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Air Toxics Hot Spots Program Risk Assessment Guidelines

The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments

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September 2014

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Preface

The draft of the *Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments* (Guidance Manual) is a description of the algorithms, recommended exposure variates, cancer and noncancer health values, and the air modeling protocols needed to perform a health risk assessment (HRA) under the Air Toxics Hot Spots Information and Assessment Act of 1987(Health and Safety Code Section 44300 et seq., see Appendix B). The Children's Environmental Health Protection Act of 1999 (Health and Safety Code Section 39606, also contained in Appendix B), which requires explicit consideration of infants and children in assessing risks from air toxics, necessitated revisions of the methods for both noncancer and cancer risk assessment, and of the exposure variates. This draft version of the Guidance Manual updates the previous version (OEHHA, 2003), and reflects advances in the field of risk assessment along with explicit consideration of infants and children.

The information presented in the draft manual is compiled from three technical support documents (TSDs) released by the Office of Environmental Health Hazard Assessment (OEHHA) for the Hot Spots Program. The three TSDs (which are also revised versions, replacing the original four Hot Spots TSDs adopted between 1999 and 2003) underwent public comment and peer review and were adopted for use in the Air Toxics Hot Spots program by the Director of OEHHA. The Technical Support Document for the Derivation of Noncancer Reference Exposure Levels (June, 2008) addressed the methodology for deriving acute, chronic and eight hour Reference Exposure Levels. The Technical Support Document for Cancer Potency Factors (May 2009) addresses the methodology for deriving cancer potency factors and adjusting cancer potency to account for the increased sensitivity of early-in-life exposure to carcinogens. The Technical Support Document for Exposure Assessment and Stochastic Analysis (June 2012) presents the exposure model for the Hot Spots program and reviews the available literature on exposure and relevant fate and transport variates. All three TSDs are available on OEHHA's web site at: http://www.oehha.ca.gov/air/hot_spots/index.html. Excerpts of these three TSDs are presented in this document. There is relatively little new information in the Guidance Manual since the adoption of theTSDs.

This draft Guidance Manual was released for public review. Public comments were received and changes were made in response to some comments. Responses were developed to all public comments. Both the Guidance Manual and OEHHA's response to comments will be reviewed by the State's Scientific Review Panel on Toxic Air Contaminants (SRP), who previously reviewed the three TSDs upon which this guidance is based. Following review by the SRP, OEHHA will finalize this Guidance Manual. When finalized, this Guidance Manual will supersede the risk assessment methods presented in the Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (OEHHA, 2003), which in turn replaced earlier guidance provided by the California Air Pollution Control Officer's Association (CAPCOA, 1993). This draft manual updates health effects values, exposure pathway

variates (e.g., breathing rates), and continues to use a tiered approach for performing HRAs based on current science and policy assessment. The Technical Support Document for Cancer Potency Factors (OEHHA, 2009) recommends a tenfold early-in-life potency factor adjustment for the third trimester and ages zero to less than two, and a threefold adjustment factor for ages two to less than sixteen. In addition, we recommend evaluating residency periods of nine, thirty and seventy years. This means that exposure variates are needed for the third trimester, ages zero to less than two, ages two to less than nine, ages two to less than 16, ages 16 to less than 30, and ages 16 to 70.

The tiered approach presented in this draft manual provides a risk assessor with flexibility and allows consideration of site-specific differences. Furthermore, risk assessors can tailor the level of effort and refinement of an HRA by using the point-estimate exposure variates or the stochastic treatment of distributions of exposure variates. The four-tiered approach to risk assessment primarily applies to residential cancer risk assessment. Compared to the OEHHA 2003 document, the exposure pathways in the Guidance Manual remain the same, the exposure and risk algorithms are similar, but they have been revised to accept new data or variables that are used in the tiered risk assessment approach.

The draft manual also contains example calculations and an outline for a modeling protocol and an HRA report. A software program, the Hot Spots Analysis and Reporting Program (HARP), has been developed by the Air Resources Board in consultation with OEHHA and Air Pollution Control/Air Quality Management District representatives. The HARP software, which is being updated with the new exposure variates and health values, is the recommended model for calculating and presenting HRA results for the Hot Spots Program. Information on obtaining the HARP software can be found on the ARB's web site at *www.arb.ca.gov* under the Hot Spots Program.

The intent of the Guidance Manual and the HARP software is to incorporate children's health concerns, update risk assessment practices, and to provide consistent risk assessment procedures. The use of consistent risk assessment methods and report presentation has many benefits, such as expediting the preparation and review of HRAs, minimizing revision and resubmission of HRAs, allowing a format for facility comparisons, and cost-effective implementation of HRAs and the Hot Spots Program. Risk assessments prepared with this Guidance Manual may be used for permitting new or modified stationary sources, or public notification, and risk reduction requirements of the Hot Spots Program. The use of uniform procedures allows comparison of risks from different facilities and enables identification of facilities that are problematic from a public health perspective. OEHHA reviews the HRAs to insure they are adequate for decision making, but does not play a role in permitting decisions that may result from the HRAs. OEHHA will provide advice to the Districts when requested on any of the risk assessment methods or health values they have used.

References

CAPCOA, 1993. CAPCOA Air Toxics Hot Spots Program Revised 1992 Risk Assessment Guidelines. California Air Pollution Control Officers Association, October 1993.

OEHHA, 2003. Air Toxics Hot Spots Risk Assessment Guidelines: The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments.

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1 - Introduction

1.1 Development of Guidelines

The Air Toxics Hot Spots Information and Assessment Act is designed to provide information to state and local agencies and to the general public on the extent of airborne emissions from stationary sources and the potential public health impacts of those emissions. The Hot Spots Act requires that the Office of Environmental Health Hazard Assessment (OEHHA) develop risk assessment guidelines for the Hot Spots program (Health and Safety Code (HSC) Section 44360(b)(2)) (see Appendix B for the text of the HSC). In addition, the Hot Spots Act specifically requires OEHHA to develop a "likelihood of risks" approach to health risk assessment. In response, OEHHA developed a tiered approach to risk assessment where a point estimate approach is first employed. If a more detailed analysis is needed, OEHHA has developed a stochastic, or probabilistic, approach using exposure factor distributions that can be applied in a stochastic estimate of the exposure. A detailed presentation of the tiered approach, risk assessment algorithms, selected exposure variates (e.g., breathing rate), and distributions with a literature review is presented in the *Air Toxics Hot Spots Program Risk Assessment Guidelines*; *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012). A summary of this information can be found in Chapter 5 of this document.

The Technical Support Document for the Derivation of Noncancer Reference Exposure Levels (OEHHA, 2008) addresses dose response relationships for noncancer health effects and the methodology for deriving acute, chronic and 8-hour Reference Exposure Levels (RELs). Currently there are 53 acute RELs, 82 chronic RELs, and 9 eight-hour RELs. The Technical Support Document for Cancer Potency Factors (OEHHA, 2009) addresses the methodology for deriving cancer potency factors and adjusting cancer potency to account for the increased sensitivity to early-in-life exposure to carcinogens. This document contains inhalation cancer potency factors and oral cancer potency factors for 142 toxicants and toxicant compound classes developed by OEHHA or developed by other authoritative bodies and endorsed by OEHHA. The OEHHA website (*www.oehha.ca.gov*) should be consulted for the most current adopted chronic, acute and 8-hour RELs and cancer potency factors. In addition, for a small subset of these substances that are subject to airborne deposition and hence human oral and dermal exposure, oral chronic RELs and oral cancer potency factors have been developed by OEHHA. A summary of cancer and noncancer health effects values can be found in Appendix L and Chapters 6 and 7 of the Guidance Manual. All three Technical Support Documents have undergone public and peer review and have been approved by the state's Scientific Review Panel on Toxic Air Contaminants and adopted by OEHHA. The Guidance Manual is undergoing the same public and peer review process.

The Guidance Manual contains a description of the algorithms, recommended exposure variates, and cancer and noncancer health values, and modeling protocols needed to perform a Hot Spots risk assessment under the Hot Spots Act (see Appendix B). The information for the Guidance Manual is taken from the three TSDs. The Guidance

Manual supersedes the risk assessment methods presented in the Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (OEHHA, 2003).

The Guidance Manual is intended to address health risks from airborne contaminants released by stationary sources. Some of the methodology used is common to other regulatory risk assessment applications, particularly for California programs. However, if the reader needs to prepare a Health Risk Assessment (HRA) under another program, the HRA may need additional analyses. Therefore, appropriate California and federal agencies should be contacted. For example, if a facility must comply with HRA requirements under the Resource Conservation and Recovery Act (RCRA) or the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), the California Department of Toxic Substances Control (DTSC) must be contacted to determine if an HRA written to comply with AB 2588 will also satisfy RCRA/CERCLA requirements.

1.2 Use of the Guidance Manual

The intent in developing this Guidance Manual is to provide HRA procedures for use in the Air Toxics Hot Spots Program or for the permitting of existing, new, or modified stationary sources. The Air Resources Board (ARB) website (*www.arb.ca.gov*) provides more information on the Hot Spots Program and risk management guidelines, including recommendations for permitting existing, new, or modified stationary sources. The use of consistent risk assessment procedures and report presentation allows comparison of one facility to another, expedites the review of HRAs by reviewing agencies, and minimizes revision and resubmission of HRAs.

OEHHA recognizes that no one risk assessment procedure or set of exposure variates could perfectly address the many types of stationary facilities in diverse locations in California. Therefore a tiered risk assessment approach was developed to provide flexibility and allow consideration of site-specific differences. The tiered approach to risk assessment is discussed in detail in Chapter 8 of this Guidance.

These guidelines should be used in conjunction with the emission data collected and reported pursuant to requirements of the ARB's *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5*), and the *Emission Inventory Criteria and Guidelines Report for the Air Toxics "Hot Spots" Program* (EICG Report), which is incorporated by reference therein (see ARB's web site: http://www.arb.ca.gov/ab2588/2588guid.htm for the most current version, which was approved on August 27, 2007). This regulation outlines requirements for the collection of emission data, based on an inventory plan, which must be approved by the Air Pollution Control or Air Quality Management District (District). The emissions reported under this program are routine or predictable and include continuous and intermittent releases and predictable process upsets or leaks. Emissions for unpredictable releases (e.g., accidental catastrophic releases) are not reported under this program.

For landfill sites, these guidelines should be applied to the results of the landfill testing required under Health and Safety Code Section 41805.5 as well as to any emissions reported under the emission inventory requirements of the Air Toxics Hot Spots Act (e.g., from flares or other on-site equipment). Districts should be consulted to determine the specific landfill testing data to be used.

1.3 Who is Required to Conduct a Risk Assessment

The Hot Spots Act requires that each local Air Pollution Control District or Air Quality Management District (hereinafter referred to as District) determine which facilities will prepare an HRA. As defined under the Hot Spots Act, an HRA includes a comprehensive analysis of the dispersion of hazardous substances in the environment, their potential for human exposure, and a quantitative assessment of both individual and population-wide health risks associated with those levels of exposure.

Districts are to determine which facilities will prepare an HRA based on a prioritization process outlined in the law. The process by which Districts identify priority facilities for risk assessment involves consideration of potency, toxicity, quantity of emissions, and proximity to sensitive receptors such as hospitals, daycare centers, schools, work-sites, and residences. The District may also consider other factors that may contribute to an increased potential for significant risk to human receptors. As part of this process Districts categorize facilities as high, intermediate, or low priority. The District prioritization process is described in the *CAPCOA Air Toxics Hot Spots Program Facility Prioritization Guidelines, July 1990* (CAPCOA, 1990), although some Districts may have adopted their own method for prioritizing facilities for the purposes of AB2588, permitting, etc. Consult the District for updates to the Prioritization Guidelines. See the Hot Spots Program on ARB's web site at *www.arb.ca.gov* for more information on facility prioritization procedures.

Facilities designated by a District as "high priority" are required to submit an HRA to the District within 150 days. Districts may grant a 30-day extension. However, a District may require any facility to prepare and submit an HRA according to the District priorities established for purposes of the Hot Spots Act.

1.4 The Hot Spots Analysis and Reporting Program (HARP) Software

The ARB and the Districts have identified a critical need for software to assist with the programmatic aspects of the Hot Spots Program. HARP is computer software used by the ARB, OEHHA, Districts, and facility operators to promote statewide consistency, efficiency, and cost-effective implementation of HRAs and the Hot Spots Program. The HARP software package includes: 1) an Emissions Inventory Database Module, 2) an Air Dispersion Modeling Module, and 3) a Risk Analysis Module. The user-friendly Windows-based package provides for:

1. Electronic implementation of the risk assessment methods presented in the OEHHA guidelines (Guidance Manual);
2. Electronic data transfer from facilities and Districts;
3. The production of reports;
4. Facility prioritization;
5. Air dispersion modeling (AERMOD) of multiple emission releases or facilities for cumulative impact evaluations;
6. A summary report of acute, 8-hour, and chronic health hazard quotients or indices, and cancer risk at the point of maximum impact (PMI), maximally exposed individual resident (MEIR), maximally exposed individual worker (MEIW) and other receptors to be evaluated as needed;
7. Mapping displays of facility property boundaries, risk isopleths, and elevation contours;
8. The ability to display combined risk contours from multiple emission sources;
9. Output of data for use in other “off-the-shelf” Geographic Information Systems (GIS) programs for additional types of analysis; and
10. Census data for determining population-related health impacts showing the number of people exposed at various cancer risk levels and cancer burden.

1.5 Risk Assessment Review Process

The Hot Spots Act risk assessments are reviewed by the local District and by OEHHA. The Districts focus their review on the emissions data and the air dispersion modeling. OEHHA provides comments on the HRA’s general concordance with the Guidelines Manual and the completeness of the reported health risks. The District, taking into account the comments of OEHHA, approves the HRA or returns it to the facility for revision and resubmission. If the HRA is not revised and resubmitted by the facility within 60 days, the District may modify the HRA and approve it as modified. Based on the approved HRA, the District determines if there is a significant health risk associated with emissions from the facility. If the District determines that facility emissions pose a significant health risk, the facility operator provides notice to all exposed individuals regarding the results of the HRA and may be required to take steps to reduce emissions by implementing a risk reduction audit and plan. Notification is to be made according to

procedures specified by the District. Each District determines its own levels of significance for cancer and noncancer health effects for notification and risk reduction. See the Hot Spots Program on ARB's web site at *www.arb.ca.gov* for more information on significance levels selected by each District.

1.6 Uncertainty in Risk Assessment

OEHHA has striven to use the best science available in developing these risk assessment guidelines. However, there is a great deal of uncertainty associated with the process of risk assessment. The uncertainty arises from lack of data in many areas necessitating the use of assumptions. The assumptions used in these guidelines are designed to err on the side of health protection in order to avoid underestimation of risk to the public. Sources of uncertainty, which may overestimate or underestimate risk, include: 1) extrapolation of toxicity data in animals to humans, 2) uncertainty in the estimation of emissions, 3) uncertainty in the air dispersion models, and 4) uncertainty in the exposure estimates. In addition to uncertainty, there is a natural range or variability in measured parameters defining the exposure scenario. Scientific studies with representative sampling and large enough sample sizes can characterize this variability. In the specific context of a Hot Spots risk assessment, the source of variability with the greatest quantitative impact is variation among the human population in such properties as height, weight, food consumption, breathing rates, and susceptibility to chemical toxicants. OEHHA captures at least some of the variability in exposure by developing data driven distributions of intake rates, where feasible, in the TSD for Exposure Assessment (OEHHA, 2012).

Interactive effects of exposure to more than one carcinogen or toxicant are addressed in the risk assessment with default assumptions of additivity. Cancer risks from all carcinogens addressed in the HRA are added. Similarly, non-cancer hazard quotients for substances impacting the same target organ/system are added to determine the hazard index (HI). Although such effects of multiple chemicals are assumed to be additive by default, several examples of synergism (interactive effects greater than additive) are known. For substances that act synergistically, the HRA could underestimate the risks. Some substances may have antagonistic effects (lessen the toxic effects produced by another substance). For substances that act antagonistically, the HRA could overestimate the risks.

Other sources of uncertainty, which may underestimate or overestimate risk, can be found in exposure estimates where little or no data are available (e.g., soil half-life and dermal penetration of some substances from a soil matrix).

The differences among species and within human populations usually cannot be easily quantified and incorporated into risk assessments. Factors including metabolism, target site sensitivity, diet, immunological responses, and genetics may influence the response to toxicants. The human population is much more diverse both genetically and culturally (e.g., lifestyle, diet) than inbred experimental animals. The intraspecies variability among humans is expected to be much greater than in laboratory animals.

In most cases, cancer potency values have been estimated only for the single most affected tumor site. This represents a source of uncertainty in the cancer risk assessment. Adjustment for tumors at multiple sites induced by some carcinogens may result in a higher potency. Some recent assessments of carcinogens include such adjustments. Other uncertainties arise 1) in the assumptions underlying the dose-response model used, and 2) in extrapolating from large experimental doses, where other toxic effects may compromise the assessment of carcinogenic potential, to usually much smaller environmental doses.

When occupational epidemiological data are used to generate a carcinogenic potency or a health protective level for a non-carcinogen, less uncertainty is involved in the extrapolation from workplace exposures to environmental exposures. When using human data, no interspecies extrapolation is necessary eliminating a significant source of uncertainty. However, children are a subpopulation who's hematological, nervous, endocrine, and immune systems, for example, are still developing and who may be more sensitive to the effects of toxicants on their developing systems. The worker population and risk estimates based on occupational epidemiological data are more uncertain for children than adults. Current risk assessment guidelines include procedures designed to address the possibly greater sensitivity of infants and children, but there are only a few compounds for which these effects have actually been measured experimentally. In most cases, the adjustment relies on default assumptions which may either underestimate or overestimate the true risks faced by infants and children exposed to toxic substances or carcinogens.

Risk estimates generated by an HRA should not be interpreted as the expected rates of disease in the exposed population but rather as estimates of potential for disease, based on current knowledge and a number of assumptions.

In the Hot Spots program, cancer risk is often expressed as the maximum number of new cases of cancer projected to occur in a population of one million people due to exposure to the cancer-causing substance over a 30-year residential period. However, there is uncertainty associated with the cancer risk estimate. An individual's risk of contracting cancer from exposure to facility emissions may be less or more than the risk calculated in the risk assessment. An individual's risk not only depends on the individual's exposure to a specific chemical but also on his or her genetic background, health, diet, lifestyle choices and other environmental and workplace exposures. OEHHA uses health-protective exposure assumptions to avoid underestimating risk. For example, the risk estimate for airborne exposure to chemical emissions uses the health-protective assumption that the individual has a high breathing rate and exposure began early in life when cancer risk is highest.

A Reference Exposure Level (REL) is the concentration level at or below which no adverse non-cancer health effects are anticipated for the specified exposure duration. RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of factors that account for uncertainties as well as individual differences in human susceptibility to chemical exposures. The

factors used in the calculation of RELs are meant to err on the side of public health protection in order to avoid underestimation of non-cancer hazards. Exceeding the REL does not automatically indicate an adverse health impact. However, increasing concentrations above the REL value increases the likelihood that the health effect will occur.

Risk assessments under the Hot Spots program are often used to compare one source with another and to prioritize concerns. Consistent approaches to risk assessment are necessary to fulfill this function.

1.7 Tiered Approach to Risk Assessment

OEHHA developed a tiered approach to accommodate consideration of site-specific data that may be more appropriate for a given facility than the default variate. The first tier is the simplest point estimate approach to estimating exposure to facility emissions. Tier 1 is the first step in conducting a comprehensive risk assessment using algorithms and point estimates of input values described in the *Technical Support Document for Exposure Assessment and Stochastic Analysis*. (OEHHA, 2012) Each facility conducts a Tier 1 risk assessment to promote consistency across the state in facility risk assessments and facilitate comparisons across facilities. To be health-protective, high-end estimates for the key intake exposure variates are used for the dominant exposure pathways.

Tier 2 allows use of site-specific point estimates of exposure variates as long as these estimates can be justified. For example, if there are data indicating that consumption of fish from an impacted body of water is lower than the OEHHA-recommended fish consumption rate, then the facility can use that data to generate a point estimate for sport-fish consumption from that body of water. The risk assessor must supply the data and methods used for the site-specific estimates, and the site-specific estimates must be reproducible and approved by both the District and OEHHA.

Tier 3 risk assessment involves stochastic analysis of exposure using data-based distributions for the key exposure variates compiled in the OEHHA (2012) *Technical Support Document*. Since a stochastic approach to risk assessment provides more information about the range of risk estimates based on the range of exposures, Tier 3 can serve as a useful supplement to the Tier 1 and 2 approaches. Variance propagation methods (e.g., Monte Carlo analysis) are used to derive a range of cancer risk estimates reflecting the known variability in the inputs. Finally, a Tier 4 approach would use distributions of exposure variates that may be more appropriate for a site, such as the distribution of fish consumption rates for a specific body of water impacted by a facility. As in a Tier 2 approach, the risk assessment must supply the data and methods used for the site-specific distributions for exposure variates, and the site-specific estimates must be justified to and reproducible by the Districts and OEHHA.

1.8 References

CAPCOA, 1990. *CAPCOA Air Toxics Hot Spots Program Facility Prioritization Guidelines.* California Air Pollution Control Officers Association, July 1990.

OEHHA, 2003. Air Toxics Hot Spots Risk Assessment Guidelines: The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments.

OEHHA, 2008. Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. Available online at: http://www.oehha.ca.gov

OEHHA, 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. May 2009. Available online at: http://www.oehha.ca.gov

OEHHA, 2012. Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis. Available online at http://www.oehha.ca.gov

2 - Overview of Health Risk Assessment

2.1 The Model for Risk Assessment

The standard approach currently used for health risk assessment (HRA) was originally proposed by the National Academy of Sciences in the 1983 book: *Risk Assessment in the Federal Government: Managing the Process* (NAS, 1983) and was updated in the Academy's 1994 book: *Science and Judgment in Risk Assessment* (NAS, 1994). In 2009 the National Academy published *Science and Decisions: Advancing Risk Assessment* (NAS, 2009), in which a number of recommendations are made on improving the risk assessment process and expanding it to include community concerns and cumulative risks. The four steps involved in the risk assessment process are 1) hazard identification, 2) exposure assessment, 3) dose-response assessment, and 4) risk characterization. These four steps are briefly discussed below.

2.2 Hazard Identification

For air toxics sources, hazard identification involves the pollutant(s) of concern emitted by a facility, and the types of adverse health effects associated with exposure to the chemical(s), including whether a pollutant is a potential human carcinogen or is associated with other types of adverse health effects. For the Air Toxics Hot Spots Program (Hot Spots), the emitted substances that are addressed in a risk assessment are found in the list of substances designated in the ARB's *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report* (EICG Report), which is incorporated by reference therein (ARB, 2007*)*. This list of substances is contained in Appendix A of this document and the EICG Report. The list of substances also identifies those substances that are considered human carcinogens or potential human carcinogens.

2.3 Exposure Assessment

The purpose of the exposure assessment is to estimate the extent of public exposure to emitted substances. For the Hot spots program, in practice this means estimating exposures for those emitted substances for which potential cancer risk or noncancer health hazards for acute, repeated 8-hour, and chronic exposures will be evaluated. This involves emission quantification, modeling of environmental transport, evaluation of environmental fate, identification of exposure routes, identification of exposed populations, and estimation of short-term (e.g., 1-hour maximum), 8-hour average, and long-term (annual) exposure levels. These activities are described in Chapters 4 and 5. Chapter 5 also discusses the tiered approach to risk assessment.

The ARB's Emission Inventory Criteria and Guidelines (EICG) Report provides assistance in determining those substances that must be evaluated in an HRA and the reporting requirements of facilities, while the Hot Spots Analysis and Reporting Program (HARP) software can be used to model ground level concentrations at specific off-site

locations resulting from facility emissions. The United States Environmental Protection Agency (U.S. EPA) has adopted the AERMOD air dispersion model into their list of regulatory approved models, in place of the previously used ISCST3 model. AERMOD is a steady-state plume model that incorporates air dispersion based on planetary boundary layer turbulence structure and scaling concepts, including treatment of both surface and elevated sources, and both simple and complex terrain (U.S. EPA, 2009). The Air Resources Board recommends AERMOD for Hot Spots risk assessments. The AERMOD air modeling software will be incorporated into the HARP software, which allows the user to input all dispersion parameters directly into the program to generate air dispersion data. Alternatively, the air dispersion data may be generated separately from HARP using other air dispersion models, and then imported into HARP to generate risk estimates. Data imported into HARP must already be in the format required by HARP. HARP has the flexibility to generate a summary of the risk data necessary for an HRA by either of the above approaches.

Most of the toxicants assessed under the Hot Spots program are volatile organic compounds that remain as gases when emitted into the air. These chemicals are not subject to appreciable deposition to soil, surface waters, or plants. Therefore, human exposure via ingestion or dermal exposure, at least at concentrations typically encountered in the ambient air, is not considered for volatile organic compounds in the Hot Spots risk assessments. While some models indicate potential for dermal exposure to certain volatile organic compounds, at this time, the Hot spots program does not consider this pathway. Significant exposure to volatile organic toxicants emitted into the air occurs through the inhalation pathway, and this pathway is the primary consideration in the Hot spots risk assessments. A small subset of Hot Spots substances consists of semi-volatile organic and metal toxicants emitted partially or totally as particles subject to deposition. Ingestion and dermal pathways as well as the inhalation pathway must be evaluated for these chemicals. A few of these semi-volatile organic and metal toxicants must also include the breast milk ingestion pathway. Additional ingestion pathways may also need to be evaluated depending on the pathways of exposure for the specific receptor of interest. Table 5.1 in Chapter 5, Table 6.4 in Chapter 6, and Table 7.1 in Chapter 7 list the substances that must be evaluated for multipathway impacts. HARP is designed to assess potential health impacts posed by substances that must be analyzed by a multipathway approach.

2.4 Dose-Response Assessment

Dose-response assessment is the process of characterizing the relationship between exposure to an agent and incidence of an adverse health effect in exposed populations. In quantitative carcinogenic risk assessment, the dose-response relationship is expressed in terms of a potency slope that is used to calculate the probability or risk of cancer associated with an estimated exposure. Cancer potency factors are expressed as the 95th percent upper confidence limit of the slope of the dose response curve estimated assuming continuous lifetime exposure to a substance. Typically, potency factors are expressed as units of inverse dose (e.g., $(\text{mg/kg BW/day})^{-1}$) or inverse concentration (e.g., $(\mu\text{g/m}^3)^{-1}$). It is assumed in cancer risk assessments that risk is directly proportional to dose and that there is no threshold for carcinogenesis.

The Office of Environmental Health Hazard Assessment (OEHHA) has compiled cancer potency factors, which should be used in risk assessments for the Hot Spots program, in Table 7.1. Cancer potency factors listed in Table 7.1 were derived either by the U.S. EPA or by OEHHA, underwent public and peer-review, and were adopted for use in the program. Chapter 8 describes procedures for use of potency values in estimating excess cancer risk. For a detailed description of cancer potency factors, refer to the *Technical Support Document for Cancer Potency Factors* (OEHHA, 2009).

For noncarcinogenic effects, dose-response data developed from animal or human studies are used to develop acute, 8-hour, and chronic noncancer Reference Exposure Levels (RELs). The acute, 8-hour and chronic RELs are defined as the concentration at which no adverse noncancer health effects are anticipated even in sensitive members of the general population, with infrequent one hour exposures, repeated 8-hour exposures over a significant fraction of a lifetime, or continuous exposure over a significant fraction of a lifetime, respectively. The most sensitive health effect is chosen to develop the REL if the chemical affects multiple organ systems. Unlike cancer health effects, noncancer health effects are generally assumed to have thresholds for adverse effects. In other words, injury from a pollutant will not occur until exposure to that pollutant has reached or exceeded a certain concentration (i.e., threshold) and/or dose. The acute, 8-hour, and chronic RELs are air concentrations intended to be below the threshold for health effects for the general population.

The actual threshold for health effects in the general population is generally not known with any precision. Uncertainty factors are applied to the Lowest Observed Adverse Effects Level (LOAEL) or No Observed Adverse Effects Level (NOAEL) or Benchmark Concentration values from animal or human studies to help ensure that the chronic, 8-hour and acute REL values are below the threshold for human health for nearly all individuals. This guidance manual provides the acute, 8-hour, and chronic Reference Exposure Levels in Tables 6.1 through 6.3. Some substances that pose a chronic or repeated 8-hour inhalation hazard may also present a chronic hazard via non-inhalation routes of exposure (e.g., ingestion of contaminated water, foods, or soils, and dermal absorption). The oral RELs for these substances are presented in Table 6.4. The methodology and derivations for acute, 8-hour, and chronic, RELs are described in the *Technical Support Document for the Derivation of Noncancer Reference Exposure Levels* (OEHHA, 2008).

2.5 Risk Characterization

This is the final step of risk assessment. In this step, modeled concentrations and exposure information, which are determined through exposure assessment, are combined with potency factors and RELs that are developed through dose-response assessment. The use of cancer potency factors to assess total cancer risk and the use of the hazard index approach for evaluating the potential for noncarcinogenic health effects are described in Chapter 8. Example calculations for determining (inhalation) cancer risk and noncancer acute, 8-hour, and chronic hazard quotients and hazard indices are presented in Appendix I. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

Under the Hot Spots Act, health risk assessments are to quantify both individual and population-wide health impacts (Health and Safety Code, Section 44306) (Appendix B). The health risk assessments are facility specific and the calculated risk should be combined for all pollutants emitted by a single facility. For example, cancer risk from multiple carcinogens is considered additive. For exposures to multiple non-carcinogen pollutants, a hazard index approach is applied for air contaminants affecting the same organ system. All substances emitted by the facility that are on the Hot Spots Act list of substances must be identified in the HRA, including those on the list that do not have a potency value or REL.

For assessing risk, OEHHA has developed two methods for determining dose via inhalation, dermal absorption, and ingestion pathways. These two methods, the point estimate approach and the stochastic exposure assessment approach, are described below and in Chapters 5 and 8. Detailed presentations of these methods can be found in*: Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012*)*.

2.5.1 *Point Estimate Approach*

OEHHA provides information in this document on average and high-end values for key exposure pathways (e.g., breathing rate for the inhalation exposure pathway). The average and high-end of point estimates in this document are defined in terms of the probability distribution of values for that variate. The mean represents the average values for point estimates and the 95th percentiles represent the high-end point estimates from the distributions identified in OEHHA (2012). Thus, within the limitations of the data, average, and high-end point estimates are supported by the distribution.

Tier 1 of the tiered approach to risk assessment, which is briefly discussed in Section 2.5.3 and presented in more detail in Chapter 8, utilizes a combination of the average and high-end point estimates to more realistically estimate exposure in multipathway risk assessments. This method uses high-end exposure estimates for the pathways that are the main drivers of exposure and the average point estimate for the other non-driving exposure pathways. This approach will lessen the issue of compounding high-end exposure estimates, while retaining a health-protective approach for the more important exposure pathways. It is unlikely that an individual receptor would be on the high-end of exposure for all exposure pathways. See Chapter 8 for detailed discussions of how this multipathway methodology is applied to cancer and noncancer calculations. The HARP software can perform this analysis (referred to as the derived approach in the HARP software).

In addition to using an estimate of average and high-end consumption rates, cancer risk evaluations at individual receptors are presented for 9, 30, and 70-year exposure durations. The 9 and 30-year durations correspond to the average and high-end of residency time recommended by U.S. EPA (1997). The California data presented in Appendix L of the Exposure TSD (OEHHA, 2012) are generally supportive of the nationwide data. The 9 and 70-year exposure durations present potential impacts over the range of residency periods, while the 30-year exposure duration is recommended

for use as the basis for estimating cancer risk at the MEIR in all HRAs. Population-wide impacts should use the 70-year exposure duration.

The parameters used for all exposure durations assume exposure begins in the last trimester of pregnancy and progress through the exposure duration of interest (e.g., 9, 30, or 70 years). These assumptions are thus protective of children. Children have higher intake rates on a per kilogram body weight basis (e.g., they breathe, drink and eat more per kg body weight than adults) and thus receive a higher dose from contaminated media. See Chapter 5 for the point estimates that can be used to estimate impacts for children. Chapters 5 and 8 discuss how to calculate cancer risk based on various exposure durations and point estimates. Appendix I contains an example calculation and Chapter 9 clarifies how to present the findings in an HRA.

2.5.2 *Stochastic Exposure Assessment*

OEHHA was directed under the Air Toxics “Hot Spots” program (SB 1731, Calderon, stat. 1992; Health and Safety Code Section 44360(b)(2)) to develop a “likelihood of risk” approach to risk assessment. To satisfy this requirement, OEHHA developed a stochastic approach to risk assessment that utilizes distributions for exposure variates such as breathing rate and water consumption rate rather than a single point estimate. The variability in exposure can be propagated through the risk assessment model using the distributions as input and a Monte Carlo or similar method. The result of such an analysis is a range of risks that at least partially characterizes variability in exposure.

Distributions of key exposure variates that are presented in the *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012) were taken from the literature, if adequate, or developed from raw data of original studies. Intake variates such as vegetable consumption are relatively data rich; for these variates reasonable probability distributions can be constructed. However, the data necessary to characterize the variability in risk assessment variates are not always available. For example, for the fate and transport variates (e.g., fish bioaccumulation factors), there are only a few measurements for a given chemical available which precludes the adequate characterization of a probability distribution. We only developed distributions for those key exposure variates that were adequately characterized by data. Development of distributions is described in detail in the *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012).

2.5.3 *Tiered Approach to Risk Assessment*

OEHHA recommends using a tiered approach to risk assessment. Tier 1 is a standard point estimate approach using the recommended point estimates presented in this document. If site-specific information is available to modify some point estimates developed in the *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012) and is more appropriate to use than the recommended point estimates in this document, then Tier 2 allows use of that site-specific information. Site-specific information should be presented to the District before being used. The District may contact OEHHA for additional advice. Note that all non-default variates need to be adequately justified to OEHHA and the Districts to be used. In Tier 3, a stochastic approach to exposure assessment is used with the data distributions developed in the TSD (OEHHA, 2012) and presented in this document. Tier 4 is also a stochastic approach but allows for utilization of site-specific distributions, if they are justifiable (to OEHHA and the Districts) and more appropriate for the site under evaluation than those recommended in this document. Persons preparing an HRA that has a Tier 2 through Tier 4 evaluation must also include the results of a Tier 1 evaluation. Tier 1 evaluations are required for all HRAs prepared for the Hot Spots Program to promote consistency across the state for all facility risk assessments and allow comparisons across facilities. Chapter 8 provides a summary of the tiered approach and the TSD (OEHHA, 2012) discusses it in detail. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

2.6 References

ARB, 2007. *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report* (EICG Report).

NAS, 1983. National Academy of Sciences. *Risk Assessment in the Federal Government: Managing the Process*. National Research Council. National Academy Press, Washington D.C.

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OEHHA, 2012. *Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis*. Available online at http://www.oehha.ca.gov

U.S. EPA (2009). AERMOD Implementation Guide. Last Revised: March 19, 2009.

U.S. EPA, 1997. *Exposure Factors Handbook, Volume I, General Factors*. EPA/600/P-95/002Fa.

AERMOD Implementation Workgroup, U. S. Environmental Protection Agency. Online at: http://www.epa.gov/ttn/scram/7thconf/aermod/aermod_implmtn_guide_19March2009.pdf

3 - Hazard Identification - Air Toxics Hot Spots Emissions

3.1 The Air Toxics Hot Spots List of Substances and Emissions Inventory

For air toxics sources, hazard identification involves identifying pollutants of concern and whether these pollutants are potential human carcinogens or associated with other types of adverse health effects. For the Air Toxics Hot Spots (Hot Spots) Program, the emitted substances that are addressed in a health risk assessment (HRA) are found in the list of hazardous substances designated in the Air Resources Board's (ARB's) *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report* (EICG Report), which is incorporated by reference therein (ARB, 2007). This list of substances is contained in both Appendix A of this document and the EICG Report. The list of substances also identifies those substances that are considered human carcinogens or potential human carcinogens.

The substances included on the Hot Spots Program list of substances are defined in the statute as those substances found on lists developed by the following sources:

- International Agency for Research on Cancer (IARC);
- U.S. Environmental Protection Agency (U.S. EPA);
- U.S. National Toxicology Program (NTP);
- ARB Toxic Air Contaminant Identification Program List;
- Hazard Evaluation System and Information Service (HESIS) (State of California);
- Proposition 65 (Safe Drinking Water and Toxic Enforcement Act of 1986) list of carcinogens and reproductive toxicants (State of California);
- Any additional substance recognized by the State Board as presenting a chronic or acute threat to public health when present in the ambient air.

All substances emitted by the facility that are on the Hot Spots Act list of substances must be identified in the HRA.

The ARB EICG Report (ARB, 2007) specifies that each facility subject to the Hot Spots Act must submit an Emission Inventory Report to the local air pollution control or air quality management district. This Emission Inventory Report must identify and account for all listed substances used, manufactured, formulated, or released by the facility. All routine, predictable releases must be reported. These inventory reports include the emission data necessary to estimate off-site levels of facility-released Hot Spots substances. These inventory reports will be discussed in further detail in Chapter 4. See Chapter 9 for an outline that specifies the content and recommended format for presenting the air dispersion modeling and HRA results. As presented in Appendix A, the EICG Report divides the list into three groups for reporting purposes. Potency or severity of toxic effects and potential for facility emission were considered in placing compounds into the three groups.

For the first group (listed in these guidelines in Appendix A-I), all emissions of these substances must be quantified in the HRA. For substances in the second group (listed in these guidelines in Appendix A-II), emissions are not quantified; however, facilities must report whether the substance is used, produced, or otherwise present on-site (i.e., these substances are simply listed in a table in the HRA). Lastly, substances in the third group (Appendix A-III) also only need to be reported in a table in the HRA if they are manufactured by the reporting facility.

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

3.2 References

ARB, 2007. *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report* (EICG Report).

4 - Air Dispersion Modeling

The information contained in this section is primarily an abbreviated version of the material found in Chapter 2 of the Air Toxics Hot Spots Risk Assessment Guidelines; Exposure Assessment and Stochastic Analysis Technical Support Document (OEHHA, 2012). Several references have been included in this section to indicate those areas that are covered in more detail in Chapter 2 of the Technical Support Document. However, some air dispersion concepts and procedures have been added to assist the reader in the health risk assessment (HRA) process. In particular, a brief summary of the Hot Spots Analysis and Reporting Program (HARP) software applicability to air dispersion analysis has been included. The HARP software has been developed by the Air Resources Board (ARB), in consultation with OEHHA and Air Pollution Control or Air Quality Management District (District) representatives. The HARP software is the recommended model for calculating and presenting HRA results for the Air Toxics Hot Spots Program (Hot Spots). Information on obtaining the HARP software can be found under the Hot Spots Program on the ARB's web site at *www.arb.ca.gov*. See Chapter 9 for an outline that specifies the content and recommended format for presenting the air dispersion modeling and HRA results.

The U.S. EPA has adopted the AERMOD air dispersion model into their list of regulatory approved models, in place of the previously used ISCST3 model. AERMOD is a steady-state plume model that incorporates air dispersion based on planetary boundary layer turbulence structure and scaling concepts, including treatment of both surface and elevated sources, and both simple and complex terrain (U.S. EPA, 2009). The Air Resources Board recommends AERMOD for Hot Spots risk assessments.

4.1 Air Dispersion Modeling in Exposure Assessment: Overview

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

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

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

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

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

1. Create an emission inventory of the toxic releases (Section 4.2)
2. Identify the source types (Section 4.3)
3. Identify the terrain type (Section 4.4)
4. Determine the detail needed for the analysis: screening or refined (Section 4.5)
5. Identify the population exposure (Section 4.6)
6. Identify the receptor network (Section 4.7)
7. Obtain meteorological data (for refined air dispersion modeling only) (Section 4.8)
8. Select an air dispersion model (Section 4.9)
9. Prepare a modeling protocol and submit to the local Air District (hereafter referred to as “the District”) (Section 4.14)
10. Complete the air dispersion analysis
11. If necessary, redefine the receptor network and return to Step 10

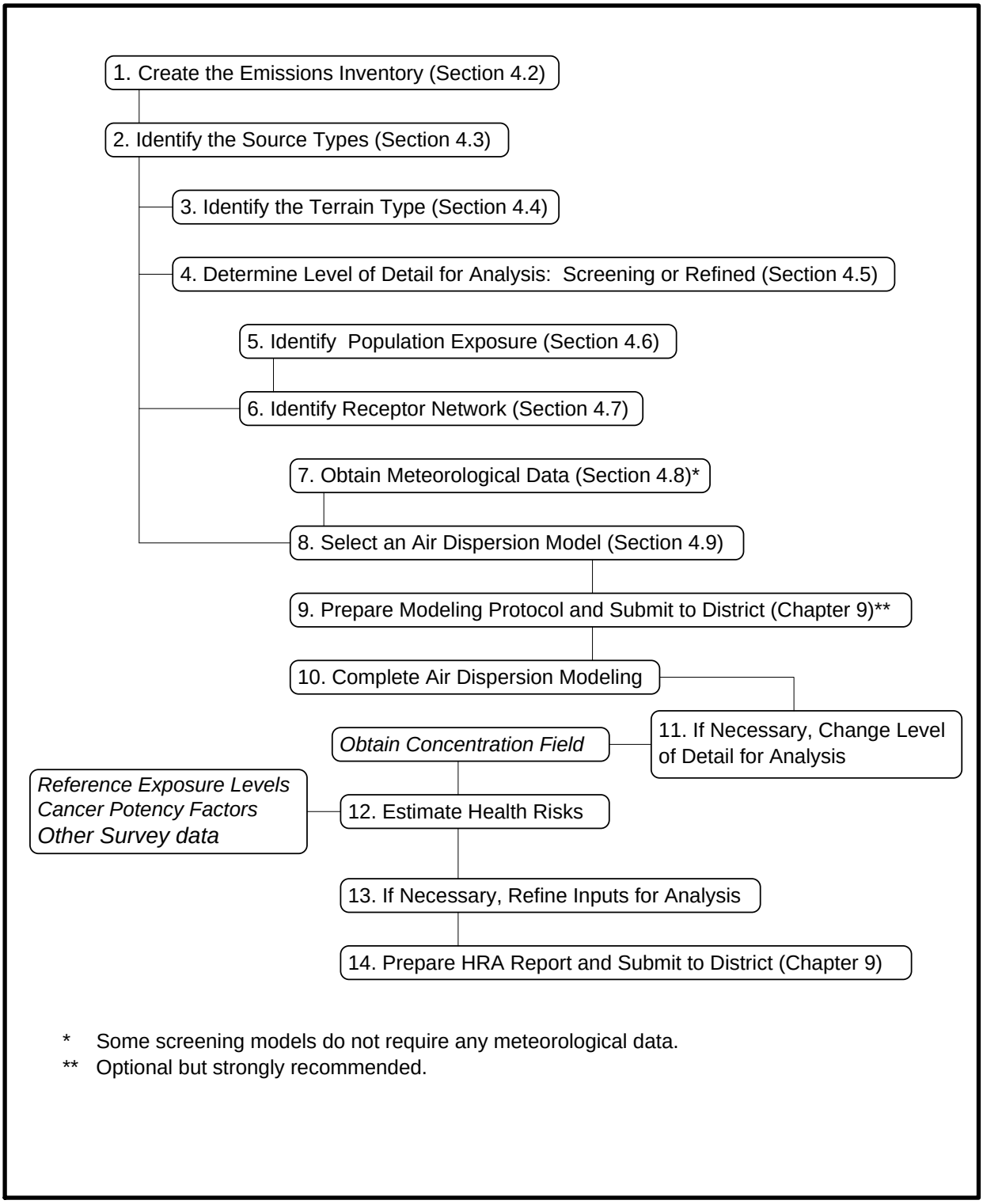
12. Complete the risk assessment

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

14. Present the HRA results (Chapter 9 provides an outline that specifies the content and recommended format of HRA results).

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

Figure 1 Overview of the Air Dispersion Modeling Process.



4.2 Emission Inventories

The Emission Inventory Reports (Inventory Reports) developed under the Hot Spots Program provide data to be used in the HRA and in the air dispersion modeling process. The Inventory Reports contain information regarding emission sources, emitted substances, emission rates, emission factors, process rates, and release parameters (area and volume sources may require additional release data beyond that generally available in Emissions Inventory reports). This information is developed according to the ARB's *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5)*, and the *Emission Inventory Criteria and Guidelines Report* (EICG Report), which is incorporated by reference therein (ARB, 2007*)*.

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

4.2.1 *Air Toxics Hot Spots Emissions*

As noted in Chapter 3, Hazard Identification, the HRA should identify all substances emitted by the facility, which are on the Hot Spots Act list of substances (see Appendix A of the Guidance Manual or the EICG Report). The EICG Report specifies that Inventory Reports must identify and account for all listed substances used, manufactured, formulated, or released by the facility. All routine, predictable releases must be reported. Under the regulations, the list is divided into three groups for reporting purposes. The first group (listed in Appendix A-I of the Inventory Guidelines Report) has all pollutants whose emissions must be quantified. The second group (listed in Appendix A-II of the Inventory Guidelines Report) includes substances where emissions do not need to be quantified; however, facilities must report whether the substance is used, produced, or otherwise present on-site. The third group (listed in Appendix A-III of the Emissions Inventory Guidelines Report) includes substances whose emissions need not be reported unless the substance is manufactured by the facility. Chemicals or substances in the second and third groups should be listed in a table in the risk assessment.

Facilities that must comply with the Resource Conservation and Recovery Act and Comprehensive Environmental Response, Compensation and Liability Act (RCRA/CERCLA) requirements for risk assessment need to consult the Department of Toxic Substances Control (DTSC) Remedial Project Manager to determine which substances must be evaluated in their risk assessment in addition to the list of "Hot Spots" chemicals. Some RCRA/CERCLA facilities may emit chemicals that are not currently listed under the "Hot Spots" Program. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.2.1.1 Emission Estimates Used in the Risk Assessment

The HRA must include emission estimates for all substances that are required to be quantified in the facility's emission inventory report. Specifically, HRAs should include both annual average emissions and maximum 1-hour emissions for each pollutant. Maximum 1-hour emissions are used for acute noncancer health impacts while annual emissions are used for chronic exposures (i.e., chronic and 8-hour noncancer health impacts or cancer risk assessment).

