, or other (specify).

RESPONSE: We added Light- Duty to Section 2.23, and Heavy-duty and Other Vehicles to Section 2.2.5.

Since this is a formal technical report, the use of first person should be avoided. Statements regarding how an analysis was done, or regarding judgments that were made, can be made without self-reference.

In general, be careful about significant figures. It is pretty rare in this type of work that data are known with more than 3 significant figures. However, in various places, numbers are reported with 5 or 6 significant figures, and often with 4. Even if the original data might be known with many significant figures, its adoption for use in representing a national fleet introduces uncertainty, since the original data may not represent the U.S. national fleet as it exists today.

RESPONSE: Significant figures for intermediate numbers were largely maintained. The final numbers were appropriately adjusted. Some of the figures/tables have reduced sig figs now as well.

Many specific comments are given below that elaborate on responses given above in response to the charge questions.

Specific Comments (numbers refer to page/paragraph/line … e.g., 3/3/1 means page 3, paragraph 3, line 1)

RESPONSE: Unless otherwise noted, all minor comments have been addressed in the report in the relevant sections. Responses are provided to the major comments.

Given that this report will be finalized perhaps in 2013 or 2014, explanation is needed for the reader as why the literature review starts off with information from 2004. Is this because more recent data are not available?

Page 2, 3rd paragraph in Section 2.1, next to last line "is acceptable" does not fit here.

Page 2, 4th paragraph in Section 2.1: while these statements seem intuitively reasonable, they are stated as if they are known facts. However, no references are cited. How do the authors know that these are accepted facts? Or are these the author's hypotheses or opinions? If factual, then cite reference(s). If these are hypotheses, then say so.

RESPONSE: we believe that these statements in this particular paragraph are obvious to anyone with a basic knowledge of vehicle brakes. While not all readers of this paper may have knowledge of brakes, some of the text in this paper are meant to be include basic instructive material as well. As such, we do not believe that relatively "basic" statements like this require references. However, the subsequent statements in the following 2 paragraphs are more specific. We've added a parenthetical when the Garg paper is first mentioned that much of the basic PM information comes from this paper. This paper is very important in the list of this study's references.

Please carefully define what is meant by "wear" and then use the definition consistently. Does "wear" refer to a mass rate of emissions or loss per tire per braking episode, or is it a time-based rate for a tire (or a vehicle), or a mileage-based rate? If this is not defined, then readers will make their own assumptions as to what this means. Does "greater wear" mean greater rate of wear, or more accumulative lost mass regardless of time period?

RESPONSE: the definition of wear versus airborne PM has been differentiated in various points in the paper. Wear means slightly different things in the literature, but in this paper it is generally, the mass of material lost, whether in the brake pads or the tires. A fraction of that wear is airborne PM. Some studies look at both wear and airborne PM, others look at one or the other. We added discussion providing a definition of brake wear, and airborne PM in Section 2.1.

3/2/1: Page 3, 2nd paragraph, a 2000 study is not "recent" in 2013… delete "In recent studies,"

3/2/6: "ranged from 3.4 mg/mile to 4.6 mg/mile" is the correct way to write the range at the end of the same paragraph.

3/3/1: "currently used" – refers to 2003 or to now?

3/3/7: this list is hard to read because apparently not all brake linings were measured in all types of tests, but the reader has to reread this a few times to really figure this out.

3/3/9: -7.9 m/s is not an acceleration. Is this supposed to be -7.9 m/s2?

3/3 – near end of paragraph… it would help to give a summary of how the PM composition varies by brake type.

RESPONSE: We added as much information as we could, but we do not have perfect information.

End of page 3 – why is there no discussion of other references, particular ones published shortly before or after Sanders et al. (2003), such as Warner et al. (2002), Abu-Allaban et al. (2002), and others. From Table 2-1, the reader infers that some of these could also be useful. If EPA judged that they are not useful, a rationale should be given.

RESPONSE: a paragraph was added to the paper here.

Related to page 3 – is the current market share of each brake lining type known? If so, please summarize. If not, then say so.

RESPONSE: We added a sentence saying that this is not known in this section.

