Air Toxics Hot Spots Program Risk Assessment Guidelines

Technical Support Document for Exposure Assessment and Stochastic Analysis

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California Environmental Protection Agency
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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.

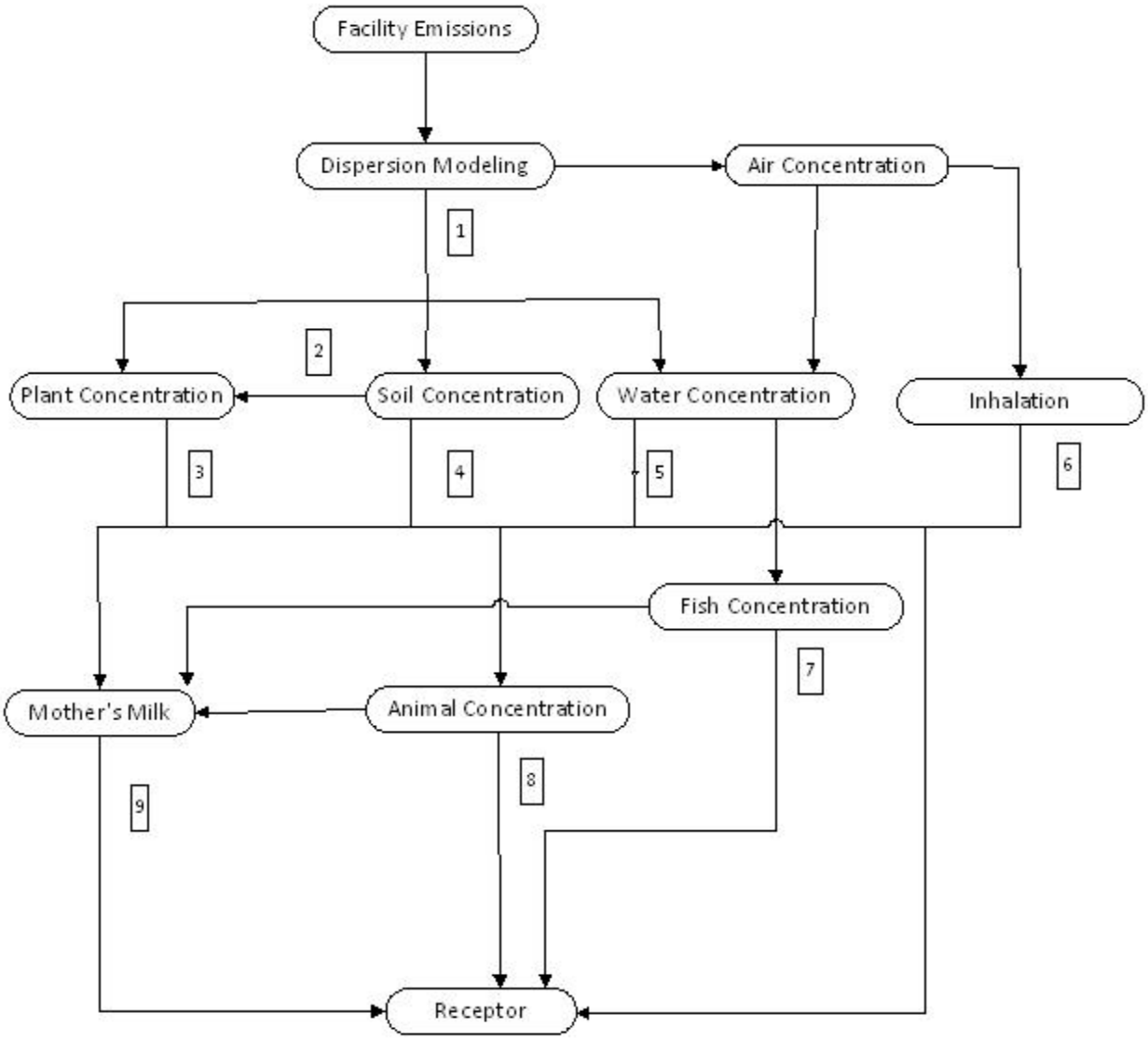
1.1 Multipathway Nature of Exposure Assessment

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)

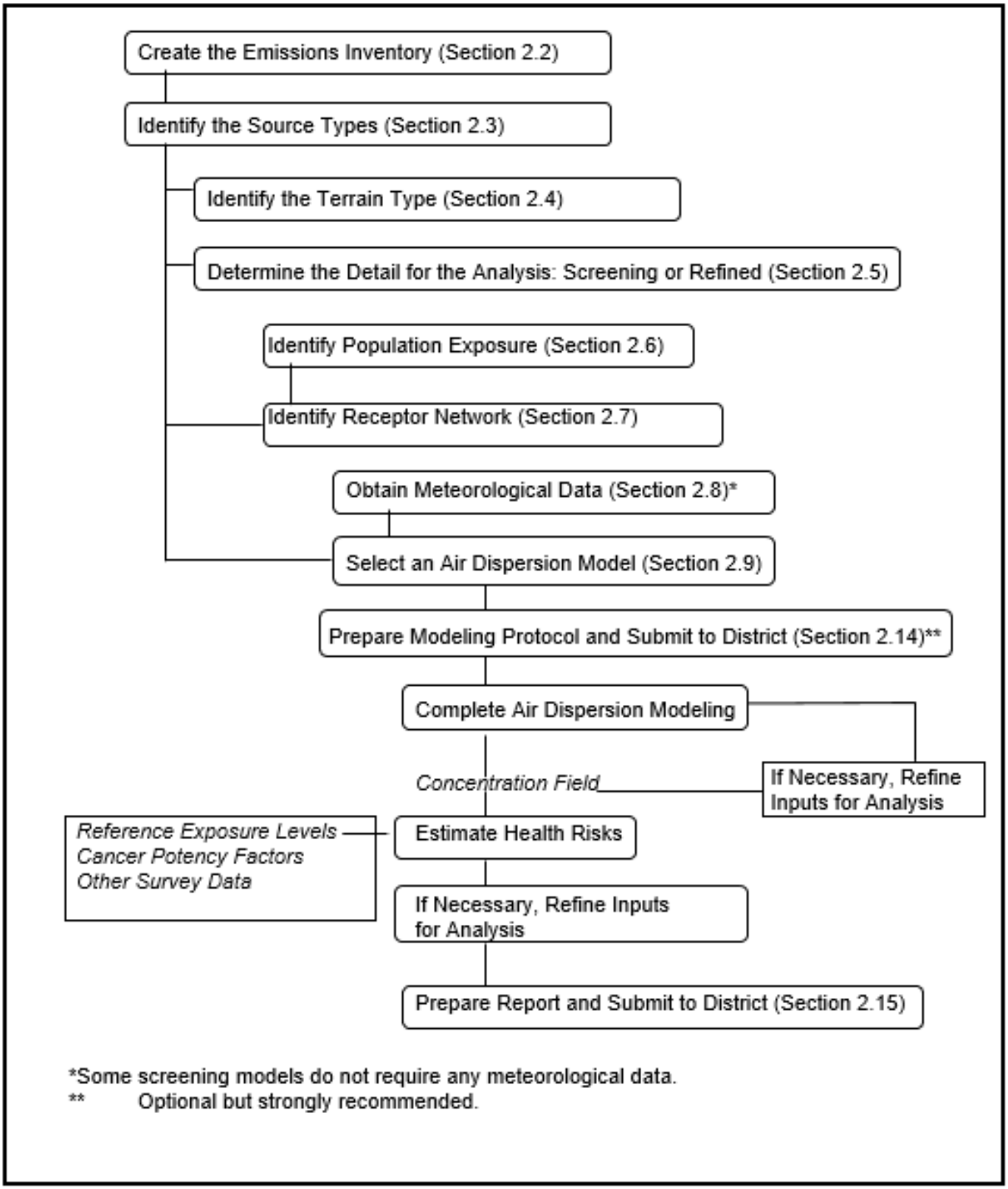
(10) Complete the air dispersion analysis

(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.



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

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

Report). The list of substances is compiled by the CARB for the Air Toxics "Hot Spots" Program.

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

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

(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 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 x 10^{-6}, 1 x 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.

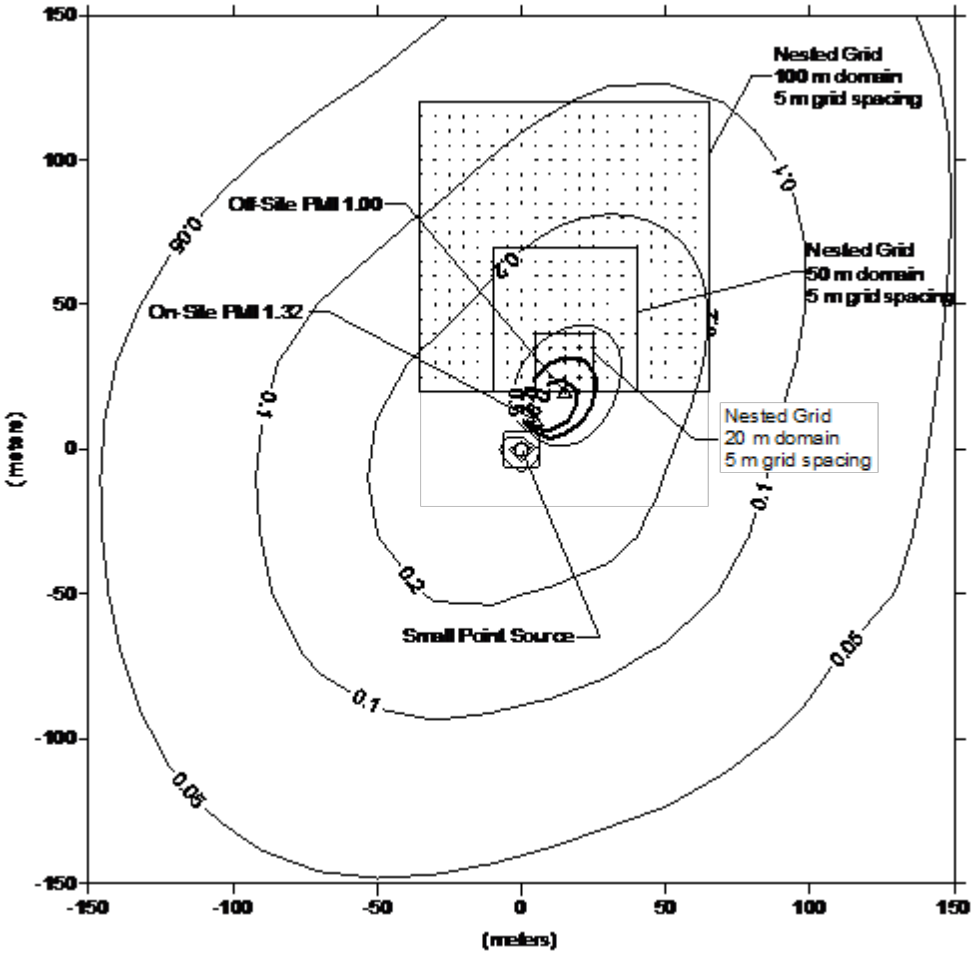
2.7.3 Spatial Averaging of Modeling Results

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. 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.

$$WAF = \frac{H_{residential}}{H_{source}} \times \frac{D_{residential}}{D_{source}} \qquad \text{Eq. 2.3}$$

Where:

WAF = the worker adjustment factor
Hresidential= 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
Dresidential = 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.

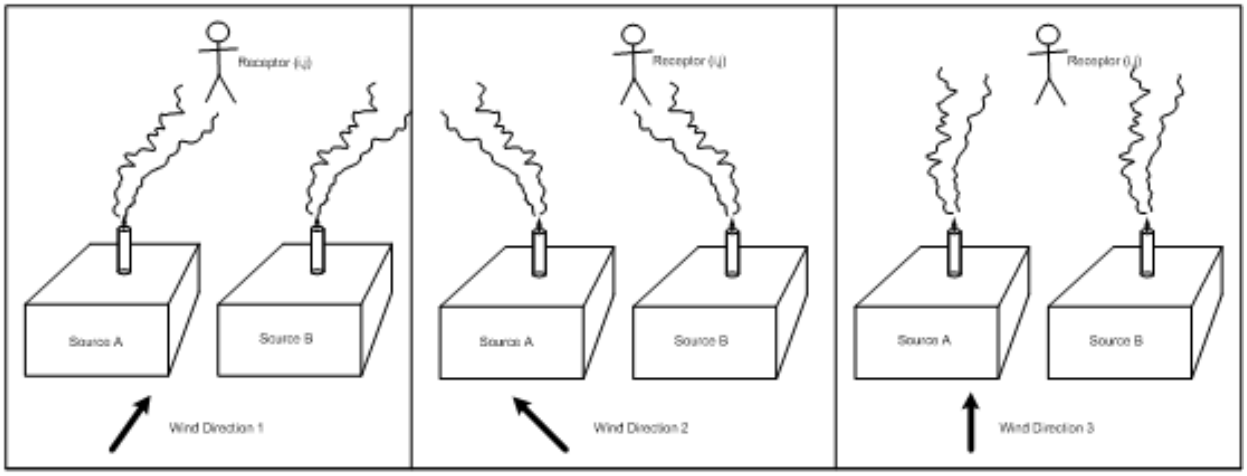
2.8.2 Modeling One-Hour Concentrations using Simple and Refined Acute Calculations

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: (I) 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.

e. 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.

f. 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.

g. 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).

h. 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)

i. 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).

j. 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.

k. 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.

l. 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.

m. 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

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Contents

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-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., 95^{th} 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

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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 95^{th} 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 95^{th} percentile value was 283 mg/day. Using the median of the 4 tracers, the mean was 7 mg/day and the 95^{th} 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 75^{th} 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

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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 $(mg/kg\text{-}day)^{-1}$.

$$RISKm_{(0<2\ yr)} = Dose_m * CPF * ASF * ED * 0.5 \quad \textbf{(Eq. 5-2)}$$

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:

$$RISK_{(lifetime)} = RISK_{(3rdtri)} + RISK_{(0<2\ yr)} + RISK_{(2<16\ yr)} + RISK_{(16\text{-}70yr)} \quad \textbf{(Eq. 5-3)}$$

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:

$$RISK_{(9\text{-}yr\ residency)} = RISK_{(3rdtri)} + RISK_{(0<2\ yr)} + RISK_{(2<9\ yr)} \quad \textbf{(Eq. 5-4)}$$

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

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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:

$$BSA\ (m^2) = (Wt^{0.51456}) \times (Ht^{0.42246}) \times 0.02350 \qquad \textbf{(Eq. 6-10)}$$

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 5^{th} 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 5^{th} 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

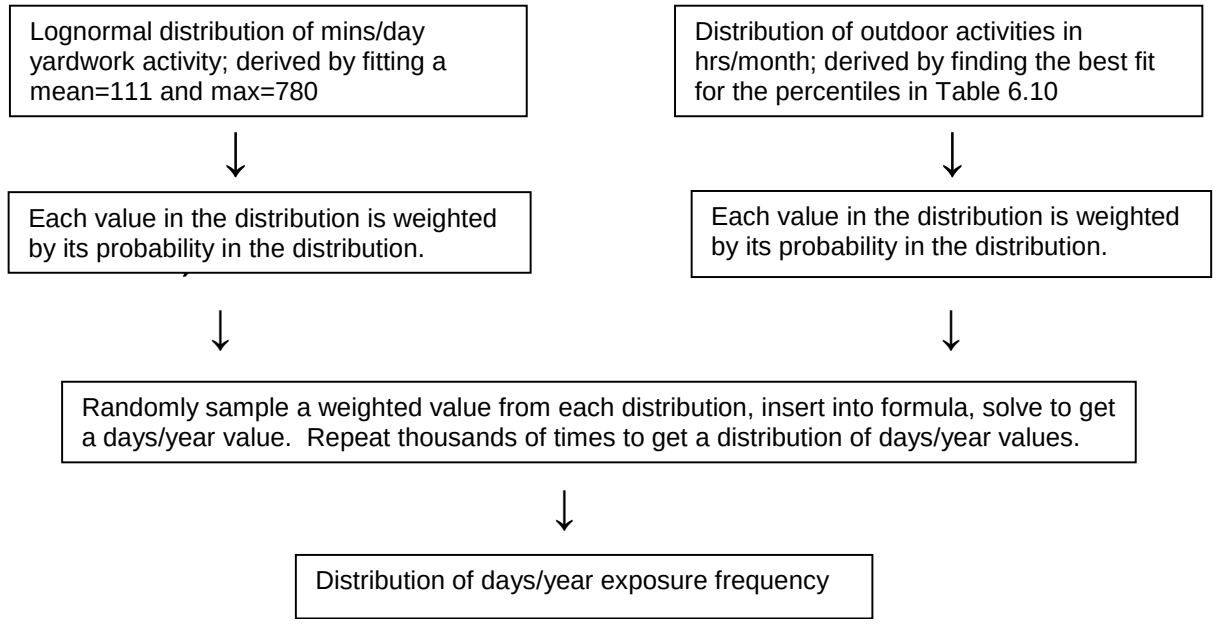
OEHHA chose to use the first three of the Garlock et al. activities (gardening, other yardwork, and team sports) for estimating soil contact frequency of adults. Using Monte Carlo simulation in Crystal Ball (Decisioneering, 2008), OEHHA calculated the best fit distribution for exposure frequency in hours/month 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)

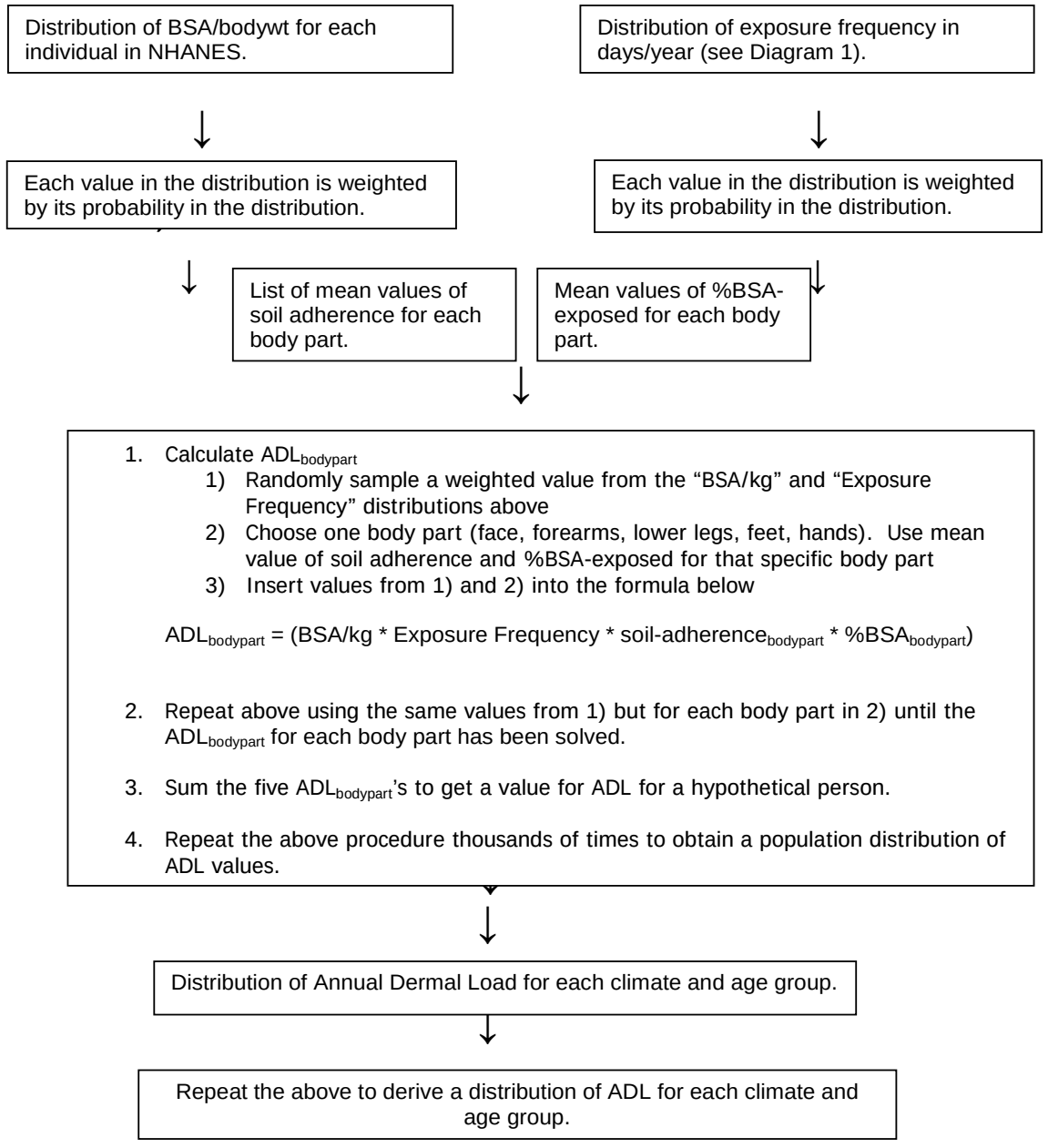
As explained in Section 6.3, OEHHA created a new variate, "annual dermal load", or ADL, which is a composite of the body surface area (BSA) per kg body weight, exposure frequency, and soil adherence variates. Point estimates from the composite "annual dermal load" can be used for point estimate assessments 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):

$$SFS_{adj} = \frac{(SA_{1-6})(AF_{1-6})(ED_{1-6})}{(BW_{1-6})} + \frac{(SA_{7-31})(AF_{7-31})(ED_{7-31})}{(BW_{7-31})} \quad \textbf{Eq. 6-14}$$

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

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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)[a]

Food Category	Third Trimester[b]		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
Pork	0.9	2.9	2.9	10.5	2.2	7.8
Poultry	1.8	4.7	4.5	11.4	3.7	9.0
Milk	5.4	15.9	50.9	116.1	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	5.5	16.6	1.9	5.9	1.8	5.6
Leafy	1.7	5.8	0.9	3.2	1.1	3.4
Protected	3.6	10.6	1.7	5.8	1.6	5.2
Root	3.0	8.7	1.7	4.6	1.5	4.2
Meat						
Beef	3.0	7.6	2.0	4.8	1.7	4.4
Pork	1.8	5.7	0.9	2.9	0.9	2.8
Poultry	3.0	7.5	1.8	4.7	1.5	3.8
Milk	16.5	48.4	5.4	15.9	4.3	13.2
Eggs	3.1	8.1	1.6	4.2	1.3	3.4

[a] April 22, 2022: Transcription errors in Table 7.1 (in Chapter 7) were corrected. In the original Table 7.1, data from Table 7.12 were incorrectly copied onto the "Ages 2<16" column. The corrected Table 7.1 replaces the data for this age group with data from Table 7.11 and replaces the column header "Ages 2>16" with "Ages 2<16". Additionally, the corrected Table 7.1 also switches the order of meat types in the Food Category column to reflect the order shown in the source data tables (Tables 7.8 – 7.13).

[b] 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

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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.

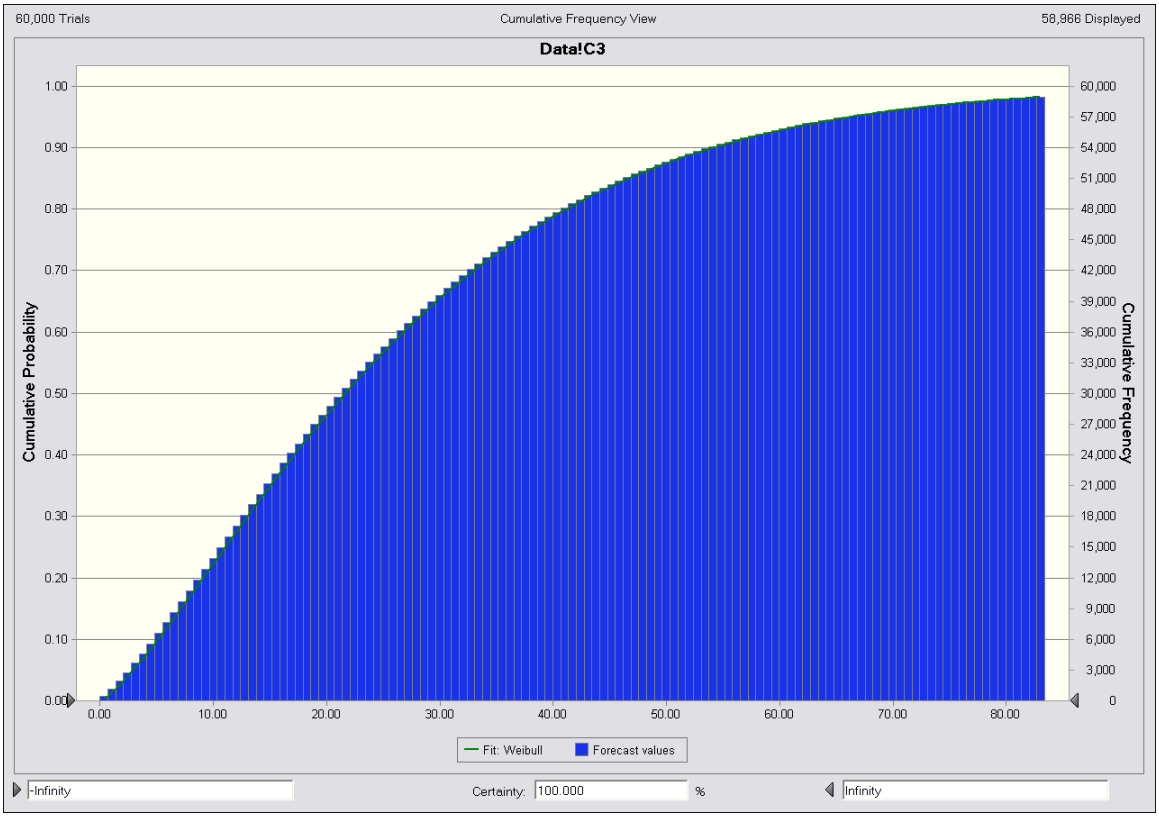
Table 8.14 Recommended Distributions of OEHHA Derived Water Intake Rates for Stochastic Analysis (ml/kg-day)

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

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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 95^{th} 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)

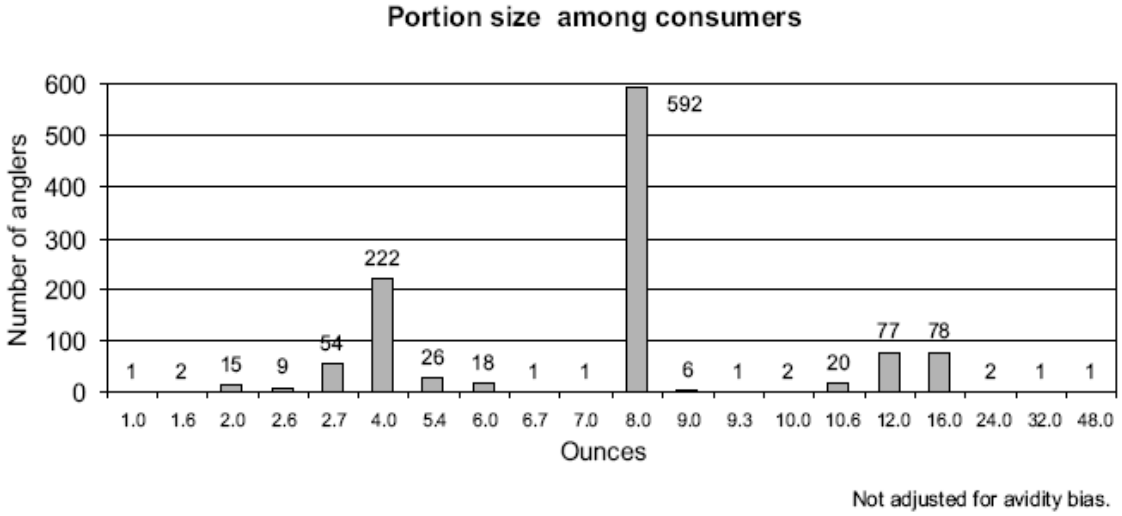
9.8.2.2 Influence of Interview Decliners on the Fish Consumption Rate

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.

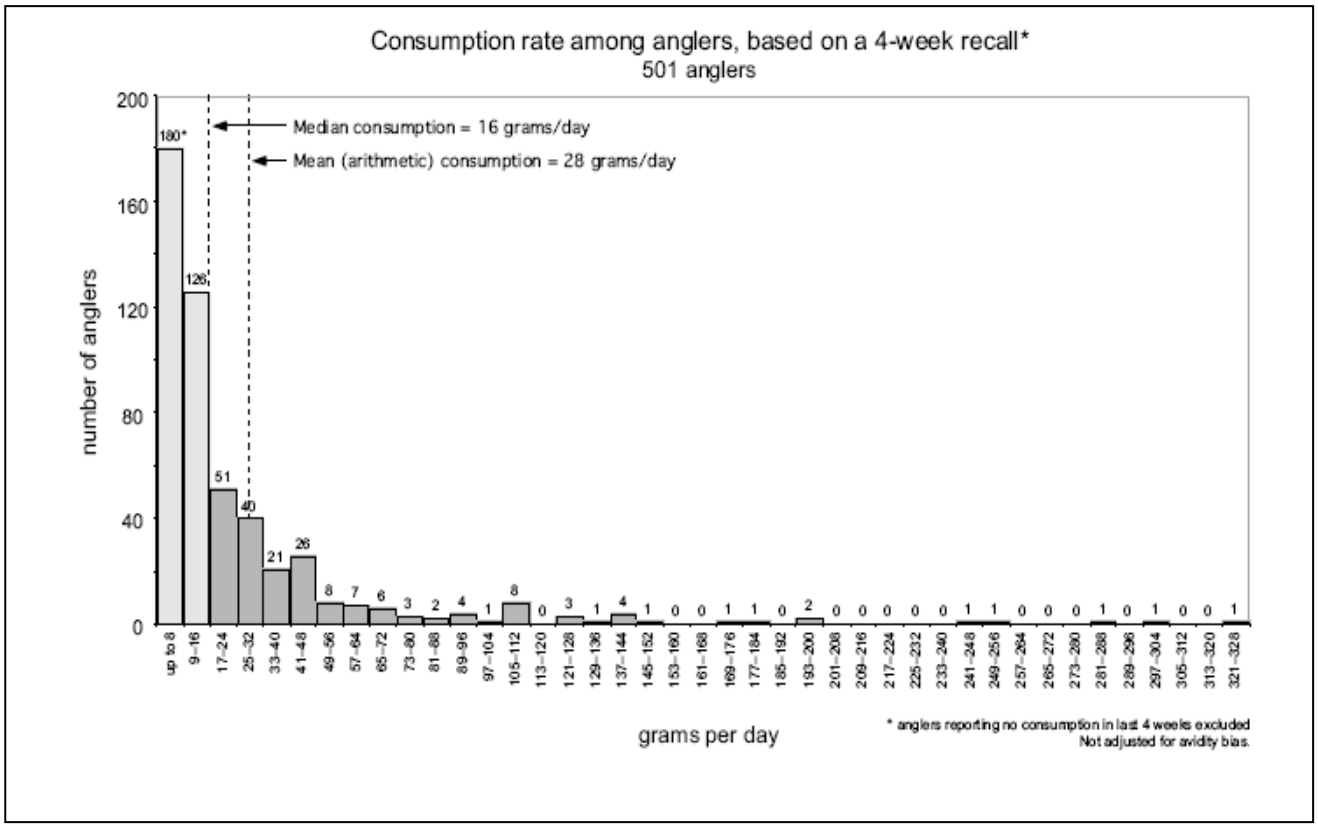
Figure 9



(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)

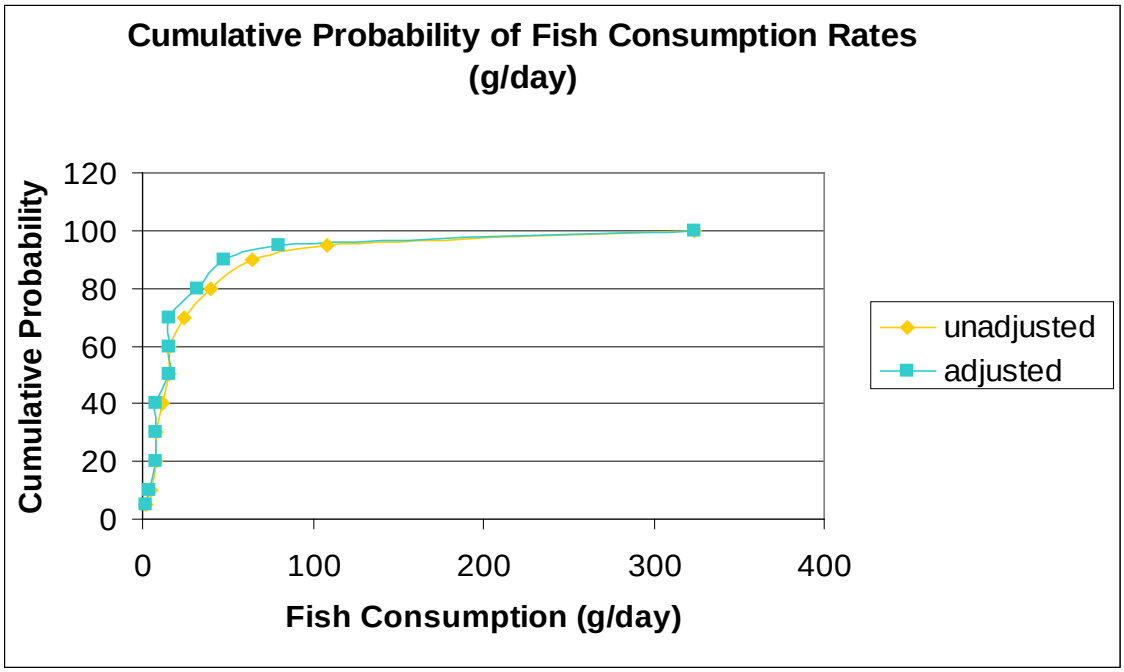
The cumulative empirical distribution curves for the rate of fish consumption for all anglers who caught Bay fish in the SF Bay survey, both unadjusted and adjusted for avidity bias, are shown in Fig. 9-3. The fish consumption rate distribution is highly skewed to the right with a long upper tail, characteristic 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



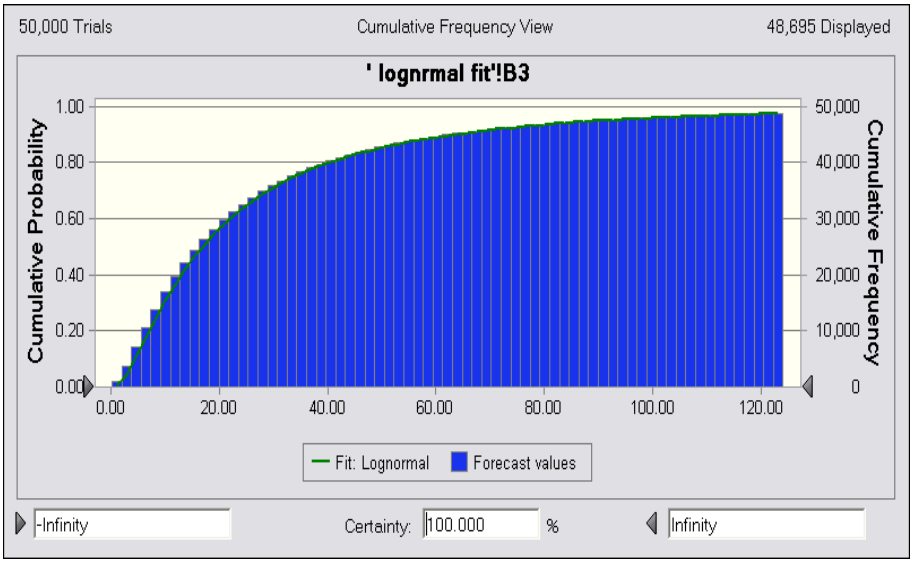
Data source: SFEI (2000)

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



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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

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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 1×10^{-6}, 1×10^{-5}, and/or 1×10^{-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.

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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

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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
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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.

Contents

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

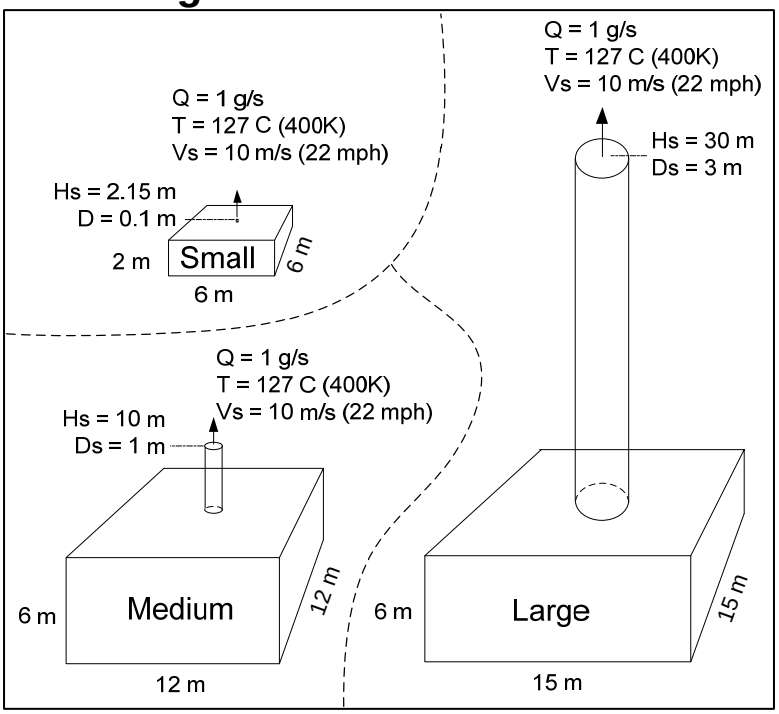


Table 1 – Point Source (Stack) Modeling Parameters

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

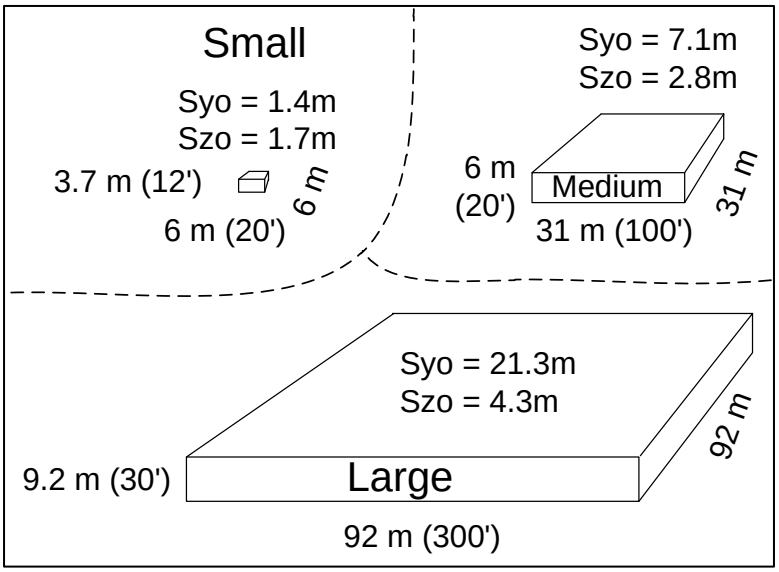


Table 2 – Volume Source Modeling Parameters					
Source Size	Qs (g/s)	Hs (m)	Syo (m)	Szo (m)	Similar Sources
Large	1	4.6	21.3 (L=92m)	4.3	Fleet Facility (300'x300'x30')
Medium	1	3.0	7.1 (L=31m)	2.8	(100'x100'x20')
Small	1	1.8	1.4 (L=6m)	1.7	Dry Cleaner (20'x20'x12')

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

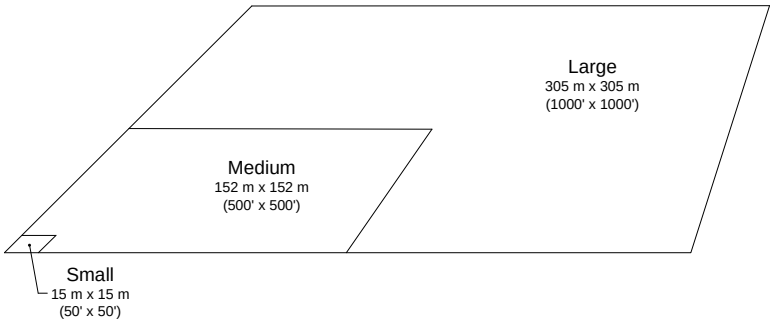


Table 3 – Area Source Modeling Parameters				
Source Size	Qs (g/s)	Hs (m)	Ls (m)	Similar Sources
Large	1	3.0	305	Rail Facility (1000'x1000')
Medium	1	3.0	152	Industrial Loading Facility (500'x500')
Small	1	2.0	15	Pile (50'x50')

Figure 4 – Line Source – Large and Small

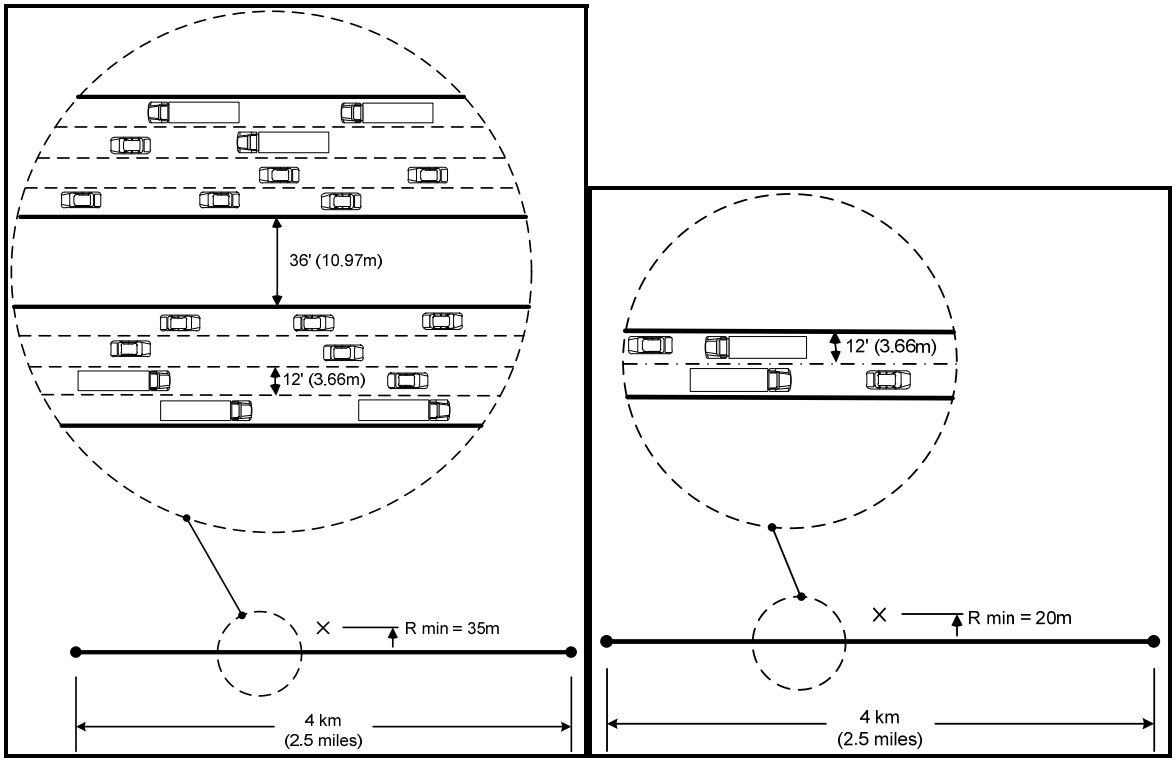


Table 4 – Line Source Modeling Parameters					
Source Size	Qs (g/s)	Vehicles per Day	Lanes	Ls (m)	Min Receptor Placement (m)
Large	1	250,000	8	4000	35
Small	1	5,000	2	4000	20

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.