Emissions for each substance must be reported for individual emitting processes associated with unique devices within a facility. Total facility emissions for an individual air contaminant will be the sum of emissions, reported by process, for that facility. Information on daily and annual hours of operation, and relative monthly activity, must be reported for each emitting process. Devices and emitting processes must be clearly identified and described and must be consistent with those reported in the emissions inventory report.

The HRA should include tables that present the emission information (i.e., emission rates for each substance released from each process) in a clear and concise manner. The District may allow the facility operator to base the HRA on more current emission estimates than those presented in the previously submitted emission inventory report (i.e., actual enforceable emission reductions realized by the time the HRA is submitted to the District). If the District allows the use of more current emission estimates, the District must review and approve the new emissions estimates prior to use in the HRA. The HRA report must clearly state what emissions are being used and when any reductions became effective. Specifically, a table presenting emission estimates included in the previously submitted emission inventory report as well as those used for the HRA should be presented. The District should be consulted concerning the specific format for presenting the emission information. Chapter 9 provides an outline that specifies the content and recommended format of HRA results. A revised emission inventory report must be submitted to the District prior to submitting the HRA and forwarded by the District to the ARB, if revised emission data are used.

4.2.1.1.1 Molecular Weight Adjustments for the Emissions of Metal Compounds

For most of the Hot Spots toxic metals, the OEHHA cancer potency factors, acute and chronic RELs apply to the weight of the toxic metal atom contained in the overall compound. Some of the Hot Spots compounds contain various elements along with the toxic metal atom (e.g., "Nickel hydroxide", CAS number 12054-48-7, has a formula of H_2NiO_2). Therefore, an adjustment to the reported pounds of the overall compound is needed before applying the OEHHA cancer potency factor for "Nickel and compounds" to such a compound. This ensures that the cancer potency factor, acute or chronic REL is applied only to the fraction of the overall weight of the emissions that are associated with health effects of the metal. In other cases, the Hot Spots metals are already reported as the metal atom equivalent (e.g., CAS 7440-02-0, "Nickel"), and these cases do not use any further molecular weight adjustment. (Refer to Note [7] in Appendix A,

List of Substances in the EICG Report for further information on how the emissions of various Hot Spots metal compounds are reported.)

The appropriate molecular weight adjustment factors (MWAF) to be used along with the OEHHA cancer potency factors, acute and chronic RELs for Hot Spots metals can be found in the MWAF column[1] of the table containing OEHHA/ARB Approved Health Values for use in Hot Spots Facility Risk Assessments that is in Appendix L of this document.

As an example, the compound "Nickel hydroxide" has a molecular formula of H_2NiO_2. The atomic weight of each of the elements in this compound, and the fraction they represent of the total weight, are therefore as follows:

Element	Number of atoms	Atomic Weight	Fraction of Total Weight = MWAF
1 x Nickel (Ni)	1 x	58.70	58.70 / 92.714 = **0.6332** (MWAF for Nickel)
2 x Oxygen (O)	2 x	15.999	
2 x Hydrogen (H)	2 x	1.008	
Total Molecular Weight of H_2NiO_2:		92.714	

So, for example, assume that 100 pounds of "Nickel hydroxide" emissions are reported under CAS number 12054-48-7. To get the Nickel atom equivalent of these emissions, multiply by the listed MWAF (0.6332) for Nickel hydroxide:

- 100 pounds x 0.6332 = 63.32 pounds of Nickel atom equivalent.

This step should be completed prior to applying the OEHHA cancer potency factor for "Nickel and compounds" in a calculation for a prioritization score or risk assessment calculation. (Note - The HARP software automatically applies the appropriate MWAF for each Hot Spots chemical (by CAS number), so the emissions should not be manually adjusted when using HARP. Therefore, if using HARP, you would use 100 pounds for Nickel hydroxide and HARP will make the MWAF adjustment for you. If not using HARP, you would use 63.32 pounds.)

[1] The value listed in the MWAF column for Asbestos is not a molecular weight adjustment. This is a conversion factor for adjusting mass and fibers or structures. See Appendix C for more information on Asbestos reporting and risk assessment information or see the EICG report for reporting guidance.

4.2.1.2 Release Parameters

Emission release parameters (e.g., stack height and inside diameter, stack gas exit velocity, release temperature and emission source location in UTM coordinates) are needed as inputs to the air dispersion model. The Inventory Guidelines specify the release parameters that must be reported for each stack, vent, ducted building, exhaust site, or other site of exhaust release. Additional information may be required to characterize releases from non-stack (volume and area) sources; see U.S. EPA dispersion modeling guidelines or specific user's manuals. This information should also be included in the air dispersion section of the risk assessment. This information must be presented in tables included in the risk assessment. Note that some dimensional units needed for the dispersion model may require conversion from the units reported in the Inventory Report (e.g., Kelvin (K) vs. degrees Fahrenheit (°F)). Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.2.1.3 Operation Schedule

The HRA should include a discussion of the facility operation schedule and daily emission patterns. For AB2588 purposes, emissions should be reported based on routine and predicable operations. Weekly or seasonal emission patterns may vary and should be discussed. This is especially important in a refined HRA. Diurnal emission patterns should be simulated in the air dispersion model because of diurnal nature of meteorological observations. Diurnal evaluations are important to include since diurnal weather patterns and emission releases may cause significant differences in the concentration at a receptor of interest.

A table should be included listing the emission schedule on an hourly and yearly basis. In addition, the emission schedule and exposure schedule should corroborate any exposure adjustment factors used for approximating an inhaled dose. For more information about exposure adjustment factors, see Section 4.8.1. Alternatively, exposure adjustments can be made through refining the air dispersion analysis. See Section 4.11.1.2(h) for special case modeling or Appendix M. An alternative to including modeling that addresses diurnal influences would be to include a sensitivity study showing, and/or text explaining, the reason(s) why there are no significant differences due to diurnal influences on the emissions from the facility or at the receptor(s) of interest. For more guidance, you can contact the district or reviewing authority**.** Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.2.1.4 Emission Controls

The HRA should include a description of control equipment, the emitting processes it serves, and its efficiency in reducing emissions of substances on the Air Toxics “Hot Spots” list. The EICG Report requires that this information be included in the Inventory Reports, along with the emission data for each emitting process. If the control equipment did not operate full-time throughout the year, then the reported overall control efficiency must be adjusted to account for any predictable downtime of the

control equipment. Any entrainment of toxic substances to the atmosphere from control equipment should be accounted for; this includes fugitive releases during maintenance and cleaning of control devices (e.g., baghouses and cyclones). Contact the District for guidance with control equipment adjustments. Recommended default deposition rates that are used when calculating potential noninhalation health impacts are listed in Section 5.3.2. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.2.2 *Landfill Emissions*

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

4.3 Source Characterization

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

4.3.1 *Source Type*

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

4.3.1.1 Point Sources

Point sources are probably the most common type of source and most air dispersion models have the capability to simulate them. Typical examples of point sources include exhaust stacks. Isolated vents from buildings are special examples of point sources.

4.3.1.2 Line Sources

The version 12345 or newer of the AERMOD can accommodate line sources. Line sources can be also treated as a special case of either an area or a volume source. Examples of line sources include: conveyor belts and rail lines, freeways, and busy roadways. Not all mobile sources may be subject to the Hot Spots program; however, non-motor vehicles that operate within a facility (e.g., ships, trains, and cranes, etc.) are subject to the Hot Spots program. For more information, see the ARB’s Emission Inventory and Criteria Guidelines document or ARB’s interpretation and guidance

memorandum to CAPCOA regarding mobile sources which are subject to the "Hot Spots" program. This memo can be found at http://www.arb.ca.gov/ab2588/motorv.pdf.

Mobile sources and rail lines are required to be evaluated under SB 352. SB 352 requires a risk assessment performed under the Hot Spots risk assessment guidance for proposed school sites within 500 feet of a busy roadway. Dedicated air dispersion models are available for motor vehicle emissions from roadways which are a special type of line source. These models (i.e., CALINE3, CAL3QHCR, and CALINE4) are designed to simulate the mechanical turbulence and thermal plume rise due to the motor vehicle activity on the roadway. However, these dedicated models use the Pasquill-Gifford dispersion stability classes for dispersion; the AERMOD dispersion model uses a more advanced continuous stability estimation method based on observations. The limitation with AERMOD is that the user needs to estimate initial mixing (Szo, and Syo) for mechanical turbulence and thermal plume rise. Consult with the District prior to conducting roadway modeling to determine model use.

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

4.3.1.3 Area Sources

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

Fugitive particulate (PM2.5, PM10, TSP) emission sources include areas of disturbed ground (e.g., open pits, parking lots) which may be present during operational phases of a facility's life. Also included are areas of exposed material (e.g., storage piles and slag dumps) and segments of material transport where potential fugitive emissions may occur (uncovered haul trucks or rail cars, emissions from unpaved roads). Fugitive emissions may also occur during stages of material handling where particulate material is exposed to the atmosphere (uncovered conveyors, hoppers, and crushers).

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

4.3.1.4 Volume Sources

Non-point sources with emissions containing an initial vertical extent should be modeled as volume sources. The initial vertical extent may be due to plume rise or a vertical

distribution of numerous smaller sources over a given area. Examples of volume sources include buildings with natural fugitive or passive ventilation, and line sources such as conveyor belts and rail lines.

4.3.2 *Quantity of Sources*

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

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

- Combining all sources into one single “representative” source

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

- Running the model for each individual source and superimposing results

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

Additional guidance regarding source merging is provided by the U.S. EPA (1995a). It should be noted that depending upon the population distribution, the total burden can actually increase when pollutants are more widely dispersed. If the total burden from the facility or zone of impact (see Section 4.6.1) could increase for the simplifying modeling assumptions described above, the District should be consulted.

4.4 Terrain Type

Two types of terrain characterizations are required to select the appropriate model. One classification is made according to land type and another one according to terrain topography.

4.4.1 *Terrain Type – Land Use*

Some air dispersion models (e.g., CALINE) use different dispersion coefficients (sigmas) depending on the land use over which the pollutants are being transported. The land use type is also used by some models to select appropriate wind profile exponents. Traditionally, the land type has been categorized into two broad divisions for the purposes of dispersion modeling: urban and rural. Accepted procedures for determining the appropriate category are those suggested by Irwin (1978): one based on land use classification and the other based on population.

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

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

AERMOD is the U.S. EPA's preferred dispersion model for a wide range of applications in rural or urban conditions. The users should refer to section 5.0 of the AERMOD Implementation Guide to determine urban or rural conditions.

The Land Use and the Population Density Procedures discussed above are described as follows.

4.4.1.1 Land Use Procedure

(1) Classify the land use within the total area A, circumscribed by a 3 km radius circle centered at the source using the meteorological land use typing scheme proposed by Auer (1978) and shown in Table 4.1.

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

4.4.1.2 Population Density Procedure

(1) Compute the average population density (*p*) per square kilometer with *A* as defined in the Land Use procedure described above. (Population estimates are also required to determine the exposed population; for more information see Section 4.6.3.)

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

Table 4.1 Identification and classification of land use types (Auer, 1978)

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

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

Rivers, lakes

4.4.2 *Terrain Type - Topography*

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

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

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

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

4.4.2.2 Intermediate Terrain

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

4.4.2.3 Complex Terrain

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

4.5 Level of Detail: Screening vs. Refined Analysis

Air dispersion models can be classified according to the level of detail which is used in the assessment of the concentration estimates as “screening” or “refined”. Refined air dispersion models use more robust algorithms capable of using representative meteorological data to predict more representative and usually less conservative

estimates. Refined air dispersion models are, however, more resource intensive than their screening counterparts. It is advisable to first use a screening model to obtain conservative concentration estimates and calculate health risks. If the health risks are estimated to be above the threshold of concern, then use of a refined model to calculate more representative concentration and health risk estimates would be warranted. There are situations when screening models represent the only viable alternative (e.g., when representative meteorological data are not available). The district or reviewing authority should be consulted to determine the appropriate method for determining the level of detail in the modeling analysis. The HARP software will incorporate the capability of using either representative meteorological data from AERMOD or the default meteorological conditions from the AERSCREEN model.

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

- with worst-case meteorology instead of representative meteorology;

- with a conservative averaging period conversion factor to calculate longer term concentration estimates (see Section 4.10 for more discussion on screening air dispersion models and adjustments factors).

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

4.6 Population Exposure

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

In addition to estimating individual cancer risk at specific points such as the MEI (maximally exposed individual), OEHHA recommends determining the number of people who reside within the 1×10^{-6}, 1×10^{-5}, 1×10^{-4}, and higher cancer risk isopleths. For noncancer population evaluations, the number of people who reside within the 0.5, one, five, or higher hazard index isopleths should be reported. The HARP software can provide population exposure estimates as cancer burden or as the number of persons exposed to a selected (user identified) health risk/impact level. Information on obtaining the HARP software can be found under the Hot Spots Program on the ARB's web site at *www.arb.ca.gov*. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.6.1 *Zone of Impact*

As part of the estimation of the population exposure for the cancer risk analysis, it is necessary to determine the geographic area affected by the facility's emissions. An initial approach to define a "zone of impact" surrounding the source is to generate an isopleth where the total excess lifetime cancer risk from inhalation exposure to all emitted carcinogens is greater than 10^{-6} (one in 1,000,000). For noncarcinogens, a second, third, and fourth isopleth (to represent the chronic, 8-hour, and acute impacts) should be created to define the zone of impact for the hazard index from both inhalation and noninhalation pathways greater than or equal to 1.0. For clarity these isopleths may need to be presented on separate maps in the HRA.

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

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

- Calculate total cancer risk and hazard index (HI) for each receptor location by using the methods provided in the risk characterization sections in Chapter 8 of the Air Toxics Hot Spots Risk Assessment Guidance Manual.

- Find the distance where the total inhalation cancer risk is equal to 10^{-6}; this may require redefining the receptor array in order to have two receptor locations that bound a total cancer risk of 10^{-6}. Next, find the distance where the chronic, 8-hour, and acute health hazard indices are declared significant by the District (e.g., acute, 8-hour, or chronic HI = 1.0).

Some Districts may prefer to use a cancer risk of 10^{-7} or an HI of 0.5 as the zone of impact. Therefore, the District should be consulted before modeling efforts are initiated. If the zone of impact is greater than 25 km from the facility at any point, then the District should be consulted. The District may specify limits on the area of the zone of impact. Ideally, these preferences would be presented in the modeling protocol (see Section 4.14).

Note that when depicting the risk assessment results, risk isopleths must present the total cancer and noncancer risk from both inhalation and noninhalation pathways. The zone of impact should be clearly shown on a map with geographic markers of adequate resolution (see Section 4.6.3.1). The text below discusses methodology for defining the zone of impact and has format recommendations. Chapter 9 provides an outline that specifies the content and recommended format of all HRA results.

The zone of impact can be defined once the exposure assessment (air dispersion modeling) process has determined the pollutant concentrations at each designated off-site receptor and a risk analysis (see Chapter 8) has been performed. For clarity,

the cancer and noncancer zone(s) of impact should be presented on separate maps. A map illustrating the carcinogenic zone of impact is required. The District may at their discretion ask for the map illustrating the potential carcinogenic zone of impact to identify the zone of impact for the minimum exposure pathways (inhalation, soil, dermal, and mothers milk) and the zone of impact for all applicable pathways of exposure (minimum pathways plus site/route dependent pathways). Two maps may be needed to accomplish this. The legend of these maps should state the level(s) used for the zone of impact and identify the exposure pathways that were included in the assessment.

The noncancer maps should also clearly identify the noncancer zones of impact. These include the acute (inhalation) zone of impact, 8-hour (inhalation) zone of impact and the chronic (including both inhalation, multipathway) zone of impact. The District may at its discretion require separate chronic inhalation and chronic multipathway zones of impact maps. For clarity, presentation of the two chronic zones of impact may also require two or more maps. The legend of these maps should state the level(s) used for the zone of impact and identify the exposure pathways (and target organs) that were included in the assessment. Further information regarding the methods for determination of hazard indices and cancer risk are discussed in Chapter 8 and Appendix I.

4.6.2 *Screening Population Estimates for Risk Assessments*

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

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

2. Calculate the potential cancer risk and hazard index for each receptor location by using the methods provided in the risk characterization sections of this document (Chapter 8).

3. Find the distance where the potential cancer risk is equal to District specified levels (e.g., 10^{-6}); this may require redefining the receptor array in order to have

two receptor locations that bound a total cancer risk of 10^{-6}. This exercise should be repeated for the noncancer health impacts.

4. Calculate cancer burden by estimating the number of people in the grid and stipulate that all are exposed at the highest level.

4.6.3 *Refined Population Estimates for Risk Assessments*

The refined HRA requires a detailed analysis of the population exposed to emissions from the facility. Where possible, a detailed population exposure analysis provides estimates of the number of individuals in residences and 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). These population analyses can include exposure estimates for workers and residents through the use of land use maps or other tools. The overall exposed residential and worker populations should be apportioned into smaller geographic subareas. The information needed for each subarea is:

1. The number of exposed persons, and
2. The receptor location at which the calculated ambient air concentration is assumed to be representative of the exposure to the entire population in the subarea.

A multi-tiered approach is suggested for the population analysis. Census tracts, which the facility could significantly impact, should be identified (see Section 4.6.3.1). A census tract should be divided into smaller subareas if it is close to the facility where ambient concentrations vary widely. The District may determine that census tracts provide sufficient resolution near the facility to adequately characterize population exposure or they may prefer the census information to be evaluated using smaller blocks. Further downwind where ambient concentrations are less variable, the census tract level may be acceptable to the District. The District may determine that the aggregation of census tracts (e.g., when the census tracts making up a city are combined) is appropriate for receptors that are considerable distances from the facility.

If a facility must also comply with the RCRA/CERCLA HRA requirements, health effects to on-site workers may also need to be addressed. The DTSC's Remedial Project Manager should be consulted on this issue. In some cases it may be appropriate to evaluate risks to on-site receptors. The district should be consulted about special cases for which evaluation of on-site receptors is appropriate, such as facilities frequented by the public or where people may reside (e.g., military facilities).

4.6.3.1 Census Tracts

For a refined risk assessment, the boundaries of census tracts can be used to define the geographic area to be included in the population exposure analysis. Digital maps showing the census tract boundaries in California can be obtained from "The Thomas

Guide"® on the World Wide Web. Statistics for each census tract can be obtained from the U.S. Census Bureau. The website address for the U.S. Census Bureau is http://www.census.gov. Numerous additional publicly accessible or commercially available sources of census data can be found on the World Wide Web. A specific example of a census tract is given in Appendix K. The HARP software includes U.S. census data and is a recommended tool for performing population exposure estimates.

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

(1) Identify the "zone of impact" (as defined previously in Section 4.6.1) on a map detailed enough to provide for resolution of the population to the subcensus tract level. (The U.S. Geological Survey (USGS) 7.5-minute series maps and the maps within the HARP software provide sufficient detail.) This is necessary to clearly identify the zone of impact, location of the facility, and sensitive receptors within the zone of impact. If significant development has occurred since the USGS survey, this should be indicated. A specific example of a 7.5-minute series map is given in Appendix K.

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

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

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

4.6.3.1.1 Subcensus Tract

Within each census tract are smaller population units. These units [urban block groups (BG) and rural enumeration districts (ED)] contain about 1,100 persons. BGs are

further broken down into statistical units called blocks. Blocks are generally bounded by four streets and contain an average of 70 to 100 persons. However, this range in population is an average and population units may vary significantly. In some cases, the EDs are very large and identical to a census tract.

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

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

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

4.6.4 *Sensitive Receptor Locations*

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

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

4.7 Receptor Siting

4.7.1 *Receptor Points*

The modeling analysis should contain a network of receptor points with sufficient detail (in number and density) to permit the estimation of the maximum concentrations. Locations that must be identified include the maximum estimated off-site impact or point of maximum impact (PMI), the maximum exposed individual at an existing residential receptor (MEIR), and the maximum exposed individual at an existing occupational worker receptor (MEIW). Note that some situations may also require that on-site receptor (worker or residential) locations be evaluated. The risk assessor should contact the District for guidance if on site exposure situations are present at the emitting

facility. These on-site locations should be included in the HRA. Some examples where the health impacts of on-site receptors may be appropriate could be military base housing, prisons, universities, day care facilities, or locations where the public may have regular access for the appropriate exposure period (e.g., a lunch time café or museum for acute exposures). When a receptor lives and works on the facility, site, or property, then these receptors should be evaluated and reported under both residential and worker scenarios and the one that is most health protective should be used for risk management decisions. The cancer risk estimates for the onsite residents may use a 30-year exposure duration while the 25-year exposure duration is used for a worker. Under a Tier 2 analysis, alternate exposure durations may be evaluated and presented with all assumptions supported.

All of these locations (i.e., PMI, MEIR, and MEIW) must be identified for potential multipathway carcinogenic and noncarcinogenic effects. It is possible that the estimated PMI, MEIR, and MEIW risk for cancer, chronic noncancer, 8-hour, acute noncarcinogenic risks occur at different locations. Some facilities will not have off-site workers in the vicinity of the facility and will not need to evaluate worker exposure. The approval to omit the MEIW receptor should be verified in writing with the District or reviewing authority and included in the HRA.

Other sensitive receptor locations may also be of interest and required to be included in the HRA. The District or reviewing authority should be consulted to determine which sensitive receptor locations must be included.

The results from a screening model (if available) can be used to identify the area(s) where the maximum concentrations are likely to occur. Receptor points should also be located at the population centroids (see Section 4.7.2) and sensitive receptor locations (see Section 4.6.4). The exact configuration of the receptor array used in an analysis will depend on the topography, population distribution patterns, and other site-specific factors. All receptor locations should be identified in the HRA using UTM (Universal Transverse Mercator) coordinates and receptor number. The receptor numbers in the summary tables should match receptor numbers in the computer output (e.g., HARP output files). In addition to actual UTM coordinates, the block/street locations (i.e., north side of 3,000 block of Smith Street) should be provided in the HRA for the PMI, MEIR, and MEIW for carcinogenic and noncarcinogenic health effects. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

4.7.1.1 Receptor Height

To evaluate localized impacts, receptor height should be taken into account at the point of maximum impact on a case-by-case basis. For example, receptor heights may have to be included to account for receptors significantly above ground level. Flagpole receptors at the height of the breathing zone of a person may need to be considered when the source receptor distance is less than a few hundred meters. Consideration must also be given to the noninhalation pathway analysis which requires modeling of chemical deposition onto soil or water at ground level. For the inhalation pathway, a health protective approach is to select a receptor height from 0 meters to 1.8 meters

that will result in the highest predicted downwind concentration. Final approval of this part of the modeling protocol should be with the District or reviewing authority.

4.7.2 *Centroid Locations*

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

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

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

4.7.3 *Spatial Averaging*

Since the inception of the “Hot Spots” and California’s Air Toxics Programs, HRA results for an individual receptor have typically been based on air dispersion modeling results at a single point or location. With a few exceptions, this method has been traditionally used for all types of receptors (e.g., PMI, MEIR, MEIW, pathway receptors, etc.). The assumptions used in risk assessment are designed to prevent underestimation of health impacts to the public resulting in a health protective approach. However, basing risk estimates on a single highest point (PMI, MEIR, or MEIW) does not take into account that a person does not remain at one location on their property, or in one location at the workplace over an extended period of time. Therefore, the average air concentration over a small area is likely to be more representative than using the air concentration at a single point, particularly in those situations where concentrations fall off rapidly around that single point. The concept of averaging air concentrations over a small area is known as spatial averaging.

In order to understand how spatial averaging can impact air dispersion modeling results with various types of facilities, the ARB, in conjunction with the OEHHA, performed sensitivity analyses to evaluate the impacts of spatially averaging air dispersion modeling results (see Appendix C of the Air Toxics Hot Spots Program Risk Assessment

Guidelines: Technical Support Document for Exposure Assessment and Stochastic Analysis (EASA)). Based on these sensitivity analyses, it is reasonable and appropriate to include spatial averaging techniques in air toxic risk assessments as supplemental information to Tier 1 information (i.e., modeling results that are based on the air concentration from a single point or location). While all risk assessments must include results based on Tier 1 methodology, the spatially averaged concentrations around the point of interest (e.g., PMI, MEIR, MEIW, multipathway exposure evaluations, etc.) could also be included as an option in risk assessments and acceptable for risk management decisions subject to approval by the District or reviewing agency. Spatial averaging is an option for the purpose of additional refinement to the risk assessment.

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

- Averaging results over a small domain will give a more representative picture of individual exposure and risk than an estimate based on one single location within their property.
- Spatial averaging will allow air dispersion modeling and risk assessment results to be characterized as the estimated concentration and risk in a discrete area of interest, rather than an exact value for a single location.
- From a risk communication standpoint, the ARB and OEHHA feel it is more appropriate to present the modeling output and the calculated health impacts as the potential impacts within a small or discrete area, rather than an exact value at a specific point on a grid or map.
- Spatial averaging is the recommended procedure in ARB's Lead Risk Management Guidelines (2001) and has been used in several complex source HRAs [e.g., Roseville Railyard (2004), Ports of LA/LB (2006), Port of Oakland (2008)].
- Spatially averaging the deposition concentrations over pasture land, a garden, or a water body for multipathway exposure scenarios is a planned upgrade for the HARP Software. This will provide an option that will refine multipathway exposure assessments. Average deposition on these types of areas (e.g., a water body) is not necessarily well represented by the single highest point of deposition, or deposition at the geographic center of the water body. Likewise, since produce is grown over the entire surface of the garden and cows graze the entire pasture, deposition is better estimated by evaluating the entire area rather than using a single point.

4.7.3.1 Spatial Averaging Methodology

The spatial averaging sensitivity study in Appendix C of the EASA is based on simulating emissions from a point, volume, area, and line sources. Most source types (e.g., point) are simulated as a small, medium or large source. Line sources are only simulated as small and large. In addition, meteorological data collected at five different locations in California were used. Nested spatial average grids of various domains

were used to study the differences on the spatial average concentration. In the case of the 20 meter by 20 meter spatial average nested grid, the spatial average concentration showed little change over the PMI for medium and large sources. In the case for small sources, the spatial average concentration is approximately 45% to 80% of the PMI concentration. Individual source type and meteorological conditions will cause variations in these results.

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

- The MEIR, MEIW, or PMI is located at the fence line or close to the emission source.
- The concentration gradient is high near the PMI. This is more associated with low level plumes such as fugitive, volume, area, or short stacks.
- A long term average is being calculated to represent a multi-year risk analysis based on one to five years of meteorological data. Note that spatial averaging should **not** be used for short term (acute) calculations.

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

1. Locate the point(s) of interest and receptor(s) (i.e., PMI, MEIW, MEIR, and any additional receptor locations of interest or concern) with a grid resolution spacing of no greater than five meters. To achieve this, two or more modeling runs with successively finer nested grid resolutions may be needed to find the final location where the nested grid that will be used for spatial averaging will be placed.

2. Center the spatial average nested grid on the each receptor's location of interest determined in step 1. Limit the nested grid to no larger than 20 meters by 20 meters or 400 square meters. Note if a portion of the centered and nested grid falls within the facility boundary and the receptor location of interest is outside of the boundary, then adjustments to the nested grid to obtain the spatially-averaged concentration for the offsite receptor are reasonable. This may be done by either repositioning the nested grid to cover 400 square meters of off-boundary area surrounding the receptor or center the nested grid and delete any on-site grid points so that only the offsite grid points surrounding the receptor are used in the spatially averaged concentration. The grid resolution spacing should be no greater than five

meters. With a five meter grid resolution, the 20 meter by 20 meter domain will result in 25 receptors. The size, shape, and placement of the domain and the resolution of points are subject to approval by the District, ARB, or other reviewing authority. See the Sections 4.7.3.1.2 and 4.7.3.1.3 below for additional discussion on domain sizing and grid spacing at worksites, pastures, gardens, and water bodies.

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

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

5. Document and include all methods, assumptions, data, maps, and files used in the spatial averaging analysis and clearly present this information in the risk assessment following the requirements of the District or reviewing authority. Note that in the update to the HARP software, functionality will be included that will assist with spatial averaging and the methodology discussed.

The following sections discuss the use of spatial averaging for various receptor types and exposure pathways.

4.7.3.1.1 Residential Receptors

Follow the steps in Section 4.7.3 outlining the spatial averaging methodology. To remain health protective when evaluating a residential receptor, spatial averaging should not take place using large nested domains. The domain used for spatial averaging should be no larger than 20 meters by 20 meters with a maximum grid spacing resolution of equal to or less than five meters. This domain represents an area that is approximately the size of a small urban lot. The size of the domain and resolution of points shall be subject to approval by the District, ARB, or other reviewing authority.

4.7.3.1.2 Worker Receptors

Offsite worker locations (e.g. MEIW) may also be a candidate for spatial averaging. However, workers can be at the same location during almost their entire daily work shift (e.g., desk/office workers). When this is the situation, then the traditional method of using a single location and corresponding modeled concentration is appropriate. If spatial averaging is used, care should be taken to determine the proper domain size and grid resolution. Follow the steps in Section 4.7.3 outlining the spatial averaging

methodology. To be consistent with the residential receptor assumptions and remain health protective, a modeling domain size no larger than 20 meters by 20 meters is recommended with a grid spacing resolution of equal to or less than five meters. However, if workers routinely and continuously move throughout the worksite over a space greater than 20 meters by 20 meters, then a larger domain may be considered. The HRA or modeling protocol shall support all assumptions used, including, but not limited to, documentation for all workers showing the area where each worker routinely performs their duties and the percentage of time spent in those areas. The final domain size should not be greater than the smallest area of worker movement. Other considerations for determining domain size and grid spacing resolution may include an evaluation of the concentration gradients across the worker area. The grid spacing used within the domain to find the concentration that will be used to calculate health impacts should be sufficient in number and detail to obtain a representative concentration across the area of interest. The size of the domain and resolution of points shall be subject to approval by the District, ARB, or other reviewing authority.

4.7.3.1.3 Pastures, Gardens, or Water Bodies

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

If spatial averaging will be done, follow the steps in Section 4.7.3.1 outlining the spatial averaging methodology. When using spatial averaging over the deposition area, care should be taken to determine the proper domain size to make sure it includes all reasonable areas of potential deposition. The size and shape of the area of interest (e.g., pasture or water body) should be identified and used for the modeling domain. The grid spacing or resolution used within the domain should be sufficient in detail to obtain a representative deposition concentration across the area of interest. One way to determine the grid resolution is to include an evaluation of the concentration gradients across the deposition area. The HRA or modeling protocol shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the pasture, garden, or water body, maps, representative coordinates, grid resolution, concentration gradients, etc.). The size of the domain and grid resolution is subject to approval by the reviewing authority.

In lieu of following the details in the paragraph above, the approach used for the other receptors (e.g., MEIR, MEIW) that uses a domain size not greater than 20 meters by 20 meters, located on the PMI within the area of interest, with a maximum grid spacing resolution of five meters, can be used. This default refined approach would apply to deposition areas greater than 20 meters by 20 meters. For smaller deposition areas, the simplified approach of using the PMI for the area, the concentration at the centroid

or a specific point of interest, or averaging over the actual smaller domain can be used. This again is subject to approval by the reviewing authority.

The HRA or modeling protocol shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the water body, pasture, or garden; all data; maps; representative coordinates, and etc.), and the details clarifying how and where the averaging was done (e.g., location and magnitude of concentration gradients, the grid spacing used).

4.8 Meteorological Data

Refined air dispersion models require hourly meteorological data. The first step in obtaining meteorological data should be to check with the District and the ARB for data availability. Other sources of data include the National Weather Service (NWS), National Climatic Data Center (NCDC), Asheville, North Carolina, ARB meteorological database (METDB), military stations and private networks. Meteorological data for a subset of NWS stations are available from the U.S. EPA Support Center for Regulatory Air Models (SCRAM). The SCRAM can be accessed at www.epa.gov/scram001/main.htm. All meteorological data sources should be approved by the District. Data not obtained directly from the District or the ARB should be checked for quality, representativeness, and completeness. It should be approved by the District before use. U.S. EPA provides guidance (U.S. EPA, 1995e) for these data. Meteorological data may need further processing. Data users can consult with the District or the ARB on how to process the raw meteorological data. The risk assessment should indicate if the District required the use of a specified meteorological data set. All memos indicating District approval of meteorological data should be attached in an appendix. If no representative meteorological data are available, screening procedures should be used as indicated in Section 4.10.

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

To determine long term average concentrations the data can be averaged. For calculation of the one-hour maximum concentrations needed to evaluate acute effects, the worst-case year should be used in conjunction with the maximum hourly emission rate. For example, the long term average concentration and one-hour maximum concentration at a single receptor for five years of meteorological data are calculated below:

Year	Annual Average ($\mu g/m^3$)	Maximum One-Hour ($\mu g/m^3$)
1	7	100
2	5	80
3	9	90
4	8	110
5	6	90
5-year average	7	

In the above example, the long-term average concentration over five years is 7 $\mu g/m^3$. Therefore, 7 $\mu g/m^3$ should be used to evaluate carcinogenic and chronic effects (i.e., annual average concentration). The one-hour maximum concentration is the highest one-hour concentration in the five-year period. Therefore, 110 $\mu g/m^3$ is the peak one-hour concentration that should be used to evaluate acute effects.

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.

4.8.1 *Meteorological Data Formats*

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

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

4.8.2 *Treatment of Calms*

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

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

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

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

4.8.3 *Treatment of Missing Data*

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

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

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

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

(4) 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.

4.8.4 *Representativeness of Meteorological Data*

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

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

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

4.8.4.1 Spatial Dependence

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

- Aspect ratio of terrain, i.e., ratio of:
    - Height of valley walls to width of valley;
    - Height of ridge to length of ridge; and
    - Height of isolated hill to width of hill at its base

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

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

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

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

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

4.8.4.2 Temporal Dependence

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

4.8.4.3 Further Considerations

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

It is important to recognize that, although certain meteorological variables may be considered unrepresentative of another site (for instance, wind direction or wind speed), other variables may be representative (such as temperature, dew point, cloud cover).

Exclusion of one variable does not necessarily exclude all. For instance, one can argue that weather observations made at different locations are likely to be similar if the observers at each location are within sight of one another - a stronger argument can be made for some types of observations (e.g., cloud cover) than others. Although by no means a sufficient condition, the fact that two observers can "see" one another supports a conclusion that they would observe similar weather conditions.

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

4.8.5 *Alternative Meteorological Data Sources*

It is necessary, in the consideration of most air pollution problems, to obtain data on site-specific atmospheric dispersion. Frequently, an on-site measurement program must be initiated. As discussed in Section 4.8.5, 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).

4.8.5.1 Recommendations

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

If data are missing from the primary source, they should be handled as follows, in order of preference: (l) substitution of other representative on-site data; (2) linear interpolation of one or two missing hours; (3) substitution of representative off-site data; or (4) coding as a missing data field, according to the discussions in Section 4.8.5.

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

4.8.6 *Quality Assurance and Control*

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

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

1. Project description - how meteorology data are to be used
2. Project organization - how data validity is supported
3. QA objective - how QA will document validity claims
4. Calibration method and frequency - for data
5. Data flow - from samples to archived valid values
6. Validation and reporting methods - for data
7. Audits - performance and system
8. Preventive maintenance
9. Procedures to implement QA objectives - details
10. Management support - corrective action and reports

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

4.9 Model Selection

There are several air dispersion models that can be used to estimate pollutant concentrations and new ones are likely to be developed. U.S. EPA added AERMOD, which incorporates the PRIME downwash algorithm, to the list of preferred models in 2005 as a replacement to ISCST3. CalPuff was added in 2003. The latest version of the U.S. EPA recommended models can be found at the SCRAM Bulletin board located at http://www.epa.gov/scram001. However, any model, whether a U.S. EPA guideline model or otherwise, must be approved for use by the local air district. Recommended models and guidelines for using alternative models are presented in this section. All air dispersion models used to estimate pollutant concentrations for risk assessment analyses must be in the public domain. Classification according to terrain, source type and level of analysis is necessary before selecting a model (see Section 4.4). The selection of averaging times in the modeling analysis is based on the health effects of

concern. Annual average concentrations are required for an analysis of carcinogenic or other chronic effects. One-hour maximum concentrations are required for analysis of acute effects.

4.9.1 *Recommended Models*

Recommended air dispersion models to estimate concentrations for risk assessment analyses are generally referenced in US EPA's Guideline on Air Quality Models available at http://www.epa.gov/scram001. Currently AERMOD is recommended for most refined risk assessments in flat or complex terrain and in rural or urban environments[1]. In addition, CalPuff is available where spatial wind fields are highly variable or transport distances are large (e.g., 50 km). AERSCREEN is a screening model based on AERMOD. AERSCREEN can be used when representative meteorological data are unavailable. CTSCREEN is available for screening risk assessments in complex terrain. The most current version of the models should be used for risk assessment analysis. Some facilities may also require models capable of special circumstances such as dispersion near coastal areas. For more information on modeling special cases see Sections 4.12 and 4.13.

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

To further facilitate the model selection, the District should be consulted for additional recommendations on the appropriate model(s) or a protocol submitted for District review and approval (see Section 4.14.1).

4.9.2 *Alternative Models*

Alternative models are acceptable if applicability is demonstrated or if they produce results identical or superior to those obtained using one of the preferred models referenced in Section 4.9.1. For more information on the applicability of alternative models refer to the following documents:

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

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

4.10 Screening Air Dispersion Models

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

Some screening models provide only 1-hour average concentration estimates. Other averaging periods can be estimated based on the maximum 1-hour average concentration in consultation and approval of the responsible air district. Because of variations in local meteorology, the exact factor selected may vary from one district to another. Table 4.2 provides guidance on the range and typical values applied. The conversion factors are designed to bias predicted longer term averaging periods towards overestimation.

Table 4.2 Recommended Factors to Convert Maximum 1-hour Avg. Concentrations to Other Averaging Periods (U.S. EPA, 2011, 1995a; ARB, 1994).

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

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

4.10.1 *AERSCREEN*

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

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

4.10.2 *Valley Screening*

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

4.10.2.1 Regulatory Options

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

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

4.10.3 *CTSCREEN*

The CTSCREEN model (Perry et al., 1990) is the screening mode of the Complex Terrain Dispersion Model (CTDMPLUS). CTSCREEN can be used to model single point sources only. It may be used in a screening mode for multiple sources on a case by case basis in consultation with the District. CTSCREEN is designed to provide conservative, yet theoretically sounder, worst-case 1-hour concentration estimates for receptors located on terrain above stack height. Internally-coded time-scaling factors are applied to obtain other averages (see Table 4.3). These factors were developed by comparing the results of simulations between CTSCREEN and CTDMPLUS for a variety of scenarios and provide conservative estimates (Perry et al., 1990). CTSCREEN produces identical results as CTDMPLUS if the same meteorology is used in both models. CTSCREEN accounts for the three-dimensional nature of the plume and terrain interaction and requires detailed terrain data representative of the modeling domain. A summary of the input parameters required to run CTSCREEN is given in Table 4.4. The input parameters are provided in three separate text files. The terrain topography file (TERRAIN) and the receptor information file (RECEPTOR) may be generated with a preprocessor that is included in the CTSCREEN package. In order to generate the terrain topography file the analyst must have digitized contour information.

Table 4.3 Time-scaling factors internally coded in CTSCREEN

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

Table 4.4 Input Parameters Required to Run CTSCREEN

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

4.11 Refined Air Dispersion Models

Refined air dispersion models are designed to provide more representative concentration estimates than screening models. In general, the algorithms of refined models are more robust and have the capability to account for site-specific meteorological conditions. For more information regarding general aspects of model selection see Section 4.9.

4.11.1 *AERMOD*

For a wide variety of applications in all types of terrain, the recommended model is AERMOD. AERMOD is a steady-state plume dispersion model for assessment of pollutant concentrations from a variety of sources. AERMOD simulates transport and dispersion from multiple point, area, or volume sources based on an up-to-date characterization of the atmospheric boundary layer. Sources may be located in rural or urban areas and receptors may be located in simple or complex terrain. AERMOD accounts for building wake effects (i.e., plume downwash) based on the PRIME building downwash algorithms. The model employs hourly sequential preprocessed meteorological data to estimate concentrations for averaging times from one hour to one year (also multiple years). AERMOD is designed to operate in concert with two pre-processor codes: AERMET processes meteorological data for input to AERMOD, and AERMAP processes terrain elevation data and generates receptor information for input to AERMOD. Guidance on input requirements may be found in the AERMOD Users Guide.

4.11.1.1 Regulatory Options

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

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

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

4.11.1.2 Special Cases

a. Building Downwash:
AERMOD automatically determines if the plume is affected by the wake region of buildings when their dimensions are given. The specification of building dimensions does not necessarily mean that there will be downwash. See

Section 4.13.1 for guidance on how to determine when downwash is likely to occur.

b. Area Sources:

The area source algorithm in AERMOD estimates source emission strength by integrating an area upwind of the receptor location. Receptors may be placed within the area itself, downwind of the area or adjacent to the area. However, since the vertical distribution parameter (σ_z) goes to zero as the downwind distance goes to zero, the plume function solution is infinite for a downwind receptor distance of zero. In order to avoid such singularity in the plume function solution, the AERMOD model arbitrarily sets the plume function to zero when the receptor distance is less than one meter. As a result, the area source algorithm will not provide reliable solutions for receptors located within or adjacent to very small areas, with dimensions on the order of a few meters across. In these cases, the receptor should be placed at least one meter outside of the area.

c. Volume Sources:

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

d. Line Sources:

Line sources are a special case of a series of volume or area sources. Where the emission source is neutrally buoyant, such as a conveyor belt, AERMOD can be used according to the user guide. In the event that the line source is a roadway, then additional considerations are required.

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

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

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

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

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

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

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

h. 8-hour Modeling for the Offsite Worker's Exposure and Residential Exposure:
If the ground level air concentrations from a facility operation 5 days a week/ 8 hours per day have been estimated by a 24 hour per day annual

average, an adjustment factor can be applied to estimate the air concentration that offsite worker with the same schedule would be exposed to. The 24-hour annual average concentration is multiplied times 4.2.

If the meteorology during the time that the facility is emitting is used, hourly model simulations need to be post-processed to cull out the data needed for the offsite worker exposure. See Appendix M for information on how to calculate the refined offsite worker concentrations using the hourly raw results from the AERMOD air dispersion model. For more discussion on worker exposure, see Section 4.8.1.

Eight-hour exposure modeling can be used to evaluate the potential for health impacts (including effects of repeated exposures) in children and teachers exposed during school hours. Although not required in the HRA, 8-hour exposure modeling could also be performed at the discretion of the District to a residential scenario (i.e., the MEIR) where a facility operates only a portion of the day and exposure to residences are not adequately reflected by averaging concentrations over a 24 hour day.

4.11.1.3 HARP Dispersion Analysis

It is highly recommended that air dispersion analysis be performed using the HARP software. HARP can perform refined dispersion analysis by utilizing the U.S. EPA standard program AERMOD. In the future, the updated version of HARP will link the AERMOD outputs with risk assessment modules.

4.11.2 *CTDMPLUS*

CTDMPLUS is a Gaussian air quality model for use in all stability conditions in complex terrain. In comparison with other models, CTDMPLUS requires considerably more detailed meteorological data and terrain information that must be supplied using specifically designed preprocessors. CTDMPLUS was designed to handle up to 40 point sources.

4.12 Modeling to Obtain Concentrations used for Various Health Impacts

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

4.12.1 *Emission Rates for Cancer, Chronic, and Acute Health Impacts*

As discussed in Section 4.2.1.1, the HRA should include both annual average emissions and maximum 1-hour emissions for each pollutant emitted by the facility. Maximum 1-hour emissions are used for acute noncancer health impacts while annual emissions are used for chronic exposures (i.e., chronic and 8-hour noncancer health impacts or cancer risk assessment). When applying the emission rates in the air

dispersion analysis, it is important not to artificially inflate or deplete the reported emission inventory.

For annual average emissions, the emissions are spread evenly over the entire year for continuous emitting sources. However, for sources where the emission patterns vary (i.e., non-continuous emitting sources), the emission rate should also account for the facility's emission schedule. If appropriate, the variable emissions rate option (e.g., hour-of-day) should be used in the air dispersion analysis. For more information consult the AERMOD User's Guide (U.S. EPA, 2004a). Also, when calculating emission rates for acute health impacts, it is important the emission rates never exceed the reported maximum 1-hour emissions.

4.12.2 *Modeling and Adjustments for Inhalation Cancer Risk at a Worksite*

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

In lieu of post-processing the hourly dispersion model output, the more typical approach is to obtain the long-term average concentration as you would for modeling a residential receptor and approximate the worker's inhalation exposure using an adjustment factor. The actual adjustment factor that is used to adjust the concentration may differ from the example below based on the specifics of the source and worker receptor (e.g., work-shift overlap). Once the worker's inhalation concentration is determined, the inhalation dose is calculated using additional exposure frequency and duration adjustments. See Chapter 5 for more information on the inhalation dose equation.

4.12.2.1 Non-Continuous Sources

When modeling a non-continuously emitting source (e.g., operating for eight hours per day and five days per week), the modeled long-term average concentrations are based on 24 hours a day and seven days per week for the period of the meteorological data set. Even though the emitting source is modeled using a non-continuous emissions schedule, the long-term concentration is still based on 24 hours a day and seven days per week. Thus, this concentration includes the zero hours when the source was not operating. For the offsite worker inhalation risk, we want to determine the long-term concentration the worker is breathing during their work shift. Therefore, the long-term concentration needs to be adjusted so it is based only on the hours when the worker is

present. For example, assuming the emitting source and worker's schedules are the same, the adjustment factor is 4.2 = (24 hours per day/8 hours per shift)x(7 days in a week/5 days in a work week). In this example, the long term residential exposure is adjusted upward to represent the exposure to a worker. Additional concentration adjustments may be appropriate depending on the work shift overlap. These adjustments are discussed below.

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

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

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

c. Calculate the worker adjustment factor (WAF) using Equation 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 present during the time that the source is emitting, the worker is not exposed to the source's emission (i.e., the DF in Equation 2.2 becomes 0).

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

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.

4.12.2.2 Continuous Sources

If the source is continuously emitting, then the worker is assumed to breathe the long-term annual average concentration during their work shift. Equation 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.

