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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Executive Summary

The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (OEHHA, 2003) (Guidance Manual) is a concise description of the algorithms, recommended exposure variates, and cancer and noncancer health values needed to perform a health risk assessment (HRA) under the Air Toxics Hot Spots Information and Assessment Act of 1987 (Hot Spots or AB 2588) (AB 2588, Connelly, Statutes of 1987; Health and Safety Code Section 44300 et seq.) (see Appendix B). The information presented in the Guidance Manual is a compilation of information presented in the four technical support documents (TSDs) released by the Office of Environmental Health Hazard Assessment (OEHHA) for the Hot Spots Program. The four TSDs underwent public comment and peer review and were adopted for use in the Air Toxics Hot Spots program by the Director of OEHHA. These four TSDs present detailed information on cancer and noncancer health effects values and exposure pathway information. Excerpts of these four documents are presented in this document. All four TSDs are available on OEHHA's web site at *www.oehha.ca.gov*. There is relatively little new information in the Guidance Manual since the previously adopted TSDs form the basis of the Guidance Manual.

The Guidance Manual supercedes the risk assessment methods presented in *The California Air Pollution Control Officer's Association (CAPCOA) Air Toxics Hot Spots Program; Revised 1992; Risk Assessment Guidelines, October 1993* (CAPCOA, 1993). The Guidance Manual scientifically updates health effects values, exposure pathway variates (e.g., breathing rates), and presents a tiered approach for performing HRAs. The tiered approach 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 assumptions or the stochastic treatment of data distributions. The four-tiered approach to risk assessment primarily applies to residential cancer risk assessment. OEHHA is not recommending a stochastic approach (Tier-3 and Tier-4) for worker receptors or for noncancer chronic evaluations. Only Tier-1 applies to acute exposure evaluations. Compared to the CAPCOA 1993 document, the exposure pathways in the Guidance Manual remain the same, the exposure algorithms are similar, and risk algorithms have been revised to accept the data needed for the tiered risk assessment approach.

The Guidance Manual also contains example calculations and an outline for a modeling protocol and a HRA report. A software program, the Hot Spots Analysis and Reporting Program (HARP), has been developed by a contractor in consultation with OEHHA, the Air Resources Board (ARB), and the 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 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 in developing this Guidance Manual and the HARP software is 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

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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.

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1. Introduction

1.1 Development of Guidelines

The Hot Spots 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 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 Risk Assessment Guidelines*; *Part IV; Exposure Assessment and Stochastic Analysis Technical Support Document (OEHHA, 2000b)* (Part IV TSD). A summary of this information can be found in Chapter 5 of this document.

Cancer and noncancer (acute and chronic) dose-response relationships (health effects values) for many Hot Spots substances are presented in the first three Technical Support Documents. *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part I; The Determination of Acute Reference Exposure Levels for Airborne Toxicants (OEHHA, 1999a)* presents acute Reference Exposure Levels (RELs) for 51 toxicants and toxicant compound classes. *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II; Technical Support Document for Describing Available Cancer Potency Factors (OEHHA, 1999b and 2002)* contains inhalation cancer potency factors and oral cancer potency factors for 122 toxicants and toxicant compound classes developed by OEHHA or developed by other authoritative bodies and endorsed by OEHHA. *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part III; Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels (OEHHA, 2000a)* documents the development of chronic noncancer inhalation RELs for 72 toxicants and toxicant classes. The OEHHA website (*www.oehha.ca.gov*) should be consulted for chronic RELs adopted subsequent to (OEHHA, 2000a). 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 are presented. 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 four Technical Support Documents have undergone public and peer review and have been endorsed by the state's Scientific Review Panel on Toxic Air Contaminants and adopted by OEHHA. The Guidance Manual has also undergone the same public and peer review process.

The Guidance Manual contains a concise description of the algorithms, recommended exposure variates, and cancer and noncancer health values 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 other four TSDs. The Guidance Manual is the successor document to *The CAPCOA Air Toxics "Hot*

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Spots" Program; Revised 1992; Risk Assessment Guidelines, October 1993 prepared by CAPCOA (CAPCOA, 1993). The Guidance Manual scientifically updates risk assessment variates and presents a tiered approach including a stochastic as well as a point-estimate approach to exposure and risk assessment. The exposure pathways remain the same and the algorithms are similar to the 1993 CAPCOA document.

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 an 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 new or modified stationary sources. See the ARB's website at *www.arb.ca.gov* for more information on the Hot Spots Program and for risk management guidelines that provide recommendations for permitting 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. However, 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.

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 (ARB, 1997*)*. 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 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 populationwide 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 are to 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)*. 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 a single integrated software package 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 consists of three modules that include: 1) the Emissions Inventory Database Module, 2) the Air Dispersion Modeling Module, and 3) the Risk Analysis and Mapping Module. 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 and identification;
5. Air dispersion modeling (ISCST3) of multiple emission releases or facilities for cumulative impact evaluations;

6. A summary report of acute and chronic health hazard quotients or indices, and cancer risk at the point of maximum impact (PMI), maximally exposed individual resident (MEIR), and the maximally exposed individual worker (MEIW). (Other receptors may be evaluated as needed.);
7. Mapping displays of facility property boundaries, risk isopleths, street maps, and elevation contours;
8. The ability to display combined risk contours from multiple facilities;
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 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 either 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. Uncertainty may be defined as what is not known and may be reduced with further scientific studies. In addition to uncertainty, there is a natural range or variability in the human population in such properties as height, weight, and susceptibility to chemical toxicants. Scientific studies with representative individuals and large enough sample size can characterize this variability.

Interactive effects of exposure to more than one carcinogen or toxicant are also not necessarily quantified in the HRA. Cancer risks from all emitted carcinogens are typically added, and hazard quotients for substances impacting the same target organ/system are added to determine the hazard index (HI). Many examples of additivity and 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. Adjustment for tumors at multiple sites induced by some carcinogens could result in a higher potency. Other uncertainties arise 1) in the assumptions underlying the dose-response model used, and 2) in extrapolating from large experimental doses, where, for example, other toxic effects may compromise the assessment of carcinogenic potential, to usually much smaller environmental doses. Also, only single tumor sites induced by a substance are usually considered. When epidemiological data are used to generate a carcinogenic potency, less uncertainty is involved in the extrapolation from workplace exposures to environmental exposures. However, children, a subpopulation whose hematological, nervous, endocrine, and immune systems, for example, are still developing and who may be more sensitive to the effects of carcinogens on their developing systems, are not included in the worker population and risk estimates based on occupational epidemiological data are more uncertain for children than adults. Finally, the quantification of each uncertainty applied in the estimate of cancer potency is itself uncertain.

Thus, 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 risk, based on current knowledge and a number of assumptions. Additionally, the uncertainty factors integrated within the estimates of noncancer RELs are meant to err on the side of public health protection in order to avoid underestimation of risk. Risk assessment is best used as a ruler to compare one source with another and to prioritize concerns. Consistent approaches to risk assessment are necessary to fulfill this function.

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). 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 identifying if a hazard exists, and if so, what are the exact pollutant(s) of concern and 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 hazardous 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, 1997). 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 each substance for which potential cancer risk or acute and chronic noncancer effects 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 and long-term exposure levels. These activities are described in Chapters 4 and 5. Chapter 5 also discusses the tiered approach to risk assessment.

The ARB's 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. Currently, the most commonly used air modeling software is the ISCST3 (Industrial Source Complex Dispersion Model). This air modeling software is incorporated into HARP, 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 does not occur to any appreciable extent via ingestion or dermal exposure. Significant exposure to these volatile organic toxicants emitted into the air only occurs through the inhalation pathway. A small subset of Hot Spots substances, semi-volatile organic and metal toxicants, is 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. Table 5.1 in Chapter 5, Table 6.3 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 at a dose of one milligram per kilogram of body weight-day and commonly expressed in units of inverse dose (i.e., $(mg/kg/day)^{-1}$). 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. For clarity, consistency, and to assure proper use in risk assessment, cancer potencies should not be modified. Cancer potency factors listed in Table 7.1 were derived either by the United States Environmental Protection Agency (U.S. EPA) or by OEHHA and 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 Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II; Technical Support Document for Describing Available Cancer Potency Factors (OEHHA, 1999b and 2002).*

For noncarcinogenic effects, dose-response data developed from animal or human studies are used to develop acute and chronic noncancer Reference Exposure Levels (RELs). The acute and chronic RELs are defined as the concentration at which no adverse noncancer adverse health effects are anticipated. The most sensitive health effect is chosen to determine the REL if the chemical affects multiple organ systems. Unlike cancer health effects, noncancer acute and chronic health effects are generally assumed to have thresholds for adverse effects. In other words, acute or chronic injury from a pollutant will not occur until exposure to that pollutant has reached or exceeded a certain concentration (i.e., threshold). The acute and chronic RELs are 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 and acute REL values are below the threshold for human health for nearly all individuals. This guidance manual provides the acute and chronic Reference Exposure Levels in Tables 6.1 and 6.2, respectively. Some substances that pose a chronic 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 6.3. The methodology and derivations for acute and chronic RELs are described in the *Air Toxics Hot Spots Program Risk Assessment Guidelines; Part I; The Determination of Acute Reference Exposure Levels for Airborne Toxicants (Part I TSD) (OEHHA 1999a)* and *Air Toxics Hot Spots Program Risk Assessment Guidelines; Part III; Technical Support Document for the Determination of Chronic Reference Exposure Levels (Part III TSD)(OEHHA 2000a).*

2.5 Risk Characterization

This is the final step of risk assessment. In this step, modeled concentrations and public 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 acute 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). 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. Any emitted toxicant, that is not included in the quantitative analysis due to lack of a potency value or REL, should be qualitatively identified.

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 *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part IV; Technical Support Document for Exposure Assessment and Stochastic Analysis (OEHHA, 2000b)* (Part IV TSD).

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2.5.1 Point-Estimate Approach

The traditional approach used in the previous *California Air Pollution Control Officer's Association (CAPCOA) Air Toxics Hot Spots Program; Revised 1992; Risk Assessment Guidelines, October 1993* (CAPCOA, 1993) (CAPCOA Guidelines) for exposure and risk assessment has been to assign a single high-end point-estimate for each exposure pathway (e.g., breathing rate). A high-end value was generally chosen so that the potential cancer risk will not be underestimated. However, in the past, the high-end point-estimate has not been well defined as to where it fell on a data distribution. An improvement over the single point-estimate approach is to select two values, one representing an average and another representing a defined high-end value. OEHHA provides information in this document on average and high-end values for key exposure pathways (e.g., breathing rate). 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 (2000b). 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. This method uses high-end exposure estimates for driving exposure pathways and the average point-estimate for non-driving exposure pathways. The HARP software can perform this analysis.

In addition to using an estimate of average and high-end consumption rates, cancer risk evaluations for 9, 30, and 70-year exposure durations can be presented instead of just a single 70-year exposure duration. While 9 and 30-year exposure durations are available to present potential impacts over a range of residency periods, all HRAs must present the results based on 70-year exposure. The 9-and 30-year durations correspond to the central tendency and high-end estimates for residency time recommended by (U.S. EPA, 1997b). The parameters used for the 9-year exposure scenario are for the first 9-years of life and are thus protective of children. Children have higher intake rates on a per kilogram body weight basis 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 Senate Bill (SB) 1731 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 Part IV TSD 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 parameters (e.g., fish bioconcentration factors), there are only a few measurements 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 Part IV TSD.

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 Part IV TSD and is more appropriate to use than the recommended point-estimates in this document, then Tier-2 allows use of that site-specific information. In Tier-3, a stochastic approach to exposure assessment is used with the data distributions developed in Part IV TSD and presented in this document. Tier-4 is also a stochastic approach but allows for utilization of site-specific distributions, if they are justifiable 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. Chapter 8 provides a summary of the tiered approach and the Part IV TSD discusses it in detail. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

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, 1997*)*. 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.

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).

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 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.

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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.

4. Air Dispersion Modeling

The information contained in this section is primarily an abbreviated version of the material found in Chapter II of the *Air Toxics Hot Spots Risk Assessment Guidelines*; *Part IV; Exposure Assessment and Stochastic Analysis Technical Support Document (OEHHA, 2000b)* (Part IV TSD). Several references have been included in this section to indicate those areas that are covered in more detail in the Part IV TSD. However, some air dispersion concepts and procedures have been added or updated 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 a contractor through the consultation of OEHHA, Air Resources Board (ARB), 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.

Additionally, there are many direct references to the United States Environmental Protection Agency (U.S. EPA) ISCST3 air dispersion model. Recently the U.S. EPA has been promoting a new air dispersion model to effectively replace the ISCST3 model. Currently this new model, AERMOD, is available for testing and review. Once the U.S. EPA adopts the AERMOD air dispersion model into their list of regulatory approved models, the references and recommendations to specific models in this document are likely to change.

4.1 Air Dispersion Modeling in Exposure Assessment: Overview

The concentration of pollutants in ambient air is needed to characterize both inhalation and noninhalation exposure pathways. Pollutant concentrations are required in HRA calculations to estimate the potential cancer risk or hazard indices associated with the emissions of any given facility. Although monitoring of a pollutant provides excellent characterization of its concentrations, it is time consuming, costly, and typically limited to a few receptor locations and snapshots in time. Air dispersion modeling has the advantage of being relatively inexpensive and is less time consuming, provided that all the model inputs are available. In addition, air dispersion modeling provides greater flexibility for placement of receptors, assessment of individual and cumulative source contributions, and characterization of concentration over greater spatial extents.

Air dispersion modeling requires the execution of the following steps (see Fig 1):

1. Complete an emission inventory of the toxic releases (Section 4.2);
2. Classify the emissions according to source type and source quantity (Section 4.3);
3. Classify the analysis according to terrain (Section 4.4);

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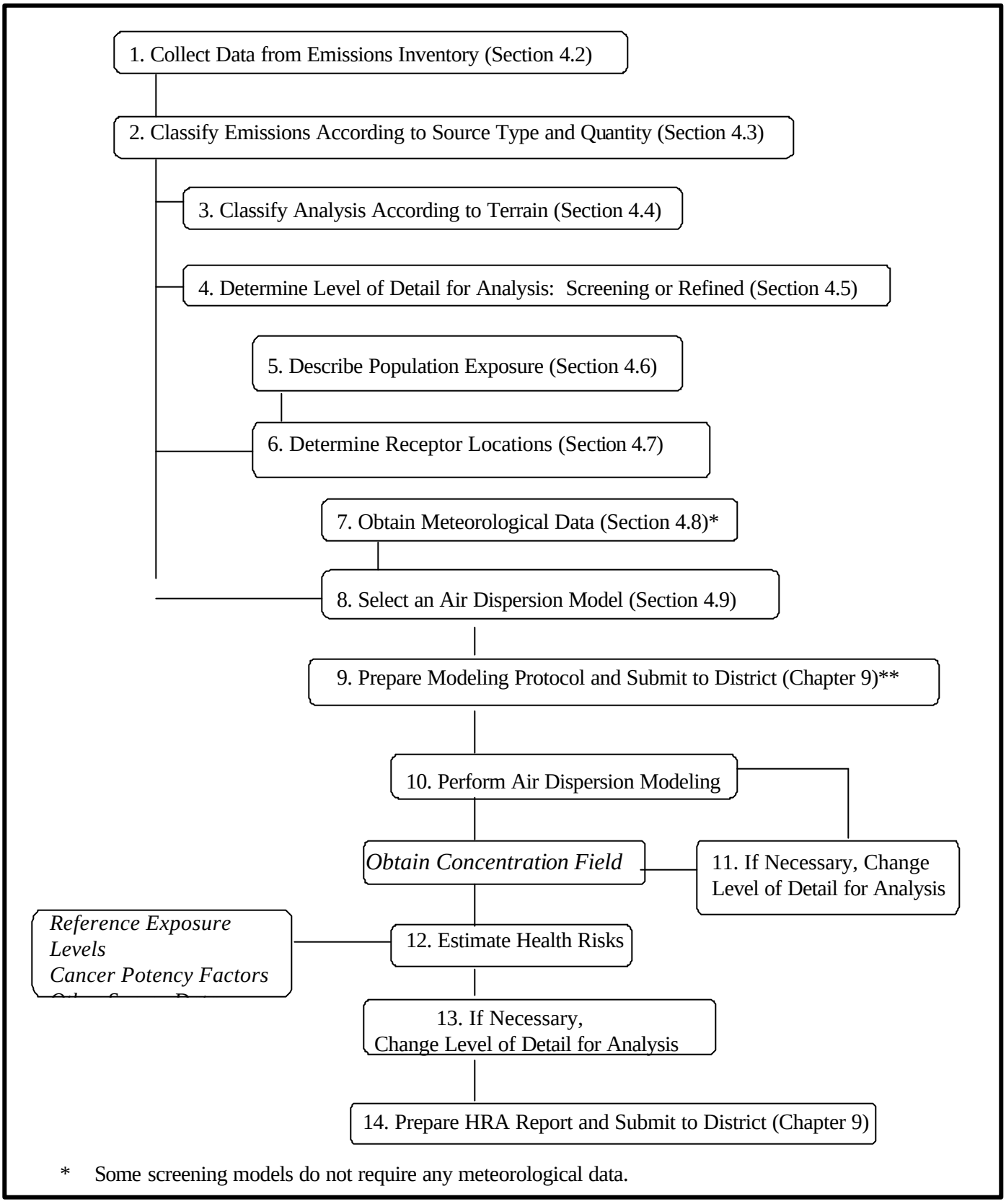
Figure 1. Overview of the Air Dispersion Modeling Process.