4/1/1: "is based on the average of the" is better than "averages"

4/3: why are only results from Sanders et al. (2003) used here… give an explanation.

5/1/1: how sensitive are results to the assumption regarding equal mix of brake types?

RESPONSE: this is answered indirectly earlier in the report where there is a summary provided from the paper regarding the differing results from the varying materials.

5/1/2: is there some basis for the assumption that 2/3 of braking power is in the front brakes? Actually, the assumption made here is that the rate of brake lining wear is twice that for front drive wheels than for rear nondrive wheels. Is the assumption really based on "power"?

RESPONSE: this information was provided by Matti Mariq from Ford who is a co-author on the Sanders paper and helped us with the data and information like this in this report. This is a "rule of thumb" in the industry. It is also consistent with what is written in Garg and that is now added to the footnote.

5/1/4: what is meant by "total PM"? is this total suspended particulate matter?

Page 8 – include definitions of opmode bins 0 and 1.

8/1: to reader it is unclear as to why 1,497 kg is used. Explain that this is a typical weight of a sedan passenger car. Is this the only selected weight? Why not others? What about larger vehicles?

RESPONSE: this is the average weight of passenger cars in MOVES. Other vehicle type weights are provided elsewhere in the report. We added this to the report.

Page 9, figure 2-7: "coastdown curves" for what, based on what data sources? Figure and table captions generally need to be more specific throughout the report. Also, the number of significant figures given for the equation borders on the absurd.

RESPONSE: PERE is a vehicle simulation model, that has undergone separate peer review. A complete description of how these plots were developed would be lengthy and distract from the focus of the report. We do not believe that it is required for what is a minor part of the quantification of the braking emissions. However, more detail was added with three figures on the PERE coast down and braking estimates in Appendix A.

9/1/3: "Eastern" not "Easter"

9/2/2: it is very unclear as to how the distribution of braking activity across speed and deceleration can be determined from the numbers given in Table 2-7.

9/2/2: "vast majority of braking" – how is this known? What is a "minor slowdown"? (give quantitative criteria for this).

RESPONSE: The following table quantitatively shows deceleration frequencies drop as braking becomes more severe. We disagree that an actual quantitative definition of mild and full decelerations is required here. This is not a distinction that is made in the MOVES model.

11: Table 2-8: in text or footnote, show the equations that define or use the constants given such as "wgt", "Cr0", "Cd", "A"… and preferably use mathematical nomenclature. Also, do not report "m^2". This should be "m2" Please use source bin terminology and please define what the columns are in this table… i.e. the last 5 columns should have a superheader of "Vehicle Type." Does MOVES have source ids for "Compact," "Mid-size," "SUV," "mddt," and "tractor"?. If these are assumptions that are meant to apply to source bin ids in MOVES, then use proper MOVES terminology to avoid confusion.

RESPONSE: MOVES does not have source types for compact, mid-size or SUV. These source type definitions are typical for vehicle simulation models like PERE. These models are capable of greater precision than MOVES, and an attempt is made here to model greater detail first, then aggregate the results up to MOVES source bins.

Table 2-8 has some definition problems, in that the first four rows of numbers are not fractions and thus should not be defined as such, and yet the last 7 rows are in a sense undefined, because there is no header in the table that defines what they are. There is inconsistency in the number of decimal places given. Also, the basis for these numbers is unclear – where do they come from and how exactly where they derived?

RESPONSE: The table has been cleaned up. Also more information has been included in the text near this table as well as appendix.

11/1: it would help to have an example calculation showing how these emission rates were estimated.

13/1: please give some rationale as to why this study was used and/or why others were not. Was this the only relevant study? The most recent study? The best study? Just stating that it was used is not sufficient to explain to the reader why it was used. Also, please indicate what type of instruments were used and at least some summary of how the sampling was done, and how it was possible to associate the measurements with a per vehicle emission rate specific to brake wear.

RESPONSE: a lengthy description has been added on page 13 and following.