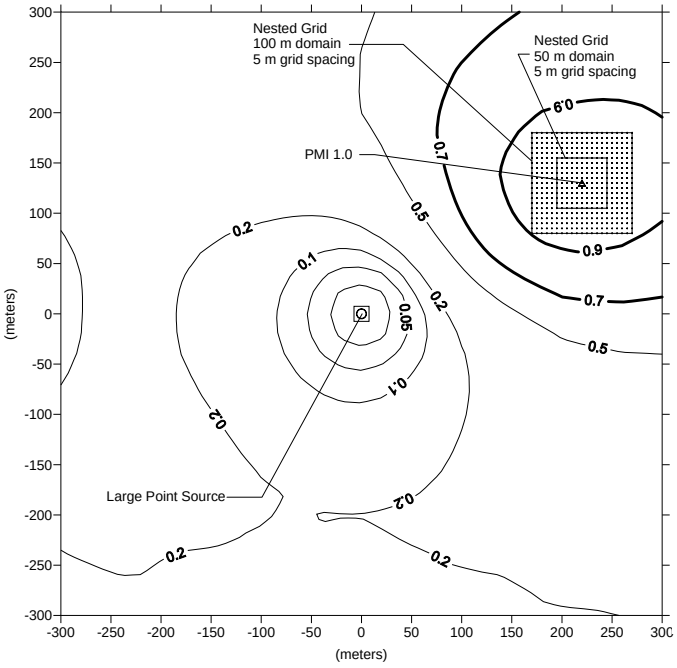
Appendix C-3 shows the PMI and two nested grids for each source (point, volume, area, and line) and for all sizes.

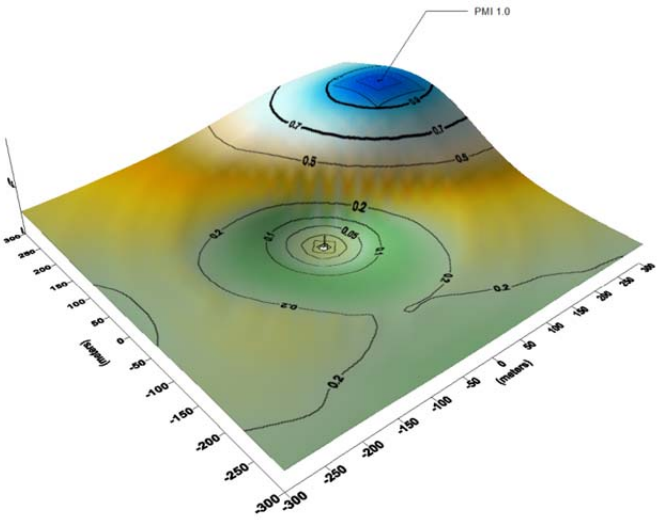
The spatial average was calculated for nested grids at ten different domains; 10m x 10m up to 100m x 100m, even though only two nested grids are shown on each plot.

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

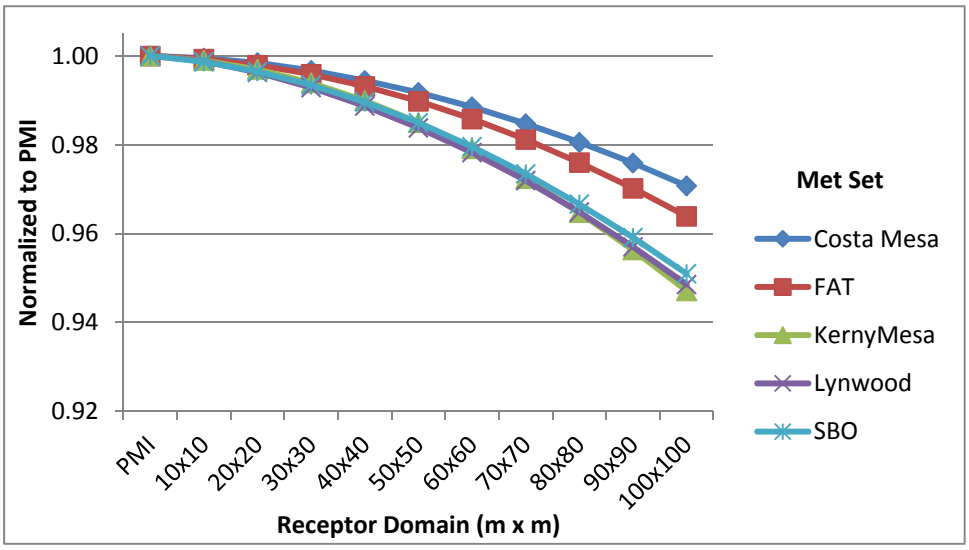


Figure 7.2
Medium Point Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

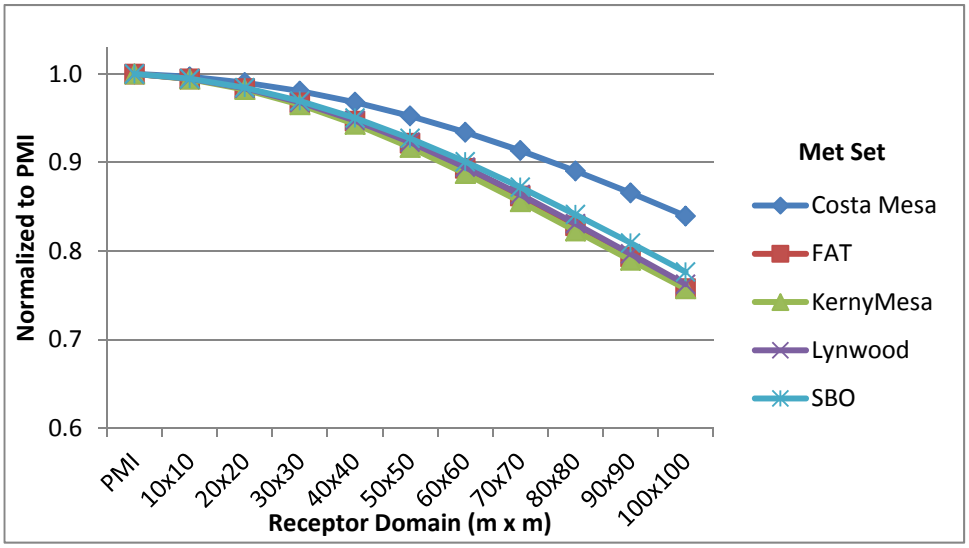


Figure 7.3
Small Point Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

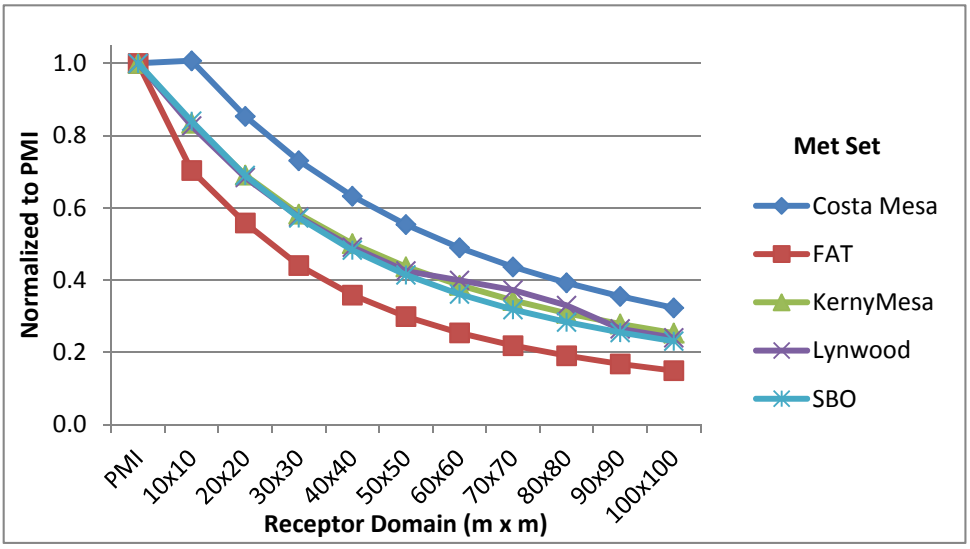


Figure 8.1
Large Volume Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

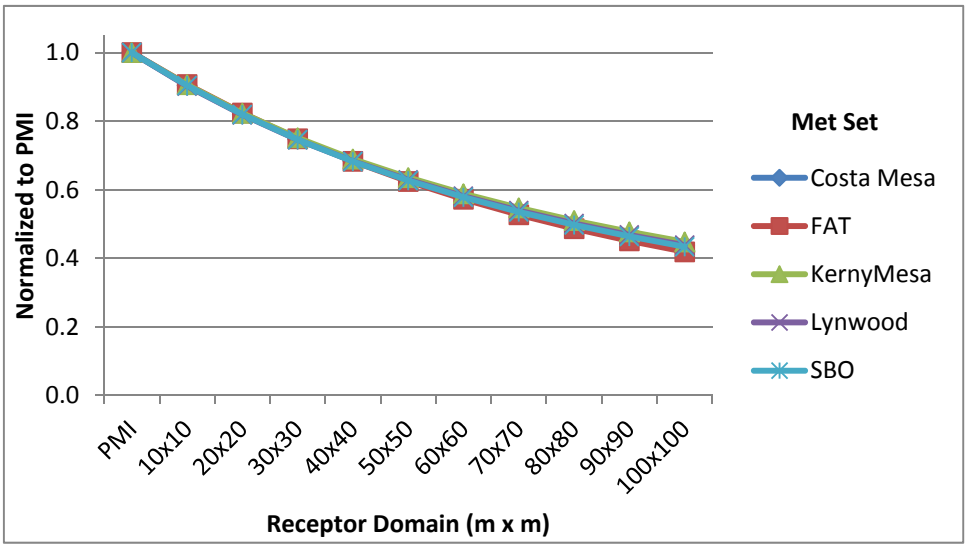


Figure 8.2
Medium Volume Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

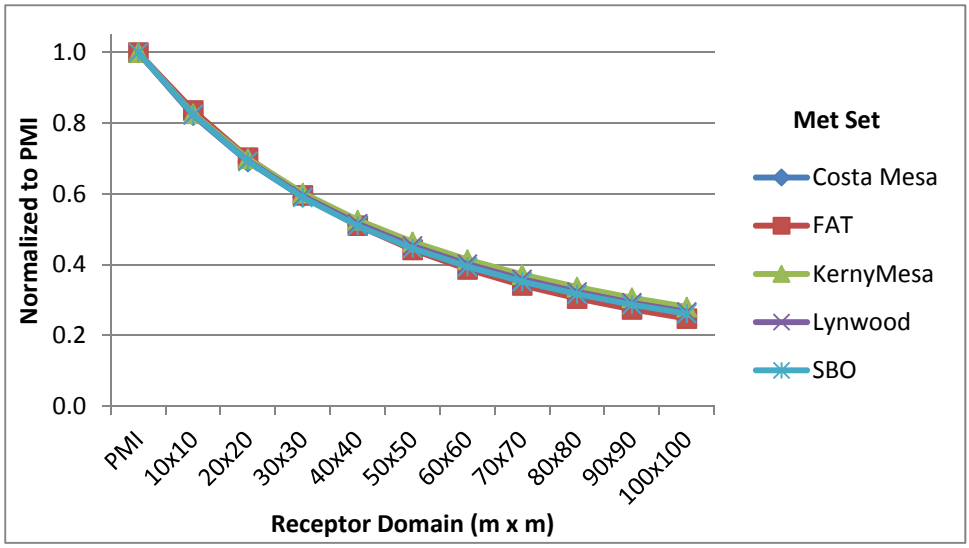


Figure 8.3
Small Volume Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

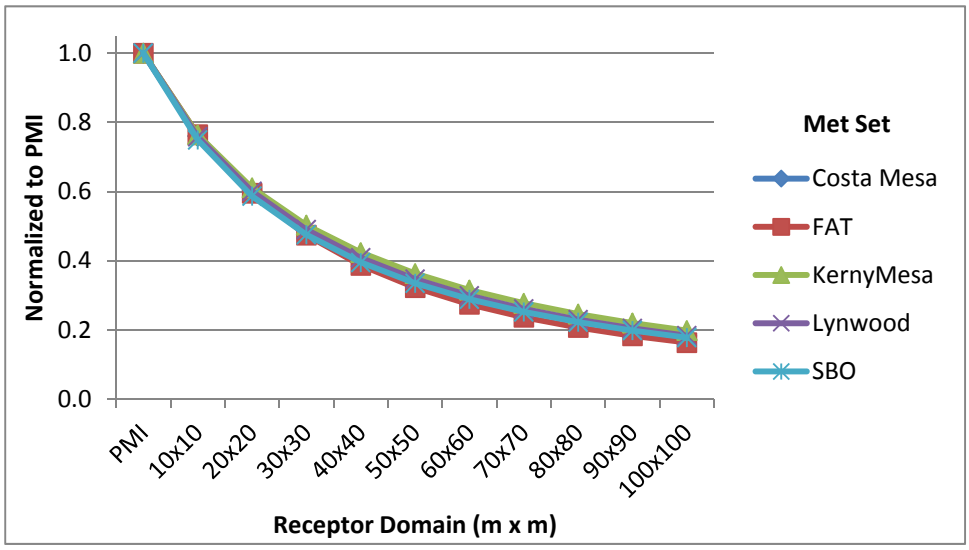


Figure 9.1
Large Area Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

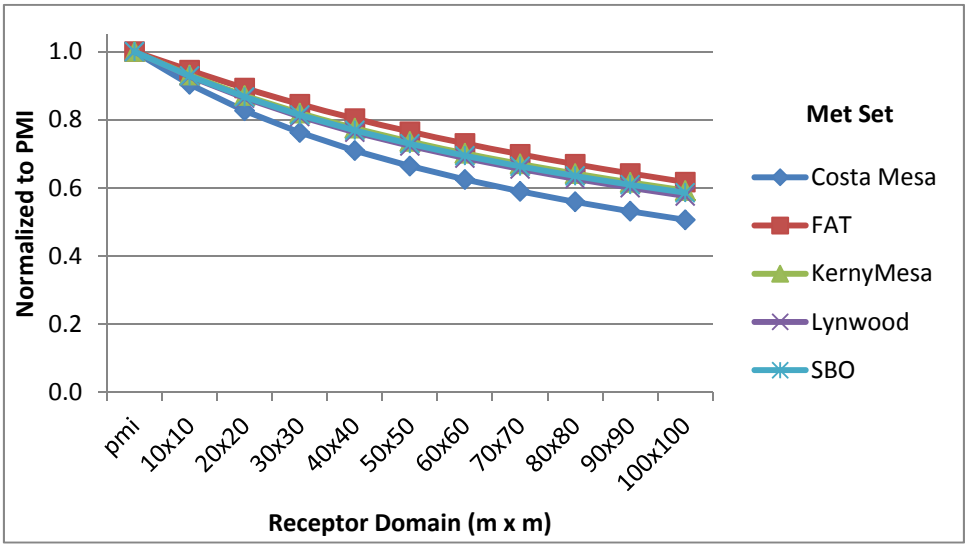


Figure 9.2
Medium Area Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

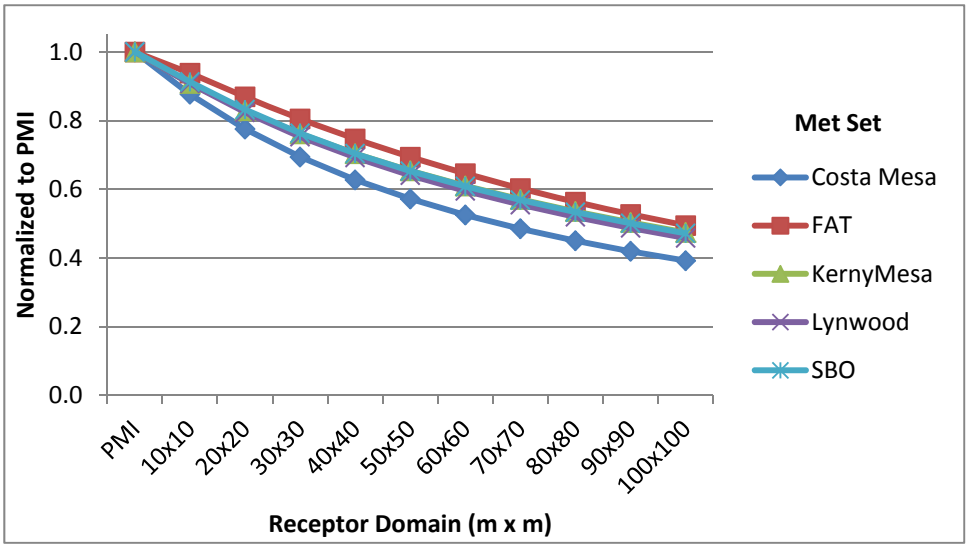


Figure 9.3
Small Area Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

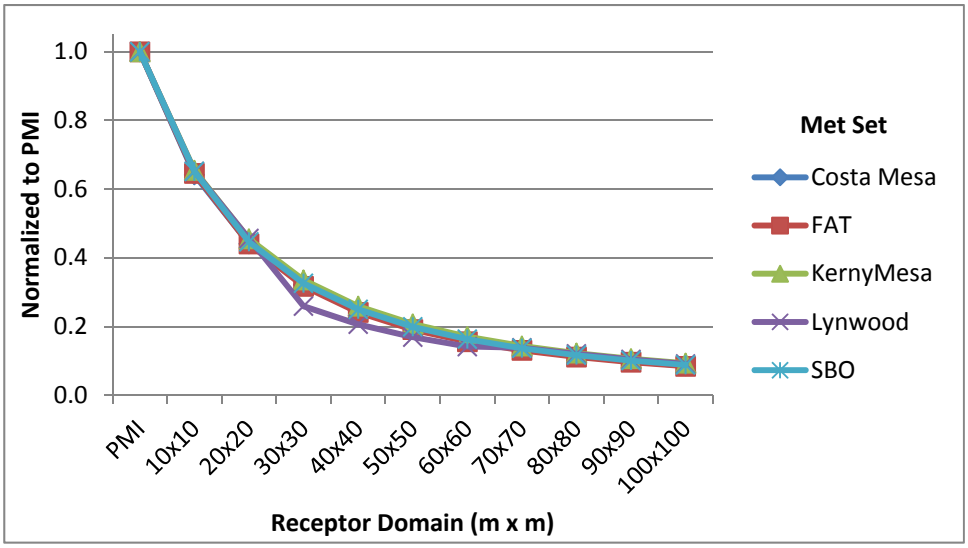


Figure 10.1
Large Line Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets

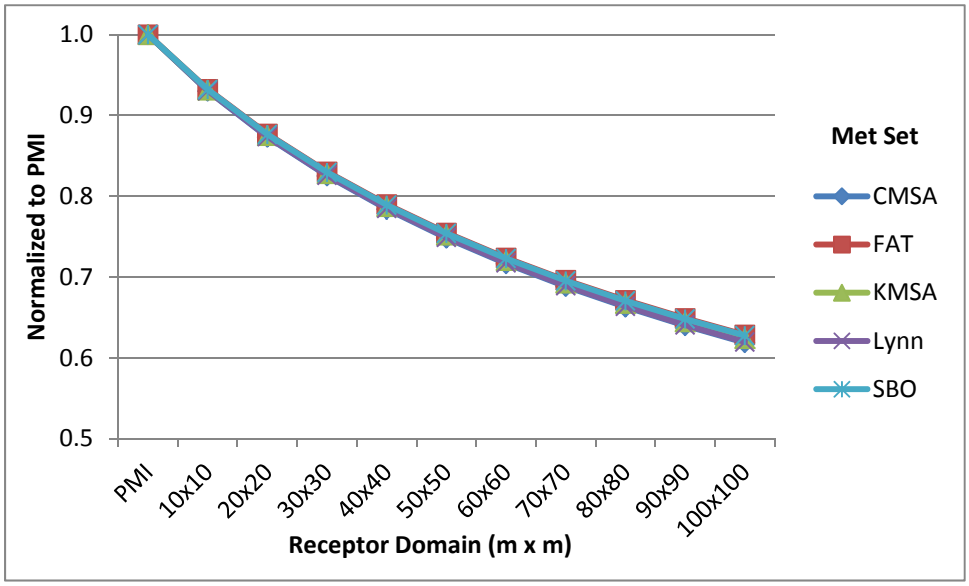
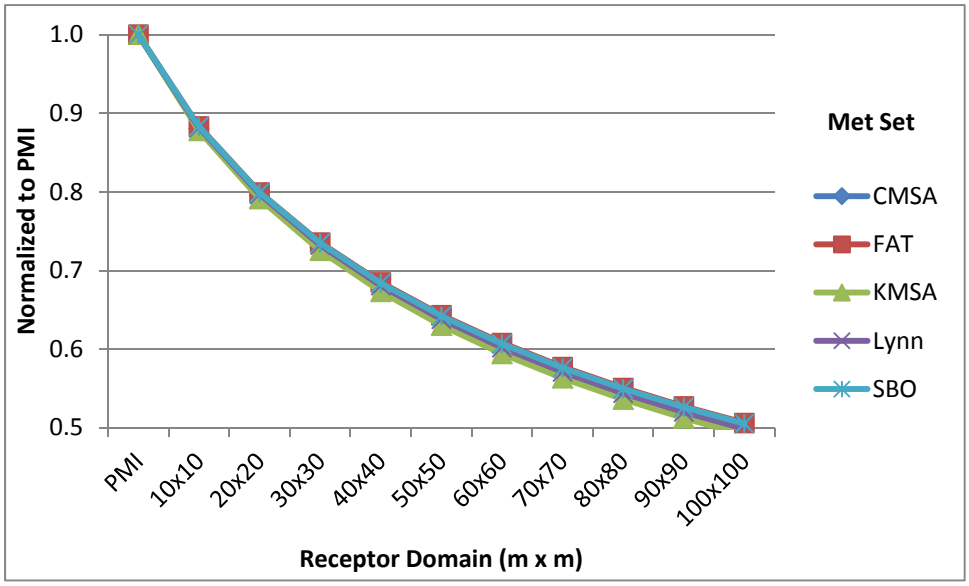
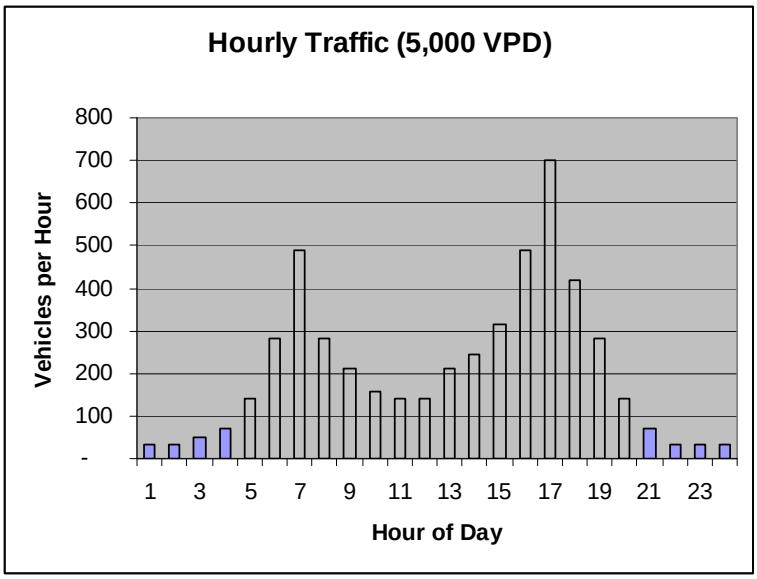


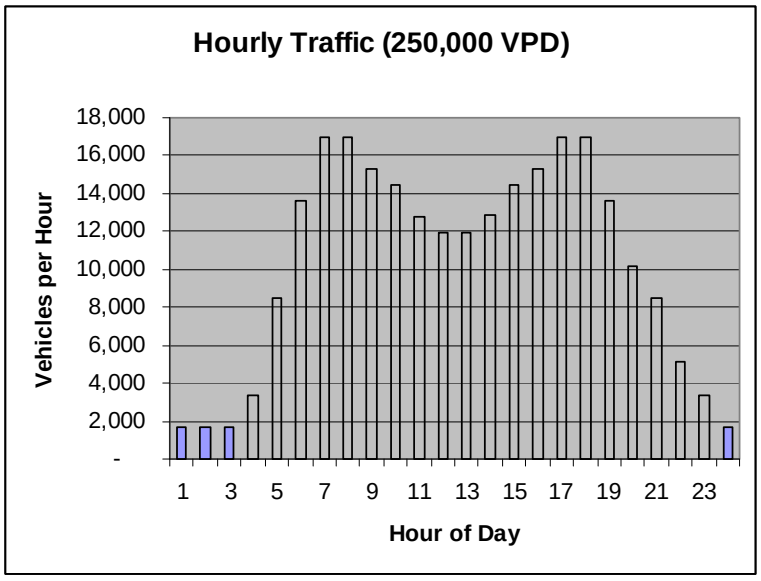
Figure 10.2
Small Line Source Spatially Averaged GLCs with Several Domain Sizes and Five Meteorological Data Sets



Appendix C-1 – Hourly Variation for Traffic Line Source

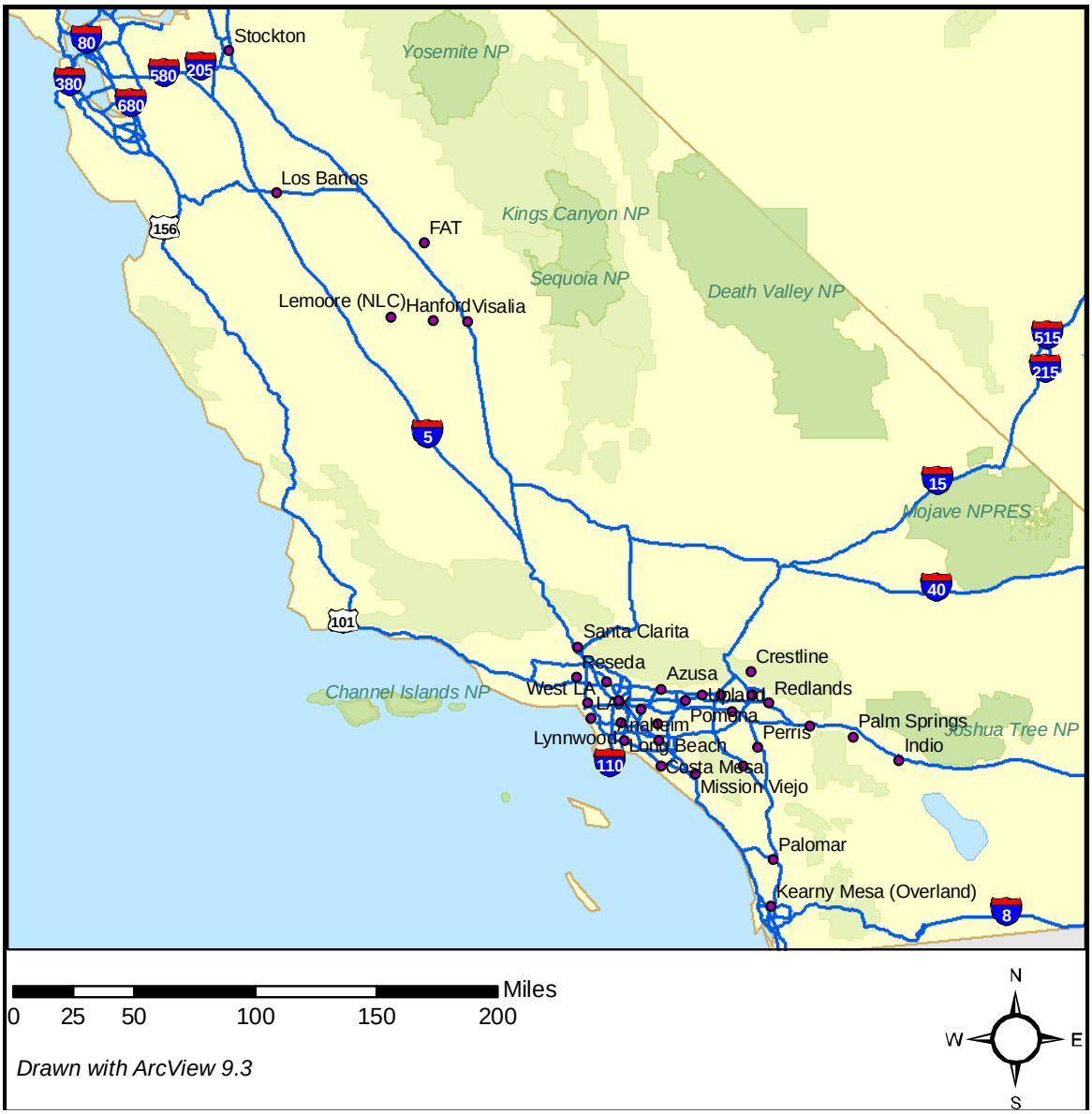
Hour	5K VPD	250K VPD
1	35	1,700
2	35	1,700
3	49	1,700
4	70	3,400
5	140	8,500
6	280	13,600
7	490	17,000
8	280	17,000
9	210	15,300
10	156	14,450
11	140	12,750
12	140	11,900
13	210	11,900
14	245	12,850
15	315	14,450
16	490	15,300
17	700	17,000
18	420	17,000
19	280	13,600
20	140	10,200
21	70	8,500
22	35	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