* Some screening models do not require any meteorological data.

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** Optional but strongly recommended.

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4. Determine level of detail for the analysis: refined or screening analysis (Section 4.5);
5. Identify the population exposure (Section 4.6);
6. Determine the receptor locations where impacts need to be analyzed (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 modeling protocol and submit to the local Air District (Chapter 9);
10. Perform an air dispersion analysis;
11. If necessary, redefine the receptor network and return to Step 10;
12. Perform HRA;
13. If necessary, change from screening to refined model and return to Step 8; and
14. Present the HRA results (Chapter 9 provides an outline that specifies the content and recommended format of HRA results).

The output of an air dispersion modeling analysis will be a receptor field of concentrations of the pollutant in ambient air. These concentrations in air need to be coupled with Reference Exposure Levels and cancer potency factors to estimate the hazard indices and potential carcinogenic risks. It should be noted that in the Hot Spots program emissions are considered inert for the purpose of transport and dispersion towards downwind receptors. Atmospheric transformations are not currently estimated.

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, 1997*)*.

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. Substances on the "list to be quantified" must be listed with emission quantities in a table in the HRA. For substances in the second and third groups, emissions do not need to be quantified; these substances should be listed in a separate table in the HRA. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

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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. 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.

Facilities that must also comply with RCRA/CERCLA requirements for HRAs need to consult the Cal/EPA Department of Toxic Substances Control (DTSC) Remedial Project Manager to determine what constitutes appropriate emissions data for use in the HRA. Source testing may be required for such facilities even if it is not required under the Hot Spots Program. Additional requirements for statistical treatment of source test results may also be imposed by DTSC on RCRA/CERCLA facilities.

A. Molecular Weight Adjustments for the Emissions of Metal Compounds

For most of the Hot Spots toxic metals, the OEHHA cancer potency factors 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 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,

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

The appropriate molecular weight adjustment factors (MWAF) to be used along with the OEHHA cancer potency factors for Hot Spots metals can be found in the MWAF column[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	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, however, that 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.**

4.2.1.2 Release Parameters

In order to use air dispersion models, release parameters (e.g., stack height and inside diameter, stack gas exit velocity, release temperature, and emission source location in actual UTM coordinates) need to be reported. The EICG Report specifies that the release parameters must be reported for each

[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 or the EICG report for reporting guidance.

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 air dispersion modeling guidelines or specific user's manuals. This information should also be included in the air dispersion portion of the HRA. This information must be presented in tables included in the HRA. Note that some dimensional units needed for the dispersion model may require conversion from the units reported in the Inventory Report (e.g., degrees K vs. degrees 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. Special weekly or seasonal emission patterns may vary and should be discussed. This is especially important in a refined HRA. Diurnal emission patterns should match the diurnal dispersion characteristics of the ambient air. Hourly emission scalars are needed to best represent emissions from facilities, especially for diurnal pattern. Air dispersion models, such as ISCST3, readily accept hourly emissions scalars and these scalars are fully functional in the HARP software with ISCST3. In addition, for the purposes of exposure adjustment for an off-site work receptor the emission schedule and exposure schedule should corroborate any exposure adjustment factors. (For example, no exposure adjustment factor should be made when an off-site receptor and the emissions are on a coincident schedule.) Some fugitive emission patterns may be continuous. Additionally, these data are used for adjustments in a screening air dispersion analysis (see Appendix H for further details). A table should be included with the emission schedule on an hourly, weekly and yearly basis. 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, the reported overall control efficiency must be adjusted to account for downtime of control equipment. Any entrainment of toxic substances to the atmosphere from control equipment should be accounted for; this includes fugitive releases during maintenance and cleaning of control devices (e.g., baghouses and cyclones). 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 8.2.4. 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

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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

The types of sources and quantity of sources at a facility need to be characterized in order to select an appropriate air dispersion model.

4.3.1 Classification According to Source Type

Air dispersion models can be classified according to the type of source that they are designed to simulate, including, but not limited to, point, line, area, and volume sources. Several models have the capability to simulate more than one type of source.

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 isolated vents from buildings and exhaust stacks from facility processes.

4.3.1.2 Line Sources

In practical terms, line sources are a special case of either an area or a volume source. Consequently, they are normally modeled using either an area or volume source model as described below. Examples of line sources include conveyor belts and rail lines. A roadway is a unique line source. Models designed to simulate the enhanced mixing due to motor vehicle movements have been developed (i.e., CALINE4 and CAL3QHCR).

4.3.1.3 Area Sources

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

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

Other fugitive emissions emanating from many points of release at the same elevation 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.

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 Classification According to Quantity of Sources

The selection of an air dispersion model also requires the consideration of the number of distinct sources. Some dispersion models are capable of simulating only one source at a time, and therefore are referred to as single-source models (e.g., SCREEN3).

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:

1. 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 the shortest distance from the receptor to the nearest stack).

2. Run the model separately for each individual source and superimposing the results.

   Superposition of results from each source is the approach used 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).

4.4 Terrain Characterization

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 Land Type Classification

Most air dispersion models use different dispersion coefficients (sigmas) depending on the land use over which the pollutants are being transported. The type of land use 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. AERMOD does not depend on the dispersion coefficients used by models such as ISCST3. Therefore AERMOD does not need to classify the land type into urban or rural. When AERMOD becomes adopted as a Guideline model and is more widely used, these recommendations on land use classifications will need to be modified. Until that time, the following recommendations are relevant.

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 (e.g., on the coast) the District should be consulted concerning the classification. 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.

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 4.6.2 and 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).

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 ash pits, 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 Topography Classification

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 various models differ in their needs to adjust for these variables.

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. The ISCST3 model uses a screening approach to complex terrain. AERMOD also provides algorithms for complex terrain.

Topography can be classified according to the following sections.

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 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.

4.5 Level of Detail: Screening vs. Refined Analysis

Air dispersion models can be classified as "screening" or "refined" according to the level of detail that is used in the assessment of the concentration estimates. Refined air dispersion models use more robust algorithms that are 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 concentrations 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 HARP software addresses these situations by incorporating the capability of using either representative meteorological data or the default meteorological conditions from the SCREEN3 model as inputs to the ISCST3 air dispersion model.

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It is acceptable to use a refined air dispersion model in a "screening" mode for this program's HRAs. In this case, worst-case hourly meteorological data are used to estimate the maximum 1-hour concentration with the ISCST3 model. Conservative conversion factors are used to estimate longer term averaging periods based on the maximum 1-hour concentration. (See Table 4.3 and Appendix H for guidance on the use of the conversion factors.)

4.6 Population Exposure

Population exposure can be assessed by determining the number of people at a particular cancer risk level such as 1×10^{-5} or 1×10^{-6}. For noncancer risk it can be the number of people exposed to the Hazard Index over a certain level such as one or five. The traditional way of estimating population exposure for cancer has been the cancer burden or the number of excess cancer cases in the exposed population.

The 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 or reviewing authority should be consulted before beginning the population exposure estimates. 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, the HARP software can provide population exposure estimates as cancer burden or as the number of persons exposed to a selected potential (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

The first step of population exposure estimate in an HRA is to define the zone of impact. The zone of impact is the area around the facility that is affected by the facility's emissions. This zone is commonly defined as the area surrounding the facility where receptors have a potential multipathway (inhalation and noninhalation exposure) cancer risk greater than 10^{-6} (one in a million), an acute (inhalation) hazard index (HI) of 1.0, and/or a chronic multipathway HI of 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, the District should be consulted. The District may specify limits on the area of the zone of impact. Ideally, these preferences would be discussed with the District before being presented in the modeling protocol and HRA.

Note that when depicting the HRA results, potential cancer and noncancer isopleths must present the total cancer and noncancer health impacts from both inhalation and noninhalation pathways, when appropriate. 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 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 Appendices I.

4.6.2 Screening Population Estimates for Risk Assessments

Not all HRAs require refined population exposure assessments and at times a screening estimate may be appropriate. A screening population estimate should include an estimate of the maximum exposed population. 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 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. The District should be consulted to determine the population estimate to be used for screening purposes. Guidance for one screening method is presented here.

1. Use a screening dispersion model (e.g., SCREEN3) 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.

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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 more detailed analysis of the population distribution that is exposed to emissions from the facility. These populations can include exposure estimates for workers and residents through the use of land use maps. The District may require that locations with high densities of sensitive individuals be identified (e.g., schools, daycare centers, hospitals). The overall exposed residential and worker populations should be apportioned into smaller geographic subareas. The information needed for each subarea is:

1. the number of exposed persons, and
2. the receptor location 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).

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4.6.3.1 Census Tracts

For a refined HRA, the boundaries of census tracts can be used to define the geographic area to be included in the population exposure analysis. Maps showing census tract boundaries and numbers can be obtained from "The Thomas Guide® - Census Tract Edition". Statistics for each census tract can be obtained from the U.S. Census Bureau. 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 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 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®). If only a portion of the census tract lies within the zone of impact, the population used in the burden calculation should include the proportion of the population in that isopleth zone. 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. Each receptor located on the Cartesian grid must be identified with actual UTM coordinates. This process may be computerized and minimizes manual mapping of centroids and census tracts. The HARP software can provide population exposure estimates as cancer burden or as the number of persons exposed at the block level to a selected potential (user identified) health risk/impact level.

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.

Subcensus Tract

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

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

It is necessary to limit the size of the detailed analysis area because inclusion of all subcensus tracts would greatly increase the resource requirements of the analysis. For example, an urban area of 100,000 persons would involve approximately 25 census tracts, approximately 100 to 150 block groups, and approximately 1,000 to 1,400 blocks. Furthermore, a high degree of resolution at large distances from a source would not significantly affect the analysis because the concentration gradient at these distances is generally small. Thus, the detailed analysis of census tracts within several kilometers of a facility should be sufficient. The District should be consulted to determine the area that requires detailed analysis.

The District should also be consulted to determine the degree of resolution required. In some cases, resolution of residential populations to the BG/ED level may be sufficient. However, resolution to the block level may also be required for those BG/EDs closest to the facility or those having maximum concentration impacts. The identified employment subareas should be resolved to a similar degree of resolution as the residential population. For each subarea analyzed, the number of residents and/or workers exposed should be estimated.

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 HRAs. 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,

hospitals). The HRA should state what the District requirements are regarding identification of sensitive receptor locations.

Although sensitive individuals are protected by general assumptions made in the dose response assessment, their identification may be useful to assure the public that such individuals are being considered in the analysis. For cancer and noncancer effects, the identification of sensitive receptor locations may be crucial in evaluating the potential impact of the toxic effect.

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, however, 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 District for guidance if on-site exposure situations are present at the emitting facility. These on-site locations should be included in the HRA. All of these locations (i.e., PMI, MEIR, and MEIW) must be identified for potential multipathway carcinogenic and noncarcinogenic effects. 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. It is possible that the estimated PMI, MEIR, and MEIW risk for carcinogenic, chronic noncarcinogenic, and acute noncarcinogenic health effects occur at different locations. Methods used to determine dose are provided in Chapter 5 and methods for calculating potential health impacts are included in Chapter 8 and Appendix I .

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 actual UTM (Universal Transverse Mercator) coordinates and receptor number. The receptor numbers in the summary tables should match receptor numbers in the computer output. In addition to actual UTM coordinates, the block/street locations (i.e., north side of 3,000 block of Smith

Street) should be provided 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.

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 to represent the breathing zone, or direct inhalation, of a person may need to be considered when the source to receptor distance is less than a few hundred meters. Consideration must also be given to the multipathway analysis, which requires the deposition at ground level. 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 lies with the District.

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. Where population is not uniformly distributed, a population-weighted centroid is needed. Another alternative could be to use the concentration at the point of maximum impact (PMI) within that census tract as the concentration to which the entire population of that census tract is exposed.

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.

4.8 Meteorological Data

Refined air dispersion models require hourly meteorological data. The first step in obtaining meteorological data should be to check with the District for data availability. Other sources of data include the National Weather Service (NWS); National Climatic Data Center (NCDC) in Asheville, North Carolina; military stations; and private networks. Meteorological data for a subset of NWS stations are available from the U.S. EPA Support Center for Regulatory Air Models (SCRAM). The SCRAM can be accessed at *www.epa.gov/scram001/main.htm*. All meteorological data sources should be approved by the District. Data not obtained directly from the District should be checked for quality, representativeness, and completeness. U.S. EPA provides guidance (U.S. EPA, 1995e) for these data. The HRA 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. The argument that "this is the nearest available meteorological data" does not justify that the data are

representative. 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 period of record, recommended for use in the air dispersion model, is five years. If it is desired to use a single year to represent long-term averages (i.e., chronic exposure), then the worst-case year should be used. The worst-case year should be the year that yields the greatest maximum chronic off-site risk. If the only adverse health effects associated with all emitted pollutants from a given facility are acute, the worst-case year should be the year that yields the greatest maximum acute off-site risk. With the increasing speeds of today's desktop computers, processing five years of data should be relatively fast. Therefore, we strongly encourage the use of five years of meteorological data when available. However, the District may determine that one year of representative meteorological data is sufficient to adequately characterize the facility's impact.

Otherwise, to determine annual average concentrations for analysis of chronic health effects, the data can be averaged, if a minimum of three years of meteorological data is available. 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 annual 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 7.0 $\mu g/m^3$. 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.

More information on sources of meteorological data, as well as representativeness and completeness of meteorological data, can be found in Chapter 2 of the Part IV TSD.

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 is in the process of adding new models to the preferred list of models: ISC-PRIME, AERMOD, AERMOD-PRIME, and CalPuff. The latest version of the U.S. EPA recommended models can be found at the SCRAM Bulletin Board located at *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. New models placed on U.S. EPA's preferred list of models (i.e., ISC-PRIME, AERMOD, AERMOD-PRIME, and CalPuff) can be considered at that time. All air dispersion models used to estimate pollutant concentrations for HRA 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 generally required for analysis of acute effects. There are a few pollutants that require averaging times up to 7 hours; these can be found in Table 6.1.

4.9.1 Recommended Models

Recommended air dispersion models to estimate concentrations for HRA analyses are shown in Table 4.2. Currently, SCREEN3 and ISCST3 are the two preferred models for HRAs. This could change when the U.S. EPA places ISC-PRIME, AERMOD, AERMOD-PRIME, and CalPuff on the preferred list. Some of the names of the air dispersion models reflect the version number at the time of the writing of this document. The most current version of the models should be used for the HRA analysis. More than one model may be necessary in some situations, for example, when modeling scenarios have receptors in simple and complex terrain. Some facilities may also require models capable of handling special circumstances such as building downwash, dispersion near coastal areas, etc. See Chapter 2 of the Part IV TSD for more information on modeling special cases and for specific information including inputs and default option settings for most of the models presented in Table 4.2.

To further facilitate the model selection, the District should be consulted for additional recommendations on the appropriate model(s) or a protocol can be submitted for District review and approval (see Chapter 9). A brief description of the preferred screening model, SCREEN 3, and the preferred refined model, ISCST3, are discussed below.

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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 shown in Table 4.2. For more information on the applicability of alternative models refer to the following documents:

- U.S. EPA (1986) *Guideline on Air Quality Models (Revised)*
- U.S. EPA (1992a) *Protocol for Determining the Best Performing Model*
- U.S. EPA (1985a) *Interim Procedures for Evaluating Air Quality Models – Experience with Implementation*
- U.S. EPA (1984) *Interim Procedures for Evaluating Air Quality Models (Revised)*

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TABLE 4.2 Recommended Air Dispersion Models

	AVERAGING PERIOD	TERRAIN TYPE	SINGLE SOURCE		MULTIPLE SOURCE	
			RURAL	URBAN	RURAL	URBAN
REFINED MODELS	SHORT TERM (1-24 hour avg)	SIMPLE	ISCST3	RAM ISCST3	ISCST3	RAM ISCST3
		COMPLEX	CTDMPLUS	CTDMPLUS	CTDMPLUS	CTDMPLUS
	LONG TERM (Monthly-Annual)	SIMPLE	ISCST3 ISCLT3	RAM ISCST3 ISCLT3	ISCST3 ISCLT3	CDM20 / RAM ISCST3 ISCLT3
		COMPLEX	CTDMPLUS	CTDMPLUS	CTDMPLUS	CTDMPLUS
SCREENING MODELS	SHORT TERM (1-24 hour avg)	SIMPLE	SCREEN3	SCREEN3	SCREEN3	SCREEN3
		COMPLEX	ISCST3 RTDM, CTSCREEN VALLEY SCRN	SHORTZ CTSCREEN VALLEY SCRN	ISCST3 CTSCREEN* VALLEY SCRN	SHORTZ CTSCREEN* VALLEY SCRN
	LONG TERM (Monthly-Annual)	SIMPLE	SCREEN3	SCREEN3	SCREEN3	SCREEN3
		COMPLEX	ISCST3 RTDM	LONGZ	ISCST3	LONGZ
	Generally speaking, ISCST3 and SCREEN3 are the models that are used in most cases in the Hot Spots Program. Other models in this list may be considered on a case-by-case basis. Additionally, newer models (e.g., ISC-PRIME, AERMOD, AERMOD-PRIME, and/or CalPuff) may be added to this list at a future date.					