4.12.3 *Modeling and Adjustments for Noncancer 8-Hour RELs*

For 8-hour noncancer health impacts, we evaluate if the receptor (e.g., worker or resident) is exposed to an 8 hour average concentration, occurring daily, that exceeds the 8-hour REL. The 8 hour RELs were derived primarily for the offsite worker scenario. Although not required in an HRA, residential receptors can be evaluated with an 8-hour REL at the discretion of the District or Reviewing authority. For ease, we use a worker receptor in this discussion and in the discussion below for a non-continuously emitting source. The daily average concentration is intended to represent the long-term average concentration the worker is breathing during 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. In lieu of post-processing the hourly dispersion model output described in Appendix M, the more typical approach is to obtain the long-term average concentration as you would for modeling a residential receptor and approximate the worker's inhalation concentration using an adjustment factor. The method for applying the adjustment factor is described in the section below.

The results from the 8-hour hazard index calculations should not be combined with the chronic or acute hazard indices. Each of the potential noncancer health impacts should be reported independently. See Chapter 8 for more discussion on calculating health impacts.

4.12.3.1 Non-Continuous Sources

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

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.

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

c. Calculate the worker adjustment factor (WAF) using Equation 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 inhalation REL do not apply if the source's emission schedule and the offsite worker's schedule do not overlap at some point.

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

Eq. 2.3

Where:

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

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

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

4.12.3.2 <u>Continuous Sources</u>

If the source is continuously emitting, then the worker is assumed to breathe the long-term annual average concentration during their work shift and no concentration adjustments are made when estimating 8-hour health impacts. Note however, if an assessor does not wish to assume the worker breathes the long-term annual average

concentration during the work shift, then a refined concentration can be post-processed as described in Appendix M. All alternative assumptions should be approved by the reviewing authority and supported in the presentation of results.

Note that 8 hr RELs are not typically used for continuously emitting sources for residential receptors. In this situation it is only necessary to estimate a chronic Hazard Index using the annual average concentrations and chronic RELs. However, there may be situations where the District may wish to assess an 8 hr Hazard Index, for example, where there are significant differences in modeled concentration of emissions during the day due to diurnal wind patterns.

4.12.4 *Modeling and Adjustment Factors for Noncancer Chronic RELs*

Potential chronic noncancer health impacts use the long-term annual average concentration regardless of the emitting facility's schedule. No adjustment factors should be used to adjust this concentration. Chronic RELs are used to assess not only residential health impacts, but in many cases worker health impacts as well. There are currently only a limited number of substances with an 8-hour inhalation REL, and a facility may emit only, or mostly, substances that currently have just a chronic REL. Until there is 8-hour RELs for many of the Hot Spots substances that have a chronic REL value, we recommend determining the chronic HI for the MEIW to adequately protect the offsite worker.

The results from the chronic hazard index calculations are not combined with the 8-hour or acute hazard indices. All potential noncancer results should be reported independently. See Chapter 8 for more discussion on calculating health impacts.

4.12.5 *Modeling and Adjustments for Oral Cancer Potencies and Oral RELs*

When estimating the cancer risk or noncancer health impacts from noninhalation pathways, no adjustment is made to the long-term annual average concentration regardless of the emitting facility's schedule. Since the media (e.g., soil) at the receptor location where deposition takes place for noninhalation pathways is continuously present, the concentrations used for all noninhalation pathways are not adjusted (up or down) by an adjustment factor. However, some adjustments are made to the concentration once the pollutants reach the media, for example, pollutants undergo decay in soils. In addition, when the dose for each pathway is calculated, exposure adjustments may also be made. See Chapter 5 of this document and the Technical Support Document for Exposure Assessment and Stochastic Analysis (August 2012) to get more information on these types of adjustments. Oral cancer potencies and oral RELs are used to assess both residential or worker health impacts.

4.12.6 *Modeling One-Hour Concentrations using Simple and Refined Acute Calculations*

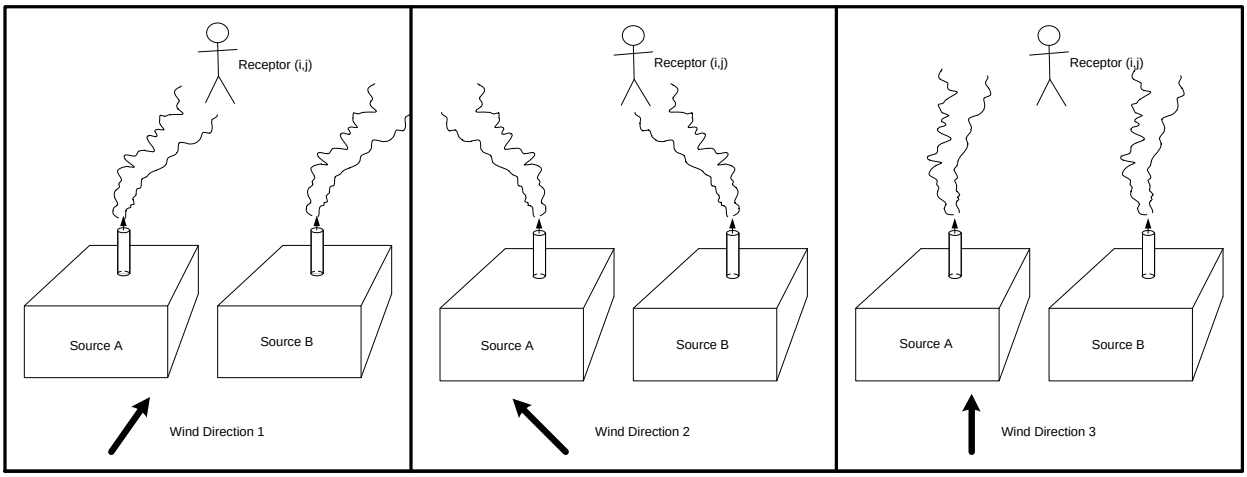
Modeled one-hour concentrations are needed for the acute health hazard index calculations. HARP has two methods to calculate this concentration; Simple and Refined. As an aid to understanding the differences between Simple and Refined,

Figure 2 shows three possible conditions showing how wind direction may vary and impact a downwind receptor (i,j) differently from just two sources (A and B).

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

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

Figure 2 Acute Scenarios



4.13 Modeling Special Cases; Specialized Models

Special situations arise in modeling some sources that require considerable professional judgment; a few of which are outlined below. It is recommended that the reader consider retaining professional consultation services if the procedures are unfamiliar. The following sections, taken mostly from the document “On-Site Meteorological Program Guidance for Regulatory Modeling Applications” (U.S. EPA,

1995e), provide general information on data formats and representativeness. Some Districts may have slightly different recommendations from those given here.

4.13.1 *Building Downwash*

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

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

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

4.13.2 *Deposition*

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

In the Air Toxics "Hot Spots" program, deposition is quantified for particle-bound pollutants and not gases. Wet deposition of water-soluble gas phase chemicals is thus not considered. When calculating pollutant mass deposited to surfaces without including depletion of pollutant mass from the plume airborne concentrations remaining in the plume and deposition to surfaces can be overestimated, thereby resulting in overestimates of both the inhalation and multi-pathway risk estimates. However, neglecting deposition in the air dispersion model, while accounting for it in the multipathway health risk assessment, is a conservative, health protective approach (CAPCOA, 1987; Croes, 1988). Misapplication of plume depletion can also lead to possible underestimates of multi-pathway risk and for that reason no depletion is the default assumption. If plume depletion is incorporated, then some consideration for possible resuspension is warranted. An alternative modeling methodology accounting

for plume depletion can be discussed with the Air District and used in an approved modeling protocol.

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

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

4.13.3 *Short Duration Emissions*

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

4.13.4 *Fumigation*

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

- Breaking up of a nocturnal radiation inversion by solar warming of the ground surface (rising warm unstable air); note that the break-up of a nocturnal radiation inversion is a short-lived event and should be modeled accordingly.
- Shoreline fumigation caused by advection of pollutants from a stable marine environment to an unstable inland environment
- Advection of pollutants from a stable rural environment to a turbulent urban environment

SCREEN3 incorporates concentrations due to inversion break-up and shoreline fumigation and is limited to maximum hourly evaluations. The Offshore and Coastal Dispersion Model incorporates overwater plume transport and dispersion as well as

changes that occur as the plume crosses the shoreline – hourly meteorological data are needed from both offshore and onshore locations.

4.13.5 *Raincap on Stack*

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

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

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

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

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

Stack tip downwash is a function of stack diameter, exit velocity, and wind speed. The maximum stack tip downwash is limited to three times the stack diameter in the AERMOD air dispersion model. In the event of a horizontal stack, stack tip downwash should be turned off and no stack height adjustments should be made. Note: This approach may not be valid for large (several meter) diameter stacks.

An alternative, more refined, approach could be considered for stack gas temperatures which are slightly above ambient (e.g., ten to twenty degrees Fahrenheit above ambient). In this approach, the buoyancy and the volume of the plume remain constant and the momentum is minimized.

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

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

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

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

4.13.6 *Landfill Sites*

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

4.14 Specialized Models

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

4.14.1 *Buoyant Line and Point Source Dispersion Model (BLP)*

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

4.14.1.1 Regulatory Application

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

- rural (IRU=l) mixing height option;
- default (no selection) for all of the following: plume rise wind shear (LSHEAR), transitional point source plume rise (LTRANS), vertical potential temperature gradient (DTHTA), vertical wind speed power law profile exponents (PEXP),

maximum variation in number of stability classes per hour (IDELS), pollutant decay (DECFAC), the constant in Briggs' stable plume rise equation (CONST2), constant in Briggs' neutral plume rise equation (CONST3), convergence criterion for the line source calculations (CRIT), and maximum iterations allowed for line source calculations (MAXIT); and

- terrain option (TERAN) set equal to 0.0, 0.0, 0.0, 0.0, 0.0, 0.0.

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

4.14.2 *Offshore and Coastal Dispersion Model (OCD)*

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

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

4.14.2.1 Regulatory Application

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

4.14.3 *Shoreline Dispersion Model (SDM)*

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

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

- tall stationary point sources located at a shoreline of any large body of water;
- rural or urban areas;

- flat terrain;
- transport distances less than 50 km;
- 1-hour to 1-year averaging times.

4.15 Interaction with the District

The risk assessor must contact the District to determine if there are any specific requirements. Examples of such requirements may include, but are not limited to: specific receptor location guidance, specific usage of meteorological data, and specific report format (input and output). See Chapter 9 for more information on the format and content of modeling protocols and HRAs.

4.15.1 *Submittal of Modeling Protocol*

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

<u>Outline for a Modeling Protocol</u>

I. Introduction

Include the facility name, address, and a brief overview describing the facility's operations.

- Provide a description of the terrain and topography surrounding the facility and potential receptors.
- Indicate the format in which data will be provided. Ideally, the report and summary of data will be on paper and all data and model input and output files will be provided electronically (e.g., compact disk or CD).
- Identify the guidelines used to prepare the protocol (e.g., District Guidelines).

II. Emissions

For each pollutant and process whose emissions are required to be quantified in the HRA, list the annual average emissions (pounds/year and grams/second) and the maximum one-hour emissions (pounds/hour and

grams/second)[2]. Maximum 1-hour emissions are used for acute noncancer health impacts while annual emissions are used for chronic exposures (i.e., chronic and 8-hour noncancer health impacts or cancer risk assessment).

- Identify the reference and method(s) used to determine emissions (e.g., source tests, emission factors, etc.). Clearly indicate any emission data that are not reflected in the previously submitted emission inventory report. In this event, a revised emission inventory report will need to be submitted to the District.

- Identify if this will be a multipathway assessment based on emitted substances.

III. Models / Modeling Assumptions

Specify the model and modeling assumptions

- Identify the model(s) to be used, including the version number.

- Identify the model options that will be used in the analysis.

- Identify the modeling domain(s) and the spacing of receptor grid(s). Grid spacing should be sufficient in number and detail to capture the concentration at all of the receptors of interest.

- Indicate complex terrain options that may be used, if applicable.

- Identify the source type(s) that will be used to represent the facility's operations (e.g., point, area, or volume sources, flare options or other).

- Indicate the preliminary source characteristics (e.g., stack height, gas temperature, exit velocity, dimensions of volume source, etc.).

- Identify and support the use of urban or rural dispersion coefficients for those models that require dispersion coefficients. For other models, identify and support the parameters required to characterize the atmospheric dispersion due to land characteristics (e.g., surface roughness, Monin-Obukhov length).

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[2] Except radionuclides, for which annual and hourly emissions are reported in Curies/year and millicuries/hour, respectively.

IV. Meteorological Data

Specify the type, source, and year(s) of hourly meteorological data (e.g., hourly surface data, upper air mixing height information).

- State how the data are representative for the facility site.
- Describe QA/QC procedures.
- Identify any gaps in the data; if gaps exist, describe how the data gaps are filled.

V. Deposition

- Specify the method to calculate deposition (if applicable).

VI. Receptors

Specify the type and location of receptors. Include all relevant information describing how the individual and population-related receptors will be evaluated.

- Identify and describe the location(s) of known or anticipated potential sensitive receptors, the point of maximum impact (PMI), and the maximum exposed individual residential (MEIR) and worker (MEIW) receptors. Identify any special considerations or grids that will be used to model these receptors. This information should correspond with information provided in Section III (e.g., fine receptor spacing of 20 meters at the fence line and centered on the maximum impacts; coarse receptor spacing of 100 meters out to 2,000 meters; extra coarse spacing of 1,000 meters out to 20,000 meters).
- Identify if spatial averaging will be used. Include necessary background information on each receptor including how the domain and spacing will be determined for each receptor or exposure pathway.
- Describe how the cancer burden or population impact estimates are calculated. Clarify the same information for the presentation of noncancer population impacts (e.g., centroids of the census tracts in the area within the zone of impact).
- Specify that actual UTM coordinates and the block/street locations (i.e., north side of 3,000 block of Smith Street), where possible, will be provided for specified receptor locations.
- Identify and support the use of any exposure adjustments (e.g., time a location, diurnal).

- Include the list of anticipated exposure pathways that will be included and indicate which substance will be evaluated in the multipathway assessment. Identify if sensitive receptors are present and which receptors will be evaluated in the HRA.

VII. Maps

Identify how the information will be graphically presented.

- Indicate which cancer risk isopleths will be plotted for the cancer zone of impact (e.g., 10^{-7}, 10^{-6} see Section 4.6.1).

- Indicate the hazard quotients or hazard indices to be plotted for the noncancer acute, 8 hour, and chronic zones of impact (e.g., 0.5, 1.0, etc.).

4.16 Health Risk Assessment Report

This section describes the information related to the air dispersion modeling process that needs to be reported in the risk assessment. This section is also presented in Chapter 9, Summary of the Requirements for a Modeling Protocol and a Health Risk Assessment Report, in Section 9.2. The District may have specific requirements regarding format and content (see Section 4.15). Sample calculations should be provided at each step to indicate how reported emissions data were used. Reviewing agencies must receive input, output, and supporting files of various model analyses on computer-readable media (e.g., CD).

4.16.1 *Information on the Facility and its Surroundings*

Report the following information regarding the facility and its surroundings:

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

4.16.2 *Source and Emission Inventory Information*[3]

4.16.2.1 Release Parameters

Report the following information for each release location in table format:

- Release location identification number
- Release name
- Release type (e.g., point, volume, area, line, pit, etc.)
- Source identification number(s) used by the facility that emit out of this release location
- Release location using UTM coordinates
- Release parameters by release type (e.g., shown for point source):
    - Stack height (m), stack diameter (building dimensions for downwash), exhaust gas exit velocity (m/s), exhaust gas volumetric flow rate (ACFM), exhaust gas exit temperature (K), etc.

4.16.2.2 Source Description and Operating Schedule

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

- Source identification number used by the facility
- Source name
- Number of operating hours per day and per year (e.g., 0800-1700, 2700 hr/yr)
- Number of operating days per week (e.g., Mon-Sat)
- Number of operating days or weeks per year (e.g., 52 wk/yr excluding major holidays)
- Release point identification number(s) for where source emissions are released
- Fraction of source emissions emitted at each release point by release point ID number

4.16.2.3 Emission Control Equipment and Efficiency

Report emission control equipment and efficiency by source and by substance

4.16.2.4 Emissions Data Grouped By Source

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

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[3] Health and Safety Code section 44346 authorizes facility operators to designate certain "Hot Spots" information as trade secret. Section 44361(a) requires districts to make health risk assessments available for public review upon request. Section 44346 specifies procedures to be followed upon receipt of a request for the release of trade secret information. See also the Inventory Guidelines Report regarding the designation of trade secret information in the Inventory Reports.

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

4.16.2.5 Emissions Data Grouped by Substance

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

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

4.16.2.6 Emission Estimation Methods

Report the methods used in obtaining the emissions data indicating whether emissions were measured or estimated. Clearly indicate any emission data that are not reflected in the previously submitted emission inventory report and submit a revised emission inventory report to the district. A reader should be able to reproduce the risk assessment without the need for clarification.

4.16.2.7 List of Substances

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

4.16.3 *Exposed Population and Receptor Location*

Report the following information regarding exposed population and receptor locations. See Chapter 9 and specific sections within this chapter for more detailed information.

- Description of zone of impact including map showing the location of the facility, boundaries of zone of impact, census tracts, emission sources, sites of maximum exposure, and the location of all appropriate receptors. This should be a true map (one that shows roads, structures, etc.), drawn to scale, and not just a schematic drawing. USGS 7.5 minute maps or GIS based maps are usually the most appropriate choices. (If significant development has occurred since the user's survey, this should be indicated.)

- Separate maps for the cancer risk zone of impact and the hazard index (noncancer) zone of impact(s). The cancer zone of impact should include isopleths down to at least the 1/1,000,000 risk level. Because some districts use a level below 1/1,000,000 to define the zone of impact, the District should be consulted. Three separate maps (to represent both chronic, 8-hour, and acute

HI) should be created to define the zone of impact for the hazard index from both inhalation and noninhalation pathways greater than or equal to 0.5. The point of maximum impact (PMI), maximum exposed individual at a residential receptor (MEIR), the maximum exposed individual worker (MEIW), and any other locations of interest for both cancer and noncancer risks should be located on the maps.

- Tables identifying population units and sensitive receptors (UTM coordinates, receptor IDs, and street addresses of specified receptors).
- Heights or elevations of the receptor points.
- For each receptor type (e.g., PMI, MEIR, MEIW, and any other location(s) of interest) that will utilize spatial averaging, the domain size and grid resolution must be clearly identified. If another domain or grid resolution other than 20 meters by 20 meters with 5-meter grid spacing will be used for a receptor, then care should be taken to determine the proper domain size and grid resolution that should be used. For a worker, the HRA shall support all assumptions used, including, but not limited to, documentation for all workers showing the area where each worker routinely performs their duties. The final domain size should not be greater than the smallest area of worker movement. Other considerations for determining domain size and grid spacing resolution may include an evaluation of the concentration gradients across the worker area. The grid spacing used within the domain should be sufficient in number and detail to obtain a representative concentration across the area of interest. When spatial averaging over the deposition area of a pasture, garden, or water body, care should be taken to determine the proper domain size to make sure it includes all reasonable areas of potential deposition. The size and shape of the pasture, garden, or water body of interest should be identified and used for the modeling domain. The grid spacing or resolution used within the domain should be sufficient in detail to obtain a representative deposition concentration across the area of interest. One way to determine the grid resolution is to include an evaluation of the concentration gradients across the deposition area. The HRA shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the pasture or water body, maps, representative coordinates, grid resolution, concentration gradients, etc.). The use or spatial averaging is subject to approval by the reviewing authority. This includes the size of the domain and grid resolution that is used for spatial averaging of a worksite or multipathway deposition area.

4.16.4 *Meteorological Data*

If meteorological data were not obtained directly from the District, then the report must clearly indicate the data source and time period used. Meteorological data not obtained from the District must be submitted in electronic form along with justification for their use including information regarding representativeness and quality assurance.

The risk assessment should indicate if the District required the use of a specified meteorological data set. All memos indicating the District's approval of meteorological data should be attached in an appendix.

4.16.5 *Model Selection and Modeling Rationale*

The report should include an explanation of the model chosen to perform the analysis and any other decisions made during the modeling process. The report should clearly indicate the name of the models that were used, the level of detail (screening or refined analysis) and the rationale behind the selection.

Also report the following information for each air dispersion model used:

- version number
- selected options and parameters in table form
- Identify the modeling domain(s) and the spacing of receptor grid(s). Grid spacing should be sufficient in number and detail to capture the concentration at all receptors of interest.

4.16.6 *Air Dispersion Modeling Results*

- Maximum hourly and annual average concentrations of chemicals at appropriate receptors such as the residential and worker MEI receptors
- Annual average and maximum one-hour (and 30-day average for lead only) concentrations of chemicals at appropriate receptors listed and referenced to computer printouts of model outputs
- Model printouts (numbered), annual concentrations, maximum hourly concentrations
- Disk with input/output files for air dispersion program (e.g., the AERMOD input file containing the regulatory options and emission parameters, receptor locations, meteorology, etc.)
- Include tables that summarize the annual average concentrations that are calculated for all the substances at each site. The use of tables that present the relative contribution of each emission point to the receptor concentration is recommended. (These tables should have clear reference to the computer model which generated the data. It should be made clear to any reader how data from the computer output 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.]

4.17 References

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5 - Exposure Assessment
Estimation of Concentration and Dose

5.1 Introduction

This chapter provides a summary of how toxicant ground level air concentrations estimated from air dispersion modeling or monitoring results are used to determine dose at receptors of interest. This chapter includes all the algorithms and data (e.g., point estimates, distributions, and transfer factors) that are needed to determine the substance-specific concentration in exposure media and the dose at a receptor of interest. The determination of exposure concentration and dose precedes the calculations of potential health impacts. See Chapter 8 and Appendix I for information on calculating potential health impacts.

At a minimum, three receptors are evaluated in Hot Spots health risk assessments (HRA) (see Section 4.7); these are:

- the Point of Maximum Impact (PMI),
- the Maximally Exposed Individual Resident (MEIR), and
- the Maximally Exposed Individual Worker (MEIW).

The PMI is defined as the receptor point(s) with the highest acute, 8-hour, chronic, or cancer health impact outside the facility boundary. The facility boundary is defined as the property line. Often the fence is on the property line. The MEIR is typically defined as the existing off-site residence(s) (i.e., house, apartment or other dwelling) with the highest acute, chronic, or cancer health impact. The MEIW is typically defined as the existing offsite workplace with the highest acute, 8-hour, chronic, or cancer health impact.

In addition, it may be necessary to determine risks at sensitive receptors (e.g., schools, day care centers, elder care centers, and hospitals). The District or reviewing authority should be consulted in order to determine the appropriate sensitive receptors for evaluation. Some situations may require that on-site receptor (worker or residential) locations be evaluated. Some examples where the health impacts of on-site receptors may be appropriate could be military base housing, prisons, universities, or locations where the public may have regular access for the appropriate exposure period (e.g., a lunch time café or museum for acute exposures). The risk assessor should contact the Air Pollution Control or Air Quality Management District (the District) for guidance about any on-site exposure situations at the emitting facility. These on-site locations should be included in the health risk assessment (HRA). If the facility emits multiple substances from two or more stacks, the acute, 8-hour, chronic, and cancer health impacts at the PMI may be located at different physical locations. The MEIR or MEIW cancer, acute, 8-hour, and chronic receptors may also be at different locations.

The process for determining dose at the receptor location, and ultimately potential health impacts, will likely include air dispersion modeling, and, with less frequency, air monitoring data. Air dispersion modeling combines the facility emissions and release parameters and uses default or site-specific meteorological conditions to estimate downwind, ground-level concentrations at various (user-defined) receptor locations. Air dispersion modeling is described in Chapter 4 and is presented in detail in the *Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis (OEHHA, 2012a*).

In summary, the process of using air dispersion modeling results as the basis of an HRA follows these four steps:

- Air dispersion modeling is used to estimate annual average and maximum one-hour ground level concentrations (GLC). The air dispersion modeling results are expressed as an air concentration or in terms of (Chi over Q) for each receptor point. (Chi over Q) is the modeled downwind air concentration based on an emission rate of one gram per second. (Chi over Q) is expressed in units of micrograms per cubic meter per gram per second, or ($\mu g/m^3$)/(g/s). (Chi over Q) is sometimes written as (χ/Q) and is sometimes referred to as the dilution factor.

- When multiple substances are evaluated, the χ/Q is normally utilized since it is based on an emission rate of one gram per second. The χ/Q at the receptor point of interest is multiplied by the substance-specific emission rate (in g/s) to yield the substance-specific ground-level concentration (GLC) in units of $\mu g/m^3$. The following equations illustrate this point.

$$\mathbf{GLC} = \left(\chi/\mathbf{Q}\right) \mathbf{x} \left(\mathbf{Q}_{\mathbf{substance}}\right)$$

$$\chi/\mathbf{Q} = \left(\mathbf{Chi\ \ over\ \ Q}\right) \mathbf{in} \left(\frac{\mu\mathbf{g}/\mathbf{m}^3}{\mathbf{g}/\mathbf{s}}\right), \mathbf{from\ model\ results\ with\ unit\ emission\ rate}$$

$$\mathbf{Q}_{\mathbf{substance}} = \mathbf{substance\ specific\ emission\ rate} \left(\mathbf{g}/\mathbf{s}\right)$$

- The applicable exposure pathways (e.g., inhalation, soil contact, fish consumption) are identified for the emitted substances, and the receptor locations are identified. This determines which exposure algorithms in this chapter are ultimately used to estimate dose. After the exposure pathways are identified, the fate and transport algorithms described in this chapter are used to estimate concentrations in the applicable exposure media (e.g., soil or water) and the exposure algorithms are used to determine the substance-specific dose.

- The dose is used with cancer and noncancer health values to calculate the potential health impacts for the receptor (Chapter 8). An example calculation using the high-end point-estimates for the inhalation (breathing) exposure

pathway can be found in Appendix I. Appendix I and Chapters 5 (this Section) and 8 also contain information on how the annual average and maximum one-hour ground level concentrations are used for chronic, 8-hour, and acute health risk calculations.

The algorithms in this chapter are also used to calculate media concentrations and dose in the rare instance, for the Hot Spots program, when monitoring equipment was used rather than air dispersion modeling to obtain a receptor's substance-specific GLC. One situation that is specific to monitored data is the treatment of results below the sampling method level of detection (LOD). In short, it is standard risk assessment practice when monitoring results are reported both above and below the LOD to use one-half of the LOD for those sample concentrations reported below the LOD. If all testing or monitoring results fall below the LOD, then assessors should contact the District for appropriate procedures. For more information about reporting emissions under the Hot Spots Program, see the ARB's *Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5),* and the *Emission Inventory Criteria and Guidelines Report* (EICG Report), which is incorporated by reference therein (ARB, 2007*)*.

The recommended model for calculating and presenting HRA results for the Hot Spots Program is the HARP software, available from the Air Resources Board (ARB). More information on HARP and directions for downloading the software can be found on the ARB's web site at http://www.arb.ca.gov/toxics/harp/downloads.htm.

5.2 Criteria for Exposure Pathway Evaluation

In order to determine total dose to the receptor the applicable pathways of exposure need to be identified. The inhalation pathway must be evaluated for all Hot Spots substances emitted by the facility. A small subset of Hot Spots substances is subject to deposition onto soil, plants, and water bodies. These substances need to be evaluated by the appropriate noninhalation pathways, as well as by the inhalation pathway, and the results must be presented in all HRAs. These substances include semi-volatile organic chemicals and heavy metals. Such substances are referred to as multipathway substances. Two steps are necessary to determine if a substance should be evaluated for multipathway impacts:

1. Determine whether the substance or its group (e.g., dioxins, PAHs) is listed in Table 5.1.
2. Determine if the substance has an oral reference exposure level (REL) listed in Table 6.4, or if it has an oral cancer slope factor listed in Table 7.1. Two other references for checking the presence of oral health factors are OEHHA's website (OEHHA, 2012b) and the *Consolidated Table of OEHHA/ARB Approved Risk Assessment Health Values* on the Air Resources Board website (ARB, 2012). Oral or noninhalation exposure pathways include the ingestion of soil, angler-caught fish, drinking water from surface water sources, mother's milk, homegrown produce, beef, pork, chicken, eggs and cow's milk. The dermal pathway is also evaluated via contact with contaminated soil.

For all multipathway substances, the minimum exposure pathways that must be evaluated at every residential site (in addition to inhalation) are soil ingestion and dermal exposure. If dioxins, furans, PCBs, PAHs or lead are emitted, then the breast-milk consumption pathway also becomes mandatory. The other exposure pathways (e.g., the ingestion of homegrown produce or angler-caught fish) are evaluated on a site-by-site basis. If the resident can be exposed through an impacted exposure pathway, then it must be included in the HRA. However, if there are no vegetable gardens or fruit trees within the zone of impact for a facility, for example, then the produce pathways need not be evaluated. Note that on-site residential receptors are potentially subject to inhalation and noninhalation exposure pathways. Table 8.2 identifies the residential and worker receptor exposure pathways that are mandatory and those that are dependent on the site-specific decisions. While residents can be exposed though several exposure pathways, worker receptors are only evaluated for inhalation, soil ingestion, and dermal exposure using point estimates.

Table 5.1 shows the multipathway substances that, based on available scientific data, can be considered for each noninhalation exposure pathway. The exposure pathways that are evaluated for a substance depend on two factors: 1) whether the substance is considered a multipathway substance for the Hot Spots Program (Table 5.1), and 2) what the site-specific conditions are. A multipathway substance may be excluded from a particular exposure pathway because its physical-chemical properties can preclude significant exposure via the pathway. For example, some water-soluble substances do not appreciably bioaccumulate in fish; therefore, the fish pathway is not appropriate. In addition, if a particular exposure pathway is not impacted by the facility or is not present at the receptor site, then the pathway is not evaluated. For example, if a fishable water body is not impacted by the facility, or the water source is impacted but no receptor uses it for fishing, then the angler-caught fish pathway is not evaluated.

Table 5.1 Specific Pathways to be Analyzed for each Multipathway Substance

Substance	Soil Ingestion	Dermal	Meat, Milk & Egg Ingestion	Fish Ingestion	Exposed Vegetable Ingestion	Leafy Vegetable Ingestion	Protected Vegetable Ingestion	Root Vegetable Ingestion	Water Ingestion	Breast Milk Ingestion
Inorganic chemicals										
Arsenic & compounds	X	X	X	X	X	X	*X*	X	X	
Beryllium & compounds	X	X	X	X	X	X	*X*	X	X	
Cadmium & compounds	X	X	X	X	X	X	*X*	X	X	
Chromium VI & compounds	X	X	X[a]	X	X	X	*X*	X	X	
Fluorides (soluble compounds)	X	X	X		X	X	*X*	X	X	
Lead & compounds	X	X	X	X	X	X	*X*	X	X	X
Mercury & compounds	X	X	X	X	X	X	*X*	X	X	
Nickel & compounds	X	X	X	X	X	X	*X*	X	X	
Selenium & compounds	X	X	X	X	X	X	*X*	X	X	
Organic chemicals										
Creosotes	X	X	X	X	X	X			X	X
Diethylhexylphthalate	X	X	X	X	X	X			X	
Hexachlorobenzene	X	X	X	X	X	X			X	
Hexachlorocyclohexanes	X	X	X	X	X	X			X	
4,4'-Methylene dianiline	X	X			X	X			X	
Pentachlorophenol[b]										
PCBs	X	X	X	X	X	X			X	X
Polychlorinated dibenzo-p-dioxins and dibenzofurans	X	X	X	X	X	X			X	X
PAHs	X	X	X	X	X	X			X	X

[a] Cow's milk only; no multipathway analysis for meat and egg ingestion
[b] To be evaluated by pathway

5.3 Estimation of Concentrations in Air, Soil, and Water

Once emissions exit the source, the substances emitted will be dispersed in the air. The substances in the exhaust gas with high vapor pressures will remain largely in the vapor phase, and substances with lower vapor pressures will tend to adsorb to fly ash or other particulate matter. The emission plume may contain both vapor phase substances and particulates. A semivolatile organic toxicant can partition into both vapor and particulate phases. Particulates will deposit on vegetation, on soil, and in water at a rate that is dependent on the particle size. Use the 0.02 m/s deposition rate for emission sources that have verifiable particulate matter control devices or for emission sources that may be uncontrolled but only emit particulate matter that is less than 2.5 microns (e.g., internal combustion engines powered by compressed natural gas). The following algorithms are used to estimate concentrations in environmental media including air, soil, water, vegetation, and animal products.

5.3.1 *Air*

The ground level concentration (GLC, or C_{air} as shown in EQ 5.3.1) of a substance in air is a function of the facility emission rate and the dilution factor (χ/Q) at the points under evaluation.

A. Equation 5.3.1: $C_{air} = Q_{substance} \times \chi/Q$

1. C_{air} = Ground level concentration ($\mu g/m^3$)
2. $Q_{substance}$ = Substance emission rate (g/sec)
3. χ/Q = Dilution factor provided by dispersion modeling ($\mu g/m^3/g/sec$)

a. Recommended values for EQ 5.3.1:

1. $Q_{substance}$ = Facility-specific, substance emission rate
2. χ/Q = For point of interest, site specific, from dispersion modeling

b. Assumptions for EQ 5.3.1:

1. No plume depletion
2. Emission rate is constant, i.e., assumes steady state

5.3.2 *Soil*

The average concentration of the substance in soil (Cs) is a function of the deposition, accumulation period, chemical specific soil half-life, mixing depth, and soil bulk density. For simplicity and health protection, the Tier 1 default assumes 70-year soil deposition for the accumulation period at end of 70-year facility lifetime. The risk assessor may also choose a supplemental Tier 2 approach, subject to District approval or reviewing authority approval, in which the assessor applies a soil accumulation period based on the facilities' start date of operation (e.g., historical date when emissions began), or the current exposure conditions, and the expected duration of operation.

A. Equation 5.3.2 A: $C_s = Dep \times X / (K_s \times SD \times BD \times T_t)$

1. C_s = Average soil concentration over the evaluation period ($\mu g/kg$)
2. Dep = Deposition on the affected soil area per day ($\mu g/m^2$-d)
3. X = Integral function for soil accumulation (d), see **EQ 5.3.2 C** below
4. K_s = Soil elimination constant (d^{-1})
5. SD = Soil mixing depth (m)
6. BD = Soil bulk density (kg/m^3)
7. T_t = Soil exposure duration or soil accumulation period (d)

a: Recommended default values for EQ 5.3.2 A:

1. Dep = Calculated in EQ 5.3.2 B

2. X = Calculated in EQ 5.3.2 C
3. K_s = Calculated in EQ 5.3.2 D
4. SD = 0.01 (m) for playground setting (soil ingestion and dermal pathways) and 0.15 (m) for agricultural setting (produce and meat pathways)
5. BD = 1,333 (kg/m^3)
6. T_t = 25,550 (d) = 70 years

b: Assumptions for EQ 5.3.2 A:

1. Substances are uniformly mixed in soil.
2. Substances are not leached or washed away, except where evidence exists to the contrary.
3. It is assumed that toxicants accumulate in the soil for 70 years from deposition over the 70 year lifespan of the facility. Use 70-year soil accumulation (T_t) for Tier 1 estimation of 9-, 30- and 70-year residential exposure, and 25-year off-site worker exposure.
4. For a receptor ingesting mother's milk, the mother is exposed from birth to 25 years of age; the infant is then born and receives mother's milk for one year. Default assumes 70-year soil accumulation for mother's milk pathway. See Table 5.1 for information on which substances or groups of substances must be evaluated by the mother's milk pathway.

B. Equation 5.3.2 B: $\mathbf{Dep = C_{air} \times Dep\text{-}rate \times 86{,}400}$

1. C_{air} = Ground level concentration ($\mu g/m^3$)
2. Dep-rate = Vertical rate of deposition (m/sec)
3. 86,400 = Seconds per day conversion factor (sec/d)

a: Recommended default values for EQ 5.3.2 B:

1. C_{air} = Calculated above in EQ 5.3.1 A
2. Dep-rate = Use 0.02 meters/second for controlled sources, or 0.05 meters/second for uncontrolled sources.

b: Assumptions for EQ 5.3.2 B:

1. Deposition rate remains constant. A deposition rate must be used when determining potential noninhalation health impacts. In the absence of facility specific information on the size of the emitted particles, the default values for deposition rate should be used. Currently, the default value of 0.02 meters per second is used for emission sources that have verifiable particulate matter control devices or for emission sources that may be uncontrolled but only emit particulate matter that is less than 2.5 microns (e.g., internal combustion engines powered by compressed natural gas). The 0.05 meters per second default value is used for risk assessment if

the emissions are uncontrolled. If other deposition rate factors are used, sufficient support documentation must be included with the HRA.

C. Equation 5.3.2 C: $X = [\{e^{-Ks * Tf} - e^{-Ks * To}\} / K_s] + T_t$

1. e = 2.718
2. K_s = Soil elimination constant
3. T_f = End of soil accumulation evaluation period (d)
4. T_o = Beginning of soil accumulation evaluation period (d)
5. T_t = Total days of soil exposure (soil accumulation period) T_f-T_o (d)

a: Recommended default values for EQ 5.3.2 C:

1: K_s = Calculated in EQ 5.3.2 D
2: T_f = 25,550 (d) = 70 years. Total soil exposure time at end of facility operation 3: T_o = 0 (d) The initial time (start period) of soil exposure to all receptors that are impacted by the soil pathway.

Note: Under a Tier 2 scenario, the risk assessor may also adjust T_f and T_t, subject to District approval, to replicate current soil accumulation and expected accumulation at the end of facility operation.

D. Equation 5.3.2 D: $K_s = 0.693 / t_{1/2}$

1. 0.693 = Natural log of 2
2. $t_{1/2}$ = Chemical specific soil half-life (d)

a: Recommended default values for EQ 5.3.2 D:

1. $t_{1/2}$ = Chemical-specific. See Table 5.2

5.3.3 *Water*

The water pathway is evaluated if a standing water body (e.g., pond or lake) is impacted by facility emissions and is used as a source for drinking water by food-producing animals or humans, or is a source of angler-caught fish. The average concentration of the substance in water (C_w) is a function of direct deposition and material carried in by surface run-off. However, only the contribution from direct deposition will be considered at this time.

A. Equation 5.3.3 A: $C_w = C_{depw}$

1. C_w = Average concentration in water (μg/kg)
2. C_{depw} = Contribution due to direct deposition (μg/kg)

B. Equation 5.3.3 B: $C_{depw} = Dep \times SA \times 365 / (WV \times VC)$

1. Dep = Deposition on water body per day ($\mu g/m^2/d$)
2. SA = Water surface area (m^2)
3. 365 = Days per year (d/yr)
4. WV = Water volume (kg)
5. VC = Number of volume changes per year

a: Recommended default values for EQ 5.3.3 B:

1. Dep = Calculated above in EQ 5.3.2 B
2. SA = Site specific water surface area (m^2)
3. WV = Site specific water volume in (kg) (1L = 1 kg)
4. VC = Site specific number of volume changes per year (SA, WV, and VC values can be obtained from the appropriate Department of Water Resources (DWR) Regional office)

b: Assumptions for EQ 5.3.3 B:

1. All material deposited into the water remains suspended or dissolved in the water column and is available for bioaccumulation in fish.

5.3.4 *Estimation of Concentrations in Vegetation, Animal Products, and Mother's Milk*

Estimates of the concentration of the substance in vegetation, animal products and mother's milk require the use of the results of the air, water, and soil environmental fate evaluation. Plants, animals and nursing mothers will be exposed to the substances at the concentrations previously calculated in Section 5.31 to 5.33 above.

5.3.4.1 Vegetation

The average concentration of a substance in and on vegetation (C_v) is a function of direct deposition of the substance onto the vegetation and of root translocation or uptake from soil contaminated by the substance. We currently recommend root translocation only for the inorganic compounds.

A. Equation 5.3.4.1 A: $C_v = C_{depv} + C_{trans}$

1. C_v = Average concentration in and on specific types of vegetation ($\mu g/kg$)
2. C_{depv} = Concentration due to direct deposition ($\mu g/kg$)
3. C_{trans} = Concentration in vegetation due to root translocation or uptake ($\mu g/kg$) – see EQ 5.3.4.1 C below

B. Equation 5.3.4.1 B: $C_{depv} = [Dep \times IF / (k \times Y)] \times (1 - e^{-kT})$

1. Dep = Deposition on affected vegetation per day ($\mu g/m^2/d$)
2. IF = Interception fraction
3. k = Weathering constant (d^{-1})
4. Y = Yield (kg/m^2)
5. e = Base of natural logarithm (2.718)
6. T = Growth period (d)

a: Recommended default values for EQ 5.3.4.1 B:

1. Dep = Calculated above in EQ 5.3.2 B
2. IF = Crop specific:
   - a: Root crops = 0.0
   - b: Leafy crops = 0.2
   - c: Protected crops = 0.0
   - d: Exposed crops = 0.1
   - e: Pasture = 0.7
3. k = 0.1 (d^{-1})
4. Y = 2 (kg/m^2) for root, leafy, protected, exposed and pasture [CA Department of Food and Agriculture dot maps]
5. T = 45 (d) for leafy crops
   T = 90 (d) for exposed crops

b: Crop-type definitions for EQ 5.3.4.1 B:

1. **Leafy** crop category consists of broad-leafed vegetables in which the leaf is the edible part. Examples include spinach, lettuce, cabbage, and kale.
2. **Root** crop category includes vegetables in which the edible portion is underground. Examples are potato, radish, and carrot.
3. **Exposed** produce category consists of crops with a small surface area subject to air deposition. Examples include strawberries, tomato, cucumber, zucchini, green bean and bell pepper.
4. **Protected** produce category consists of crops in which the edible part is not exposed to air deposition (e.g., the exposed skin of the crop is removed and not eaten). Examples are corn, pea, pumpkin and oranges.

Tables H-9 through H-15 in Appendix H provides more examples of various leafy, root, exposed and protected crop types.

c: Assumptions for EQ 5.3.4.1 B:

1. No deposition on root or protected crops
2. No uptake and translocation of deposited chemicals onto crops

C. Equation 5.3.4.1 C: (for inorganic compounds)

$$C_{trans} = C_s \times UF_2$$

1. C_s = Average soil concentration (μg/kg)
2. UF_2 = Uptake factor based on soil concentration

a: Recommended default values for EQ 5.3.4.1 C:

1. C_s = Calculated above in EQ 5.3.2 A
2. UF_2 = See Table 5.2

D. Equation 5.3.4.1 D: (for organic compounds)

$$UF_2 = [(0.03 \times K_{ow}^{0.77}) + 0.82] / [(K_{oc})(F_{oc})]$$

1. 0.03 = Empirical constant
2. K_{ow} = Octanol:water partition factor
3. 0.77 = Empirical constant
4. 0.82 = Empirical constant
5. K_{oc} = Organic carbon partition coefficient
6. F_{oc} = Fraction organic carbon in soil

a: Recommended default values for EQ 5.3.4.1 D:

1. K_{ow} = Chemical specific, see Table 5.2
2. K_{oc} = Chemical specific, see Table 5.2
3. F_{oc} = 0.1

b: Assumptions for EQ 5.3.4.1 D:

1. OEHHA currently has no recommended root uptake factors for organic compounds listed in Table 5.2. Evidence suggests this route is insignificant compared to airborne deposition. Nevertheless, if it becomes necessary in specific cases to assess root uptake for an organic compound, Equation 5.3.4.1 D would be the algorithm OEHHA recommends using to assess root uptake.

5.3.4.2 Animal Products

The average concentration of the substance in animal products (C_{fa}) depends on which routes of exposure exist for the animals. Animal exposure routes include inhalation, soil ingestion, ingestion of contaminated feed and pasture, and ingestion of contaminated water.

A. Equation 5.3.4.2:

$$C_{fa} = (\text{Inhalation} + \text{Water ingestion} + \text{Feed ingestion} + \text{Pasture/Grazing ingestion} + \text{Soil ingestion}) * Tco$$

1. C_{fa} = Average concentration in farm animals and their products (μg/kg)
2. Inhalation, water ingestion, etc. = Dose through inhalation, water ingestion, etc. (μg/d)
3. Tco = Chemical-specific transfer coefficient of contaminant from diet to animal product (d/kg)

a: Recommended default values for EQ 5.3.4.2:

1. Tco = See Tables 5.3a and 5.3b

b: Assumptions for EQ 5.3.4.2:

1. The Tco for a given chemical is the same for all exposure routes

5.3.4.2.1 Inhalation

A. Equation 5.3.4.2.1:

$$\text{Inhalation} = BR_a \times C_{air}$$

1. Inhalation = Dose through inhalation (μg/d)
2. BR_a = Breathing rate for animal (m^3/d)
3. C_{air} = Ground-level concentration (μg/m^3)

a: Recommended default values for EQ 5.3.4.2.1:

1. BR_a = See Table 5.4
2. C_{air} = Calculated above in EQ 5.3.1 A

b: Assumptions for EQ 5.3.4.2.1:

1. All material inhaled is 100% absorbed

5.3.4.2.2 Water ingestion

The water ingestion pathway is applied if there are surface water sources of drinking water, such as springs, ponds or lakes, which are exposed to airborne deposition of facility emissions. Due to the site-specific nature for this exposure pathway, OEHHA recommends that the risk assessor conduct a survey at the site to estimate the fraction of contaminated drinking water ingested by the animals, if such sources exist.

A. Equation 5.3.4.2.2: **Water ingestion = $WI_a \times FSW \times C_w$**

1. Water ingestion = Dose through water ingestion (μg/d)
2. WI_a = Water ingestion for animal (kg/d)
3. FSW = Fraction of water ingested from a contaminated body of water (site-specific)
4. C_w = Average concentration in water (μg/kg)
   For water 1 kg = 1 L

a: Recommended default values for EQ 5.3.4.2.2:

1. WI_a = See Table 5.4
2. FSW = Site specific fraction, need to survey water ingestion practices in affected area
3. C_w = Calculated above in EQ 5.3.3 A

5.3.4.2.3 Feed Ingestion

The fraction of feed intake by cattle, pigs and poultry that is contaminated by facility emissions can vary considerably depending on the manner in which the animals are raised. Due to the site-specific nature for this exposure pathway, OEHHA recommends that the risk assessor conduct a survey at the site to estimate the fraction of contaminated feed eaten by the animals. For a Tier 1 assessment, default values are provided by OEHHA (see Table 5.4 and Table 5.4 footnotes) for estimation of exposure to the animals.

Agricultural mixing depth should be used for calculating soil concentration for feed and pasture contamination.