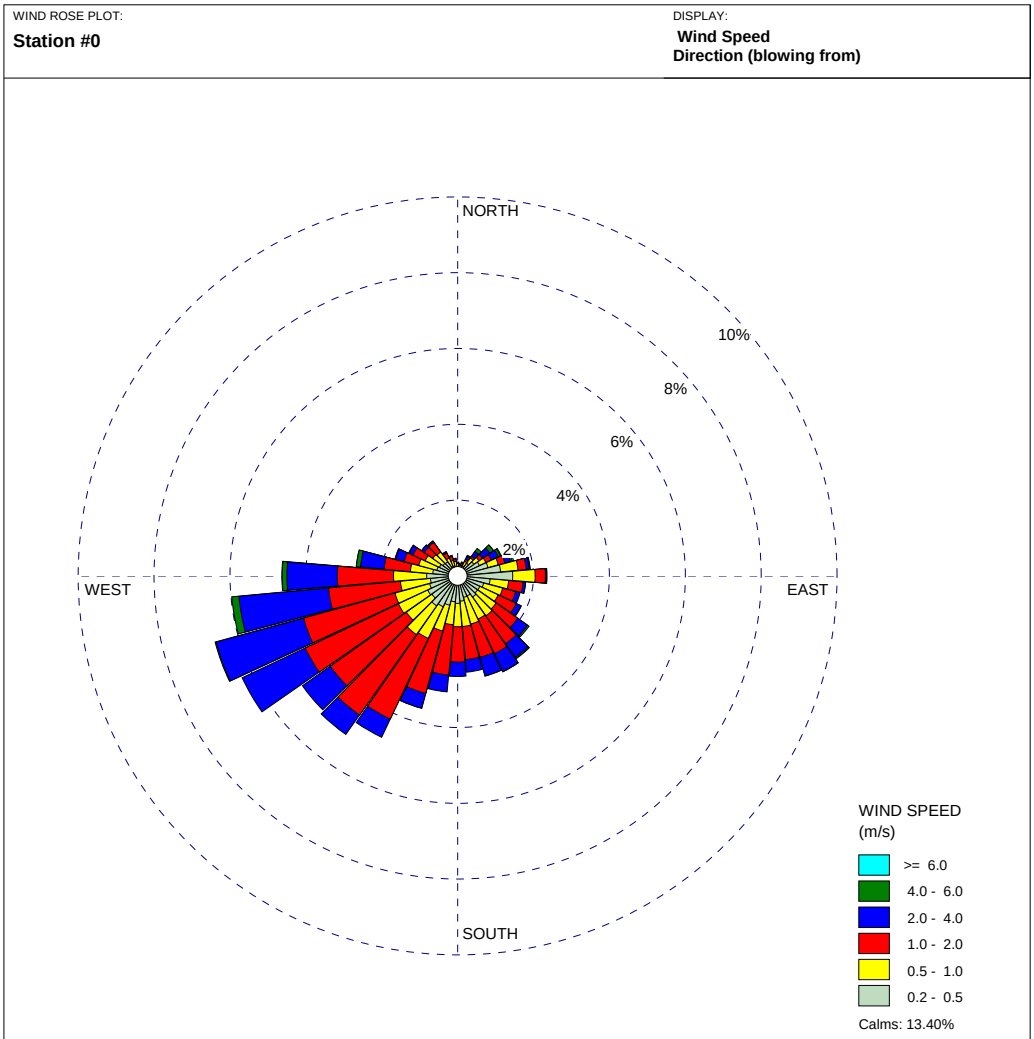


Figure AP C-2.3 – Fresno Air Terminal – Wind Rose Summary

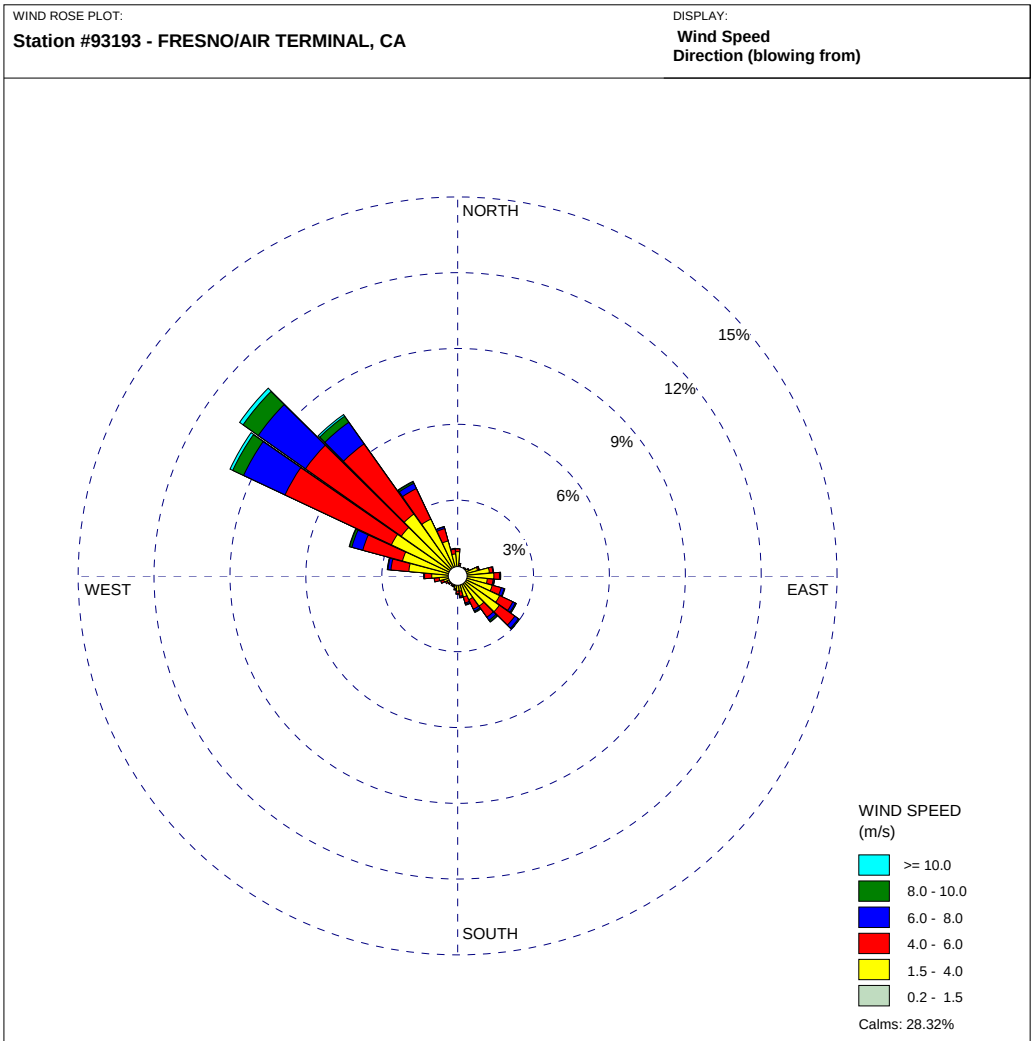


Figure AP C-2.4 – Kearny Mesa – Wind Rose Summary

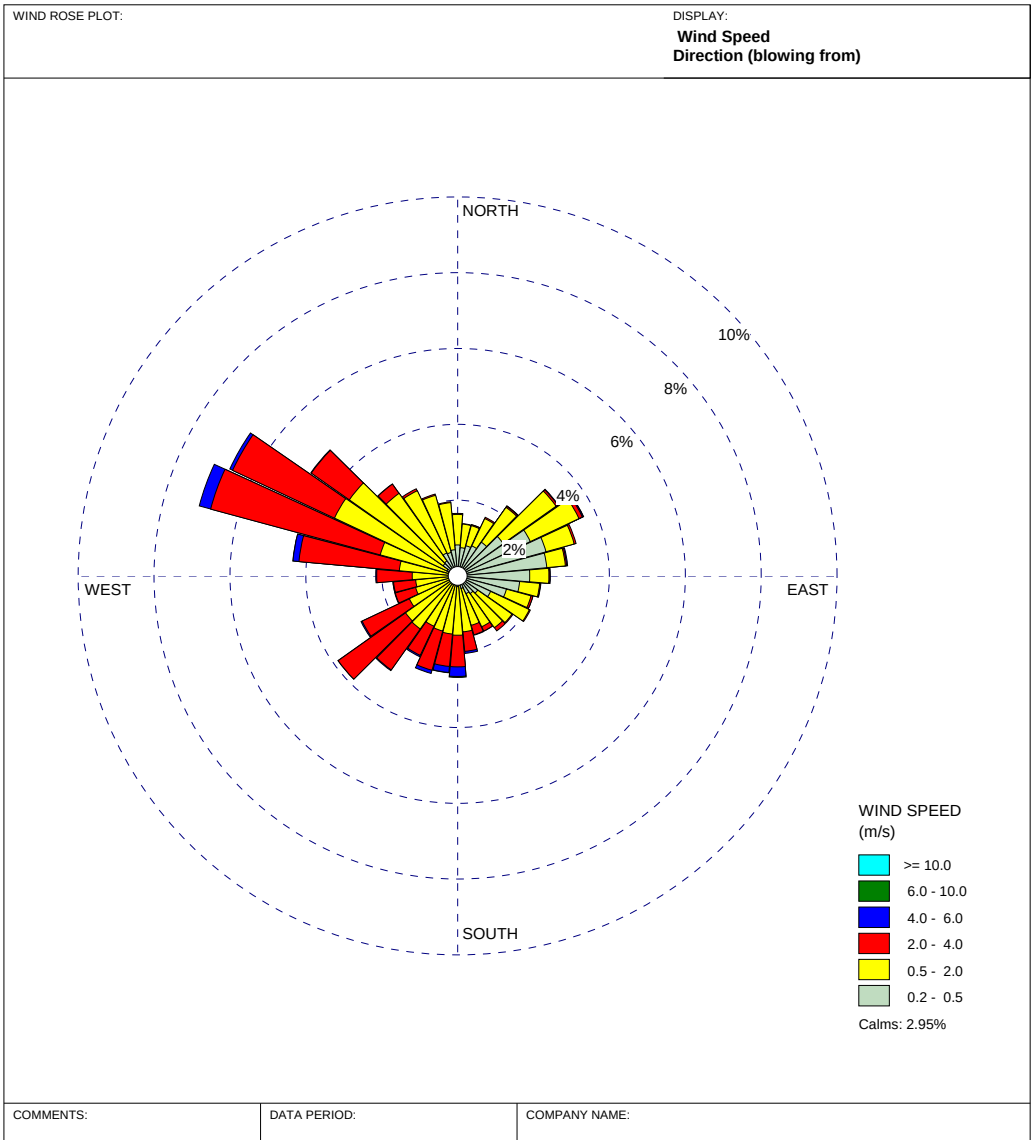


Figure AP C-2.5 – Lynwood – Wind Rose Summary

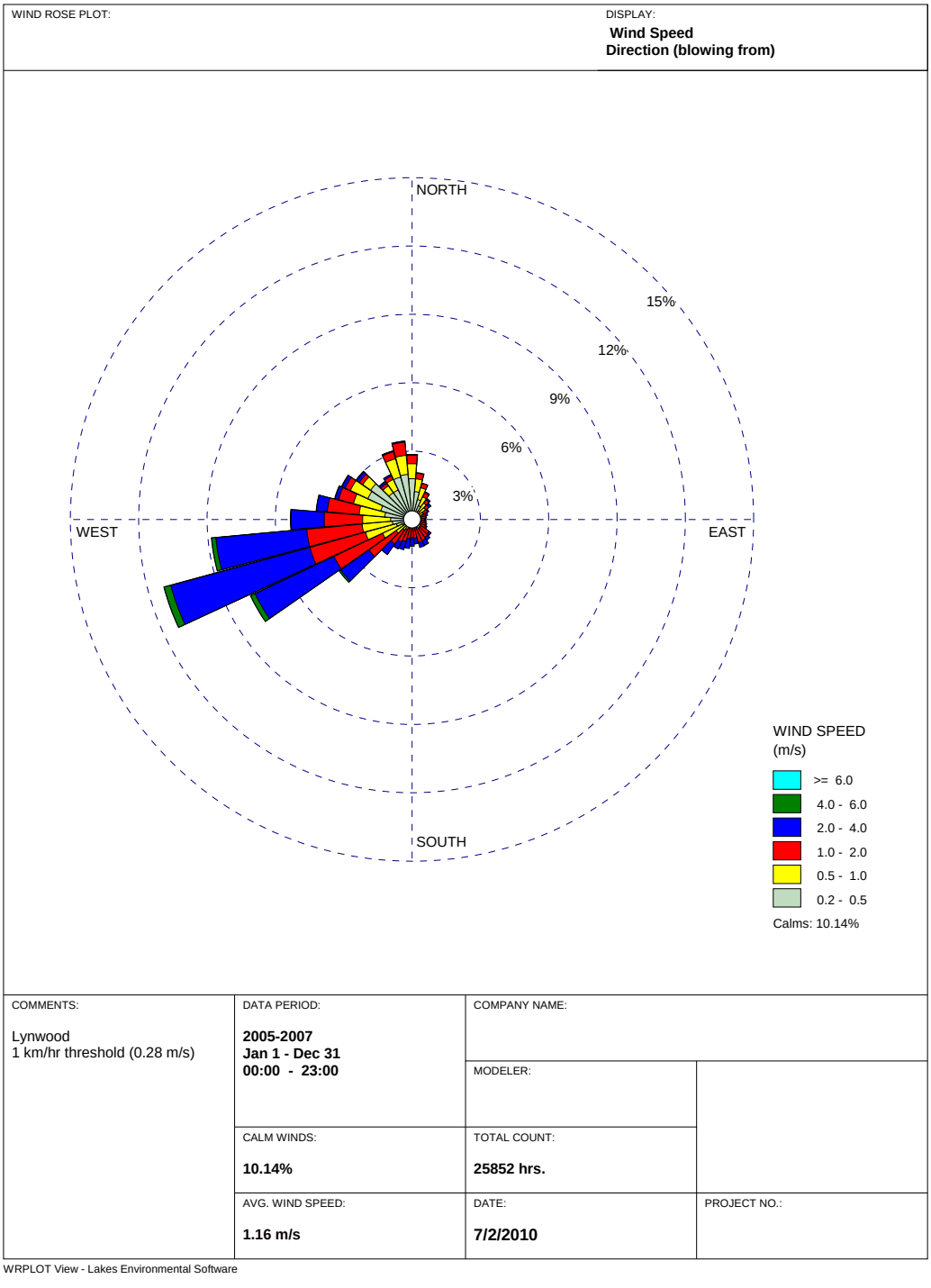
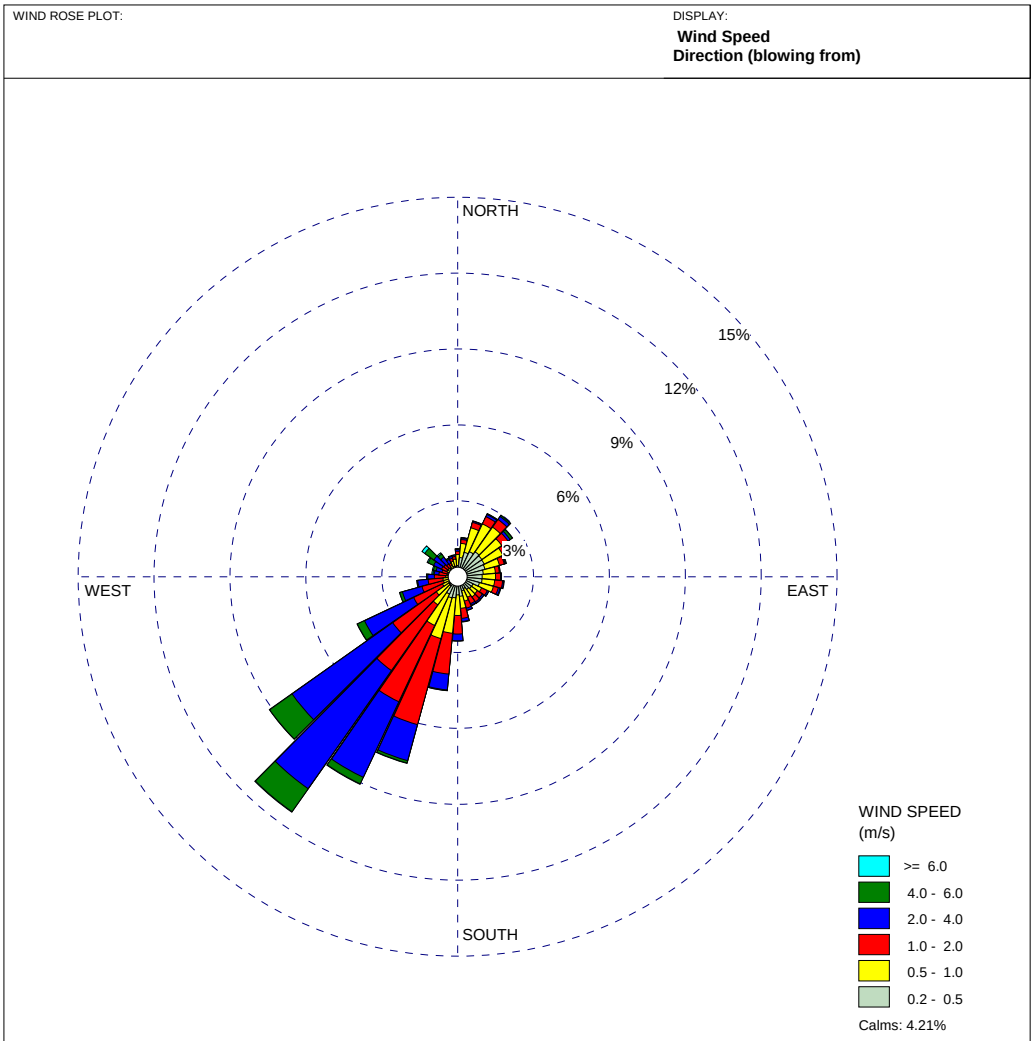


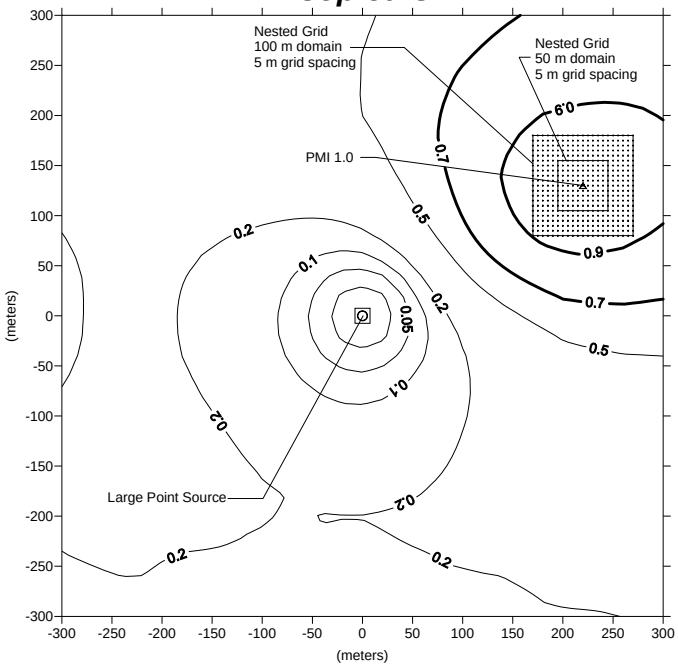
Figure AP C-2.6 – San Bernardino – Wind Rose Summary



Appendix C-3 – Sources, Receptors, Concentrations

Figure AP C-3.1.1 – Large Point Source – Costa Mesa

Isopleths



Surface

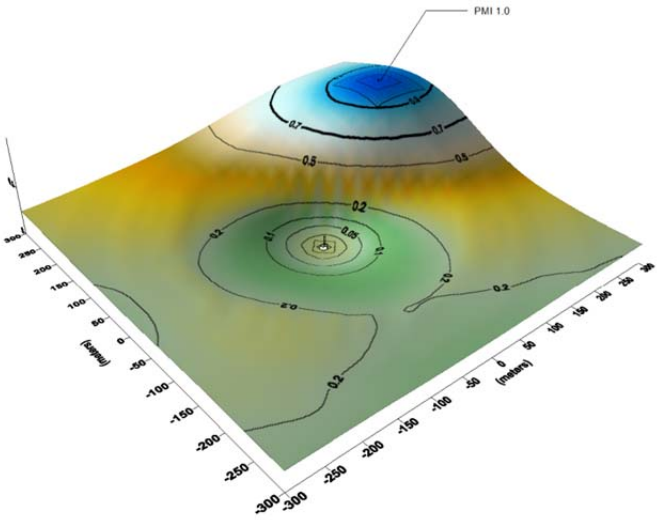
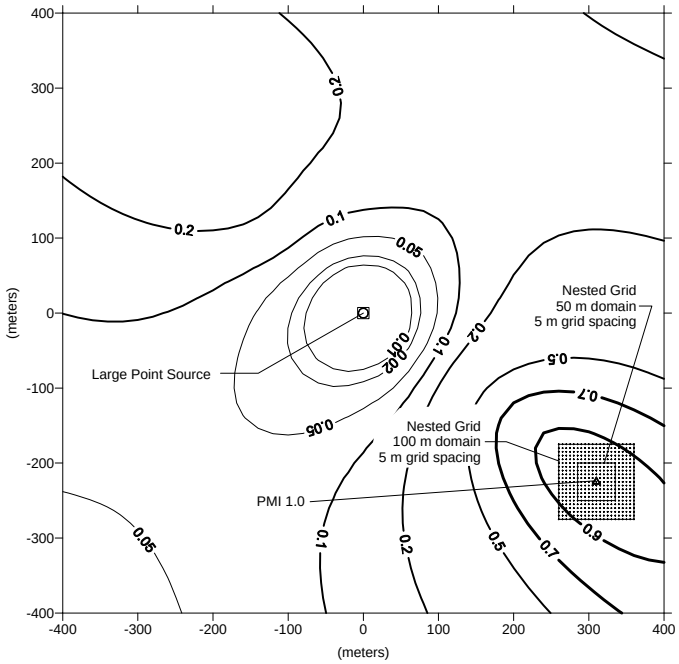


Figure AP C-3.1.2 – Large Point Source – Fresno Air Terminal

Isopleths



Surface

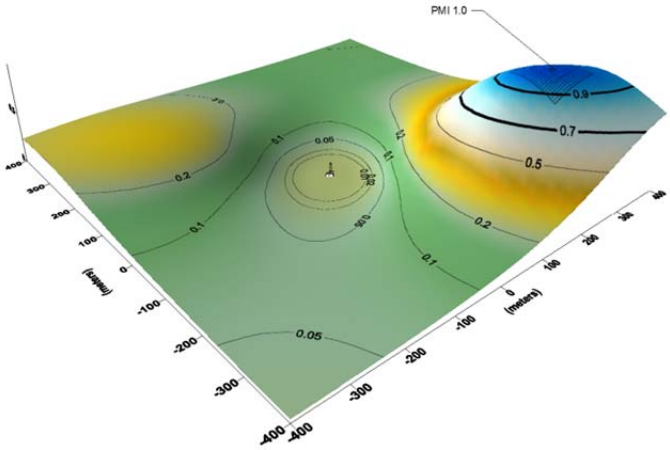


Figure AP C-3.1.3 – Large Point Source – Kearny Mesa

Isopleths ***Surface***

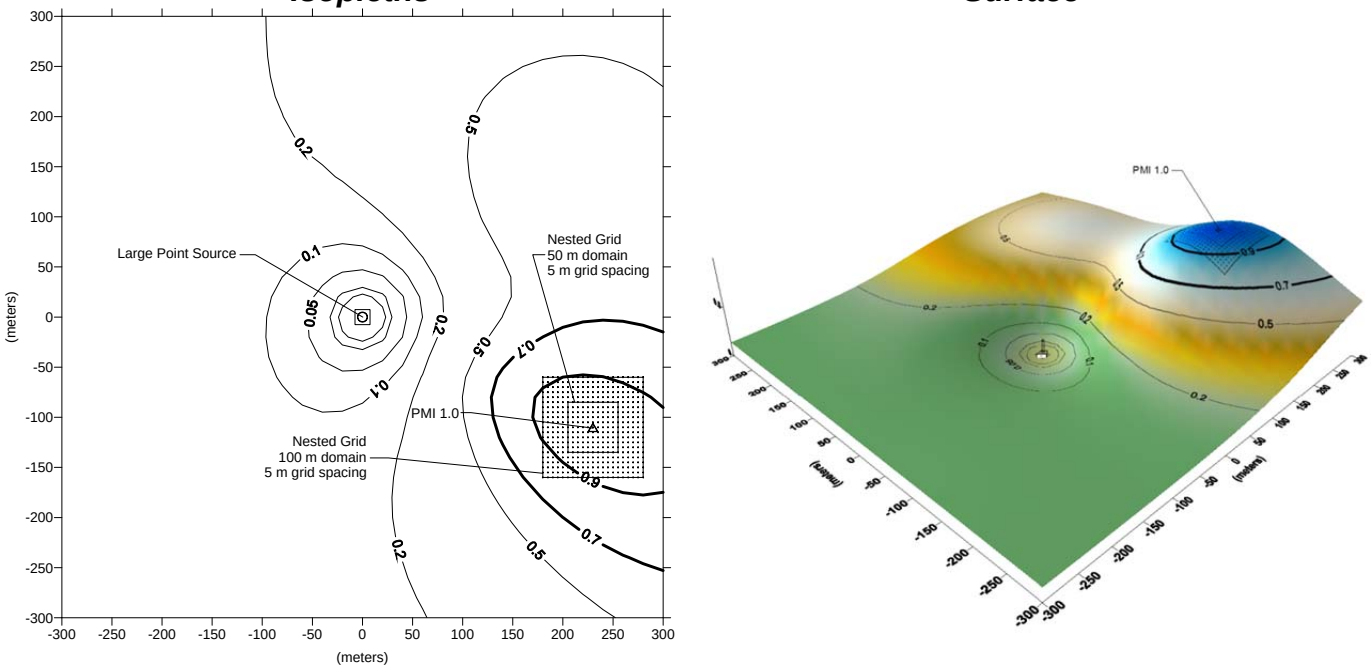


Figure AP C-3.1.4 – Large Point Source – Lynwood

Isopleths ***Surface***

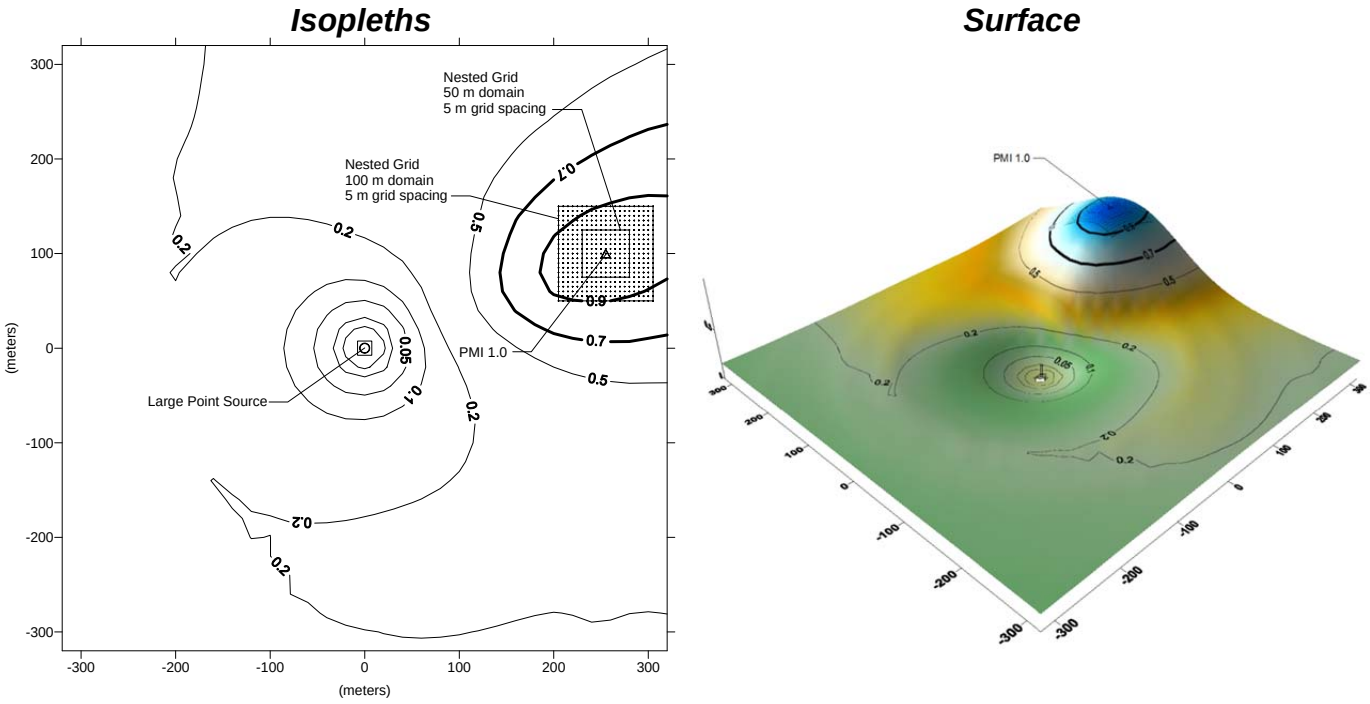
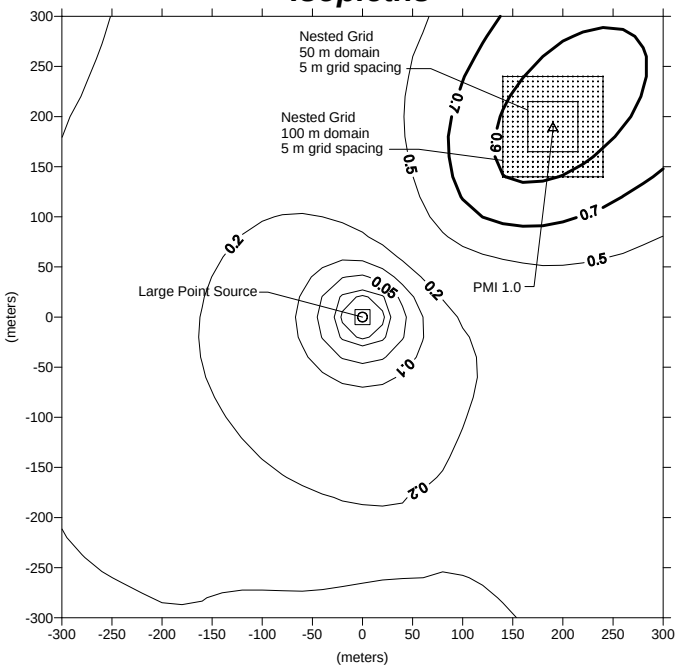


Figure AP C-3.1.5 – Large Point Source – San Bernardino

Isopleths



Surface

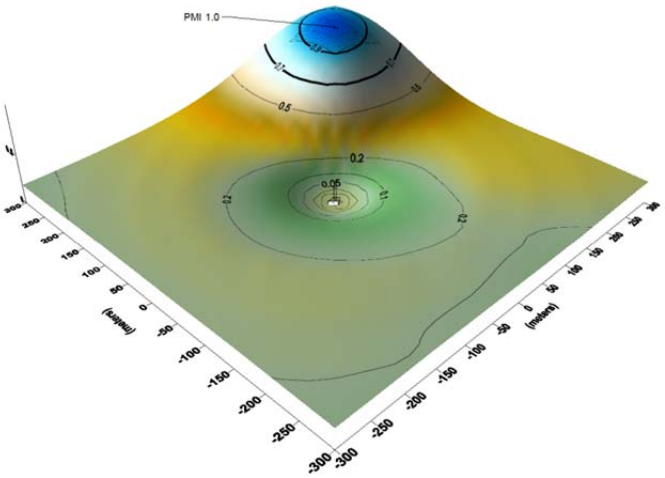


Figure AP C-3.2.1 – Medium Point Source – Costa Mesa

Isopleths ***Surface***

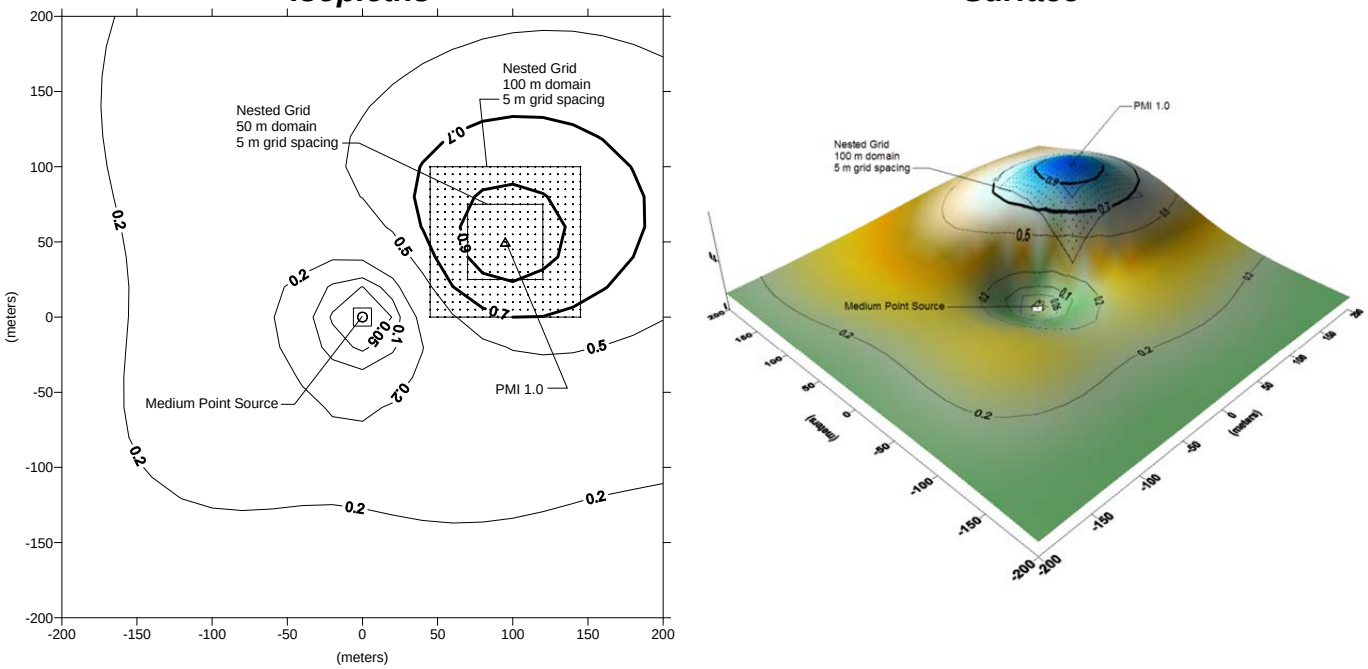


Figure AP C-3.2.2 – Medium Point Source – Fresno Air Terminal

Isopleths ***Surface***

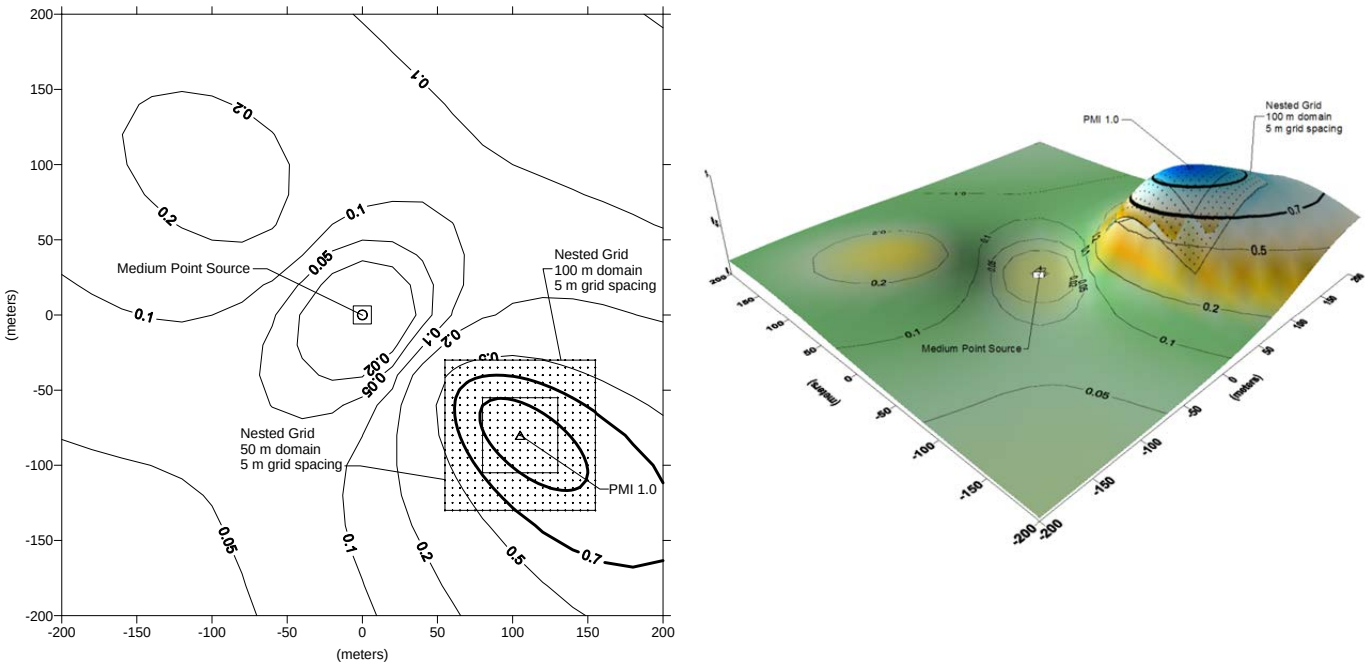
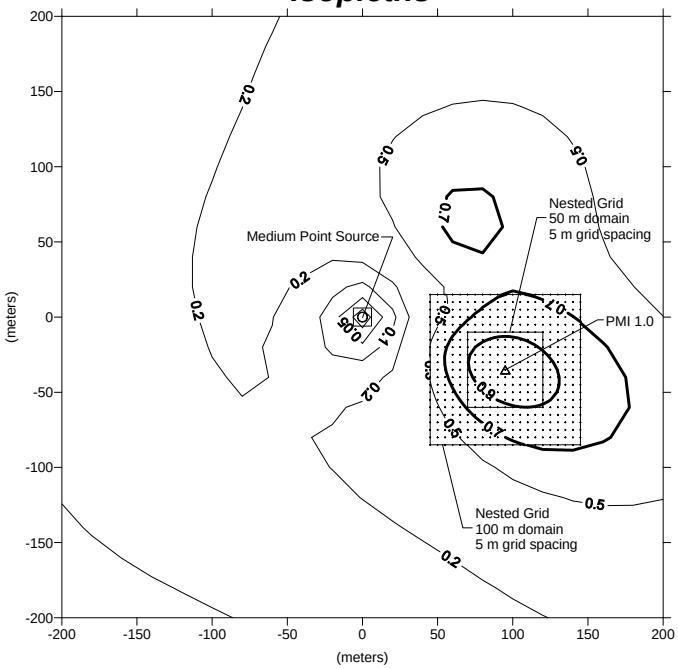


Figure AP C-3.2.3 – Medium Point Source – Kearny Mesa

Isopleths



Surface

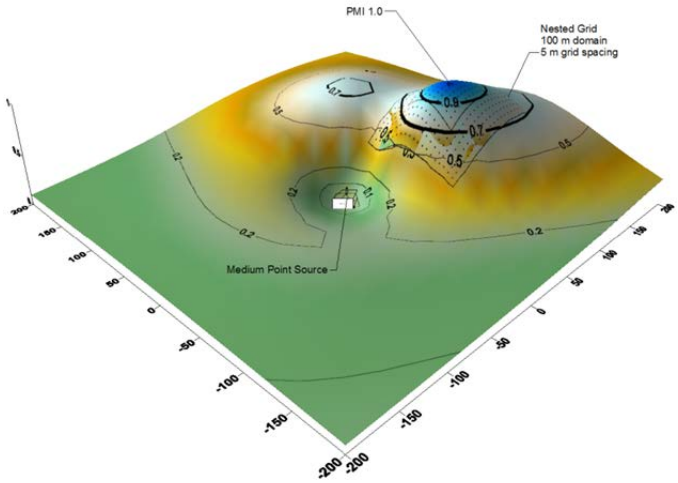
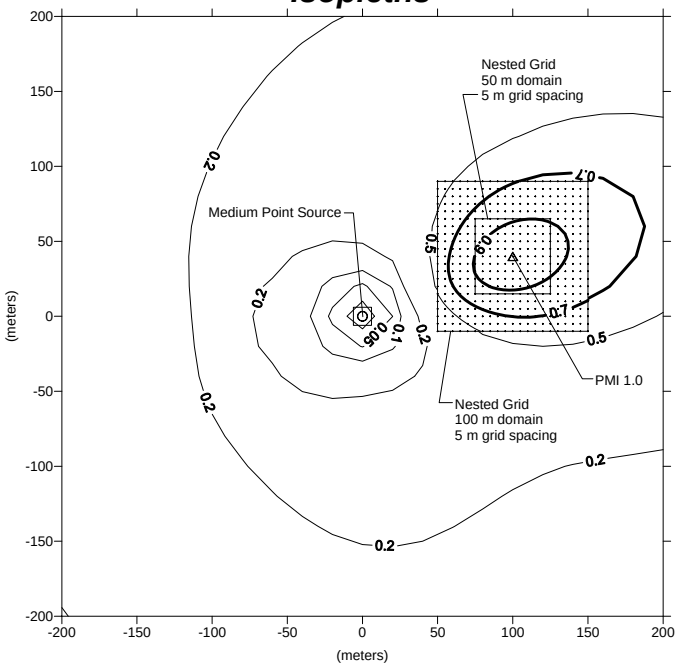


Figure AP C-3.2.4 – Medium Point Source – Lynwood

Isopleths



Surface

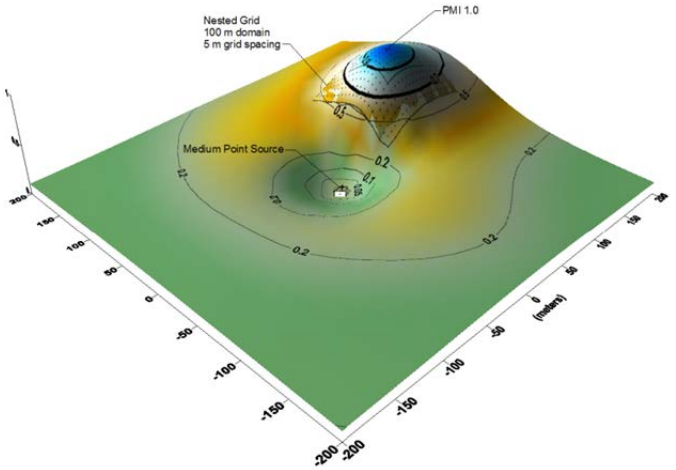
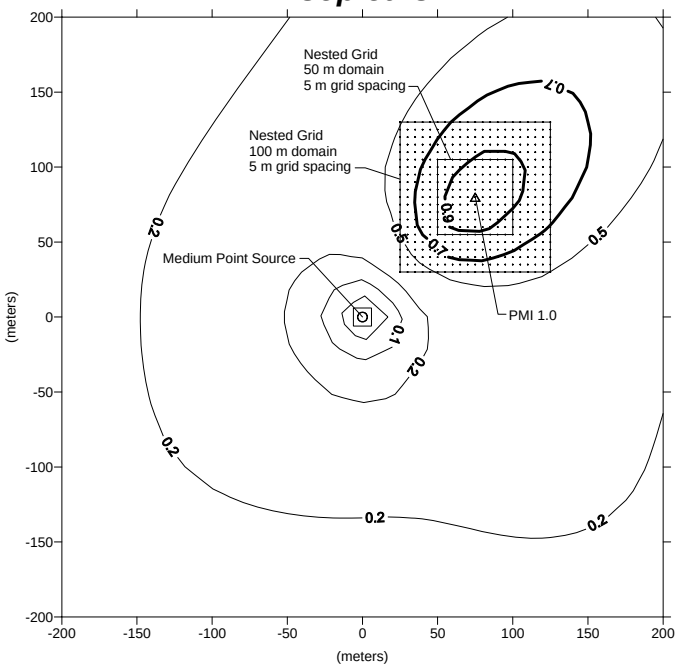


Figure AP C-3.2.5 – Medium Point Source – San Bernardino

Isopleths



Surface

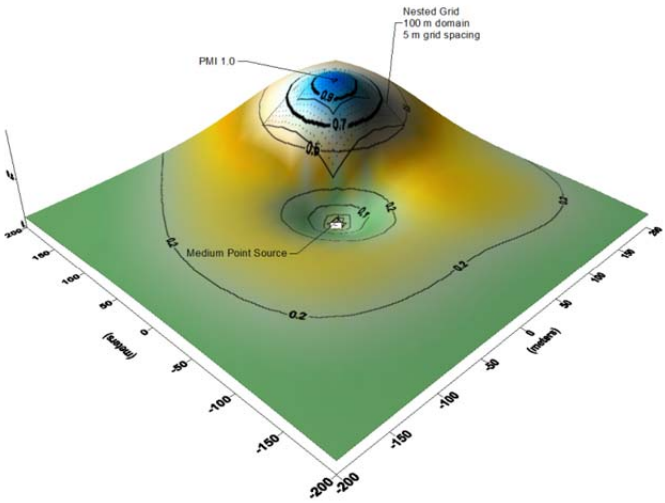
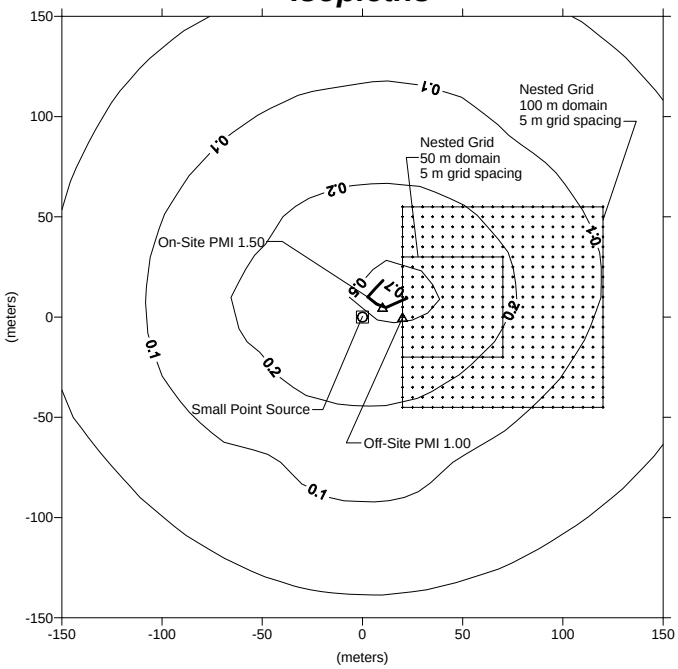


Figure AP C-3.3.1 – Small Point Source – Costa Mesa

Isopleths



Surface

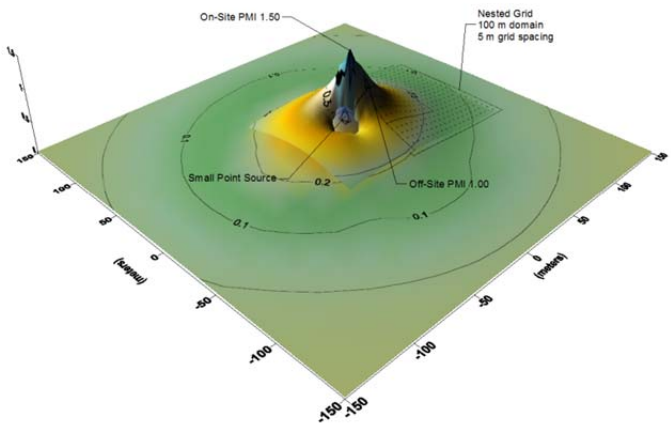
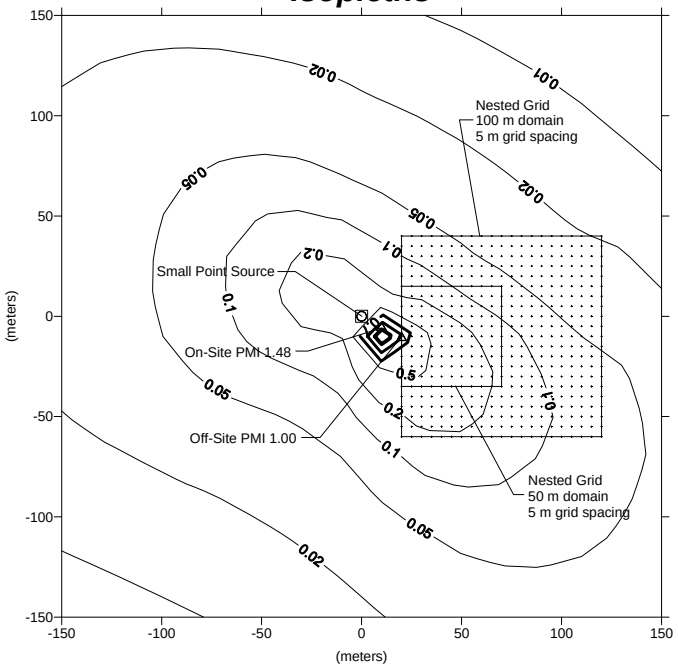