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4.10 Screening Air Dispersion Models

A screening model may be used to estimate 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. Maximum 1-hour concentration averages can be converted to other averaging periods through consultation and approval by the District. Appendix H describes the use of the conversion factors. Because of variations in local meteorology and source types, the exact factor selected may vary from one district to another. Table 4.3 provides guidance on the range and typical values applied. The conversion factors are designed to bias predicted longer-term averaging periods towards overestimation.

Table 4.3. Recommended Factors to Convert Maximum 1-hour Avg. Concentrations to Other Averaging Periods (U.S. EPA, 1995a; ARB, 1994).

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

4.10.1 SCREEN3

The SCREEN3 model is among the most widely used model primarily because it has been periodically updated to reflect changes in air dispersion modeling practices and theories. The SCREEN3 model represents a good balance between ease of use and the capabilities and flexibility of the algorithms. In addition, the calculations performed by the model are very well documented (U.S. EPA, 1995a). The SCREEN3 User's Guide (U.S. EPA, 1995d) also presents technical information and provides references to other support documents. The dispersion algorithms used in SCREEN3 are consistent with ISCST3. (With the implementation of AERMOD, which is expected in the future, SCREEN3 may need to be superseded with a model that is compatible with AERMOD.)

The most important difference between the SCREEN3 model and refined models such as ISCST3 is the meteorological data used to estimate pollutant concentrations. The SCREEN3 model can assume worst-case meteorology, which greatly simplifies the resources and time normally

associated with obtaining meteorological data. Consequently, more conservative (higher concentration) estimates are normally obtained. Alternatively, a single stability class and wind speed may also be entered.

Number of Sources and Type

SCREEN3 was designed to simulate only a single source at a time. However, more than one source may be modeled by consolidating the emissions into one emission point or by individually running each point source and adding the results. SCREEN3 can be used to model point sources, flare releases, and simple area and volume sources. Input parameters required for various source-types are shown in Tables 4.4 (point), 4.5 (flare release), 4.6 (area), and 4.7 (volume).

Table 4.4. Required Input Parameters to Model a Point Source Using SCREEN3.

Emission Rate (g/s)	
Stack Height (m)	
Stack Inside Diameter (m)	
Stack Gas Exit Velocity (m/s) or Volumetric Flow Rate (ACFM, m^3/s)	
Stack Gas Temperature (K)	
Ambient Temperature (K)	
Receptor Height Above Ground (m)	
Receptor Distance from the Source (m)	[discrete distance or automated array]
Land Type	[urban or rural]
Meteorology	[option "1" (full meteorology) is normally selected]
In Addition, for building downwash calculations	
Building Height (m)	
Minimum Horizontal Dimension (m)	
Maximum Horizontal Dimension (m)	

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Table 4.5. Required Input Parameters to Model a Flare Using SCREEN3.

Emission Rate (g/s)	
Flare Stack Height (m)	
Total Heat Release (cal/s)	
Receptor Height Above Ground (m)	
Receptor Distance from the Source (m)	
Land Type	[urban or rural]
Meteorology	[option "1" (full meteorology) is normally selected]
In Addition, for building downwash calculations	
Building Height (m)	
Minimum Horizontal Dimension (m)	
Maximum Horizontal Dimension (m)	

Table 4.6. Required Input Parameters to Model an Area Source Using SCREEN3.

Emission Rate (g/s-m^2)	
Source Release Height (m)	
Length of Larger Side of the Rectangular Area (m)	
Length of Smaller Side of the Rectangular Area (m)	
Receptor Height Above Ground (m)	
Receptor Distance from the Source (m)	
Land Type	[urban or rural]
Meteorology	[option "1" (full meteorology) is normally selected] [wind direction optional]

Table 4.7. Required Input Parameters to Model a Volume Source Using SCREEN3.

Emission Rate (g/s)	
Source Release Height (m)	
Initial Lateral Dimension of Volume (m)	
Initial Vertical Dimension of Volume (m)	
Receptor Height Above Ground (m)	
Receptor Distance from the Source (m)	
Land Type	[urban or rural]
Meteorology	[option "1" (full meteorology) is normally selected]

Regulatory Options

SCREEN3 algorithms contain all regulatory options internally coded including stack-tip downwash and buoyancy-induced dispersion. These regulatory options are the default settings of the parameters so the user does not need to set any switches during a run.

Special Cases

SCREEN3 has the capability to model several special cases by setting switches in the input file or by responding to on-screen questions (if run interactively). The special cases include:

- simple elevated terrain
- plume impaction in complex terrain using VALLEY model 24-hr screening procedure
- building downwash (only for flat and simple elevated terrain)
- cavity region concentrations (The PRIME algorithms included with ISCST3-PRIME should be used for estimates in the cavity zone)
- inversion break-up fumigation (only for rural inland sites with stack heights greater than or equal to 10 m and flat terrain)
- shoreline fumigation (for sources within 3,000 m from a large body of water)
- plume rise for flare releases

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.

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4.11.1 ISCST3

The ISCST3 model (U.S. EPA, 1995b; 1995c) is a steady-state Gaussian plume model, which can be used to assess pollutant concentrations from a wide variety of sources associated with an industrial source complex. The ISCST3 model can be used for multiple sources in urban or rural terrain. The model includes the algorithms of the complex terrain model COMPLEX I. The user can specify if calculations are to be made for simple terrain, complex terrain, or both. However since COMPLEX 1 is a screening model, the ISCST3 model is only a screening tool for receptors in complex terrain. The ISCST3 model can calculate concentration averages for 1-hour or for the entire meteorological data period (e.g., annual or intermediate time periods such as 24-hour averages). A summary of basic input parameters needed to model a point source is shown in Table 4.8. Guidance on additional input requirements (e.g., for area and volume sources) may be found in the ISC Users Guide. (ISCST3 may be replaced with AERMOD in the future pending promulgation by the U.S. EPA.)

Table 4.8. Basic Input Parameters Required to Model a Point Source Using ISCST3.

Land Use	Urban or Rural
Averaging Period	
Emission Rate (g/s)	
Stack Height (m)	
Stack Gas Exit Temperature (K)	
Stack Gas Exit Velocity (m/s)	
Stack Diameter (m)	
Receptor Locations (x,y) coordinates (m)	discrete points; polar array; Cartesian array;
Meteorology	may be supplied by preprocessor, e.g., PCRAMMET
Anemometer Height (m)	

4.11.1.1 Regulatory Options

Regulatory application of the ISCST3 model 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 regulatory options, automatically selected when the DFAULT keyword is used, are:

- Stack-tip downwash (except for Schulman-Scire downwash)
- Buoyancy-induced dispersion (except for Schulman-Scire downwash)
- Final plume rise (except for building downwash)
- Treatment of calms
- Default values for wind profile exponents

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- Default values for vertical potential temperature gradients
- Use upper-bound concentration estimates for sources influenced by building downwash from super-squat buildings

4.11.1.2 Special Cases

a. Building Downwash

The ISC models automatically determine if the plume is affected by the wake region of buildings when their dimensions are given. Including building dimensions in the model input does not necessarily mean that there will be downwash. See Chapter 2 of the Part IV TSD for guidance on how to determine when downwash is likely to occur.

b. Area Sources

The area source algorithms in ISCST3 use an integration technique that allows placement of receptors within the area source. Additionally, initial dispersion in the vertical can be included to simulate sources with vertical extent.

c. Volume Sources

The volume source algorithms in ISCST3 require an estimate of the initial distribution of the emission source in the horizontal and the vertical. Tables that provide information on how to estimate the initial distribution for different sources are given in the ISC3 User's Guide (U.S. EPA, 1995b; 1995c).

d. Intermediate Terrain

When simple and complex terrain algorithms are selected by the user, ISCST3 will select the higher impact from the two algorithms on an hour-by-hour, source-by-source, and receptor-by-receptor basis for all receptors located in intermediate terrain (U.S. EPA, 1995b).

Alternatively, the pollution concentrations in the receptor field may be generated separately from HARP using other approved air dispersion models. HARP has the flexibility to generate a summary of the risk data necessary for an HRA by either approach: ISCST3 internal to HARP or the use of other approved models outside of HARP.

In addition, the HARP software also incorporates the capability of using either user supplied representative meteorological data or the worst-case meteorological conditions from the SCREEN3 model as inputs to the ISCST3 air dispersion model. Information on obtaining the HARP software can be found 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.

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e. Deposition

The ISC models contain algorithms to model settling and deposition and require additional information such as the particle size distribution. For more information consult the ISC3 User's Guide (U.S. EPA, 1995b). Note that, when performing the HRA modeling, a deposition rate will be requested and used for the noninhalation pathway exposure (see Section 8.2.5.A).

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 ISCST3 (Industrial Source Complex – Short Term 3). In addition, HARP directly links the ISCST3 outputs with risk assessment modules eliminating the need for intermediate processing by the user.

4.12 Modeling Special Cases; Specialized Models

Special situations arise in modeling some sources that require considerable professional judgment; these include building down-wash effects, wet and dry deposition, short term emissions (i.e., significantly less than 1-hour), fumigation effects, rain-cap on stack, and landfill sites. Details for these special modeling situations and specific models can be found in Chapter 2 of the Part IV TSD. It is recommended that the reader consider retaining professional consultation services if the procedures are unfamiliar. 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.

4.13 Interaction with the District

The risk assessor must contact the District to determine if there are any specific modeling requirements. Examples of such requirements may include specific receptor location guidance, specific usage of meteorological data, and specific report format (input and output). See Chapter 9 for information on the format and content of modeling protocols and HRAs.

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 concentrations and dose precede the calculations of potential health impacts. See Chapter 8 and Appendix I for information on calculating potential health impacts.

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

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

The PMI is defined as the receptor point(s) with the highest acute, chronic, or cancer health impacts outside the facility boundary. The facility boundary is defined as the property line. Often the fence is on the property line. The MEIR is defined as the existing off-site residence(s) (e.g., house or apartment) with the highest acute, chronic, or cancer health impacts. The MEIW is defined as the highest acute, chronic, or cancer health impacts at an existing off-site workplace. Note, however, that occasionally 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 (District) for guidance if on-site exposure situations exist 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, chronic, and cancer health impacts at the PMI may be located at different physical locations. The MEIR or MEIW cancer, acute, and chronic receptors may also be at different locations. In addition, it may be necessary to determine risks at sensitive receptors (e.g., schools, daycare, eldercare, and hospitals). The District or reviewing authority should be consulted in order to determine the appropriate sensitive receptors for evaluation.

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*

The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments. August 2003.

Guidelines; Part IV; Technical Support Document for Exposure Assessment and Stochastic Analysis (OEHHA, 2000b) (Part IV TSD).

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 an annual-average and maximum one, four, six, and seven-hour ground level concentrations. 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.

$$GLC = \left(\chi/Q\right)\left(Q_{\text{substance}}\right)$$

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

$$Q_{\text{substance}} = \text{substance specific emission rate} \left(g/s\right)$$

- The applicable exposure pathways (e.g., inhalation, soil, fish) 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.

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 were 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, 1997*)*.

The HARP software is the recommended model for calculating and presenting HRA results for the Hot Spots Program. A contractor, through consultation with OEHHA, Air Resources Board (ARB), and District representatives, developed the HARP software. 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.

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 on to the 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 used to determine if a substance should be evaluated for multipathway impacts:

- Step one is to see if the substance or its group (e.g., dioxins, PAHs) is listed in Table 5.1.

- Step two is to determine if the substance has an oral reference exposure level (REL) listed in Table 6.3, or if it has an oral cancer slope factor listed in Table 7.1. Oral or noninhalation exposure pathways include the ingestion of soil, fisher caught fish, drinking water from surface waters, 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, or PCBs are emitted, then the breast-milk consumption pathway also becomes mandatory. The other exposure pathways (e.g., the ingestion of homegrown produce or 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 were no vegetable gardens or fruit trees within the zone of impact for a facility, for example, then the produce pathways would 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 single 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 chemicals 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 surface waters are not impacted by the facility, or the water source is impacted but never used for drinking water, then the drinking water 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
4,4'-Methylene dianiline	X	X		X	X	X	X	X	X	
Creosotes	X	X	X	X	X	X			X	
Diethylhexylphthalate	X	X		X	X	X	X	X	X	
Hexachlorocyclohexanes	X	X		X	X	X			X	
PAHs	X	X	X	X	X	X			X	
PCBs	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	X	X	X	X	X	X	
Inorganic arsenic & compounds	X	X	X	X	X	X	X	X	X	
Beryllium & compounds	X	X	X	X	X	X	X	X	X	
Lead & compounds	X	X	X	X	X	X	X	X	X	
Mercury & compounds	X	X	X	X	X	X	X	X	X	
Nickel	X	X	X		X	X	X	X	X	
Fluorides (Including hydrogen fluoride)	**To be determined**									
Dioxins & furans	X	X	X	X	X	X			X	X

5.3 Estimation of Concentrations in Air, Soil, and Water

Once emissions exit the source, the substances 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 single semivolatile organic toxicant can partition as a vapor and into a particulate. Particulates will deposit at a rate that is dependent on the particle size. The substances will deposit on vegetation, on soil, and in water. Use the 0.02 m/s factor 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 concentration of the substance in air at ground level (GLC) is a function of the facility emission rate and the dilution factor (χ/Q) at the points under evaluation.

a. Formula 5.3.1 A: **GLC = E-rate * χ/Q (EQ 5.3.1 A)**

1> GLC = Ground-level concentration ($\mu g/m^3$)
2> E-rate = Substance emission rate (g/sec)
3> χ /Q = Dilution factor provided by dispersion modeling ($\mu g/m^3$/g/sec)

b. Recommended values for EQ 5.3.1 A:

1> E-rate = Facility specific, substance emission rate
2> χ/Q = For point of interest, site specific, from dispersion modeling

c. Assumptions for EQ 5.3.1 A:

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.

a. Formula 5.3.2 A: $C_s = Dep * X / (K_s * SD * BD * T_t)$ **(EQ 5.3.2 A)**

1> C_s = Average soil concentration over the evaluation period ($\mu g/kg$)
2> Dep = Deposition on the affected soil area per day ($\mu g/m^2/d$)

a> Formula 5.3.2 B: $$Dep = GLC * Dep\text{-}rate * 86{,}400 \quad \text{(EQ 5.3.2 B)}$$

1: GLC = 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)

b> Recommended default values for EQ 5.3.2 B:

1: GLC = Calculated above in EQ 5.3.1 A
2: Dep-rate = Use 0.02 meters/second for controlled or
0.05 meters/second for uncontrolled sources.

c> Assumptions for EQ 5.3.2 B:

1: Deposition rate remains constant

3> **X** = Integral function

a> Formula 5.3.2 C: $$X = [\{e^{-Ks * Tf} - e^{-Ks * To}\} / K_s] + T_t \quad \text{(EQ 5.3.2 C)}$$

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

a: Formula 5.3.2 D: $$K_s = 0.693 / t_{1/2} \quad \text{(EQ 5.3.2 D)}$$

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

b: Recommended default values for EQ 5.3.2 D:

1) $t_{1/2}$ = See Table 5.3

b> Recommended default values for EQ 5.3.2 C:

1: K_s = Calculated above in EQ 5.3.2 D
2: T_f = 25,550 (d) = 70 yr (for 9, 30 and 70 years). Identifies the total number of days of soil deposition.

= 9,490 (d) = 26 years for nursing mother in mother's milk pathway
3: T_o = 0 (d) The initial time (start period) of exposure to all receptors that are impacted by the soil pathway. Used for direct soil exposure to a worker, residential adults (9, 30, and 70-years), and children. Also used as the initial time for

determining the concentration in soil that is used for estimating the dose from the ingestion of breast milk.

4> SD = Soil mixing depth (m)
5> BD = Soil bulk density (kg/m^3)

b. Recommended default values for EQ 5.3.2 A:

1> Dep = Calculated above in EQ 5.3.2 B
2> X = Calculated above in EQ 5.3.2 C
3> K_s = Calculated above 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 (yr) for 9, 30 and 70 year exposure durations and mother's milk pathway
= 25,550 (d) for adult in mother's milk pathway

c. 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> For a receptor ingesting mother's milk, the mother is exposed for 26 years, the child receives milk for one year (the last year of maternal exposure), and then is exposed to all other pathways for 9, 30 or 70 years.
4> It is assumed that toxicants accumulate in the soil for 70 years from deposition.