5.3.4.2.3.1 Feed Ingestion

A. Equation 5.3.4.2.3.1: Feed ingestion = $(1.0 - FG) \times FI \times L \times C_v$

1. Feed ingestion = Dose through the ingestion of feed (μg/d) that is harvested after it is impacted by source emissions
2. FG = Fraction of diet provided by grazing (site-specific)
3. FI = Feed ingestion rate (kg/d)
4. L = Fraction of locally grown (source impacted) feed that is not pasture (site-specific)
5. C_v = Concentration in feed (μg/kg)

a: Recommended default values EQ 5.3.4.2.3.1:

1. FG = Default values in Table 5.4 footnote b, although a site-specific survey for the fraction of diet provided by grazing is recommended
2. FI = See **Error! Reference source not found.** 5.4
3. L = Default values in Table 5.4 footnote b, although a site-specific survey for fraction of locally grown (source impacted) feed that is not pasture is recommended
4. C_v = As calculated above in EQ 5.3.4.1 A

b: Assumptions for EQ 5.3.4.2.3.1:

1. Feed (FI) transported from an off-site location (i.e., not grown locally) is not contaminated by facility emissions.

5.3.4.2.3.2 Pasture/Grazing ingestion

A. Equation 5.3.4.2.3.2: $\text{Pasture/Grazing ingestion} = FG \times C_v \times FI$

1. Pasture/Grazing ingestion = Dose through pasture/grazing (μg/d)
2. FG = Fraction of diet provided by grazing (site-specific)
3. C_v = Concentration in pasture/grazing material (μg/kg)
4. FI = Feed ingestion rate (kg/d)

a: Recommended default values EQ 5.3.4.2.3.2:

1. FG = Default values in Table 5.4 for fraction of diet provided by grazing, although a site-specific survey is recommended
2. C_v = As calculated above in EQ 5.3.4.1 A
3. FI = See Table 5.4

5.3.4.2.4 Soil ingestion

The feeds provided to dairy and beef cattle may contain small quantities of soil. A larger fraction of soil by weight of food is taken up during grazing. Rooting behavior by pigs with access to soil will result in soil ingestion. Likewise, poultry with free access to soil or pasture will also ingest soil. Defaults for soil ingestion are shown in Table 5.4.

A. Equation 5.3.4.2.4 A: $\textbf{Soil ingestion} = SI_a \times C_s$

1. Soil ingestion = Dose through soil ingestion (μg/d)
2. SI_a = Soil ingestion rate for animal (kg/d)
3. C_s = Average soil concentration (μg/kg)

a: Recommended default values for EQ 5.3.4.2.4 A:

1. SI_a = Calculated below
2. C_s = Calculated above in EQ 5.3.2 A

B. Equation 5.3.4.2.4 B: $SI_a = [(1 - FG) \times FS_f \times FI] + [FG \times FS_p \times FI]$

1. FG = Fraction of diet provided by grazing
2. FS_f = Soil ingested as a fraction of feed ingested
3. FI = Feed ingestion rate (kg/d)
4. FS_p = Soil ingested as a fraction of pasture ingested

a: Recommended default values for EQ 5.3.4.2.4 B:

1. FG = Site specific fraction of diet provided by grazing
2. FS_f = See Table 5.4
3. FI = See Table 5.4
4. FS_p = See Table 5.4

b: Assumptions for EQ 5.3.4.2.4 B:

1. The transfer coefficient is the same for all exposure routes.
2. Soil ingested in feed (FS_f) transported from an off-site location (i.e., not grown locally) is assumed not to be contaminated by facility emissions.

5.3.4.3 Bioaccumulation in Angler-Caught Fish

The average concentration in fish (C_f) is based on the concentration in water and a chemical-specific bioaccumulation factor.

A. Equation 5.3.4.3: $Ct = Cw \times BAF$

1. Ct = Concentration in wet weight tissue (muscle) of fish (µg/kg)
2. Cw = Concentration in water (µg/kg)
3. BAF = Fish bioaccumulation factor (unitless)

a: Recommended default values for Equation 5.3.4.3:

1. Cw = As calculated above in Equation 5.3.3 A
2. BAF = Chemical-specific; see Table 5.2

b: Assumptions for Equation 5.3.4.3:

1. For conversion of a chemical concentration in a volume of water shown as µg/L, 1 L water = 1 kg water; thus, for concentration of chemical in water, µg/L = µg/kg.
2. For organic chemicals, BAFs lipid-normalized to adult rainbow trout with 4% lipid content in muscle tissue
3. For organic chemicals, BAFs based on the freely dissolved fraction in water under conditions of average particulate organic carbon and dissolved organic carbon in U.S. lakes and other water bodies
4. For inorganic compounds, BAFs based on wet weight muscle tissue concentration and on the total water concentration of the inorganic compound in water.
5. Contaminant concentrations are uniform in water based on dispersion

5.3.4.4 Bioaccumulation in Mother's Milk

The average concentration of a chemical in mother's milk (C_f) is a function of the mother's exposure through all exposure routes (i.e., inhalation, ingestion via food, drinking water, and soil, and dermal absorption via skin contact with soil contaminated with the chemical), the contaminant half-life in the mother's body, and transfer of absorbed chemical to mother's milk. The contaminant half-life in the body and transfer to mother's milk is incorporated in biotransfer coefficients (Tco) in Equation 5.3.4.8. See the TSD (OEHHA, 2012a), Appendix J for details on development of biotransfer factors. The substances assessed by the mother's milk pathway are shown in Table 5.1.

A. Equation 5.3.4.4: $$C_m = [(D_{inh} \times Tco_{m_inh}) + (D_{ing} \times Tco_{m_ing})] \times BW$$

1. C_m = Concentration in mother's milk (mg/kg-milk)
2. D_{inh} = The sum of DOSEair + DOSEdermal through inhalation and dermal absorption (mg/kg-BW-day)
3. D_{ing} = The sum of DOSEfood + DOSEsoil + DOSEwater through ingestion (mg/kg-BW-day)
4. $Tco_{m\ inh}$ = Biotransfer coefficient from inhalation and dermal absorption to mother's milk (d/kg-milk)
5. $Tco_{m\ ing}$ = Biotransfer coefficient from ingestion to mother's milk (d/kg-milk)
6. BW = Body weight of mother (Kg)

a: Recommended cancer risk default values for EQ 5.3.4.4:

1. D_{ing} = As calculated through ingestion of soil in EQ 5.4.3.1.1 + home-grown produce in EQ 5.4.3.2.1 + home-raised animal products in EQ 5.4.3.2.2 + drinking water in EQ 5.4.3.3.1 + angler-caught fish in EQ 5.4.3.4.1
2. D_{inh} = As calculated through inhalation in EQ 5.4.1.1 + dermal exposure in EQ 5.4.2.1
3. $Tco_{m\ inh}$ = See Table 5.5
4. $Tco_{m\ ing}$ = See Table 5.5

b: Recommended noncancer risk default values for EQ 5.3.4.4:

1. D_{ing} = As calculated through ingestion of soil in EQ 5.4.3.1.2 + home-grown produce and home-raised animal products in EQ 5.4.3.2.3 + drinking water in EQ 5.4.3.3.2 + angler-caught fish in EQ 5.4.3.4.2
2. D_{inh} = As calculated through inhalation in EQ 5.4.1.1 + dermal exposure in EQ 5.4.2.2
3. $Tco_{m\ inh}$ = See Table 5.5
4. $Tco_{m\ ing}$ = See Table 5.5

c: Assumptions for EQ 5.3.4.4:

1. Default age of mother at birth is 25 years of age, then nurses the infant for 1 year; Use 16<30 year old high-end (95th percentile) daily breathing rate and intake rates for D_{ing} and D_{inh} for estimating dose to mother.
2. For inhalation dose to mother's milk, it is recommended that the EF variate in EQ 5.4.1.1 is left out for calculation of inhalation dose in the mother's milk pathway.
3. Biotransfer coefficient, $Tco_{m\ inh}$, the same for both inhalation and dermal pathways based on lack of first-pass metabolism through the liver for both of these pathways.
4. Biotransfer coefficient, $Tco_{m\ ing}$, the same for all ingestion pathways based on first-pass metabolism through the liver.
5. For chemicals in Table 5.5 lacking either an oral or inhalation Tco, use the oral Tco for the absent inhalation Tco (i.e., for PCDDs and PCDFs and dioxin-like PCBs), or the inhalation Tco for the absent oral Tco (i.e., for lead) in Equation 5.3.4.8.
6. The concentration in the mother's milk is determined using the derived approach to risk assessment. This method allows use of the high-end dose point estimate for driving exposure pathways and the average dose point estimates for other exposure pathways. See Sections 8.2.6 (cancer) and 8.3.3 (noncancer) for the description of the methodology on how to implement the derived methodology.

Table 5.2 Substance Specific Default Values for Multipathway Substances[1]

				Root Uptake Factors (for inorganic compounds only)					
Multipathway Substance	Log K_{oc}[2]	Log K_{ow}[2]	Fish BAF	Root	Leafy	Exposed	Protected	GRAF[3]	Soil HalfLife (days)
Inorganic Compounds									
Arsenic & Inorganic Compounds	NA[6]	NA	$2 \times 10^{+1}$	8×10^{-3}	1×10^{-2}	2×10^{-2}	7×10^{-2}	1.0	$1.0 \times 10^{+8}$
Beryllium & Compounds	NA	NA	$4 \times 10^{+1}$	5×10^{-3}	2×10^{-4}	8×10^{-3}	3×10^{-4}	1.0	$1.0 \times 10^{+8}$
Cadmium & Compounds	NA	NA	$4 \times 10^{+1}$	8×10^{-2}	1×10^{-1}	2×10^{-2}	1×10^{-2}	1.0	$1.0 \times 10^{+8}$
Chromium VI & Compounds	NA	NA	$2 \times 10^{+1}$	$3 \times 10^{+0}$	3×10^{-1}	2×10^{-2}	7×10^{-2}	1.0	$1.0 \times 10^{+8}$
Fluorides (soluble compounds)			NA	9×10^{-3}	4×10^{-2}	4×10^{-3}	4×10^{-3}	1.0	$1.0 \times 10^{+8}$
Lead & Compounds	NA	NA	$2 \times 10^{+1}$	4×10^{-3}	8×10^{-3}	7×10^{-3}	3×10^{-3}	1.0	$1.0 \times 10^{+8}$
Mercury & Inorganic Compounds	NA	NA	$8 \times 10^{+1}$	2×10^{-2}	2×10^{-2}	9×10^{-3}	1×10^{-2}	1.0	$1.0 \times 10^{+8}$
Nickel and compounds	NA	NA	$2 \times 10^{+1}$	6×10^{-3}	1×10^{-2}	3×10^{-3}	3×10^{-2}	1.0	$1.0 \times 10^{+8}$
Selenium & compounds			$1 \times 10^{+3}$	7×10^{-2}	6×10^{-2}	4×10^{-2}	3×10^{-1}	1.0	$1.0 \times 10^{+8}$

Table 5.2 Substance Specific Default Values for Multipathway Substances[1]

				Root Uptake Factors (for inorganic compounds only)					
Multipathway Substance	**Log K_{oc}[2]**	**Log K_{ow}[2]**	**Fish BAF**	**Root**	**Leafy**	**Exposed**	**Protected**	**GRAF[3]**	**Soil HalfLife (days)**
Organic Compounds									
Creosotes	NA	NA	$8 \times 10^{+2}$	NA	NA	NA	NA	1.0	$4.3 \times 10^{+2}$
Diethylhexyl-phthalate	4.72	5.11	$4 \times 10^{+1}$	NA	NA	NA	NA	1.0	$1.5 \times 10^{+1}$
Dioxins and Furans	NA	NA	$3 \times 10^{+5}$	NA	NA	NA	NA	0.43	$7.0 \times 10^{+3}$
Hexachlorobenzene	NA	NA	$8 \times 10^{+4}$				NA	1.0	$1.0 \times 10^{+8}$
Hexachlorocyclo-hexanes	NA	NA	$3 \times 10^{+3}$	NA	NA	NA	NA	1.0	$9.4 \times 10^{+1}$
4,4'-Methylene dianiline	2.24	1.59	NA	NA	NA	NA	NA	1.0	$4.6 \times 10^{+2}$
Pentachlorophenol[4]									
Polycylcic Aromatic Hydrocarbons (PAHs)	NA	NA	$8 \times 10^{+2}$	NA	NA	NA	NA	1.0	$4.3 \times 10^{+2}$
Polychlorinated Biphenyls	NA	NA	$2 \times 10^{+6}$	NA	NA	NA	NA	1.0	$3.2 \times 10^{+3}$

(1)Values based on South Coast AQMD Multi-Pathway Assessment Input Parameters Guidance Document as adapted and modified by OEHHA.

(2)See Tables 5.17 and 5.18 for derivation and references for Kow and Koc values.

(3) GRAF (Gastrointestinal Relative Absorption Factor). The guidelines allow for adjusting for bioavailability where the evidence warrants. For example, there are good data which indicate that dioxin is not as available to an organism when bound to soil or fly ash matrices relative to when it is in solution or in food. Therefore, a bioavailability factor is incorporated into the model to account for this difference. When information becomes available for other chemicals of concern, this type of bioavailability will be incorporated into the model.

(4) To be evaluated for specific default values

NA - Data Not Available or Not Applicable.

Table 5.3a Food Animal 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
Hexachlorocyclohexanes	0.01	7	5	0.2	0.09
PAHs	0.01	0.003	0.003	0.07	0.06
Polychlorinated biphenyls					
Congener 77	0.001	6	4	0.07	0.4
81	0.004	10	7	0.2	0.4
105	0.01	10	7	0.6	0.7
114	0.02	10	7	0.9	0.7
118	0.03	10	7	1	0.7
123	0.004	10	7	0.2	0.7
126	0.04	10	7	2	0.7
156	0.02	10	8	0.9	2
157	0.01	10	8	0.5	2
167	0.02	10	8	1	2
169	0.04	10	8	2	2
189	0.005	10	8	0.2	1
Unspeciated (PCB 126)[b]	0.04	10	7	2	0.7
PCDD/Fs					
Congener 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 (2378-TCDD)[b]	0.02	10	9	0.7	0.1

[a] All Tco values were rounded to the nearest whole number.

[b] For unspeciated mixtures, use PCB 126 Tcos to represent the class of PCBs, and 2378-TCDD Tcos to represent the class of PCDDs/Fs.

Table 5.3b 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

Table 5.4 Point Estimates for Animal Pathway

Parameter	Beef Cattle	Lactating Dairy Cattle	Pigs	Meat Poultry	Egg-laying Poultry
BW (body weight in kg)	533	575	55	1.7	1.6
BR_a (inhalation rate in m^3/d)	107	115	7	0.4	0.4
WI_a (water consumption in kg/d)	45	110	6.6	0.16	0.23
FI (Food Intake in kg/d) DMI [a] and/or pasture grazing[b]	9	22	2.4	0.13	0.12
FS_f (soil fraction of feed)	0.01	0.01	NA	NA	NA
FS_p (soil fraction of pasture)	0.05	0.05	0.04	0.02	0.02

[a] Dry matter intake
[b] For beef and dairy cattle, pasture grazing is assumed to be leafy vegetation (grasses, including greenchop) and accounts for half of the cattle's diet (FG 0.5 in Section 5.3.4.2.3). The default assumes on-site pasture grazing contaminated by facility emissions. Fraction of feed or dry matter intake (e.g., hay, grain) grown on-site is assumed to be contaminated by facility emissions and fraction of feed that is grown off-site is not assumed to be contaminated. A default may be used that assumes all feed is grown off-site (L=0 in Section 5.3.4.2.3), but a survey is recommended to verify the fractions of feed grown on-site and off-site.

For pigs with access to soil, but usually confined to a pen, default assumes no pasture grazing (FG=0 in Section 5.3.4.2.3). For feed, estimated intake consists of equal portions of all plant types including exposed, leafy, protected and root in which 10% (L=0.1 in Section 5.3.4.2.3) of the diet is homegrown and contaminated by facility emissions. The fraction of feed that was transported from an off-site location is assumed not to be contaminated by facility emissions.

For poultry including egg-laying and broiler chickens that have access to soil, default assumes no pasture grazing (FG=0 in Section 5.3.4.2.3). Estimated feed intake is composed of equal proportions of all plant types with 5% (L=0.05 in Section 5.3.4.2.3) homegrown and contaminated by facility emissions.

The fraction of feed grown off-site and transported to the receptor was not contaminated by facility emissions.

NA - Not applicable. Assume FS_f is equal to zero.

Table 5.5 Mother's Milk Transfer Coefficients (Tco_m)

Chemical/chem. group	Tcom (day/kg-milk)
PCDDs - oral[a]	3.7
PCDFs - oral[a]	1.8
Dioxin-like PCBs - oral[a]	1.7
PAHs – inhalation	1.55
PAHs – oral	0.401
Lead - inhalation[b]	0.064

[a] Due to limited data available, the oral Tco_m is also used for the inhalation and dermal pathways
[b] Due to limited data available, the inhalation Tco_m is also used for the ingestion pathways

5.4 Estimation of Dose

Once the concentrations of substances are estimated in air, soil, water, plants, and animal products, they are used to evaluate estimated exposure to people. Exposure is evaluated by calculating the daily dose in milligrams per kilogram body weight per day (mg/kg/d). The following algorithms calculate this dose for exposure through inhalation, dermal absorption, and ingestion pathways. All chemicals must be assessed for exposure through inhalation. If there are emissions of one or more of the subset of semi- or non-volatile multipathway substances, the soil ingestion pathway and the dermal soil exposure pathway are also assessed. The mother's milk pathway may also be a mandatory pathway depending on the multipathway substance released (See Table 5.1). The other exposure pathways may also need to be assessed if a survey of the exposure site shows they are present (e.g., ingestion of water, home-grown crops, home-raised animal products, and angler-caught fish).

This section contains average and high-end point estimates and data distributions for adults and children for many exposure pathways. The point-estimates and data distributions for children fall within the 3rd trimester, 0<2, 2<9, and 2<16 year age groupings. The point-estimates and data distributions for adults fall within the 16<30 and 16-70 year age groupings. When evaluating 9-, 30-, and 70-year exposure durations for cancer risk assessment, assessors will use distributions starting at the third trimester.

Workers are assessed for cancer risk as adults using 8-hour breathing rate point estimates (See Table 5.8). Point estimates for workers are listed under "offsite worker." OEHHA has not developed stochastic distributions for worker exposure. Therefore, there is no Tier 3 stochastic approach for offsite worker cancer risk assessment.

5.4.1 *Estimation of Exposure through Inhalation*

The dose through the inhalation route is estimated for cancer risk assessment and noncancer hazard assessment. Both residential and offsite worker exposures are considered. Since residential exposure includes near-continuous long-term exposure at a residence and workers are exposed only during working hours (i.e., 8 hours/day), different breathing rate distributions are used.

5.4.1.1 Residential Inhalation Dose for Cancer Risk Assessment

Exposure through inhalation is a function of the breathing rate, the exposure frequency, and the concentration of a substance in the air. For residential exposure, the breathing rates are determined for specific age groups, so inhalation dose (Dose-air) is calculated for each of these age groups, 3rd trimester, 0<2, 2<9, 2<16, 16<30 and 16-70 years. These age-specific groupings are needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8). A Tier 1 evaluation uses the high-end point estimate (i.e., the 95th percentiles) breathing rates for the inhalation pathway in order to avoid underestimating cancer risk to the public, including children.

A possible exception for using high-end breathing rates are when there is exposure to multipathway substances and two of the non-inhalation pathways drive the risk, rather than the inhalation pathway (see Chapter 8).

A. Equation 5.4.1.1: $$\text{Dose-air} = C_{air} \times \{BR/BW\} \times A \times EF \times 10^{-6}$$

1. Dose-air = Dose through inhalation (mg/kg/d)
2. C_{air} = Concentration in air ($\mu g/m^3$)
3. {BR/BW} = Daily Breathing rate normalized to body weight (L/kg body weight - day)
4. A = Inhalation absorption factor (unitless)
5. EF = Exposure frequency (unitless), days/365 days
6. 10^{-6} = Micrograms to milligrams conversion, liters to cubic meters conversion

a: Recommended default values for EQ 5.4.1.1:

1. {BR/BW} = Daily breathing rates by age groupings, see As supplemental information, the assessor may wish to evaluate the inhalation dose by using the mean point estimates in Table 5.6 to provide a range of breathing rates for cancer risk assessment to the risk manager.
2. Table (point estimates) and Table 5.7 (parametric model distributions for Tier III stochastic risk assessment). For Tier 1 residential estimates, use 95th percentile breathing rates in Table 5.6.
3. A = 1
4. EF = 0.96 (350 days/365 days in a year for a resident)

b: Assumption for EQ 5.4.1.1:

1. The fraction of chemical absorbed (A) is the same fraction absorbed in the study on which the cancer potency or Reference Exposure Level is based.

As supplemental information, the assessor may wish to evaluate the inhalation dose by using the mean point estimates in Table 5.6 to provide a range of breathing rates for cancer risk assessment to the risk manager.

Table 5.6 Point Estimates of Residential Daily Breathing Rates for 3rd trimester, 0<2, 2<9, 2<16, 16<30 and 16-70 years (L/kg BW-day)

	3rd Trimester	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

Table 5.7 Daily Breathing Rate Distributions by Age Group for Residential Stochastic Analysis (L/kg BW-day)

	3rd Trimester	0<2 years	2<9 years	2<16 years	16<30 years	16-70 years
Distribution	Max extreme	Max extreme	Max extreme	Log-normal	Logistic	Logistic
Minimum	78	196	156	57	40	13
Maximum	491	2,584	1,713	1,692	635	860
Scale	59.31	568.09	125.59		40.92	36.19
Likeliest	191.50	152.12	462.61			
Location				-144.06		
Mean	225	658	535	452	210	185
Std Dev	72	217	168	172	75	67
Skewness	0.83	2.01	1.64	1.11	0.83	1.32
Kurtosis	3.68	10.61	7.88	6.02	5.17	10.83
Percentiles						
5%	127	416	328	216	96	86
10%	142	454	367	259	118	104
25%	179	525	427	331	161	141
50%	212	618	504	432	207	181
75%	260	723	602	545	252	222
80%	273	758	631	572	261	233
90%	333	934	732	659	307	262
95%	361	1090	861	745	335	290
99%	412	1430	1,140	996	432	361

5.4.1.2 Offsite Worker (MEIW) Inhalation Dose for Cancer Risk Assessment

For worker exposure, the default assumes working age begins at 16 years, and that exposures to facility emissions occur during the work shift, typically up to 8 hours per day during work days. Breathing rates that occur over an 8-hour period vary depending on the intensity of the activity (See Table 5.8), and are used to estimate the inhalation dose. The 8-hour breathing rates may also be useful for cancer risk assessment of children and teachers exposed at schools during school hours.

Another risk management consideration for the offsite worker scenario for cancer assessment of a Hot Spots facility is whether there are women of child-bearing age at the MEIW location and whether the MEIW has a daycare center. Since the third trimester is only a short segment of the 25 year exposure duration used for the MEIW, the resulting risk estimate would not differ significantly. An exception to this assumption is high exposure to carcinogens over a short period, as might occur during short-term projects (see Section 8.2.10). In this case, risk assessment during the third trimester may be warranted. However, if there is onsite daycare at the MEIW, then the risks to the children will be underestimated using the offsite adult worker scenario due to increased exposure (per kg body weight) and increased sensitivity to carcinogen exposure (see Section 8.2.1). In this case, the Districts may wish to include a calculation of inhalation dose for the children in the onsite daycare, assuming they could be there from 0 to age 6 years.

Exposed workers may be engaged in activities ranging from desk work, which would reflect breathing rates of sedentary/passive or light activities, to farm worker activities, which would reflect breathing rates of moderate intensity (See Table 5.9). OEHHA recommends default (Tier 1) point estimate 8-hour breathing rates in L/kg-8-hrs based on the mean and 95th percentile of moderate intensity activities, 170 and 230 L/kg-8-hrs, respectively, for adults 16-70 years old.

Many facilities operate non-continuously, as in only 8-10 hours per day, but the air dispersion modeling is performed as if the emissions were uniformly emitted over 24 hours a day, 7 days per week. The air dispersion computer model used, including AERMOD and other models, typically calculate an annual average air concentration based on actual operating conditions but also include the hours of nonoperation in the average concentration.

Therefore, there are two components that determine the worker exposure to facility emissions:

1) What is the estimated concentration the worker is exposed to (i.e., breathes), during their work shift, and

2) What is the amount of time the offsite worker's schedule overlaps with the facilities' emission schedule?

There are two approaches to estimating the modeled concentration the worker is breathing during their work shift. The first approach uses a worker adjustment factor (i.e., the WAF) to approximate what the worker is breathing based on the modeling run used for residential receptors. The second approach uses a special modeling run with the hourly raw results from an air dispersion analysis and is described in Appendix M.

The first and more basic approach is to obtain the long term average concentration as you would for modeling a residential receptor, then adjusting this exposure concentration using the calculated WAF (EQ 5.4.1.2 B) to estimate the concentration the offsite worker is would be exposed to during their work shift (shown as (C_{air} × WAF) in EQ 5.4.1.2 A). This method is characteristic of a default approach used in a Tier 1 assessment. Once the exposure concentration is determined, the workers inhalation dose (Dose-air) can be calculated as shown in EQ 5.4.1.2 A.

The second approach for determining the air concentration the worker is exposed to uses a refined modeling run where the hourly raw dispersion model output are post processed to examine the hourly concentrations that fall within the offsite worker's shift. This method provides a more representative estimate of the air concentration, but is more complex, and time consuming than the first method. See Appendix M for information on how to simulate the long term concentration for the offsite worker that can be used to estimate inhalation cancer risk.

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

If the off-site workers shift does not completely overlap the emission schedule of the facility, then a Discount Factor (DF) may be applied to the WAF. Calculation of the DF is shown in EQ 5.4.1.2 C. The default assumption is that the offsite workers shift falls completely within the emission schedule of the facility, in which case DF=1. Use of a DF less than 1 requires a survey at the MIEW to verify that some portion of the off-site worker shift is not subject to the facility emissions.

A. Equation 5.4.1.2 A: $\text{Dose-air} = (C_{air} \times WAF) \times \{BR/BW\} \times A \times EF \times 10^{-6}$

1. Dose-air = Dose through inhalation (mg/kg/d)
2. C_{air} = Concentration in air ($\mu g/m^3$)
3. WAF = Worker air concentration adjustment factor (unitless)
4. {BR/BW} = Eight-hour breathing rate normalized to body weight (L/kg body weight - day)
5. A = Inhalation absorption factor (unitless)
6. EF = Exposure frequency (unitless), days/365 days)
7. 10^{-6} = Micrograms to milligrams conversion, Liters to cubic meters conversion

a: Recommended default values for EQ 5.4.1.2 A:

1. WAF = See EQ. 5.4.1.2 B for formula to calculate WAF, or App. M for refined post-processing modeling to calculate WAF.
2. {BR/BW} = For workers, use age16-70 year, 95th percentile, moderate intensity 8-hour point estimate breathing rates (see Table 5.8). No worker breathing rate distributions exist for stochastic risk assessment.
3. A = 1
4. EF = 0.68 (250 days / 365 days). Equivalent to working 5 days/week, 50 weeks/year.

b: Assumption for EQ 5.4.1.2 A:

1. The fraction of chemical absorbed (A) through the lungs is the same fraction absorbed in the study on which the cancer potency factor is based.
2. The source emits during the daylight hours. Calculate WAF (EQ 5.4.1.2 B) if a special post-processing modeling run described in App. M was not completed. For nighttime emissions and exposure scenarios, see Appendix N.

B. Equation 5.4.1.2 B: $$WAF = (H_{res} / H_{source}) \times (D_{res} / D_{source}) \times DF$$

1. WAF = Worker adjustment factor (unitless)
2. H_{res} = Number of hours per day the annual average residential air concentration is based on (always 24 hours)
3. H_{source} = Number of hours the source operates per day
4. D_{res} = Number of days per week the annual average residential air concentration is based on (always 7 days)
5. D_{source} = Number of days the emitting source operates per week
6. DF = Discount factor, for when the offsite worker's schedule partially overlaps the source's emission schedule

b: Recommended default values for EQ 5.4.1.2 B:

1. DF = 1 for offsite worker's schedule occurring within the source's emission schedule. A site-specific survey may be used to adjust the DF using EQ 5.4.1.2 C.

C. Equation 5.4.1.2 C: $$DF = (H_{coincident} / H_{worker}) \times (D_{coincident} / D_{worker})$$

1. $H_{coincident}$ = Number of hours per day the offsite worker's schedule and the source's emission schedule coincide
2. H_{worker} = Number of hours the offsite worker works per day
3. $D_{coincident}$ = Number of days per week the offsite worker's schedule and the source's emission schedule coincide
4. D_{worker} = Number of days the offsite worker works per week

Tier 2 adjustments for EQ 5.4.1.2 A-C may be used for:

1. Eight-hour breathing rate. Point estimates in Table 5.8 for lower breathing rates of sedentary/passive and light intensity work activities may be substituted in site-specific Tier 2 scenarios. Table 5.9 can be used to estimate breathing rate intensities for various job activities. Use of different breathing rates requires a survey of the exposed workplace and approval by Air District, ARB and OEHHA.
2. Discount Factor (DF) in EQ 5.4.1.2 C. If a site-specific survey of the offsite worker schedule only partially overlaps with the source's emission schedule, then a DF less than 1 may be calculated. Use of a DF less than 1 requires a survey of the exposed workplace and approval by the Air District or ARB.

The 8-hour breathing rates are based on minute ventilation rates derived by U.S. EPA (2009). U.S. EPA employed a metabolic equivalent (METS) approach for estimating breathing rates. This method determines daily time-weighted averages of energy expenditure (expressed as multipliers of the basal metabolic rate) across different levels of physical activity. The 8-hour breathing rates shown in Table 5.8 are divided into three categories:

Sedentary & Passive Activities (METS $\leq$ 1.5)

Light Intensity Activities (1.5 < METs $\leq$ 3.0)

Moderate Intensity Activities (3.0 < METs $\leq$ 6.0)

For example, a METS = 1 is roughly equivalent to energy expenditure during sleep and is close to the basal metabolic rate. A METS activity that is two to three times greater (METS = 2 to 3) is characteristic of light intensity activities, such as administrative office work or sales work as shown in Table 5.9.

Table 5.8. Eight Hour Breathing Rate (L/kg-8 Hr) Point Estimates for Males and Females Combined[a]

	0<2 years	**2<9 years**	**2<16 years**	**16<30 years**	**16-70 years**
	Sedentary & Passive Activities (METS $\leq$ 1.5)				
Mean	200	100	80	30	30
95th Percentile	250	140	120	40	40
	Light Intensity Activities (1.5 < METs $\leq$ 3.0)				
Mean	490	250	200	80	80
95th Percentile	600	340	270	100	100
	Moderate Intensity Activities (3.0 < METs $\leq$ 6.0)				
Mean	890	470	380	170	170
95th Percentile	1200	640	520	240	230

[a] For pregnant women, OEHHA recommends using the mean and 95th percentile 8-hour breathing rates based on moderate intensity activity of 16<30 year-olds for 3rd trimester.

Table 5.9. METS Distributions for Workplace and Home Activities

Activity Description	Mean	Median	SD	Min	Max
Workplace Activities					
Administrative office work	1.7	1.7	0.3	1.4	2.7
Sales work	2.9	2.7	1.0	1.2	5.6
Professional	2.9	2.7	1.0	1.2	5.6
Precision/production/craft/repair	3.3	3.3	0.4	2.5	4.5
Technicians	3.3	3.3	0.4	2.5	4.5
Private household work	3.6	3.5	0.8	2.5	6.0
Service	5.2	5.3	1.4	1.6	8.4
Machinists	5.3	5.3	0.7	4.0	6.5
Farming activities	7.5	7.0	3.0	3.6	17.0
Work breaks	1.8	1.8	0.4	1.0	2.5
Household/Neighborhood Activities					
Sleep or nap	0.9	0.9	0.1	0.8	1.1
Watch TV	1.0	1.0	-	1.0	1.0
General reading	1.3	1.3	0.2	1.0	1.6
Eat	1.8	1.8	0.1	1.5	2.0
Do homework	1.8	1.8	-	1.8	1.8
General personal needs and care	2.0	2.0	0.6	1.0	3.0
Indoor chores	3.4	3.0	1.4	2.0	5.0
Care of plants	3.5	3.5	0.9	2.0	5.0
Clean house	4.1	3.5	1.9	2.2	5.0
Home repairs	4.7	4.5	0.7	4.0	6.0
General household chores	4.7	4.6	1.3	1.5	8.0
Outdoor chores	5.0	5.0	1.0	2.0	7.0
Walk/bike/jog (not in transit) age 20	5.8	5.5	1.8	1.8	11.3
Walk/bike/jog (not in transit) age 30	5.7	5.7	1.2	2.1	9.3
Walk/bike/jog (not in transit) age 40	4.7	4.7	1.8	2.3	7.1

Table 5.10 lists some WAFs for a few typical scenarios. For example, if the source is continuously emitting, then the offsite worker is assumed to breathe the long-term annual average concentration during their work shift. The WAF then becomes one and no concentration adjustments are necessary in this situation when estimating the inhalation cancer risk. If the source is non-continuously emitting for 8 hours/day, 5 days/week and the offsite workers' shift completely overlaps the emitting facility's operating schedule, then the WAF would be 4.2:

$$(24 \text{ hrs/day} / 8 \text{ hrs/day}) \times (7 \text{ days/week} / 5 \text{ days/week}) = 4.2$$

If the offsite workers' 8 hour/day shift only overlaps the emitting facility's operation schedule for 4 hrs/day, then the WAF is 2.1 because the DF = 0.5 will reduce the WAF by half:

DF = (4 hrs/day / 8 hrs/day) x (5 days/week / 5 days/week) = 0.5

Table 5.10: Example Worker Adjustment Factors (WAF) to Convert a Long-Term Daily Average Emission Concentration to an Off-Site Worker Receptor Exposure

Off-Site Workers' Shift Overlap with Facility's Emission Schedule[a]	Facility Operating Schedule	Adjustment Factor
8 hrs/day, 5 days/week	Continuous (24 hrs/7 days/week)	1.0
8 hrs/day, 5 days/week[b]	Non-continuous (8 hrs/5 days/week)	4.2
4 hrs/day, 5 days/week	Non-continuous (8 hrs/5 days/week)	2.1

[a] Worker works 8 hours per day, 5 days per week
[b] Workers work hours completely overlaps the facilities operating hours

5.4.1.3 Inhalation Dose for Children at Schools and Daycare Facilities for Cancer Risk Assessment

The 8-hour breathing rates and inhalation dose equations (EQ 5.4.1.2 A-C) may also be used to estimate risk to children when exposures occur while at school or at day care facilities. Breathing rate point estimates to use in Table 5.8 depend on the ages of the children at the exposed schools and day cares. As a Tier 1 default, moderate intensity breathing rates are recommended. Equations 5.4.1.2 A-C is used in the same way to estimate dose in children as it is for workers.

5.4.1.4 Non-Cancer Inhalation Exposure for Workers and Residents

For typical daily work shifts of 8-9 hours, acute, 8-hour and chronic Reference Exposure Levels (RELs) described in Chapter 8 are used in health risk assessments to characterize the noncancer risks using the Hazard Index approach described in Chapter 8 and in OEHHA (2008). Uncertainty factors are already incorporated into the RELs used to assess noncancer risk, as explained in Chapter 8, so all that is needed to evaluate the noncancer hazard is the air concentration that the worker is exposed to. The modeled maximum 1-hour air concentration is determined for acute hazard assessment and the annual average air concentration is determined for chronic hazard assessment. The modeled average air concentration during a work shift is determined for 8-hour hazard assessment using the adjusted annual average air concentration described below.

The 8-hour RELs are primarily designed to address offsite worker inhalation exposure at the MEIW because they better characterize the daily intermittent exposures of workers than the chronic RELs do. They are used in estimating the 8 hour Hazard Index for

offsite workers. Note, however, there are only a handful of 8-hour RELs currently adopted for use in the Hot Spots program. Therefore, we also recommend performing chronic noncancer exposure assessment for the offsite worker (MEIW) based on the annual average air concentration at the MEIW. Evaluation of the chronic Hazard Index should help protect workers who routinely work longer than 8 hour shifts. Exposure to multipathway substances also requires noncancer hazard assessment for the dermal and oral soil exposure pathways for offsite workers. Because there are few 8-hour RELs currently available, hazard assessment for the noninhalation pathways for multipathway substances is only applied when estimating the chronic Hazard Index.

In addition, the Districts may wish to determine if there is an onsite daycare at the MEIW and include a calculation of the chronic and 8-hour inhalation dose for children, although onsite hazard assessment is not a requirement for a Hot Spots risk assessment.

As explained in Section 5.4.1.2 for cancer risk, the modeled annual average air concentration is adjusted to the air concentration that the worker is actually exposed to if the facility operates non-continuously. The typical method for this adjustment is by calculating the Worker Adjustment Factor (WAF) shown in EQ 5.4.1.4 B and multiplying this value by the annual average air concentration (C_{air}, in $\mu g/m^3$) in EQ 5.4.1.4 A.

Unlike cancer risk assessment, no discount factor (DF) is applied in noncancer assessment for partial overlap between the worker's schedule and the source's emission schedule. Adjustments for worker vacations, work shifts for shortened weeks (e.g., 1 - 4 days), and worker time away on weekends are also not appropriate.

An alternative refined post-processing method, described in Appendix M, may be used to estimate the air concentration the worker is exposed to during their work schedule. OEHHA may be consulted about the particular chemical involved if it is important to make a more refined analysis.

The equation to adjust the annual average air concentration to a worker 8-hour exposure concentration (i.e., the adjusted annual average ground level concentration) is expressed as:

A. Equation 5.4.1.4 A:

$$\text{Adjusted } C_{air} \ (\mu g/m^3) = C_{air} \times WAF$$

Where WAF is determined as:

B. Equation 5.4.1.4 B:

$$WAF = (H_{res} / H_{source}) \times (D_{res} / D_{source})$$

a: Assumptions for EQ 5.4.1.4 B:

1. No adjustment of the WAF allowed for partial overlap of the worker's schedule and the source's emission schedule.

Alternatives for calculating off-site worker Adjusted C_{air} in EQ 5.4.1.4 A-B:

1. Rather than calculate the WAF for a non-continuous emitting facility, a post-processing of the hourly raw dispersion model output and examination of the hourly concentrations that fall within the offsite worker's shift can be conducted to estimate the air concentration the worker is exposed to. This method is a more refined, complex, and time consuming approach, but should result in a more representative exposure concentration. See Appendix M for information on how to simulate the exposure concentration for the off-site worker.
2. For continuously-emitting facilities (i.e., 24 hrs/day, 7 days/week), if an assessor does not wish to assume the worker breathes the long-term annual average concentration during the work shift, then a refined concentration can also be post-processed as described in Appendix M. All alternative assumptions should be approved by the reviewing authority and supported in the presentation of results.

For residential exposure to non-continuously operating facilities, the modeled maximum 1-hour and chronic air concentrations at the MEIR are determined for noncancer hazard assessment. Hazard assessment for repeated 8-hour exposure at the MEIR is not required. Chronic exposure assessment based on the annual average air concentration should adequately protect individuals, in part because residents are considered to be present at the MEIR at or near 24 hrs per day. Many facilities operate for periods longer than 8 hours per day and the hazards are better characterized based on chronic exposure. Nevertheless, differences between 8-hour and chronic exposures (i.e., higher daily 8-hour exposures vs. lower longer daily exposure 24 hrs/day) may result in different toxicological responses including potentially greater toxicological responses with either 8-hour or chronic exposure. There may also be cases such as special meteorological situations (e.g., significant diurnal-nocturnal meteorological differences) where the 8-hour REL will be more protective than the chronic REL. Thus, the air districts may also elect to have an 8-hour hazard assessment performed at the MEIR, using daily 8 hour exposures and the 8 hr RELs.

Eight-hour exposure assessment is not recommended for continuously emitting sources for residential receptors. In this situation it is only necessary to estimate chronic exposure based on the annual average concentration. However, there may be situations where the air district may wish to assess an 8-hour residential exposure to continuously operating facilities, for example, where there are significant differences in modeled concentration of emissions during the day due to diurnal wind patterns.

For estimating the air concentration from non-continuously operating facilities, EQ 5.4.1.4.A is also used to adjust the annual average concentration to what the residents are exposed to. This is the air concentration that the 8-hour REL will be compared to as discussed in Chapter 8. The alternative refined post-processing method described in Appendix M may also be used to estimate residential exposure.

In summary, the requirements for noncancer hazard assessment using the Hazard Index approach at the MEIW and MEIR are as follows.

For offsite worker exposure:

- Acute hazard assessment based on the maximum 1-hour air concentrations and 1-hour RELs
- Eight-hour hazard assessment based on daily average 8-hour exposure (estimated using adjusted annual average air concentration in EQ 5.4.1.4 A and B or by post-processing method in App. M) for those substances with 8-hour RELs
- Chronic hazard assessment based on annual average exposure and chronic RELs, and oral chronic RELs for noninhalation routes of multipathway substances

For residential exposure:

- Acute hazard assessment based on the maximum 1-hour air concentration and 1-hour RELs
- Eight-hour hazard assessment based on daily average 8-hour exposure not required, but can be performed at the discretion of the air districts for exposure to non-continuously operating facilities based on the adjusted annual average air concentration (EQ 5.4.1.4 A and B or method in App. M). Eight-hour assessments not recommended for exposure to continuously operating facilities
- Chronic hazard assessment based on annual average exposure and chronic RELs, and oral chronic RELs for noninhalation routes of multipathway substances

5.4.1.5 Exposure Frequency and Age Groupings for Noncancer Hazard Assessment

For cancer risk, the basic assumption is that risk is associated with cumulative dose of carcinogen. Thus, the dose used to estimate cancer risk can be adjusted for exposure frequency, as well as time spent within the MEIR or MEIW location. Chronic RELs are not necessarily related to cumulative dose. Thus, adjusting the estimated dose used to calculate hazard index for exposure frequency or time away from the MEIR or MEIW is not appropriate.

The average daily dose for chronic noncancer assessment is based on exposure beginning at birth to 70 years of age, necessitating calculation of a time-weighted average for age 0-2, 2-16 and 16-70 years. Since we are not applying Age Sensitivity Factors for assessing non-cancer hazard, the 3rd trimester is not explicitly called out for determining dose, as it is for cancer risk assessment. Rather adult exposure is considered, which would include pregnant women in any trimester. Both inhalation and oral RELs incorporate safety factors to protect sensitive human populations.

5.4.2 *Estimation of Exposure through Dermal Absorption*

Exposure through dermal absorption (dose-dermal) is a function of the soil or dust loading of the exposed skin surface, the amount of skin surface area exposed, and the

concentration and availability of the substance. In the previous edition of OEHHA's exposure guidelines document (OEHHA, 2000), we recommended using specified average and high-end point estimate values for four of the variates (body weight, exposed surface area of skin, soil load on skin and frequency of exposure) in the stochastic analysis for dermal dose. This equation required multiplying values together, which could lead to overly conservative exposure estimates when high-end values were used. By combining information from the four variates into one composite distribution, over-conservatism may be avoided.

To this end, OEHHA created a new variate, "annual dermal load", or ADL, which is a composite of the body surface area (BSA) per kg body weight, exposure frequency, and soil adherence variates. Point estimates from the composite "annual dermal load" can be used for point estimate assessments while parameters and information on the type of distribution (e.g., lognormal) can be used for Tier III stochastic risk assessments. For details on the development of the ADL, refer to the Technical Support Document for Exposure and Stochastic Analysis (OEHHA, 2012).

5.4.2.1 Dermal Dose for Cancer Risk Assessment

The dose through residential dermal exposure to contaminated soil varies by age and is calculated for each age group (e.g., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 and 16-70 yrs). These age-specific groupings are needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8). This pathway is also assessed for exposure to offsite workers; a separate ADL for offsite workers is presented in Table 5.11. Children at a MEIW daycare, if present, may also be assessed for exposure if the District deems it advisable.

A. Equation 5.4.2.1:

$$\mathbf{Dose_{dermal} = ADL \times Cs \times ABS \times 10^{-9} \ / \ 365}$$

1. $Dose_{dermal}$ = Exposure dose through dermal absorption (mg/kg-d)
2. ADL = Annual dermal load (mg soil/kg BW-yr)
3. C_s = Average soil concentration (µg/kg)
4. ABS = Fraction absorbed across skin (unitless)
5. 10^{-9} = Conversion factor for chemical & soil (µg to mg, mg to kg)
6. 1/365 = Conversion factor for ADL from yrs to days

a: Recommended default values for EQ 5.4.2.1:

1. ADL = See Table 5.11 (point estimates) & Table 5.12 a-d (distributions)
2. C_s = Calculated above in EQ 5.3.2 A
3. ABS = See Table 5.13

b: Assumption for EQ 5.4.2.1:

1. The ADL for the third trimester of the fetus is based on the ADL of the mother; when normalized to body weight, we assume that exposure to the mother and the fetus will be the same. The mother's exposure is based on the adults age 16-30 years of age in Table 5.11 and 5.12d.
2. Exposure frequency (EF) for vacation time spent away from exposure does not appear as a variate in EQ 5.4.2.1, as it is incorporated in the ADL and includes a 2-week vacation per year away from dermal soil exposure for both residents and offsite workers.

Climate will strongly influence people's choice of clothing. Due to California's varied climatic regions and existing data on clothing choices at different temperatures, three levels of climatic conditions, warm, mixed, and cold, are used to describe California's climate regions:

1. A warm climate is characteristic of Southern California areas such as Los Angeles, which can have warm to hot temperatures throughout the year.
2. A "mixed" climate is one that has warm-to-hot temperatures during much of the year (daily highs over 80 degrees are common), roughly from April to October, and cold temperatures (lows near or below freezing) during the remainder of the year. The mountains and central valley are examples of a mixed climate.
3. A cold climate is representative of San Francisco, Eureka, and other northern coastal communities, which have cool temperatures (daily highs of less than 65 degrees) for the majority of the year and can receive a considerable amount of fog and rainfall.

OEHHA recommends consulting the local air district for assistance on selecting the most appropriate climate.