Figure AP C-3.3.2 – Small Point Source – Fresno Air Terminal

Isopleths



Surface

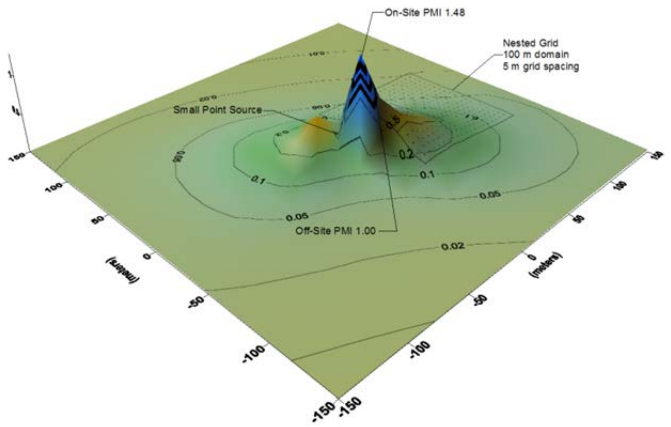
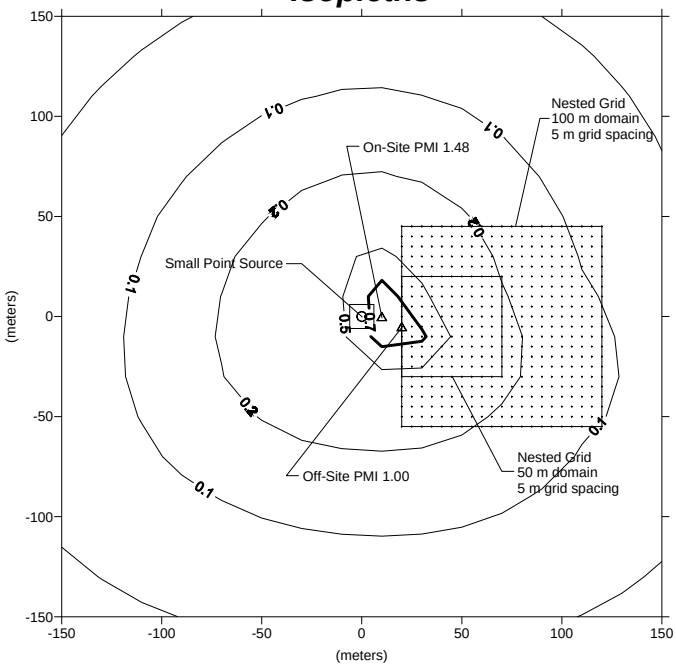


Figure AP C-3.3.3 – Small Point Source – Kearny Mesa

Isopleths



Surface

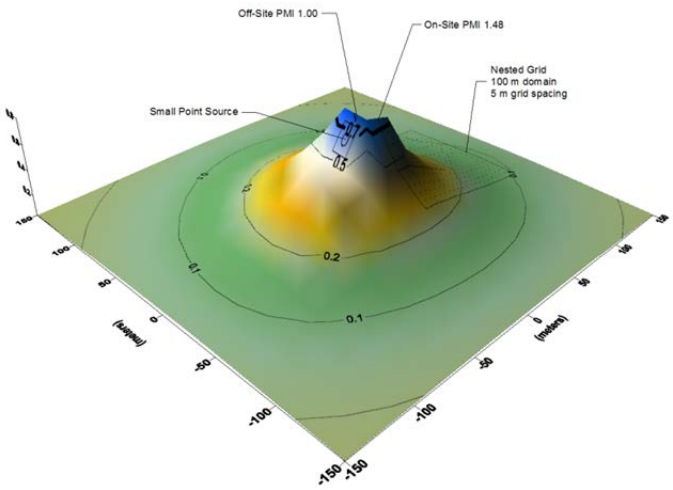
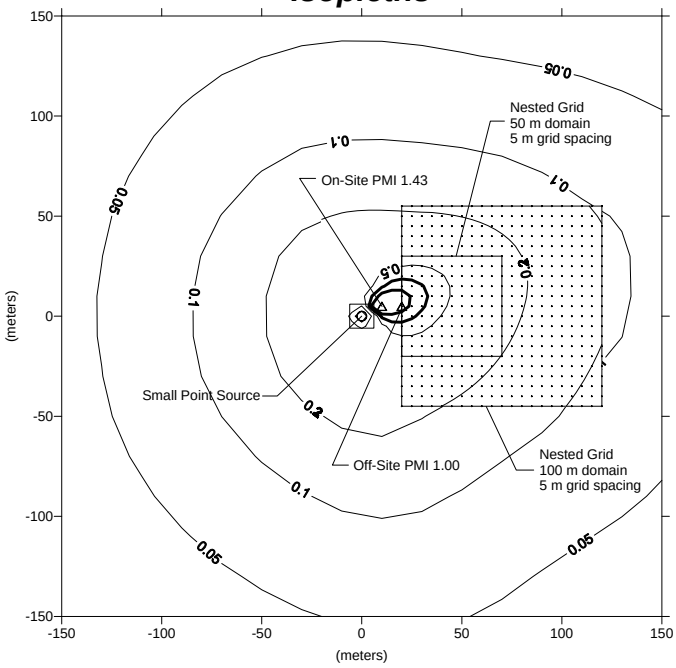


Figure AP C-3.3.4 – Small Point Source – Lynwood

Isopleths



Surface

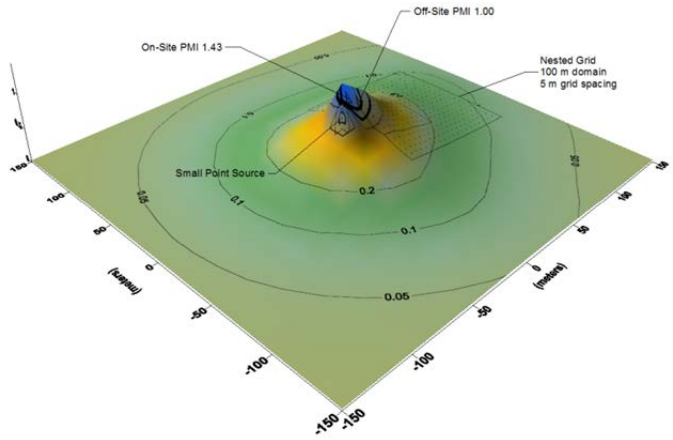
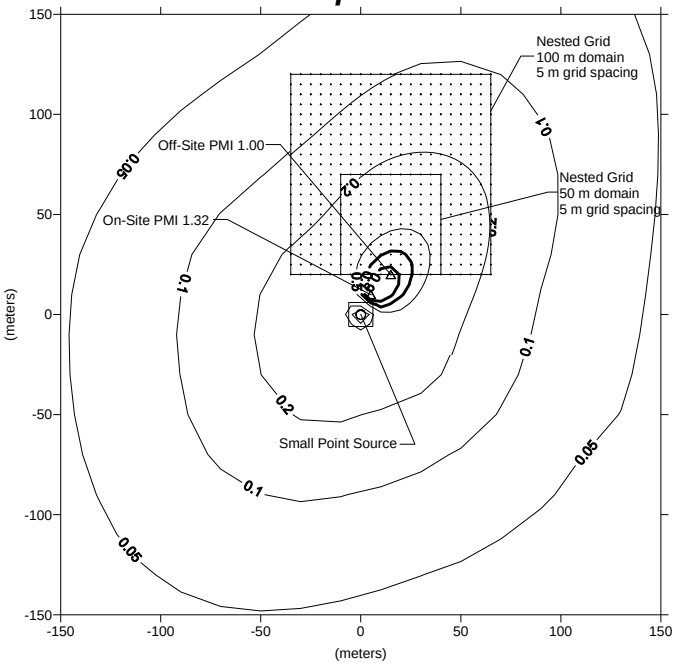


Figure AP C-3.3.5 – Small Point Source – San Bernardino

Isopleths



Surface

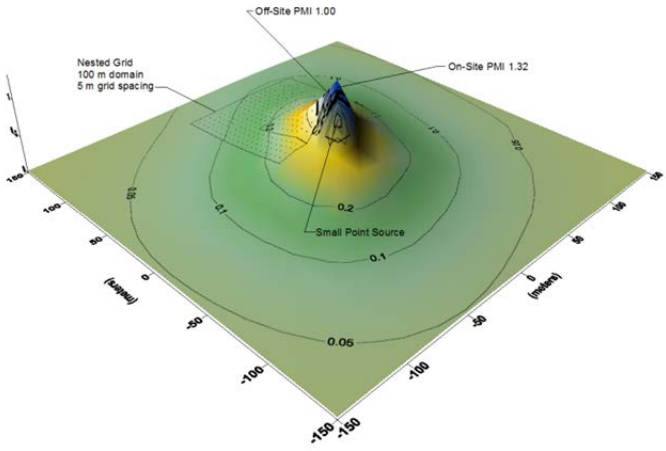
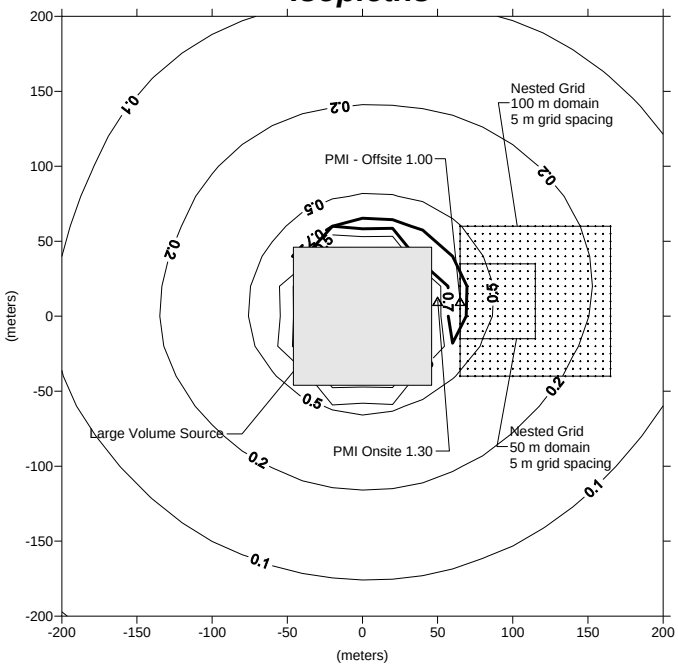


Figure AP C-3.4.1 – Large Volume Source – Costa Mesa

Isopleths



Surface

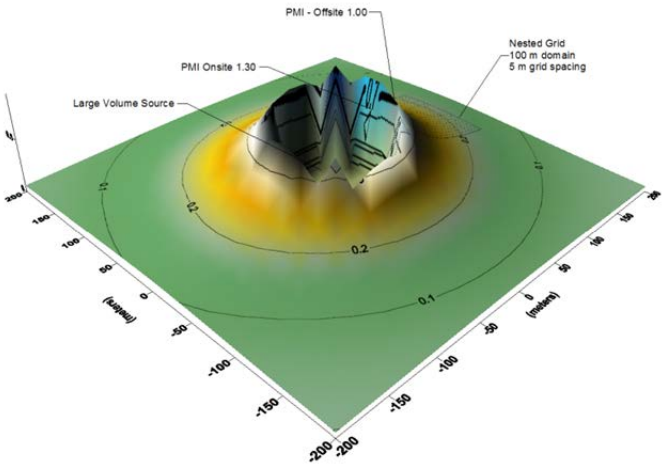
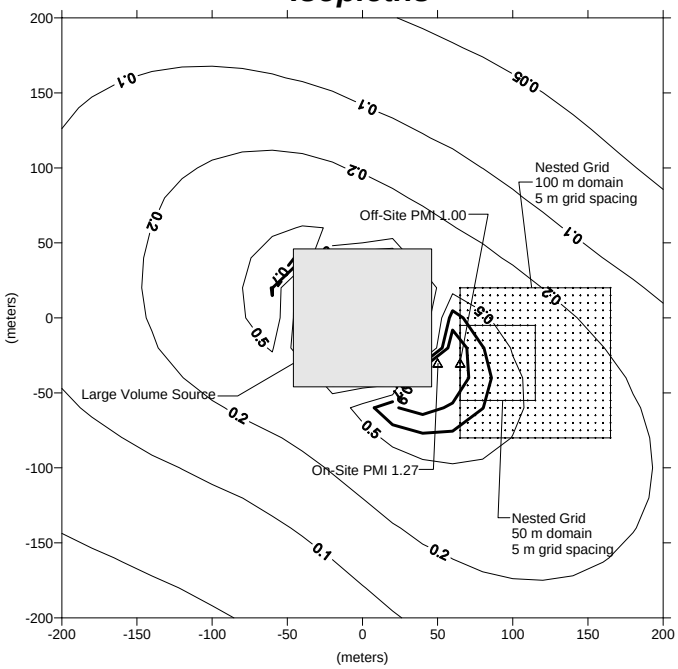


Figure AP C-3.4.2 – Large Volume Source – Fresno Air Terminal

Isopleths



Surface

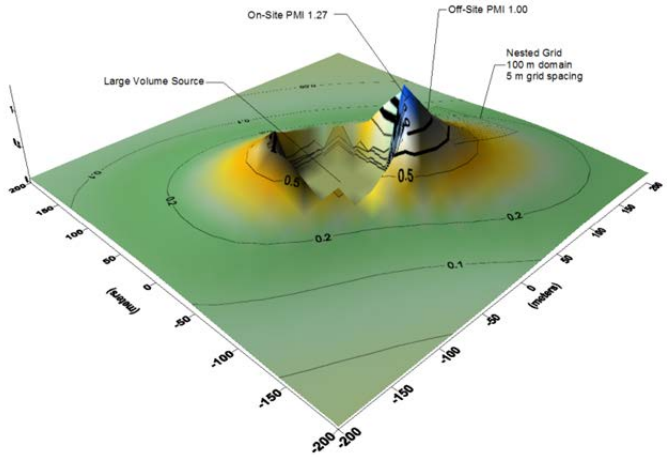
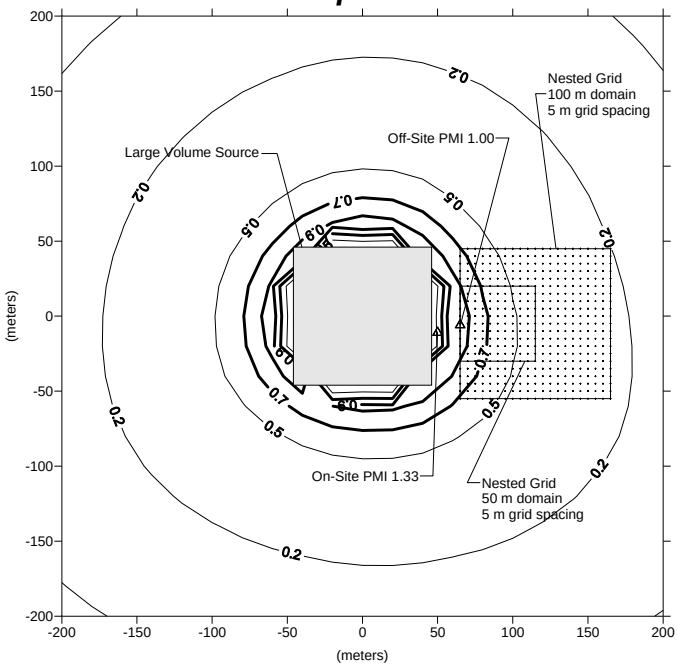


Figure AP C-3.4.3 – Large Volume Source – Kearny Mesa

Isopleths



Surface

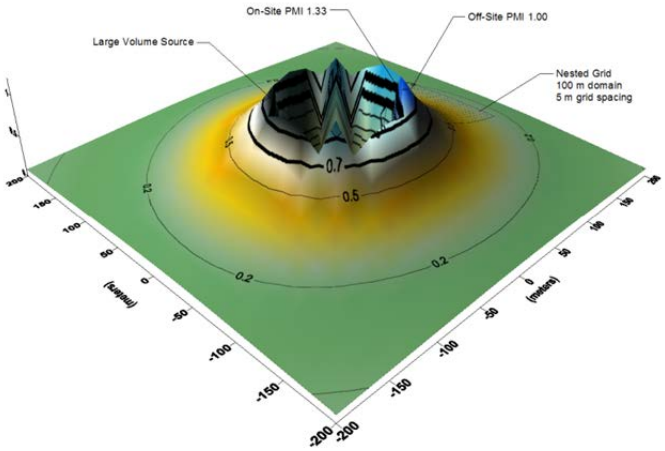
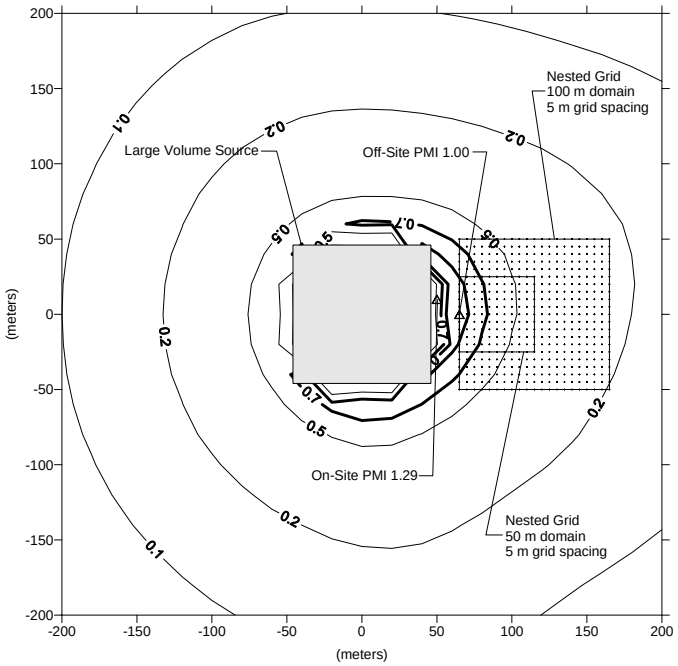


Figure AP C-3.4.4 – Large Volume Source – Lynwood

Isopleths



Surface

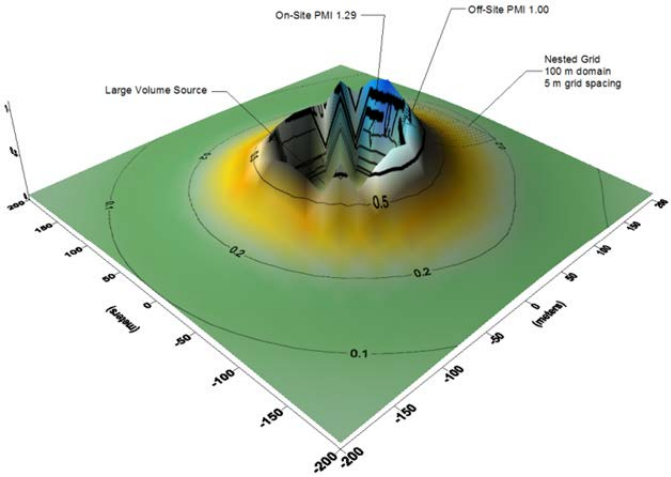
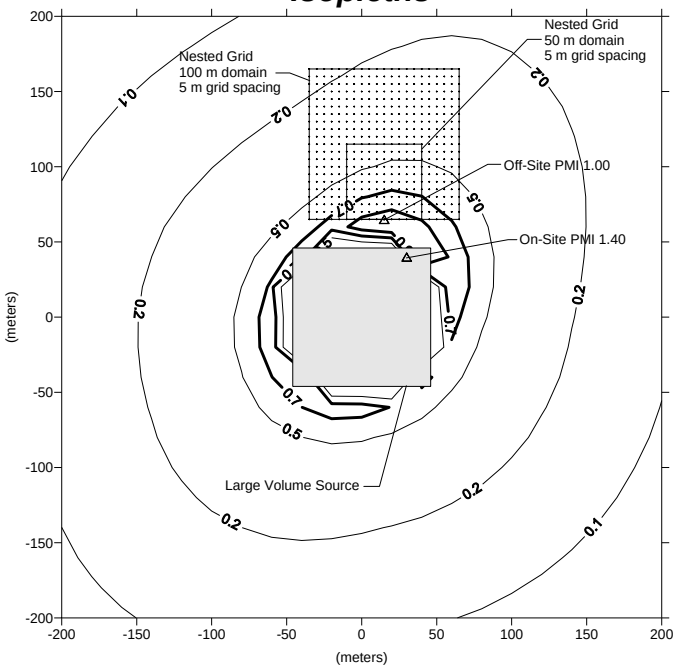


Figure AP C-3.4.5 – Large Volume Source – San Bernardino

Isopleths



Surface

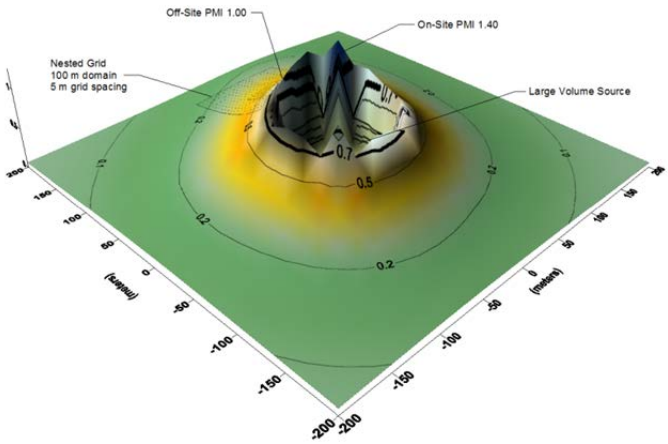
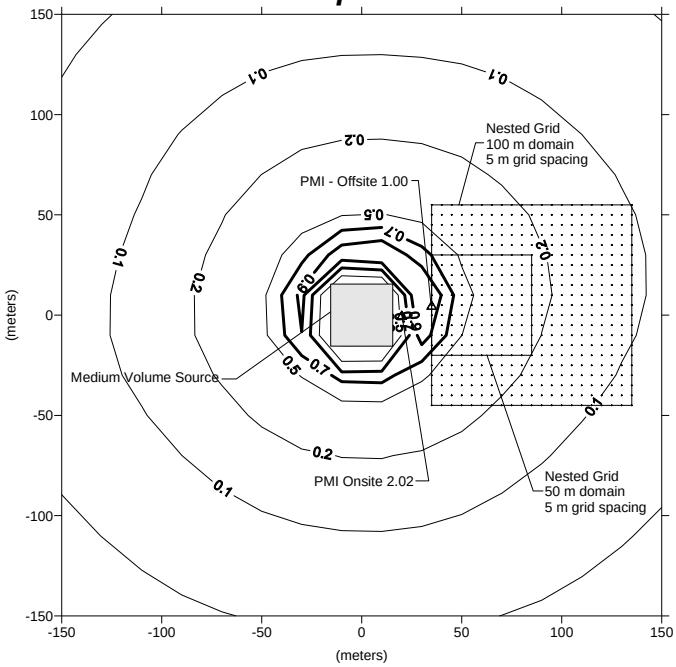


Figure AP C-3.5.1 – Medium Volume Source – Costa Mesa

Isopleths



Surface

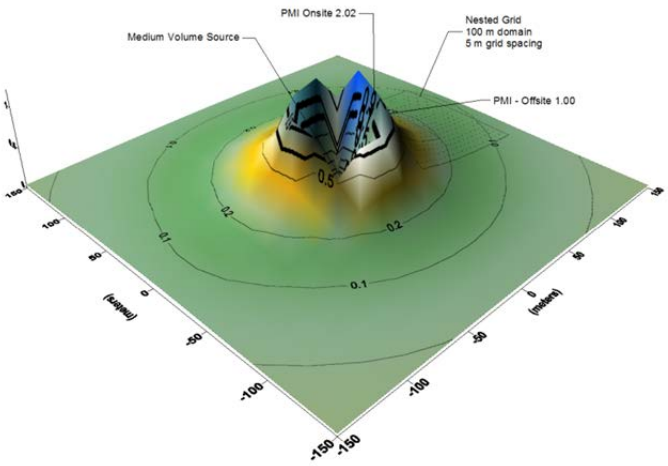
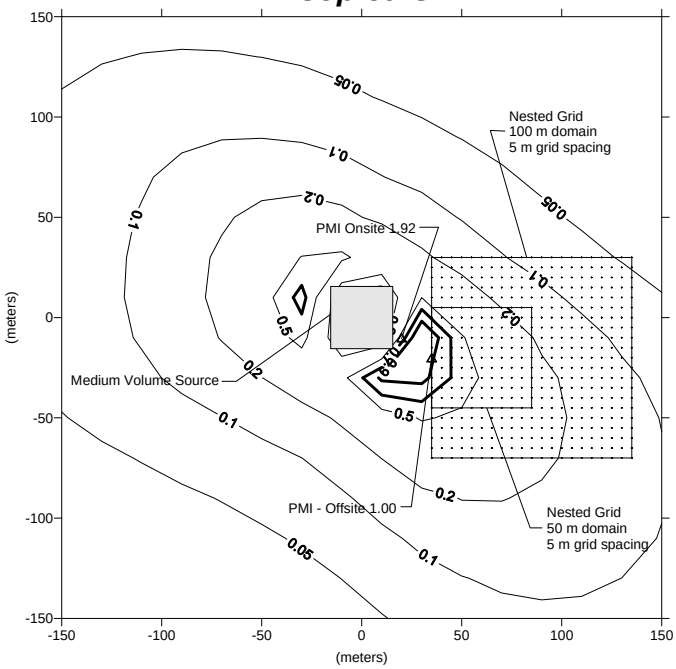


Figure AP C-3.5.2 – Medium Volume Source – Fresno Air Terminal

Isopleths



Surface

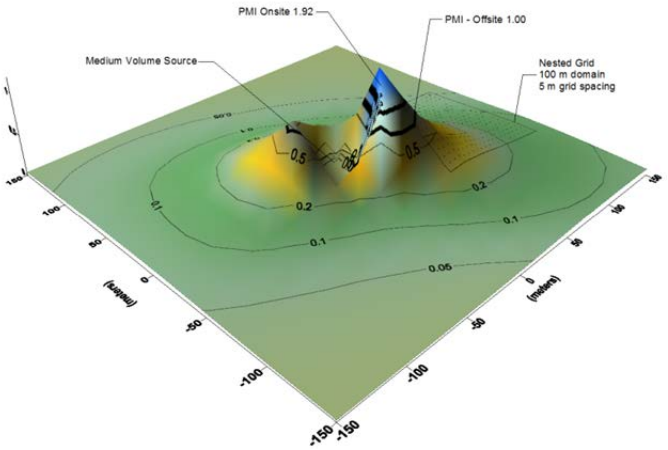
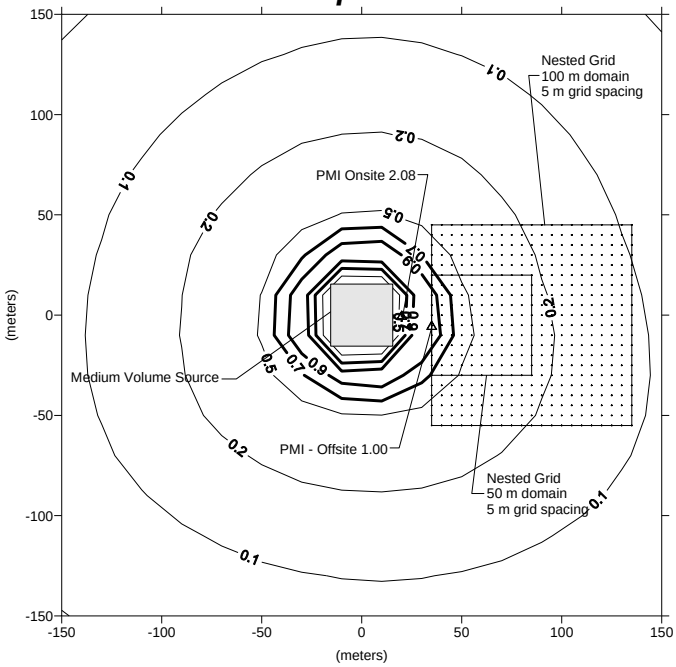


Figure AP C-3.5.3 – Medium Volume Source – Kearny Mesa

Isopleths



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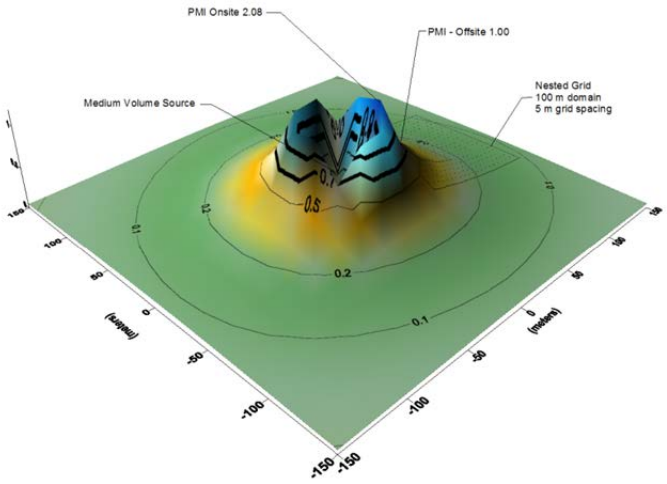
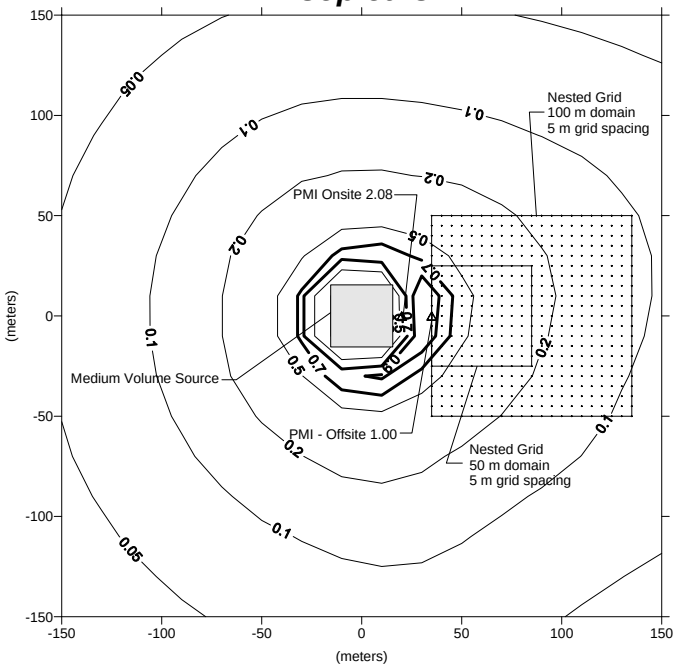


Figure AP C-3.5.4 – Medium Volume Source – Lynnwood

Isopleths



Surface

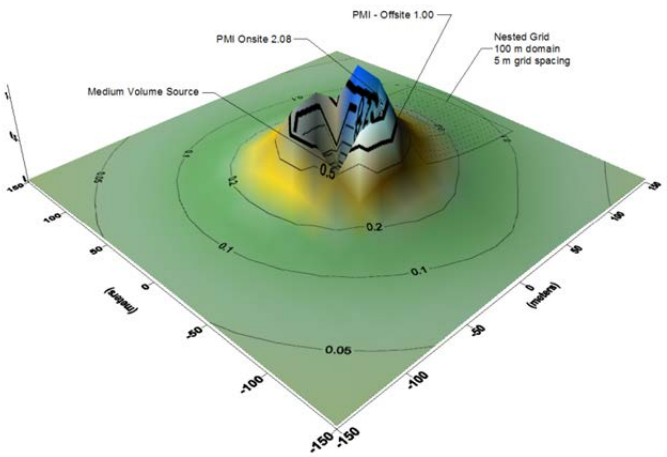
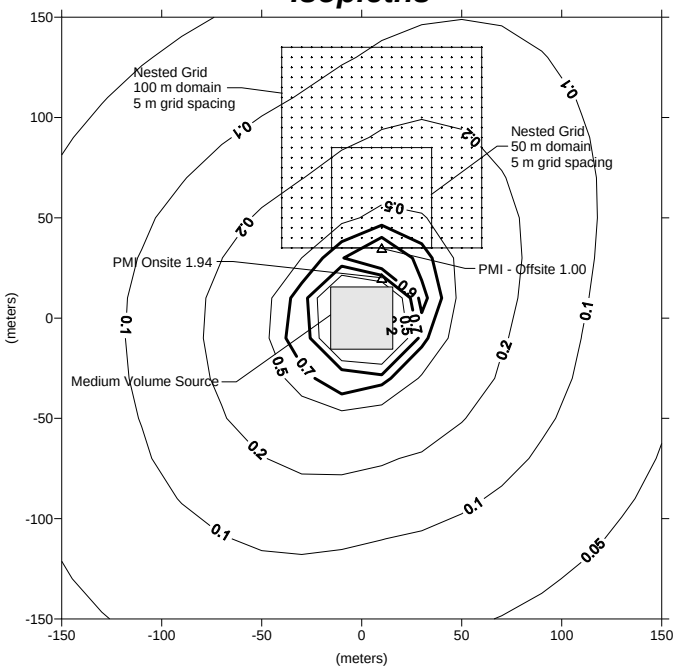


Figure AP C-3.5.5 – Medium Volume Source – San Bernardino

Isopleths



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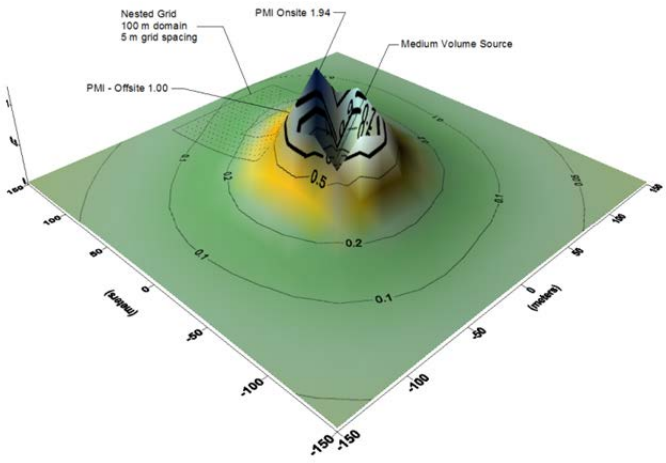
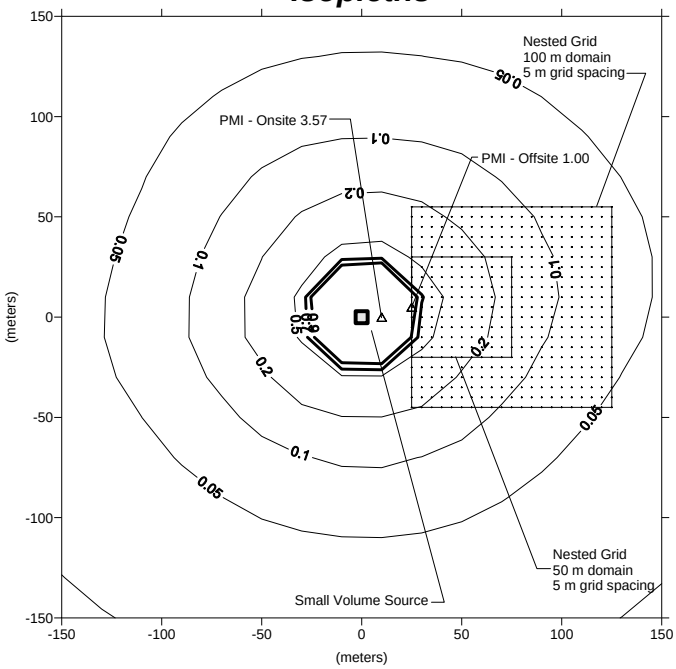


Figure AP C-3.6.1 – Small Volume Source – Costa Mesa

Isopleths



Surface

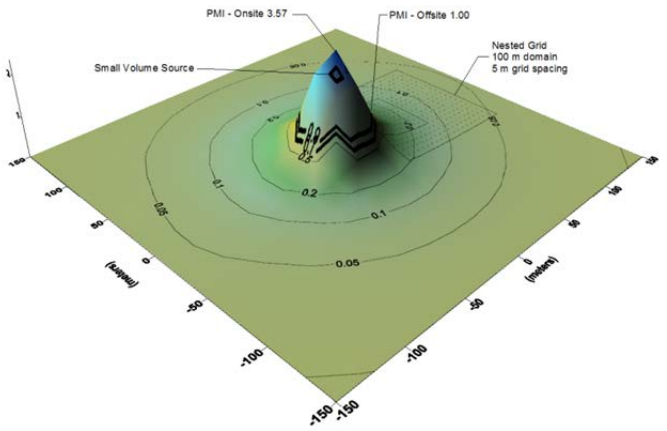
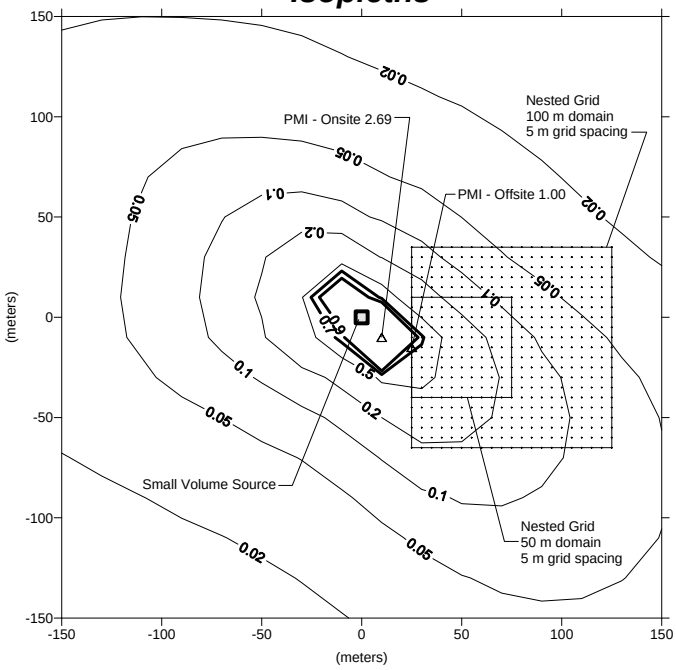


Figure AP C-3.6.2 – Small Volume Source – Fresno Air Terminal

Isopleths



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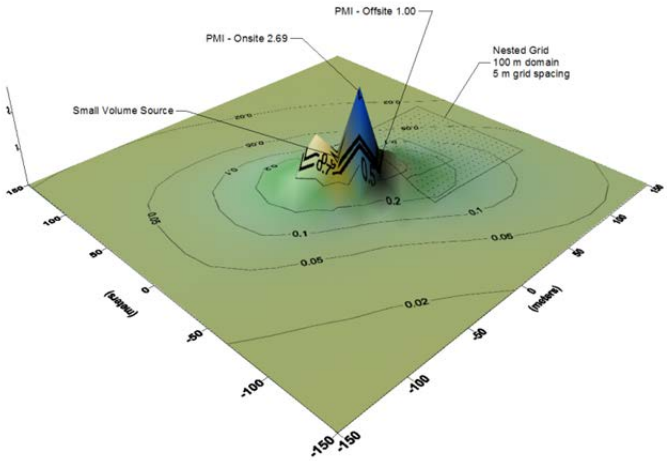
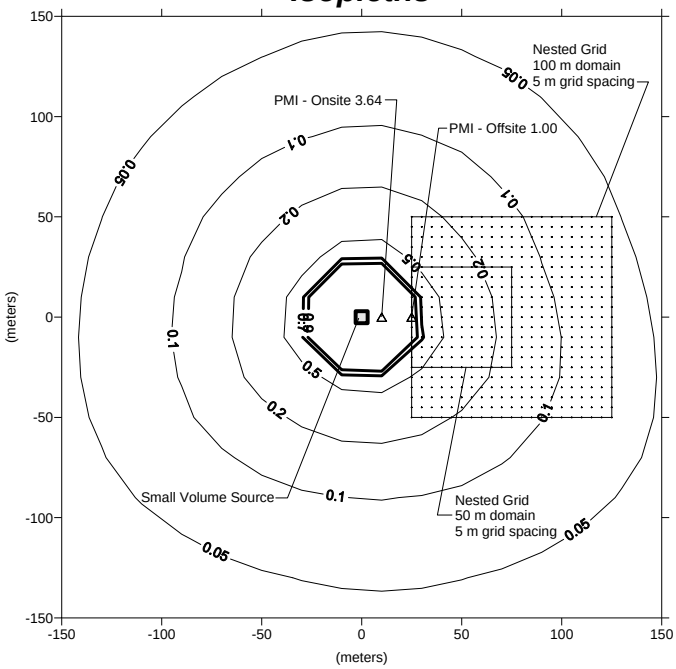