5.3.3 In Water

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. Formula 5.3.3 A:

$$C_w = C_{depw} \quad \text{(EQ 5.3.3 A)}$$

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

a> Formula 5.3.3 B:

$$C_{depw} = Dep * SA * 365 / (WV * VC) \quad \text{(EQ 5.3.3 B)}$$

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

b> 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)
4: VC = Site specific number of volume changes per year
(SA, WV, and VC values can be acquired from the applicable Department of Water Resources (DWR) Regional office)

c> 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 bioconcentration in fish.

5.3.4 Estimation of Concentrations in Vegetation and Animal Products

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

1. **Vegetation**

The average concentration of a substance in and on vegetation (C_f) is a function of direct deposition of the substance onto the vegetation and of root translocation or uptake from soil contaminated by the substance.

a. Formula 5.3.4.1 A:

$$C_v = C_{depv} * GRAF + C_{trans} \qquad \text{(EQ 5.3.4.1 A)}$$

1> C_v = Average concentration in and on specific types of vegetation (μg/kg)
2> C_{depv} = Concentration due to direct deposition (μg/kg)
3> GRAF = Gastrointestinal Relative Absorption Fraction

a> Formula 5.3.4.1 B:

$$C_{depv} = [Dep * IF / (k * Y)] * (1 - e^{-kT})] \qquad \text{(EQ 5.3.4.1 B)}$$

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)

b> 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
  b: Leafy crops = 0.2
  c: Protected crops = 0
  d: Exposed crops = 0.1
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

c> Assumptions for EQ 5.3.4.1 B:

1: No deposition on root or protected crops

3> GRAF = Gastrointestinal Relative Absorption Fraction
0.43 for dioxins; 1.0 for all other chemicals

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 5.3).

4> C_{trans} = Concentration due to root translocation or uptake (μg/kg)

a> Formula 5.3.4.1 C: $$C_{trans} = C_s * UF_2 \qquad \text{(EQ 5.3.4.1 C)}$$

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

b> Recommended default values for EQ 5.3.4.1 C:

1: C_s = Calculated above in EQ 5.3.2 A
2: UF_2 = Inorganic compounds--see Table 5.3

1) Formula 5.3.4.1 D: (for organic compounds)

$$UF_2 = [(0.03 * K_{ow}^{0.77}) + 0.82] / [(K_{oc})(F_{oc})] \quad \text{(EQ 5.3.4.1 D)}$$

a) 0.03 = Empirical constant
b) K_{ow} = Octanol: water partition factor
c) 0.77 = Empirical constant
d) 0.82 = Empirical constant
e) K_{oc} = Organic carbon partition coefficient
f) F_{oc} = Fraction organic carbon in soil

2) Recommended default values for EQ 5.3.4.1 D:

a) K_{ow} = Chemical specific, see Table 5.3
b) K_{oc} = Chemical specific, see Table 5.3
c) F_{oc} = 0.1

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. Formula EQ 5.3.4.2 E:

$$C_{fa} = (\text{Inhalation} + \text{Water ingestion} + \text{Feed ingestion} + \text{Pasture/Grazing ingestion} + \text{Soil ingestion}) * Tco \quad \text{(EQ 5.3.4.2 E)}$$

1> C_{fa} = Average concentration in farm animals and their products (µg/kg)
2> **Inhalation** = Dose through inhalation (µg/d)

a> Formula 5.3.4.2 F:

$$\text{Inhalation} = BR_A * GLC \quad \text{(EQ 5.3.4.2 F)}$$

1: BR_A = Inhalation rate for animal (m^3/d)
2: GLC = Ground-level concentration (µg/m^3)

b> Recommended default values for EQ 5.3.4.2 F:

1: BR_A = See Table 5.2
2: GLC = Calculated above in EQ 5.3.1 A

c> Assumptions for EQ 5.3.4.2 F:

1: All material inhaled is 100% absorbed

3> **Water ingestion** = Dose through water ingestion (μg/d)

a> Formula EQ 5.3.4.2 G:

$$\textbf{Water ingestion} = WIR_a * FSW * C_w \quad \textbf{(EQ 5.3.4.2 G)}$$

1: WIR_a = Water ingestion for animal (kg/d)
2: FSW = Fraction of water ingested from a contaminated body of water
3: C_w = Average concentration in water (μg/kg) For water 1 kg = 1 L

b>Recommended default values for EQ 5.3.4.2 G:

1: WIR_a = See Table 5.2
2: FSW = Site specific, need to survey, fraction of water ingestion practices in affected area

3: C_w = Calculated above in EQ 5.3.3 A

Table 5.2 Point Estimates for Animal Pathway*

Parameter	Beef Cattle	Lactating Dairy Cattle	Pigs	Poultry
BW (body weight) (kg)	500	500	60	2
BR_A (inhalation rate) (m^3/d)	100	100	7	0.4
WIR_a (water ingestion) (kg/d)**	40	80	8	0.2
FIR (feed ingestion) (kg/d)	8	16	2	0.1
FS_f (soil fraction of feed)	0.01	0.01	NA	NA
FSp (soil fraction of pasture)	0.05	0.05	0.04	0.02

Beef and dairy cattle food from pasture grazing is assumed to be leafy vegetation (grass) and account for 0.5 of the cattle's diet. For pigs, the default assumes a pig's diet consists of equal portions of all plant types exposed, leafy, protected and root. The default assumption is that 0.1 of the diet is homegrown. The default assumption for chickens is that pasture is composed of equal proportions all plant types with 0.05 homegrown.
Agricultural mixing depth should be used for calculating soil concentration for feed and pasture contamination.

NA Not applicable. Assume F S_f is equal to zero.

* See Section 7 of *Technical Support Document for Exposure Assessment and Stochastic Analysis (OEHHA, 2000b)* for source of these values.

** 1 kg=1 L for water

4> **Feed ingestion** = Dose through the ingestion of feed (μg/d) that is harvested after it is impacted by source emissions

a> Formula EQ 5.3.4.2 H:

$$\textbf{Feed ingestion} = (1 - FG) * FIR * L * C_f \quad \textbf{(EQ 5.3.4.2 H)}$$

1: FG = Fraction of Diet provided by grazing
2: FIR = Feed ingestion rate (kg/d)
3: L = fraction of locally grown (source impacted) feed that is not pasture
4: C_f = Concentration in feed (μg/kg)

b> Recommended default values EQ 5.3.4.2 H:

1: FG = Site specific fraction of diet provided by grazing (need to survey)
2: FIR = See Table 5.2
3 L = Site specific, fraction of locally grown (source impacted) feed that is not pasture
4: C_f = As calculated above in EQ 5.3.4.1 A

5> **Pasture/Grazing ingestion** = Dose through pasture/grazing (μg/d)

a> Formula EQ 5.3.4.2 I:

Pasture/Grazing ingestion = FG * C_v * FIR (EQ 5.3.4.2 I)

1: FG = Fraction of Diet provided by grazing
2: C_v = Concentration in pasture/grazing material (μg/kg)
3: FIR = Feed ingestion rate (kg/d)

b> Recommended default values EQ 5.3.4.2 J:

1: FG = Site specific fraction of diet provided by grazing (need to survey)
2: C_v = As calculated above in EQ 5.3.4.1 A
3: FIR = See Table 5.2

6> **Soil ingestion**= Dose through soil ingestion (μg/kg)

a> Formula EQ 5.3.4.2 K: **Soil ingestion = SI_a * C_s (EQ 5.3.4.2 K)**

1: SI_a = Soil ingestion rate for animal (kg/d)

a: Formula EQ 5.3.4.2 L:

SI_a = [(1 - FG) * FS_f * FIR] +[FG * FS_p * FIR] (EQ 5.3.4.2 L)

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

4) FS_p = Soil ingested as a fraction of pasture ingested

b: Recommended default values for EQ 5.3.4.2 L:

1) FG = Site specific fraction of diet provided by grazing
2) FS_f = See Table 5.2
3) FIR = See Table 5.2
4) FS_p = See Table 5.2

2: **C_s** = Average soil concentration (μg/kg)

b> Recommended default values for EQ 5.3.4.2 K:

1: SI_a = Calculated above
2: C_s = Calculated above in EQ 5.3.2 A

7> **Tco** = Transfer coefficient of contaminant from diet to animal product (d/kg)

a> Recommended default values:

1: TCO = SEE TABLE 5.3

b> Recommended default values EQ 5.3.4.2 J:

1: FG = Site specific fraction of diet provided by grazing (need to survey)
2: C_f = As calculated above in EQ 5.3.4.1 A
3: FIR = See Table 5.2

Table 5.3 Substance Specific Default Values for Multipathway Substances(1)

				Feed to meat, milk, eggs Transfer Coefficients [3][Tco (d/kg)]			Root Uptake Factors (for inorganic compounds)					
Multipathway Substance	**Log K_{oc} [2]**	**Log K_{ow} [2]**	**Fish Biocon. Factor**	**Tco Meat**	**Tco Milk**	**Tco[3] Egg**	**Root**	**Leafy**	**Exposed & Protected**	**GRAF[4]**	**Dermal[5] Absorp. Fact.(ABS)**	**Soil Half Life (days)**
Arsenic (inorganic)	NA[6]	NA	$4.0 \times 10^{+0}$	2.0×10^{-3}	6.2×10^{-5}	2.0×10^{-3}	4.0×10^{-4}	4.0×10^{-3}	9.0×10^{-4}	1.0	0.04	$1.0 \times 10^{+8}$
Beryllium & Compounds	NA	NA	$1.9 \times 10^{+1}$	1.0×10^{-3}	9.1×10^{-7}	1.0×10^{-3}	2.0×10^{-3}	1.0×10^{-3}	2.0×10^{-4}	1.0	0.01	$1.0 \times 10^{+8}$
Cadmium & Compounds	NA	NA	$3.66 \times 10^{+2}$	5.5×10^{-4}	1.0×10^{-3}	5.5×10^{-4}	4.0×10^{-2}	6.0×10^{-2}	2.0×10^{-2}	1.0	0.001	$1.0 \times 10^{+8}$
Creosotes	NA	NA	$5.83 \times 10^{+2}$	3.4×10^{-2}	1.6×10^{-2}	3.4×10^{-2}	NA	NA	NA	1.0	0.13	$5.7 \times 10^{+2}$
Chromium VI & Cmpds	NA	NA	$2.0 \times 10^{+0}$	9.2×10^{-3}	1.0×10^{-5}	9.2×10^{-3}	1.0×10^{-3}	8.0×10^{-4}	7.0×10^{-4}	1.0	0.01	$1.0 \times 10^{+8}$
Diethylhexylphthalate	4.72	5.11	$4.83 \times 10^{+2}$	NA	NA	NA	NA	NA	NA	1.0	0.10	$2.3 \times 10^{+1}$
Dioxins and Furans	NA	NA	$1.9 \times 10^{+4}$	4.0×10^{-1}	4.0×10^{-2}	4.0×10^{-1}	NA	NA	NA	0.43	0.02	$4.72 \times 10^{+3}$
Hexachlorocyclohexanes	NA	NA	$4.56 \times 10^{+2}$	NA	NA	NA	NA	NA	NA	1.0	0.10	$6.7 \times 10^{+1}$
Lead & Compounds (inorganic)	NA	NA	$1.55 \times 10^{+2}$	4.0×10^{-4}	2.6×10^{-4}	4.0×10^{-4}	2.0×10^{-3}	5.0×10^{-3}	1.0×10^{-3}	1.0	0.01	$1.0 \times 10^{+8}$
Mercury (inorganic)	NA	NA	$5.0 \times 10^{+3}$	2.7×10^{-2}	9.7×10^{-6}	2.7×10^{-2}	5.0×10^{-2}	9.0×10^{-2}	3.0×10^{-2}	1.0	0.10	$1.0 \times 10^{+8}$
Nickel and compounds	NA	NA	NA	2.0×10^{-3}	1.0×10^{-3}	2.0×10^{-3}	2.0×10^{-2}	6.0×10^{-3}	9.0×10^{-3}	1.0	0.04	$1.0 \times 10^{+8}$
4,4'-Methylene dianiline	2.24	1.59	$1.11 \times 10^{+1}$	NA	NA	NA	NA	NA	NA	1.0	0.10	$4.0 \times 10^{+0}$
PAH as Benzo(a)pyrene	NA	NA	$5.83 \times 10^{+2}$	3.4×10^{-2}	1.6×10^{-2}	3.4×10^{-2}	NA	NA	NA	1.0	0.13	$5.7 \times 10^{+2}$
Polychlorinated Biphenyls	NA	NA	$9.97 \times 10^{+4}$	5.0×10^{-2}	1.0×10^{-2}	5.0×10^{-2}	NA	NA	NA	1.0	0.14	$9.4 \times 10^{+2}$

(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) Values for the Egg Transfer Coefficients have not been developed but are assumed to be similar to meat transfer coefficients cited in the SCAQMD document.
(4) 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.
(5) Dermal absorption of many compounds is limited. The guidelines have incorporated dermal absorption factors to account for the decreased absorption relative to other routes of exposure, for estimates of dermal dose used to assess both cancer and noncancer health hazards. The dermal absorption values come from literature describing absorption of chemicals across the skin. In some cases, there are good data available for specific compounds. In other cases, an absorption fraction is inferred from data for similar chemicals. In a few cases the effects of adsorption to a soil or fly ash matrix on dermal bioavailability have been studied. In these rare instances, the dermal absorption factor used in the guidelines accounts for this decreased bioavailability (e.g., the dermal absorption value for dioxins/furans accounts for decreased bioavailability).
NA - Data Not Available or Not Applicable.

b> Assumptions:

1: The transfer coefficient is the same for all exposure routes.
2: The transfer coefficient for all meat is the same.
3: The transfer coefficient for eggs is the same as for meat.

3. Fish Products

The average concentration in fish (C_f) is based on the concentration in water and a bioconcentration factor.

a. Formula EQ 5.3.4.3 M: $$C_f = C_w * BCF \quad \text{(EQ 5.3.4.3 M)}$$

1> C_f = Concentration in fish (μg/kg)
2> C_w = Concentration in water (μg/kg)
3> BCF = Bioconcentration factor

b. Recommended default values for EQ 5.3.4.3 M:

1> C_w = Calculated above in EQ 5.3.3 A
2> BCF = See Table 5.3

c. Assumptions for EQ 5.3.4.3 M:

1> All contaminants in water are available for bioconcentration.
2> Contaminant is present in a soil or fly ash matrix.
Contaminant concentrations are uniform in water based on dispersion.
4> Only bioconcentration is currently considered. Bioaccumulation from the food chain is not considered.

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 lifetime average daily dose (LADD). The following algorithms calculate this dose for exposure through inhalation, dermal absorption, and ingestion pathways. 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 that should be used for children are listed under the nine-year exposure duration. The point-estimates and data distributions that should be used for adults are listed under the 30 and 70-year exposure duration. Workers are addressed as adults using single point-estimates for three exposure pathways. Point-estimates for workers are listed under "worker (single value)."
OEHHA has not generated or endorsed 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

Exposure through inhalation (Dose-inh) is a function of the respiration rate and the concentration of a substance in the air.

1. Formula EQ 5.4.1 A:

$$\text{Dose-inh} = \frac{C_{air} * \{DBR\} * A * EF * ED * 10^{-6}}{AT} \qquad \text{(EQ 5.4.1 A)}$$

where:

Dose-inh	=	Dose through inhalation (mg/kg/d)
10^{-6}	=	Micrograms to milligrams conversion, Liters to cubic meters conversion
C_{air}	=	Concentration in air ($\mu g/m^3$)
{DBR}	=	Daily breathing rate (L/kg body weight - day)
A	=	Inhalation absorption factor
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
AT	=	Averaging time period over which exposure is averaged, in days (e.g., 25,550 d for 70 yr for cancer risk)

2. Recommended default values for EQ 5.4.1 A:

a. EF = 350 d/y
b. ED = 9; 30; or 70 yr
c. AT = 25,550 days
d. A = 1
e. {DBR} 9, 30 & 70 year exposure = see Table 5.4
f. {DBR} 30 and 70 year exposure = see Table 5. 5 for parametric models (distributions for Tier 3 stochastic risk assessment)

Table 5.4 Point Estimates for Daily Breathing Rate for 9, 30, and 70-year Exposure Durations (DBR) (L/kg BW * Day)

9-Year Exposure Duration		30 & 70-Year Exposure Duration		Off-site[1] Worker
Average	High End	Average	High End	(Single Value)
452	581	271	393	149

[1]This value corresponds to a 70 kg worker breathing 1.3 m^3/hour for an eight hour day. 1.3 m^3/hr is the breathing rate recommended by U.S.EPA, (1997a) as an hourly average for outdoor workers.