Table 5.11 Recommended Annual Dermal Load Point Estimates (in mg/kg-yr) for Dermal Exposure

	3rd Trimester[a]	Children 0<2 yrs	Children 2<9 yrs	Children 2<16 yrs	Adults[b]	Offsite Worker[c]
Warm climate						
Mean	1.2×10^3	3.6×10^3	7.5×10^3	6.4×10^3	1.2×10^3	2.6×10^3
95th percentile	2.6×10^3	4.3×10^3	9.1×10^3	8.5×10^3	2.6×10^3	5.0×10^3
Mixed climate						
Mean	1.1×10^3	2.2×10^3	6.6×10^3	5.7×10^3	1.1×10^3	2.6×10^3
95th percentile	2.4×10^3	2.9×10^3	8.7×10^3	8.1×10^3	2.4×10^3	5.0×10^3
Cold climate						
Mean	0.7×10^3	1.2×10^3	3.1×10^3	2.8×10^3	0.7×10^3	2.6×10^3
95th percentile	2.1×10^3	1.9×10^3	5.2×10^3	5.1×10^3	2.1×10^3	5.0×10^3

[a] The ADL for the 3rd trimester of the fetus is based on the ADL of the mother; when normalized to body weight, we assume that exposure to the mother and the fetus will be the same
[b] Residential adult ADLs are for both 16<30 and 16-70 year age groups
[c] Assumes exposure only to face, hands and forearms regardless of climate region

Tables 5.12a - d Annual Dermal Load Distributions by Age Group and Climate for Stochastic Analysis

Table 5.12a Annual Dermal Load (mg/kg-yr) Distributions for the 0<2 Year Age Group

Climate Type	Warm climate	Mixed climate	Cold climate
Distribution	Student's t	Logistic	Triangular
Minimum			0.2×10^3
Likeliest			0.7×10^3
Maximum			2.6×10^3
Scale	0.41	0.28	
Deg. freedom	3		
Midpoint	3.6×10^3		
Mean	3.6×10^3	2.2×10^3	1.2×10^3
50th percentile	3.6×10^3	2.2×10^3	0.9×10^3
90th percentile	4.1×10^3	2.8×10^3	1.9×10^3
95th percentile	4.3×10^3	2.9×10^3	1.9×10^3
99th percentile	4.7×10^3	3.1×10^3	2.1×10^3

Table 5.12b Annual Dermal Load (mg/kg-yr) Distributions for the 2<9 Year Age Group

Climate Type	Warm climate	Mixed climate	Cold climate
Distribution	Min extreme	Min extreme	Triangular
Minimum			0.4×10^3
Likeliest	8.0×10^3	7.3×10^3	1.9×10^3
Maximum			6.9×10^3
Scale	0.1	1.3	
Mean	7.5×10^3	6.6×10^3	3.1×10^3
50th percentile	7.7×10^3	6.5×10^3	2.3×10^3
90th percentile	8.7×10^3	8.4×10^3	5.1×10^3
95th percentile	9.1×10^3	8.7×10^3	5.2×10^3
99th percentile	9.7×10^3	9.4×10^3	5.7×10^3

Table 5.12c Annual Dermal Load (mg/kg-yr) Distributions for the 2<16 Year Age Group

Climate Type	Warm Climate	Mixed climate	Cold climate
Distribution	Min extreme	Logistic	Triangular
Minimum			0.3×10^3
Likeliest	7.2×10^3		1.6×10^3
Maximum			6.9×10^3
Scale	1.29	0.91	
Mean	6.4×10^3	5.7×10^3	2.8×10^3
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 5.12d Annual Dermal Load (mg/kg-yr) Distributions for Residential Adults (Age 16-30 and 16-70 Years) [a] and Offsite Workers

Receptor	Residential Adult			Offsite Worker
Climate Type	Warm	Mixed	Cold	All Climates[b]
Distribution	Beta	Beta	Gamma	Lognormal
Minimum	0.2×10^3	0.02×10^3		
Maximum	3.3×10^3	0.3×10^3		
Scale			0.07	
Mean	1.2×10^3	1.1×10^3	0.7×10^3	2.6×10^3
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] The ADL distribution for the 3rd trimester is based on the ADL distribution of the mother; we assume the same ADL distribution for residential adult (the mother) and the fetus

[b] Face, hands and forearms are exposed only, regardless of climate

Table 5.13 Dermal Absorption Fraction Factors (ABS) as Percent from Soil for Semi-Volatile and Solid Chemicals under the OEHHA "Hot Spots" Program

Chemical	ABS
Inorganic chemicals	
Arsenic	6
Beryllium	3
Cadmium	0.2
Chromium (VI)	2
Fluorides (soluble compounds)	3
Lead	3
Mercury	4
Nickel	2
Selenium	3
Organic chemicals	
Creosotes	13
Diethylhexylphthalate	9
Hexachlorobenzene	4
Hexachlorocyclohexanes	3
4,4'methylene dianiline	10
Pentachlorophenol	[a]
Polychlorinated biphenyls	14
Polychlorinated dibenzo-p-dioxins and dibenzofurans	3
Polycyclic aromatic hydrocarbons	13

[b] To be assessed for dermal absorption

Skin permeability is related to the solubility or strength of binding of the chemical in the delivery matrix (soil or other particles) versus the receptor matrix, the skin's stratum corneum. Fractional dermal absorption point estimate values were derived by OEHHA from available literature sources for the semi-volatile and nonvolatile chemicals in the "Hot Spots" program. The rationale for the chemical-specific dermal absorption fraction values, and the use of default values in cases where sufficient data are lacking, can be found in Appendix F of the Technical Support Document for Exposure and Stochastic Analysis (OEHHA, 2012).

5.4.2.2 Chronic Noncancer Dermal Dose

Dermal exposure, and thus annual dermal load (ADL), varies by age group. Therefore, a time-weighted average ADL for age 0-70 years (0-2, 2-16, and 16-70 years) is estimated for chronic residential exposure using ADL values in Table 5.12. This exposure pathway is also assessed for offsite workers using the offsite worker ADL values in Table 5.12d. Children at a MEIW daycare, if present, may also be assessed for exposure if the District deems it advisable. The contribution to the dermal dose is determined for each age group in EQ 5.4.2.2:

A. Equation 5.4.2.2: $\mathbf{Dose_{dermal} = ADL \times Cs \times ABS \times 10^{-9} \times ED/AT \times (1/350)}$

1. $Dose_{dermal}$ = Exposure dose through dermal absorption (mg/kg/d)
2. ADL = Annual dermal load (mg/kg-yr), age-specific
3. Cs = Average soil concentration (μg/kg)
4. ABS = Fraction absorbed across skin (unitless)
5. 10^{-9} = Conversion factor for chemical & soil (μg to mg, mg to kg)
6. 1/350 = Conversion factor for ADL from yrs to days (Note: this conversion is needed to remove EF, expressed as 350 days/365 days, from the ADLs in Table 5.12a-d)
7. ED = Exposure duration for specified age groups: 2 yrs for 0<2, 14 yrs for 2<16, 54 yrs for 16-70 for residential exposure,
8. AT = Averaging time for residential exposure – 70 yrs

a: Recommended default values for EQ 5.4.2.2:

1. ADL = See Table 5.11 for point estimates by age group, climate region and receptor type (resident or worker)
2. Cs = Calculated above in EQ 5.3.2 A
3. ABS = See Table 5.13

b: Recommended off-site worker default modifications to EQ 5.4.2.2:

1. Chronic dermal dose to the off-site worker assumes only adult exposure and is incorporated into the off-site worker ADL in Table 5.12d.
2. A time-weighted average estimate of dose is not necessary and the ED and AT variates are left out of EQ 5.4.2.2 for dermal dose to the worker.

c: Recommended nursing mother default modifications to EQ 5.4.2.2:

1. For dermal dose to mother's milk, use the ADL for age 16-30 years in Table 5.12d.
2. The ED and AT variates in EQ 5.4.2.2 are left out for dermal dose in the mother's milk pathway.

d: Assumptions for EQ 5.4.2.2:

1. For cancer risk assessment, Exposure Frequency (EF) for vacation time away from exposure is incorporated into the ADLs shown in Tables 5.11 and 5.12 using the basic assumption that cancer risk is associated with cumulative dose of carcinogen. The dose used to estimate cancer risk can be adjusted for EF, and for time spent within the MEIR or MEIW location. Chronic RELs are not necessarily related to cumulative dose. Thus, adjusting the estimated dose for EF at the MEIR or MEIW is not appropriate, and the unadjusted daily rate is used in EQ 5.4.2.2.

2. For worker exposure, the annual average concentration should not be adjusted to account for worker and facility emission schedules, as done for inhalation cancer risk assessment. The pollutant will be deposited and accumulate in the soil in the absence or presence of the worker; therefore, the total deposition and soil concentration will be dependent on the annual average air concentration.

For residential chronic exposure, the dermal dose contribution for each age group is summed together to obtain the time-weighted average daily dermal dose for chronic hazard assessment:

$$(\text{ADL age } 0<2 \times Cs \times ABS \times 10^{-9} \times 2 / 70 \times (1/350)) +$$

$$(\text{ADL age } 2<16 \times Cs \times ABS \times 10^{-9} \times 14 / 70 \times (1/350)) +$$

$$(\text{ADL age } 16\text{-}70 \times Cs \times ABS \times 10^{-9} \times 54 / 70 \times (1/350)) = \text{Chronic Dose}_{dermal}$$

5.4.3 *Estimation of Exposure through Ingestion*

Exposure through ingestion is a function of the concentration of the substance in the ingested soil, water, and food, the gastrointestinal absorption of the substance, and the amount ingested.

5.4.3.1 Exposure through Ingestion of Soil

There are no distributions for soil ingestion currently recommended. Tier III stochastic risk assessments should include a high-end point estimate of soil ingestion, soil loading, exposure frequency and soil area.

5.4.3.1.1 Soil Ingestion Dose for Cancer Risk

The exposure dose through residential soil ingestion varies by age and is calculated for each age group ((e.g., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 and 16-70 yrs). These age-specific groupings are needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8). This pathway is also assessed for exposure to off-site workers. Children at a MEIW daycare, if present, may also be assessed for exposure if the District deems it advisable. The dose from inadvertent soil ingestion can be estimated by the point estimate approach using the following general equation:

A. Equation 5.4.3.1.1: $\mathbf{DOSE_{soil} = C_{soil} \times GRAF \times SIR \times 10^{-9} \times EF}$

1. $DOSE_{soil}$ = Dose from soil ingestion (mg/kg BW-day)
2. 10^{-9} = Conversion factor (μg to mg, mg to kg)
3. C_{soil} = Concentration of contaminant in soil (μg/kg)
4. GRAF = Gastrointestinal relative absorption fraction, chemical-specific (unitless)
5. SIR = Soil ingestion rate (mg/kg BW-day)
6. EF = Exposure frequency (unitless), (days/365 days)

a: Recommended default values for EQ 5.4.3.1.1:

1. C_{soil} = Calculated above in EQ 5.3.2 A
2. GRAF = See Table 5.2
3. SIR = See Table 5.14
4. EF = 350 d/year resident, 250 d/year worker

In this approach, it is assumed that the soil ingested contains a representative concentration of the contaminant(s) and the concentration is constant over the exposure period.

The term **GRAF**, or gastrointestinal relative absorption factor, is defined as the fraction of contaminant absorbed by the GI tract relative to the fraction of contaminant absorbed from the matrix (feed, water, other) used in the study(ies) that is the basis of either the cancer potency factor (CPF) or the Reference Exposure Level (REL). If no data are available to distinguish absorption in the toxicity study from absorption from the environmental matrix in question (i.e., soil), then GRAF = 1. The GRAF allows for adjustment for absorption from a soil matrix if it is known to be different from absorption across the GI tract in the study used to calculate the CPF or REL. In most instances, the GRAF will be 1.

Table 5.14 Recommended Soil Ingestion Rate (SIR) Estimates for Adults and Children (mg/kg-day)*

Age Groups (years)	Mean (mg/kg-day)	95th % (mg/kg-day)
3rd Trimester[a]	0.7	3
0<2	20	40
2<9	5	20
2<16	3	10
16<30	0.7	3
16 to 70	0.6	3
PICA adult	NR	-

[a] Assumed to be the mother's soil ingestion rate (adult age 16 <30)
* Soil includes outdoor settled dust
NR = No recommendation

5.4.3.1.2 Chronic Noncancer Dose for Soil Ingestion

The soil ingestion rate varies by age. A time-weighted average approach is used to combine soil intake rates of the age groupings (i.e., 0<2 yrs, 2<16 yrs, and 16-70 yrs) to determine the residential soil ingestion dose for chronic noncancer hazard assessment. This pathway is also assessed for exposure to offsite workers using the adult intake values for age 16-70 years in Table 5.14. Children at a MEIW daycare, if present, may also be assessed for exposure if the District deems it advisable. The contribution to the soil ingestion dose by each age group is determined in EQ 5.4.3.1.2:

A. Equation 5.4.3.1.2: $$DOSE_{soil} = C_{soil} \times GRAF \times SIR \times 10^{-9} \times ED/AT$$

1. $DOSE_{soil}$ = Dose from soil ingestion (mg/kg BW-day)
2. 10^{-9} = Conversion factor (μg to mg, kg to mg)
3. C_{soil} = Concentration of contaminant in soil (μg/kg)
4. GRAF = Gastrointestinal relative absorption fraction, unitless; chemical-specific
5. SIR = Soil ingestion rate (mg/kg BW-day)
6. ED = Exposure duration for a specified age group: 2 yrs for 0<2, 14 yrs for 2<16, 54 yrs for 16-70
7. AT = Averaging time for lifetime exposure – 70 yrs

a: Recommended default values for EQ 5.4.3.1.2:

1. C_{soil} = Calculated above in EQ 5.3.2 A
2. GRAF = See Table 5.2
3. SIR = See Table 5.14; use 16-70 age group SIR for workers

b: Recommended off-site worker default modifications to EQ 5.4.3.1.2:

1. A time-weighted average estimate of dose is not necessary and the ED and AT variates are left out of EQ 5.4.3.1.2 for oral soil dose to the worker.

c: Recommended nursing mother default modifications to EQ 5.4.3.1.2:

1. For mother's ingested soil dose to milk, use the SIR for age 16-30 years in Table 5.14.
2. The ED and AT variates in EQ 5.4.3.1.2 are left out for soil ingestion dose in the mother's milk pathway.

d: Assumptions for EQ 5.4.3.1.2:

1. For worker exposure, the annual average concentration should not be adjusted to account for overlap of worker and facility emission schedules. The pollutant will be deposited and accumulate in the soil in the absence or presence of the worker; therefore, the total deposition and soil concentration will be dependent on the annual average air concentration.

For residential exposure, the soil ingestion dose contribution for each age group is summed together to obtain the time-weighted average daily soil intake dose for chronic hazard assessment:

(SIR for age 0<2 yrs × C_{soil} × GRAF × 10^{-9} × 2 / 70) +

(SIR for age 2<16 yrs × C_{soil} × GRAF × 10^{-9} × 14 / 70) +

(SIR for age 16-70 yrs × C_{soil} × GRAF × 10^{-9} × 54 / 70) = Chronic $Dose_{soil}$

5.4.3.2 Exposure through Ingestion of Food

The exposure through food ingestion can be through ingestion of home-grown plant products (categorized as leafy, protected, exposed and root produce), home-raised animals (categorized as meat, cow's milk and eggs), angler-caught fish and mother's milk. When a specific food pathway is a dominant pathway (e.g. homegrown produce), and multiple pathways such as home raised meat, milk, and eggs categories all need to be assessed, the 95th percentile default consumption rate for the driving exposure pathway is used, while the mean consumption values for the remaining exposure pathways (i.e., food categories) are used. See Section 8.2.6 for a complete discussion of the methodology on how to implement the derived methodology.

5.4.3.2.1 Dose for Cancer Risk from Home-Grown Produce

Exposure through ingesting home-grown produce (DOSEp) is a function of the type of crop (i.e., exposed, leafy, protected, root), gastrointestinal absorption factor, bioavailability and the fraction of plant ingested that is homegrown. The calculation is

done for each type of crop, then summed to get total dose for this pathway. The exposure dose through ingestion home-grown produce varies by age and is calculated for each age group (e.g., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 and 16-70 yrs). These age-specific groupings are needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8).

A. Equation 5.4.3.2.1: $$DOSEp = C_v \times IP \times GRAF \times L \times EF \times 10^{-6}$$

1. DOSEp = Exposure dose through ingestion of home-grown produce (mg/kg/d)
2. C_v = Concentration in specific type of crop, i.e., exposed, leafy, protected, root (μg/kg)
3. IP = Consumption of specific type of crop (g/kg BW*day)
4. GRAF = Gastrointestinal relative absorption factor (unitless)
5. L = Fraction of plant type consumed that is home-grown or locally grown (unitless)
6. EF = Exposure frequency (unitless, days/365 days)
7. 10^{-6} = Conversion factors (μg/kg to mg/g)

a: Recommended default values for Equation 5.4.3.2.1:

1. C_v = Calculated above in EQ 5.3.4.1 A
2. IP = See Table 5.15 (point estimates) and 5.16a-e (distributions)
3. GRAF = GRAF = See Table 5.2
4. L = Site-specific survey is recommended. Otherwise, see Table 5.17 for Tier I default values
5. EF = 0.96 (350 d/365 d in a yr)

Once the dose for each type of crop that applies is calculated (See Section 5.3.4.1 for definition of crops types), the doses are summed to get the total dose for the home-grown produce pathway:

Total DOSEp = DOSEp (leafy) + DOSEp (root) + DOSEp (exposed) + DOSEp (protected)

The total home-grown produce dose will need to be calculated for each age group that applies.

5.4.3.2.2 Dose for cancer risk from home-raised meat, eggs, and cow's milk

Exposure through ingesting home-raised or farm animal products ($DOSE_{fa}$) is a function of the type of food (meat, eggs and cow's milk), gastrointestinal absorption factor, bioavailability and the fraction of food ingested that is home-raised. The only meat sources considered here are beef, pork and poultry. Unlike the home-grown produce pathway, the dose is calculated and presented separately for each type of home-raised food. The age-specific groupings to determine dose (3rd trimester, 0<2 yrs, 2<9 yrs,

2<16 yrs, 16<30 yrs or 16-70 yrs) is needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8).

A. Equation 5.4.3.2.2:

$$DOSE_{fa} = C_{fa} \times I_{fa} \times GRAF \times L \times EF \times 10^{-6}$$

1. $DOSE_{fa}$ = Exposure dose through ingestion of home-raised animal product (mg/kg/d)
2. C_{fa} = Concentration in animal product, e.g., beef, pork, poultry, diary, eggs (μg/kg)
3. I_{fa} = Consumption of animal product (g/kg BW-day)
4. GRAF = Gastrointestinal relative absorption factor (unitless)
5. L = Fraction of animal product consumed that is home-raised or locally produced (unitless)
6. EF = Exposure frequency (unitless, days/365 days)
7. 10^{-6} = Conversion factors (μg/kg to mg/g)

a: Recommended default values for EQ 5.4.3.2.2:

1. C_{fa} = Calculated above in EQ 5.3.4.2 A
2. I_{fa} = See Table 5.15 (point estimates) and Table 5.16a-e (distributions)
3. GRAF = See Table 5.2
4. L = Site-specific survey is recommended. Otherwise, see Table 5.17 for Tier I default values
5. EF = 0.96 (350 days / 365 days in a year)

5.4.3.2.3 Chronic Noncancer Dose for Ingestion of Food

For oral noncancer hazard assessment, a time-weighted average approach is used to combine food ingestion rates for the age groups (i.e., 0<2, 2<16 and 16-70 yrs) to estimate the chronic dose for residential exposure. The equation used to estimate dose through home-grown produce and home-raised meat/eggs/cow's milk are similar and is shown below in one equation. Similar to the cancer risk dose calculation, home-grown produce is presented as a total dose for all types of crops (See Section 5.4.3.2.1) and home-raised animal product dose is presented separately for each type of animal product that applies (See Section 5.4.3.2.2).

The contribution to the food intake dose is determined for each age group in EQ 5.4.3.2.3:

A. Equation 5.4.3.2.3: $DOSE_{food} = C_{food} \times I_{food} \times GRAF \times L \times 10^{-6} \times ED/AT$

1. $DOSE_{food}$ = Exposure dose through ingestion of home-grown produce or home-raised animal product (mg/kg/d)
2. C_{food} = Concentration (μg/kg) in produce (e.g., exposed, leafy, protected, root) or animal product (e.g., beef, pork, poultry, diary, eggs)
3. I_{food} = Consumption of produce or animal product (g/kg BW-day)
4. GRAF = Gastrointestinal relative absorption factor (unitless)
5. L = Fraction of produce or animal product consumed that is home-grown (unitless)
6. 10^{-6} = Conversion factors (μg/kg to mg/g)
7. ED = Exposure duration for a specified age group (2 yrs for 0<2, 14 yrs for 2<16, 54 yrs for 16-70
8. AT = Averaging time for lifetime exposure – 70 yrs

a: Recommended default values for EQ 5.4.3.2.3:

1. C_{food} = Calculated above in EQ 5.3.4.1 A (for home-grown produce) or EQ 5.3.4.2 A (for home-raised animal products)
2. I_{food} = Age-specific, see Table 5.15 for point estimates
3. GRAF = See Table 5.2
4. L = Site-specific survey is recommended. Otherwise, see Table 5.17 for Tier I default values

b: Recommended nursing mother default modifications to EQ 5.4.3.2.3:

1. For the mother's dose to milk through ingested food, use the food intake rates for age 16-30 years in Table 5.15 and 5.16d.
2. The ED and AT variates in EQ 5.4.3.2.3 are left out for ingested food dose in the mother's milk pathway.

Following calculation of the intake dose contributions for each age group, the intake rates for home-grown produce and the intake rates for home-raised animal products are summed separately to obtain the residential time-weighted average intake dose for chronic residential exposure to home-grown produce and to home-raised animal products:

(I_{food} for age 0<2 yrs × C_{food} × GRAF × L × 10^{-6} × 2 / 70) +

(I_{food} for age 2<16 yrs × C_{food} × GRAF × L × 10^{-6} × 14 / 70) +

(I_{food} for age 16-70 yrs × C_{food} × GRAF × L × 10^{-6} × 54 / 70) = Chronic $Dose_{food}$

Table 5.15 Recommended Average and High End Point Estimate Values for Home Produced Food Consumption (g/kg-day)

Food Category	Third Trimester		Ages 0<2		Ages 2<9	
Produce	Avg.	High End	Avg.	High End	Avg.	High End
Exposed	1.9	5.9	11.7	30.2	7.4	21.7
Leafy	0.9	3.2	3.8	10.8	2.5	7.9
Protected	1.7	5.8	5.9	17.5	4.7	13.3
Root	1.7	4.6	5.7	15.3	3.9	10.8
Meat						
Beef	2.0	4.8	3.9	11.3	3.5	8.6
Poultry	0.9	2.9	2.9	10.5	2.2	7.8
Pork	1.8	4.7	4.5	11.4	3.7	9.0
Milk	5.4	15.9	50.9	116	23.3	61.4
Eggs	1.6	4.2	6.1	15.0	3.9	9.4
	Ages 2>16		**Ages 16<30**		**Ages 16-70**	
Produce	Avg.	High End	Avg.	High End	Avg.	High End
Exposed	1.9	5.9	1.9	5.9	1.8	5.6
Leafy	0.9	3.2	0.9	3.2	1.1	3.4
Protected	1.7	5.8	1.7	5.8	1.6	5.2
Root	1.7	4.6	1.7	4.6	1.5	4.2
Meat						
Beef	2.0	4.8	2.0	4.8	1.7	4.4
Poultry	0.9	2.9	0.9	2.9	0.9	2.8
Pork	1.8	4.7	1.8	4.7	1.5	3.8
Milk	5.4	15.9	5.4	15.9	4.3	13.2
Eggs	1.6	4.2	1.6	4.2	1.3	3.4

[a] Food consumption values for 3rd trimester calculated by assuming that the fetus receives the same amount of contaminated food on a per kg BW basis as the mother (adult age 16 to less than 30).

Table 5.16a - e Parametric Models of Per Capita Food Consumption by Age Group for Stochastic Analysis

Table 5.16a Per Capita Food Consumption (g/kg-day) for 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 5.16b Per Capita Food Consumption (g/kg-day) for Ages 2<9

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape	Rate
Produce								
Exposed	Exponential	206						0.14
Leafy	LogN	127	2.64	3.89				
Protected	Weibull	68			0.02	4.76	1.063	
Root	LogN	60	3.95	3.85				
Meat								
Beef	LogN	35	3.55	2.79				
Poultry	LogN	17	3.71	2.67				
Pork	LogN	66	2.25	2.84				
Milk	LogN	12	23.4	20.78				
Eggs	LogN	38	3.93	3.00				

Table 5.16c Per Capita Food Consumption (g/kg-day) for Ages 2<16

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	Gamma	60			0.01	6.54	0.8325
Leafy	LogN	68	1.83	2.91			
Protected	Gamma	47			0.00	3.69	0.9729
Root	LogN	51	3.10	3.44			
Meat							
Beef	LogN	10	2.96	2.49			
Poultry	LogN	27	2.98	2.52			
Pork	LogN	48	1.84	2.79			
Milk	LogN	35	16.8	19.2			
Eggs	LogN	71	3.16	2.95			

Table 5.16d Per Capita Food Consumption (g/kg-day) for Ages 16-30[a]

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	Gamma	70			0.01	2.05	0.9220
Leafy	Weibull	191			0.00	0.88	0.8732
Protected	LogN	93	1.81	3.31			
Root	LogN	43	1.69	1.69			
Meat							
Beef	LogN	26	1.98	1.54			
Poultry	LogN	26	1.80	1.42			
Pork	LogN	242	1.01	1.74			
Milk	Gamma	22			0.02	5.66	0.9421
Eggs	LogN	29	1.55	1.36			

[a] These distributions are also recommended for the third trimester. Food consumption values for 3rd trimester are calculated by assuming that the fetus receives the same amount of contaminated food on a per kg BW basis as the mother (adult age 16<30).

Table 5.16e Per Capita Food Consumption (g/kg-day) for Ages 16-70

Food Category	Distribution Type	Anderson-Darling Statistic	Mean	Std. Dev	Location	Scale	Shape
Produce							
Exposed	Gamma	148			0.01	2.07	0.8628
Leafy	Gamma	83			0.00	1.15	0.9713
Protected	Gamma	78			0.01	1.90	0.8325
Root	Gamma	14			0.00	1.28	1.166
Meat							
Beef	LogN	20	1.75	1.40			
Poultry	LogN	18	1.53	1.18			
Pork	LogN	190	0.97	1.59			
Milk	Gamma	20			0.00	4.50	0.9627
Eggs	LogN	30	1.3	1.01			

Table 5.17 Default Values for L in EQs 5.4.3.2.1., 5.4.3.2.2 and 5.4.3.2.3: Fraction of Food Intake that is Home-Produced

Food Type	Households that Garden[a]	Households that Farm[a]
Avg. Total Veg & Fruits	0.137	0.235
	Households that Garden/Hunt[b]	**Households that Farm[b]**
Beef	0.485	0.478
Pork	0.242	0.239
Poultry	0.156	0.151
Eggs	0.146	0.214
Total Dairy (Cow's milk)	0.207	0.254

[a] As a default for home-produced leafy, exposed, protected and root produce, OEHHA recommends 0.137 as the fraction of produce that is home-grown. The households that grow their own vegetables and fruits are the population of concern. In rural situations where the receptor is engaged in farming, OEHHA recommends 0.235 as the default value for fraction of leafy, exposed, protected and root produce that is home-grown.

[b] OEHHA recommends the fraction home-raised under "Households that raise animals/hunt" (for beef, pork, poultry (chicken), eggs and dairy (cow's milk), with the exception of rural household receptors engaged in farming. OEHHA recommends that the fractions listed under "Households that farm" be used for the rural household receptors.

5.4.3.3 Exposure through Ingestion of Water

Intake of drinking water varies by age on a ml per kg body weight per day basis resulting in differences in exposure dose by age. The age-specific groupings to

determine dose is needed in order to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8) and to calculate a time-weighted average dose for chronic noncancer assessment.

5.4.3.3.1 Dose for Cancer Risk through Ingestion of Water

$DOSE_{water}$ is calculated for each age group (i.e., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 and 16-70 yrs), then incorporated into EQ 8.2.5 in Chapter 8 to determine cancer risk through exposure in drinking water.

A. Equation 5.4.3.3.1: $\mathbf{DOSE_{water} = C_w \times WIR \times ABS_{wa} \times F_{dw} \times EF \times 10^{-6}}$

1. $DOSE_{water}$ = Exposure dose through ingestion of water (mg/kg BW/d)
2. C_w = Water concentration (μg/L)
3. WIR = Water ingestion rate (ml/kg BW-day)
4. ABS_{wa} = Gastrointestinal absorption factor (unitless)
5. Fdw = Fraction of drinking water from contaminated source
6. EF = Exposure frequency (unitless, days/365 days)
7. 10^{-6} = Conversion factors (mg/μg)(L/ml)

a: Recommended default values for EQ 5.4.3.3.1:

1. C_w = Calculated above 5.3.3 A
2. WIR = See 5.18 (point estimates) and Table 5.19 (distributions)
3. ABS_{wa} = Default set to 1
4. Fdw = Default set to 1, although a site-specific survey is recommended for this variate
5. EF = 0.96 (350 days/365 days in a year)

5.4.3.3.2 Chronic Noncancer Dose through Ingestion of Water

Because water intake varies by age group, a time-weighted average intake approach is used to determine the daily water ingestion dose for chronic residential exposure. The contribution to the water ingestion dose is determined for each age group (i.e., 0<2, 2<16 and 16-70 yrs) in EQ 5.4.3.3.2.

A. Equation 5.4.3.3.2:

$$DOSE_{water} = C_w \times WIR \times ABS_{wa} \times Fdw \times 10^{-6} \times ED/AT$$

1. $DOSE_{water}$ = Exposure dose through ingestion of water (mg/kg BW/d)
2. C_w = Water concentration (μg/L)
3. WIR = Water ingestion rate (ml/kg BW-day)
4. ABS_{wa} = Gastrointestinal absorption factor
5. Fdw = Fraction of drinking water from contaminated source (site-specific)
6. 10^{-6} = Conversion factors (mg/μg)(L/ml)
7. ED = Exposure duration for a specified age group: 2 yrs for 0<2, 14 yrs for 2<16, 54 yrs for 16-70
8. AT = Averaging time for residential exposure – 70 yrs

a: Recommended default values for EQ 5.4.3.3.2:

1. C_w = Calculated above in 5.3.3 A
2. WIR = See 5.18 (point estimates)
3. ABS_{wa} = Default set to 1
4. Fdw = Default set to 1, although a site-specific survey is recommended for this variate

b: Recommended nursing mother default modifications to EQ 5.4.3.3.2:

1. For the dose to mother's milk through water ingestion, use the WIR for age 16-30 years in Table 5.18.
2. The ED and AT variates in EQ 5.4.3.3.2 are left out for ingested food dose in the mother's milk pathway.

The water intake dose contribution for each age group is summed together to obtain the time-weighted average daily residential water ingestion dose:

(WIR for age 0<2 yrs × C_w × ABS_{wa} × Fdw × 10^{-6} × 2 / 70) +

(WIR for age 2<16 yrs × C_w × ABS_{wa} × Fdw × 10^{-6} × 14 / 70) +

(WIR for age 16-70 yrs × C_w × ABS_{wa} × Fdw × 10^{-6} × 54 / 70) = Chronic $Dose_{water}$

Table 5.18 Recommended Point Estimate Tap Water Intake Rates (ml/kg-day)

Point Estimates				
Using Mean Values	**For the Age Period**	**9-year scenario**	**30-year scenario**	**70-year scenario**
	3rd trimester	18	18	18
	0<2 years	113	113	113
	2<9 years	26	-	-
	2<16 years	-	24	24
	16-30 years	-	18	-
	16-70 years	-	-	18
Using 95th-percentile values	**For the Age Period**	**9-year scenario**	**30-year scenario**	**70-year scenario**
	3rd trimester	47	47	47
	0<2 years	196	196	196
	2<9 years	66	-	-
	2<16 years	-	61	61
	16-30 years	-	47	-
	16-70 years	-	-	45

Table 5.19 Recommended Distributions of Tap Water Intake Rates (ml/kg-day) for Stochastic Risk Assessment

	9-year scenario	30-year scenario	70-year scenario
0<2 years	**Max Extreme** Likeliest = 93 Scale = 35	**Max Extreme** Likeliest = 93 Scale = 35	**Max Extreme** Likeliest = 93 Scale = 35
2<9 years	**Weibull** Location = 0.02 Scale = 29 Shape = 1.3		
2<16 years		**Gamma** Location = 0.19 Scale = 15.0 Shape = 1.6	**Gamma** Location = 0.19 Scale = 15.0 Shape = 1.6
16-30 years		**Gamma** location=0.49 scale=13.6 shape=1.26	
16-70 years			**Beta** min=0.17 max=178 alpha=1.5 beta= 12.9

5.4.3.4 Exposure through Ingestion of Angler-caught Fish

Exposure through ingestion of angler-caught fish ($DOSE_{fish}$) is a function of the fraction of fish ingested that is caught in the exposed water body, which differs for each age grouping, and the gastrointestinal absorption factor. Ingestion of angler-caught fish on a mg/kg body weight per day basis varies by age resulting in differences in exposure dose by age. The age-specific groupings to determine dose is needed primarily to properly use the age sensitivity factors for cancer risk assessment (see Chapter 8) and to calculate a time-weighted average dose for chronic noncancer assessment.

5.4.3.4.1 Cancer Risk Dose via Ingestion of Angler-Caught Fish

$DOSE_{fish}$ is calculated for each age group separately (i.e., 3rd trimester, 0<2 yrs, 2<9 yrs, 2<16 yrs, 16<30 and 16-70 yrs), then incorporated into EQ 8.2.5 in Chapter 8 to determine cancer risk through exposure to angler-caught fish.

A. Equation 5.4.3.4.1:

$$DOSE_{fish} = C_t \times I_{fish} \times Gf \times L \times EF \times 10^{-6}$$

1. $DOSE_{fish}$ = Dose via ingestion of angler-caught fish (mg/kg BW-day)
2. Ct = Concentration in fish muscle tissue (μg/kg)
3. I_{fish} = Angler-caught fish ingestion rate (g/kg BW per day)
4. Gf = Gastrointestinal absorption factor (unitless)
5. L = Fraction of fish caught at exposed site (unitless)
6. EF = Exposure frequency (days/365 days)
7. 10^{-6} = Conversion factor (mg/μg, kg/g)

a: Recommended default values for Equation 5.4.3.4.1:

1. Ct = Calculated above in Equation 5.3.4.7
2. I_{fish} = See Table 5.20 (point estimates) and Table 5.21 (distributions)
3. Gf = Default set to 1
4. L = Default set to 1 for fraction of fish caught locally, although a site-specific survey is recommended for this variate
5. EF = 0.96 (350 days/365 days in a yr)

5.4.3.4.2 Chronic Noncancer Dose via Ingestion of Angler-Caught Fish

Angler-caught fish consumption varies by age group. A time-weighted average intake for residential consumption over 70 years is used to determine dose for average and high-end exposure. The contribution to the angler-caught fish consumption dose is determined for each age group in EQ 5.4.3.4.2:

A. Equation 5.4.3.4.2: $DOSE_{fish} = C_t \times I_{fish} \times Gf \times L \times 10^{-6} \times ED/AT$

1. $DOSE_{fish}$ = Dose via ingestion of angler-caught fish (mg/kg BW-day)
2. C_t = Concentration in fish muscle tissue (μg/kg)
3. I_{fish} = Angler-caught fish ingestion rate (g/kg BW per day)
4. Gf = Gastrointestinal absorption factor (unitless)
5. L = Fraction of fish caught at exposed site (unitless)
6. 10^{-6} = Conversion factor (mg/μg, kg/g)
7. ED = Exposure duration for a specified age group: 2 yrs for 0<2, 14 yrs for 2<16 and 54 yrs for 16-70
8. AT = Averaging time for chronic exposure – 70 yrs

a: Recommended default values for Equation 5.4.3.4.2:

1. C_t = Calculated above in Equation 5.3.4.7
2. I_{fish} = See Table 5.20 (point estimates)
3. Gf = Default set to 1
4. L = Default set to 1 for fraction of fish caught locally, although a site-specific survey is recommended for this variate

b: Recommended nursing mother default modifications to EQ 5.4.3.4.2:

1. For the dose to mother's milk through fish consumption, use the I_{fish} for age 16-30 years in Table 5.20.
2. The ED and AT variates in EQ 5.4.3.4.2 are left out for the dose via fish consumption in the mother's milk pathway.

Following calculation of the angler-caught fish consumption dose contribution for each age group, 0<2 yr, 2<16 yr and 16-70 yr fish consumption doses are summed together to obtain the residential chronic dose:

(I_{fish} for age 0<2 yrs × C_t × Gf × L × 10^{-6} × 2 / 70) +

(I_{fish} for age 2<16 yrs × C_t × Gf × L × 10^{-6} × 14 / 70) +

(I_{fish} for age 16-70 yrs × C_t × Gf × L × 10^{-6} × 54 / 70) = Chronic $Dose_{fish}$

Table 5.20 Point Estimate Values for Angler-Caught Fish Consumption (g/kg-day) by Age Group

	Third Trimester	0 <2 Years	2<9 Years	2<16 Years	16<30 Years	16-70 Years
Mean	0.38	0.18	0.36	0.36	0.38	0.36
95th Percentile	1.22	0.58	1.16	1.16	1.22	1.16

Table 5.21 Empirical distribution for Angler-Caught fish consumption (g/kg-day)

Mean	Percentile									
	10th	20th	30th	40th	50th	60th	70th	80th	90th	95th
Third trimester, 2<9, 2<16, 16<30 and 16-70-year age groups										
0.36	0.06	0.09	0.12	0.16	0.21	0.27	0.36	0.50	0.79	1.16
0<2-year age group										
0.18	0.03	0.05	0.06	0.08	0.11	0.14	0.18	0.25	0.40	0.58

5.4.3.5 Mother's Milk

Exposure through mother's milk ingestion (Dose-Im) is a function of the average concentration of the substance in mother's milk and the amount of mother's milk ingested. The minimum pathways that the nursing mother is exposed to include inhalation, soil ingestion, and dermal, since the chemicals evaluated by the mother's milk pathway are multipathway chemicals. Other pathways may be appropriate depending on site conditions (e.g., the presence of vegetable gardens or home grown chickens). The compounds currently considered for the mother's milk pathway are:

1. Dioxins and Furans (PCDDS and PCDFs)
2. Polychlorinated biphenyls (PCBs)
3. Polycyclic Aromatic Hydrocarbons (PAHs), including creosotes
4. Lead

The nursing mother in the mother's milk pathway is not herself subject to the mother's milk pathway. The summed average daily dose (mg/kg BW-day) from all pathways is calculated for the nursing mother using the equations that follow.

5.4.3.5.1 Cancer Risk Dose to Infant via Mother's Milk

A. Equation 5.4.3.5.1: $$\text{Dose-Im} = C_m \times BMI_{bw} \times EF \times 10^{-3}$$

1. Dose-Im — Dose to infant through ingestion of mother's milk (mg/kg BW per day)
2. C_m = Concentration of contaminant in mother's milk (mg/kg milk)
3. BMI_{bw} = Daily breast-milk ingestion rate (g/kg BW-day)
4. EF = Frequency of exposure (days / 365 days)
5. 10^{-3} = Conversion factor (kg to g)

a: Recommended default values for EQ 5.4.3.5.1:

1. C_m = See EQ 5.3.4.8
2. BMI_{bw} = See Table 5.22 for point estimates. For distribution (parametric model) for Tier 3 stochastic risk assessments see Table 5.23.
3. EF = 1 (all 365 days of the first year of birth)

b: Assumptions for EQ 5.4.3.5.1:

8. For the MEIR, mother is exposed from birth up to 25 years of age when the infant is born. The exposed infant is then fully breastfed only during the first year of life.
9. For cancer risk assessment, exposure of breast-feeding infants to contaminants in breast milk applies only to the first year of the 0<2 yr age group for calculation of risk to this group, which then can be summed with the risk calculated for the other age groups (See Chapter 8).

5.4.3.5.2 Chronic Noncancer Dose to Infant via Mother's Milk

For oral noncancer hazard assessment, exposure of the infant through mother's milk ingestion occurs during the first year of life. After one year of age, the mother's milk pathway is not a factor for noncancer assessment.

A. Equation 5.4.3.5.2: $\text{Dose-Im} = C_m \times BMI_{bw} \times 10^{-3}$

1. Dose-Im Dose to infant through ingestion of mother's milk (mg/kg BW/d)
2. C_m = Concentration of contaminant in mother's milk (mg/kg milk)
3. BMI_{bw} = Daily breast-milk ingestion rate (g/kg BW-day)
4. 10^{-3} = Conversion factor (kg to g)

a: Recommended default values for EQ 5.4.3.5.2:

1. C_m = See EQ 5.3.4.8
2. BMI_{bw} = See Table 5.22 for point estimates

Table 5.22 Default Point Estimates for Breast Milk Intake (BMI_{bw}) for Breastfed Infants

Infant Group	Intake (g/kg-day)
Fully breastfed over the first year (i.e., fed in accordance with AAP recommendations)	
Mean	101
95th percentile	139

Table 5.23 Recommended Distribution of Breast Milk Intake Rates Among Breastfed Infants for Stochastic Assessment* (Averaged Over an Individual's First Year of Life)

	Mean (SD)	Percentile							
		5	10	25	50	75	90	95	99
Intake (g/kg-day)	101 (23)	62	71	85	101	116	130	139	154

* For stochastic analysis, the mother's milk data is normally distributed.

5.5 References

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6 - Dose-Response Assessment for Noncarcinogenic Endpoints

6.1 Derivation of Toxicity Criteria for Noncancer Health Effects

Dose-response assessment describes the quantitative relationship between the amount of exposure to a substance (the dose) and the incidence or occurrence of an adverse health impact (the response). Dose-response information for noncancer health effects is used to determine Reference Exposure Levels (RELs). Inhalation RELs are air concentrations or doses at or below which adverse noncancer health effects are not expected even in sensitive members of the general population under specified exposure scenarios. The acute RELs are for infrequent 1 hour exposures that occur no more than once every two weeks in a given year, although this time frame of exposure does not necessarily apply to chemicals that can bioaccumulate (e.g., dioxins and furans, PCBs, and various metals). The chronic RELs are for 24 hour per day exposures for at least a significant fraction of a lifetime, defined as about 8 years (≥12 percent of a 70-year lifespan). The 8-hour RELs are for repeated 8-hour exposures for a significant fraction of a lifetime such as the exposures that offsite workers might typically receive. Eight-hour RELs are only available for 10 chemicals at present, but OEHHA will develop 8-hour RELs as we re-evaluate our existing RELs to ensure they are protective of children's health, and as we develop RELs for new chemicals. There are oral chronic RELs for some chemicals in the Hot Spots program that are semivolatile or nonvolatile and thus subject to deposition and oral ingestion or dermal exposure. The methodology for developing RELs is similar to that used by U.S. EPA in developing the inhalation Reference Concentrations (RfCs) and oral Reference Doses (RfDs).

The first step in determining an acute, 8-hour, or chronic REL is to determine a point of departure. The point of departure is preferably determined by the benchmark concentration procedure applied to human or animal studies, but if this method of calculation cannot be used with a particular data set, a no observed adverse effect level (NOAEL) or lowest observed adverse effect level (LOAEL) may be used as the point of departure. The benchmark concentration method (also referred to as the benchmark dose method for oral exposures) is a preferred method to estimate a point of departure because it takes all of the available dose-response data into account to statistically estimate, typically, a 5 percent response rate.

Dosimetric of toxicokinetic adjustments are often made to the point of departure to adjust for differences in dosimetry or kinetics across species or among humans. Time adjustments are generally applied to adjust experimental exposure to the exposure of interest for the REL (e.g., 1 hour for acute, continuous for chronic). A modified Haber's equation is used where needed to adjust studies with different exposure times to the one-hour period needed for acute RELs. A simple Haber's law (C x T) adjustment for exposure period duration is used for most 8-hour and chronic RELs.

The time and dosimetry adjusted point of departure is divided by uncertainty factors that reflect the limitations in the current toxicology of the chemical. For example, an interspecies uncertainty factor is applied to account for the differences between humans and animals when an animal study is used. An intraspecies uncertainty factor is usually included to account for differences in susceptibility among the human population. In addition, where benchmark dose modeling is not suitable and a NOAEL is not available, a LOAEL to NOAEL uncertainty factor may be applied when the LOAEL serves as the point of departure. If a chronic study is not available to serve as a basis for a chronic REL, than a subchronic uncertainty factor (for chronic and 8-hour RELs only) may also be applied. Finally, if there are data deficiencies, for example, lack of a developmental toxicity study for a chemical, then a database deficiency factor may be applied. The individual uncertainty factors, which range from 2 to 10 depending on the limitations in the data, are multiplied together for a total uncertainty factor. The point of departure is then divided by the total UF to obtain the REL.

The most sensitive toxicological end point is selected as the basis for the REL when there are multiple adverse health effects. The selection of the most sensitive endpoint as the basis for a REL helps ensure that the REL is protective for all health effects. The use of uncertainty factors helps ensure that the REL is protective for nearly all individuals, including sensitive subpopulations, within the limitations of current scientific knowledge. For detailed information on the methodology and derivations for RELs, including guidance on selection of uncertainty factors, see the Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document for the Derivation of Noncancer Reference Exposure Levels (OEHHA, 2008).

It should be emphasized that exceeding the acute or chronic REL does not necessarily indicate that an adverse health impact will occur. The REL is not the threshold where population health effects would first be seen. However, levels of exposure above the REL have an increasing but undefined probability of resulting in an adverse health impact, particularly in sensitive individuals (e.g., depending on the toxicant, the very young, the elderly, pregnant women, and those with acute or chronic illnesses). The significance of exceeding the REL is dependent on the seriousness of the health endpoint, the strength and interpretation of the health studies, the magnitude of combined safety factors, and other considerations. In addition, there is a possibility that a REL may not be protective of certain small, unusually sensitive human subpopulations. Such subpopulations can be difficult to identify and study because of their small numbers, lack of knowledge about toxic mechanisms, and other factors. It may be useful to consult OEHHA staff when an REL is exceeded (hazard quotient or hazard index is greater than 1.0). Chapter 8 discusses the methods used for determining potential noncancer health impacts and Appendix I presents example calculations used to determine a hazard quotient (HQ) and hazard indices (HI).