Figure AP C-3.6.3 – Small Volume Source – Kearny Mesa

Isopleths



Surface

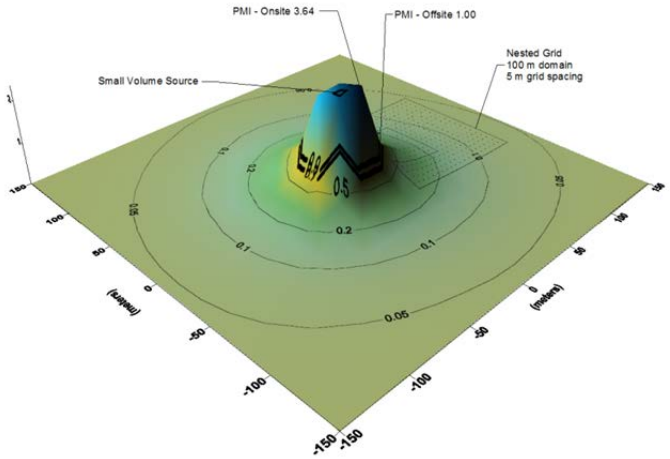
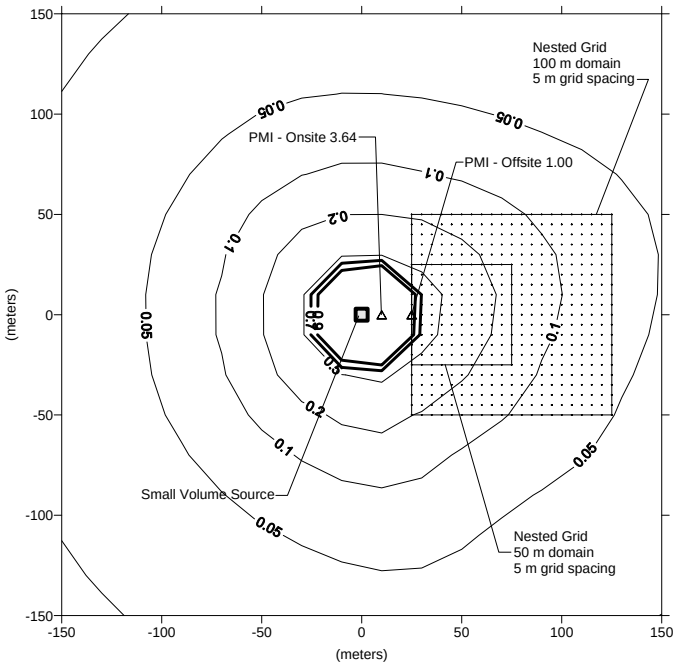


Figure AP C-3.6.4 – Small Volume Source – Lynnwood

Isopleths



Surface

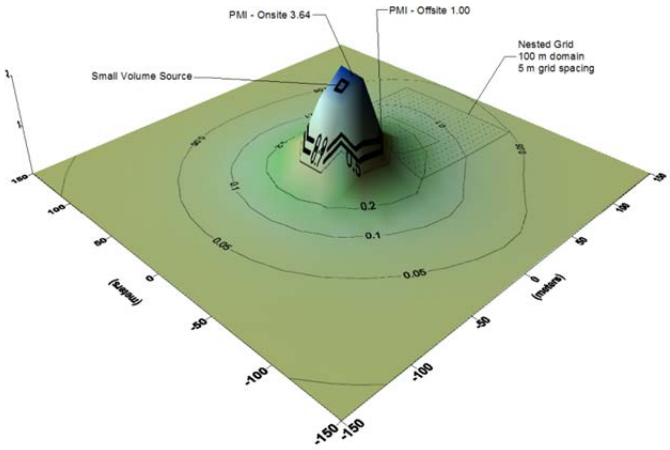
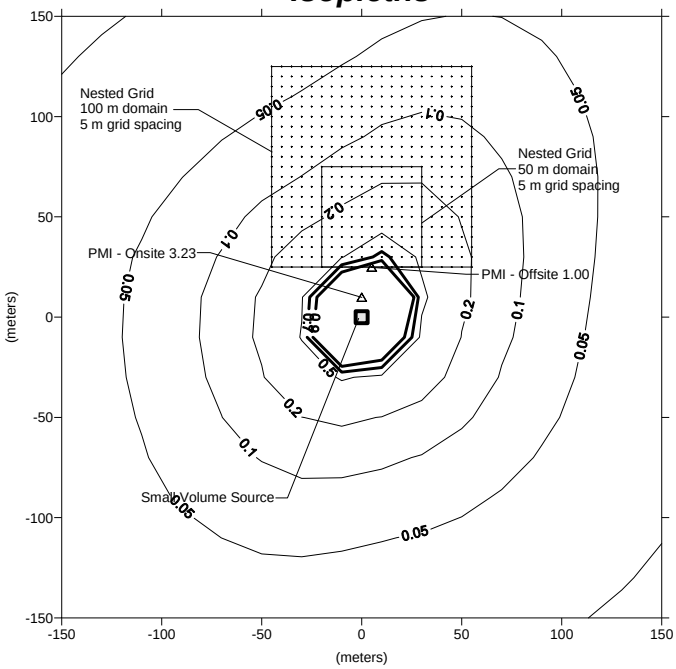


Figure AP C-3.6.5 – Small Volume Source – San Bernardino

Isopleths



Surface

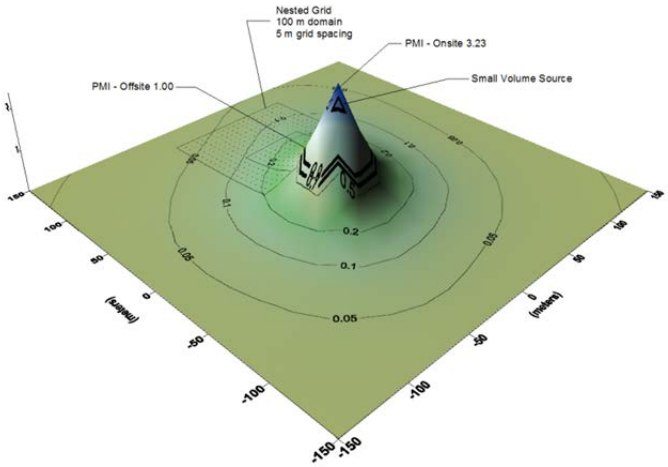
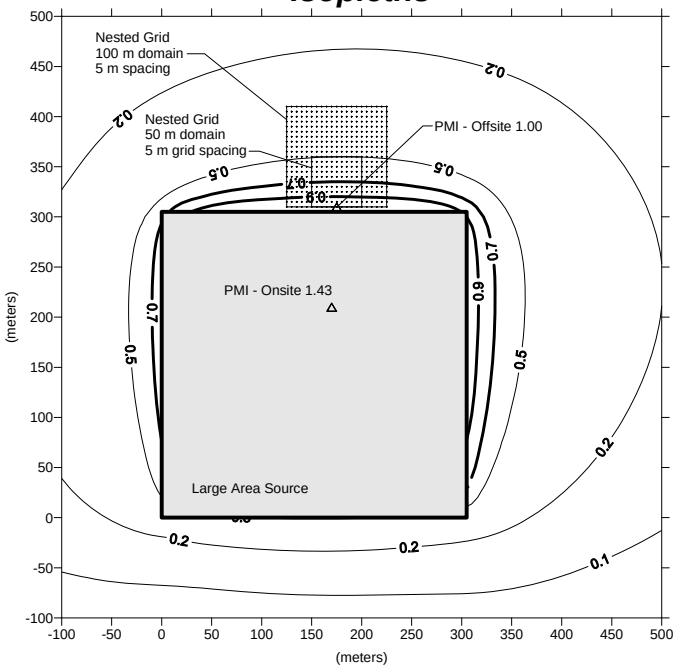


Figure AP C-3.7.1 – Large Area Source – Costa Mesa

Isopleths



Surface

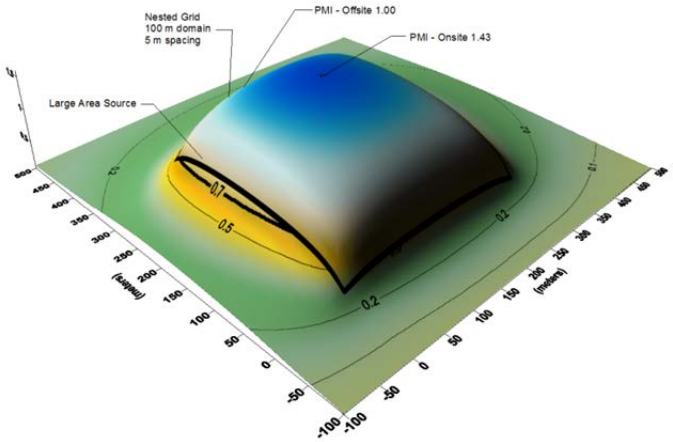
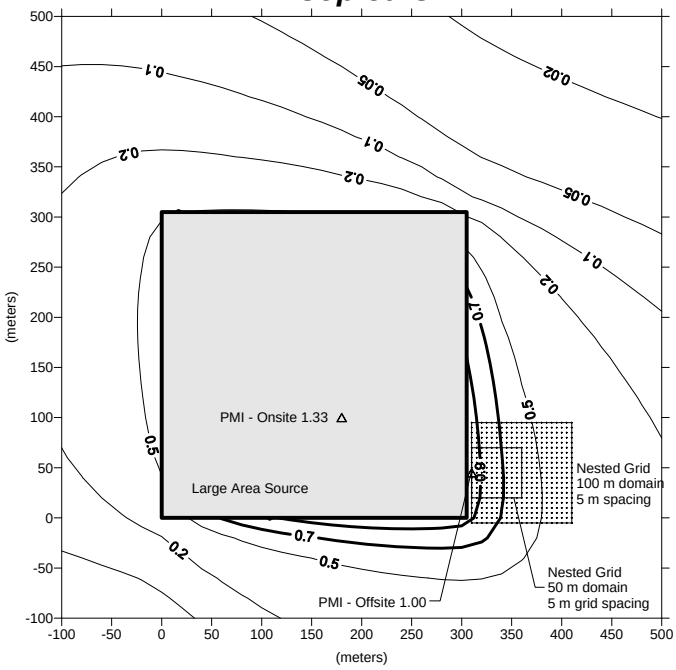


Figure AP C-3.7.2 – Large Area Source – Fresno Air Terminal

Isopleths



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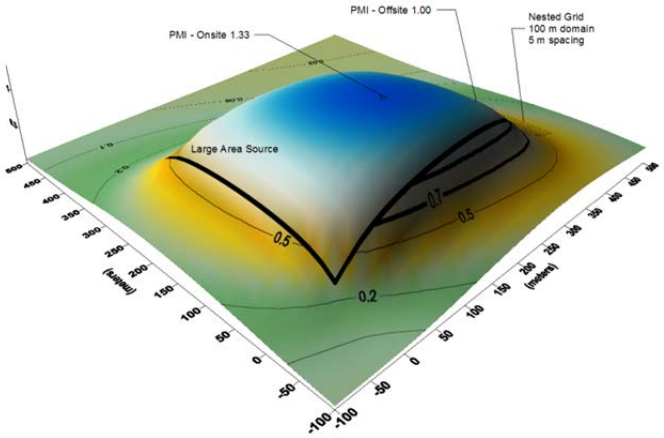
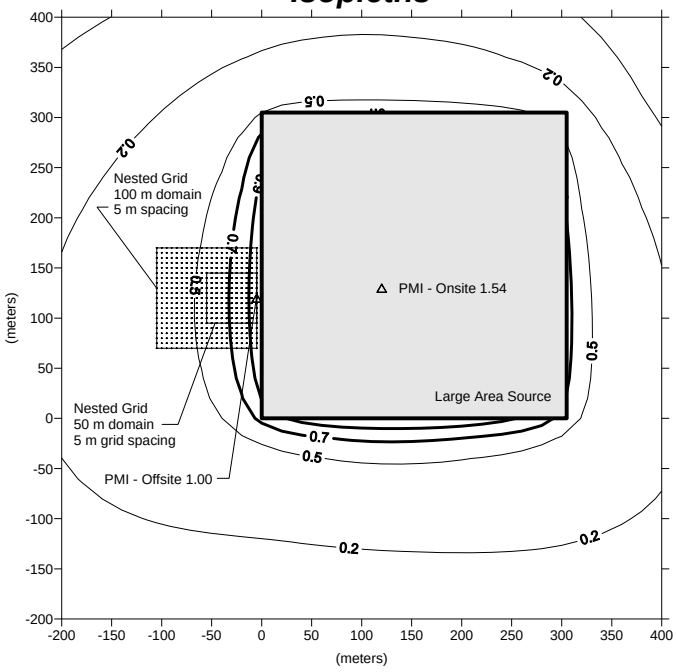


Figure AP C-3.7.3 – Large Area Source – Kearny Mesa

Isopleths ***Surface***



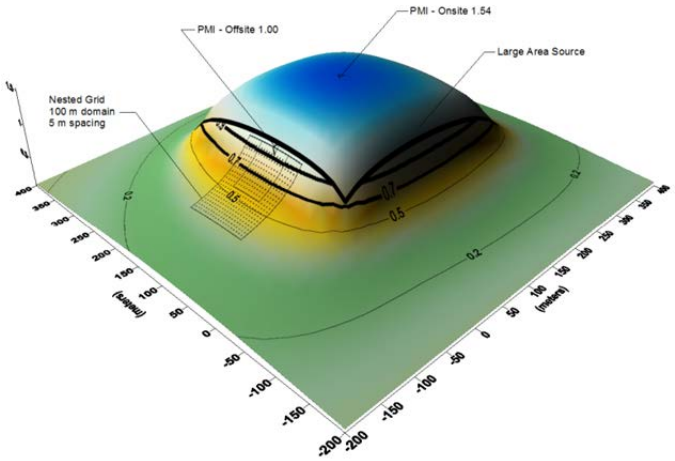
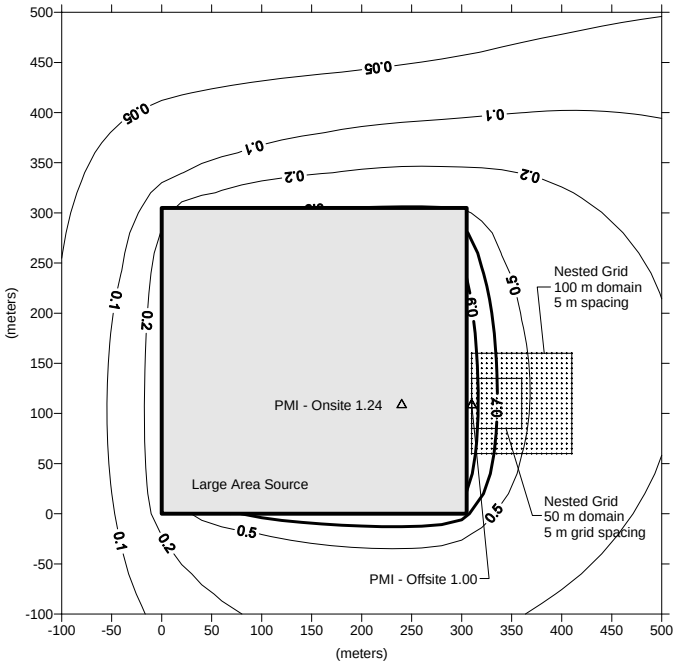


Figure AP C-3.7.4 – Large Area Source – Lynwood

Isopleths ***Surface***



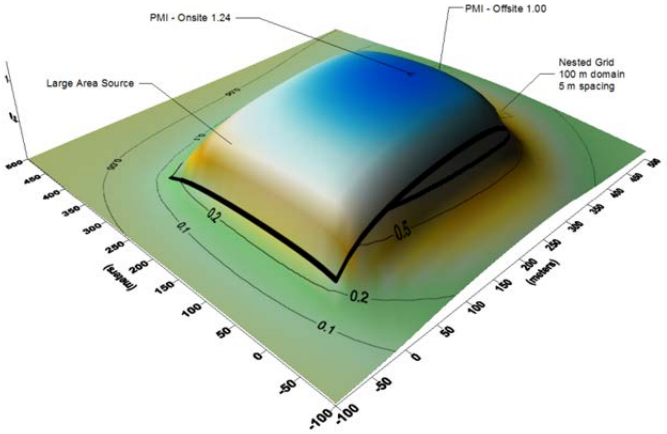
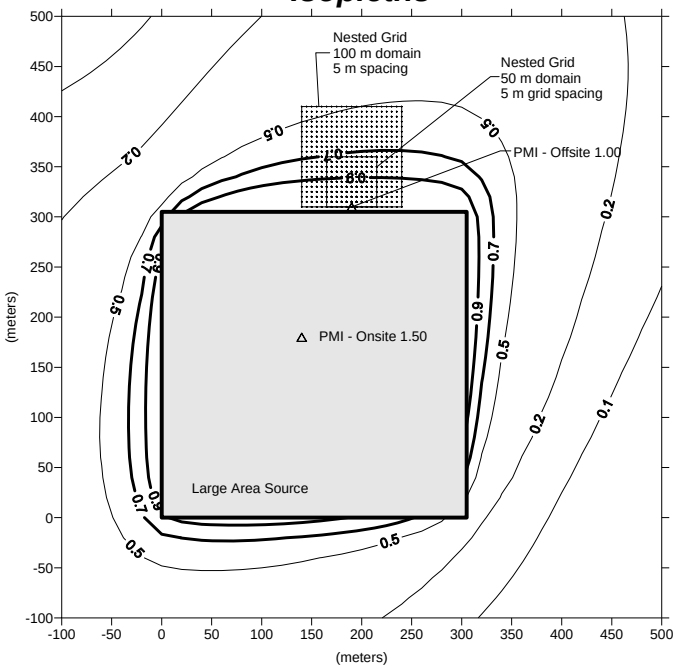


Figure AP C-3.7.5 – Large Area Source – San Bernardino

Isopleths



Surface

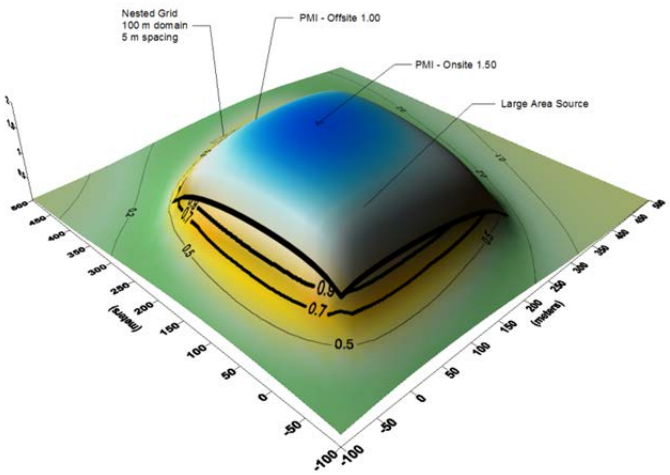
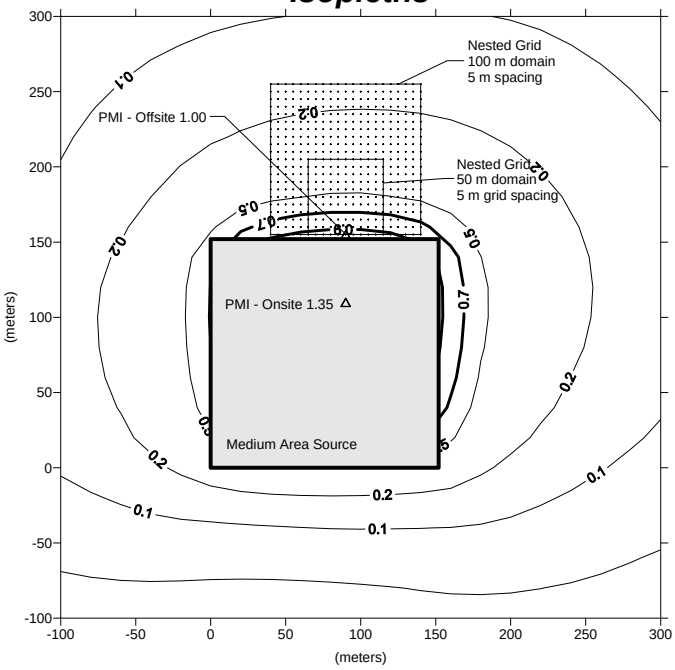


Figure AP C-3.8.1 – Medium Area Source – Costa Mesa

Isopleths



Surface

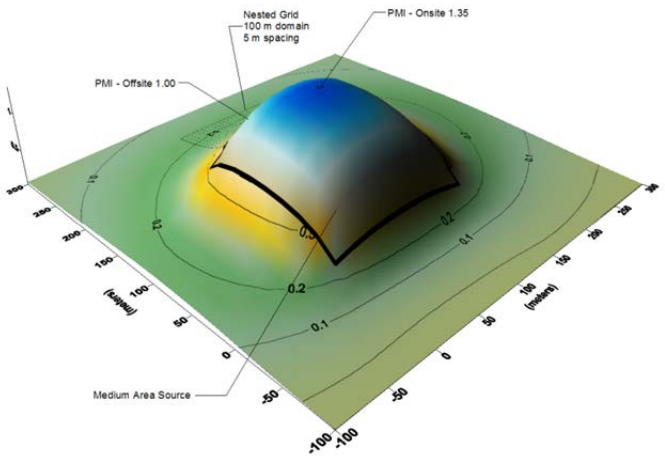
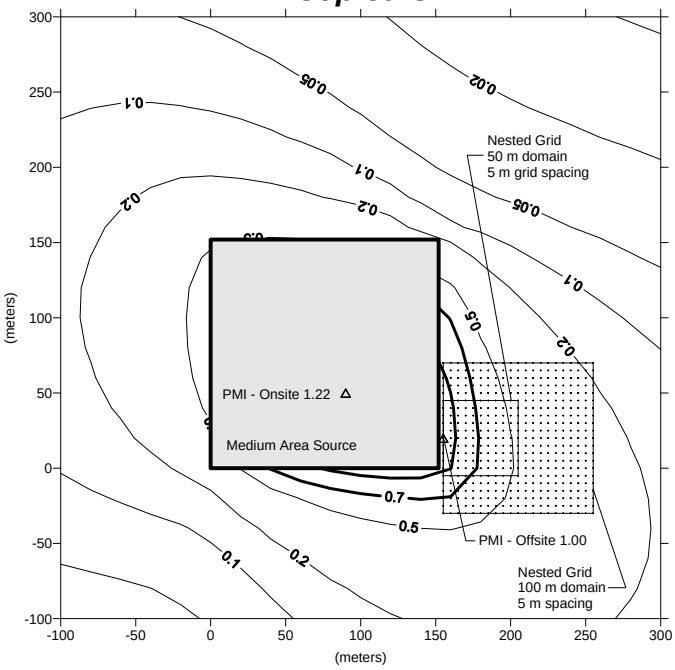


Figure AP C-3.8.2 – Medium Area Source – Fresno Air Terminal

Isopleths



Surface

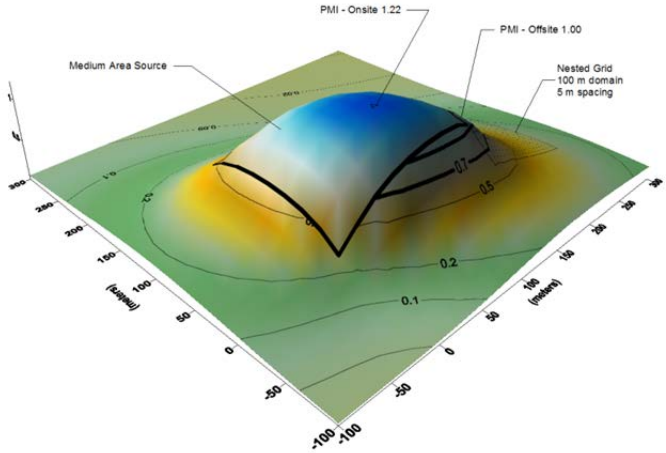
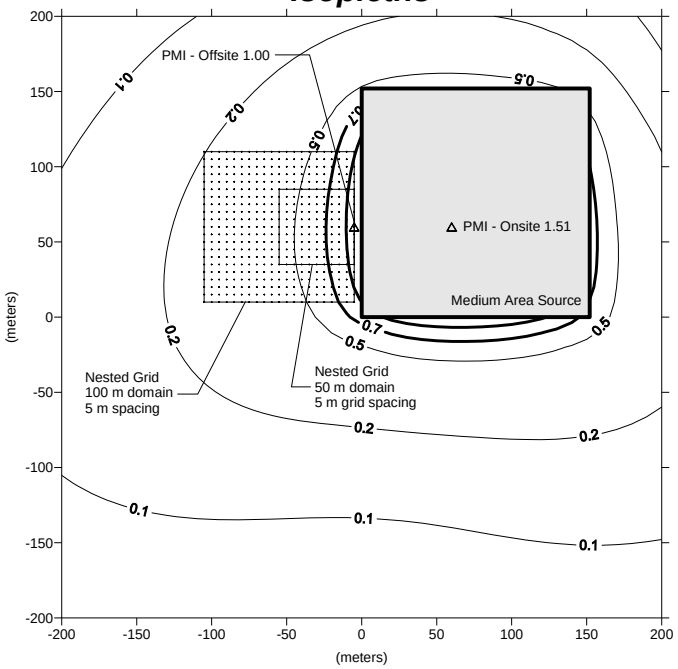


Figure AP C-3.8.3 – Medium Area Source – Kearny Mesa

Isopleths



Surface

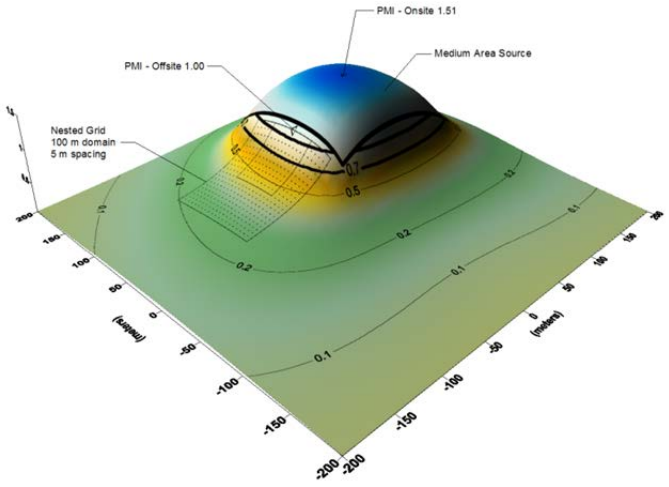
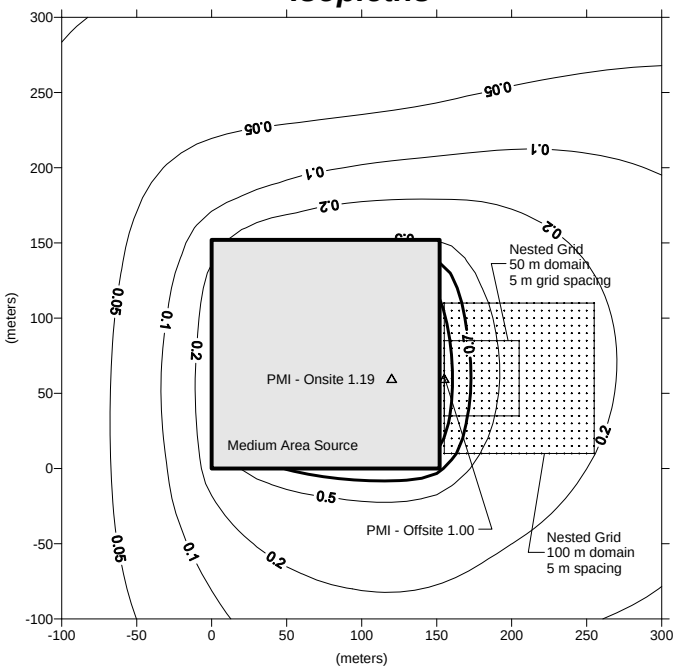


Figure AP C-3.8.4 – Medium Area Source – Lynwood

Isopleths



Surface

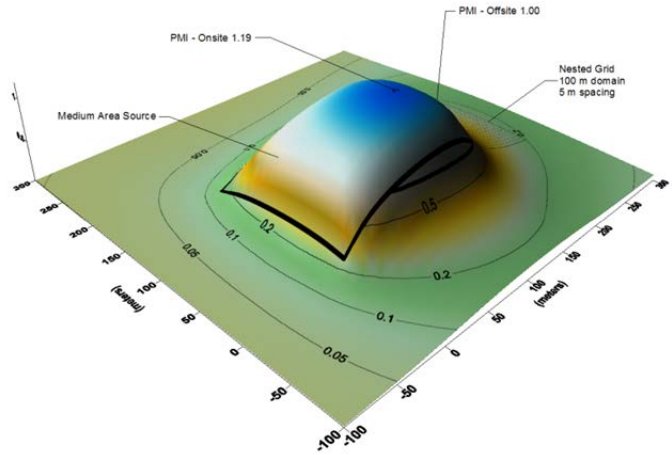
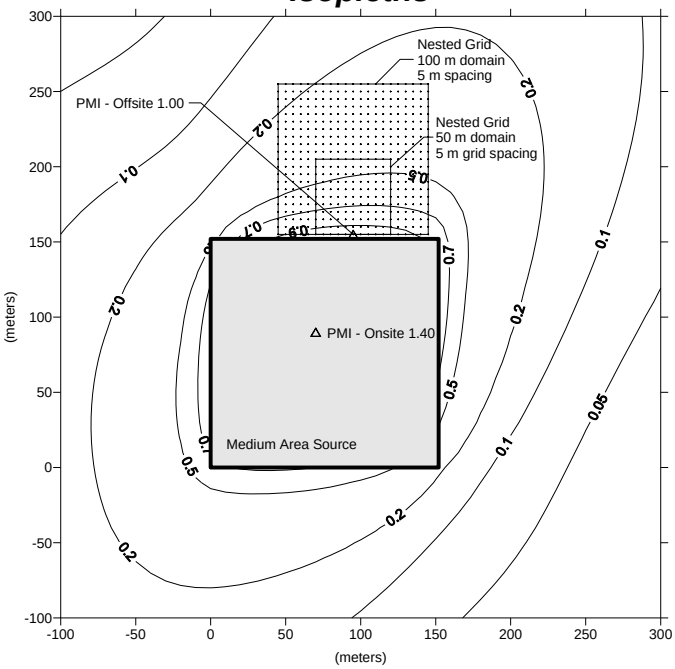


Figure AP C-3.8.5 – Medium Area Source – San Bernardino

Isopleths



Surface

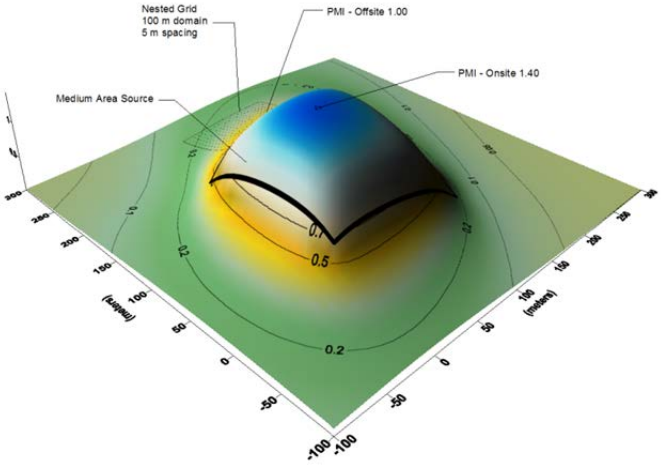
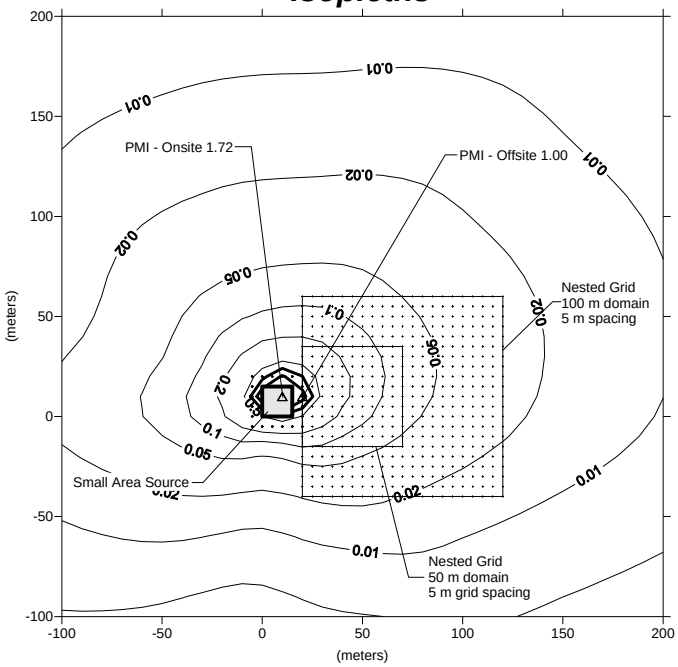


Figure AP C-3.9.1 – Small Area Source – Costa Mesa

Isopleths



Surface

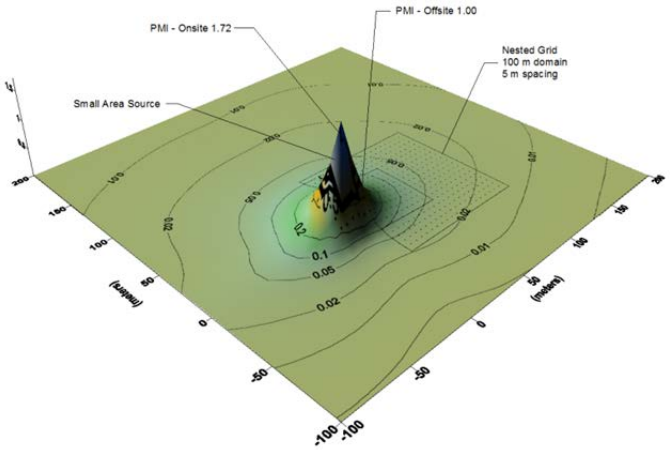
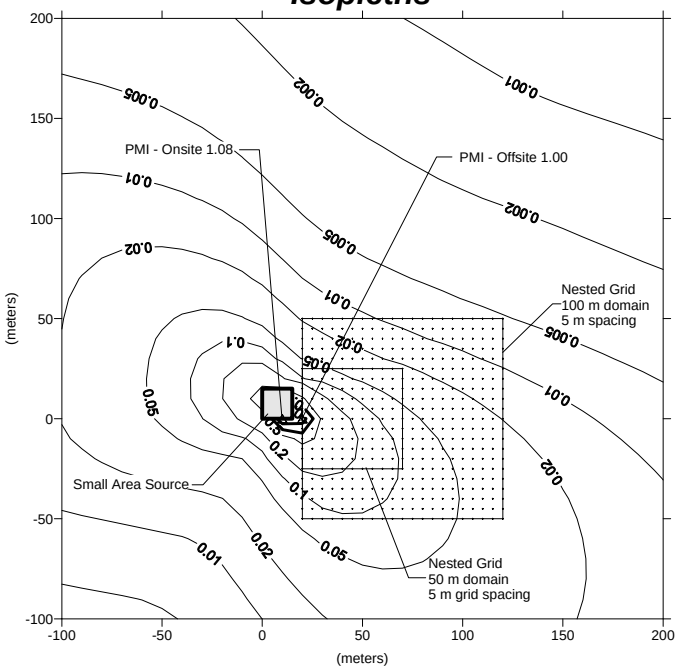


Figure AP C-3.9.2 – Small Area Source – Fresno Air Terminal

Isopleths



Surface

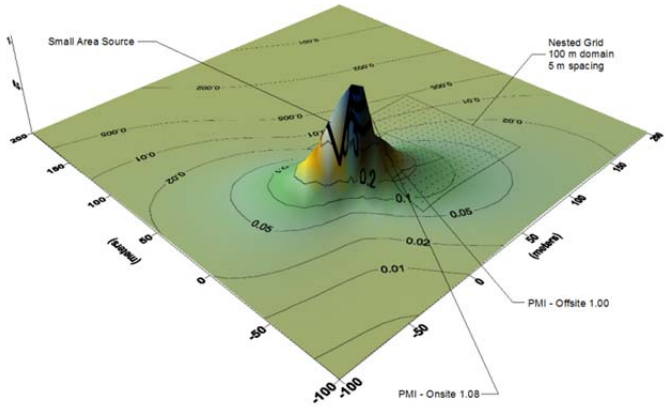
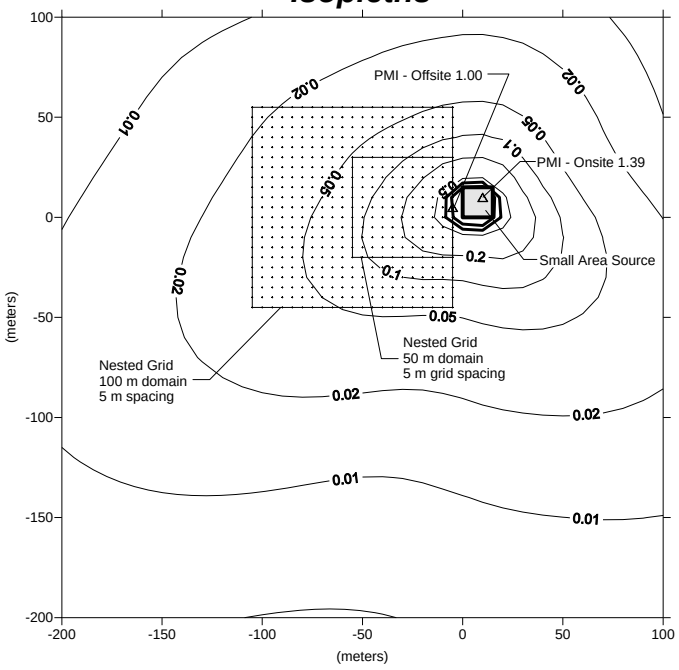


Figure AP C-3.9.3 – Small Area Source – Kearny Mesa

Isopleths



Surface

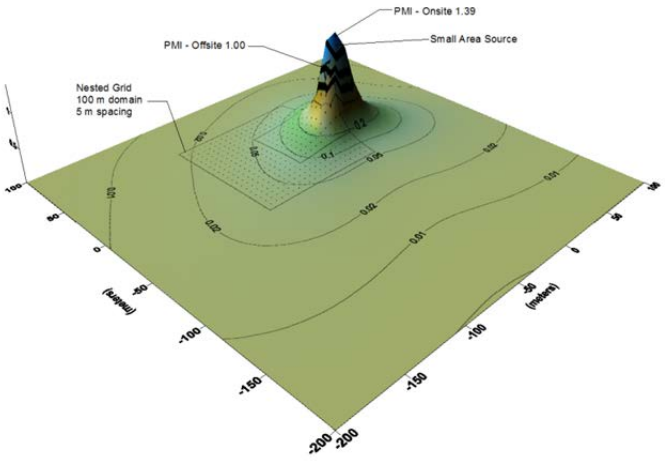
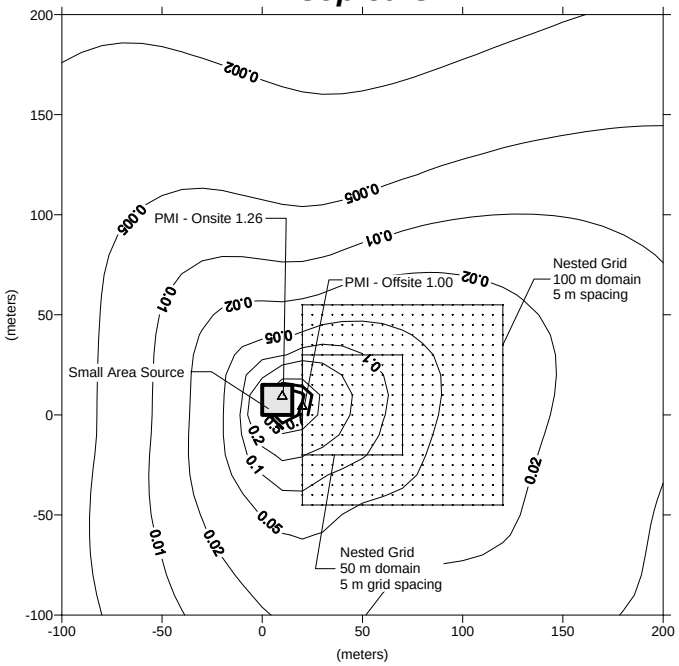