Table 5.5 Breathing Rate Distributions for 9, 30, and 70-Year Exposure Durations for Stochastic Analysis (L/kg BW * Day)

	9-Year Exposure Duration	30 & 70-Year Exposure Duration
Distribution Type	Gamma	Gamma
Location	301.67	193.99
Scale	29.59	31.27
Shape	5.06	2.46

3. Assumption for EQ 5.4.1 A:

a. 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.

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. Distributions are not available for stochastic analysis. Tier III stochastic risk assessments should include the dermal pathway as a high end point estimate.

1. Formula EQ 5.4.2 A:

$$\textbf{Dose-dermal} = C_s * SA * SL * Ef * ABS * 10^{-9} * ED/BW * AT \quad \textbf{(EQ 5.4.2 A)}$$

Where:

Dose-dermal	=	Exposure dose through dermal absorption (mg/kg/d)
C_s	=	Average soil concentration (µg/kg)
SA	=	Surface area of exposed skin (cm^2)
SL	=	Soil loading on skin (mg/cm^2-d)
ABS	=	Fraction absorbed across skin
BW	=	Body weight (kg)
10^{-9}	=	Micrograms to kilogram conversion factor (µg/kg)
EF	=	(EF defined in Table 5.6) (days/year)
AT	=	25,550 days (70 years)
ED	=	Exposure Duration (years)

2. Recommended default values for EQ 5.4.2 A:

a. C_s = Calculated above in EQ 5.3.2 A
b. SA = See Table 5.6
c. SL = See Table 5.6
d. ABS = See Table 5.3
e. BW = See Table 5.6
f. f = See Table 5.6

Table 5.6 Recommended Point Estimate Values for Dermal Pathway for 9, 30, and 70 Year Exposure Durations and Worker[1]

	9 Year[1] Exposure Duration		30 & 70 Year Exposure Duration		Worker[2] (Single Value)
BW Body Weight (kg)	18		63		70
	Average	High End	Average	High End	
SL Soil Loading (mg/cm^2-day)[3]	0.2	1.0	0.2	1.0	1.0
EF Exposure Frequency (d/yr)	228	350	121	350	245
SA Surface Area Exposed (cm^2)	2,778	3,044	4,700	5,500	5,800

1. OEHHA, 2000b, page 6-10 contains surface area exposed and exposure frequency recommended values for children (1- 6) and adults (>6). For the 9 year average surface area exposed, a time weighted average value for ages 0-9 was derived with following formula (5/9 x 2000) + (3/9 x 5000) = 2,778 cm^2. For the 9 year high-end surface area exposed, (5/9 x 2000) + (3/9 x 5800) = 3,044 cm^2. It is assumed that dermal exposure to outdoor soil does not occur the first year of life. For exposure frequency the same approach was used:
(5/9 x 350) + (3/9 x 100) = 228 (d/yr) for average.
2. Worker values for surface area exposed and soil loading are the high end adult values from page 6-10, OEHHA, 2000b. The exposure frequency assumes that the worker works 49 weeks per year, 5 days per week and that he or she is exposed everyday at work.
3. For Hot Spots risk assessments it is assumed that one event occurs per day.

5.4.3 Estimation of Exposure Through Ingestion

Exposure through ingestion is a function of the concentration of the substance in the substance ingested (soil, water, and food), the gastrointestinal absorption of the substance in a soil or fly ash matrix, and the amount ingested.

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. The dose from inadvertent soil ingestion can be estimated by the point estimate approach using the following general equation:

$$\text{Dose} = \frac{C_{soil} \times GRAF \times SIR \times EF \times ED \times 10^{-9}}{AT} \qquad \text{(EQ 5.4.3.1 A)}$$

where:

Dose = dose from soil ingestion (mg/kg BW *day)
10^{-9} = conversion factor (mg/μg) (kg/mg)
C_{soil} = concentration of contaminant in soil (μg/g)

GRAF = gastrointestinal relative absorption fraction, unitless; chemical-specific (see Table 5.3)
SIR = soil ingestion rate (mg/kg BW * day) (see Table 5.7)
EF = exposure frequency (days/year)
ED = exposure duration (years)
AT = averaging time, period of time over which exposure is averaged (days); for noncancer endpoints, AT = ED x 365 d/yr; for cancer risk estimates, AT = 70 yr x 365 d/yr = 25,550 days

b. Recommended default values for EQ 5.4.3.1 A:

a. GRAF = Table 5.3
b. SIR = Table 5.7
c. EF = 350 d/year resident, 245 d/year worker
d. ED = 9, 30, or 70 yr
e. AT = 25,550 days

Table 5.7 Soil Ingestion Rates (SIR) for 9, 30 and 70-Year Exposure Durations and Off-site Worker.

	9-Year Exposure Duration	30 & 70-Year Exposure Duration	Off-site[1] Worker
Soil Ingestion Rate (mg/kg BW *Day)	8.7	1.7	1.4

1. The soil ingestion rate of 1.4 (mg/kg BW * day) corresponds to the OEHHA, 2000b recommendation of 100 mg/day for a 70 kg adult.

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.

2. **Exposure through Ingestion of Water**

a. Formula EQ 5.4.3.2 B:

$$\textbf{Dose-w} = C_w * \textbf{WIR} * AB_{ing} * F_{dw} * \textbf{EF} * \textbf{ED} * 10^{-6} / \textbf{AT} \quad \textbf{(EQ 5.4.3.2 B)}$$

where:

Dose-w	=	Exposure dose through ingestion of water (mg/kg/d)
C_w	=	Water concentration (μg/kg)
WIR	=	Water ingestion rate (ml/kg BW/day)
AB_{ing}	=	Gastrointestinal absorption factor
F_{dw}	=	Fraction of drinking water from contaminated source
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
10^{-6}	=	Conversion factor (μg/mg)(L/ml)

b. Recommended default values for EQ 5.4.3.2 B:

1>	C_w	=	Calculated above 5.3.3 A
2>	WIR	=	See Tables 5.8 and 5.9
3>	AB_{ing}	=	Default set to 1
4>	EF	=	350 d/yr
5>	ED	=	9, 30, or 70 yrs
6>	AT	=	25,550 days

Table 5.8
Point Estimate Water Consumption Ingestion Rates (WIR) for 9, 30, and 70-Year Exposure Durations (ml/kg BW * day)

9-Year Exposure Duration		30 and 70-Year Exposure Duration	
Average	High End	Average	High End
40	81	24	54

Table 5.9
Water Ingestion Lognormal Distributions for 9, 30, and 70-Year Exposure Durations (ml/kg BW * day) (Stochastic Analysis)

Distribution Type	9-Year Exposure Duration		30 & 70-Year Exposure Duration	
Lognormal	Mean ± S.D.	μ ± σ	Mean ± S.D.	μ ± σ
Lognormal	40.03 ± 21.45	3.57 ± 0.50	24.2 ± 17.0	2.99 ± 0.63

3. Exposure through Ingestion of Food

The exposure through food ingestion can be through ingestion of plant products, animal products (including fish), and mother's milk.

a. Plant products

Exposure through ingesting plants (Dose-p) is a function of the type of plant, gastrointestinal absorption factor, bioavailability and the fraction of plants ingested that are homegrown. The calculation is done for each type of plant, then summed to get total dose for this pathway.

1> Formula EQ 5.4.3.3.a C:

$$\text{Dose-p} = (C_f * IP * GRAF * L * EF * ED * 10^{-6}) / AT \quad \text{(EQ 5.4.3.3.a C)}$$

a> Dose-p = Exposure dose through ingestion of plant products (mg/kg/d)
b> C_f = Concentration in plant type (μg/kg)
c> IP = Consumption of exposed, leafy, protected, or root produce (g/kg*day)
d> GRAF = Gastrointestinal relative absorption factor
e> L = Fraction of exposed, leafy, protected, or root produce homegrown
f> EF = Exposure frequency (days/year)
g> ED = Exposure duration (years)
h> 10^{-6} = Conversion factor (μg/kg to mg/g)
i> AT = Averaging time, period over which exposure is averaged (days)

2> Recommended default values for EQ 5.4.3.3.a C:

a> C_f = Calculated above in EQ 5.3.4.1 A
b> IP = See Tables 5.10 to 5.12
c> GRAF = See Table 5.3
d> L = Site specific fraction of produce homegrown or locally produced. For nonurban sites 0.15 may be used as a default. For urban sites 0.052 may be used (USEPA, 1997b).
e> EF = 350 d/yr
f> ED = 70 yrs
g> AT = 25,550 days

Table 5.10
Point Estimates for Per Capita Food Consumption Rates (g/Kg BW * Day)

	9-Year Exposure Duration		30 & 70-Year Exposure Durations	
	Average	High End	Average	High End
Produce				
Exposed	4.16	15.7	3.56	12.1
Leafy	2.92	10.9	2.90	10.6
Protected	1.63	6.66	1.39	4.88
Root	4.08	14.9	3.16	10.5
Meat				
Beef	2.24	7.97	2.25	6.97
Chicken	1.80	4.77	1.46	5.02
Pork	1.31	5.10	1.39	4.59
Dairy	12.0	51.9	5.46	17.4
Eggs	3.21	10.3	1.80	5.39

Table 5.11
Parametric Models for Ages 0-9 Food Consumption Distributions (g/kg BW * Day) (Stochastic Analysis)

Food Category	Distribution Type	Mean	Std. Dev.	Location	Scale	Shape	$\mu \pm \sigma$
Produce							
Exposed	Lognormal	3.93	5.49				exp(0.83±1.04)
Leafy	Lognormal	2.83	3.89				exp(0.43±1.03)
Protected	Weibull			0.13	1.21	0.71	
Root	Lognormal	4.08	5.91				exp(0.84±1.06)
Meat							
Beef	Weibull			0.24	1.72	0.77	
Chicken	Gamma			0.25	2.94	0.53	
Pork	Weibull			0.18	0.97	0.78	
Dairy	Lognormal	11.32	18.3				exp(1.78±1.13)
Eggs	Weibull			0.26	2.67	0.82	

Table 5.12
Parametric Models for Ages 0-70 Food Consumption Distributions (g/kg BW * Day) (Stochastic Analysis)

Category of Food	Mean	Standard Deviation	Distribution Type	$\mu \pm \sigma$
Produce				
Exposed	3.43	6.16	Lognormal	Exp (0.51±1.20)
Leafy	2.97	4.95	Lognormal	Exp (0.42±1.15)
Protected	1.39	2.43	Lognormal	Exp (-0.37±1.18)
Root	3.07	5.23	Lognormal	Exp (0.44±1.17)
Meat				
Beef	2.32	3.50	Lognormal	Exp (0.25±1.09)
Chicken	1.44	2.19	Lognormal	Exp (-0.23±1.09)
Pork	1.42	2.30	Lognormal	Exp (-0.29±1.13)
Dairy	5.57	10.5	Lognormal	Exp (0.96±1.23)
Eggs	1.84	2.60	Lognormal	Exp (0.061±1.05)

Table 5.13
Default Values for Fisher–caught Fish Consumption (g/kg BW * Day)

	9, 30, & 70-Year Exposure Scenario
Average	0.48
High-End	1.35

Table 5.14
Parametric Model for Fisher-caught Fish Consumption Distribution for 9, 30 and 70-Year Exposure Scenarios (g/kg BW *Day) (stochastic analysis).

Mean	Standard. Deviation	Distribution Type	$\mu \pm \sigma$
0.48	0.71	Lognormal	exp(-1.31 ± 1.08)

b. Animal Products (Including Fisher-caught Fish)

Exposure through animal product ingestion (Dose-ap) is a function of what type of meat and/or fish is ingested, as well as animal milk products and eggs. The calculation is done for each type and then summed to get the total dose for this pathway.

1> Formula 5.4.3.3.b D:

$$\textbf{Dose-ap} = C_{fa} * If * GI * L * EF * ED * 10^{-6} / AT \qquad \textbf{(EQ 5.4.3.3.b D)}$$

a> Dose-ap = Exposure dose through ingestion of animal or fish products (mg/kg BW * day)
b> C_{fa} = Concentration in animal product (μg/kg)
c> If = Consumption of animal product (g/kg BW per day), e.g, beef, chicken, pork, diary, eggs, fish
d> GI = Gastrointestinal absorption factor
e> L = Fraction of animal product homegrown
f> EF = Exposure frequency (days/year)
g> ED = Exposure duration (years)
h> AT = Averaging time (days)
i> 10^{-6} = Conversion factor (μg/kg to mg/g) for C_f term

2> Recommended default values for EQ 5.4.3.3.b D:

a> C_{fa} = Calculated above in EQ 5.3.4.2 E
b> If = See Tables 5.10, 5.11, and 5.12. For fish ingestion rates see Table 5.13. For distributions (parametric models) for Tier 3 risk assessments see Tables 5.11, 5.12, and 5.14.
c> GI = Default set to 1.
d> L = Site specific fraction of product locally produced.
e> EF = 350 d/yr
f> ED = 70 yrs
G> AT = 25,550 DAYS

c. Mother's Milk

Exposure through mother's milk ingestion (Dose-Im) is a function of the average substance concentration in mother's milk and the amount of mother's milk ingested. The minimum pathways that the nursing mother is exposed to include inhalation, soil ingestion and dermal, since the chemicals evaluated by the mother's milk pathway are multipathway chemicals. Other pathways may be appropriate depending on site conditions (e.g. presence of vegetable gardens or home grown chickens). 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 on pages 20-26.

1> Formula 5.4.3.3.c E:

$$\text{Dose-Im} = C_m * BMI_{bw} * F * yr / 25{,}550 \quad \text{(EQ 5.4.3.3.c E)}$$

a> Dose-Im = Exposure dose through ingestion of mother's milk (mg/kg BW/d)

b> C_m = Concentration of contaminant in mother's milk is a function of the mother's exposure through all routes and the contaminant half-life in the body (mg/g milk)

1: Formula 5.4.3.3.c F:

$$Cm = E_{mi} * t_{1/2} * f_1 * f_3 * 10^{-3} / (f_2 * 0.693) \quad \text{(EQ 5.4.3.3.c F)}$$

a: E_{mi} = Average daily maternal intake of contaminant from all routes (mg/kg/d)
b: $t_{1/2}$ = Half-life of contaminant in mother (d)
c: f_1 = Fraction of contaminant that partitions to mother's fat
d: f_3 = Fraction of fat of mother's milk (kg fat/kg milk)
e: f_2 = Fraction of mother's weight that is fat(kg fat/kg bw)
f: 10^{-3} = Conversion factor (g to kg milk)
g: 0.693 = Natural log of 2

2: Recommended default values for EQ 5.4.3.3.c F:

a: E_{mi} = Sum of doses
b: $t_{1/2}$ = 2,117 (d) for PCDDs/PCDFs = 5.8 yr
1,460 (d) for both PCBs
c: f_1 = 0.8
d: f_3 = 0.04 (kg fat/kg milk)
e: f_2 = 0.33 (kg fat/kg BW)

c> BMI_{bw} = Daily breast-milk ingestion rate (g/kg BW*day)
d> F = Frequency of exposure (d/yr)
e> yr = Breast-feeding period (yr)

f> 25,550 = Exposure period (d)

2> Recommended default values for EQ 5.4.3.3.c E:

a> BMI_{bw} = see Table 5.15
For distribution (parametric model) for Tier 3 stochastic

risk assessments see Table 5.16

b> F = 365 (d)

c> yr = 1(yr)

3> Assumptions for EQ 5.4.3.3.c E:

a> For the MEIR, mother is exposed for 25 years, the child receives milk for another year, and then the nursing infant is exposed for 9, 30, or 70 years.

b> For the 9, 30, and 70 year exposure duration scenarios, the total toxicant dose from the breast-feeding in the first year of life is assumed to be spread over 70 years in order to calculate an average daily dose.

Table 5.15
Point Estimate Values for Breast Milk Consumption Rate
(g/kg BW *day)

	9, 30, and 70-Year Exposure Durations
Average	102
High End	138

Table 5.16
Parametric Model for Breast Milk Consumption Rate for
9, 30, and 70 Year Exposure Durations (Stochastic Analysis) (g/kg BW *day)

Distribution Type	Mean ± S.D.
Normal	102 ± 21.8

5.5 References for Kow and Koc Values in Table 5.3

Table 5.17 References for Kow Values

Compound	Notes	Reference
Diethylhexlyphthalate	Level 1 calculation	Mackay *et al*. (1995)
4,4'-Methylene dianiline	Measured	Hansch *et al*. (1985)

Table 5.18 References for Koc Values

Compound	Notes	Reference
Diethylhexylphthalate	Level 1 calculation	Mackay *et al*. (1995)
4,4'-Methylene dianiline	Estimated according to methodology of Lyman *et al*. (1990)	Lyman *et al*. (1990)

6. Dose-Response Assessment for Noncarcinogenic Endpoints

6.1 Derivation of Toxicity Criteria

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). For noncarcinogens, dose-response information is presented in the form of Reference Exposure Levels (RELs). RELs are concentrations or doses at or below which adverse effects are not likely to occur following specified exposure conditions. The methodology for developing chronic RELs is fundamentally the same as that used by U.S. EPA in developing the inhalation Reference Concentrations (RfCs) and oral Reference Doses (RfDs).