Tables 6.1 through 6.3 list the currently adopted acute, 8-hour, and chronic inhalation RELs. Some substances that pose a long-term inhalation hazard may also present a chronic hazard via non-inhalation (oral, dermal) routes of exposure. The oral RELs for these substances are presented in Table 6.3. Appendix L provides a consolidated listing of all the acute, 8-hour, and chronic RELs with the respective target organs that

are approved for use by OEHHA and ARB for the Hot Spots Program. Periodically, new or updated RELs are adopted by OEHHA and these guidelines will be updated to reflect those changes. See OEHHA's web site at *www.oehha.ca.gov* (look under "Air", then select "Hot Spots Guidelines") to determine if any new or updated RELs have been adopted since the last guideline update.

6.2 Acute Reference Exposure Levels

OEHHA developed acute RELs for assessing potential noncancer health impacts for short-term, one-hour peak exposures to facility emissions (OEHHA, 2008; http://www.oehha.ca.gov/air/allrels.html). By definition, an acute REL is an exposure that is not likely to cause adverse health effects in a human population, including sensitive subgroups, exposed to that concentration (in units of micrograms per cubic meter or $\mu g/m^3$) for the specified exposure duration on an intermittent basis.

The target organ systems and the acute RELs for each substance are presented in Table 6.1. Many acute RELs are based on mild adverse effects, such as mild irritation of the eyes, nose, or throat, or may result in other mild adverse physiological changes. For most individuals, it is expected that the mild irritation and other adverse physiological changes will not persist after exposure ceases. For RELs that have been recently developed or revised, the notation "sensory irritation" has been added in parenthesis in Table 6.1 for those chemicals that have an acute REL based on sensory irritation of the respiratory system (i.e., nose , throat) and/or eyes.

Other acute RELs are based on reproductive/developmental endpoints, such as teratogenicity or fetotoxicity, which are considered severe adverse effects. The inhalation pathway is the only pathway to assess for acute exposure. Other non-inhalation pathways of exposure are evaluated for worker and residential scenarios where the exposures are chronic or repeated daily in nature. The oral RELs are used to evaluate the non-inhalation pathways of exposure. Noninhalation (oral) RELs are discussed in Section 6.5. Chapter 8 discusses the methods used for determining noncancer acute health impacts. Appendix I presents an example calculation used to determine an HQ and HI.

Table 6.1 Acute Inhalation Reference Exposure Levels (RELs) and Acute Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Acute Inhalation REL ($\mu g/m^3$)	Acute Hazard Index Target Organ Systems(s)
Acetaldehyde	75-07-0	$4.7 \times 10^{+2}$	Eyes; Respiratory System (sensory irritation)
Acrolein	107-02-8	$2.5 \times 10^{+0}$	Eyes; Respiratory System (sensory irritation)
Acrylic Acid	79-10-7	$6.0 \times 10^{+3}$	Eyes; Respiratory System
Ammonia	7664-41-7	$3.2 \times 10^{+3}$	Eyes; Respiratory System
Arsenic and Inorganic Arsenic Compounds (including arsine)	7440-38-2	2.0×10^{-1}	Development; Cardiovascular System; Nervous System
Benzene	71-43-2	$2.7 \times 10^{+1}$	Reproductive/Developmental; Immune System; Hematologic System
Benzyl Chloride	100-44-7	$2.4 \times 10^{+2}$	Eyes; Respiratory System
1,3-Butadiene	106-99-0	$6.6 \times 10^{+2}$	Development
Caprolactam	105-60-2	$5.0 \times 10^{+1}$	Eyes (sensory irritation)
Carbon Disulfide	75-15-0	$6.2 \times 10^{+3}$	Nervous System; Reproductive/Developmental
Carbon Monoxide [a]	630-08-0	$2.3 \times 10^{+4}$	Cardiovascular System
Carbon Tetrachloride	56-23-5	$1.9 \times 10^{+3}$	Alimentary System (Liver); Nervous System Reproductive/Developmental
Chlorine	7782-50-5	$2.1 \times 10^{+2}$	Eyes; Respiratory System
Chloroform	67-66-3	$1.5 \times 10^{+2}$	Nervous System; Respiratory System; Reproductive/Developmental
Chloropicrin	76-06-2	$2.9 \times 10^{+1}$	Eyes; Respiratory System
Copper and Compounds	7440-50-8	$1.0 \times 10^{+2}$	Respiratory System
1,4-Dioxane	123-91-1	$3.0 \times 10^{+3}$	Eyes; Respiratory System
Epichlorohydrin	106-89-8	$1.3 \times 10^{+3}$	Eyes; Respiratory System
Ethylene Glycol Monobutyl Ether	111-76-2	$1.4 \times 10^{+4}$	Eyes; Respiratory System
Ethylene Glycol Monoethyl Ether	110-80-5	$3.7 \times 10^{+2}$	Reproductive/Developmental
Ethylene Glycol Monoethyl Ether Acetate	111-15-9	$1.4 \times 10^{+2}$	Nervous System; Reproductive/Developmental
Ethylene Glycol Monomethyl Ether	109-86-4	$9.3 \times 10^{+1}$	Reproductive/Developmental
Formaldehyde	50-00-0	$5.5 \times 10^{+1}$	Eyes (sensory irritation)
Hydrogen Chloride	7647-01-0	$2.1 \times 10^{+3}$	Eyes; Respiratory System
Hydrogen Cyanide	74-90-8	$3.4 \times 10^{+2}$	Nervous System
Hydrogen Fluoride	7664-39-3	$2.4 \times 10^{+2}$	Eyes; Respiratory System
Hydrogen Selenide	7783-07-5	$5.0 \times 10^{+0}$	Eyes; Respiratory System
Hydrogen Sulfide [a]	7783-06-4	$4.2 \times 10^{+1}$	Nervous System
Isopropanol	67-63-0	$3.2 \times 10^{+3}$	Eyes; Respiratory System
Mercury and Inorganic Mercury Compounds	7439-97-6	6.0×10^{-1}	Nervous System; Development
Methanol	67-56-1	$2.8 \times 10^{+4}$	Nervous System
Methyl Bromide	74-83-9	$3.9 \times 10^{+3}$	Nervous System; Respiratory System; Reproductive/Developmental

Substance	Chemical Abstract Service Number (CAS)	Acute Inhalation REL ($\mu g/m^3$)	Acute Hazard Index Target Organ Systems(s)
Methyl Chloroform	71-55-6	$6.8 \times 10^{+4}$	Nervous System
Methyl Ethyl Ketone	78-93-3	$1.3 \times 10^{+4}$	Eyes; Respiratory System
Methylene Chloride	75-09-2	$1.4 \times 10^{+4}$	Nervous System; Cardiovascular System
Nickel and Nickel Compounds	7440-02-0	2.0×10^{-1}	Immune System
Nitric Acid	7697-37-2	$8.6 \times 10^{+1}$	Respiratory System
Nitrogen Dioxide [a]	10102-44-0	$4.7 \times 10^{+2}$	Respiratory System
Ozone [a]	10028-15-6	$1.8 \times 10^{+2}$	Eyes; Respiratory System
Perchloroethylene (Tetrachloroethylene) [a]	127-18-4	$2.0 \times 10^{+4}$	Eyes; Nervous System; Respiratory System
Phenol	108-95-2	$5.8 \times 10^{+3}$	Eyes; Respiratory System
Phosgene	75-44-5	$4.0 \times 10^{+0}$	Respiratory System
Propylene Oxide	75-56-9	$3.1 \times 10^{+3}$	Eyes; Respiratory System; Reproductive/Developmental
Sodium Hydroxide	1310-73-2	$8.0 \times 10^{+0}$	Eyes; Skin; Respiratory System
Styrene	100-42-5	$2.1 \times 10^{+4}$	Eyes; Respiratory System; Reproductive/Developmental
Sulfates [a]	N/A	$1.2 \times 10^{+2}$	Respiratory System
Sulfur Dioxide [a]	7446-09-5	$6.6 \times 10^{+2}$	Respiratory System
Sulfuric Acid and Oleum	7664-93-9 8014-95-7	$1.2 \times 10^{+2}$	Respiratory System
Tetrachloroethylene [a] (Perchloroethylene)	127-18-4	$2.0 \times 10^{+4}$	Eyes; Nervous System; Respiratory System
Toluene	108-88-3	$3.7 \times 10^{+4}$	Nervous System; Respiratory System; Eyes; Reproductive/Developmental
Triethylamine	121-44-8	$2.8 \times 10^{+3}$	Nervous System; Eyes
Vanadium Pentoxide	1314-62-1	$3.0 \times 10^{+1}$	Eyes; Respiratory System
Vinyl Chloride	75-01-4	$1.8 \times 10^{+5}$	Nervous System; Eyes; Respiratory System
Xylenes (m,o,p-isomers)	1330-20-7	$2.2 \times 10^{+4}$	Eyes; Respiratory System; Nervous System

[a] California Ambient Air Quality Standard

6.3 8-hour Reference Exposure Levels

OEHHA has developed 8-hour RELs for assessing potential noncancer health impacts for exposures to the general public that occur on a recurrent basis, but only during a portion of each day (OEHHA, 2008; http://www.oehha.ca.gov/air/allrels.html). Eight-hour RELs are compared to air concentrations that represent an average (daily) 8-hour exposure. They were designed to address off-site worker exposure at the MEIW, but may also be used at the Districts discretion to characterize 8-hour residential noncancer exposures, particularly for non-continuous facility operations where exposure is based on air concentrations during facility operation (i.e., the zero emission hours are not included) rather than averaged over 24-hours/day, 7 days/week as assessed for chronic exposure. The 8-hour RELs can also be used to assess exposure of students and teachers while at school (OEHHA, 2008). These RELs were developed because of concerns that applying the chronic REL in some scenarios was overly conservative. By

definition, an 8-hour REL is an exposure that is not likely to cause adverse health effects in a human population, including sensitive subgroups, exposed to that concentration (in units of micrograms per cubic meter or $\mu g/m^3$) for an 8-hour exposure duration on a regular (including daily) basis.

The RELs, target organ systems, and the averaging time for substances that can present a potential hazard from inhalation for 8 hours on a daily basis are presented in Table 6.2. Chapter 8 discusses the methods used for determining noncancer 8-hour health impacts. Appendix I presents an example calculation used to determine an HQ and HI.

Any substances in Table 6.2 with Development or Reproductive System as a target organ system are represented in HARP and in the Appendix L REL tables under the single endpoint "Reproductive/Development".

Table 6.2 Eight-Hour Inhalation Reference Exposure Levels (RELs) and 8-Hour Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Acetaldehyde	75-07-0	$3.0 \times 10^{+2}$	Respiratory System
Acrolein	107-02-8	7.0×10^{-1}	Respiratory System
Arsenic & Inorganic Arsenic Compounds	7440-38-2	1.5×10^{-2}	Cardiovascular System; Development [a]; Nervous System; Respiratory System; Skin
Benzene	71-43-2	$3.0 \times 10^{+0}$	Hematologic System
1,3-Butadiene	106-99-0	$9.0 \times 10^{+0}$	Reproductive System
Caprolactam	105-60-2	$7.0 \times 10^{+0}$	Respiratory System
Formaldehyde	50-0-0	$9.0 \times 10^{+0}$	Respiratory System
Manganese & Manganese Compounds	7439-96-5	1.7×10^{-1}	Nervous System
Mercury & Inorganic Mercury Compounds	7439--97-6	6.0×10^{-2}	Nervous System; Development [a]; Kidney
Nickel & Nickel Compounds	7440-02-0	6.0×10^{-2}	Respiratory System; Immune System

6.4 Chronic Reference Exposure Levels

OEHHA has developed chronic RELs for assessing noncancer health impacts from long-term exposure. (OEHHA, 2008; see also http://www.oehha.ca.gov/air/allrels.html) A chronic REL is a concentration level (expressed in units of micrograms per cubic meter ($\mu g/m^3$) for inhalation exposure and in a dose expressed in units of milligrams per kilogram-day (mg/kg-day) for oral exposures) at or below which no adverse health effects are anticipated following long-term exposure. Long-term exposure for these purposes has been defined by U.S. EPA as at least 12% of a lifetime, or about eight years for humans. Table 6.3 lists the chronic noncancer RELs that should be used in

the assessment of chronic health effects from inhalation exposure. Appendix L provides a consolidated listing of all the acute, 8-hour and chronic RELs and target organs that are approved for use by OEHHA and ARB for the Hot Spots Program. Periodically, new or updated RELs are adopted by OEHHA. See OEHHA's web site http://www.oehha.ca.gov/air/allrels.html to determine if any new or updated RELs have been adopted since the last guideline update.

The organ system(s) associated with each chronic REL are also presented in Table 6.3. Any substances in Table 6.3 with Development or Reproductive System as a target organ system are represented in HARP and in the Appendix L REL tables under the single endpoint "Reproductive/Development". Chapter 8 discusses the methods used for determining potential noncancer health impacts and Appendix I presents example calculations used to determine a HQ and HI.

Table 6.3 Chronic Inhalation Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Acetaldehyde [a]	75-07-0	$1.4 \times 10^{+2}$	Respiratory System
Acrolein	107-02-8	3.5×10^{-1}	Respiratory System
Acrylonitrile	107-13-1	$5.0 \times 10^{+0}$	Respiratory System
Ammonia	7664-41-7	$2.0 \times 10^{+2}$	Respiratory System
Arsenic & Inorganic Arsenic Compounds	7440-38-2	1.5×10^{-2}	Cardiovascular System; Development; Nervous System; Respiratory System; Skin
Benzene	71-43-2	$3.0 \times 10^{+0}$	Hematologic System
Beryllium and Beryllium Compounds	7440-41-7	7.0×10^{-3}	Immune System; Respiratory System
1,3-Butadiene	106-99-0	$2.0 \times 10^{+0}$	Reproductive System
Cadmium and Cadmium Compounds	7440-43-9	2.0×10^{-2}	Kidney; Respiratory System
Caprolactam	105-60-2	$2.2 \times 10^{+0}$	Respiratory System
Carbon Disulfide	75-15-0	$8.0 \times 10^{+2}$	Nervous System; Reproductive System
Carbon Tetrachloride	56-23-5	$4.0 \times 10^{+1}$	Alimentary System (Liver); Development; Nervous System
Chlorine	7782-50-5	2.0×10^{-1}	Respiratory System
Chlorine Dioxide	10049-04-4	6.0×10^{-1}	Respiratory System
Chlorinated Dibenzo-*p*-dioxins [b]			
2,3,7,8-Tetrachlorodibenzo-*p*-dioxin [b]	1746-01-6	4.0×10^{-5}	Alimentary System (Liver); Development; Endocrine System; Hematologic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzo-*p*-dioxin [b]	40321-76-4	4.0×10^{-5}	
1,2,3,4,7,8-Hexachlorodibenzo-*p*-dioxin [b]	39227-28-6	4.0×10^{-4}	
1,2,3,6,7,8-Hexachlorodibenzo-*p*-dioxin [b]	57653-85-7	4.0×10^{-4}	
1,2,3,7,8,9-Hexachlorodibenzo-*p*-dioxin [b]	19408-74-3	4.0×10^{-4}	
1,2,3,4,6,7,8-Heptachlorodibenzo-*p*-dioxin [b]	35822-46-9	4.0×10^{-3}	
1,2,3,4,6,7,8,9-Octachlorodibenzo-*p*-dioxin [b]	3268-87-9	1.3×10^{-1}	

Table 6.3 Chronic Inhalation Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Chlorinated Dibenzofurans [b]			
2,3,7,8-Tetrachlorodibenzofuran [b]	5120-73-19	4.0×10^{-4}	Alimentary System (Liver); Development; Endocrine System; Hematologic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzofuran [b]	57117-41-6	1.3×10^{-3}	
2,3,4,7,8-Pentachlorodibenzofuran [b]	57117-31-4	1.3×10^{-4}	
1,2,3,4,7,8-Hexachlorodibenzofuran [b]	70648-26-9	4.0×10^{-4}	
1,2,3,6,7,8-Hexachlorodibenzofuran [b]	57117-44-9	4.0×10^{-4}	
1,2,3,7,8,9-Hexachlorodibenzofuran [b]	72918-21-9	4.0×10^{-4}	
2,3,4,6,7,8-Hexachlorodibenzofuran [b]	60851-34-5	4.0×10^{-4}	
1,2,3,4,6,7,8-Heptachlorodibenzofuran [b]	67562-39-4	4.0×10^{-3}	
1,2,3,4,7,8,9-Heptachlorodibenzofuran [b]	55673-89-7	4.0×10^{-3}	
1,2,3,4,6,7,8,9-Octachlorodibenzofuran [b]	39001-02-0	1.3×10^{-1}	
Chlorobenzene	108-90-7	$1.0 \times 10^{+3}$	Alimentary System (Liver); Kidney; Reproductive System
Chloroform	67-66-3	$3.0 \times 10^{+2}$	Alimentary System (Liver); Development; Kidney
Chloropicrin	76-06-2	4.0×10^{-1}	Respiratory System
Chromium VI & Soluble Chromium VI Compounds (except chromic trioxide)	18540-29-9	2.0×10^{-1}	Respiratory System
Chromic Trioxide (as chromic acid mist)	1333-82-0	2.0×10^{-3}	Respiratory System
Cresol Mixtures	1319-77-3	$6.0 \times 10^{+2}$	Nervous System
1,4-Dichlorobenzene	106-46-7	$8.0 \times 10^{+2}$	Alimentary System (Liver); Kidney; Nervous System; Respiratory System
1,1-Dichloroethylene (Vinylidene Chloride)	75-35-4	$7.0 \times 10^{+1}$	Alimentary System (Liver)
Diesel Exhaust [a]	N/A	$5.0 \times 10^{+0}$	Respiratory System
Diethanolamine	111-42-2	$3.0 \times 10^{+0}$	Hematologic System; Respiratory System
N,N-Dimethylformamide	68-12-2	$8.0 \times 10^{+1}$	Alimentary System (Liver); Respiratory System
1,4-Dioxane	123-91-1	$3.0 \times 10^{+3}$	Alimentary System (Liver); Cardiovascular System; Kidney
Epichlorohydrin	106-89-8	$3.0 \times 10^{+0}$	Eyes; Respiratory System
1,2-Epoxybutane	106-88-7	$2.0 \times 10^{+1}$	Cardiovascular System; Respiratory System
Ethylbenzene	100-41-4	$2.0 \times 10^{+3}$	Alimentary System (Liver); Kidney; Development; Endocrine System
Ethyl Chloride	75-00-3	$3.0 \times 10^{+4}$	Alimentary System (Liver); Development
Ethylene Dibromide	106-93-4	8.0×10^{-1}	Reproductive System
Ethylene Dichloride	107-06-2	$4.0 \times 10^{+2}$	Alimentary System (Liver)
Ethylene Glycol	107-21-1	$4.0 \times 10^{+2}$	Development; Kidney; Respiratory System
Ethylene Glycol Monoethyl Ether	110-80-5	$7.0 \times 10^{+1}$	Hematologic System; Reproductive System
Ethylene Glycol Monoethyl Ether Acetate	111-15-9	$3.0 \times 10^{+2}$	Development

Table 6.3 Chronic Inhalation Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Ethylene Glycol Monomethyl Ether	109-86-4	$6.0 \times 10^{+1}$	Reproductive System
Ethylene Glycol Monomethyl Ether Acetate	110-49-6	$9.0 \times 10^{+1}$	Reproductive System
Ethylene Oxide	75-21-8	$3.0 \times 10^{+1}$	Nervous System
Fluorides (except hydrogen fluoride)	N/A	$1.3 \times 10^{+1}$	Bone and Teeth; Respiratory System
Formaldehyde	50-00-0	$9.0 \times 10^{+0}$	Respiratory System
Glutaraldehyde	111-30-8	8.0×10^{-2}	Respiratory System
Hexane (n-)	110-54-3	$7.0 \times 10^{+3}$	Nervous System
Hydrazine	302-01-2	2.0×10^{-1}	Alimentary System (Liver); Endocrine System
Hydrogen Chloride	7647-01-0	$9.0 \times 10^{+0}$	Respiratory System
Hydrogen Cyanide	74-90-8	$9.0 \times 10^{+0}$	Cardiovascular System; Endocrine System; Nervous System
Hydrogen Fluoride	7664-39-3	$1.4 \times 10^{+1}$	Bone and Teeth; Respiratory System
Hydrogen Sulfide	7783-06-4	$1.0 \times 10^{+1}$	Respiratory System
Isophorone	78-59-1	$2.0 \times 10^{+3}$	Alimentary System (Liver); Development
Isopropanol	67-63-0	$7.0 \times 10^{+3}$	Development; Kidney
Maleic Anhydride	108-31-6	7.0×10^{-1}	Respiratory System
Manganese & Manganese Compounds	7439-96-5	9.0×10^{-2}	Nervous System
Mercury & Inorganic Mercury Compounds	7439-97-6	3.0×10^{-2}	Nervous System; Development; Kidney
Methanol	67-56-1	$4.0 \times 10^{+3}$	Development
Methyl Bromide	74-83-9	$5.0 \times 10^{+0}$	Development; Nervous System; Respiratory System
Methyl Chloroform	71-55-6	$1.0 \times 10^{+3}$	Nervous System
Methyl Isocyanate	624-83-9	$1.0 \times 10^{+0}$	Reproductive System; Respiratory System
Methyl tertiary-Butyl Ether	1634-04-4	$8.0 \times 10^{+3}$	Alimentary System (Liver); Eyes; Kidney
Methylene Chloride	75-09-2	$4.0 \times 10^{+2}$	Cardiovascular System; Nervous System
4,4'-Methylene Dianiline (& its dichloride)	101-77-9	$2.0 \times 10^{+1}$	Alimentary System (Liver); Eyes
Methylene Diphenyl Isocyanate	101-68-8	7.0×10^{-1}	Respiratory System
Naphthalene	91-20-3	$9.0 \times 10^{+0}$	Respiratory System
Nickel & Nickel Compounds (except nickel oxide)	7440-02-0	1.4×10^{-2}	Hematologic System; Respiratory System
Nickel Oxide	1313-99-1	2.0×10^{-2}	Respiratory System
Perchloroethylene (Tetrachloroethylene)[a]	127-18-4	$3.5 \times 10^{+1}$	Alimentary System (Liver); Kidney
Phenol	108-95-2	$2.0 \times 10^{+2}$	Alimentary System (Liver); Cardiovascular System; Kidney; Nervous System
Phosphine	7803-51-2	8.0×10^{-1}	Alimentary System (Liver); Hematologic System; Kidney; Nervous System; Respiratory System

Table 6.3 Chronic Inhalation Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Inhalation Hazard Index Target Organ System(s)
Phosphoric Acid	7664-38-2	$7.0 \times 10^{+0}$	Respiratory System
Phthalic Anhydride	85-44-9	$2.0 \times 10^{+1}$	Respiratory System
Polychlorinated biphenyls (PCBs)[b]			
3,3',4,4'-Tetrachlorobiphenyl (77) [b]	35298-13-3	4.0×10^{-1}	Alimentary System (Liver); Developmental; Endocrine System; Hematologic System; Reproductive System; Respiratory System
3,4,4',5-Tetrachlorobiphenyl (81) [b]	70362-50-4	1.3×10^{-1}	
2,3,3',4,4'- Pentachlorobiphenyl (105) [b]	32598-14-4	$1.3 \times 10^{+0}$	
2,3,4,4'5- Pentachlorobiphenyl (114) [b]	74472-37-0	$1.3 \times 10^{+0}$	
2,3'4,4',5- Pentachlorobiphenyl (118) [b]	31508-00-6	$1.3 \times 10^{+0}$	
2',3,4,4',5- Pentachlorobiphenyl (123) [b]	65510-44-3	$1.3 \times 10^{+0}$	
3,3',4,4',5- Pentachlorobiphenyl (126) [b]	57465-28-8	4.0×10^{-4}	
2,3,3',4,4',5-Hexachlorobiphenyl (156) [b]	38380-08-4	$1.3 \times 10^{+0}$	
2,3,3',4,4',5'-Hexachlorobiphenyl (157) [b]	69782-90-7	$1.3 \times 10^{+0}$	
2,3',4,4',5,5'-Hexachlorobiphenyl (167) [b]	52663-72-6	$1.3 \times 10^{+0}$	
3,3',4,4'5,5'- Hexachlorobiphenyl (169) [b]	32774-16-6	1.3×10^{-3}	
2,3,3'4,4',5,5'-Heptachlorobiphenyl (189) [b]	39635-31-9	$1.3 \times 10^{+0}$	
Propylene	115-07-1	$3.0 \times 10^{+3}$	Respiratory System
Propylene Glycol Monomethyl Ether	107-98-2	$7.0 \times 10^{+3}$	Alimentary System (Liver)
Propylene Oxide	75-56-9	$3.0 \times 10^{+1}$	Respiratory System
Selenium and Selenium compounds (other than Hydrogen Selenide)	7782-49-2	$2.0 \times 10^{+1}$	Alimentary System (Liver); Cardiovascular System; Nervous System
Silica (crystalline, respirable)	N/A	$3.0 \times 10^{+0}$	Respiratory System
Styrene	100-42-5	$9.0 \times 10^{+2}$	Nervous System
Sulfuric Acid	7664-93-9	$1.0 \times 10^{+0}$	Respiratory System
Toluene	108-88-3	$3.0 \times 10^{+2}$	Development; Nervous System; Respiratory System
2,4-Toluene Diisocyanate	584-84-9	7.0×10^{-2}	Respiratory System
2,6-Toluene Diisocyanate	91-08-7	7.0×10^{-2}	Respiratory System
Trichloroethylene [a]	79-01-6	$6.0 \times 10^{+2}$	Eyes; Nervous System
Triethylamine	121-44-8	$2.0 \times 10^{+2}$	Eyes
Vinyl Acetate	108-05-4	$2.0 \times 10^{+2}$	Respiratory System
Xylenes (m, o, p-isomers)	1330-20-7	$7.0 \times 10^{+2}$	Nervous System; Respiratory System; Eyes

[a] These peer-reviewed values were developed under the Toxic Air Contaminant (TAC) Program mandated by AB1807 (California Health and Safety Code Sec. 39650 *et seq*.).

[b] The OEHHA has adopted the World Health Organization Toxicity Equivalency Factor (TEF) scheme for evaluating the cancer risk and noncancer hazard due to exposure to samples containing mixtures of polychlorinated dibenzo-*p*-dioxins (PCDD) (also referred to as chlorinated dioxins and dibenzofurans), polychlorinated dibenzofurans (PCDF) and polychlorinated biphenyls (PCBs). The TEF values are revised from time to time to reflect new data and increased scientific knowledge. Currently OEHHA recommends use of the 2005 revision to the WHO TEF values (WHO_{05}-TEF). See Appendix E for more information about the scheme and for the methodology for calculating 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) equivalents for PCDD and PCDFs. For

convenience, OEHHA has calculated chronic REL values for speciated PCDDs, PCDFs and PCBs based on the WHO_{05} TEF values and the chronic REL for 2,3,7,8-TCDD using the procedure discussed in Appendix E. The chronic REL values can be used to calculate a hazard index when the mixtures are speciated from individual congener ground level concentrations. In those cases where speciation of dioxins and furans has not been performed, then 2,3,7,8-TCDD serves as the surrogate for dioxin and furan emissions.

N/A Not Applicable

6.5 Chronic Oral (Noninhalation) Reference Exposure Levels

As specified throughout the guidelines, estimates of long-term exposure resulting from facility air emissions of specific compounds must be analyzed for both inhalation and noninhalation (multipathway) pathways of exposure for humans. Facilities often emit substances under high temperature and pressure in the presence of particulate matter. While some of these substances are expected to remain in the vapor phase, other substances such as metals and semi-volatile organics can be either emitted as particles, form particles after emission from the facility, or adhere to existing particles. Some substances will partition between vapor and particulate phases. Substances in the particulate phase can be removed from the atmosphere by settling and, thus, potentially present a significant hazard via noninhalation pathways.

Particulate-associated chemicals can be deposited directly onto soil, onto the leaves or fruits of crops, or onto surface waters. Exposure via the oral route is the predominant noninhalation pathway, resulting in the noninhalation RELs being referred to as ‘oral RELs’ in this document. The oral RELs are used for both ingestion and dermal exposures, and are applied using the chronic non-inhalation exposures in the residential scenario and the worker scenarios. The oral RELs are expressed as doses in milligrams of substance (consumed and dermally absorbed) per kilogram body weight per day (mg/kg-day).

Table 6.4 lists the chronic noncancer RELs to be used in the assessment of chronic health effects from noninhalation pathways of exposure. Any substances in Table 6.4 with Development or Reproductive System as a target organ system are represented in HARP and in the Appendix L REL tables under the single endpoint “Reproductive/Development”. Appendix L provides a consolidated listing of all chronic RELs and target organs that are approved for use by OEHHA and ARB for the Hot Spots Program. Periodically, new or updated RELs are adopted by OEHHA and these guidelines will be updated to reflect those changes. See OEHHA’s web page at http://www.oehha.ca.gov/air/allrels.html to determine if any new or updated RELs have been adopted since the last guideline update. Chapter 8 discusses the methods used for determining potential noncancer health impacts and Appendix I presents example calculations used to determine a HQ and HI.

Table 6.4 Chronic Noninhalation 'Oral' Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service No. (CAS)	Chronic Oral REL (mg/kg-day)	Chronic Oral Hazard Index Target Organ System(s)
Arsenic & Inorganic Arsenic Compounds	7440-38-2	3.5×10^{-6}	Development; Nervous System; Respiratory System; Cardiovascular System; Skin
Beryllium and Beryllium Compounds	7440-41-7	2.0×10^{-3}	Alimentary System (Gastrointestinal Tract)
Cadmium and Cadmium Compounds	7440-43-9	5.0×10^{-4}	Kidney
Chlorinated Dibenzo-*p*-dioxins [a]			
2,3,7,8-Tetrachlorodibenzo-*p*-dioxin [a]	1746-01-6	1.0×10^{-8}	Alimentary System (Liver); Developmental; Endocrine System; Hematologic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzo-*p*-dioxin [a]	40321-76-4	1.0×10^{-8}	
1,2,3,4,7,8-Hexachlorodibenzo-*p*-dioxin [a]	39227-28-6	1.0×10^{-7}	
1,2,3,6,7,8-Hexachlorodibenzo-*p*-dioxin [a]	57653-85-7	1.0×10^{-7}	
1,2,3,7,8,9-Hexachlorodibenzo-*p*-dioxin [a]	19408-74-3	1.0×10^{-7}	
1,2,3,4,6,7,8-Heptachlorodibenzo-*p*-dioxin [a]	35822-46-9	1.0×10^{-6}	
1,2,3,4,6,7,8,9-Octachlorodibenzo-*p*-dioxin [a]	3268-87-9	3.3×10^{-5}	
Chlorinated Dibenzofurans [a]			
2,3,7,8-Tetrachlorodibenzofuran [a]	5120-73-19	1.0×10^{-7}	Alimentary System (Liver); Development; Endocrine System; Hematologic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzofuran [a]	57117-41-6	3.3×10^{-7}	
2,3,4,7,8-Pentachlorodibenzofuran [a]	57117-31-4	3.3×10^{-8}	
1,2,3,4,7,8-Hexachlorodibenzofuran [a]	70648-26-9	1.0×10^{-7}	
1,2,3,6,7,8-Hexachlorodibenzofuran [a]	57117-44-9	1.0×10^{-7}	
1,2,3,7,8,9-Hexachlorodibenzofuran [a]	72918-21-9	1.0×10^{-7}	
2,3,4,6,7,8-Hexachlorodibenzofuran [a]	60851-34-5	1.0×10^{-7}	
1,2,3,4,6,7,8-Heptachlorodibenzofuran [a]	67562-39-4	1.0×10^{-6}	
1,2,3,4,7,8,9-Heptachlorodibenzofuran [a]	55673-89-7	1.0×10^{-6}	
1,2,3,4,6,7,8,9-Octachlorodibenzofuran [a]	39001-02-0	3.3×10^{-5}	
Chromium VI & Soluble Chromium VI Compounds (including chromic trioxide)	18540-29-9	2.0×10^{-2}	Hematologic System
Fluorides (including hydrogen fluoride)	7664-39-3	4.0×10^{-2}	Bone and Teeth
Mercury & Mercury Inorganic Compounds	7439-97-6	1.6×10^{-4}	Kidney; Nervous System; Development
Nickel & Nickel Compounds (including nickel oxide)	7440-02-0	1.1×10^{-2}	Development
Polychlorinated biphenyls (PCBs) (speciated)[a]			
3,3',4,4'-Tetrachlorobiphenyl (77)[a]	35298-13-3	1.0×10^{-4}	Alimentary System (Liver); Developmental; Endocrine System; Hematologic System; Reproductive System; Respiratory System
3,4,4',5-Tetrachlorobiphenyl (81)[a]	70362-50-4	3.3×10^{-5}	
2,3,3',4,4'- Pentachlorobiphenyl (105)[a]	32598-14-4	3.3×10^{-4}	
2,3,4,4'5- Pentachlorobiphenyl (114)[a]	74472-37-0	3.3×10^{-4}	
2,3'4,4',5- Pentachlorobiphenyl (118)[a]	31508-00-6	3.3×10^{-4}	
2',3,4,4',5- Pentachlorobiphenyl (123)[a]	65510-44-3	3.3×10^{-4}	
3,3',4,4',5- Pentachlorobiphenyl (126)[a]	57465-28-8	1.0×10^{-7}	
2,3,3',4,4',5-Hexachlorobiphenyl (156)[a]	38380-08-4	3.3×10^{-4}	
2,3,3',4,4',5'-Hexachlorobiphenyl (157)[a]	69782-90-7	3.3×10^{-4}	
2,3',4,4',5,5'-Hexachlorobiphenyl (167)[a]	52663-72-6	3.3×10^{-4}	
3,3',4,4'5,5'- Hexachlorobiphenyl (169)[a]	32774-16-6	3.3×10^{-7}	

Table 6.4 Chronic Noninhalation 'Oral' Reference Exposure Levels (RELs) and Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service No. (CAS)	Chronic Oral REL (mg/kg-day)	Chronic Oral Hazard Index Target Organ System(s)
2,3,3'4,4',5,5'- Heptachlorobiphenyl (189)[a]	39635-31-9	3.3×10^{-4}	
Selenium and Selenium Compounds (other than hydrogen selenide)	7782-49-2	5.0×10^{-3}	Alimentary system (Liver); cardiovascular system; nervous system

[a] The OEHHA has adopted the World Health Organization Toxicity Equivalency Factor (TEF) scheme for evaluating the cancer risk and noncancer risk due to exposure to samples containing mixtures of polychlorinated dibenzo-*p*-dioxins (PCDD) (also referred to as chlorinated dioxins and dibenzofurans), polychlorinated dibenzofurans (PCDF), and polychlorinated biphenyls (PCBs). The TEF values are revised from time to time to reflect new data and increased scientific knowledge. Currently OEHHA recommends use of the 2005 revision to the WHO TEF values (WHO_{05}-TEF). See Appendix E for more information about the scheme and for the methodology for calculating 2,3,7,8-equivalents for PCDD and PCDFs. For convenience, OEHHA has calculated chronic 'oral' REL values for speciated PCDDs, PCDFs, and PCBs based on the WHO_{05} TEF values and the chronic 'oral' REL for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin using the procedure discussed in Appendix E. The chronic 'oral' REL values can be used to calculate a hazard index when the mixtures are speciated from individual congener ground level concentrations. In those cases where speciation of dioxins and furans has not been performed, then 2,3,7,8-TCDD serves as the surrogate for dioxin and furan emissions.

6.6 References

OEHHA, 2008. Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. Available online at: http://www.oehha.ca.gov

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7 - Dose-Response Assessment for Carcinogens

7.1 Introduction

Dose-response assessment characterizes the quantitative relationship between the amount of exposure to a substance (the dose) and the incidence or occurrence of injury (the response). The process often involves establishing a toxicity value or criterion to use in assessing potential health risk. The toxicity criterion, or health guidance value, for carcinogens is the cancer potency slope (potency factor), which describes the potential risk of developing cancer per unit of average daily dose over a 70-year lifetime. Cancer inhalation and oral potency factors have been derived by the Office of Environmental Health Hazard Assessment (OEHHA) or by the United States Environmental Protection Agency (U.S. EPA) and approved by the State's Scientific Review Panel on Toxic Air Contaminants. They are available for many of the substances listed in Appendix A (List of Substances) as carcinogens. Table 7.1 and Appendix L list the inhalation and oral cancer potency factors that should be used in multipathway health risk assessments (HRAs) for the Hot Spots Program.

The details on the methodology of dose-response assessment for carcinogens and the approved cancer potency factors are provided in the Air Toxics Hot Spots Risk Assessment Guidelines. Part II. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. May, 2009. (OEHHA, 2009; see http://www.oehha.ca.gov/air/hot_spots/tsd052909.html).

7.2 Carcinogenic Potency

Cancer potency factors used for both the inhalation and oral routes in the Hot Spots program are generally the 95% upper confidence limits (UCL) on the modeled dose-response slope at the low dose range. The cancer slope factor assumes continuous lifetime exposure to a substance, and is expressed in units of inverse dose [i.e., $(mg/kg/day)^{-1}$]. Another common potency expression is in units of inverse concentration [$(\mu g/m^3)^{-1}$)] when the slope is based on exposure concentration rather than dose; this is termed the unit risk factor. To accommodate the use of age-specific exposure variates, the Hot Spots program has translated the unit risk factors based on concentration to units of inverse dose. This allows calculation of risk for age groupings, as exposure varies with age. It also allows for application of Age Sensitivity Factors for early life exposures.

It is assumed in cancer risk assessments that risk is directly proportional to dose and that, for most carcinogens, there is no threshold for carcinogenesis. The derivation of inhalation and oral cancer potency factors takes into account information on pharmacokinetics, when available, and on the mechanism of carcinogenic action.

Table 7.1 and Appendix L list inhalation and oral cancer potency factors that should be used in risk assessments for the Hot Spots Program. Chapter 8 describes procedures for use of potency factors in estimating potential cancer risk.

7.2.1 *Inhalation Cancer Potency Factor*

The risk assessment methodology and algorithms presented in Chapter 8 express the inhalation cancer slope factors in units of inverse dose (i.e., $(mg/kg/day)^{-1}$). Breathing rates, expressed in units of liters per kilogram of body weight-day (L/kg-day), are multiplied with the air concentrations, coupled with the appropriate unit conversion factor, to estimate dose in mg/kg-day. This allows estimation of average and high-end cancer risk point estimates. Estimation of a distribution of cancer risk based on variability in breathing rate can be obtained by Monte Carlo methods using the distributions of breathing rates in L/kg-day, which can then be converted to a dose distribution in mg/kg BW based on the intake rate. Unit risk factors [in the units of inverse concentration (i.e., $(\mu g/m^3)^{-1}$], which were used in previous guidelines for the Hot Spots program, are still listed in the TSD (OEHHA, 2009) and may prove useful in other risk assessment applications.

The average daily inhalation dose (mg/kg-day) multiplied by the cancer potency factor $(mg/kg\text{-}day)^{-1}$ will give the inhalation cancer risk (unitless), which is an expression of the chemical's cancer risk during a 70-year lifespan of exposure. For example, an inhalation cancer risk of 5×10^{-6} is the same as stating that an individual has an estimated probability of developing cancer from their exposure of 5 chances per million people exposed. A more complete description of how potential cancer risk is calculated from the exposure dose and cancer potency factors is provided in Chapter 8. Appendix I presents an example calculation for determining cancer risk.

A list of current inhalation potency factors is provided in Table 7.1. Periodically, new or revised cancer potency factors will be peer reviewed by the State's Scientific Review Panel on Toxic Air Contaminants (SRP) and adopted by the Director of OEHHA. For new or updated numbers, consult the OEHHA web site at (http://www.oehha.ca.gov/air/hot_spots/tsd052909.html) to determine if any new or updated cancer potency factors have been adopted since this guideline update. New cancer potency factors that have been approved by the SRP and adopted by the Director of OEHHA should be incorporated into Hot Spots risk assessment for facilities that emit those chemicals.

7.2.2 *Oral Cancer Potency Factor*

Under the Hot Spots Program, a few substances are evaluated for exposure and risk from non-inhalation pathways – these are referred to as multipathway substances. Multipathway substances have the potential to impact a receptor through inhalation and noninhalation (oral and dermal) exposure routes. These substances include heavy metals and semi-volatile organic substances such as dioxins, furans, and polycyclic aromatic hydrocarbons (PAHs). These substances commonly exist in the particle

phase or partially in the particle phase when emitted into the air. They can therefore be deposited onto soil, vegetation, and water. Noninhalation exposure pathways considered under the Hot Spots Program include the ingestion of soil, homegrown produce, meat, milk, surface water, breast milk, and fish as well as dermal exposure to contaminants deposited in the soil. See Table 5.1 for a list of the multipathway substances.

Table 7.1 and Appendix L list oral cancer potency factors in units of $(mg/kg\text{-}day)^{-1}$ that should be used for assessing the potential cancer risk for these substances through noninhalation exposure pathways. The cancer risk from these individual pathways is calculated by multiplying the dose (mg/kg-day) times the oral cancer potency factor $(mg/kg\text{-}day)^{-1}$ to yield the potential cancer risk (unitless) from non-inhalation exposures. Chapter 5 provides all of the algorithms to calculate exposure dose through all of the individual exposure pathways. Appendix I provides a sample calculation for dose and cancer risk using the inhalation exposure pathway.

Three carcinogens (cadmium, beryllium, and nickel), although subject to deposition, are only treated as carcinogenic by the inhalation route and not by the oral route. Therefore, there are no oral cancer potency factors for these substances. However, the oral doses of these substances need to be estimated because of their noncancer toxicity. See Chapters 6 and 8, and Appendices I and L for dose-response factors, and calculations to address these substances.