Figure AP C-3.9.4 – Small Area Source – Lynwood

Isopleths



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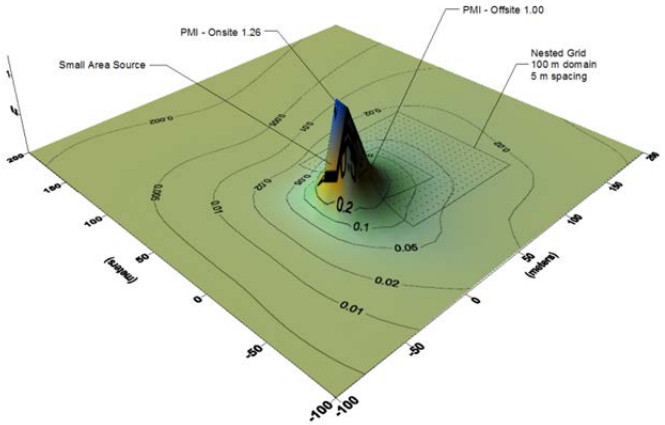
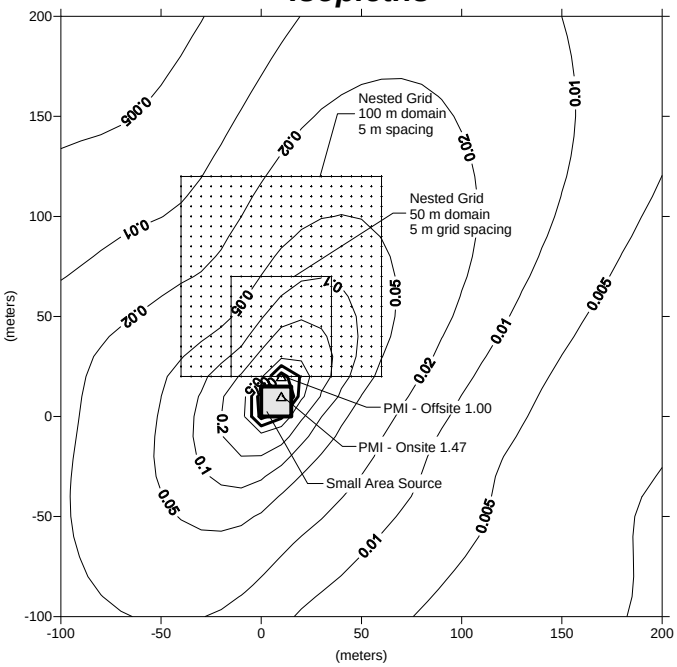


Figure AP C-3.9.5 – Small Area Source – San Bernardino

Isopleths



Surface

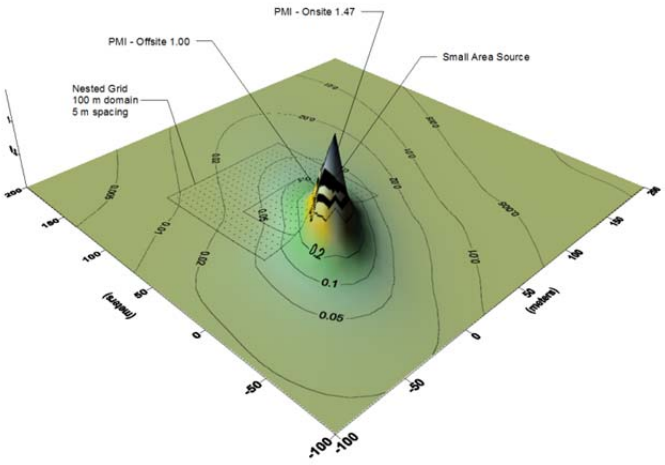
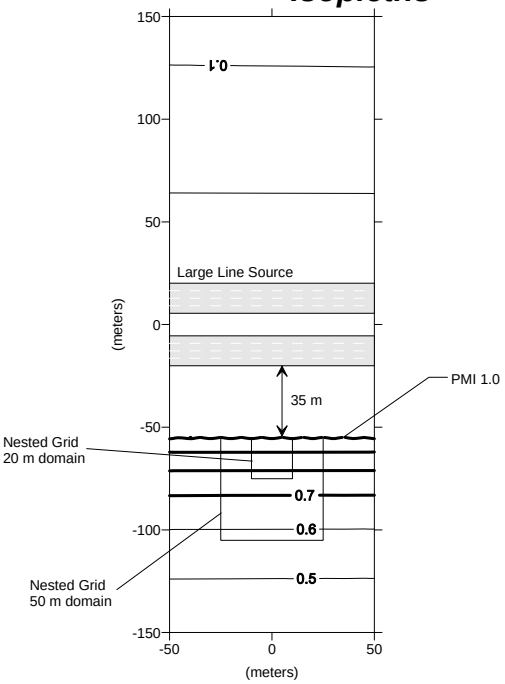


Figure AP C-3.10.1 – Large Line Source, CALINE – Costa Mesa

Isopleths



Surface

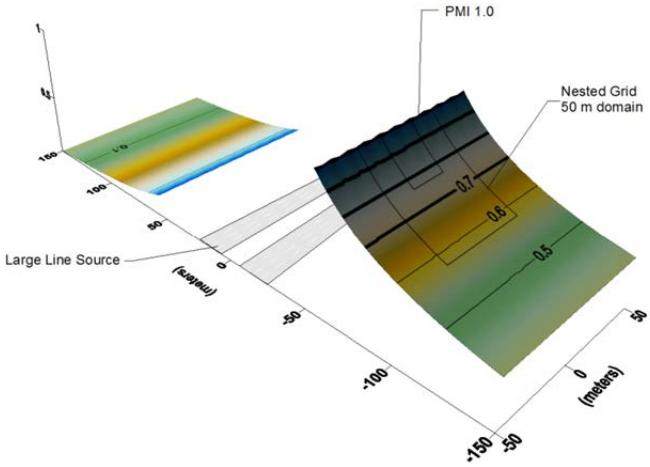
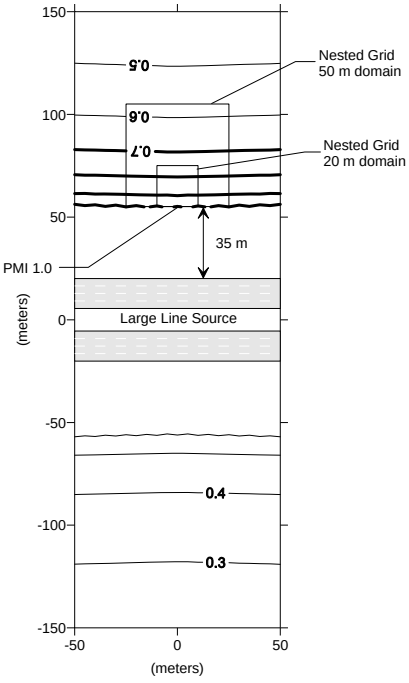


Figure AP C-3.10.2 – Large Line Source, CALINE – Fresno Air Terminal

Isopleths



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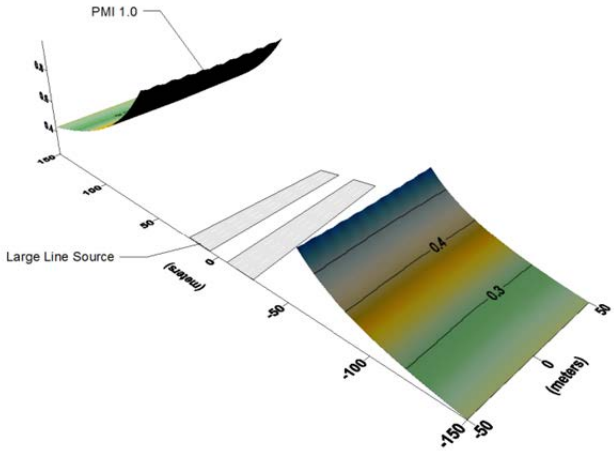


Figure AP C-3.10.3 – Large Line Source, CALINE – Kearny Mesa

Isopleths ***Surface***

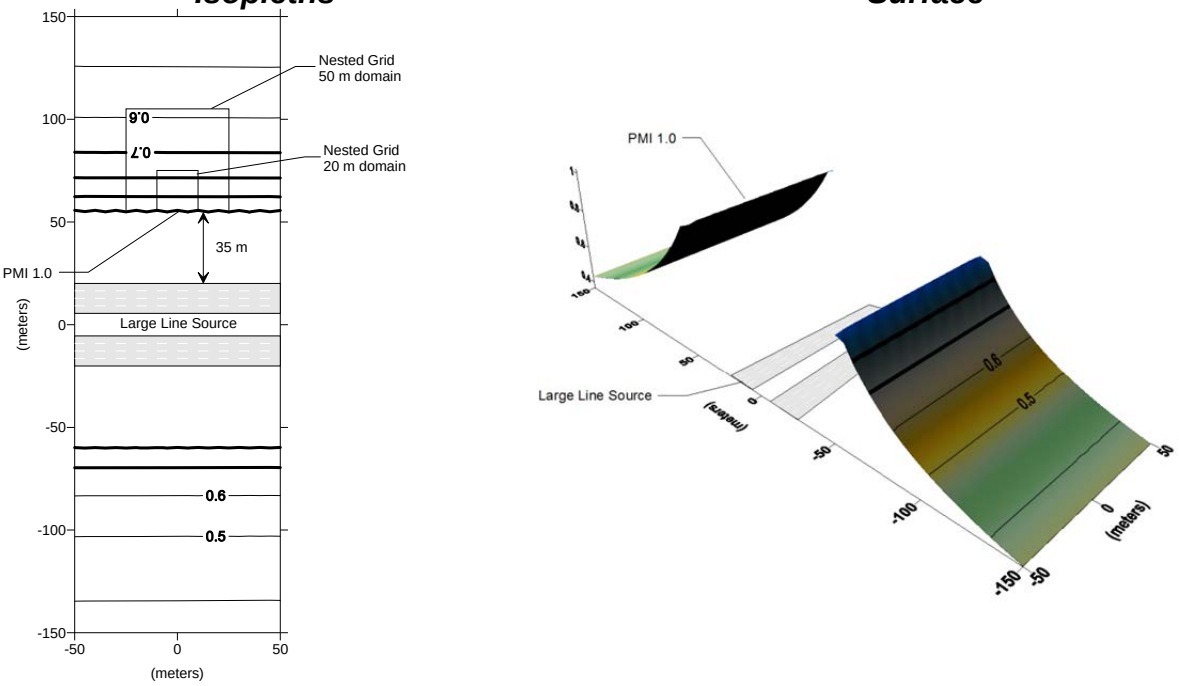


Figure AP C-3.10.4 – Large Line Source, CALINE – Lynwood

Isopleths ***Surface***

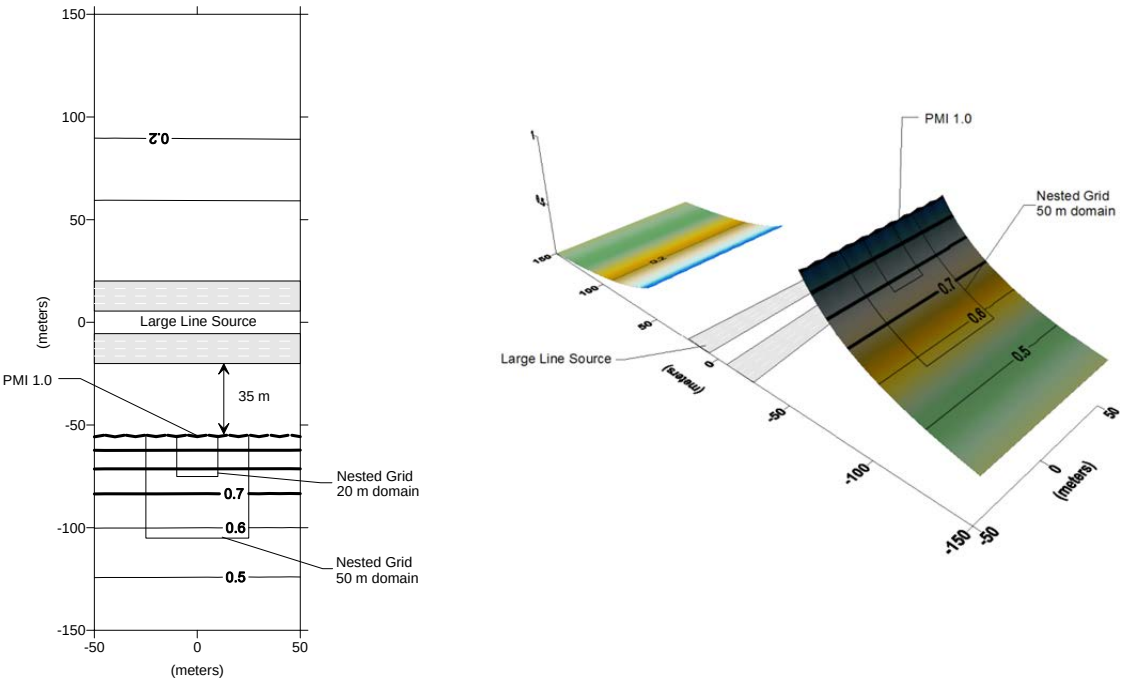
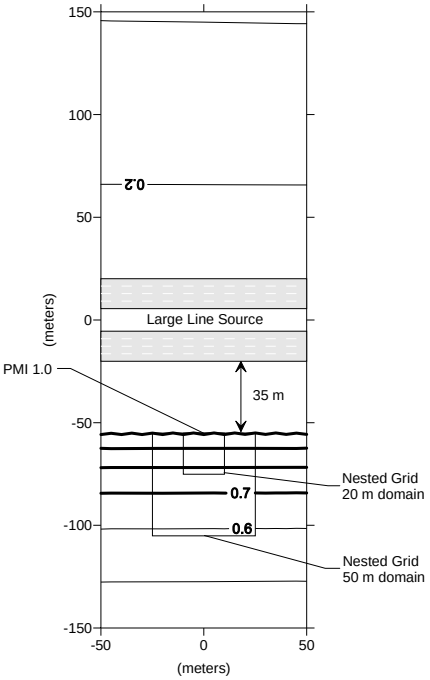


Figure AP C-3.10.5 – Large Line Source, CALINE – San Bernardino

Isopleths ***Surface***



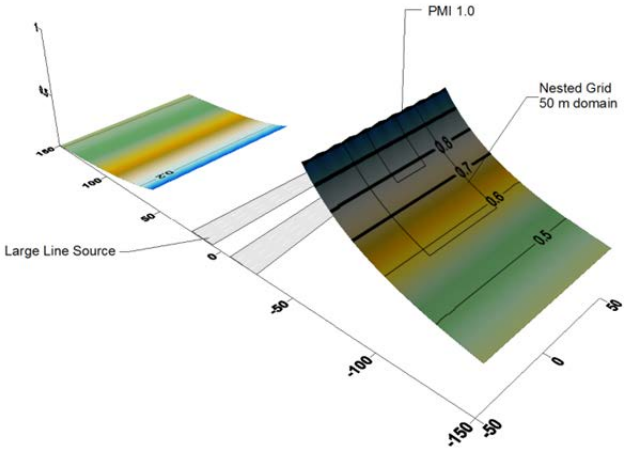


Figure AP C-3.11.1 – Small Line Source, CALINE – Costa Mesa

Isopleths ***Surface***

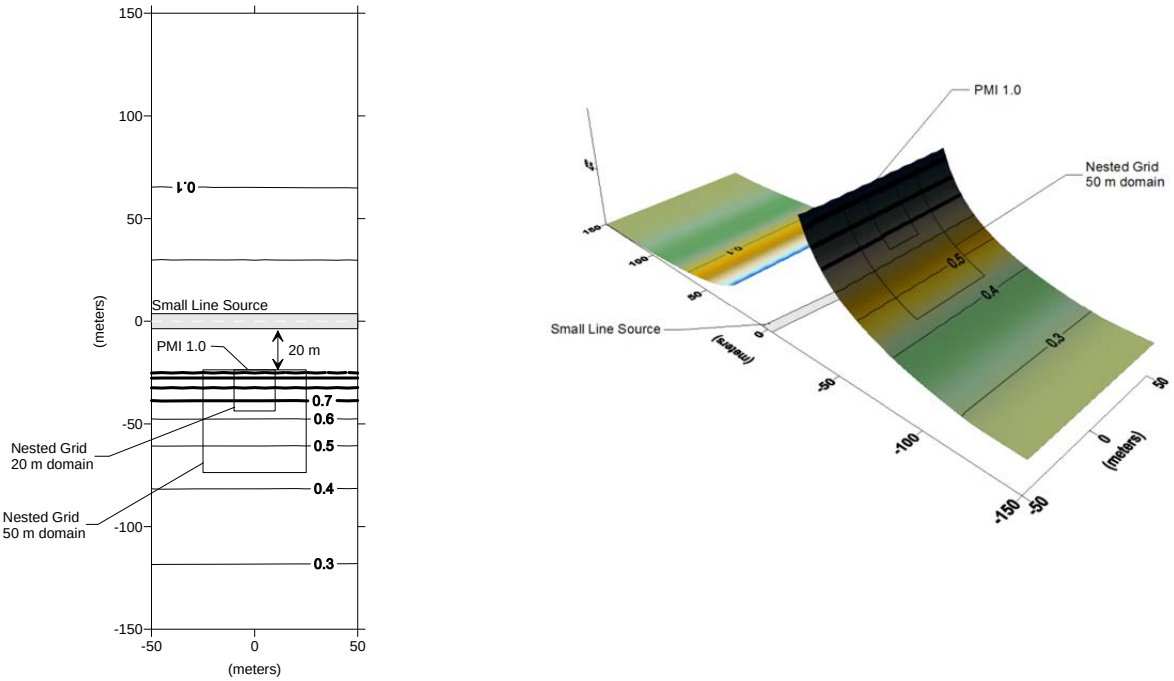


Figure AP C-3.11.2 – Small Line Source, CALINE – Fresno Air Terminal

Isopleths ***Surface***

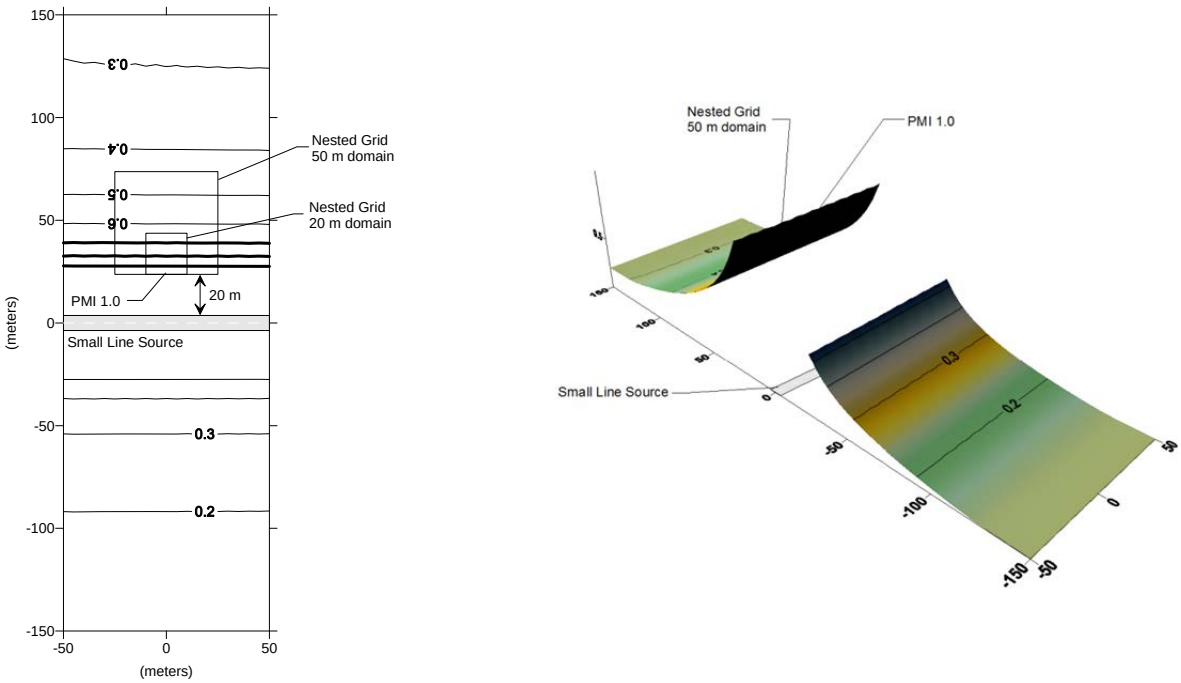
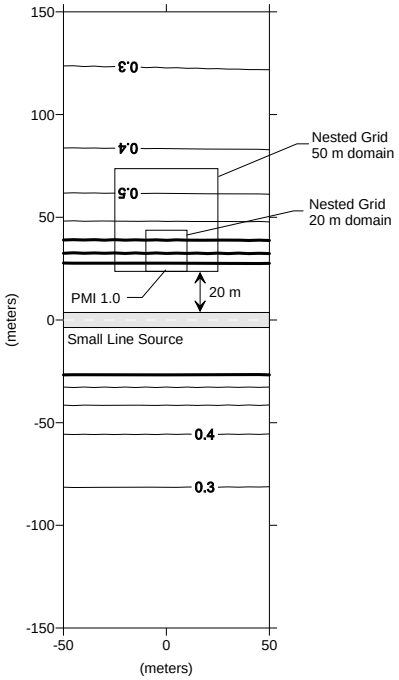


Figure AP C-3.11.3 – Small Line Source, CALINE – Kearny Mesa

Isopleths ***Surface***



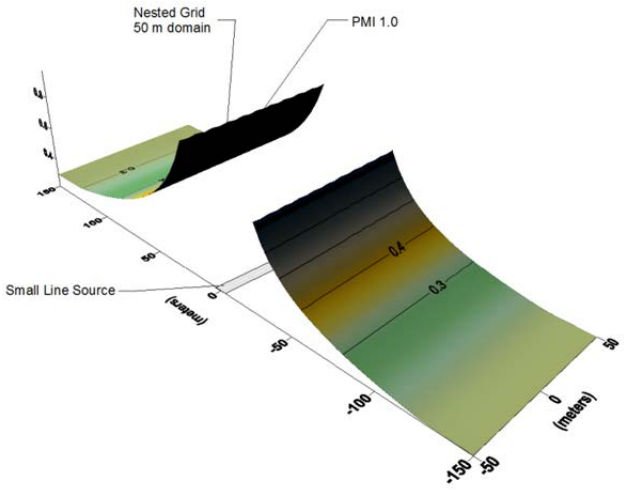
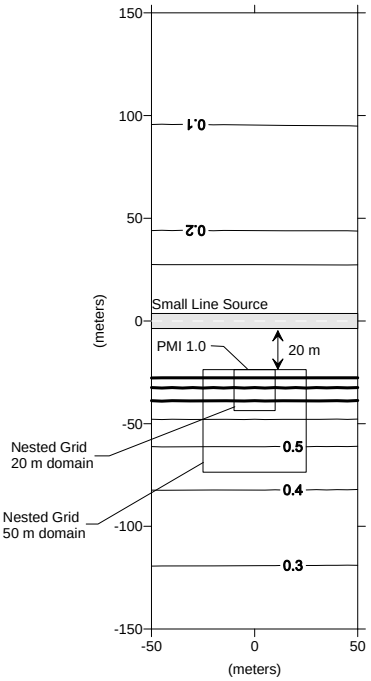


Figure AP C-3.11.4 – Small Line Source, CALINE – Lynwood

Isopleths ***Surface***



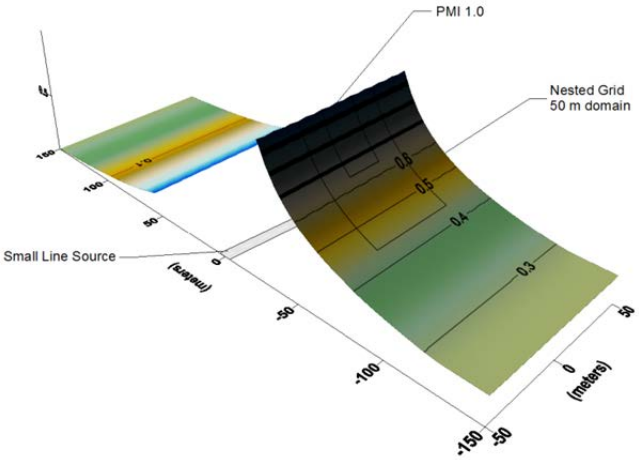
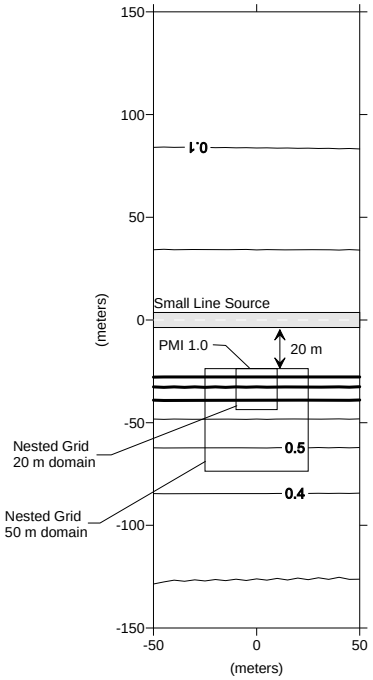
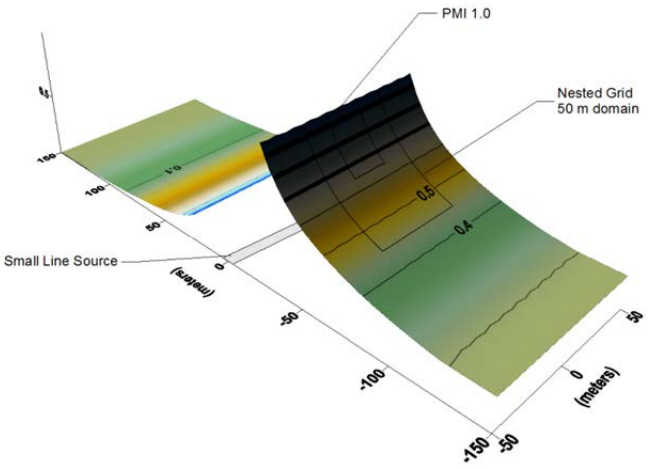


Figure AP C-3.11.5 – Small Line Source, CALINE – San Bernardino

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

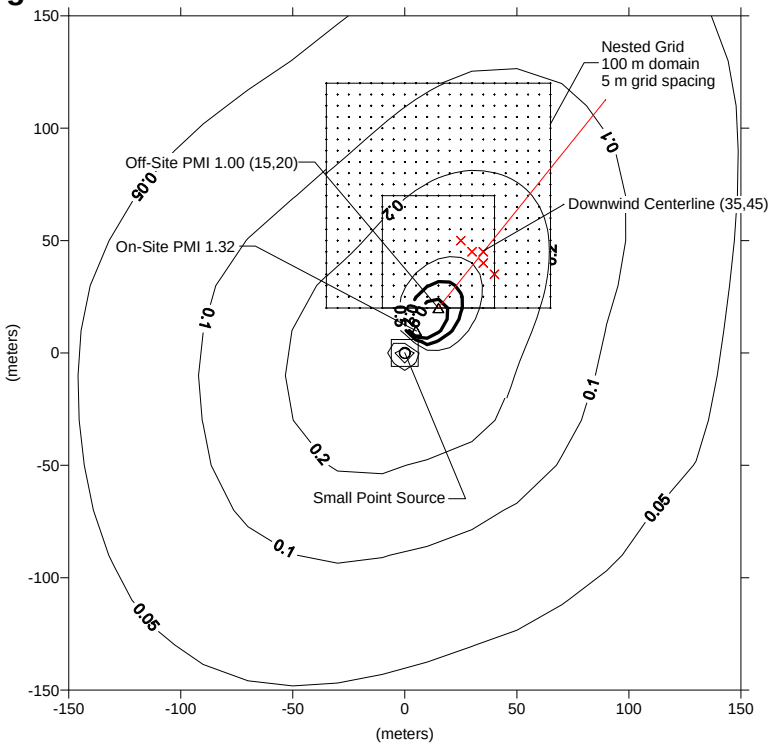
Tilted Spatial Averaging

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

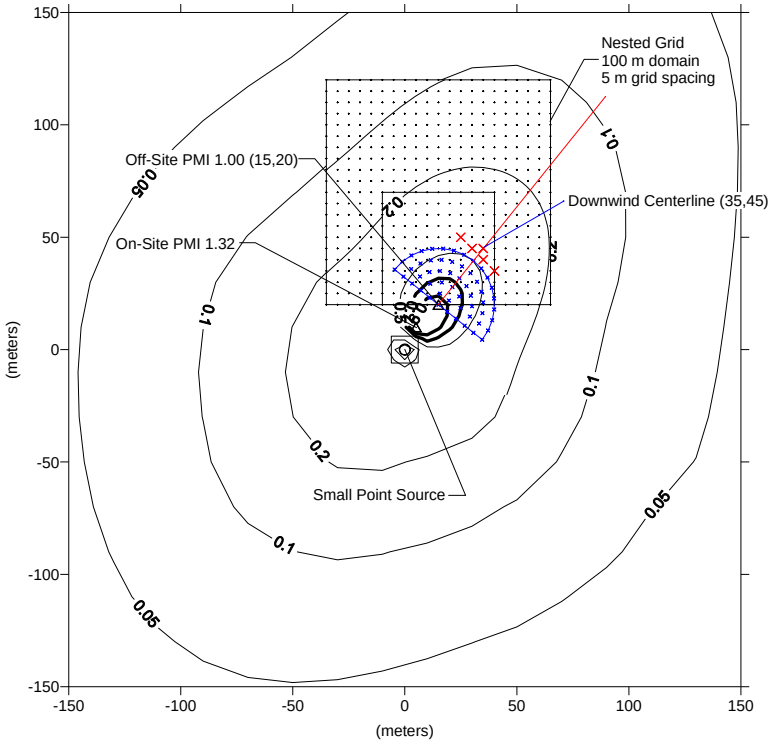
As a result of the above receptor spacing, the following field of polar receptors in Table AP **C-5.2** is needed for the San Bernardino example.

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

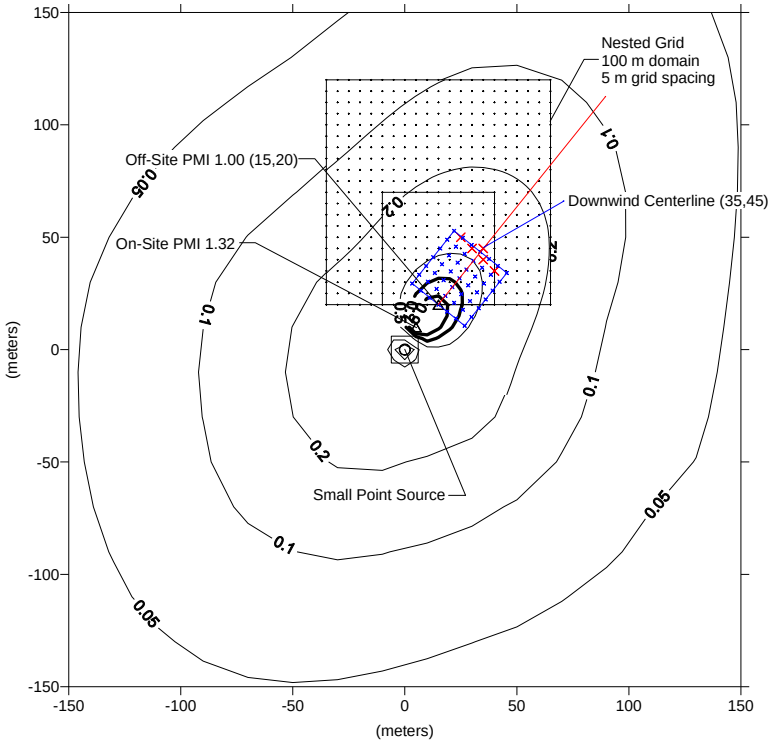


Table AP C-5.3.1 – Spatial Average – San Bernardino – Small Point Source

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

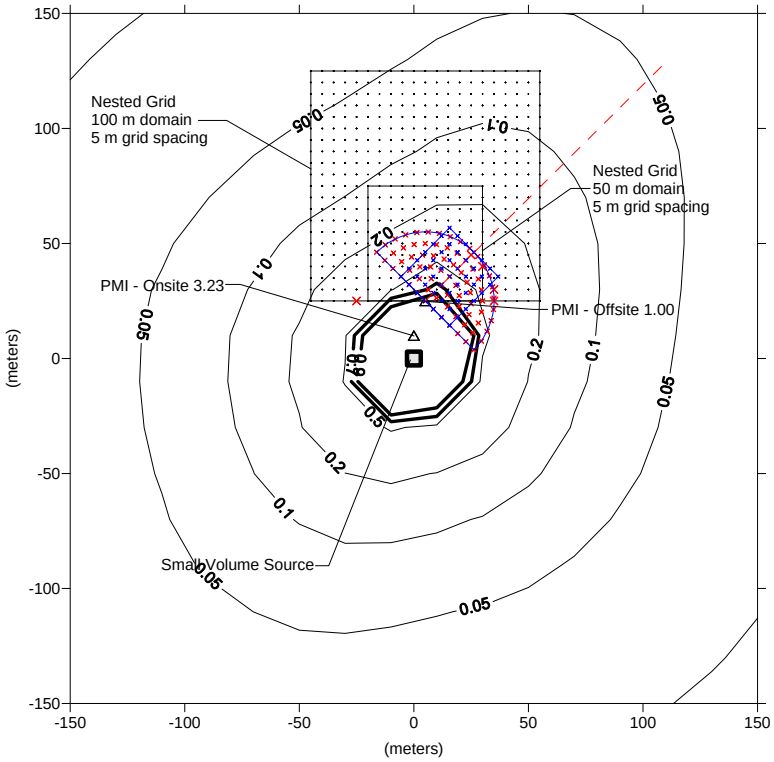


Table AP C-5.3.2 – Spatial Average – San Bernardino – Small Volume Source

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

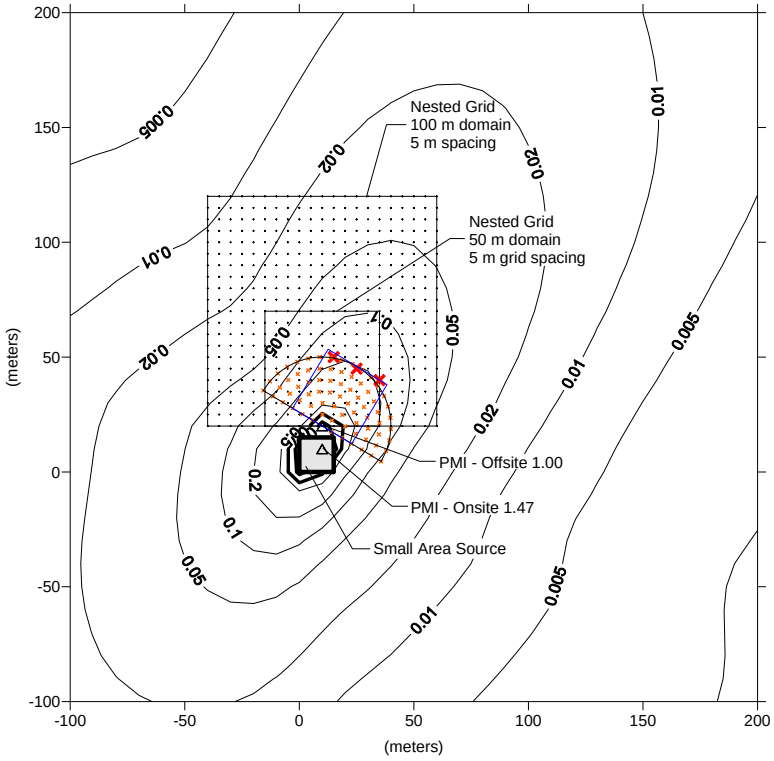


Table AP C-5.3.3 – Spatial Average – San Bernardino – Small Area Source

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

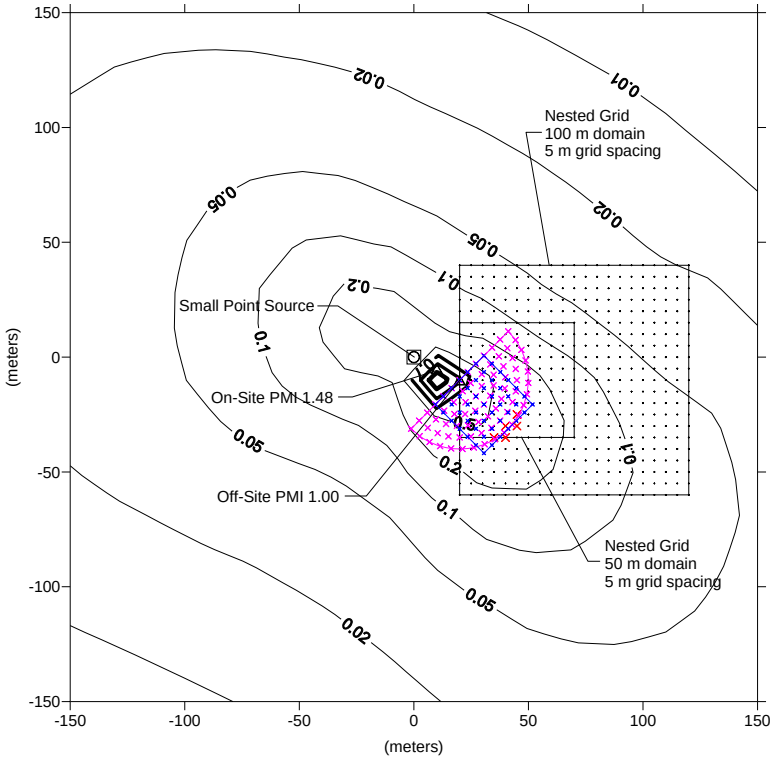


Table AP C-5.4.1 – Spatial Average – Fresno Air Terminal – Small Point Source

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

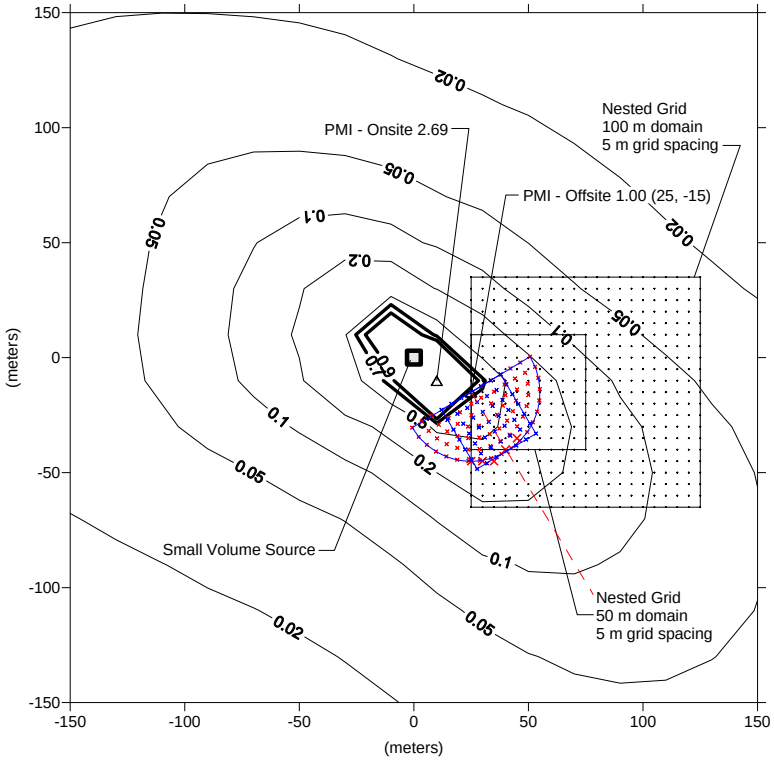


Table AP C-54.2 – Spatial Average – Fresno Air Terminal – Small Volume Source

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

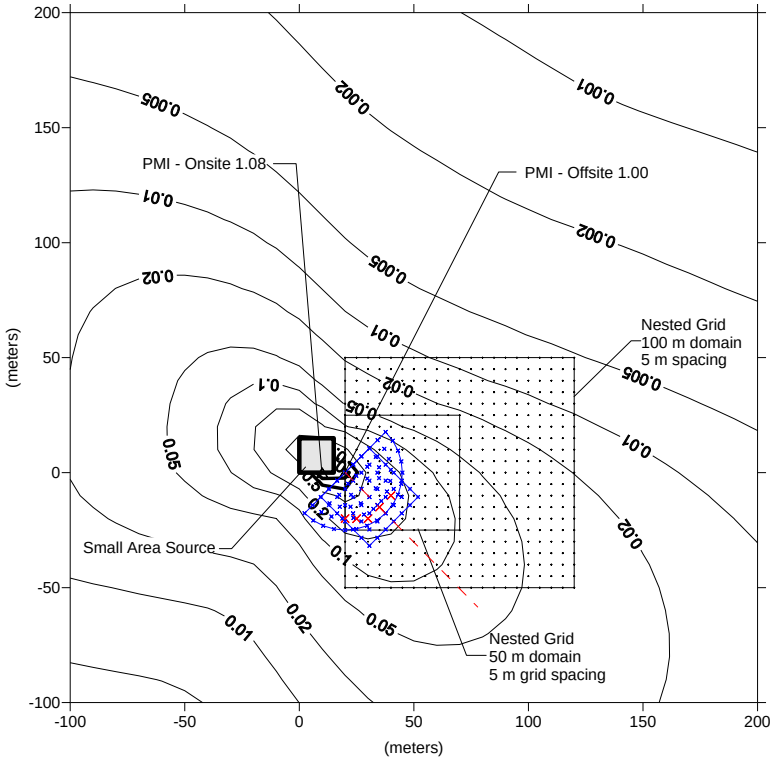


Table AP C-5.4.3 – Spatial Average – Fresno Air Terminal – Small Area Source

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

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
33.0	Vegetable stew without meat	75439010
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

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Walnuts	42116000
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

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Pomegranate, raw	63145010
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

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Corn, cooked, NS as to form, NS as to color, NS as to fat ad	75216000
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

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
100.0	Corn, white, cooked, from frozen, fat added in cooking	75216222
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

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, strained	76405010
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

Table D.3 Food Codes for Protected Produce

% Protected Produce in Food Item	Food Item Description	USDA Food Code
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	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. 6 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. 6 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	2.14×10^{-6}	2.14×10^{-4}	3.46×10^{-4}
Acenaphthene (3 fused rings)	3.02×10^{-3}*	25	7	2.23×10^{-5}	2.23×10^{-3}	4.34×10^{-3}
Acenaphthylene (3 fused rings)	6.67×10^{-3}	25	7	1.00×10^{-4}	0.01	7.55×10^{-3}
Anthracene (3 fused rings)	4.2×10^{-6}*	25	7	1.57×10^{-2}	1.57	6.78×10^{-2}
Benzo[a]anthracene (4 fused rings)	4.07×10^{-6}*	25	7	1.42×10^{-1}	14.2	8.15
Chrysene (4 fused rings)	8.81×10^{-8}**	25	7	8.84×10^{-1}	88.4	4.82×10^{-5}
Benzo[a]pyrene (5 fused rings)	9.23×10^{-8}	25	7	8.79×10^{-1}	87.9	60.2
Benzo[b]fluoranthene (5 fused rings)	1.59×10^{-7}	25	7	8.09×10^{-1}	80.9	NA****
Benzo[k]fluoranthene (5 fused rings)	3.7×10^{-8}*	25	7	9.48×10^{-1}	94.8	79.9
Dibenz[a,h]-anthracene (5 fused rings)	6.07×10^{-11}**	25	7	1.00×10^{0}	100	NA****
Indeno[1,2,3cd]-pyrene (6 fused rings)	1.19×10^{-9}**	25	8	9.98×10^{-1}	99.8	NA****
Chlorophenols						
Pentachlorophenol	1.73×10^{-3}*	25	2	3.88×10^{-4}	3.88×10^{-2}	76.9
2,4,6-Trichlorophenol	2.8×10^{-02}*	25	2	2.34×10^{-5}	2.34×10^{-3}	NA****
2,4,5-Trichlorophenol	4.59×10^{-02}*	25	2	1.46×10^{-5}	1.46×10^{-3}	NA****
Nitrosoamines						
N-Nitrosodiethylamine	8.60×10^{-1}***	20	1	7.81×10^{-7}	7.81×10^{-5}	2.67×10^{-5}
N-Nitroso-dimethylamine	8.1***	20	2	8.29×10^{-8}	8.29×10^{-6}	NA****
N-Nitroso-diphenylamine	4.12×10^{2}**	25	2	1.63×10^{-9}	1.63×10^{-7}	NA****
N-Nitrosodi-n-butylamine	3.0×10^{-2}***	20	9	2.24×10^{-5}	2.24×10^{-3}	NA****
N-Nitrosodi-n-propylamine	4.15×10^{-1}***	20	2	1.62×10^{-6}	1.62×10^{-4}	2.75×10^{-4}
N-Nintrosopyrrolidine	7.2×10^{-02}***	20	9	9.2×10^{-6}	9.2×10^{-4}	NA****

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
PCBs						
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

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[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 Ingest	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.