Acute and chronic RELs are frequently calculated by dividing the no observed adverse effect level (NOAEL) or lowest observed adverse effect levels (LOAEL) in human or animal studies by uncertainty factors. Uncertainty factors are applied to account for interspecies extrapolation, intraspecies variability, the use of subchronic studies to extrapolate to chronic effects, and use of a LOAEL instead of a NOAEL. Total uncertainty factors range from one to three thousand for current RELs. Haber's equation is used, where needed, to adjust studies with different exposure times to the one-hour period needed for most acute RELs. Currently, there are eight acute RELs with reproductive health endpoints, which have exposure time periods different from one-hour; these alternative exposure periods include four, six, and seven hours. The most sensitive toxicological end point is selected as the basis for the REL when there are multiple adverse health effects. A slightly more complicated methodology, the Benchmark Concentration approach, is described in OEHHA, 1999a. 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.

It should be emphasized that exceeding the acute or chronic REL does not necessarily indicate that an adverse health impact will occur. 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 an 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). For detailed information on the methodology and derivations for acute RELs, see the *Air Toxics Hot Spots Program Risk Assessment Guidelines; Part I; The Determination of Acute Reference Exposure Levels for Airborne Toxicants (OEHHA 1999a)* (Part I TSD). For information on chronic RELS see the *Air Toxics Hot Spots Program*

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Risk Assessment Guidelines; Part III; Technical Support Document for the Determination of Chronic Reference Exposure Levels (OEHHA 2000a) (Part III TSD*).*

Tables 6.1 and 6.2 list the currently adopted acute and chronic inhalation RELs. Some substances that pose a chronic inhalation hazard may also present a chronic hazard via non-inhalation (oral) 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 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 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 Description of Acute Reference Exposure Levels

OEHHA developed acute RELs for assessing potential noncancer health impacts for short-term, generally one-hour peak exposures to facility emissions. (A few RELs are for 4 to 7-hour peak exposures.) 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. 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. Some acute RELs are based on reproductive/developmental endpoints, such as teratogenicity or fetotoxicity, which are considered severe adverse effects. The RELs, target organ systems, and the averaging time for substances that can present a potential acute hazard from inhalation are presented in Table 6.1. Unlike the chronic RELs discussed in the following section, there are no acute noninhalation RELs. 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.

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Table 6.1 Acute Reference Exposure Levels and Target Organ Systems Impacted

Substance	Chemical Abstract Service Number (CAS)	Acute Inhalation REL ($\mu g/m^3$)	Averaging [a] Time (hour)	Acute Hazard Index Target Organ Systems(s)
Acrolein	107-02-8	1.9×10^{-1}	1	Eyes; Respiratory System
Acrylic Acid	79-10-7	$6.0 \times 10^{+3}$	1	Eyes; Respiratory System
Ammonia	7664-41-7	$3.2 \times 10^{+3}$	1	Eyes; Respiratory System
Arsenic and Inorganic Arsenic Compounds	7440-38-2	1.9×10^{-1}	4	Reproductive/Developmental
Arsine	7784-42-1	$1.6 \times 10^{+2}$	1	Hematologic System
Benzene	71-43-2	$1.3 \times 10^{+3}$	6	Hematologic System; Immune System; Reproductive/Developmental
Benzyl Chloride	100-44-7	$2.4 \times 10^{+2}$	1	Eyes; Respiratory System
Carbon Disulfide	75-15-0	$6.2 \times 10^{+3}$	6	Nervous System; Reproductive/Developmental
Carbon Monoxide [b]	630-08-0	$2.3 \times 10^{+4}$	1	Cardiovascular System
Carbon Tetrachloride	56-23-5	$1.9 \times 10^{+3}$	7	Alimentary Tract; Nervous System; Reproductive/Developmental
Chlorine	7782-50-5	$2.1 \times 10^{+2}$	1	Eyes; Respiratory System
Chloroform	67-66-3	$1.5 \times 10^{+2}$	7	Nervous System; Reproductive/Developmental
Chloropicrin	76-06-2	$2.9 \times 10^{+1}$	1	Eyes; Respiratory System
Copper and Compounds	7440-50-8	$1.0 \times 10^{+2}$	1	Respiratory System
1,4-Dioxane	123-91-1	$3.0 \times 10^{+3}$	1	Eyes; Respiratory System
Epichlorohydrin	106-89-8	$1.3 \times 10^{+3}$	1	Eyes; Respiratory System
Ethylene Glycol Monobutyl Ether	111-76-2	$1.4 \times 10^{+4}$	1	Eyes; Respiratory System
Ethylene Glycol Monoethyl Ether	110-80-5	$3.7 \times 10^{+2}$	6	Reproductive/Developmental
Ethylene Glycol Monoethyl Ether Acetate	111-15-9	$1.4 \times 10^{+2}$	6	Nervous System; Reproductive/Developmental
Ethylene Glycol Monomethyl Ether	109-86-4	$9.3 \times 10^{+1}$	6	Reproductive/Developmental
Formaldehyde	50-00-0	$9.4 \times 10^{+1}$	1	Eyes; Immune System; Respiratory
Hydrogen Chloride	7647-01-0	$2.1 \times 10^{+3}$	1	Eyes; Respiratory System
Hydrogen Cyanide	74-90-8	$3.4 \times 10^{+2}$	1	Nervous System
Hydrogen Fluoride	7664-39-3	$2.4 \times 10^{+2}$	1	Eyes; Respiratory System
Hydrogen Selenide	7783-07-5	$5.0 \times 10^{+0}$	1	Eyes; Respiratory System
Hydrogen Sulfide [b]	7783-06-4	$4.2 \times 10^{+1}$	1	Nervous System
Isopropyl Alcohol	67-63-0	$3.2 \times 10^{+3}$	1	Eyes; Respiratory System
Mercury (Inorganic)	7439-97-6	$1.8 \times 10^{+0}$	1	Reproductive/Developmental
Methanol	67-56-1	$2.8 \times 10^{+4}$	1	Nervous System

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Table 6.1 Acute Reference Exposure Levels and Target Organ Systems Impacted

Substance	Chemical Abstract Service Number (CAS)	Acute Inhalation REL ($\mu g/m^3$)	Averaging Time (hour) [a]	Acute Hazard Index Target Organ Systems(s)
Methyl Bromide	74-83-9	$3.9 \times 10^{+3}$	1	Nervous System; Respiratory Irritation; Reproductive/Developmental
Methyl Chloroform	71-55-6	$6.8 \times 10^{+4}$	1	Nervous System
Methyl Ethyl Ketone	78-93-3	$1.3 \times 10^{+4}$	1	Eyes; Respiratory System
Methylene Chloride	75-09-2	$1.4 \times 10^{+4}$	1	Nervous System
Nickel and Nickel Compounds	7440-02-0	$6.0 \times 10^{+0}$	1	Immune System; Respiratory System
Nitric Acid	7697-37-2	$8.6 \times 10^{+1}$	1	Respiratory System
Nitrogen Dioxide [b]	10102-44-0	$4.7 \times 10^{+2}$	1	Respiratory System
Ozone [b]	10028-15-6	$1.8 \times 10^{+2}$	1	Eyes; Respiratory System
Perchloroethylene	127-18-4	$2.0 \times 10^{+4}$	1	Eyes; Nervous System; Respiratory System
Phenol	108-95-2	$5.8 \times 10^{+3}$	1	Eyes; Respiratory System
Phosgene	75-44-5	$4.0 \times 10^{+0}$	1	Respiratory System
Propylene Oxide	75-56-9	$3.1 \times 10^{+3}$	1	Eyes; Respiratory System; Reproductive/Developmental
Sodium Hydroxide	1310-73-2	$8.0 \times 10^{+0}$	1	Eyes; Skin; Respiratory System
Styrene	100-42-5	$2.1 \times 10^{+4}$	1	Eyes; Respiratory System
Sulfates [b]	N/A	$1.2 \times 10^{+2}$	1	Respiratory System
Sulfur Dioxide [b]	7446-09-5	$6.6 \times 10^{+2}$	1	Respiratory System
Sulfuric Acid and Oleum	7664-93-9 8014-95-7	$1.2 \times 10^{+2}$	1	Respiratory System
Toluene	108-88-3	$3.7 \times 10^{+4}$	1	Nervous System; Eyes; Respiratory System; Reproductive/Developmental
Triethylamine	121-44-8	$2.8 \times 10^{+3}$	1	Nervous System; Eyes
Vanadium Pentoxide	1314-62-1	$3.0 \times 10^{+1}$	1	Eyes; Respiratory System
Vinyl Chloride	75-01-4	$1.8 \times 10^{+5}$	1	Nervous System; Eyes; Respiratory System
Xylenes (m,o,p-isomers)	1330-20-7	$2.2 \times 10^{+4}$	1	Eyes; Respiratory System

a. The averaging period of noncancer acute RELs is generally a one-hour exposure. However, some are based on several hour exposure for reproductive/developmental endpoints (see section 1.6 of the Part I TSD). The RELs for the following substances must be compared to modeled emission concentrations of the same duration rather than maximum one-hour concentrations (e.g., a 4-hour REL should be compared to the maximum 4-hour average concentration from the air dispersion model).

b. California Ambient Air Quality Standard

6.3 Description of Chronic Reference Exposure Levels

OEHHA has developed chronic RELs for assessing noncancer health impacts from long-term exposure. (See the Part III TSD for detailed information on the development of noncancer chronic inhalation and oral RELs.) A chronic REL is a concentration level (that is expressed in units of micrograms per cubic meter ($\mu g/m^3$) for inhalation exposure and in a dose expressed in units of milligram 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 as 12% of a lifetime, or about eight years for humans. Table 6.2 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 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 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.

The most sensitive organ system(s) associated with each chronic REL are also presented in Table 6.2. 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 6.2 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	$9.0 \times 10^{+0}$	Respiratory System
Acrolein	107-02-8	6.0×10^{-2}	Eyes; 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	3.0×10^{-2}	Cardiovascular System; Developmental; Nervous System
Benzene	71-43-2	$6.0 \times 10^{+1}$	Developmental; Hematopoietic System; Nervous System
Beryllium and Beryllium Compounds	7440-41-7	7.0×10^{-3}	Immune System; Respiratory System
Butadiene	106-99-0	$2.0 \times 10^{+1}$	Reproductive System
Cadmium and Cadmium Compounds	7440-43-9	2.0×10^{-2}	Kidney; 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; Developmental; 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; Developmental; Endocrine System; Hematopoietic 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	4.0×10^{-1}	
Chlorinated Dibenzofurans [b]			
2,3,7,8-Tetrachlorodibenzofuran [b]	5120-73-19	4.0×10^{-4}	Alimentary System; Developmental; Endocrine System; Hematopoietic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzofuran [b]	57117-41-6	8.0×10^{-4}	
2,3,4,7,8-Pentachlorodibenzofuran [b]	57117-31-4	8.0×10^{-5}	
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}	

Table 6.2 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)
1,2,3,7,8,9-Hexachlorodibenzofuran [b]	72918-21-9	4.0×10^{-4}	Alimentary System; Developmental; Endocrine System; Hematopoietic System; Reproductive System; Respiratory System
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	4.0×10^{-1}	
Chlorobenzene	108-90-7	$1.0 \times 10^{+3}$	Alimentary System; Kidney; Reproductive System
Chloroform	67-66-3	$3.0 \times 10^{+2}$	Alimentary System; Developmental; 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; Kidney; Nervous System; Respiratory System;
1,1-Dichloroethylene (Vinylidene Chloride)	75-35-4	$7.0 \times 10^{+1}$	Alimentary System
Diesel Exhaust [a]	N/A	$5.0 \times 10^{+0}$	Respiratory System
Diethanolamine	111-42-2	$3.0 \times 10^{+0}$	Cardiovascular System; Nervous System
N,N-Dimethylformamide	68-12-2	$8.0 \times 10^{+1}$	Alimentary System; Respiratory System
1,4-Dioxane	123-91-1	$3.0 \times 10^{+3}$	Alimentary System; 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); Developmental; Endocrine System; Kidney
Ethyl Chloride	75-00-3	$3.0 \times 10^{+4}$	Alimentary System; Developmental
Ethylene Dibromide	106-93-4	8.0×10^{-1}	Reproductive
Ethylene Dichloride	107-06-2	$4.0 \times 10^{+2}$	Alimentary System (Liver)
Ethylene Glycol	107-21-1	$4.0 \times 10^{+2}$	Developmental; Kidney; Respiratory System
Ethylene Glycol Monoethyl Ether	110-80-5	$7.0 \times 10^{+1}$	Hematopoietic System; Reproductive System
Ethylene Glycol Monoethyl Ether Acetate	111-15-9	$3.0 \times 10^{+2}$	Developmental

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Table 6.2 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
Formaldehyde	50-00-0	$3.0 \times 10^{+0}$	Eyes; Respiratory System
Fluorides		$1.3 \times 10^{+1}$	Bone and Teeth, 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; 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; Developmental
Isopropanol	67-63-0	$7.0 \times 10^{+3}$	Developmental; Kidney
Maleic Anhydride	108-31-6	7.0×10^{-1}	Respiratory System
Manganese & Manganese Compounds	7439-96-5	2.0×10^{-1}	Nervous System
Mercury & Mercury Compounds (inorganic)	7439-97-6	9.0×10^{-2}	Nervous System
Methanol	67-56-1	$4.0 \times 10^{+3}$	Developmental
Methyl Bromide	74-83-9	$5.0 \times 10^{+0}$	Developmental; Nervous System; Respiratory System
Methyl tertiary-Butyl Ether	1634-04-4	$8.0 \times 10^{+3}$	Alimentary System; Eyes; Kidney
Methyl Chloroform	71-55-6	$1.0 \times 10^{+3}$	Nervous System
Methyl Isocyanate	624-83-9	$1.0 \times 10^{+0}$	Reproductive; Respiratory System
Methylene Chloride	75-09-2	$4.0 \times 10^{+2}$	Cardiovascular System; Nervous System
4,4'-Methylene Dianiline (and its dichloride)	101-77-9	$2.0 \times 10^{+1}$	Alimentary System; 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	5.0×10^{-2}	Hematopoietic System; Respiratory System

Table 6.2 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)
Nickel Oxide	1313-99-1	1.0×10^{-1}	Hematopoietic System; Respiratory System
Phenol	108-95-2	$2.0 \times 10^{+2}$	Alimentary System; Cardiovascular System; Kidney; Nervous System
Phosphine	7803-51-2	8.0×10^{-1}	Alimentary System; Hematopoietic System; Kidney; Nervous System; Respiratory System
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[P4] (PCBs) (speciated)[b]			
3,3',4,4'-Tetrachlorobiphenyl (77)[b]	35298-13-3	4.0×10^{-1}	Alimentary System; Developmental; Endocrine System; Hematopoietic System; Reproductive System; Respiratory System
3,4,4',5-Tetrachlorobiphenyl (81)[b]	70362-50-4	4.0×10^{-1}	
2,3,3',4,4'- Pentachlorobiphenyl (105)[b]	32598-14-4	4.0×10^{-1}	
2,3,4,4'5- Pentachlorobiphenyl (114)[b]	74472-37-0	8.0×10^{-2}	
2,3'4,4',5- Pentachlorobiphenyl (118)[b]	31508-00-6	4.0×10^{-1}	
2',3,4,4',5- Pentachlorobiphenyl (123)[b]	65510-44-3	4.0×10^{-1}	
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	8.0×10^{-2}	
2,3,3',4,4',5'-Hexachlorobiphenyl (157)[b]	69782-90-7	8.0×10^{-2}	
2,3',4,4',5,5'-Hexachlorobiphenyl (167)[b]	52663-72-6	4.0×10^{-0}	
3,3',4,4'5,5'- Hexachlorobiphenyl (169)[b]	32774-16-6	4.0×10^{-3}	
2,3,3'4,4',5,5'- Heptachlorobiphenyl (189)[b]	39635-31-9	4.0×10^{-1}	
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
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; Cardiovascular System; Nervous System
Styrene	100-42-5	$9.0 \times 10^{+2}$	Nervous System
Sulfuric Acid	7664-93-9	$1.0 \times 10^{+0}$	Respiratory System
Tetrachloroethylene [a] (Perchloroethylene)	127-18-4	$3.5 \times 10^{+1}$	Alimentary System; Kidney

Table 6.2 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)
Toluene	108-88-3	$3.0 \times 10^{+2}$	Developmental; 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

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*.).

N/A Not Applicable

b The OEHHA has adopted the World Health Organization 1997 Toxicity Equivalency Factor (WHO_{97}-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). 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 REL values for speciated PCDDs, PCDFs and PCBs based on the WHO_{97} TEF values and the chronic REL for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin 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.

6.4 Description of 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 expressed as doses in milligrams of substance (consumed and dermally absorbed) per kilogram body weight per day (mg/kg-day).