13/1/7: "by ratioing"… ratio is not a verb. "by taking the ratio of…" More importantly, please explain the empirical basis for these ratios. Upon what measurements are they based, and how was it possible to distinguish among the vehicle categories – e.g., how was it possible to apportion measurements to vehicle types?

Table 2-2: the caption "scaling to other vehicle class" may mean something to the author, but lacks sufficient detail and specificity to mean much to the reader. What exactly is contained in this table and what is the source or basis of the information? Tables and figures should be self-documented to the extent possible, to make very clear as to what is the content. Please define or explain specialized terms – e.g., "regclasswt" and "regclassid" are variables used in MOVES (define them). What is the basis of the weight ratio? Is this based on weights given in MOVES (for clarity, report the weights so that the basis of the weight ratios is more clear). What is the source of the "mg/mi" and why is there no description of what this is in the column header? Similarly, "gms/hr**" has no explanatory title nor is there any explanation of whether "**" is meant to be a footnote. This type of sloppy and incomplete documentation of tables leads to long-term confusion as to the basis of data contained in MOVES, and causes a lot of problems for MOVES users for many years to come. Thus, it is essential that these reports be well documented.

RESPONSE: A paragraph has been added before Figure 2-4 in addition to a Figure 2-4 and additional tables (Tables 2-12, 2-13) to help clarify.

13/2/2: "a PM10/PM2.5 ratio" – is one ratio used regardless of opmode bin? Source category? Etc.? Needs to be more clear.

RESPONSE: this has been clarified in the report that there is one ratio used for simplification.

13/2: The PM10/PM2.5 ratio is "based on the assumption that the mass fraction of particles below PM10 is 0.80, and that the mass fraction of particles below PM2.5 is 0.1" ☐ this is suggested revised text. I do not think that the authors really mean to refer to the fraction of

particle number, but rather mean to refer to fraction of particle mass, yet the text implies that these ratios are by particle number. There is a big difference. Also, explain the basis of these assumptions. Are these mass fractions of 0.8 (why 0.80 – 2 sig figs) and 0.1 (why only 1 sig fig) based on measurements, a wild guess?

RESPONSE: Additional clarification on the size distribution used to derive the PM10/PM2.5 fraction from Sanders et al. (2003) is provided in Section 2.2.4.

13/2/6: what are examples of cases either in which the PM2.5 values are known, or are not known, so that the reader has some idea of how extensive is the reliance on the assumption?

13/3: could delete "Tires are an essential part of any vehicle and"

RESPONSE: this is a matter of style. We disagree on this deletion.

14/2: this paragraph ("Tire wear occurs through…") needs references. Some of the points here are repetitive of previous text.

14/4 ("The key influences…") this text comes across as repetitive although it does more specifically refer to vehicle characteristics, whereas other lists given previously seem not be specifically about vehicle characteristics. However, in general, this material could be better and more clearly organized with less repetition and with citation to literature to support statements of apparent fact that presumably have an empirical basis.

14/5 "Retreads are considered…" "considered" is extremely vague. "estimated" would be better. However, the text that follows does not support this claim. It merely indicates that retreads have less tire volume than the original tread. Why does this imply more wear? And what does more wear really mean – a higher rate of wear in terms of mass per stop, mass per time, mass per mile?

RESPONSE: this is another issue that is commonly known. The references include some other interesting facts, however, the report now lists that modeling emissions from retreads is beyond the scope of the report in Section 3.1 .

14/6: any statement that starts with "According to the literature" must end with one or more cited references.

RESPONSE the subsequent paragraphs and table have many references on the technique that is introduced in this sentence.

Page 15: Table 3-1 appears but I cannot find anywhere in the text where it is mentioned or discussed. If it is not mentioned or discussed, why is it here, or if it is here, why isn't it mentioned or discussed? Also, clarify specifically what is meant by the tire wear rates – are these rates per vehicle and, if so, based on how many tires per vehicle? The mass reported here is for what PM size range? Why isn't Luhana et al. (2004) summarized in this table, since it is the only reference really used?

15/1/1-2: delete “that have been carried out”

15/2/2: “m9ass” should be “mass”

15/3/2: delete “That being said” (too colloquial for a formal technical report).