Table 7.1 Inhalation and Oral Cancer Potency Factors

Substance	Chemical Abstract Service Number (CAS)	Inhalation Potency Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
Acetaldehyde	75-07-0	1.0×10^{-2}	
Acetamide	60-35-5	7.0×10^{-2}	
Acrylamide	79-06-1	$4.5 \times 10^{+0}$	
Acrylonitrile	107-13-1	$1.0 \times 10^{+0}$	
Allyl chloride	107-05-1	2.1×10^{-2}	
2-Aminoanthraquinone	117-79-3	3.3×10^{-2}	
Aniline	62-53-3	5.7×10^{-3}	
Arsenic (inorganic)	7440-38-2	$1.2 \times 10^{+1}$	$1.5 \times 10^{+0}$
Asbestos [#]	1332-21-4	$2.2 \times 10^{+2}$ [#]	
Benz[*a*]anthracene [BaP]	56-55-3	3.9×10^{-1}	$1.2 \times 10^{+0}$
Benzene	71-43-2	1.0×10^{-1}	
Benzidine	92-87-5	$5.0 \times 10^{+2}$	
Benzo[*a*]pyrene	50-32-8	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
Benzo[*b*]fluoranthrene [BaP]	205-99-2	3.9×10^{-1}	$1.2 \times 10^{+0}$
Benzo[*j*]fluoranthrene [BaP]	205-82-3	3.9×10^{-1}	$1.2 \times 10^{+0}$
Benzo[*k*]fluoranthrene [BaP]	207-08-9	3.9×10^{-1}	$1.2 \times 10^{+0}$
Benzyl chloride	100-44-7	1.7×10^{-1}	
Beryllium	7440-41-7	$8.4 \times 10^{+0}$	
Bis(2-chloroethyl) ether	111-44-4	$2.5 \times 10^{+0}$	
Bis(chloromethyl)ether	542-88-1	$4.6 \times 10^{+1}$	
1,3-Butadiene	106-99-0	6.0×10^{-1}	
Cadmium (and compounds)	7440-43-9	$1.5 \times 10^{+1}$	
Carbon tetrachloride	56-23-5	1.5×10^{-1}	
Chlorinated Dibenzo-p-dioxins [A]			
2,3,7,8-Tetrachlorodibenzo-*p*-dioxin	1746-01-6	$1.3 \times 10^{+5}$	$1.3 \times 10^{+5}$
1,2,3,7,8-Pentachlorodibenzo-*p*-dioxin	40321-76-4	$1.3 \times 10^{+5}$	$1.3 \times 10^{+5}$
1,2,3,4,7,8-Hexachlorodibenzo-*p*-dioxin	39227-28-6	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,6,7,8-Hexachlorodibenzo-*p*-dioxin	57653-85-7	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,7,8,9-Hexachlorodibenzo-*p*-dioxin	19408-74-3	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,4,6,7,8-Heptachlorodibenzo-*p*-dioxin	35822-46-9	$1.3 \times 10^{+3}$	$1.3 \times 10^{+3}$
1,2,3,4,,6,7,8,9-Octachlorodibenzo-*p*-dioxin	3268-87-9	$3.9 \times 10^{+1}$	$3.9 \times 10^{+1}$
Chlorinated Dibenzofurans [A]			
2,3,7,8-Tetrachlorodibenzofuran	5120-73-19	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,7,8-Pentachlorodibenzofuran	57117-41-6	$3.9 \times 10^{+3}$	$3.9 \times 10^{+3}$
2,3,4,7,8-Pentachlorodibenzofuran	57117-31-4	$3.9 \times 10^{+4}$	$3.9 \times 10^{+4}$
1,2,3,4,7,8-Hexachlorodibenzofuran	70648-26-9	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,6,7,8-Hexachlorodibenzofuran	57117-44-9	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
1,2,3,7,8,9-Hexachlorodibenzofuran	72918-21-9	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
2,3,4,6,7,8-Hexachlorodibenzofuran	60851-34-5	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$

Table 7.1 Inhalation and Oral Cancer Potency Factors

Substance	Chemical Abstract Service Number (CAS)	Inhalation Potency Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
1,2,3,4,6,7,8-Heptachlorodibenzofuran	67562-39-4	$1.3 \times 10^{+3}$	$1.3 \times 10^{+3}$
1,2,3,4,7,8,9-Heptachlorodibenzofuran	55673-89-7	$1.3 \times 10^{+3}$	$1.3 \times 10^{+3}$
1,2,3,4,,6,7,8,9-Octachlorodibenzofuran	39001-02-0	$3.9 \times 10^{+1}$	$3.9 \times 10^{+1}$
Chlorinated paraffins	108171-26-2	8.9×10^{-2}	
Chloroform	67-66-3	1.9×10^{-2}	
4-Chloro-*o*-phenylenediamine	95-83-0	1.6×10^{-2}	
p-Chloro-*o*-toluidine	95-69-2	2.7×10^{-1}	
Chromium (hexavalent)	18540-29-9	$5.1 \times 10^{+2}$	5×10^{-1}
Chrysene [BaP]	218-01-9	3.9×10^{-2}	1.2×10^{-1}
Creosote	8001-58-9	*	
p-Cresidine	120-71-8	1.5×10^{-1}	
Cupferron	135-20-6	2.2×10^{-1}	
2,4-Diaminoanisole	615-05-4	2.3×10^{-2}	
2,4-Diaminotoluene	95-80-7	$4.0 \times 10^{+0}$	
Dibenz[*a,h*]acridine [BaP]	226-36-8	3.9×10^{-1}	$1.2 \times 10^{+0}$
Dibenz[*a,j*]acridine [BaP]	224-42-0	3.9×10^{-1}	$1.2 \times 10^{+0}$
Dibenz[*a,h*]anthracene [BaP]	53-70-3	$4.1 \times 10^{+0}$	$4.1 \times 10^{+0}$
Dibenzo[*a,e*]pyrene [BaP]	192-65-4	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
Dibenzo[*a,h*]pyrene [BaP]	189-64-0	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
Dibenzo[*a,l*]pyrene [BaP]	189-55-9	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
Dibenzo[*a,l*]pyrene [BaP]	191-30-0	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
7H-Dibenzo[*c,g*]carbazole [BaP]	194-59-2	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
1,2-Dibromo-3-chloropropane	96-12-8	$7.0 \times 10^{+0}$	
1,4-Dichlorobenzene	106-46-7	4.0×10^{-2}	
3,3'-Dichlorobenzidine	91-94-1	$1.2 \times 10^{+0}$	
1,1-Dichloroethane	75-34-3	5.7×10^{-3}	
Diesel exhaust [B]	NA	$1.1 \times 10^{+0}$	
Diethylhexylphthalate	117-81-7	8.4×10^{-3}	8.4×10^{-3}
p-Dimethylaminoazobenzene	60-11-7	$4.6 \times 10^{+0}$	
7,12-Dimethylbenz[*a*]anthracene [BaP]	57-97-6	$2.5 \times 10^{+2}$	$2.5 \times 10^{+2}$
1,6-Dinitropyrene [BaP]	42397-64-8	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
1,8-Dinitropyrene [BaP]	42397-65-9	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
2,4-Dinitrotoluene	121-14-2	3.1×10^{-1}	
1,4-Dioxane	123-91-1	2.7×10^{-2}	
Epichlorohydrin	106-89-8	8.0×10^{-2}	
Ethyl benzene	100-41-4	8.7×10^{-3}	1.1×10^{-2}
Ethylene dibromide	106-93-4	2.5×10^{-1}	
Ethylene dichloride	107-06-2	7.2×10^{-2}	
Ethylene oxide	75-21-8	3.1×10^{-1}	

Table 7.1 Inhalation and Oral Cancer Potency Factors

Substance	Chemical Abstract Service Number (CAS)	Inhalation Potency Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
Ethylene thiourea	96-45-7	4.5×10^{-2}	
Formaldehyde	50-00-0	2.1×10^{-2}	
Hexachlorobenzene	118-74-1	$1.8 \times 10^{+0}$	
Hexachlorocyclohexanes (technical grade)	608-73-1	$4.0 \times 10^{+0}$	$4.0 \times 10^{+0}$
Hydrazine	302-01-2	$1.7 \times 10^{+1}$	$3.0 \times 10^{+0}$
Indeno[1,2,3-*cd*]pyrene [BaP]	193-39-5	3.9×10^{-1}	$1.2 \times 10^{+0}$
Lead and lead compounds	7439-92-1	4.2×10^{-2}	8.5×10^{-3}
Lindane	58-89-9	$1.1 \times 10^{+0}$	$1.1 \times 10^{+0}$
Methyl tertiary-butyl ether	1634-04-4	1.8×10^{-3}	
3-Methylcholanthrene [BaP]	56-49-5	$2.2 \times 10^{+1}$	$2.2 \times 10^{+1}$
5-Methylchrysene [BaP]	3697-24-3	$3.9 \times 10^{+0}$	$1.2 \times 10^{+1}$
4, 4'-Methylene bis(2-chloroaniline) (MOCA)	101-14-4	$1.5 \times 10^{+0}$	
Methylene chloride	75-09-2	3.5×10^{-3}	
4,4'-Methylenedianiline	101-77-9	$1.6 \times 10^{+0}$	$1.6 \times 10^{+0}$
Michler's ketone	90-94-8	8.6×10^{-1}	
Naphthalene	91-20-3	1.2×10^{-1}	
Nickel (and compounds)	7440-02-0	9.1×10^{-1}	
5-Nitroacenaphthene [BaP]	602-87-9	1.3×10^{-1}	1.3×10^{-1}
6-Nitrochrysene [BaP]	7496-02-8	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
2-Nitrofluorene [BaP]	607-57-8	3.9×10^{-2}	1.2×10^{-1}
1-Nitropyrene [BaP]	5522-43-0	3.9×10^{-1}	$1.2 \times 10^{+0}$
4-Nitropyrene [BaP]	57835-92-4	3.9×10^{-1}	$1.2 \times 10^{+0}$
N-Nitroso-n-butylamine	924-16-3	$1.1 \times 10^{+1}$	
N-Nitroso-N-methylethylamine	10595-95-6	$2.2 \times 10^{+1}$	
N-Nitrosodi-*n*-propylamine	621-64-7	$7.0 \times 10^{+0}$	
N-Nitrosodiethylamine	55-18-5	$3.6 \times 10^{+1}$	
N-Nitrosodimethylamine	62-75-9	$1.6 \times 10^{+1}$	
N-Nitrosodiphenylamine	86-30-6	9.0×10^{-3}	
p-Nitrosodiphenylamine	156-10-5	2.2×10^{-2}	
N-Nitrosomorpholine	59-89-2	$6.7 \times 10^{+0}$	
N-Nitrosopiperidine	100-75-4	$9.4 \times 10^{+0}$	
N-Nitrosopyrrolidine	930-55-2	$2.1 \times 10^{+0}$	
Pentachlorophenol	87-86-5	1.8×10^{-2}	
Perchloroethylene	127-18-4	2.1×10^{-2}	5.1×10^{-2}
Polychlorinated biphenyls (PCBs) (unspeciated mixture)	1336-36-3		
(high risk) [P1]		$2.0 \times 10^{+0}$	$2.0 \times 10^{+0}$
(low risk) [P2]		4.0×10^{-1}	4.0×10^{-1}
(lowest risk) [P3]		7.0×10^{-2}	7.0×10^{-2}

Table 7.1 Inhalation and Oral Cancer Potency Factors

Substance	Chemical Abstract Service Number (CAS)	Inhalation Potency Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
Polychlorinated biphenyls[P4] (PCBs) (speciated)			
3,3',4,4'-Tetrachlorobiphenyl (77)	35298-13-3	$1.3 \times 10^{+1}$	$1.3 \times 10^{+1}$
3,4,4',5-Tetrachlorobiphenyl (81)	70362-50-4	$3.9 \times 10^{+1}$	$3.9 \times 10^{+1}$
2,3,3',4,4'- Pentachlorobiphenyl (105)	32598-14-4	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2,3,4,4'5- Pentachlorobiphenyl (114)	74472-37-0	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2,3'4,4',5- Pentachlorobiphenyl (118)	31508-00-6	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2',3,4,4',5- Pentachlorobiphenyl (123)	65510-44-3	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
3,3',4,4',5- Pentachlorobiphenyl (126)	57465-28-8	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
2,3,3',4,4',5-Hexachlorobiphenyl (156)	38380-08-4	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2,3,3',4,4',5'-Hexachlorobiphenyl (157)	69782-90-7	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
2,3',4,4',5,5'-Hexachlorobiphenyl (167)	52663-72-6	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
3,3',4,4'5,5'- Hexachlorobiphenyl (169)	32774-16-6	$3.9 \times 10^{+3}$	$3.9 \times 10^{+3}$
2,3,3'4,4',5,5'- Heptachlorobiphenyl (189)	39635-31-9	$3.9 \times 10^{+0}$	$3.9 \times 10^{+0}$
Potassium bromate	7758-01-2	4.9×10^{-1}	
1,3-Propane sultone	1120-71-4	$2.4 \times 10^{+0}$	
Propylene oxide	75-56-9	1.3×10^{-2}	2.4×10^{-1}
1,1,2,2-Tetrachloroethane	79-34-5	2.0×10^{-1}	
Thioacetamide	62-55-5	$6.1 \times 10^{+0}$	
2,4-Toluene diisocyanate	584-84-9	3.9×10^{-2}	
2,6-Toluene diisocyanate	91-08-7	3.9×10^{-2}	
1,1,2-Trichloroethane (vinyl trichloride)	79-00-5	5.7×10^{-2}	
Trichloroethylene	79-01-6	7.0×10^{-3}	1.5×10^{-2}
2,4,6-Trichlorophenol	88-06-2	7.0×10^{-2}	
Urethane	51-79-6	$1.0 \times 10^{+0}$	
Vinyl chloride	75-01-4	2.7×10^{-1}	

Notes for Table 7.1

#	Asbestos: [100 PCM fibers/m^3]$^{-1}$ A unit risk factor of 2.7 x 10^{-6} ($\mu g/m^3$)$^{-1}$ and an inhalation cancer potency factor of 2.2 x 10^{+2} (mg/kg BW*day)$^{-1}$ are available (see Appendix C for explanation).
BaP	PAHs and PAH Derivatives: Many have potency equivalency factors relative to benzo[*a*]pyrene (see Appendix G). For multipathway chemicals, including PAHs, the oral slope factor is considered the same as the inhalation potency factor unless otherwise noted in the Table.
A	Polychlorinated Dibenzo-*p*-dioxins, Polychlorinated Dibenzofurans and speciated poly chlorinated biphenyls: (see Appendix E). For convenience, OEHHA has calculated cancer potency factors for speciated polychlorinated dibenzo-*p*-dioxin, polychlorinated dibenzofuran and poly chlorinated biphenyl congeners using the procedure in Appendix E.
B	Diesel Exhaust is listed as a Toxic Air Contaminant by the Air Resources Board as "Particulate Matter from Diesel-Fueled Engines". (See Appendix D)
*	Creosote: Can be calculated using Potency Equivalency Factors contained in the benzo[*a*]pyrene Toxic Air Contaminant document and in Appendix G of these guidelines.
P1	Polychlorinated Biphenyls (PCBs): High Risk is for use in cases where congeners with more than four chlorines do not comprise less (are greater) than one-half percent of total PCBs. The high risk number is the default for unspeciated PCB mixtures.
P2	The low risk number is generally not applicable to the Hot Spots program. The Hot Spots program addresses PCBs emitted by stationary facilities. It cannot be assumed that such emissions would occur by simple evaporation. There is a dermal absorption factor applied in evaluation of the dermal pathway for PCBs so the medium risk would not apply to dermal exposure (OEHHA, 2009). The water pathway does not include an assumption that PCB isomers are water soluble, so the medium number would not apply to the water pathway.
P3	Polychlorinated Biphenyls (PCBs): Lowest Risk is for use in cases where congeners with more than four chlorines comprise less than one-half percent of total PCBs. In order for the low number to be used, scientific justification needs to be presented.
P4	Number in parentheses is the IUPAC #, the PCB nomenclature is IUPAC. For multipathway chemicals, including PCBs, the oral slope factor is considered the same as the inhalation potency factor unless otherwise noted in the Table.

7.3 References

OEHHA, 2009. Air Toxics Hot Spots Risk Assessment Guidelines. Part II. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. May, 2009. . Available online at: http://www.oehha.ca.gov/air/hot_spots/tsd052909.html

8 - Risk Characterization for Carcinogens and Noncarcinogens and the Requirements for Hot Spots Risk Assessments

8.1 Introduction

Risk characterization is the final step of the health risk assessment (HRA). In this step, information developed through the exposure assessment is combined with information from the dose-response assessment to characterize risks to the general public from emissions. In the Hot Spots program, OEHHA conducts the dose-response assessment during the development of cancer potency factors and Reference Exposure Levels. These are used in conjunction with the exposure estimates to estimate cancer risk and evaluate hazard from noncancer toxicity of emitted chemicals. Under the Air Toxics Hot Spots (Hot Spots) Act, risk characterizations should present both individual and population-wide health risks (Health and Safety Code Section (HSC) 44306). Persons preparing HRAs for the Hot Spots Program should consult the local Air Pollution Control or Air Quality Management District (District) to determine if the District has special guidelines to assist with HRA format or other requirements of the Hot Spots Program.

OEHHA is recommending that a 30-year exposure duration be used as the basis for estimating cancer risk at the maximum exposed individual resident (MEIR) in the Hot Spots Program. This exposure duration represents the time of residency for 90 to 95% of Californians at a single location and should provide adequate public health protection against individual risk. We also recommend including the 9 and 70-year cancer risk at the MEIR as supplemental information. Note that a 70-year exposure duration is required to estimate cancer burden or provide an estimate of population-wide risk.

This chapter provides guidance on how to evaluate the risk characterization component of risk assessments required by the Hot Spots Program. A general summary of the risk characterization components includes the following items and information.

- The locations of the point of maximum impact (PMI), the MEIR, and the maximum exposed individual worker (MEIW) are to be identified. The PMI, MEIW, and MEIR for cancer risk and for noncancer hazard indices (averaging times for acute 1-hour, repeated 8-hour, and chronic hazard indices) may not be the same location; all should be identified.

- The location of any specified sensitive receptors (e.g., schools, hospitals, daycare, or eldercare facilities - contact the District or reviewing authority for more information) should be identified

- Estimates of population-wide cancer risk and noncancer hazard

This information must be clearly presented in cross-referenced text, tables, figures, and maps. Chapter 9 provides an outline that specifies the content and recommended format of HRA results. The HARP software is the recommended model for calculating HRA results for the Hot Spots Program. Information on obtaining the HARP software can be found under the Air Toxics Program on the ARB's web site at *www.arb.ca.gov*.

8.1.1 *Tiered Approach to Risk Assessment*

The tiered approach for risk assessment that is presented in detail in the TSD (OEHHA, 2012) and summarized here should be reviewed prior to conducting the health risk assessment. The tiered approach to risk assessment and the health impacts evaluation described here are included in the HARP software.

The tiered approach provides a risk assessor with flexibility and allows consideration of site-specific differences (Table 8.1). The four-tiered approach to risk assessment is intended to primarily apply to residential cancer risk assessment, both for inhalation and noninhalation pathways. Risk assessors can tailor the level of effort and refinement of an HRA by using either the point estimate exposure assumptions as the basis of the exposure and risk assessment, or both the point estimate and a stochastic treatment of exposure factor distributions.

Table 8.1 The Tiered Approach to Risk Assessment

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

Tier 1 is a standard point estimate approach that uses the recommended exposure variate (e.g., breathing or water ingestion rate) point estimates presented in this document. Derivations of these values are described in detail in OEHHA (2012). The results of the Tier 1 evaluations are required to be presented in the risk characterization section for all HRAs prepared for the Hot Spots Program. Thus, persons preparing an HRA using Tier 2 through Tier 4 evaluations must also include the risk characterization results of a Tier 1 evaluation in the HRA.

As discussed in OEHHA (2012), if the risk characterization results from a Tier 1 assessment are above a regulatory level of concern, the risk assessor may want to proceed with more site-specific analysis as described in Tier 2, or use a more resource-intensive stochastic modeling effort described in Tier 3 and Tier 4 (for cancer risk). While further evaluation may provide more information to the risk manager on which to base decisions, the Tier 1 evaluation is useful in comparing risks among a large number of facilities and must be included in all HRAs.

Tier 2 analysis allows the use of available and justifiable site-specific exposure variates (e.g., fish consumption), when presenting the potential health impacts. The site-specific information applied in a Tier 2 assessment must be adequately justified and approved by OEHHA and the District. In Tier 3, a stochastic approach to exposure assessment is taken using the distributions for the exposure pathways presented in the TSD (OEHHA, 2012) and in Chapter 5 of this Guidance Manual. The exposure distributions apply only to a residential receptor and are used only for the determination of cancer risk. OEHHA has not developed exposure intake distributions for workers to use in the offsite worker exposure scenario. Tier 4 is also a stochastic approach for the residential exposure scenario but allows for utilization of site-specific exposure variate distributions if they are justifiable and more appropriate for the site under evaluation than those derived in OEHHA (2012). Alternative site-specific distributions must be approved by OEHHA and the District. For an off-site worker cancer risk evaluation, Tiers 3 and 4 do not apply. Tier 3 and Tier 4 analyses show what a distribution of potential cancer risk may be to an individual or population based on a distribution of exposure inputs (e.g., water ingestion rate) rather than specific point estimates of exposure.

Table 8.2 summarizes OEHHA's recommendations for use of the four Tiers in cancer and noncancer risk assessment.

Table 8.2 Tiers for Residential and Offsite Worker Cancer and Noncancer Hot Spots Risk Assessments

Tier	Cancer		Non Cancer Chronic and 8-Hour	
	Inhalation	Noninhalation	Inhalation	Noninhalation
Tier-1	X	X	X	X
Tier-2	X	X		X[b]
Tier-3	X[a]	X[a]		
Tier-4	X[a]	X[a]		

[a] Applies to residential exposure scenario only
[b] Applies to chronic noncancer exposure only

OEHHA has not developed a stochastic approach (Tier 3 or 4) for estimating noncancer health impacts using acute, 8-hour, and chronic Reference Exposure Levels (RELs). Tier 1 is the only option for determining noncancer health impacts from inhalation exposure since calculating the hazard quotient involves dividing the ground level air concentrations for the specified exposure duration by the appropriate RELs. However, chronic noninhalation noncancer risks involve a calculation of dose from oral or dermal pathways to which site-specific evaluations could be considered under a Tier 2 approach.

Small foot-print facilities – Tier 2 or Tier 4

Some facilities subject to the Air Toxics Hot Spots Act (e.g., some in the industry-wide categories such as gas stations or dry cleaners) have very small zones of impact. In some of these instances, there will be very few receptors within the zone of impact. It isn't possible to develop special recommendations for exposure variates for all possible exposure scenarios. Alternative breathing rates (point estimates or distributions) may be used as part of Tier 2 or Tier 4 risk assessments with appropriate supporting justification in the case of a very small zone of impact. OEHHA is willing to work with risk managers at ARB and the Districts on this issue.

8.2 Risk Characterization for Carcinogens

Cancer risk is calculated by multiplying the daily inhalation or oral dose (calculated in Chapter 5), by a cancer potency factor, the age sensitivity factor, the frequency of time spent at home (for residents only), and the exposure duration divided by averaging time, to yield the excess cancer risk (see section 8.2.4). As described below, the excess cancer risk is calculated separately for each age grouping and then summed to yield cancer risk at the receptor location. A brief description of the age sensitivity factors, exposure duration, and frequency of time spent at home are included in Sections 8.2.1 to 8.2.3 below. These factors are discussed in detail in OEHHA (2009) and OEHHA (2012).

8.2.1 *Adjustment for Early Life Stage Exposures to Carcinogens*

Studies have shown that young animals are more sensitive than adult animals to exposure to many carcinogens (OEHHA, 2009). Therefore, OEHHA developed age sensitivity factors (ASFs) to take into account the increased sensitivity to carcinogens during early-in-life exposure (Table 8.3). These factors were developed and described in detail in OEHHA (2009). In the absence of chemical-specific data, OEHHA recommends a default ASF of 10 for the third trimester to age 2 years, and an ASF of 3 for ages 2 through 15 years to account for potential increased sensitivity to carcinogens during childhood.

Table 8.3 Age Sensitivity Factors by Age Group for Cancer Risk Assessment

Age Group	Age Sensitivity Factor (unitless)
3rd Trimester	10
0<2 years	10
2<9 years	3
2<16 years	3
16<30 years	1
16-70 years	1

For specific carcinogens where data indicate enhanced sensitivity during life stages other than the immediate postnatal and juvenile periods, or for which data demonstrate ASFs different from the default ASFs, the chemical-specific data should be used in order to adequately protect public health.

The risk assessments generated under the Air Toxics Hot Spots Act are reviewed by OEHHA. If a risk assessor had data indicating there are no windows of susceptibility early in life or that a different ASF should be used for a specific carcinogen and wanted to use these data, OEHHA would review the material as part of the review of the risk assessment.

8.2.2 *Fraction of Time Spent at Home for Cancer Risk Assessment*

OEHHA and ARB evaluated information from activity patterns databases to estimate the fraction of time at home (FAH) during the day (OEHHA, 2012). This information can be used to adjust exposure duration and cancer risk from a specific facility's emissions, based on the assumption that exposure to the facility's emissions are not occurring away from home. From the third trimester to age <2 years, 85% of time is spent at home (Table 8.4). From age 2 through <16 years, 72% of time is spent at home. From age 16 years and greater, 73% of time is spent at home. Facilities with a school within the 1×10^{-6} (or greater) isopleth should use FAH = 1 for the child age groups (3rd Trimester, 0<2 years, and 2<16 years).

Table 8.4 Recommendations for Fraction of Time at Home (FAH) for Evaluating Residential Cancer Risk

Age Range	Fraction of Time at Residence
3rd Trimester, and 0<2 years	0.85[1]
2<16 years[2]	0.72[1]
16-70 years[3]	0.73

[1] Use FAH = 1 if a school is within the 1×10^{-6} (or greater) cancer risk isopleth
[2] Also use FAH = 0.72 for 2<9 yr age group.

[3] Also use FAH = 0.73 for 16<30 yr age group.

The FAH is calculated based on a diary of trips taken over a 24-hour period on the survey day. Ninety-five percent of the diary days were on weekdays. Participants can select "vacation" as one of their trips. However, vacation time represented only a fraction (0.68%) of the over 175,000 trips recorded in the survey. Because much of these vacation trips were presumed to be within-day trips and were only a small fraction of total trips, there is likely little overlap with the Exposure Frequency (EF) variate used in the dose equations in Chapter 5.

8.2.3 *Exposure Duration for Estimating Cancer Risk to Residents and Off-Site Workers*

OEHHA recommends that an exposure duration (residency time) of 30 years be used to estimate individual cancer risk for the maximally exposed individual resident (MEIR) (Table 8.5). OEHHA also recommends that the 30-year exposure duration be used as the basis for public notification and risk reduction audits and plans. The Districts, however, may opt to use the 70 year cancer risk for notification and risk reduction audits and plans.

Note that the 30-year exposure duration starts in the third trimester to accommodate the increased susceptibility of exposures in early life (OEHHA, 2009), and would apply to both the point estimate and stochastic approaches.

Table 8.5 Summary of Recommendations for Exposure Duration for Individual Cancer Risk at the MEIR and MEIW

Receptor	***Recommendation***
Resident (MEIR)	30 years
Resident (supplemental Information)	9 years for central tendency; 70 years for maximum (lifetime)
Worker (MEIW)	25 years

Exposure durations of 9-years and 70-years are also recommended to be evaluated for the MEIR to show the range of cancer risk based on residency periods. If a facility is notifying the public regarding cancer risk, the 9- and 70-year cancer risk estimates are useful for people who have resided in their current residence for periods shorter and longer than 30 years.

The 9-, 30-, and 70-year exposures are chosen to coincide with U.S. EPA's estimates of the average (9 years), high-end estimates (30-years) of residence time, and a lifetime residency (70 years). These estimates are also consistent with what is known about residence time in California. Together, the 9-, 30-, and 70-year cancer risk calculations provide a useful presentation of cancer risk and the relationship to duration of residency and, thus, exposure to a facility's emissions.

For the maximally exposed individual worker (MEIW), OEHHA recommends using an exposure duration of 25 years to estimate individual cancer risk for the off-site worker scenario (Table 8.5). This duration represents approximately the 95th percentile of job tenure with the same employer in the U.S.

8.2.4 *Calculating Residential and Offsite Worker Inhalation Cancer Risk*

Residential Receptors

For residential inhalation exposure, cancer risk must be separately calculated for specified age groups (Eq. 8.2.4A, see Section 8.2.1), because of age differences in sensitivity to carcinogens and age differences in intake rates (per kg body weight). Separate risk estimates for these age groups provides a health-protective estimate of cancer risk by accounting for greater susceptibility in early life, including both age-related sensitivity and amount of exposure. The following equation illustrates the formula for calculating residential inhalation cancer risk. See Appendix I for a detailed example calculation.

A. Equation 8.2.4 A: $$RISK_{inh\text{-}res} = DOSE_{air} \times CPF \times ASF \times ED/AT \times FAH$$

7. $RISK_{inh\text{-}res}$ = Residential inhalation cancer risk
8. $DOSE_{air}$ = Daily inhalation dose (mg/kg-day)
9. CPF = Inhalation cancer potency factor (mg/kg-day^{-1})
10. ASF = Age sensitivity factor for a specified age group (unitless)
11. ED = Exposure duration (in years) for a specified age group
12. AT = Averaging time for lifetime cancer risk (years)
13. FAH = Fraction of time spent at home (unitless)

a: Recommended default values for EQ 8.2.4 A:

5. $DOSE_{air}$ = Calculated for each age group from Eq. 5.4.1
6. CPF = Substance-specific (see Table 7.1)
7. ASF = See Section 8.2.1
8. ED = 0.25 years for 3rd trimester, 2 years for 0<2, 7 years for 2<9, 14 years for 2<16, 14 years for 16<30, 54 years for 16-70
9. AT = 70 years*
10. FAH = See Table 8.4

*Although AT actually sums to 70.25 years when the 3rd trimester (0.25 years) is included, OEHHA recommends rounding AT = 70 years (and rounding residential exposure durations at 9- and 30-years rather than 9.25- and 30.25-years) because it is an insignificant adjustment.

Cancer risk calculated above for individual age groups are summed to estimate cancer risk for 9-, 30- and 70-year exposures as shown below. Note that this example includes the Fraction of Time Spent at Home (FAH) for each age grouping.

Calculation of Inhalation Cancer Risk from the Third Trimester to Age Nine:

$RISK_{inh\text{-}res}$ = ($DOSE_{air}$ third trimester × CPF × 10 × 0.25/70 years × $FAH_{3rd\ tri\ <2}$) + ($DOSE_{air}$ age 0<2 × CPF × 10 × 2/70 × $FAH_{3rd\ tri\ <2}$) + ($DOSE_{air}$ age 2<9 × CPF × 3 × 7/70 years × $FAH_{2<9}$)

Calculation of Inhalation Cancer Risk from Third Trimester to Age 30:

$RISK_{inh\text{-}res}$ = ($DOSE_{air}$ third trimester × CPF × 10 × 0.25/70 years × $FAH_{3rd\ tri\ <2}$) + ($DOSE_{air}$ age 0<2 × CPF × 10 × 2/70 × $FAH_{3rd\ tri\ <2}$) + ($DOSE_{air}$ age 2<16 × CPF × 3 × 14/70 × $FAH_{2<16}$) + ($DOSE_{air}$ age 16<30 × CPF × 1 × 14/70 years × $FAH_{16\text{-}30}$)

Calculation of Inhalation Cancer Risk from Third Trimester to Age 70:

$RISK_{inh\text{-}res}$ = ($DOSE_{air}$ third trimester × CPF × 10 ×0.25/70 years× $FAH_{3rd\ tri\ <2}$) + ($DOSE_{air}$ age 0<2 × CPF × 10 × 2/70 × $FAH_{3rd\ tri\ <2}$) + ($DOSE_{air}$ age 2<16 × CPF × 3 × 14/70 × $FAH_{2<16}$) + ($DOSE_{air}$ age 16<70 × CPF × 1 × 54/70 years × $FAH_{16\text{-}70}$)

Expressing cancer risk in "chances per million" is useful as a risk communication tool for the public, but cancer risk can also be expressed in other ways, such as "chances per 100,000" (cancer risk × 10^5) or "chances per 10 million" (cancer risk × 10^7). To convert the resulting cancer risk estimate to chances of developing cancer per million individuals exposed, multiply the cancer risk by 10^6:

$$\text{Cancer risk} \times 10^6 = \text{chances per million}$$

For exposure to multiple carcinogenic substances, Table 8.7 and Table I.5 in Appendix I are examples of how cancer risks of individual substances are summed to determine the total cancer risk.

Worker Receptors

For assessment of off-site worker cancer risk at the MEIW, the default assumes working age begins at 16 years. Note that the residential FAH factor in Eq. 8.2.4.A above does not apply for workers. The daily inhalation dose ($DOSE_{air}$) (as calculated in Chapter 5, EQ 5.4.1.2) is based on the adjusted 8-hour concentration at the MEIW (for non-continuous sources) and amount of time the offsite worker's schedule overlaps with the facilities' emission schedule. The duration of exposure at the MEIW receptor is 25 years, as discussed in the TSD (OEHHA, 2012).

B. Equation 8.2.4 B: $$RISK_{inh\text{-}work} = DOSE_{air} \times CPF \times ASF \times ED/AT$$

1. $RISK_{inh\text{-}work}$ = Worker inhalation cancer risk

a: Recommended default values for EQ 8.2.4 B:

1. $DOSE_{air}$ = Calculated for workers in Eq. 5.4.1.2
2. CPF = Substance specific (see Table 7.1)
3. ASF = 1 for working age 16-70 yrs (See Section 8.2.1)
4. ED = 25 years
5. AT = 70 yrs for lifetime cancer risk

Work Locations with Daycare Facilities:

An additional risk management consideration for offsite worker cancer risk assessment of a Hot Spots facility is whether there are women of child bearing age at the MEIW location and whether the MEIW has a daycare center. In the case of women of child-bearing age at the MEIW, the Districts may wish to treat the off-site MEIW in the same way as the residential scenario to account for the higher susceptibility during the third trimester of pregnancy (i.e., use of an ASF=10 for third trimester exposure). If there is onsite daycare at the MEIW, then the risks to the children will be underestimated using the offsite adult worker scenario. In this case, the Districts may wish to include a cancer risk assessment for the children in the onsite daycare, assuming they could be there from 0 to age 6 years (ED = 6 years) and using the appropriate exposure factors to calculate $DOSE_{air}$, fraction of time at worksite (e.g., hrs at daycare per 24 hrs), and ASFs in EQ 8.2.4 B to account for the higher susceptibility of infants and children to carcinogens.

Children at a MEIW daycare may also be assessed for noninhalation exposures. Typically, soil ingestion and dermal exposure will be the most common noninhalation pathways. However, all pathways that are present at the daycare should be included. See section 8.2.6 for more discussion of multipathway risk assessment methods.

8.2.5 *Calculation of Noninhalation Cancer Risk*

A small subset of Hot Spots substances is subject to deposition onto the soil, plants, and water bodies (see Table 5.1). These substances need to be evaluated by the appropriate noninhalation pathways, as well as by the inhalation pathway, and the risk characterization results must be presented in all HRAs. These substances include semi-volatile organic chemicals and heavy metals.

For all multipathway substances, the exposure pathways that must be evaluated at every residential and worker site (in addition to inhalation) are soil ingestion and dermal exposure. If PAHs (and creosotes), lead, dioxins, furans, or PCBs are emitted, then the breast-milk consumption pathway becomes mandatory for residential receptors. OEHHA has developed transfer coefficients for these chemicals from the mother to breast milk (see OEHHA, 2012 for details). The other exposure pathways (e.g.,

ingestion of homegrown produce or fish) are only evaluated for residential receptors if the facility impacts that exposure medium and the receptor under evaluation can be exposed to that medium or pathway. For example, if the facility does not impact a fishable body of water within the isopleth of the facility, or the impacted water body does not sustain fish that are consumed by fishers, then the fish pathway will not be considered for that facility or receptor.

Table 8.6 identifies the residential receptor exposure pathways that are mandatory and those that are dependent on the available routes of exposure. Table 8.6 also identifies the three exposure pathways that are relevant for a worker receptor. The cancer risk estimates should be presented in the risk characterization section of the risk assessment for all the appropriate pathways.

Table 8.6 Mandatory and Site/Route Dependent Exposure Pathways

Mandatory Exposure Pathways	Site/Route Dependent Exposure Pathways
• Inhalation[w] • Soil Ingestion[w] • Dermal Exposure to Contaminated Soil[w] • Breast Milk Consumption[*]	• Homegrown Produce Ingestion • Angler-Caught Fish Ingestion • Drinking Water Ingestion • Home-Raised Animal Product Ingestion (Dairy (Cow's) Milk, Meat (Beef, Pork, Chicken) and Egg).

(w) Identifies the appropriate exposure pathways that should be evaluated for a worker. These pathways are inhalation, dermal exposure, and the soil ingestion pathway.
(*) If PAHs (including creosotes), lead, dioxins, furans, or PCBs are emitted, then the breast-milk consumption pathway becomes mandatory.

The noninhalation residential cancer risk is calculated using the same steps as inhalation cancer risk described in Section 8.2.4. A dose (see Chapters 4 and 5) from the pathway under evaluation (e.g., soil ingestion) is multiplied by the substance-specific oral slope factor, expressed in units of inverse dose (i.e., $(mg/kg/day)^{-1}$) (Table 7.1), the appropriate age sensitivity factor (ASF), and exposure duration divided by averaging time to yield the cancer risk for a specified age grouping. Cancer risk for each age group is summed as appropriate for the exposure duration. The FAH factor is relevant only to the inhalation pathway and is not appropriate to use in the noninhalation pathways.

Equation 8.2.5 illustrates the formula for calculating noninhalation cancer risk. Details (data, algorithms, and guidance) for each exposure pathway are presented in Chapter 5 and in OEHHA (2012).

A. Equation 8.2.5: $\mathbf{RISK_{noninh} = DOSE_{noninh} \times CPF_{oral} \times ASF \times ED/AT}$

1. $RISK_{noninh}$ = Noninhalation pathway cancer risk
2. $DOSE_{noninh}$ = Daily dose (mg/kg-day) for a specified non-inhalation pathway for each age group
3. CPF_{oral} = Oral cancer potency (slope) factor (mg/kg-day^{-1})
4. ASF = Age sensitivity factor for a specified age group (unitless)
5. ED = Exposure duration (in years) for a specified age group
6. AT = Averaging time for lifetime cancer risk

a: Recommended default values for EQ 8.2.5:

1. $DOSE_{noninh}$ = Calculated in Chapter 5 dose algorithms for each age group and for each noninhalation route in Table 8.6 the receptor is exposed to
2. CPF_{oral} = Substance-specific (see Table 7.1)
3. ASF = See Section 8.2.1
4. ED = Residents: 0.25 years for 3rd trimester, 2 years for 0<2, 7 years for 2<9, 14 years for 2<16, 14 years for 16<30, 54 years for 16-70
   = Offsite worker: 25 yrs
5. AT = 70 years

Estimating cancer risk for 9-, 30- and 70-years by summing the individual age-group cancer risks is the same as that shown for the inhalation route in Section 8.2.4. The exception is that the FAH variate is only appropriate for the residential inhalation pathway and is not a factor for oral and dermal exposure pathways.

Calculation of Noninhalation Cancer Risk from Third Trimester to Age 30:

$RISK_{noninh\text{-}res}$ = ($DOSE_{noninh}$ third trimester × CPF × 10 × 0.25/70 years) + ($DOSE_{noninh}$ age 0<2 × CPF × 10 × 2/70) + ($DOSE_{noninh}$ age 2<16 × CPF × 3 × 14/70) + ($DOSE_{noninh}$ age 16<30 × CPF × 1 × 14/70 years)

To convert this estimated probability of risk to chances per million of developing cancer, multiply the estimated cancer risk for each noninhalation exposure route by 10^6. This result is useful communication tool to compare risks for each pathway of exposure.

$$\text{Cancer risk} \times 10^6 = \text{cancer risk expressed as chances per million}$$

For assessment of the offsite worker the typical noninhalation pathways that apply for worker cancer risk are the dermal exposure pathway and the soil ingestion pathway.

Children at a MEIW daycare may also be assessed for noninhalation exposures. Typically, soil ingestion and dermal exposure will be the most common noninhalation pathways. However, all pathways that are present at the daycare should be included.

8.2.6 *Multipathway Cancer Risk Methodology*

Under a Tier 1 assessment, it is necessary to calculate the total cancer risk from both inhalation and noninhalation exposures if multipathway substances are emitted from the facility. The calculation of cancer risk that includes exposure to a multipathway substance or substances has three steps:

1) Calculate cancer risk for the inhalation pathway (EQ 8.2.4 A for residents, EQ 8.2.4 B for off-site workers) for all substances, and the noninhalation pathways that apply (EQ 8.2.5) for all multipathway substances, using high-end point estimates of intake rates.
2) For each multipathway substance, identify the two exposure pathways with the highest risk. These are the dominant pathways that are to be assessed using high-end point estimates of intake rates for the total cancer risk. For all other pathways, the average point estimate of intake rates may be used to calculate the pathway cancer risk (See OEHHA (2012) for more information).
3) To calculate total cancer risk, all inhalation and noninhalation pathways are summed together for all substances.

The final cancer risk calculation using a combination of high-end and average exposure parameters is referred to as the derived risk in the HARP software. This is described in Chapter 1, Section 1.4.1 of OEHHA (2012). The inhalation route is almost always one of the two dominant pathways in a multipathway cancer risk assessment. Therefore, in most cases only one noninhalation pathway would be calculated using a high-end dose point estimate. For all other pathways, the average point estimate may be used to calculate the pathway cancer risk.

For example, if dermal exposure and soil ingestion risks are calculated, then the cancer risks from these pathways would be summed along with the inhalation cancer risks to give the total cancer risk for the single multipathway substance:

Cancer Risk (inhalation) + Cancer Risk (dermal) + Cancer Risk (soil) = Total Risk

The mother's milk pathway also becomes a mandatory pathway to assess risk in nursing infants if the mother is exposed to specific substances (see Table 5.1).

Many facilities will emit multiple carcinogenic substances. If multiple substances are emitted, the substance-specific cancer risks for all exposure pathways are summed to give the (total) multipathway cancer risk at the receptor location. The HARP software will display not only the multipathway risk for each carcinogenic substance, but also show a breakdown of the cancer risk from each exposure pathway. Table 8.7 shows the results of a multipathway risk assessment for a hypothetical facility. While not presented in the following table, it is critical to identify the driving exposure pathways and the driving substances in a multipathway cancer risk assessment when summarizing and presenting the HRA results. See Chapter 9 for more information.

Table 8.7 Multipathway Assessment of a Hypothetical Facility 30-Year Cancer Risk

Substance	Cancer Risk[a]	Cancer risk[b] (chances per million)
Arsenic	1.1×10^{-5} (i) 3×10^{-7} (ni)	11 (i) 0.3 (ni)
Benzene	2.92×10^{-4} (i)	292 (i)
2,3,7,8-TCDD (dioxin)	1.06×10^{-4} (i) 5.7×10^{-5} (ni)	106 (i) 57 (ni)
1,3-Butadiene	6.0×10^{-6} (i)	6 (i)
Total Facility Cancer Risk	**4.723×10^{-4}**	**472**

[a] As calculated in EQ 8.2.4 A or EQ 8.2.5
[b] Calculated as: cancer risk × 10^6 = chances per million
i = inhalation pathway contribution
ni = noninhalation pathway contribution

Cancer risk in Table 8.7 for the multipathway substances, arsenic and 2,3,7,8-TCDD, is arranged by the inhalation pathway risk and the sum of all noninhalation pathway risks. The total facility multipathway cancer risk is the sum of all inhalation and noninhalation pathways.

Cancer risks from different substances are treated additively in risk assessment generally, and in the Hot Spots Program in part because many carcinogens act through the common mechanism of DNA damage. The additive assumption is reasonable from a public health point of view. Other possible interactions of multiple carcinogens include synergism (effects are greater than additive) or antagonism (effects are less than additive). The type of interaction is both chemical and dose dependent and in most cases the data are not available to adequately characterize these interactions.

8.2.7 *Multipathway Cancer Risk for Infant Exposure to Mother's Milk*

The mother' milk pathway becomes mandatory if the nursing mother is exposed to one or more of the following multipathway substances: dioxins and furans, PCBs, PAHs including creosotes, and lead. The default assumption inherent in the intake rate is that the infant's only source of food is breast for the first year (e.g., is fully breastfed, see OEHHA, 2012, for details), which is one-half of the 0<2 year age group used in the Hot Spots program. Thus, the cancer risk by the mother's milk pathway will need to be calculated with a modified cancer risk equation using a different exposure duration:

A. Equation 8.2.7: $\mathbf{RISK_{mm}} = \text{Dose-Im} \times \mathbf{CPF_{oral}} \times \mathbf{ASF} \times \mathbf{ED/AT}$

1. $RISK_{mm}$ = Infant cancer risk via mother's milk pathway
2. Dose-Im = Daily dose (mg/kg-day) to infant from mother's milk
3. CPF_{oral} = Oral cancer slope factor (mg/kg-day^{-1})
4. ASF = Age sensitivity factor for infant (unitless)
5. ED = Exposure duration (in years) for infant
6. AT = Averaging time for lifetime cancer risk

a: Recommended default values for EQ 8.2.7:

6. Dose-Im = Calculated from EQ 5.4.3.5.2, dose to infant via mother's milk
7. CPF_{oral} = Substance-specific (see Table 7.1)
8. ASF = 10 (See Section 8.2.1)
9. ED = 1 yr (1st yr of 0<2 yr age group)
10. AT = 70 years

Once the cancer risk is determined for the mother's milk pathway for each applicable substance, the pathway risk is summed with other pathway risks.

For Tier 1, the derived approach for cancer risk assessment should be used if the mother's milk pathway applies. As outlined in Section 8.2.6, the two dominant pathways will be calculated using high-end point estimates of intake rates; all additional pathways may be calculated using average point estimates of intake rates. There will be four mandatory pathways to assess (inhalation, mother's milk, soil ingestion and dermal exposure) for cancer risk when exposure to dioxins/furans, PCBs, PAHs including creosotes, and/or lead occurs. Therefore, if the infant is exposed to no other additional site-specific noninhalation pathway(s), only the two dominant pathways among the four will be assessed for cancer risk using high-end point estimates of intake rates; and the others would be assessed using the average point estimate of intake rate.

In short, multipathway cancer risk for a substance is estimated by summing the potential inhalation and noninhalation cancer risks for the receptor location of interest. See the discussion of Tier 1 in Section 8.2.6 or the TSD for more information on the method used to determine the multipathway cancer risk.

8.2.8 *Cancer Risk Characterization for Stochastic Risk Assessment*

Risk characterization for a stochastic risk assessment is similar to that described for the point-estimate approach. However, the stochastic risk assessment produces a distribution of risk that accounts for some of the natural variability in exposure-related factors, such as breathing rates or water intake. The cancer risk distribution for inhalation cancer risk, for example, is generated by multiplying randomly selected values from the breathing rate distribution by the ground level air concentration, and the cancer potency factor. A variation of the Monte Carlo method called Latin hypercube sampling is the method by which the values from the breathing rate distribution are

selected. If noninhalation pathways need to be evaluated, the same process is followed for each pathway and the risk is summed to give an overall inhalation and noninhalation cancer risk distribution. Further, the specification of Age Sensitivity Factors and need to separately calculate risks requires that a Monte Carlo sampling be conducted for each age group and the cancer risk distributions are then summed across age groups.

The HARP software will perform an HRA using either OEHHA-provided or user-provided data distributions using a Monte Carlo analysis and will include the statistics for the distributions. In risk assessments that have chosen to use the distribution of exposure variates, the cancer risk distribution for a 30-year residential exposure duration (MEIR) should be presented in the risk characterization section We also recommend including the 9 and 70-year cancer risk at the MEIR as supplemental information. Note that a 70-year exposure duration is required to estimate cancer burden or provide an estimate of population-wide risk. A stochastic approach has not been developed for acute, 8-hour, and chronic noncancer health impacts or worker (MEIW) exposures.

8.2.9 *Use of Individual Cancer Risk and Population-wide Cancer Risk*

Cancer risk for an individual receptor and a representation of population-wide cancer risk are both important components of a risk assessment. The individual receptor approach reflects the exposures that may occur to an individual receptor over a period of time at a specific location. The individual cancer risk approach has some inherent limitations in terms of illustrating and potentially protecting population-based public health. For example, a facility with a small emissions footprint may impact a few individuals with a high individual potential cancer risk; whereas, a facility with a larger emission footprint may have a lower potential cancer risk for an individual receptor but expose many more people to those levels. Since this larger emitting facility can impact many more people, the population-wide health impacts are magnified due to the larger number of people exposed to the facility's emissions. This potential for higher population impacts is not captured by the individual receptor risk methodology. Therefore, the individual and population-wide heath impacts should be presented for all facilities to provide a more complete illustration of the facility's health impacts.

8.2.9.1 Population Risk

For facilities with large emission footprints (e.g., refineries, ports, or rail yards, etc.), population-based health impacts are critical to provide a better illustration of the potential impacts of emissions since large numbers of people may be exposed to the emissions. The individual cancer risk approach has some inherent limitations in terms of protecting public health. A small facility with a single stack can impact a few individuals with an individual cancer risk that is unacceptable, whereas a large facility may have an individual cancer risk that is below the acceptable limit for individual risk but exposes many more people. Thus, the population-wide impacts are larger for the large facility. Population-wide risk is independent of individual risk, and assumes a population (not necessarily the same individuals) will live in the impacted zone over a

70-year period. Thus, a 70-year exposure duration is required for estimates of population-wide risks.

To evaluate population risk, one method that regulatory agencies have used is the cancer burden method to account for the number of excess cancer cases that could occur in a population.

Cancer Burden

The cancer burden can be calculated by multiplying the cancer risk at a census block centroid by the number of people who live in the census block, and adding up the estimated number of potential cancer cases across the zone of impact. The result of this calculation is a single number that is intended to estimate of the number of potential cancer cases within the population that was exposed to the emissions for a lifetime (70 years).
The cancer burden is calculated on the basis of lifetime (70-year) risks (whereas individual cancer risk at the MEIR is based on 30-year residential exposure). Cancer burden is independent of how many people move in or out of the vicinity of an individual facility. For example, if 10,000 people are exposed to a carcinogen at a concentration with a 1×10^{-5} cancer risk for a lifetime the cancer burden is 0.1, and if 100,000 people are exposed to a 1×10^{-5} risk the cancer burden is 1.