Table 6.3 lists the chronic noncancer RELs to be used in the assessment of chronic health effects from noninhalation pathways of exposure. 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 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. 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 Noninhalation 'Oral' Reference Exposure Levels (RELs) And Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Oral REL (mg/kg-day)	Chronic Oral Hazard Index Target Organ System(s)
Arsenic & Inorganic Arsenic Compounds	7440-38-2	3.0×10^{-4}	Cardiovascular System; Skin
Beryllium and Beryllium Compounds	7440-41-7	2.0×10^{-3}	Alimentary System
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; Developmental; Endocrine System; Hematopoietic 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	1.0×10^{-4}	
Chlorinated Dibenzofurans [a]			
2,3,7,8-Tetrachlorodibenzofuran [a]	5120-73-19	1.0×10^{-7}	Alimentary System; Developmental; Endocrine System; Hematopoietic System; Reproductive System; Respiratory System
1,2,3,7,8-Pentachlorodibenzofuran [a]	57117-41-6	5.0×10^{-7}	
2,3,4,7,8-Pentachlorodibenzofuran [a]	57117-31-4	5.0×10^{-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	1.0×10^{-4}	
Chromium VI & Soluble Chromium VI Compounds (except chromic trioxide)	18540-29-9	2.0×10^{-2}	Hematologic
Fluorides (including hydrogen fluoride)		4.0×10^{-2}	Bones and Teeth
Mercury & Mercury Compounds (inorganic)	7439-97-6	3.0×10^{-4}	Immune System; Kidney
Nickel & Nickel Compounds (except nickel oxide)	7440-02-0	5.0×10^{-2}	Alimentary System
Nickel Oxide	1313-99-1	5.0×10^{-2}	Alimentary System
Polychlorinated biphenyls[P4] (PCBs) (speciated)[b]			Alimentary System; Developmental; Endocrine System; Hematopoietic System; Reproductive System; Respiratory System
3,3',4,4'-Tetrachlorobiphenyl (77)[b]	35298-13-3	1.0×10^{-4}	
3,4,4',5-Tetrachlorobiphenyl (81)[b]	70362-50-4	1.0×10^{-4}	
2,3,3',4,4'- Pentachlorobiphenyl (105)[b]	32598-14-4	1.0×10^{-4}	

Table 6.3 Chronic Noninhalation 'Oral' Reference Exposure Levels (RELs) And Chronic Hazard Index Target Organ System(s)

Substance	Chemical Abstract Service Number (CAS)	Chronic Oral REL (mg/kg-day)	Chronic Oral Hazard Index Target Organ System(s)
2,3,4,4'5- Pentachlorobiphenyl (114)[b]	74472-37-0	2.0×10^{-5}	Alimentary System; Developmental; Endocrine System; Hematopoietic System; Reproductive System; Respiratory System
2,3'4,4',5- Pentachlorobiphenyl (118)[b]	31508-00-6	1.0×10^{-4}	
2',3,4,4',5- Pentachlorobiphenyl (123)[b]	65510-44-3	1.0×10^{-4}	
3,3',4,4',5- Pentachlorobiphenyl (126)[b]	57465-28-8	1.0×10^{-7}	
2,3,3',4,4',5-Hexachlorobiphenyl (156)[b]	38380-08-4	2.0×10^{-5}	
2,3,3',4,4',5'-Hexachlorobiphenyl (157)[b]	69782-90-7	2.0×10^{-5}	
2,3',4,4',5,5'-Hexachlorobiphenyl (167)[b]	52663-72-6	1.0×10^{-3}	
3,3',4,4'5,5'- Hexachlorobiphenyl (169)[b]	32774-16-6	1.0×10^{-6}	
2,3,3'4,4',5,5'- Heptachlorobiphenyl (189)[b]	39635-31-9	1.0×10^{-4}	

a The OEHHA has adopted the World Health Organization 1997 Toxicity Equivalency Factor (WHO_{97}-TEF) scheme for evaluating the cancer 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). For convenience, OEHHA has calculated chronic REL values for speciated PCDDs, PCDFs and PCBs based on the WHO97 TEF values and the chronic REL for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin using the procedure discussed in Appendix E. See Appendix E for more information about the scheme and for the methodology for calculating 2,3,7,8-equivalents for PCDD, PCDFs and PCBs. The oral chronic RELs for these compounds may be used if the mixtures are speciated to calculate a hazard index from individual congener doses.

7. Dose-Response Assessment for Carcinogens

7.1 Introduction

Dose-response assessment describes 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 determined by the Office of Environmental Health Hazard Assessment (OEHHA) or by the United States Environmental Protection Agency (U.S. EPA) and endorsed by OEHHA. 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 are provided in the 1985 California Department of Health Services publication *Guidelines for Chemical Carcinogen Risk Assessments and their Scientific Rationale* (CDHS, 1985). Substance-by-substance information is presented in OEHHA's document entitled, *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II; Technical Support Document for Describing Available Cancer Potency Factors (OEHHA 1999b)* (Part II TSD).

7.2 Definition of Carcinogenic Potency

Cancer potency factors are expressed as the upper bound probability of developing cancer assuming continuous lifetime exposure to a substance at a dose of one milligram per kilogram of body weight, and are expressed in units of inverse dose as a potency slope [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. It is assumed in cancer risk assessments that risk is directly proportional to dose and that there is no threshold for carcinogenesis. The derivation of carcinogenic inhalation and oral cancer potency factors takes into account the available information on pharmacokinetics and on the mechanism of carcinogenic action. These values are generally the 95% upper confidence limits (UCL) on the dose-response slope. 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 Description of the Inhalation Cancer Potency Factor

Under the new risk assessment methodology and algorithms presented in Chapters 5 and 8, inhalation cancer slope factors must be expressed in units of inverse dose (i.e., $(mg/kg/day)^{-1}$). Unit

risk factors, in the units of inverse concentration as micrograms per cubic meter (i.e., $(\mu g/m^3)^{-1}$), which have been used in previous guidelines for the Hot Spots program, can also be used for assessing cancer inhalation risk directly from air concentrations. However, breathing rates, expressed in units of liters per kilogram of body weight-day (L/kg*BW-day or L/kg-day), can be coupled with the air concentrations to estimate dose in mg/kg-day. This allows estimation of average, high-end, and distributions of cancer risk. Therefore for the Hot Spots Program, inhalation cancer potency factors are now recommended for determining cancer risk instead of unit risk factors. Unit risk factors are still listed in the Part II TSD and may prove useful in other risk assessment applications.

Multiplication of the average daily inhalation dose over 70 years (mg/kg-day) with the cancer potency factor $(mg/kg\text{-}day)^{-1}$ will give inhalation cancer risk (unitless). A more complete description of how 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 potential (inhalation) 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 and adopted by the Director of OEHHA. At that time, these guidelines will be updated to reflect those changes. However, in the interim between the adoption of new or updated numbers and a guideline update, consult the OEHHA web site at *www.oehha.ca.gov* (look under "Air", then select "Hot Spots Guidelines") to determine if any new or updated cancer potency factors have been adopted since the last guideline update. If so, these too should be used in the HRA.

7.2.2 Description of the Oral Cancer Potency Factor

Under the Hot Spots Program, a few substances are considered multipathway substances. Multipathway substances have the potential to impact a receptor through inhalation and noninhalation (oral) 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 substances that must be evaluated for multipathway exposure.

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 oral potential cancer risk (unitless). 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.

Four carcinogens (cadmium, hexavalent chromium, 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, J, 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	1.9×10^{-4} [#]	
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	$1.3 \times 10^{+1}$	$1.3 \times 10^{+1}$
Chlorinated Dibenzofurans [A]			
2,3,7,8-Tetrachlorodibenzofuran	5120-73-19	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$

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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 $(mg/kg\text{-}day)^{-1}$
1,2,3,7,8-Pentachlorodibenzofuran	57117-41-6	$6.5 \times 10^{+3}$	$6.5 \times 10^{+3}$
2,3,4,7,8-Pentachlorodibenzofuran	57117-31-4	$6.5 \times 10^{+4}$	$6.5 \times 10^{+4}$
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	$1.3 \times 10^{+4}$	$1.3 \times 10^{+4}$
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	$1.3 \times 10^{+1}$	$1.3 \times 10^{+1}$
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	$5.1 \times 10^{+2}$	
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*,*I*]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	$2.5 \times 10^{+2}$	$2.5 \times 10^{+2}$

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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 $(mg/kg\text{-}day)^{-1}$
1,6-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}	
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}	
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	$1.7 \times 10^{+1}$	
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	$1.1 \times 10^{+0}$	
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}	
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	3.7×10^{0}	
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	$6.7 \times 10^{+0}$	

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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 $(mg/kg\text{-}day)^{-1}$
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}	
Polychlorinated biphenyls (PCBs) (unspeciated mixture)	1336-36-3		
(high risk) [P1]		$2.0 \times 10^{+0}$	$2.0 \times 10^{+0}$
(mediumlow risk) [P2]		4.0×10^{-1}	4.0×10^{-1}
(lowest risk) [P3]		7.0×10^{-2}	7.0×10^{-2}
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	$1.3 \times 10^{+1}$	$1.3 \times 10^{+1}$
2,3,3',4,4'- Pentachlorobiphenyl (105)	32598-14-4	$1.3 \times 10^{+1}$	$1.3 \times 10^{+1}$
2,3,4,4'5- Pentachlorobiphenyl (114)	74472-37-0	$6.5 \times 10^{+1}$	$6.5 \times 10^{+1}$
2,3'4,4',5- Pentachlorobiphenyl (118)	31508-00-6	$1.3 \times 10^{+1}$	$1.3 \times 10^{+1}$
2',3,4,4',5- Pentachlorobiphenyl (123)	65510-44-3	$1.3 \times 10^{+1}$	$1.3 \times 10^{+1}$
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	$6.5 \times 10^{+1}$	$6.5 \times 10^{+1}$
2,3,3',4,4',5'-Hexachlorobiphenyl (157)	69782-90-7	$6.5 \times 10^{+1}$	$6.5 \times 10^{+1}$
2,3',4,4',5,5'-Hexachlorobiphenyl (167)	52663-72-6	$1.3 \times 10^{+0}$	$1.3 \times 10^{+0}$
3,3',4,4'5,5'- Hexachlorobiphenyl (169)	32774-16-6	$1.3 \times 10^{+3}$	$1.3 \times 10^{+3}$
2,3,3'4,4',5,5'- Heptachlorobiphenyl (189)	39635-31-9	$1.3 \times 10^{+1}$	$1.3 \times 10^{+1}$
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}	
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}	
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}	

#	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).
A	Polychlorinated Dibenzo-*p*-dioxins and Polychlorinated Dibenzofurans: The World Health Organization 1997 (WHO-97) Toxicity Equivalency Factors are used for polychlorinated dibenzo-*p*-dioxins, polychlorinated dibenzofurans and polychlorinated biphenyls. (see Appendix E). For convenience, OEHHA has calculated cancer potency factors for speciated poly chlorinated biphenyls 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 Polychlorinated Biphenyls (PCBs).
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. 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.

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 (e.g., monitored or modeled concentrations, inhalation or oral doses, and exposure pathway information) is combined with cancer potency factors and Reference Exposure Levels (RELs) to quantify the cancer risk and noncancer health impacts, respectively. Under the Air Toxics Hot Spots (Hot Spots) Act, comprehensive risk assessments should quantify 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. Note that, for the Hot Spots Program, the 70-year exposure duration should continue to be used as the basis for estimating risk.

This chapter provides guidance on how to evaluate the risk characterization components required by the Hot Spots Program. A general summary of the HRA components includes the following items or information. This information should be clearly presented in cross-referenced text, tables, figures, and/or maps.

- The location and potential acute noncancer, and multipathway (inhalation and noninhalation) cancer and noncancer chronic health impacts at the point of maximum impact (PMI), at the maximum exposed individual resident (MEIR), at the maximum exposed individual worker (MEIW), and at specified (contact District or reviewing authority) sensitive receptors (e.g., schools, hospitals, daycare, or eldercare facilities).

- Estimates of population exposure for potential cancer risk and noncancer acute and chronic health impacts.

To perform the HRA and create the information listed above, OEHHA recommends using a tiered approach to risk assessment. The tiered approach 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 assumptions or the stochastic treatment of exposure factor distributions. Tier-1 evaluations are required for all HRAs prepared for the Hot Spots Program. Persons preparing an HRA using Tier-2 through Tier-4 evaluations must also include the results of a Tier-1 evaluation in the HRA. The four-tiered approach to risk assessment is intended to primarily apply to residential cancer risk assessment, both for inhalation and noninhalation pathways. OEHHA is not recommending a stochastic approach (Tier-3) for worker exposure, or noncancer inhalation chronic evaluations. A Tier-2 evaluation could be used for off-site worker risk assessments.

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There is only a Tier-1 option for determining acute noncancer risks since calculating the hazard quotient only involves the acute REL and short-term maximum ground level air concentrations. There is only a Tier-1 option for evaluating inhalation noncancer chronic risks since calculating the chronic hazard quotient only involves the chronic Reference Exposure Level and the annual average concentration (not exposure parameter distributions). Chronic noninhalation noncancer risks involve a calculation of dose from oral pathways. It is possible that site-specific intake variates (e.g., fish consumption) could be appropriate for a particular site and therefore a Tier-2 analysis could be useful. See the *Air Toxics Hot Spots Program Risk Assessment Guidelines; Part IV; Technical Support Document for Exposure Assessment and Stochastic Analysis (OEHHA, 2000b)* (Part IV TSD) for a detailed discussion of the tiered approach. Table 8.1 summarizes OEHHA's recommendations for the four Tiers.

Table 8.1 Tiers for Cancer and Noncancer Hot Spots Risk Assessments

Tier	Cancer		Chronic Non Cancer		Acute
	Inhalation	Noninhalation	Inhalation	Noninhalation	Inhalation
Tier-1	X	X	X	X	X
Tier-2	X	X		X	
Tier-3	X	X			
Tier-4	X	X			

Cancer risk assessment as currently practiced involves estimating exposure to carcinogenic chemicals and multiplying the dose times the cancer potency factor. There are often questions regarding the validity of applying the cancer potency factors to less than lifetime exposures. The cancer potency or unit risk factors are estimated from long-term animal studies approaching lifetime, or from worker epidemiological studies involving long term exposure usually over decades.

8.2 Risk Characterization for Cancer Health Effects

8.2.1 Calculating Inhalation Cancer Risk

A 70-year inhalation cancer risk evaluation is required for all carcinogenic risk assessments (see Sections 8.2.2 and 8.2.3 for exposure duration information). There are two pieces of information needed to assess inhalation cancer risk. These are the inhalation cancer potency for the substance, expressed in units of inverse dose as a potency slope (i.e., $(mg/kg/day)^{-1}$) from Table 7.1, and an estimate of average daily inhalation dose in units of milligram per kilogram-day (mg/kg-day) (see Chapters 4 and 5). Cancer risk is calculated by multiplying the inhalation dose by the inhalation cancer potency factor to yield the potential inhalation excess cancer risk. The following equation illustrates the formula for calculating cancer risk. See Appendix I for an example calculation.

(Inhalation Dose (mg/kg-day)) x (Cancer Potency $(mg/kg-day)^{-1}$) = Cancer Risk

To convert this to chances per million of developing cancer, multiply the potential cancer risk by 10^6. This result is useful as a risk communication tool.

Tier-1 is a standard point-estimate approach that uses the recommended exposure pathway (e.g., breathing rate) point-estimates presented in this document. A Tier-1 evaluation must use the high-end point-estimate for the inhalation pathway to present the inhalation cancer risk. For the Hot Spots Program, the 70-year exposure duration should be used as the basis for public notification and risk reduction audits and plans. Sections 8.2.2 and 8.2.3 describe the use of exposure duration adjustment factors for residential and worker receptors. As supplemental information, the assessor may wish to evaluate the cancer risk by using the average point-estimate to provide a range of cancer risk to the risk manager. The assessor may also decide to further supplement the HRA by performing a Tier-3 evaluation using the daily breathing rate data distribution in a stochastic analysis. See Chapter 5 for the algorithms and exposure information used for all exposure pathways for Tier-1 and Tier-3 evaluations. The HARP software will perform all of these analyses. Specifically, the required high-end, 70-year inhalation cancer risk evaluation can be performed in HARP by selecting either the high-end point-estimate/cancer risk analysis or by selecting the derived/70-year cancer risk analysis.

The risk assessment guidelines require the use of the 95th percentile (i.e., high end) breathing rate for all assessments of cancer risk by the inhalation route in Tier-1 risk assessments in order to avoid underestimating risk to the public, including children. In general, the risk management of facilities in the Air Toxics Hot Spots program is based on the 70-year risk at the highest exposed receptor point using high-end estimates of breathing rate. Some facilities subject to the Air Toxics Hot Spots Act (e.g., some in the industry-wide categories) 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 all possible exposure scenarios. Alternative breathing rates (point estimates or distributions) may be used as part of Tier-2 or Tier-4 risk assessments. Thus, the risk manager should take this into account during any risk management decisions. OEHHA is willing to work with risk managers at ARB and the Districts on this issue. Further examination of the issue is warranted.