15/4/2: change to “with results reported either as”

16/1: there is a disconnect between stating that data are primarily from one reference and yet that reference is not included in the summary table just above.

16/1/middle – there are syntax problems here.

Table 3-2: caption is too vague. What type(s) of vehicles? What type(s) of tires? What is the source of these data? The table is not very clear either – e.g., mean tire weight loss “mg/km” – does this refer to per tire or per vehicle? The numbers reported in the table have inconsistent numbers of decimal places and/or significant figures.

17/1: does mg/vkm refer to all tires on a vehicle, or one tire? Clarify throughout.

17/2/1: “the tables below” – cite the specific table(s).

Table 3-3. The caption “Data used for the analysis” takes the prize for vagueness. I don’t think there could be a more meaningless caption. The caption should specifically indicate the content of the table. The lack of thought that went into this table is frustrating for the reader. These are some examples: “Front-wheel drive vehicles only” is not a valid column header, it should be part of the caption; “units” does not describe what is in the first column, yet why not use the same labels for both Tables 3-2 and 3-3 to denote the same tests (i.e. be consistent); the average trip speed of the first data row of 90.3 km/hr is not the same as the corresponding number in Table 3-2; is the tire weight loss per tire or the sum of both wheels on the axles? i.e. are the units g/km-axle or g/km-tire?; total weight loss for all tires (could be more clear) and could have one superheader over both of the columns that contain this information. Why is average speed given in the 2nd and 7th columns, rather than in adjacent columns if the only difference is a units conversion?; where do these data come from (what reference), and why not (in the footnote) specifically state the year, make, and model of each vehicle?

RESPONSE: This and other tables throughout the report have more descriptive captions now. The table has also been reformatted and redone so that the information is more clear to the reader.

Figure 3-1 and 3-2: the caption should indicate the vehicle type and whether the g/mi is per tire or per vehicle. If per vehicle, based on the assumption of how many tires/vehicle? Also, are mean trips speeds based on a particular time period of travel? One could divide a trip arbitrarily into short segments and obtain a wider range of mean trip speeds. However, if the averaging time is something like 60 seconds, 600 seconds, or 3000 seconds, that would affect the appropriate use of these rates.

RESPONSE: The paper does not specify the trip times.

Figure 3-2 – this figure seems unnecessary. The fitted curve is in Figure 3-2. If the purpose is to show the curve extrapolated beyond the range of the observed data, this could be done in figure 3-1 by using a dashed line for the fitted curve when it is outside the range of mean trip speeds.

RESPONSE: this figure has been removed.

19/1/1: rewrite as "Based on extrapolation of the fitted curve, wear is highest at zero speed and decreases as the speed increases." Why is this counter-intuitive? Lower mean trip speed might imply more variability in speed and, hence, more acceleration/deceleration(braking) that could increase tire wear. Thus, to me, this curve is intuitive. Delete "it is important to note" which is a passive phrase that has no content. The statement that "the relationship" does not take accelerations (and turning) into account is probably false. Aren't these data based on real-world driving? If so, then the observed wear rates implicitly take into account these factors, and the curve is fitted to these data – thus, the curve implicitly accounts for these.

RESPONSE: more text is included in this section to help clarify why the authors believe why some readers may believe this to be counterintuitive.

20/1/2: how is it known that the vehicle is "braking hard"?

RESPONSE: this is a descriptive term, not scientific. We do not feel that change to the text is required.

20/1/2-4: text here is a bit awkward… could say that there is insufficient data to characterize tire wear on a second by second basis to enable binning by operating mode bins.

20/2/1: this first sentence needs to be stated up front – i.e. that this section focuses on LDVs. Tire wear rates for other than LDVs should be in a new section. The assumption that tire wear is just based on the number of tires also presumes that tire wear for larger tires is the same as for LDV tires, and that tire wear is the same regardless of the ratio of weight/number of tires on a vehicle. These assumptions should be more clearly enumerated and discussed. Might it be the case that all else being equal (e.g., weight, acceleration, speed, road surface, etc.), tires with larger tread surface in contact with the ground would have less wear, but that more weight per tire would increase wear?