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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 µg/cm^2/min during a 5 minute exposure, to 2.5 µg/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

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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:

$$Csoil = [GLC \cdot (Dep\text{-}rate) \cdot (86{,}400) \cdot (X)] / [Ks \cdot (SD) \cdot (BD) \cdot (Tt)] \quad \textbf{(Eq. G-1)}$$

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:

$$X = [\{Exp \cdot (-Ks \times Tf) - Exp \cdot (-Ks \times To)\} / Ks] + Tt \quad \textbf{(Eq. G-2)}$$

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

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Contents

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)

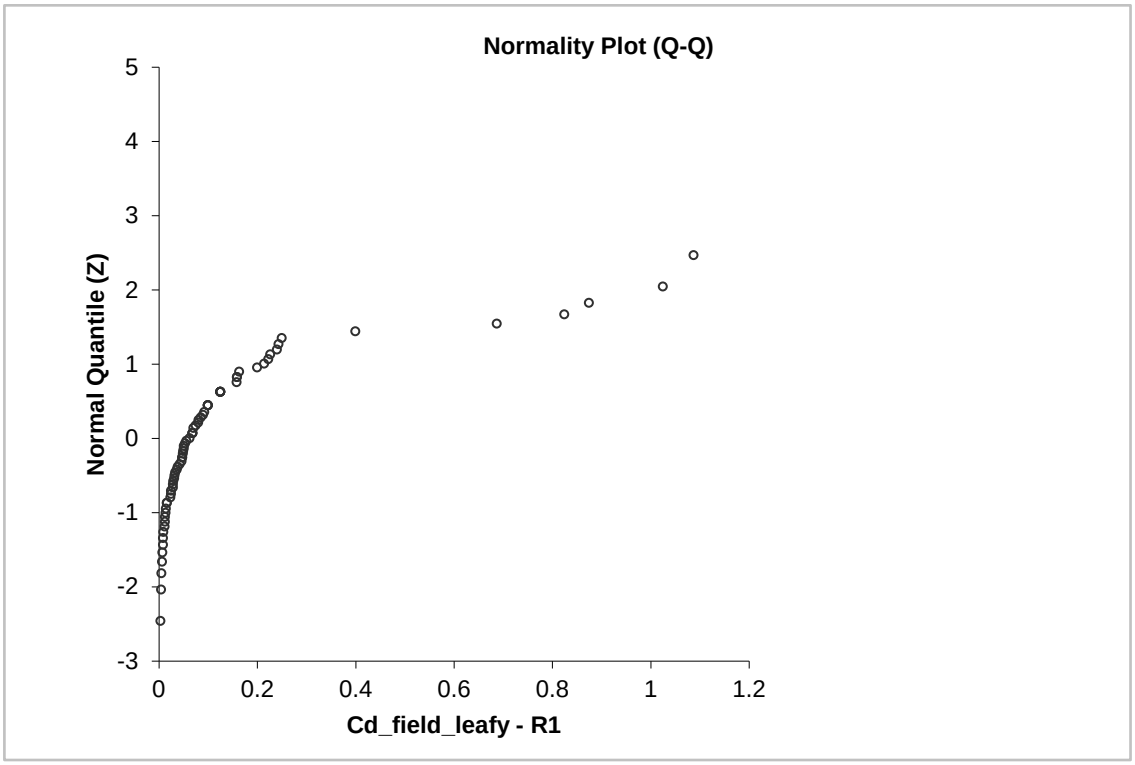

Table H.2 presents the UF distributions from field data only. UFs calculated from laboratory studies in which Cd salts were added to soils were not included in Table H.2, although there are a considerable number of these types of studies. Comparison of UFs calculated from field and Cd salt studies showed 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

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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

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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).

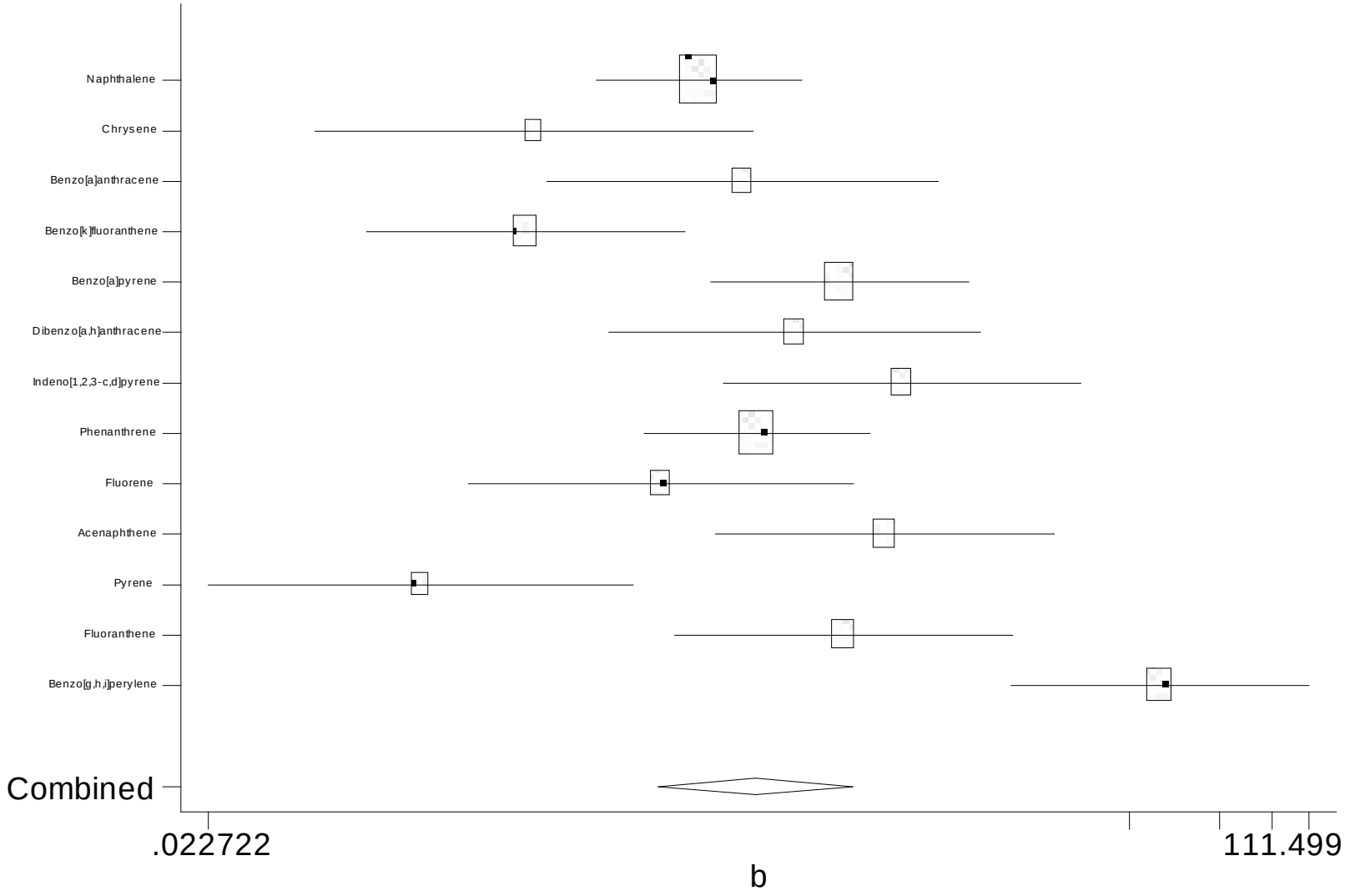
Table J.3-7: Random Effects Estimate and 95% Confidence Intervals of Tcos Stratified by Intake Route and Data Source

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

$$Tco_{hma}=(Cma/C_{blood}^{+})x(C_{blood}^{+}/C_{blood})x(C_{blood}/(C_{air} \times BR))xF_{c1}\,xF_{c2} \qquad \textbf{Eq. J-9}$$

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.

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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.

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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

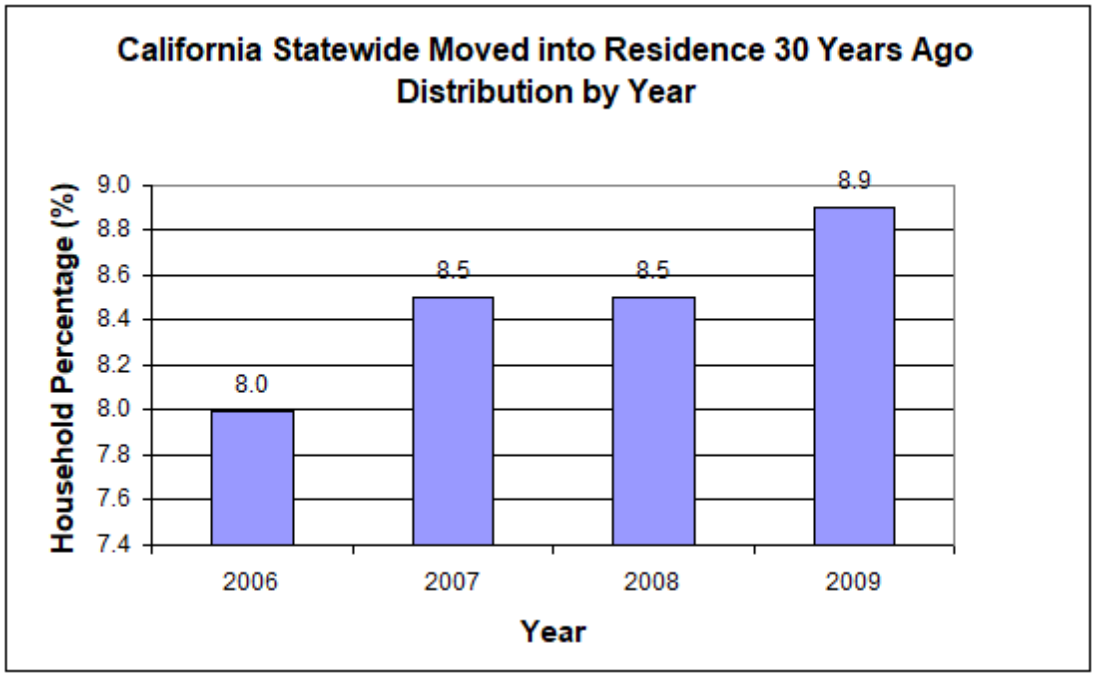
(Weighted Household Percent and Number)

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*

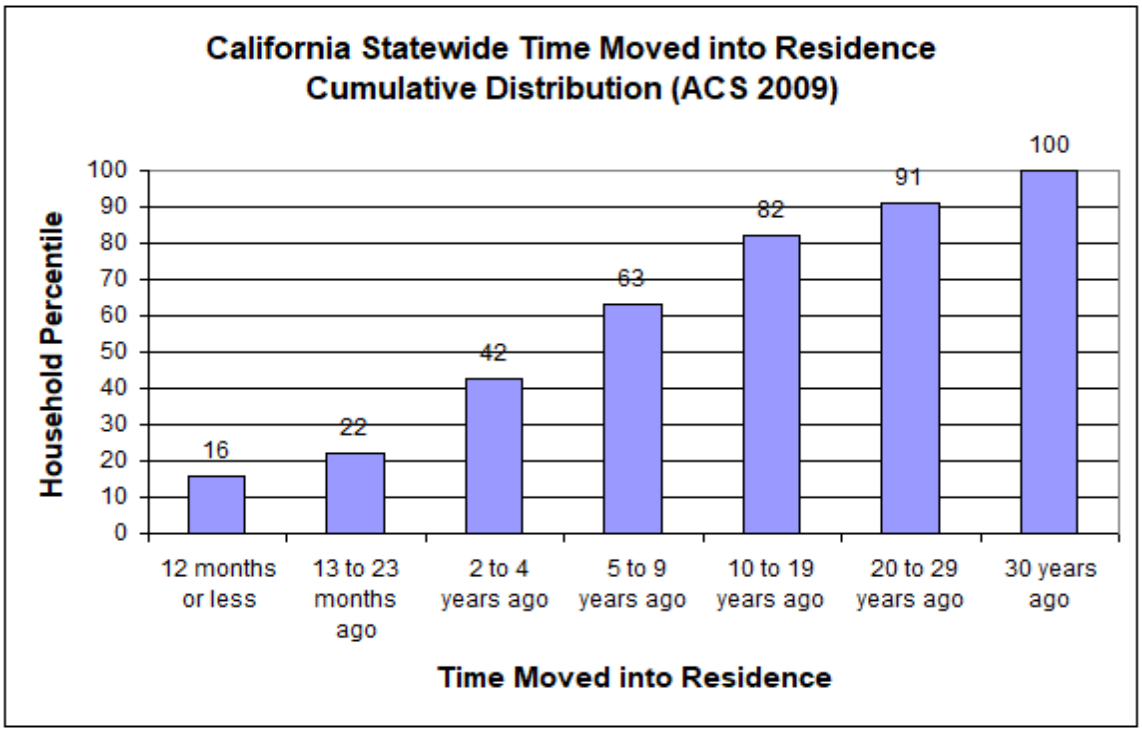


* IPUMS-USA ACS 2006, 2007, 2008, and 2009 single year samples with household weight applied.

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.

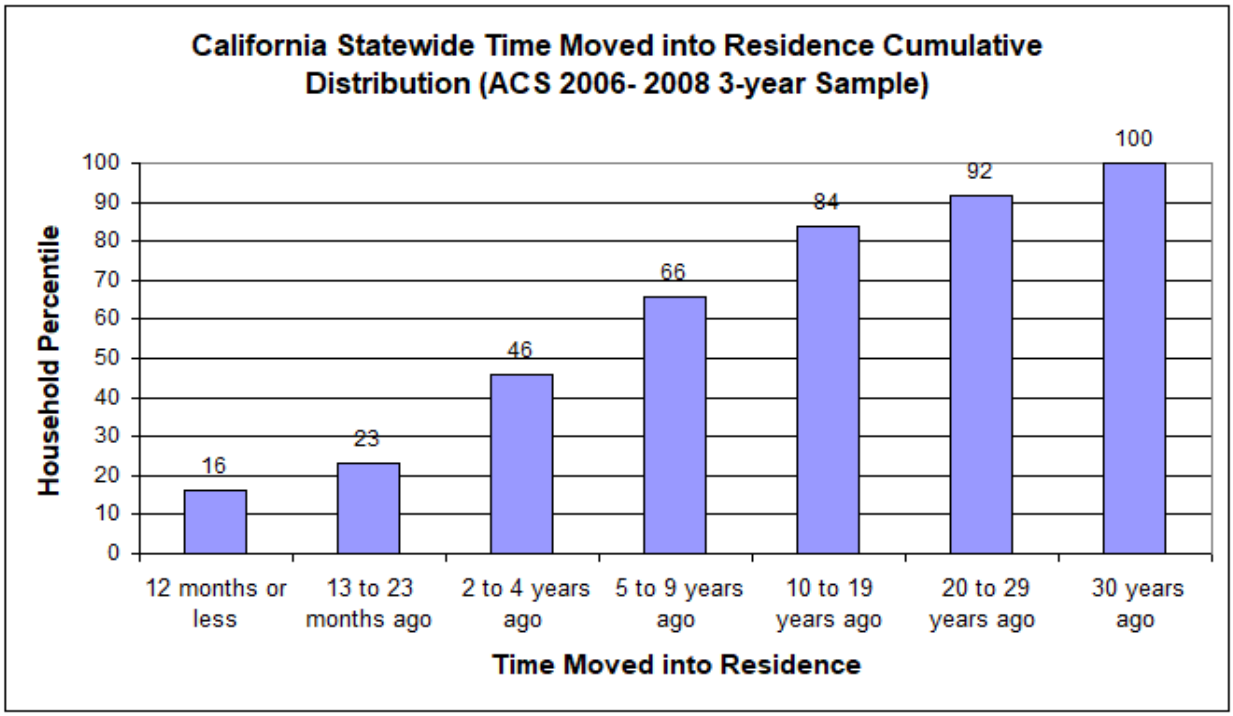
See Supplemental Information section (page 29) for additional information on time moved into residence distributions by California householder's ethnicity, age, and household income from IPUMS-USA ACS 2009 data.

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.

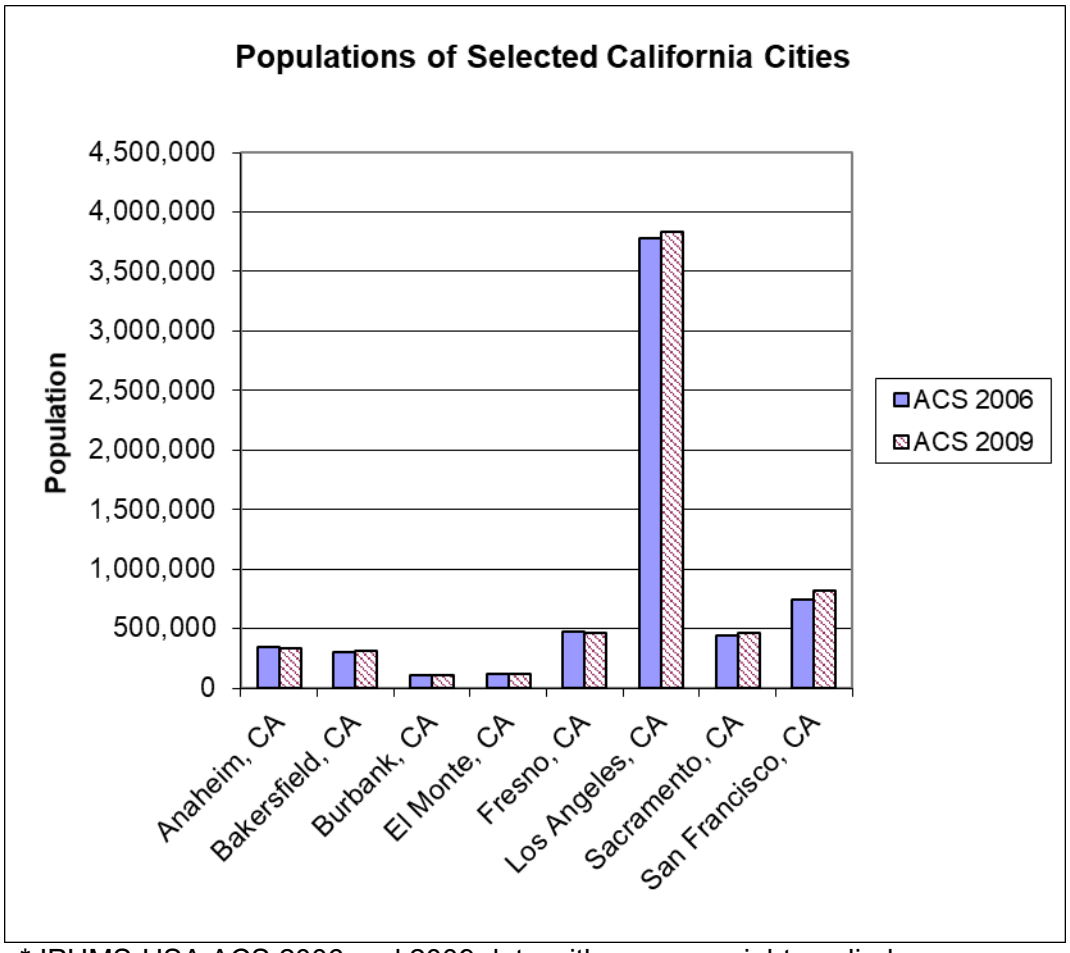
Table L.3* Comparison of Populations of Selected California Cities

(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.

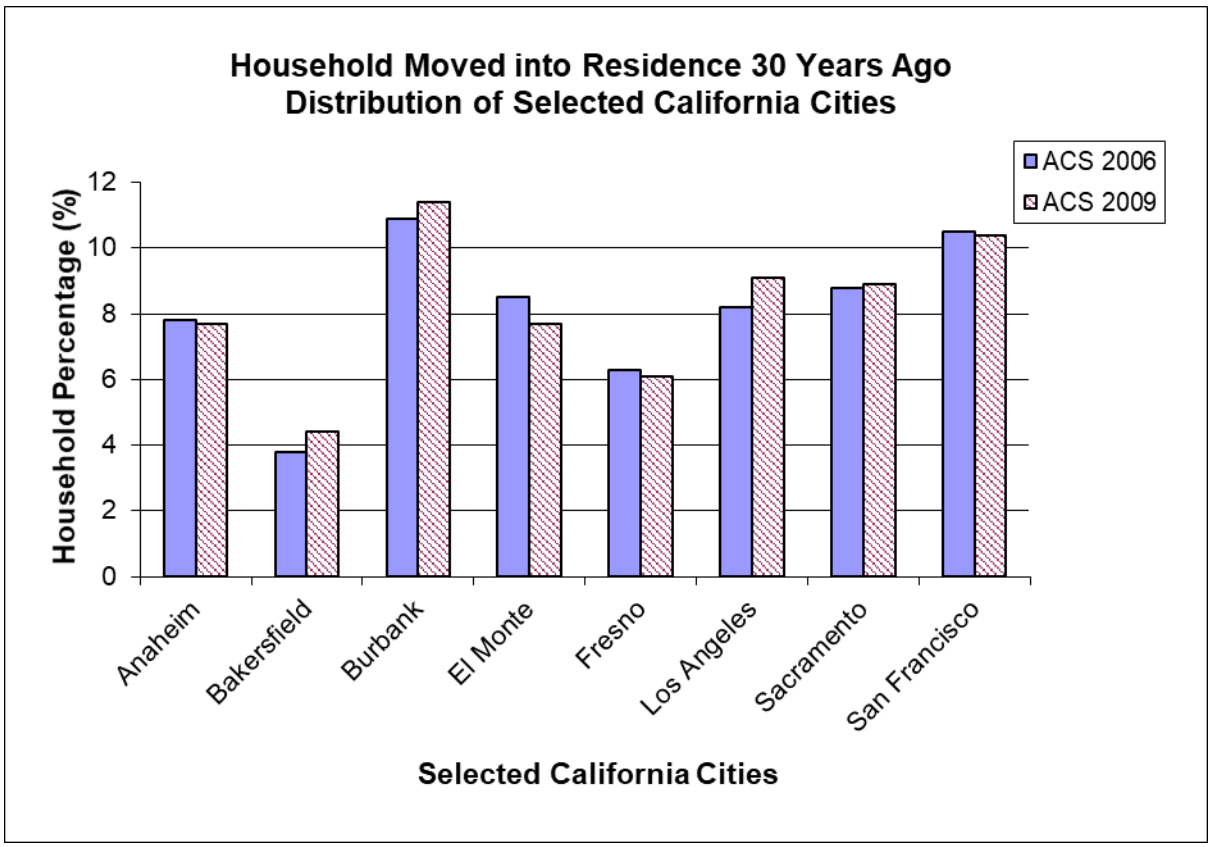
Table L.5* Time Moved into Residence Distribution for Selected California Cities Weighted Household Percent and Samples (IPUMS-USA ACS 2009)

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.

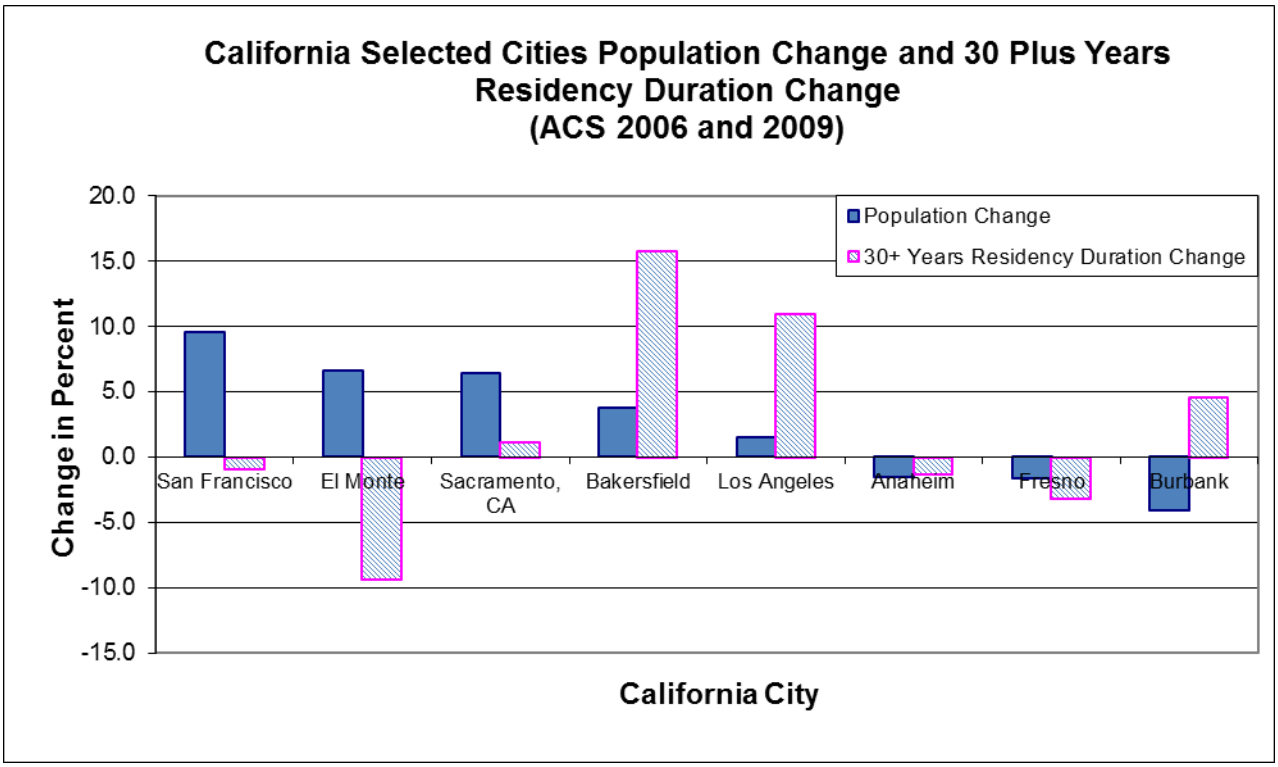
Figure L.5*



* IPUMS-USA ACS 2006 and 2009 data with household weight applied.

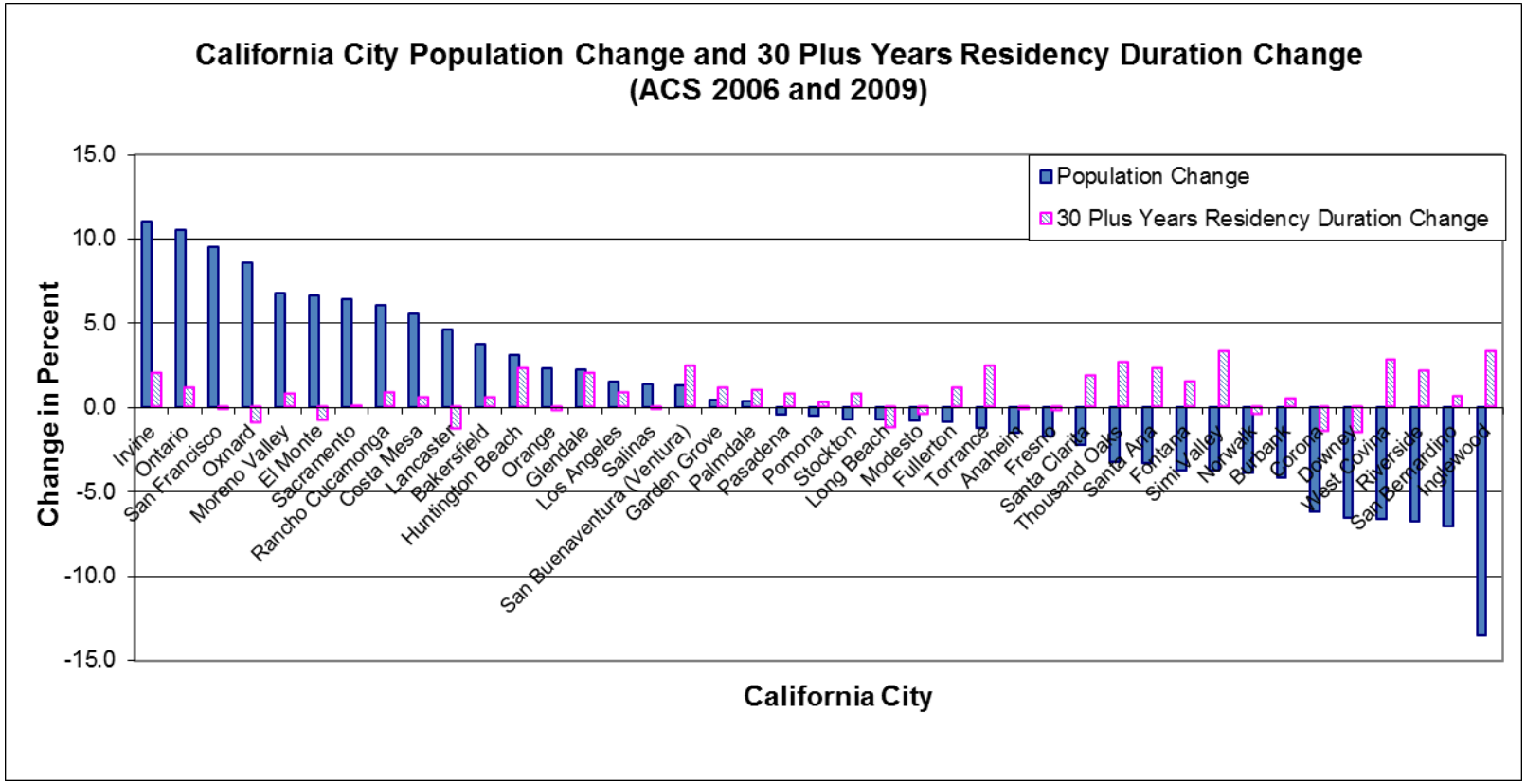
Staff also analyzed the population changes and the 30 years or greater residency duration changes for both the 8 selected cities and the 41 identifiable California cities using IPUMS-USA ACS 2006 to ACS 2009 data. The purpose of this analysis is to see if a rapidly growing city has a different pattern 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).

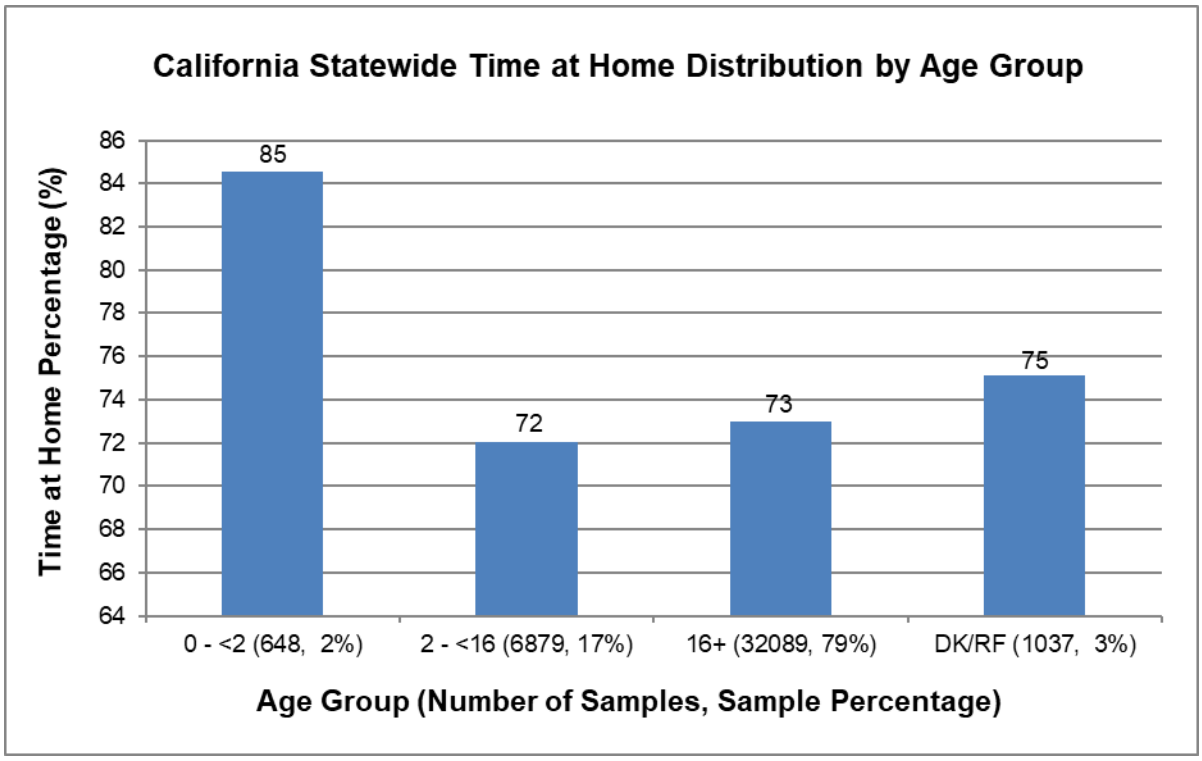
Table L.6 California Statewide Time at Home Distribution by Age Group

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.

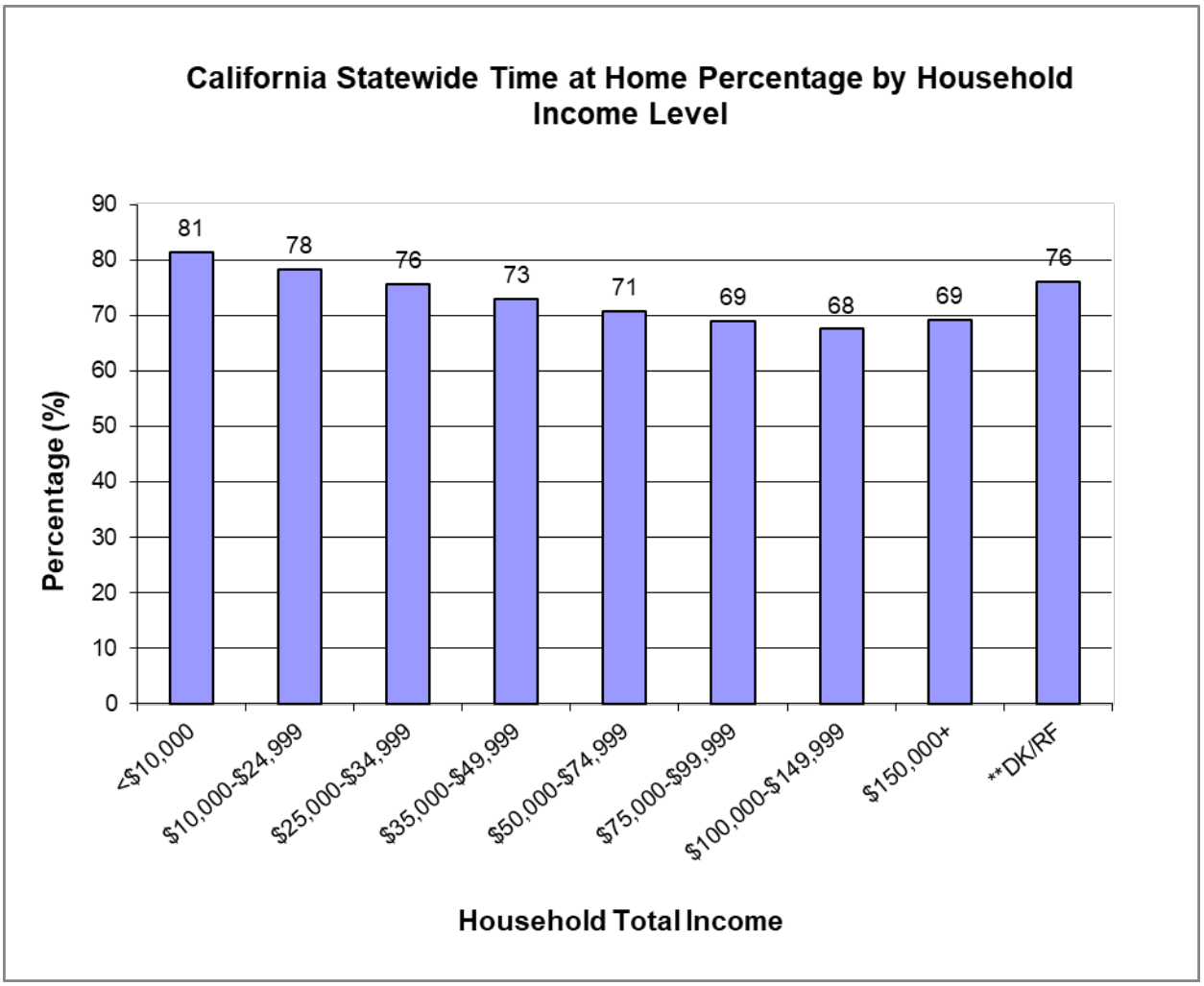
Table L.7 California Statewide Time at Home Distribution by Household Income Level

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.