Estimate of Population Wide Risk

An estimate of the number of people exposed at various cancer risk levels can provide perspective on the magnitude of the potential public health threat posed by a facility. This approach is intended as a replacement for or addition to the cancer burden calculation used by some Districts in the past. The new approach provides a much easier way for the general public to interpret results when compared to cancer burden estimates. A facility in a sparsely populated area can have a public health impact different from the same facility in a highly populated area; however, under the cancer burden method, those differences may not be seen. Some suggested approaches and methods for performance of a screening or refined population exposure analyses are provided in Section 4.6.

The District or reviewing authority should be consulted before beginning the population exposure estimates and, as results are generated, further consultation may be necessary. Note that a 70-year exposure duration is required to estimate cancer burden or provide an estimate of population-wide risk.

The zone of impact for estimating the number of persons exposed to a cancer risk from facility emissions should be set at a minimum of a 10^{-6} cancer risk level (see Section 4.6.1). Some Districts may prefer to use a cancer risk of 10^{-7} to define the carcinogenic zone of impact. The total number of persons exposed to a series of potential risk levels can be presented to aid risk managers in understanding the magnitude of the potential public health impacts.

The HARP software can provide population-level risk estimates as cancer burden or as the number of persons exposed to a selected (user-identified) cancer risk level at block level centroids.

8.2.9.2 Population Estimates for Noncancer Health Impacts

A noncancer chronic, 8-hour, and acute population estimate of the number of people exposed to acute, 8-hour, and chronic HQs or HIs exceeding 0.5 or 1.0, in increments of 1.0, should also be presented. For example, a facility with a maximum chronic HI of 4.0 would present the number of people exposed to a chronic HI of 0.5, 1.0, 2.0, 3.0, and 4.0. The isopleths used in this determination should be drawn using the smallest feasible grid size. The same methods that are described in Chapter 4 and Section 8.2.9 (for the population exposure estimate for cancer risk) should be used in the chronic, 8-hour and acute population estimates. Population estimates for acute, 8-hour, and chronic health impacts should be presented separately.

8.2.9.3 Factors That Can Impact Population Risk – Cumulative Impacts

Although the Hot Spots program is designed to address the impacts of single facilities and not aggregate or cumulative impacts, there are a number of known factors that influence the susceptibility of the exposed population and thus may influence population risk. Socioeconomic status influences access to health care, nutrition, and outcome after cancer diagnosis. Community unemployment can affect exposure and residency time near a facility. Factors that affect the vulnerability of the population are discussed in the report *Cumulative Impacts Building a Scientific Foundation* (OEHHA, 2010). Information on many of these factors is relatively easy to obtain at the census tract level. The OEHHA recommends that these types of factors be considered by the risk manager, along with the quantitative measures of population risk. OEHHA is in the process of developing guidance on quantification of the impact of these factors.

8.2.10 *Cancer Risk Evaluation of Short Term Projects*

The local air pollution control districts sometimes use the risk assessment guidelines for the Hot Spots program in permitting decisions for short-term projects such as construction or waste site remediation. Frequently, the issue of how to address cancer risks from short-term projects arises.

Cancer potency factors are based on animal lifetime studies or worker studies where there is long-term exposure to the carcinogenic agent. There is considerable uncertainty in trying to evaluate the cancer risk from projects that will only last a small fraction of a lifetime. There are some studies indicating that dose rate changes the potency of a given dose of a carcinogenic chemical. In others words, a dose delivered over a short time period may have a different potency than the same dose delivered over a lifetime.

The OEHHA's evaluation of the impact of early-in-life exposure has reduced some of the uncertainty in evaluating the cancer risk to the general population for shorter-term exposures, as it helps account for susceptibility to carcinogens by age at exposure (OEHHA, 2009).

Due to the uncertainty in assessing cancer risk from very short-term exposures, we do not recommend assessing cancer risk for projects lasting less than two months at the MEIR. We recommend that exposure from projects longer than 2 months but less than 6 months be assumed to last 6 months (e.g., a 2-month project would be evaluated as if it lasted 6 months). Exposure from projects lasting more than 6 months should be evaluated for the duration of the project. In all cases, for assessing risk to residential receptors, the exposure should be assumed to start in the third trimester to allow for the use of the ASFs (OEHHA, 2009). Thus, for example, if the District is evaluating a proposed 5-year mitigation project at a hazardous waste site, the cancer risks for the residents would be calculated based on exposures starting in the third trimester through the first five years of life.

For the MEIW, we recommend using the same minimum exposure requirements used for the residential receptor (i.e., no evaluation for projects less than 2 months; projects longer than 2 months but less than 6 months be assumed to last 6 months; projects longer than 6 months would be evaluated for the duration of the project). Although the off-site worker scenario assumes that the workers are 16 years of age or older with an Age-Sensitivity Factor of 1, another risk management consideration for short-term project cancer assessment is whether there are women of child bearing age at the worksite and whether the MEIW receptor has a daycare center. In this case, the Districts may wish to treat the off-site MEIW in the same way as the residential scenario to account for the higher susceptibility during the third trimester of pregnancy, and for higher susceptibility of infants and children.

Finally, the risk manager may want to consider a lower cancer risk threshold for risk management for very short-term projects. Typical District guidelines for evaluating risk management of Hot Spots facilities range around a cancer risk of 1 per 100,000 exposed persons as a trigger for risk management. Permitting thresholds also vary for each District. There is valid scientific concern that the rate of exposure may influence the risk – in other words, a higher exposure to a carcinogen over a short period of time may be a greater risk than the same total exposure spread over a much longer time period. In addition, it is inappropriate from a public health perspective to allow a lifetime acceptable risk to accrue in a short period of time (e.g., a very high exposure to a carcinogen over a short period of time resulting in a 1×10^{-5} cancer risk). Thus, consideration should be given for very short term projects to using a lower cancer risk trigger for permitting decisions.

8.3 Noncancer Acute, 8-Hour, and Chronic Inhalation Health Impacts – the Hazard Index Approach

All substances in the Hot Spots Program that have noncancer health impacts at a receptor must be evaluated through the inhalation pathway. Estimates of noncancer inhalation health impacts are determined by dividing an airborne concentration at the receptor by the appropriate Reference Exposure Level (REL). This is termed the Hazard Index Approach. A REL is used as an indicator of potential noncancer health impacts and is defined as the concentration at which no adverse noncancer health effects are anticipated. When a health impact calculation is performed for a single substance, then it is called the hazard quotient (HQ). Each REL for a substance will have one or more target organ systems (e.g., respiratory system, nervous system, etc.) where the substance can have a noncancer health impact. Thus, all HQs have specified target organ systems associated with them. The sum of the Hazard Quotients of all chemicals emitted that impact the same target organ is termed the Hazard Index.

Inhalation RELs for noncancer health impacts have been developed for acute, 8-hour, and chronic exposures to a number of Hot Spots substances. Acute RELs are designed to protect against the maximum 1-hour ground level concentration at the receptor. Eight-hour RELs are designed to protect people with daily 8-hour schedules, such as offsite workers, in an impacted zone. Chronic RELs protect against long-term exposure to the annual average air concentration spread over 24 hours/day, 7 days/week.

OEHHA has added 8-hour RELs to the set of noncancer RELs that were previously comprised of acute and chronic RELs (OEHHA, 2008). Specifically, 8-hour RELs are air concentrations at or below which health impacts would not be expected even for sensitive subpopulations in the general population with repeated daily 8-hour exposures over a significant fraction of a lifetime. The 8-hour RELs can be used to evaluate the potential for health impacts (including effects of repeated exposures) in offsite workers, and to children and teachers exposed during school hours. Although not required in the HRA, they could also be applied by the Districts to a residential scenario where a facility operates only a portion of the day and exposure to residences is not adequately reflected by averaging concentrations over a 24 hour day. The number of chemicals with 8-hour RELs will increase as OEHHA re-evaluates RELs for chemicals under SB-25 to ensure that they are protective of children's health.

Acute, 8-hour, and chronic RELs are needed because the dose metrics and even the health impact endpoints may be different with the different exposure durations of acute, daily 8-hour, and chronic exposures. Also, although chronic REL values are lower or set the same as 8-hour RELs, there are some cases such as special meteorological situations (e.g., significant diurnal-nocturnal meteorological differences) or intermittent exposures where the 8-hour REL may be more protective than the chronic REL.

Chapter 4 describes air dispersion modeling and both Chapter 6 and Appendix L list the needed dose-response information to evaluate non-cancer hazards. Appendix I presents sample calculations for determining acute HQs and HIs, 8-hour HQs and HIs, and chronic multipathway HQs and HIs. Chapter 9 provides an outline of information

required for risk characterization. The HARP software will calculate the HQ and HI for Hot Spots risk assessments.

8.3.1 *Calculation of Noncancer Inhalation Hazard Quotient and Hazard Index*

To calculate the acute HQ, the maximum 1-hour ground level concentration (in $\mu g/m^3$) of a substance at a receptor is divided by the acute 1-hour REL (in $\mu g/m^3$) for the substance:

$$\text{Acute Hazard Quotient} \quad \frac{\text{1-Hour Max Concentration } (\mu g/m^3)}{\text{Acute REL } (\mu g/m^3)}$$

To calculate the chronic HQ, the annual average ground level concentration of a substance is divided by the chronic REL for the substance:

$$\text{Chronic Hazard Quotient} \quad \frac{\text{Annual Average Concentration } (\mu g/m^3)}{\text{Chronic REL } (\mu g/m^3)}$$

To calculate the 8-hour HQ, the adjusted annual average ground level concentration of a substance (represented as "Adjusted C_{air}" in EQ 5.4.1.4 A) is divided by the 8-hour REL for the substance:

$$\text{8-hour Hazard Quotient} \quad \frac{\text{Adjusted Annual Average Concentration } (\mu g/m^3)}{\text{8-hour REL } (\mu g/m^3)}$$

The daily 8-hour average ground level concentrations used for calculating the 8-hour HQs are derived as described in Chapter 4.

An HQ of 1.0 or less indicates that adverse health effects are not expected to result from exposure to emissions of that substance. As the HQ increases above one, the probability of human health effects increases by an undefined amount. However, it should be noted that a HQ above one is not necessarily indicative of health impacts due to the application of uncertainty factors in deriving the RELs.

If a receptor is exposed to multiple substances that target the same organ system, then the HQs for the individual substances are summed to obtain a Hazard Index (HI) for that target organ.

Table 8.8 is an example of an HRA spreadsheet showing acute inhalation HQs arranged by target organ system for several substances. The bottom row shows the summed HQs by target organ system to derive the HIs.

Table 8.8 Individual Hazard Quotients and Total Hazard Index for Acute Inhalation Exposure

Substance	Reproductive/ Developmental	Nervous System	Cardiovascular System	Respiratory System	Eye
Ammonia				0.6	0.6
Arsenic	0.2	0.2	0.2		
Benzene	0.02				
Chlorine				0.7	0.7
Total Hazard Index	**0.22**	**0.2**	**0.2**	**1.3**	**1.3**

A more detailed example of calculating HQs and HIs, and determining noncancer health impacts is shown in Appendix I.

Hazard quotients or HIs for different target organs are not summed together (e.g., do not add the impacts for the eye to the cardiovascular system). Chapter 6 and Appendix L have lists of the organ systems affected by each substance. Unlike the cancer risk algorithms, no exposure duration adjustment (e.g., 9 yrs / 70 yrs) should be made for noncancer assessments.

There are limitations to this method of assessing cumulative noncancer health impacts. The impact on organ systems may not be additive if health effects occur by different mechanisms. However, the impact on organ systems could also be synergistic. An analysis by a trained health professional familiar with the substance's toxicological literature is usually needed to determine the public health significance of an HQ or HI above one. It is recommended that the Air District contact OEHHA if this situation presents itself. For assessing the noncancer health impacts of lead, different procedures are used; please see Appendix F.

8.3.2 *Calculating Noninhalation (oral) Noncancer Hazard Quotient and Hazard Index*

Similar to the situation with multipathway carcinogenic substances, multipathway substances that present a noncancer hazard are assessed by noninhalation routes of exposure (see Table 8.6). Noninhalation routes of exposure are assessed only for chronic exposure. There are no oral acute RELs since it is generally anticipated that health effects from a single exposure via the oral route at typical environmental levels resulting from deposition of facility emissions would be insignificant relative to the inhalation route. The multipathway substances with noninhalation RELs, called chronic oral RELs, are shown in Table 6.4. Similar to inhalation exposure, the hazard quotient

for a noninhalation pathway is obtained by dividing the dose in milligrams per kilogram-day (mg/kg-day) by the oral REL also expressed in units of mg/kg-day:

$$\text{Chronic Non-inhalation HQ} = \frac{\text{Chronic Noninhalation Dose (mg/kg-day)}}{\text{Chronic Oral REL (mg/kg-day)}}$$

The calculated chronic oral HQs are combined with the chronic inhalation HQs for determining the chronic HIs for each affected target organ (see Section 8.3.4). The point estimates and algorithms for calculating the oral dose for all applicable exposure pathways and receptors (e.g., workers or residents) are explained in Chapter 5.

The chronic oral dose calculated in mg/kg-day is based on a time-weighted average 70-year residential exposure combining the 0<2, 2<16 and 16-70 year age groups. Unlike the assessment of cancer risk, no exposure duration adjustment should be made when estimating HQs. In other words, the variates ED and AT in the cancer risk EQ 8.2.5 in Section 8.2.5 is not used for estimating the noncancer HQs. See Appendix I for an example calculation.

8.3.3 *Multipathway Noncancer Risk Methodology*

To determine multipathway chronic noncancer health impacts, it is necessary to calculate the total hazard index from both inhalation and noninhalation exposures. The calculation of HIs has several steps:

1) First, the inhalation HQ is calculated for each substance emitted (Section 8.3.1).
2) Second, if the substance has an oral REL, then the non-inhalation HQ is calculated as shown above using high-end point-estimates for intake rates for each noninhalation pathway that applies.
3) Third, if there are more than two noninhalation pathways to consider for a multipathway substance, then the oral HQ is calculated using high-end point estimates in the dose equation for the two dominant pathways. For any additional noninhalation pathways, the HQs are calculated using average point estimates in the dose equation. This step applies only to residential receptors.
4) Fourth, all noninhalation pathway HQs for a multipathway substance are then summed together by target organ to obtain the total noninhalation HQ for a multipathway substance.
5) The final step is to sum the inhalation and noninhalation HQs together by target organ to determine the HIs. This step is displayed in Table 8.9. If there is only one substance, then the multipathway HQ is the same as the HI.

Table 8.9 Substance-Specific Chronic Inhalation and Noninhalation Hazard Quotients and the Hazard Index by Target Organ System

Substance	Respiratory System	Hematologic System	Alimentary System	Endocrine System	Development	Reproductive System	Nervous System	Cardiovascular System	Skin
Ammonia	0.8								
Arsenic					0.04(i) 0.1(ni)		0.04(i) 0.1(ni)	0.04(i) 0.1(ni)	0.04(i) 0.1(ni)
Benzene		0.08			0.08		0.08		
2,3,7,8-TCDD (dioxin)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)			
Nickel	0.4(i)	0.4(i)	0.1(ni)						
Hazard Index	**1.50**	**0.78**	**0.40**	**0.3**	**0.52**	**0.30**	**0.94**	**0.14**	**0.14**

i = inhalation pathway contribution

ni = noninhalation pathway contribution

Table 8.9 shows the calculated chronic HIs by combining the chronic inhalation HQs and chronic oral HQs. The HQs or HIs for different target organs are not added together (e.g., do not add the impacts for the respiratory system to the nervous system). The noninhalation pathways for TCDD and arsenic in Table 8.9 have all the noninhalation pathways that apply incorporated into their HQ values. For example, the noninhalation value for arsenic (HQs = 0.1) include at least the soil ingestion and dermal soil pathways in the HQs because these are the mandatory noninhalation pathways to take into account with exposure to a multipathway substance. For TCDD, the mother's milk pathway is an additional mandatory noninhalation pathway to take into account (See Table 5.1). If there are exposures to any of the site-specific pathways, then these would be included too. A more detailed example calculation of HIs is shown in Appendix I.

When exposure to more than two noninhalation pathways occur, using the high-end point estimates of intake rates for only the two dominant noninhalation pathways will lessen the issue of compounding high-end exposure estimates, while retaining a health-protective approach for the more important exposure pathways. It is unlikely that an individual receptor would be on the high-end of exposure for all the non-inhalation intake parameters (exposure pathways).

8.3.4 *Summary - Acute, 8-Hour and Chronic Hazard Index Calculation at the MEIR and MEIW*

Eight-hour RELs were developed principally for exposure of individuals during 8-hour work schedules. There are currently only a limited number of substances with an 8-hour inhalation REL. Over time as the science supporting REL values for individual substances are reviewed and revised by OEHHA, more 8-hour RELs will be developed.

Therefore, for the MEIR, we recommend:

- Estimating the acute Hazard Index based on the maximum 1-hour air concentration and 1-hour RELs.
- Estimating the chronic Hazard Index based on the annual average air concentration and the chronic RELs, and the oral RELs for multipathway substances

An 8-hour hazard index based on the daily average 8-hour exposure is not required for the MEIR, but can be performed at the discretion of the District for exposure to non-continuously operating facilities using the adjusted annual average air concentration (See EQ 5.4.1.4 A and B or method in App. M). Eight-hour hazard assessments are not recommended for exposure to continuously operating facilities.

For the MEIW, we recommend:

- Estimating the acute Hazard Index based on the maximum 1-hour air concentration and 1-hour RELs.
- Estimating the 8-hour Hazard Index based on daily average 8-hour exposure for those chemicals with 8-hour RELs
- Estimating the chronic Hazard Index based on the annual average air concentration and chronic RELs, and oral RELs for multipathway substances

Until there are 8-hour RELs for many of the Hot Spots substances that have a chronic REL value, we recommend determining the chronic HI for the MEIW to adequately protect the offsite worker.

8.3.5 *Evaluation of Background Criteria Pollutants*

The District should be contacted to determine if the contribution of background criteria pollutants to respiratory health effects is required to be included in an HRA for the Hot Spots Program. If inclusion is required, the methods for calculating the health impact from acute and chronic exposure (respiratory endpoint) is the standard HI approach (see Sections 8.3.1 and 8.3.4). There are currently no 8-hour RELs for criteria pollutants, so 8-hour health impacts from criteria pollutants are not assessed in HRAs. The background criteria pollutant contribution should be calculated if the HI from the facility's emissions exceeds 0.5 in either the acute or chronic assessment for the respiratory endpoint.

The most recent criteria pollutant concentration data should be obtained from the ARB's ambient air monitoring network and can be found in the *California Almanac of Emissions and Air Quality* on their web site at *www.arb.ca.gov*. For determining the criteria pollutant contribution in HI calculations, the annual average concentration data should be taken from a monitoring site near the facility. If background contributions are unavailable, the District may direct the risk assessor to make an alternative assumption. The criteria pollutants that should be included in acute and chronic assessments for the respiratory endpoint are ozone, nitrogen dioxide, sulfur dioxide, sulfates, and hydrogen sulfide.

8.4 Uses of Exposure Duration Adjustments for Onsite Receptors

Onsite workers are protected by CAL OSHA and typically are not evaluated under the Hot Spots program. Exceptions may include a worker who also lives on the facility property such as at prisons, military bases, and universities that have worker housing within the facility. Another scenario where the District may require assessment of on-site worker exposure and risk is when a facility (e.g., airport) has multiple businesses owned by different entities within the facility/property (e.g., rental car agencies, restaurants, etc.). In these situations the evaluation of onsite cancer risks, and/or acute, 8-hour, and chronic noncancer hazard indices is appropriate under the Hot Spots program. If the onsite receptor under evaluation can be exposed through a noninhalation exposure pathway, then that exposure pathway must also be included. When a receptor lives and works on the facility, site, or property, then these receptors should be evaluated and reported under both residential and worker scenarios and the one that is most health-protective should be used for risk management decisions.

The cancer risk estimates for the on-site residents may use a 30-year exposure duration while the 25-year exposure duration is used for a worker. Under a Tier 2 analysis, alternate exposure durations may be evaluated and presented with all assumptions supported. See section 8.2.10 for more discussion of short-term exposures.

Other situations that may require on-site receptor assessment include the presence of locations where the public may have regular access for the appropriate exposure period (e.g., a lunchtime café, store, or museum for acute exposures). The District or reviewing authority should be consulted on the appropriate evaluations for the risk for all onsite receptors.

8.5 References

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9 - Summary of the Requirements for a Modeling Protocol and a Health Risk Assessment Report

The AB 2588 program is a community right-to-know act. Although risk assessment is a technical field, AB 2588 risk assessments need to be clear and understandable to the educated lay person. An Executive Summary that explains the process and the results of the risk assessment in lay terms is necessary. Clear risk communication is imperative in situations where the facility is required to notify the surrounding community. In addition, the risk assessment is by law reviewed by the local Air Pollution Control or Air Quality Management District (District) and OEHHA in order to ensure that AB 2588 risk assessment procedures have been followed. This chapter clarifies the type of information that is needed for District and OEHHA review of modeling protocols and health risk assessments (HRAs).

The material presented here is intended to promote transparent, consistent presentation and efficient review of the modeling protocol and the health risk assessment report (products). We recommend that persons preparing these products consult with the local District to determine if the District has modeling or HRA guidelines that supersede these products. If the District does not have guidelines for these products, then we recommend Section 9.1 be used for modeling protocols and Section 9.2 be used for the presentation of HRAs. Persons preparing modeling protocols and HRAs should specify the guidelines that were used to prepare their products.

9.1 Submittal of a Modeling Protocol

It is strongly recommended that a modeling protocol be submitted to the District for review and approval prior to extensive analysis with an air dispersion model. The modeling protocol is a plan of the steps to be taken during the air dispersion modeling and risk assessment process. We encourage people who are preparing protocols to take advantage of the protocol step and fully discuss anticipated methodologies for any portion of your project that may need special consideration. Below, we have provided an example of the format that may be followed in the preparation of the modeling protocol. **Consult with the District to confirm format and content requirements or to determine the availability of District modeling guidelines before submitting the protocol.**

9.1.1 *Outline for a Modeling Protocol*

I. Introduction

Include the facility name, address, and a brief overview describing the facility's operations.

- Provide a description of the terrain and topography surrounding the facility and potential receptors.
- Indicate the format in which data will be provided. Ideally, the report and summary of data will be on paper and all data and model input and output files will be provided electronically (e.g., compact disk or CD).
- Identify the guidelines used to prepare the protocol (e.g., District Guidelines).

II. Emissions

For each pollutant and process whose emissions are required to be quantified in the HRA, list the annual average emissions (pounds/year and grams/second) and the maximum one-hour emissions (pounds/hour and grams/second)[1]. Maximum 1-hour emissions are used for acute noncancer health impacts while annual emissions are used for chronic exposures (i.e., chronic and 8-hour noncancer health impacts or cancer risk assessment).

- Identify the reference and method(s) used to determine emissions (e.g., source tests, emission factors, etc.). Clearly indicate any emission data that are not reflected in the previously submitted emission inventory report. In this event, a revised emission inventory report will need to be submitted to the District.
- Identify if this will be a multipathway assessment based on emitted substances.

[1] Except radionuclides, for which annual and hourly emissions are reported in Curies/year and millicuries/hour, respectively.

III. Models / Modeling Assumptions

Specify the model and modeling assumptions

- Identify the model(s) to be used, including the version number.
- Identify the model options that will be used in the analysis.
- Identify the modeling domain(s) and the spacing of receptor grid(s). Grid spacing should be sufficient in number and detail to capture the concentration at all of the receptors of interest.
- Indicate complex terrain options that may be used, if applicable.
- Identify the source type(s) that will be used to represent the facility's operations (e.g., point, area, or volume sources, flare options or other).
- Indicate the preliminary source characteristics (e.g., stack height, gas temperature, exit velocity, dimensions of volume source, etc.).
- Identify and support the use of urban or rural dispersion coefficients for those models that require dispersion coefficients. For other models, identify and support the parameters required to characterize the atmospheric dispersion due to land characteristics (e.g., surface roughness, Monin-Obukhov length).

IV. Meteorological Data

Specify the type, source, and year(s) of hourly meteorological data (e.g., hourly surface data, upper air mixing height information).

- State how the data are representative for the facility site.
- Describe QA/QC procedures.
- Identify any gaps in the data; if gaps exist, describe how the data gaps are filled.

V. Deposition

- Specify the method to calculate deposition (if applicable).

VI. Receptors

Specify the type and location of receptors. Include all relevant information describing how the individual and population-related receptors will be evaluated.

- Identify and describe the location(s) of known or anticipated potential sensitive receptors, the point of maximum impact (PMI), the maximum exposed individual residential (MEIR), and worker (MEIW) receptors. Identify any special considerations or grids that will be used to model these receptors. This information should correspond with information provided in Section III (e.g., fine receptor spacing of 20 meters at the fence line and centered on the maximum impacts; coarse receptor spacing of 100 meters out to 2,000 meters; extra coarse spacing of 1,000 meters out to 20,000 meters).

- Identify if spatial averaging will be used. Include necessary background information on each receptor including how the domain and spacing will be determined for each receptor or exposure pathway.

- Describe how the cancer burden or population impact estimates are calculated. Clarify the same information for the presentation of noncancer population impacts (e.g., centroids of the census tracts in the area within the zone of impact).

- Specify that actual UTM coordinates and the block/street locations (i.e., north side of 3,000 block of Smith Street), where possible, will be provided for specified receptor locations.

- Identify and support the use of any exposure adjustments (e.g., time at location, diurnal).

- Include the list of anticipated exposure pathways that will be included and indicate which substance will be evaluated in the multipathway assessment. Identify if sensitive receptors are present and which receptors will be evaluated in the HRA.

VII. Maps

Identify how the information will be graphically presented.

- Indicate which cancer risk isopleths will be plotted for the cancer zone of impact (e.g., 10^{-7}, 10^{-6} see Section 4.6.1).

- Indicate the hazard quotients or hazard indices to be plotted for the noncancer acute, 8 hour, and chronic zones of impact (e.g., 0.5, 1.0, etc.).

9.2 Health Risk Assessment Report

The purpose of this section is to provide an outline to assist with the preparation and review of HRAs. This outline specifies the key components that should be included in HRAs. All information used for the report must be presented in the HRA. Ideally, the HRA report and a summary of data used in the HRA will be on paper and all data and model input and output files will be provided electronically (e.g., CD). Persons preparing HRAs for the Hot Spots Program should consult the District to determine if HRA guidelines or special formats are to be followed when preparing and presenting the HRA's results.

If District guidelines or formats do not exist that supersede this outline, then the HRA should follow the format presented here. If the HRA is prepared for other programs, the reviewing authority should be consulted for clarification of format and content. We recommend that those persons preparing HRAs specify the guidelines that were used to prepare their product. **The HRA may be considered deficient by the reviewing authority if components that are listed here are not included**.

9.2.1 *Outline for the Health Risk Assessment Report*

I. Table of Contents

- Section headings with page numbers indicated.
- Tables of tables and Table of figures with page numbers indicated.
- Appendices with page numbers indicated.

II. Executive Summary

Overview of all relevant information regarding the project or facility.

- Facility identifier number (consult the District).

- Description of facility operations and a list identifying emitted substances including table of maximum 1-hour emissions, and annual average emissions.

- Provide a brief description of acute, 8-hour, chronic, and cancer health impacts of the emitted substances, based on OEHHA's descriptions in the appropriate Technical Support Documents.

- Text presenting overview of dispersion modeling and exposure assessment.

- Text describing estimated cancer risk for carcinogens, noncancer Hazard Quotients and Hazard Indices and a table showing target organ systems by substance for noncancer impacts.

- Summarize the individual and population-wide health impacts including the driving substance(s) and the driving exposure pathways:

  - Location (block/street location; e.g., north side of 3,000 block of Smith Street) and description of the off-site point of maximum impact (PMI), maximum exposed individual resident (MEIR), and maximum exposed individual worker (MEIW).

  - Location (block/street location; e.g., north side of 3,000 block of Smith Street) and description of any on-site receptors that were evaluated at the facility (consult District or agency).

  - Location (block/street location; e.g., north side of 3,000 block of Smith Street) and description of any sensitive receptors that are required by the district or reviewing authorities (consult District or agency).

    NOTE: When presenting information described in the following bullets, cancer risk should be presented separately for a residential 30-year, Tier–1 analysis. Results of other exposure assumptions (e.g., 9 or 70-year) or other tier evaluations should also be presented, and must be clearly labeled. For the Hot Spots Program, while the 30-year exposure duration is recommended as the basis for public notification and risk reduction audits and plans, the District has discretion to use the 70 year exposure scenario for its decisions. In addition, the 70 year cancer risk must be calculated to estimate population-wide impacts.

  - Text presenting an overview of the total cancer risk (including multipathway substances, if present) at the PMI, MEIR, MEIW, and sensitive receptors. Provide a table of cancer risk by substance for the MEIR and MEIW (if applicable). Include a statement indicating which of the substances appear to contribute most to (drive) the potential health impacts. In addition, identify the exposure pathways evaluated in the HRA.

  - Provide a map of the facility and surroundings and identify the location of the MEIR, MEIW, PMI, and other locations or receptors of interest.

  - Provide a map of 30-year and 70-year cancer risk zone of impact(s), if applicable.

  - Text presenting an overview of the acute and chronic noncancer hazard quotients and the (total) hazard indices for the PMI, MEIR,

MEIW, and sensitive receptors. Additionally, include 8-hour hazard quotients and hazard indices for the MEIW. Include separate statements (for acute, 8-hour, and chronic exposures) indicating which of the substances appear to drive the potential health impacts. In addition, clearly identify the primary target organ(s) that are impacted from acute, 8-hour, and chronic exposures.

- Identify any sensitive subpopulations (e.g., child daycare facilities, schools, nursing homes) of concern.

- Table and text presenting an overview of estimates of population exposure (e.g., cancer burden or population estimates from HARP) (consult District or agency) (see Section 8.4).

- Version of the Risk Assessment Guidelines and computer program(s) used to prepare the risk assessment (e.g., HARP).

III. Risk Assessment Procedures

*A. **Hazard identification***

- Table and text identifying all substances emitted from the facility, plus any other substances required by the District or reviewing authority. Include the CAS number of the substance and the physical form of the substance if possible. [The Hot Spots substances are listed in Appendix A, and also in the ARB's Emission Inventory Criteria and Guidelines Regulations (Title 17, California Code of Regulations, Sections 93300-93300.5), and the Emission Inventory Criteria and Guidelines Report (EICG Report), which is incorporated by reference therein (ARB, 1997)].

- Table and text identifying all substances that are evaluated for cancer risk and/or noncancer acute, 8-hour, and chronic health impacts. In addition, identify any multipathway substances that present a cancer risk or chronic noncancer hazard via noninhalation routes of exposure.

- Describe the types and amounts of continuous or intermittent predictable emissions from the facility that occurred during the reporting year. As required by statute, releases from a facility include spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping (fugitive), leaching, dumping, or disposing of a substance into ambient air. Include the substance(s) released and a description of the processes that resulted in long-term and continuous releases.

*B. **Exposure Assessment***

This section describes the information related to the air dispersion modeling process that needs to be reported in the risk assessment; the information is also

presented in Chapter 4 (see Section 4.15). The District may have specific requirements regarding format and content (see Section 4.14). Sample calculations should be provided at each step to indicate how reported emissions data were used. Reviewing agencies must receive input, output, and supporting files of various model analyses on computer-readable media (e.g., CD).

1. Information on the Facility and its Surroundings

Report the following information regarding the facility and its surroundings:

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

2. Source and Emission Inventory Information

a. Release Parameters

Report the following information for each release location in table format:

- Release location identification number
- Release name
- Release type (e.g., point, volume, area, line, pit, etc.)
- Source identification number(s) used by the facility that emit out of this release location
- Release location using UTM coordinates
- Release parameters by release type (e.g., shown for point source):
- Stack height (m), stack diameter (building dimensions for downwash, exhaust gas exit velocity (m/s), exhaust gas volumetric flow rate (ACFM), exhaust gas exit temperature (K), etc.

b. Source Description and Operating Schedule

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

- Source identification number used by the facility
- Source name

- Number of operating hours per day and per year (e.g., 0800-1700, 2700 hr/yr)
- Number of operating days per week (e.g., Mon-Sat)
- Number of operating days or weeks per year (e.g., 52 wk/yr excluding major holidays)
- Release point identification number(s) for where source emissions are released
- Fraction of source emissions emitted at each release point by release point ID number

c. *Emission Control Equipment and Efficiency*

- Report emission control equipment and efficiency by source and by substance

d. *Emissions Data Grouped By Source*

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

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

e. *Emissions Data Grouped by Substance*

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

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

f. *Emission Estimation Methods*

Report the methods used in obtaining the emissions data indicating whether emissions were measured or estimated. Clearly indicate any emission data that are not reflected in the previously submitted emission inventory report and submit a revised emission inventory report to the

district. A reader should be able to reproduce the risk assessment without the need for clarification.

g. *List of Substances*

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

h. *Exposed Population and Receptor Location*

Report the following information regarding exposed population and receptor locations:

- Description of zone of impact including map showing the location of the facility, boundaries of zone of impact, census tracts, emission sources, sites of maximum exposure, and the location of all appropriate receptors. This should be a true map (one that shows roads, structures, etc.), drawn to scale, and not just a schematic drawing. USGS 7.5 minute maps or GIS based maps are usually the most appropriate choices. (If significant development has occurred since the user's survey, this should be indicated.)

- Separate maps for the cancer risk zone of impact and the hazard index (noncancer) zone of impact(s). The cancer zone of impact should include isopleths down to at least the 1/1,000,000 risk level. Because some districts use a level below 1/1,000,000 to define the zone of impact, the District should be consulted. For the noncancer zone of impact, three separate isopleths (to represent chronic, 8-hour, and acute HI) should be created to define the zone of impact for the hazard index from both inhalation and noninhalation pathways greater than or equal to 0.5. The point of maximum impact (PMI), maximum exposed individual at a residential receptor (MEIR), and maximum exposed individual worker (MEIW) for both cancer and noncancer risks should be located on the maps.

- Tables identifying population units and sensitive receptors (UTM coordinates, receptor IDs or index from the modeling, and street addresses of specified receptors)

- Heights or elevations of the receptor points.

- **Spatial averaging**: For each receptor type (e.g., PMI, MEIR, and MEIW, or other location of interest) that will utilize spatial averaging, the domain size and grid resolution must be clearly identified. If another domain or grid resolution other than 20 meters by 20 meters with 5-meter grid spacing will be used for a receptor, then care should

be taken to determine the proper domain size and grid resolution that should be used. For a worker, the HRA shall support all assumptions used, including, but not limited to, documentation for all workers showing the area where each worker routinely performs their duties. The final domain size should not be greater than the smallest area of worker movement. Other considerations for determining domain size and grid spacing resolution may include an evaluation of the concentration gradients across the worker area. The grid spacing used within the domain should be sufficient in number and detail to obtain a representative concentration across the area of interest. When spatial averaging over the deposition area of a pasture, garden, or water body, care should be taken to determine the proper domain size to make sure it includes all reasonable areas of potential deposition. The size and shape of the pasture, garden, or water body of interest should be identified and used for the modeling domain. The grid spacing or resolution used within the domain should be sufficient in detail to obtain a representative deposition concentration across the area of interest. One way to determine the grid resolution is to include an evaluation of the concentration gradients across the deposition area. The HRA shall support all assumptions used, including, but not limited to, documentation of the deposition area (e.g., size and shape of the pasture or water body, maps, representative coordinates, grid resolution, concentration gradients, etc.). The use or spatial averaging is subject to approval by the reviewing authority. This includes the size of the domain and grid resolution that is used for spatial averaging of a worksite or multipathway deposition area.

3. Meteorological Data

If meteorological data were not obtained directly from the District, then the report must clearly indicate the data source and time period used. Meteorological data not obtained from the District must be submitted in electronic form along with justification for their use including information regarding representativeness and quality assurance.

The risk assessment should indicate if the District required the use of a specified meteorological data set. All memos indicating the District's approval of meteorological data should be attached in an appendix.

4. Model Selection and Modeling Rationale

The report should include an explanation of the model chosen to perform the analysis and any other decisions made during the modeling process. The report should clearly indicate the name of the models that were used, the level of detail (screening or refined analysis) and the rationale behind the selection.

Also report the following information for each air dispersion model used:

- Version number
- Selected options and parameters in table form
- Identify the modeling domain(s) and the spacing of receptor grid(s). Grid spacing should be sufficient in number and detail to capture the concentration at all receptors of interest.

5. Air Dispersion Modeling Results

The report should include tables, text, and appendices that clearly present all of the following information

- Maximum hourly and annual average concentrations of chemicals at appropriate receptors such as the residential and worker MEI receptors

- Annual average and maximum one-hour (and 30-day average for lead only) concentrations of chemicals at appropriate receptors listed and referenced to computer printouts of model outputs

- Model printouts (numbered), annual concentrations, maximum hourly concentrations

- Disk with input/output files for air dispersion program (e.g., the AERMOD input file containing the regulatory options and emission parameters, receptor locations, meteorology, etc.)

- Include tables that summarize the annual average concentrations that are calculated for all the substances at each site. The use of tables that present the relative contribution of each emission point to the receptor concentration is recommended. (These tables should have clear reference to the computer model which generated the data. It should be made clear to any reader how data from the computer output 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.]

C. ***Health Values Used in Dose-Response and Dose Estimates***

- Provide tables of the acute, 8-hour and chronic inhalation RELs, chronic oral RELs (if applicable), and cancer potency factors for each substance that is quantified in the HRA.

- Identify the guidelines (title and date) that were used to obtain these factors, or indicate whether newly approved values obtained from the OEHHA website were used.

- Provide a table of target organ systems for each noncancer substance, including acute (1 hour), 8-hour, and chronic inhalation, and chronic oral (if applicable).

- Include tables of the estimated dose for each substance by each exposure pathway at the PMI, MEIR, MEIW, and at any sensitive receptor locations (required by the District).

D. ***Risk Characterization***

The Hot Spots Analysis and Reporting Program (HARP) will generate the risk characterization data needed for the outline below. Any data needed to support the risk characterization findings should be clearly presented and referenced in the text and appendices. A listing of HARP output files that meet these HRA requirements are provided in this outline under the section entitled "Appendices". All HARP files should be included in the HRA. Ideally, the HRA report and a summary of data used in the HRA will be on paper and all data and model input and output files will be provided electronically (e.g., CD). Information on obtaining copies of HARP is available on the California Air Resources Board's Internet web site under the Air Toxics Program at *www.arb.ca.gov.*

NOTE: The cancer risk for the PMI, MEIR, and sensitive receptors of interest must be presented in the HRA's text, tables, and maps. OEHHA recommends that cancer risk for a 30-year exposure duration be presented for the MEIR, and that cancer risk for 9-year and 70-year exposure durations for the MEIR be presented to provide the risk managers with supplemental information. Note that the assessment of population impacts must be based on a 70-year exposure duration; thus all risk assessments need to estimate cancer risk for a 70-year exposure duration in order to report the number of individuals residing in the risk isopleths, or to calculate cancer burden if the District so requires. In addition, some Districts may opt to make risk management decisions based on a 70-year exposure duration. The MEIW location should use a 25-year exposure period.

All HRAs must include the results of a Tier-1 exposure assessment (see Chapter 2 and 8, or the 2012 TSD). If the reviewing authority specifies that additional exposure periods should be presented, or if persons preparing the HRA would like to present additional information (i.e., exposure duration adjustments or the inclusions of risk characterizations using Tier-2 through Tier-4 exposure data), then this information should be presented in separate, clearly titled, sections, tables, and text.

The following information should be presented in this section of the HRA. If not fully presented here, then by topic, clearly identify the section(s) and pages within the HRA where this information is presented.

- Description of receptors to be quantified.

- Table and text providing the location [UTM coordinates, receptor ID number or index from the modeling, and the block/street address (e.g., north side of 3,000 block of Smith Street)] and description of the PMI, MEIR, and MEIW for both cancer and noncancer risks.

- Separate tables and text providing description of the PMI and MEIR for 30-year cancer risk, and 9- or 70-year cancer risk,.

- Tables and text describing MEIW 25-year cancer risk.

- Table and text providing the location [UTM coordinates, receptor ID number or index from the modeling, and the block/street address (e.g., north side of 3,000 block of Smith Street)] and description of any sensitive receptors that is of interest to the District or reviewing authorities (consult District or agency).

- Provide any exposure information that is used for risk characterization (e.g., concentrations at receptors, emissions information, census information, figures, zone of impact maps, etc.). If multipathway substances are emitted, identify the site/route dependent exposure pathways (e.g., water ingestion) for the receptor(s), where appropriate (e.g., MEIR).

- Provide a summary of the site-specific inputs used for each exposure pathway (e.g., water or grazing intake assumptions). This information may be presented in the appendix with the information clearly presented and cross-referenced to the text. In addition, provide reference to the appendix (section and page number) that contains the modeling (i.e., HARP/dispersion modeling) files that show the same information.

- If any exposure parameters were used other than those provided in the Air Toxics Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis (2012), they must be presented in detail. The derivation and data used must be presented so that it is clear to the reviewer. The justification for using site-specific exposure parameters must be clearly presented.

- Table and text presenting the potential multipathway cancer risk by substance, by pathway, and total, at the PMI, MEIR, MEIW, and sensitive receptor locations (required by the District).

- Table and text presenting the acute (inhalation only) and chronic noncancer (inhalation and oral) hazard quotients (by substance, exposure pathways, and target organs) and the (total) hazard indices by substance and target organs for the PMI, MEIR, MEIW, and sensitive receptors. For 8-hour exposure at the MEIW (inhalation only), table and text presenting

hazard quotients (by substance, exposure pathways, and target organs) and the (total) hazard indices by substance and target organs. Note: Chronic noncancer results should be shown with inhalation and oral contributions (shown separately) and for the combined (multipathway) impact.

- Identify any sensitive subpopulations (e.g., child daycare facilities, schools, nursing homes) of concern.

- Table and text presenting estimates of population exposure (e.g., population exposure estimates or cancer burden from HARP) (consult District or agency). Tables should indicate the number of persons exposed to a (total) cancer risk greater than 10^{-7}, 10^{-6}, 10^{-5}, 10^{-4}, etc., and total hazard quotient or hazard index greater than 0.5, 1.0, 2.0, and 3.0, etc. Provide a table that shows excess cancer burden for each population unit and the total excess cancer burden, if cancer burden calculation is required.

- Provide maps that illustrate the HRA results for the three sub-bullet points below. These maps should be an actual street map of the area impacted by the facility with elevation contours and actual UTM coordinates, and the facility boundaries clearly labeled. In some cases the elevation contours will make the map too crowded and should therefore not appear. This should be a true map (one that shows roads, structures, etc.), drawn to scale, and not just a schematic drawing. USGS 7.5-minute maps are usually the most appropriate choice (see Section 4.6).

    - The facility (emission points and boundaries), the locations of the PMI, MEIR, MEIW, and sensitive receptors.

    - Maps of the cancer zone of impacts (e.g., 10^{-6} or 10^{-7} levels - consult District or Agency). The map should clearly identify the zone of impact for the inhalation pathway, the minimum exposure pathways (soil ingestion, dermal exposure, and breast-milk consumption) if multipathway substances are emitted, and the zone of impact for all the applicable exposure pathways (minimum exposure pathways plus any additional site/route specific pathways) for multipathway analyses. Two maps may be needed to accomplish this. The legend of these maps should state the level(s) used for the zone of impact and identify the exposure pathways that were included in the assessment.

    - Maps of the noncancer hazard index (HI) zone of impacts (e.g., 0.5 or 1.0 - consult District or Agency). The noncancer maps should clearly identify the noncancer zones of impact. These include the acute (inhalation), 8-hour (inhalation), chronic (inhalation), and chronic (multipathway) zones of impact. For clarity, presentation of the

noncancer zones of impact may require two or more maps. The legend of these maps should state the level(s) used for the zone of impact and identify the exposure pathways.

- The risk assessor may want to include a discussion of the strengths and weaknesses of the risk analyses and associated uncertainty directly related to the facility HRA.

- If appropriate, comment on the possible alternatives for control or remedial measures. How do the risks compare?

- If possible, identify any community concerns that influence public perception of risk.

- Sample calculations may be needed for all analyses in the HRA if proprietary software other than HARP was used. The District should be consulted. These calculations should be clearly presented and referenced to the findings they are supporting in the HRA text.

- Version of the Risk Assessment Guidelines and computer program used to prepare the risk assessment.

- If software other than HARP is used for the health assessment modeling, all supporting material must be included with the HRA (e.g., all algorithms and parameters used in a clear, easy to review format).

E. ***References***

Include any references used for the HRA in this section.

F. ***Appendices***

The appendices should contain all data, sample calculations, assumptions, and all modeling and risk assessment files that are needed to reproduce the HRA results. Ideally, a summary of data used in the HRA will be on paper and all data and model input and output files will be provided electronically (e.g., CD), unless otherwise specified by the district or reviewing authority. All appendices and the information they contain should be referenced, clearly titled, and paginated.

Potential Appendix Topics (if not presented elsewhere in the HRA report):

- List of all receptors locations (UTM coordinates, receptor ID number or index from the modeling, and the block/street address (e.g., north side of 3,000 block of Smith Street)) for the PMI, MEIR, MEIW, and sensitive receptors.
- List of all emitted substances.

- All emissions files.
- List of dose-response factors (Reference Exposure Levels and cancer potency factors).
- All air dispersion modeling input and output files. Detailed discussions of meteorological data, regulatory options, emission parameters, receptor locations, etc.
- Census data.
- Maps.
- Identify the site/route dependent exposure pathways for the receptor(s), where appropriate (e.g., MEIR). Provide a summary of the site-specific inputs used for each pathway (e.g., water or grazing intake assumptions) and the data to support them.
- All calculations used to determine emissions, concentrations, and potential health impacts at the PMI, MEIR, MEIW, and sensitive receptors.
- All HRA model input and output (HARP) files for receptors of concern.
- (Total) cancer and noncancer impacts by receptor, substance, and exposure pathway (by endpoint for noncancer) at all receptors.
- Presentation of alternate risk assessment methods (e.g., alternate exposure durations, or Tier-2 to Tier-4 evaluations with supporting information).

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