RESPONSE: We added a sentence at the beginning of Section 3.2 stating that the analysis starts with light-duty, and then is extended to other vehicle types. A number of engineering assumptions were required here. There is clearly a lack of information in the literature (as of when this study was completed) that would give emission rates for a variety of regulatory classes. A paragraph was added on future work.

20/2/4: Should say "The latter data were found in the …" s

Table 3-4: does the 2002 VIUS Survey report the number of tires per truck? Or the number of axles? If the latter, then it is not valid to imply that the number of tires is from the survey.

However, in either case, the survey can be cited with appropriate explanatory text in a footnote. The report is inconsistent in how vehicle categories are defined and described. If "regclass" is actually used here, which doesn't' seem quite right (aren't these source id?), then also give the MOVES code associated with each for complete clarity. There is no way that there are 519 billion LDVs in the U.S. – something is wrong with all of the numbers in the 3rd and 4th columns of this table. Some explanation is needed of the "Tires Per Vehicle" – this must be "Average Tires Per Vehicle," since no vehicle can have 5.5 or 14.9 tires, and 7 tires would also be quite unusual. Some explanation of the basis for the number of tires per vehicle for each vehicle type is needed. The assumption that the number of tires for LDT is the same as for LDV seems reasonable, but should be explained – i.e. LDT includes SUV, minivan, and pickup trucks which are typically 4 wheels. Larger pickup trucks that might have 6 wheels are not considered or do they fall into another category?

> *RESPONSE: We added clarification in the Table heading, that the tires were calculated from the 2002 VIUS Survey axle count. We also removed the survey weighting factors that were not helpful from for the reader. We also, specify that the Tires are calculated as the averages in the Table headings, which can yield the non-integer numbers. As discussed in the MOVES2014 Population and Activity Report, trucks with 6 wheels are classified in RegClassID 41, not as LDT.*

20/3/1: text refers to "Table 5" – should this be Table 3-5? (but Table 3-5 seems to be on a different topic).

20/4: This paragraph is unclear. It starts by stating that probably less than 10% of car tire wear is emitted as PM10 based on Boulter (2005a) and then goes on to say that results of Kupiainen et al. (2005) were used by Boulter. If Kupiainen is the original source of the data/information, then this could be more clear and conveyed consistently in the paragraph. Also, here and in general throughout the report, clarify if the mg/vkm values are per tire or per vehicle.

> *RESPONSE: this sentence was removed. Kupiainen and Boulter are research collaborators and co-authors, judging from their series of publications.*

20/5: what is the basis for assuming 8% of total tire wear is PM10 and that 15% of PM10 is PM2.5 For clarity, what happens to the other 92% of tire wear? Is it assumed that none of it ever re-emitted to the atmosphere?

> *RESPONSE: References and description of how the 8%, and 15% mass fractions were selected are added in Section 3.2. We also added a footnote (h) describing how the PM10/PM2.5 ratio is derived from the PM mass fractions in Section 3.2.1.*

Table 3-5: please clarify if the g/mi tire wear is per vehicle or per tire. What is "avgbinspeed" and "RegclassID" – should define/explain in footnotes. The last 8 columns need a superheader of "Regulatory Class" and it would help if each of the individual columns included descriptive

text in the header. Avebinspeed should have units. SpeedBinID and OpModeID also need to be defined/explained.

Response: The previous table is removed, and is replaced with Table 3-4, which includes output by MOVES2014 by sourcetypeID. We also specify that the tire wear emission rates are per/vehicle-mile. We also included information on the exact rates used in MOVES (g/hour), in Appendix B.

21/1: for clarity, insert information that 15% of the mass of PM10 is estimated to be emitted as PM2.5.

For the references, the reference format does not seem to follow any standard convention.

Response: We have provided information such that readers can find the sources.

D.7.2 Dr. Joe Zietsman

Overall, this report on brake and tire wear emissions in MOVES 2014 is well documented and satisfactory. Most of my comments are related to clarifications that are needed rather than methodological issues.