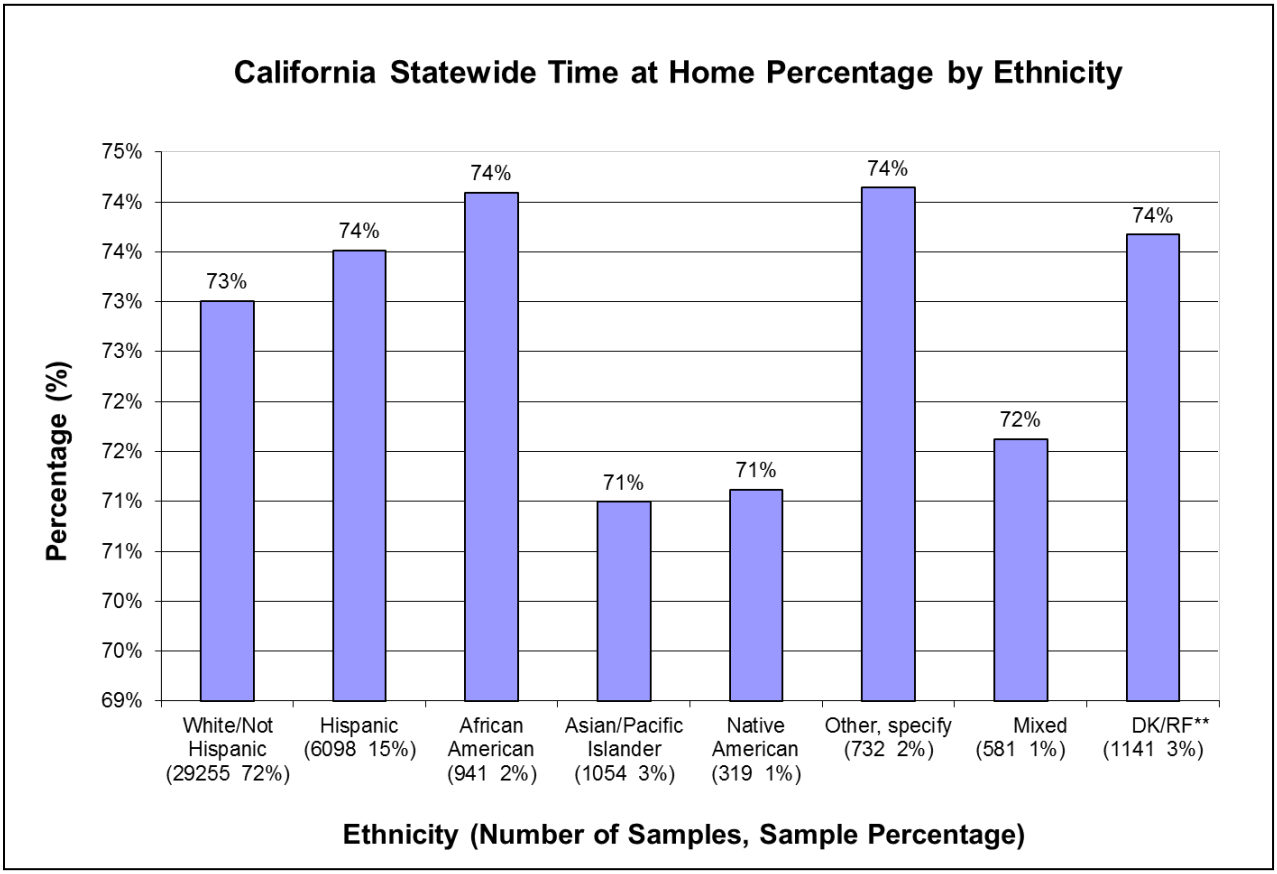
Table L.8 and Figure L.10 show California statewide time spent at home distribution by ethnicity. They depict that all the ethnic groups spend 71% to 74% time at home per day. The N/A in the ethnicity group in Table L.8 means the description of the ethnicity code 6 in the database is not available. The 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.

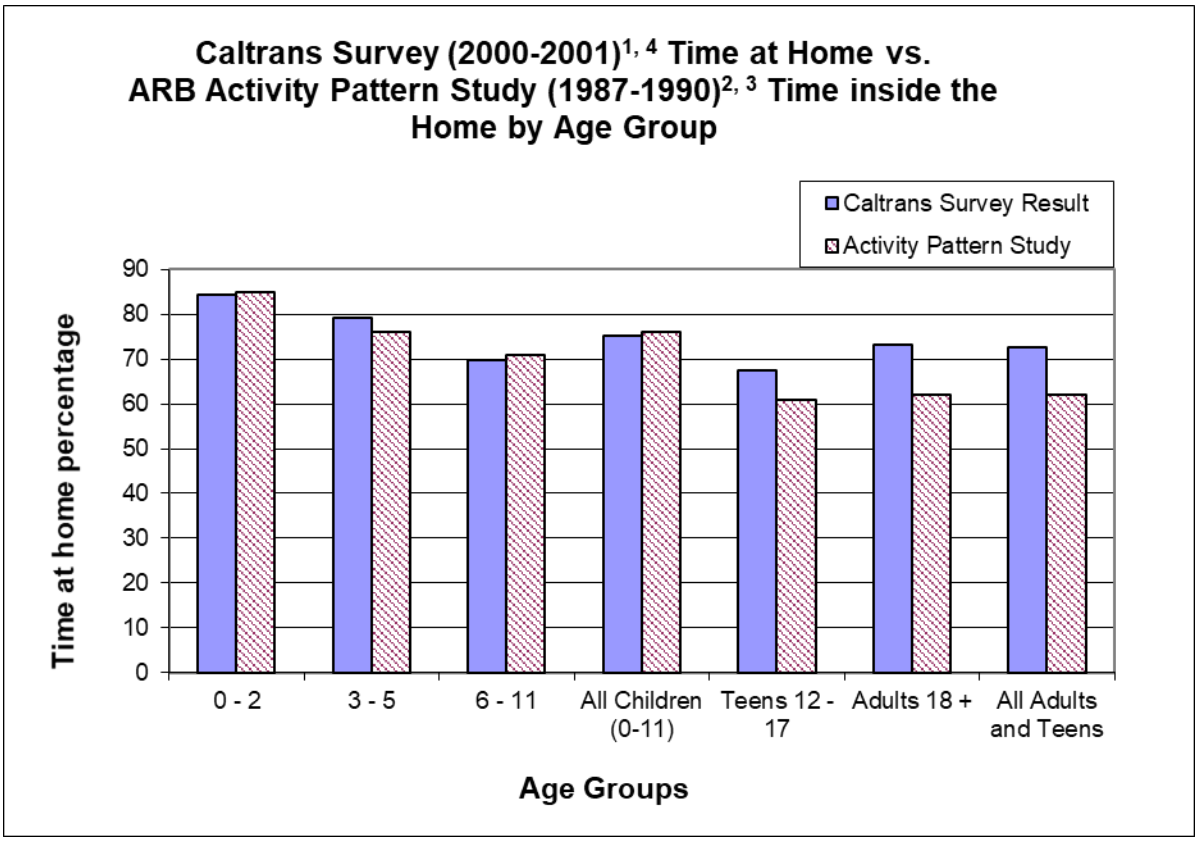
Table L.9 Caltrans Survey (2000-2001) Time at Home vs. ARB Activity Pattern Study (1987-1990) Time inside the Home by Age Group

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.

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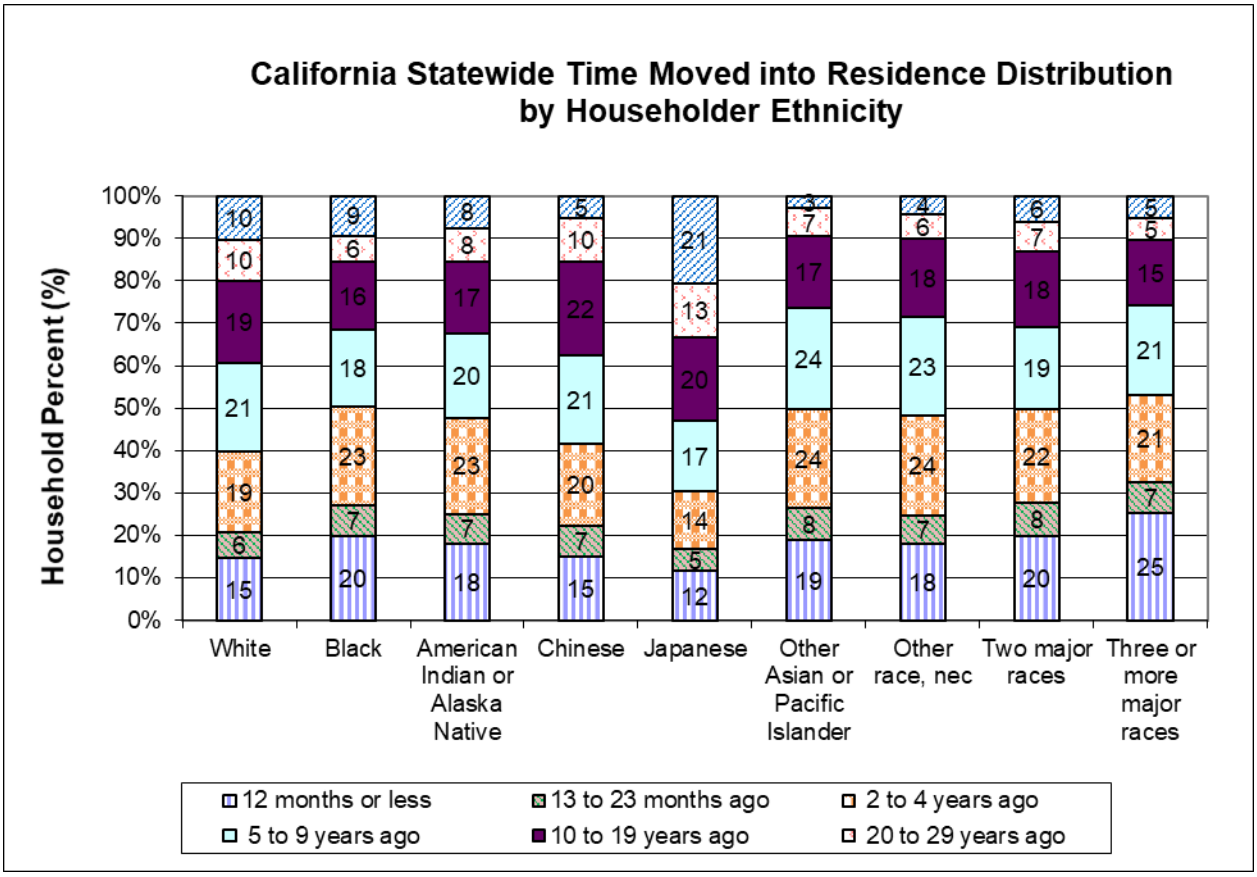
(Klepeis NE, Nelson W C, Ott W R, Robinson, J-Pm Tsang A M, Switzer P, Bhar J, Hem S C, Engelmann W H. (1995) National Human Activity Pattern Survey. Klepeis et al., 1995. http://eetd.lbl.gov/ie/viaq/pubs/LBNL-47713.pdf

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.

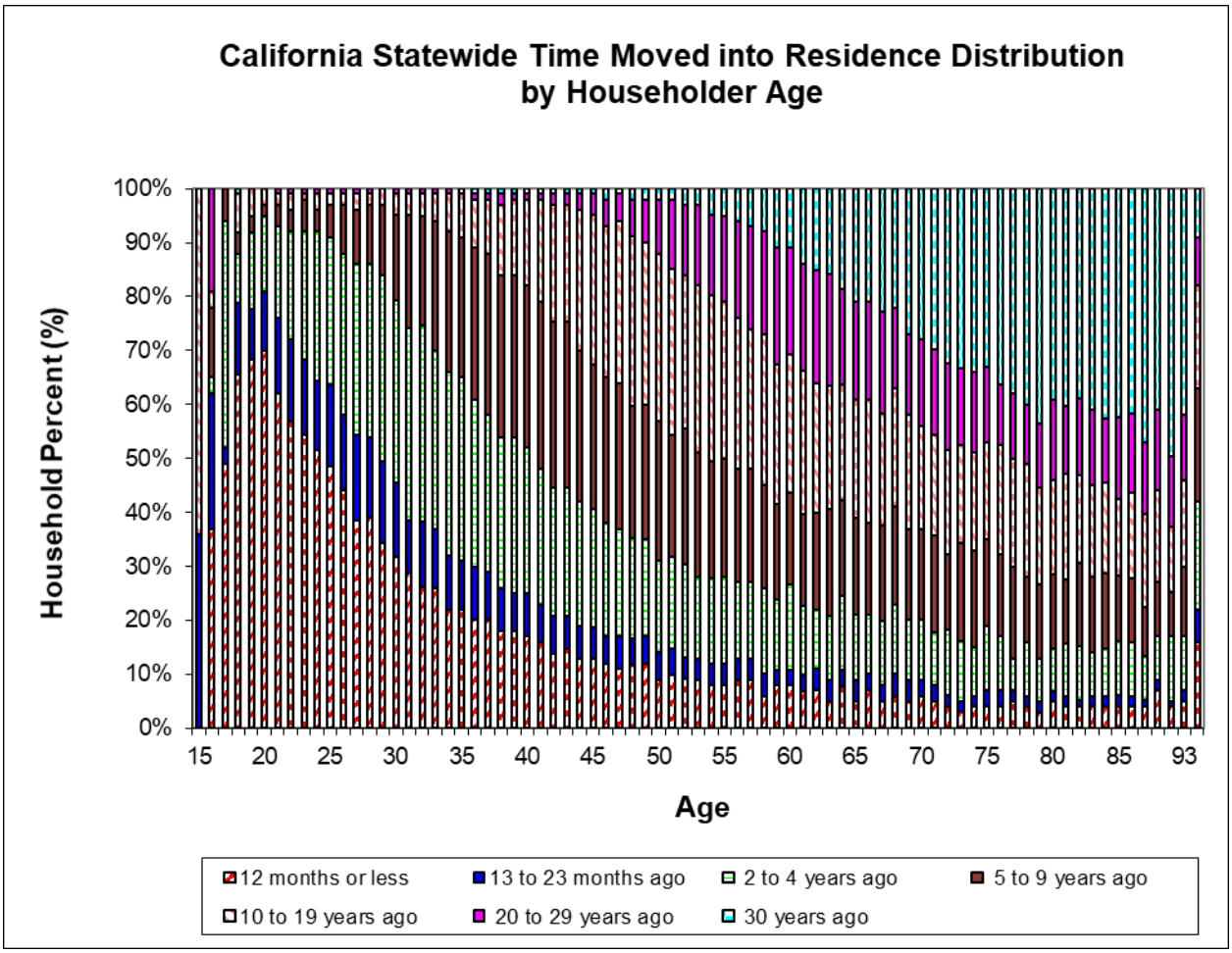
Figure A.1*



* 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.

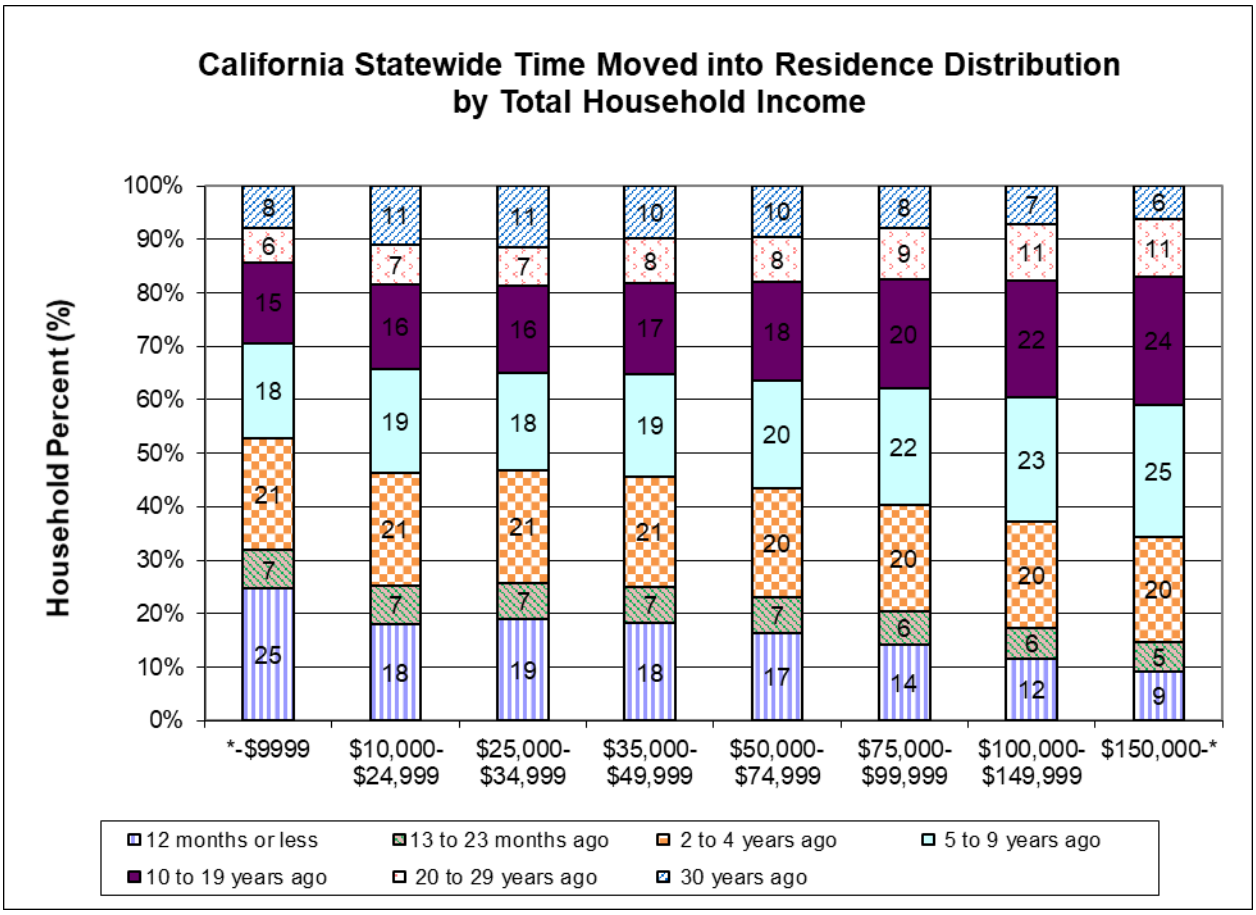
Figure A.2*



* 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 µg/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 (µg/m^3)	5	7	8	0	9	11	5	1	12	3
Source 2 Concentration(µg/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

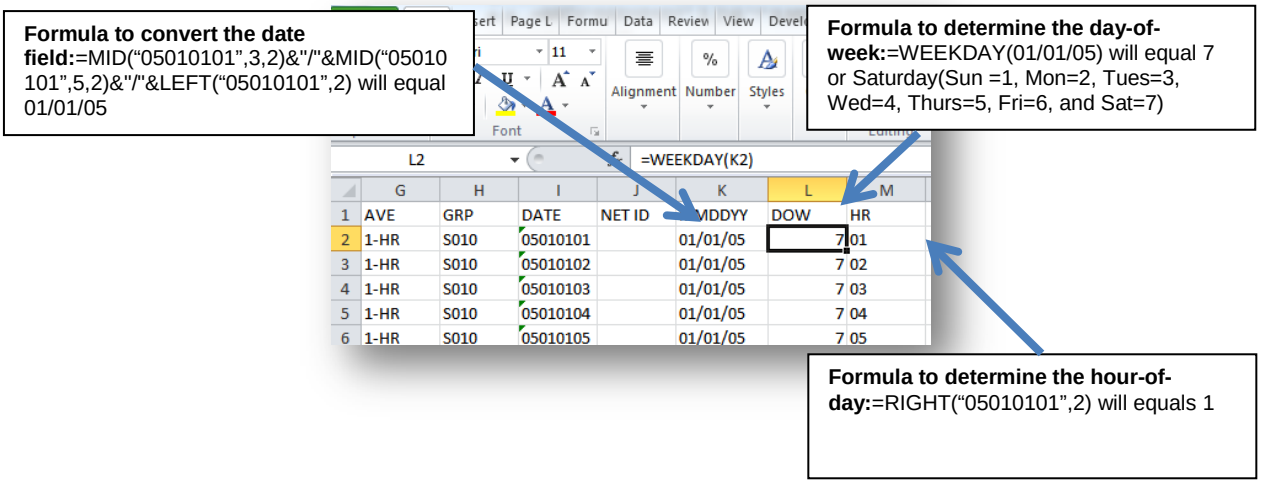
Figure M.3.1. Sample of an AERMOD POSTFILE

```
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).

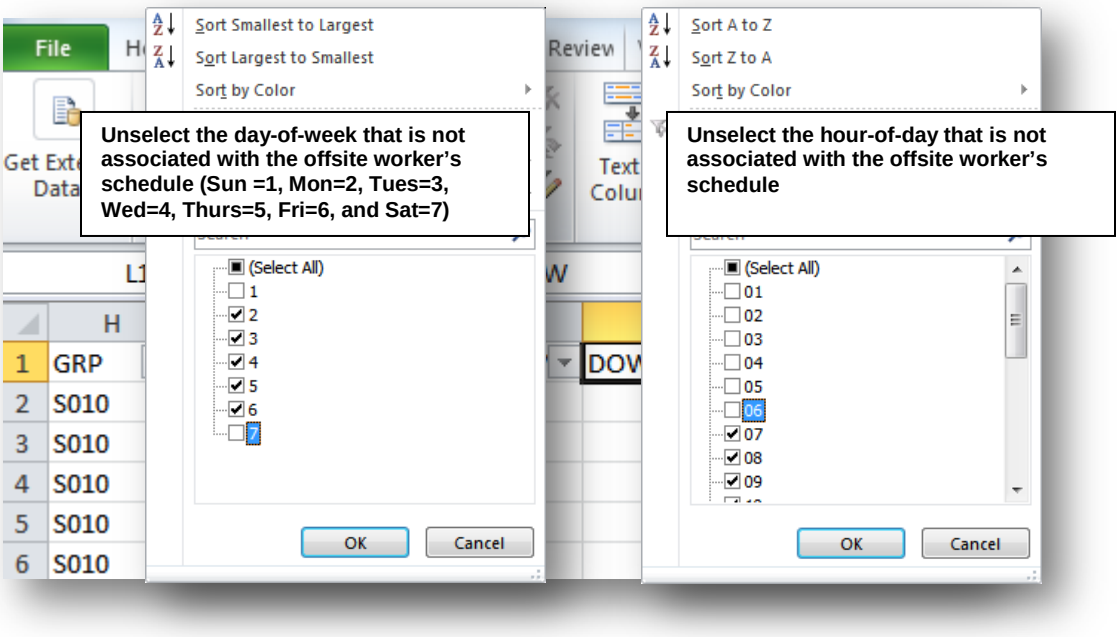
Figure M.3.2. How to Determine the Day-of-Week and Hour-of-Day in Microsoft Excel



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

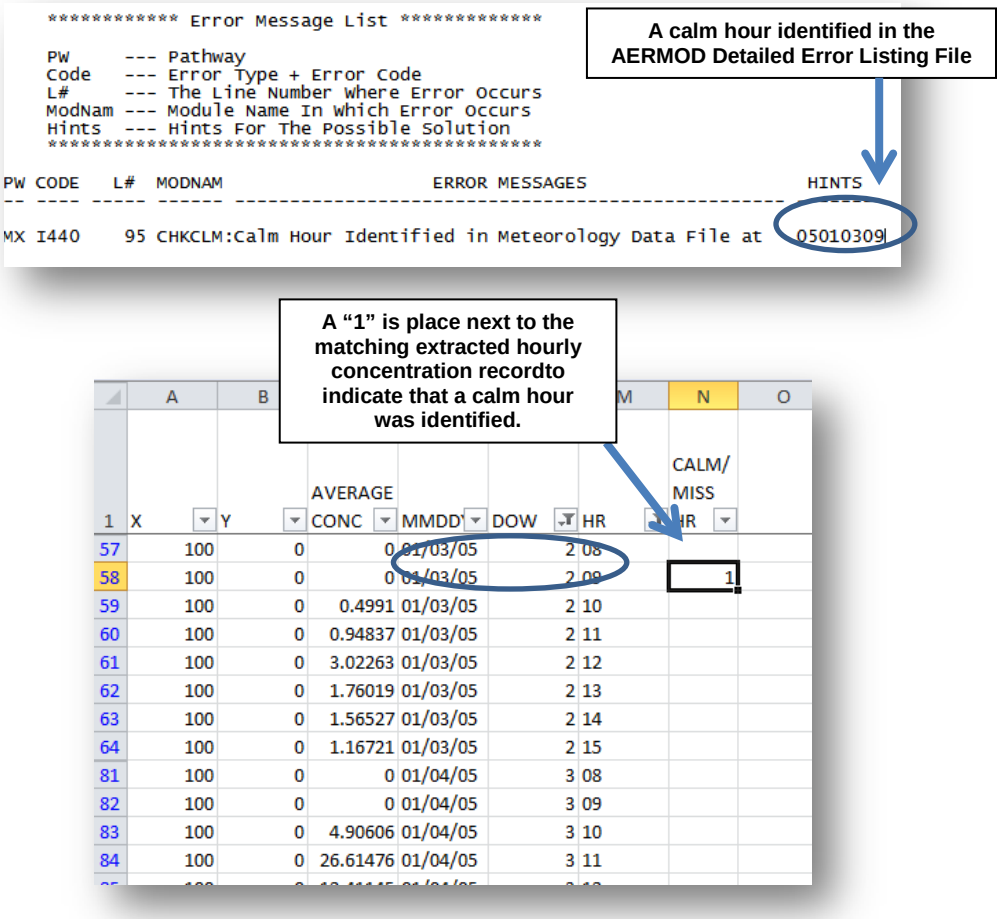
A	B	C	D	E	F	G	H	I	J	K	L
X	Y	AVERAGE CONC	ZELEV	ZHILL	ZFLAG	AVE	GRP	DATE	NET ID	MMDD	DOW
100	0	110.2656	10	10	1.2	1-HR	S010	05082610		08/26/05	
100	0	105.365	10	10	1.2	1-HR	S010	05082315		08/23/05	
100	0	105.1168	10	10	1.2	1-HR	S010	05080512		08/05/05	
100	0	103.7613	10	10	1.2	1-HR	S010	05071310		07/13/05	
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.

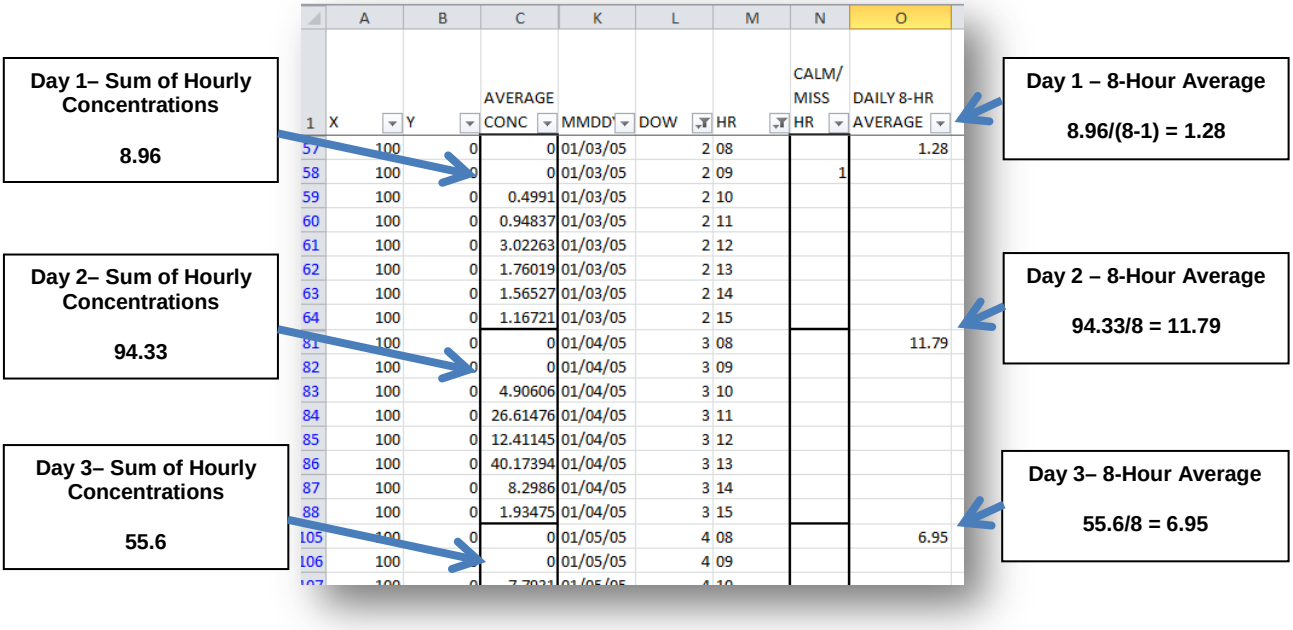
- Using the extracted hourly concentrations based on the steps from Section 3.0, identify any calm and missing hours with a “1”. To do this, use the Detailed Error Listing File that was created from the air dispersion analysis (See Section 2.1 for more information).The Detailed Error Listing File will 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.

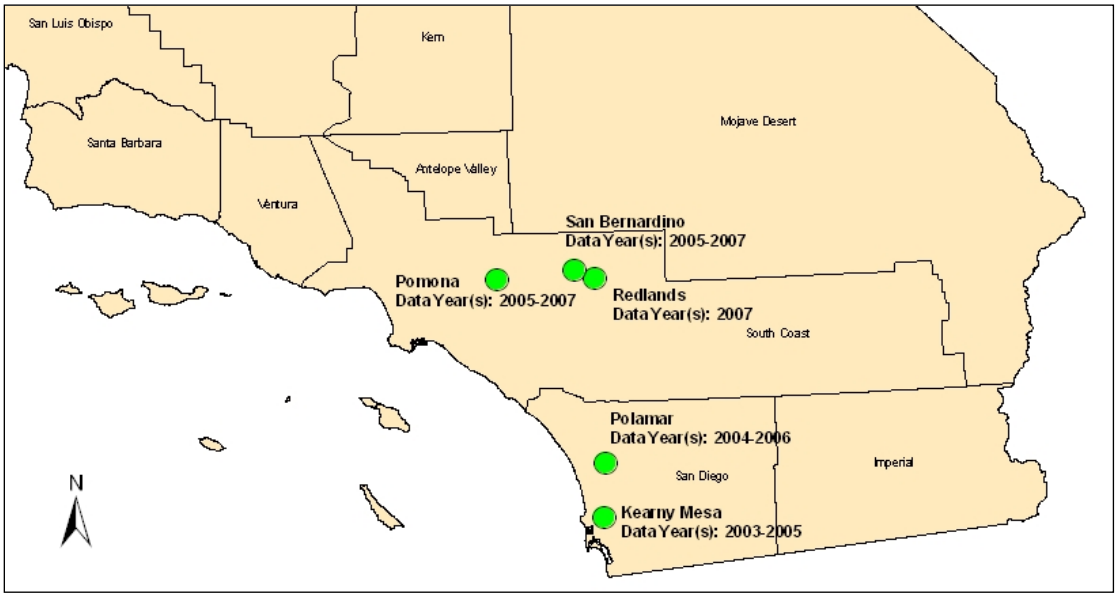
N.3.4. Meteorological Data

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

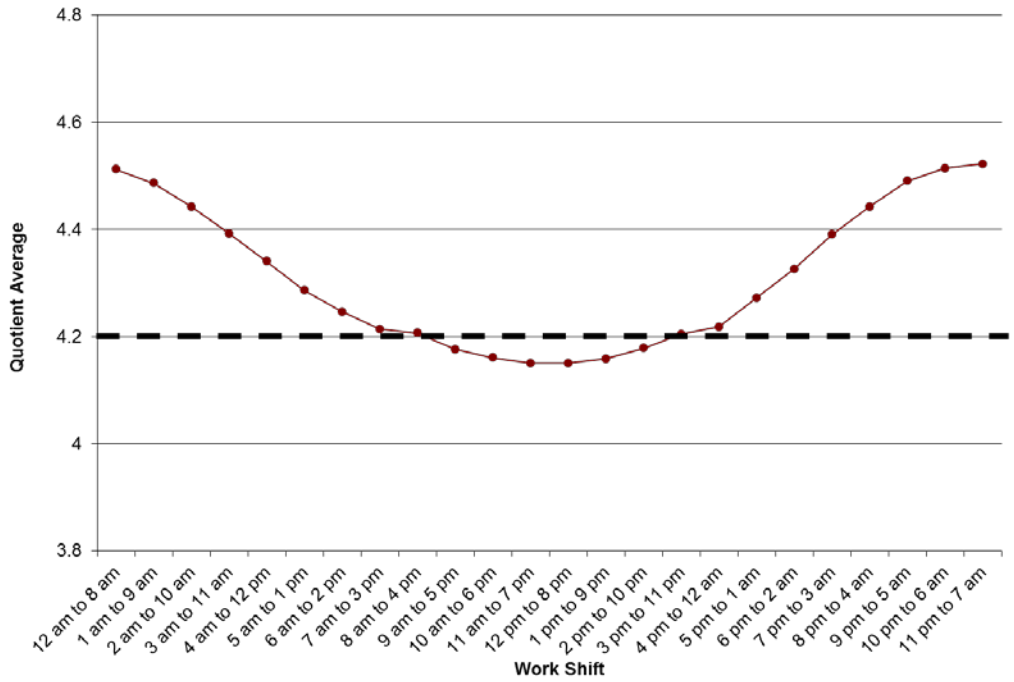


Figure N.3 shows relationship between the worker schedule and the percent of calm and missing hours that occurred during 8-hr work shifts. The figure shows the percent of calm and missing hours are higher during the early morning and evening hour start hours.

Figure N.3. Average Percent of Calm and Missing Hours for 8-Hour Work Shifts

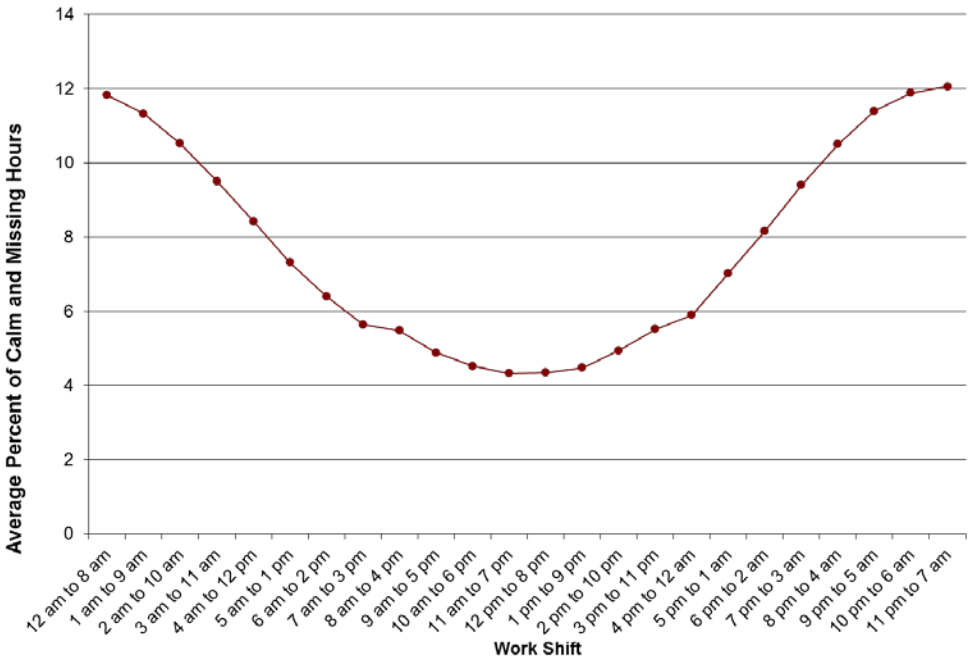


Figure N.4 shows how the post-processed period averages change over 10-hour rolling work shifts. The value at each 10-hour work shift represents the quotient average across the five meteorological data sets. Values that fall on or below the thick dashed line (i.e., the 3.4 worker adjustment factor) indicate 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

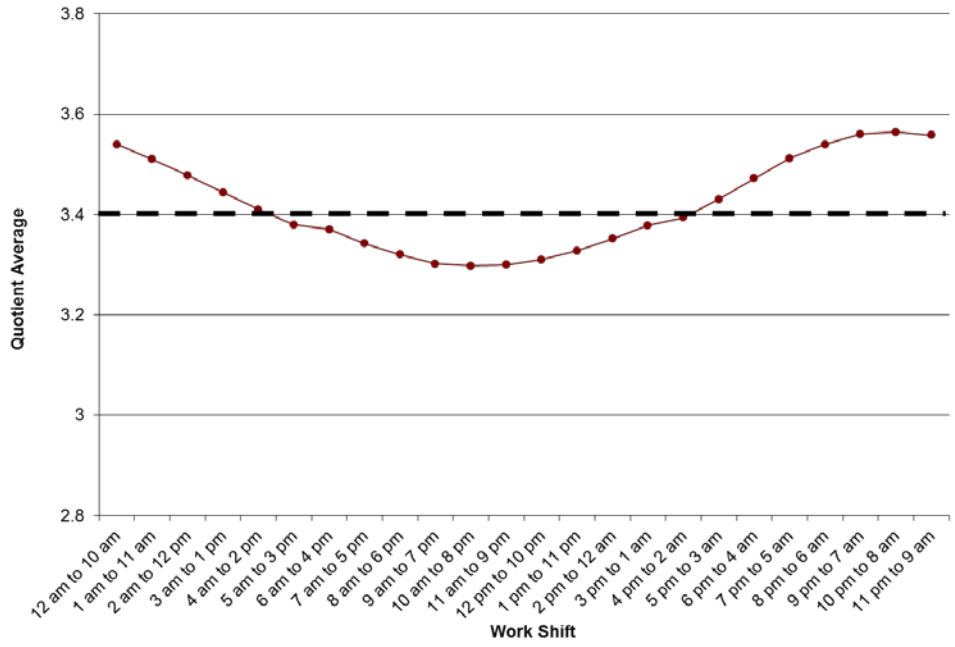


Figure N.5 shows relationship between the worker schedule and the percent of calm and missing hours that occurred during 10-hr work shifts. The figure shows the percent of calm and missing hours are higher during the early morning and evening hour start hours.

Figure N.5. Average Percent of Calm and Missing Hours for 10-Hour Work Shifts

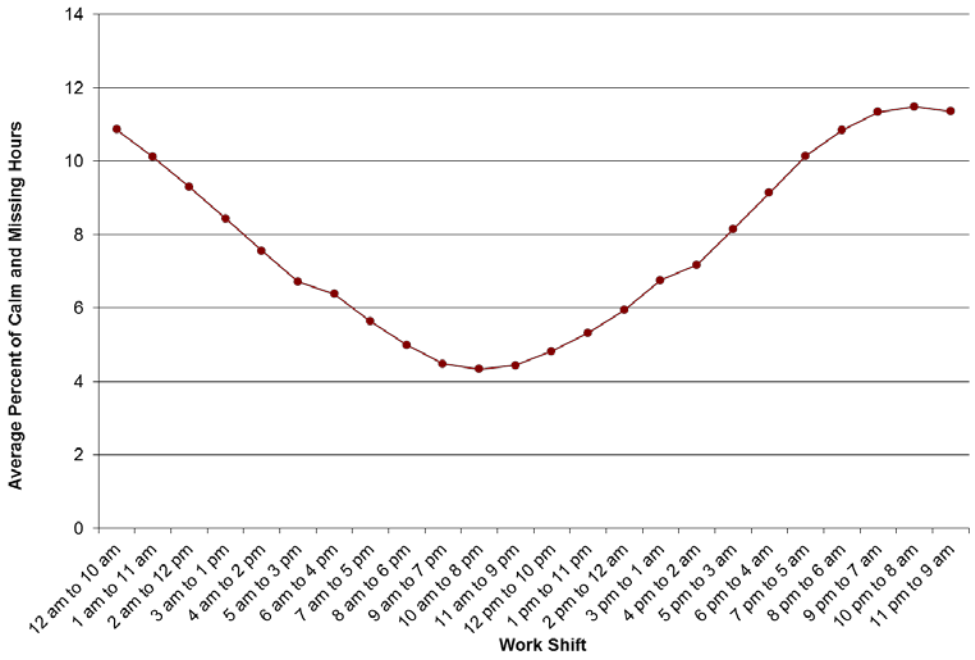


Table N.5 shows the average, minimum, and maximum quotients across all 24 8-hour work shifts for each point source size (i.e., small, medium, and large). The values in the parentheses are the range across the 24 work shifts for each meteorological data set.

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**