References

[1] Harrison, R.M., A. M. Jones, J. Gietl, J. Yin, D. C. Green, "Estimation of the Contributions of Brake Dust, Tire Wear, and Resuspension to Nonexhaust Traffic Particles Derived from Atmospheric Measurements," Environmental Science & Technology, 2012.

[2] Edmunds.com, http://www.edmunds.com/car-technology/brakes-drum-vs-disc.html

[3] Cha, S., P. Carter, R. Bradow., "Simulation of Automobile Brake Wear Dynamics and Estimation of Emissions", SAE Paper 831036. Society of Automotive Engineers, Warrendale, PA, 1983.

[4] EPA, PART5 Documentation

[5] Garg, B. D., S. H. Cadle, P. A. Mulawa, P. J. Groblicki, C. Laroo, G. A. Parr, "Brake Wear Particulate Matter Emissions", Environmental Science and Technology, 34(21), 4463-4469, 2000.

[6] Sanders, P.G., N. Xu,T. M. Dalka, M. M. Maricq, "Airborne Brake Wear Debris: Size Distributions, Composition, and a Comparison of Dynamometer and Vehicle Tests", Environmental Science & Technology,37 (18), 4060-4069, 2003.Note that 7 is missing. Maybe this will be cleaned up after all track changes are accepted.

[7] EPA, Development of Emission Rates for Light-Duty Vehicles in the Motor Vehicle Emissions Simulator (MOVES2010),198 pp, EPA-420-R-11-011, August 2011)

[8] Nam, E.K., Giannelli, R., *"Fuel Consumption Modeling of Conventional and Advanced Technology Vehicles in the Physical Emission Rate Estimator (PERE)"*, EPA document number EPA420-P-05-001, 2004

[9] EPA report, "Kansas City PM Characterization Study". EPA420-R-08-009. 2005.

[10] Sierra Report No. SR02-07-04, "Task Order No. 2 SCF Improvement – Field Data Collection," July 2002

[11] Sierra Report No. SR02-07-03, "Task Order No. 3 SCF Improvement – Vehicle Instrumentation and Instrumented Vehicles," July, 2002

[12] Sierra Report No. SR02-07-05, "Task Order No. 7 SCF Improvement – Driving Data Collection, South Coast Air Basin," July, 2002

[13] Abu-Allaban, M., Gillies, J.A.,Gertler,A.W., Clayton ,R., Proffitt,D., "Tailpipe, re-suspended road dust, and brake wear emission factors from on-road vehicles," Atmospheric Environment, 37(1),5283-5293, 2002.

[14] National Research Council" Modeling Mobile-Source Emissions Committee to Review EPA's Mobile Source Emissions Factor (MOBILE) Model, Board on Environmental Studies and Toxicology", Transportation Research Board, National Research Council 200.

[15] Dunlop South Pacific tires report to the Ministry of Transport, Government of New Zealand

[16] Maître, O., Süssner, M., Zarak, C., "Evaluation of Tire Wear Performance", SAE Technical Paper 980256, doi:10.4271/980256, 1998.

[17] Cenek, "Tyre Wear Modleing for HDM4", 1993.

[18] Lowne, R. W., "The Effect of Road Surface Texture on Tire Wear," Vol. 15, pp. 57-70, 1970.

[19] Bennett, C.R., Greenwood, I.D., "Modeling road use and environmental effects in HDM -4". HDM-4 Ref. Vol. 7. World Road Association, Paris, 2001.

[20] Carpenter, P., P. Cenek, "Tyre Wear Modeling for HDM4," Opus International Consultants, Limited, New Zealand, 1999.

[21] Hildemann L. M., Markowski G. R., Cass G. R., "Chemical composition of emissions from urban sources of fine organic aerosol", Environmental Science & Technology 25, 744-759, 1991.

[22] Rauterberg-Wulff A., "Determination of emission factors for tyre wear particles up to 10µm by tunnel Measurements", Proceedings of 8th International Symposium 'Transport and Air Pollution', 1999.

[23] Luhana L., Sokhi R., Warner L., Mao H., Boulter P., McCrae I., Wright J., Osborn D., "Measurement of Non-exhaust Particulate Matter", Deliverable 8 of Particulates-project. European Commission, DG TrEn, 5th Framework Programme. 96 p, 2004.

[24] Kupiainen, K.J., Tervahattu, H., Räisänen, M., Mäkelä, T., Aurela, M., Hillamo, R., Size and composition of airborne particles from pavement wear, tyres, and traction sanding. Environmental Science & Technology 39, 699e706, 2005.

[25] Councell, T.B., Duckenfield, K.U., Landa, E.R., Callender, E.,. Tire-wear particles as a source of zinc to the environment. Environmental Science & Technology 38, 4206–4214, 2004.

[26] Warner L., R.S. Sokhi, L.Luhana, P.G. Boulter, I. McCrae, Dept. of Environmental Sciences Univ. of Hertfordshire,UK, Non-exhaust particle emissions from road transport, 11th International Conference "Transport and air pollution", 2004.

[27] Kolioussis, M., Pouftis, C., "Calculation of tyre mass loss and total waste material from road transport", Diploma Thesis, Laboratory of Applied Thermodynamics, Report No. 0010, Thessaloniki, Greece 2000.

[28] EMPA "Anteil des Strassenverkehrs an den PM10 und PM2.5 Imissionen", NFP41, Verkehr und Umwelt, Dubendorf, Switzerland, 2000.

[29] SENCO (Sustainable Environment Consultants Ltd.) "Collation of information on particulate pollution from tyres, brakes, and road surfaces", 23 March, 1999, Colchester, Essex, UK 2000.

[30] Exemplarische Erfassung der Umweltexposition Ausgewaehlter Kauschukderivate bei der bestimmungsdemaessen Verwendung in Reifen uind deren Entsirgung. UBA-FB 98-003

[31] Garben et al. Emissionkataster Kraftfahrzeugverkehr Berlin 1993, IVU GmbH Berlin, Gutachten im Auftrag der Senatsverwaltung fur Stadtenwicklung, Umweltschutz und Technologie, Berlin, unveroeffentlich, 1997.

[32] Gebbe et al. Quantifizierung des Reifenabriebs von Kraftfahrzeugen in Berlin, ISS-Fahrzeugtechnik, TU Berlin, i.A. der Senatsverwaltung fur Stadtenwicklung, Umweltschutz und Technologie, Berlin, 1997.

[33] Lee, P.K., Touray, J.C., Baillif, P., Ildefonce J.P., "Heavy metal contamination of settling particles in a retention pond along the A-71 motorway in Sologne, France", The Science of the Total Environment, 201, 1-15, 1997.

[34] Baekken, T. , "Environmental effects of asphalt and tyre wear by road traffic", Nordisk Seminar-og Arbejdsrapporter 1992:628 Copenhagen, Denmark 1993.

[35] Schuring, D. J., Clark, J. D., Rubber Chemistry and Technology, 61, 669-687, 1988.

[36] Malmqvist, P.A. "Urban storm water pollutant sources", Chalmers University, Gothenberg, Sweden, 1983.

[37] Gottle, A.,Ursachen und Mechanismen der Regenwasserverschmutzung - Ein Beitrag zur Modellierung der Abflussbeschaffenheit in st dt. Gebieten. Berichte aus Wassergutewirtschaft und Gesundheitsingenieurwesen, TU Munchen H.23, 1979.

[38] Cadle, S. H., Williams, R. L., "Gas and particle emissions from automobile tyres in laboratory and field studies", Rubber Chemistry and Technology, 52(1), 146-158, 1978.

[39] Dannis, M. L., "Rubber dust from the normal wear of tyres", Rubber Chemistry and Technology, 47, 1011-1037, 1974.

[40] Luhana, L., Sokhi, R., Warner, L., Mao, H , Boulter, P., McCrae, I.S., Wright, J. Osborn, D,, "Non-exhaust particulate measurements:results," *Deliverable 8 of the European Commission DG TrEn, 5th Framework PARTICULATES project , Contract No.* 2000 -RD.11091, Version 2.0 , October 2004.