8.2.2 Calculating Cancer Risk Using Different Exposure Durations

A. Residential

OEHHA recommends the 70-year exposure duration (ED) be used for determining residential cancer risks. For the Hot Spots Program, the 70-year exposure duration should be used as the basis for public notification and risk reduction audits and plans. This will ensure that a person residing in the vicinity of a facility for a lifetime will be included in the evaluation of risk posed by that facility. Exposure durations of 9-years and 30-years may also be evaluated as supplemental information to show the range of cancer risk based on residency periods. Lifetime or 70-year exposure is the historical benchmark for comparing facility impacts on receptors and for evaluating the effectiveness of air pollution control measures. Although it is not likely that most people will reside at a single residence for 70 years, it is

common that people will spend their entire lives in a major urban area. While residing in urban areas it is very possible to be exposed to the emissions of another facility at the next residence. In order to help ensure that people do not accumulate an excess unacceptable cancer risk from cumulative exposure to stationary facilities at multiple residences, OEHHA recommends the 70-year exposure duration for risk management decisions. However, if a facility is notifying the public regarding cancer risk, it is useful information for a person who has resided in his current residence for less than 70 years to know that the calculated estimate of his or her cancer risk is less than that calculated for a 70-year risk.

Cancer risk assessment as currently practiced involves estimating exposure to carcinogenic chemicals and multiplying the dose times the cancer potency factor. There are often questions regarding the validity of applying the cancer potency factors to less than lifetime exposures. The cancer potency or unit risk factors are estimated from long-term animal studies approaching lifetime, or from worker epidemiological studies involving long term exposure usually over decades.

OEHHA has presented in this document exposure variates for estimating 9, 30 and 70-year exposures. These 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 typical lifetime (70 years). We support the use of cancer potency factors for estimating cancer risk for these exposure durations. However, as the exposure duration decreases the uncertainties introduced by applying cancer potency factors derived from very long term studies increases. Short-term high exposures are not necessarily equivalent to longer-term lower exposures even when the total dose is the same. OEHHA therefore does not support the use of current cancer potency factor to evaluate cancer risk for exposures of less than 9 years. If such risk must be evaluated, we recommend assuming that average daily dose for short-term exposure is assumed to last for a minimum of 9 years. OEHHA is evaluating cancer risk assessment methodologies over the next several years to address a number of issues including methods to evaluate short-term exposures to carcinogens.
If children younger than age 9 can be exposed to the emissions of a short term project, then the point estimates for a child should be used for an exposure period of 9 years to calculate a child's potential cancer risk. OEHHA is evaluating cancer risk assessment methodologies over the next several years to address a number of issues including methods to evaluate short-term exposures to carcinogens.

As presented in Chapter 5 and explained in the Part IV TSD, the 9-year (child) exposure duration is intended to represent the first 9-years of life. Children, for physiological as well as behavioral reasons, have higher rates of exposure (mg/kg-day) than adults. Therefore, the daily point-estimate (e.g., inhalation rate, soil ingestion rates) for the 9-year exposure duration is higher than for the 30 and 70-year (adult) exposure durations. When assessing the impacts specifically for children, the 9-year point-estimates and exposure factor distributions should be used. If a 9-year adult exposure duration is desired, then the 30 and 70-year point-estimates could be used and the cancer risk is adjusted using a factor of 9/70.

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The 30 and 70-year exposure durations are intended to represent the first 30 and first 70 years of life, respectively. However, in the interest of simplicity, the 30-year exposure duration scenario uses the same exposure point-estimates and data distributions as the 70-year exposure duration scenario. This assumption to use the 70-year exposure point-estimate for both 30 and 70-year exposures probably results in a small underestimation of dose for the 30-year exposure scenario, since the exposure parameters for earlier years are higher than years spent as an adult.

The mother's milk pathway is unlike other pathways because the (entire) dose to the breastfed infant is received in the first year of life. In evaluating risk from the pathway for 9, 30 and 70 years, it is assumed that the cancer risk from the one-year exposure to contaminants in mother's milk is equally spread over 70 years to obtain a lifetime risk. If an assessor wants to calculate the multipathway risk for a 9-year exposure duration, then the cancer risk for this exposure pathway is adjusted using a $9/70^{th}$ factor.

B. Worker

The general approach for estimating the potential health impacts to an offsite worker (e.g., MEIW) includes estimating the concentration at the receptor and identifying the duration of that exposure. The best way to determine potential impacts for a worker is to use the algorithms and exposure information in Chapter 5 and the HARP software.

There are three factors that affect worker exposure for cancer risk determination. The first is the offsite worker's schedule. For example, some workers such as teachers have three months off during the summer and some workers work throughout the year except for weekends, holidays and vacation. The second factor is the operating schedule of the emitting facility under consideration. This is important because the ISCST-3 air dispersion computer model, or other models typically calculate an annual average air concentration based on actual operating conditions. For example, the facility may operate 365 days a year, 24 hours a day or may operate eight hours a day, five days a week. The third factor is the coincidence of the offsite worker's schedule with the time that the facility is emitting. For example, if the facility emits during the day, five days a week, and the offsite worker is working only at night, then no inhalation exposure would occur.

If an adjustment needs to be made for the time that the worker is present (coincident with the emissions), then the standard default assumption is the worker is present for 5 days per week, 49 weeks per year, for 40 years. The 40-year working lifetime is the same assumption used under the Proposition 65 Regulation. The worker is assumed to breath 149 L/kg BW* day for an 8-hour workday. Other adjustments may be appropriate, such as for teachers or other workers. If the offsite worker only works part time, for example 4 hours per day, a factor of 0.5 (4/8) may be used to adjust the daily inhalation exposure proportionally.

If the annual average concentration of pollutants from the emitting facility (determined by the air model) is different than the air concentration that the worker breathes when present at the site, then the

annual average concentration for the worker inhalation pathway will need to be adjusted. For example, if the offsite worker and emitting facility are on concurrent schedules (i.e., the worker has a standard working schedule of eight hours per day, 5 days a week, and the facility emits 5 days a week, 8 hours per day), then the annual average air concentrations for the worker inhalation pathway would need be approximated by adjusting it upward using a factor of 4.2 (7/5 x 24/8). The annual average determined by the air modeling program is a 24 hour per day , 7 days per week, 365 days per year regardless of the actual operating schedule of the facility. The adjustment simply reflects the air concentration that the worker breathes. If the worker is only present some of the time that the facility is operating, then the average concentration that the worker breathes over his or her working day may be used.

For the chemicals where noninhalation pathways (e.g., soil ingestion and dermal exposure) need to be evaluated for workers, the annual average concentration should not be adjusted to account for the operating schedule of the emitting facility or the worker schedule (even if the facility emits only 5 days per week 8 hours per day while the offsite worker is present). 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.

If the calculation for determining a MEIW inhalation risk are not able to be performed using the original algorithms or the HARP software, then the adjustment factors in Table 8.2 may be of use for inhalation assessments only. The algorithms and assumptions in Chapter 5 must be used to determine multipathway impacts to a worker receptor.

Table 8.2: Adjustment Factors to Convert Inhalation Based Cancer Risk Estimates for a Residential Receptor to a Worker Receptor

Worker Receptor Type (Hrs/Days/Weeks/Years)	Facility Operating Schedule (Hrs/Days/Weeks/Years)	Adjustment Factor	
		(High End)*	(Average)*
Worker (8/5/49/40)	Continuous (24/7/52/70)	0.1516	0.2199
Worker (8/5/49/40)	Standard (8/5/52/70)	0.6366	0.9234
Teacher (8/5/36 T/40)	Continuous (24/7/52/70)	0.1114	0.1616
Teacher (8/5/36 T/40)	Standard (8/5/52/70)	0.4679	0.6787

* High End adjustment factors convert the residential receptor risk based on the high-end breathing rate point-estimate to a worker receptor risk. Average adjustment factors convert the residential receptor risk based on the average breathing rate point-estimate to a worker receptor risk.

T Number of weeks is based on school days per year reported by school district representatives.

C. Uses of Exposure Duration Adjustments for On-site Receptors

On-site workers are protected by CAL OSHA and do not have to be evaluated under the Hot Spots program, unless the worker also lives on the facility site, or property. Occasionally, facilities like

prisons, military bases, and universities have worker housing within the facility. In these situations the evaluation of on-site cancer risks, and/or acute and chronic noncancer hazard indices is appropriate under the Hot Spots program.

If the receptor lives and works on the facility, site, or property, then they should be evaluated under both scenarios and the one that is most health protective should be used.

The cancer risk estimates for the onsite residents may be done using the 70-year exposure variates and 40-year exposure duration. The use of the 70 year exposure variates will overestimate exposure to adult workers to a small extent because higher inhalation rates, etc., during the portion of a 70 year lifetime that a person is a child are incorporated. If the on-site resident under evaluation can be exposed through an impacted exposure pathway (other than inhalation), then that exposure pathway must be included. 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). No exposure adjustments apply to acute exposure analyses. The District may be consulted on the appropriate evaluations for the risk assessment.

8.2.3 Speciation for Specific Classes of Compounds: Polycyclic Aromatic Hydrocarbons (PAHs),Polychlorinated Dibenzo-p-dioxins (PCDDs) and Dibenzofurans (PCDFs), and Polychlorinated Biphenyls (PCBs)

Health values and potency equivalency factors (PEFs) have been developed for approximately 26 PAHs (see Appendix G). When speciation of PAHs has been performed on facility emissions, these health values and PEFs should be used. In those cases where speciation of PAHs has not been performed, then benzo(a)pyrene or B(a)P serves as the surrogate carcinogen for all PAH emissions. A similar method has been developed for PCDDs and PCDFs, and PCBs known as toxicity equivalency factors (WHO TEFs), based on the number of chlorines and their position on the molecule (see Appendix E). Where speciation of PCDDs and PCDFs, and PCBs has been performed on facility emissions, the WHO TEFs should be used. In those cases where speciation of PCDDs and PCDFs has not been performed, then 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) serves as the surrogate for PCDD and PCDF emissions. Similarly, where only total PCBs are available, then the cancer potency factor for PCBs should be applied.

When using the HARP software, the emission contribution of speciated PAHs and PCDDs/PCDFs that have health values can be entered into the software. Unknown contributions of the PAH or PCDD/PCDF mixtures, or PAHs without a health value, should be assigned the appropriate surrogate. If a surrogate substance is used in the report, the facility-emitted substance (PAH mixture or PCDDs/PCDF mixture) must also be clearly indicated in the risk assessment as the actual substance emitted.

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Since the surrogates for total PAH (B(a)P) and total PCDD/PCDF (2,3,7,8-tetrachlorodibenzo-*p*-dioxin) are the most or nearly-the-most potent carcinogens in the class, use of the cancer potency factors for these with total emissions will overestimate the risk.

Given that speciation data on these classes of compounds can result in significant capital investment, it may be reasonable to run a screening estimate of risk on the unknown mixture using the appropriate surrogate compound to represent the class. If the resulting risk estimate is deemed significant enough to trigger health concerns, it would then be advisable to speciate the mixture and run a screening estimate using the speciated data.

8.2.4 Determination of Noninhalation (Oral) Cancer Risk

A small subset of Hot Spots substances is subject to deposition onto the 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.

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, or PCBs are emitted, then the breast-milk consumption pathway becomes mandatory. The other exposure pathways (e.g., ingestion of homegrown produce or fish) are only evaluated 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, then the fish pathway will not be considered for that facility or receptor. Table 5.1 lists the multipathway substances and the pathways that can be considered for each substance. Table 8.3 identifies the residential receptor exposure pathways that are mandatory and those that are dependent on the available routes of exposure. Table 8.3 also identifies the three exposure pathways that are appropriate for a worker receptor.

Table 8.3 Mandatory and Site/Route Dependant Exposure Pathways

Mandatory Exposure Pathways	Site/Route Dependent Exposure Pathways
• Inhalation[w] • Soil Ingestion[w] • Dermal Exposure[w] • Breast-Milk or Mother's Milk Consumption*	• Homegrown Produce Ingestion • Fish Ingestion • Drinking Water Ingestion • Dairy (Cow's) Milk Ingestion • Meat (Beef, Pork, Chicken, and Egg) Ingestion

(*) If dioxins, furans, or PCBs are emitted, then the breast-milk consumption pathway becomes mandatory.
(w) Identifies the only appropriate exposure pathways that should be evaluated for a worker. These pathways are

inhalation, dermal exposure, and the soil ingestion pathways.

The oral cancer risk is calculated using the same steps as inhalation cancer risk described in Section 8.2.1. The only difference is that the inhalation dose is replaced by a noninhalation pathway dose (e.g., soil ingestion) and consideration is given to determining the dominant exposure pathways for the proper use of point-estimates (see Section 8.2.5).

In summary, an oral 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 as a potency slope (i.e., $(mg/kg/day)^{-1}$) from Table 7.1 or Appendix L, to yield the soil ingestion cancer risk. The following equation illustrates the formula for calculating cancer risk. Details (data, algorithms, and guidance) for each exposure pathway are presented in Chapter 5 and the Part IV TSD. See the discussion of Tier-1 in Section 8.2.6 or the Part IV TSD for the method used to determine the multipathway cancer risk. See Appendix I for an example calculation for the inhalation exposure pathway.

$$\left(\text{Oral Dose}\ \frac{mg}{kg-day}\right)\left(\text{Oral Slope Factor}\ \frac{kg-day}{mg}\right) = \quad \text{Potential Cancer Risk}$$

To convert this to chances per million of developing cancer, multiply the cancer risk by 10^6. This result is useful as a risk communication tool.

$$\text{Cancer risk x } 10^6 = \text{ chances per million}$$

8.2.5 Evaluation of Multipathway (Inhalation and Noninhalation) Cancer Risk

A. Deposition Rate

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.

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B. Use of Air Dispersion Modeling Results for Pastures and Water Bodies in Risk Assessment and the HARP Software

The substance or pollutant deposition to a drinking or pasture water body source and pastureland will be evaluated if an HRA includes the drinking water, fish ingestion, and cow's milk or meat (beef) exposure pathways. Two approaches are recommended for determining the deposition impacts to water bodies and pastureland. A simple approach is to select the results from a single receptor point on the grid laid over the area covered by the water body or pasture and assume that the modeled concentration at that grid-point is uniform across the water or pasture area. To make this first approach health protective, the grid-point within the area of the water body or pastureland with the highest modeled concentration should be used. A more refined approach is to average the air dispersion modeling results for all of the grid-points covering the area of the pasture or water body.

C. Summary of the Tiered Approach to Risk Assessment

The tiered approach for risk assessment that is presented in detail in the Part IV TSD and summarized here should be reviewed prior to estimating multipathway cancer risk. The tiered approach to risk assessment and the evaluation described here are included in the HARP software. 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*.

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. If an HRA cancer risk assessment involves multipathway residential exposures, then the risk assessor needs to first calculate the cancer risk from each pathway using the high-end exposure variates for all pathways. Then a second calculation is performed in which the pathways with the two highest cancer risks are added to the cancer risks from the rest of the pathways (if any) calculated with the average exposure variates. Dominant pathways are defined as the two exposure pathways that contribute the most to the total cancer risk estimate when using high-end point-estimates for all the exposure pathways under consideration. The final cancer risk calculation using a combination of high end and average exposure variates is referred to as derived risk in the HARP software and applies only to the residential receptor. There are only single values for exposure variates for the worker for the three pathways considered.

A similar procedure is used to determine the hazard index for the noncancer noninhalation pathways. The doses from all pathways (noninhalation) are calculated using the high-end exposure variate. The dose is used to calculate the hazard quotient for all noninhalation pathways. The hazard quotient for the inhalation pathway is calculated from the ground level concentration and the chronic REL. The three pathways with the highest hazard quotient are the dominant pathways. The remaining noninhalation pathways (if any) hazard quotients may be recalculated using the average exposure variates. The total hazard quotient for the chemical may be calculated by adding the

individual hazard quotients from the dominant pathways and those calculated with the average exposure variates.

Using the derived estimate of dose and risk will lessen the issue of compounding high-end exposure estimates, while retaining a health-protective approach for the more important exposure pathway(s). It is unlikely that an individual receptor would be on the high-end of exposure for all the intake variates (exposure pathways). Usually, inhalation is the dominant pathway posing the most cancer risk and noncancer chronic health impacts in the HRAs prepared for the Hot Spots Program. Occasionally, risks from other exposure pathways may also be dominant for lipophilic (fat-loving) compounds or metals. Therefore, for many facilities emitting volatile and multipathway chemicals, the inhalation pathway will be at least one of the two exposure pathways for which cancer risks are assessed using a high-end estimate (see Section 8.2.1).

The relatively health-protective assumptions incorporated into the Tier-1 risk assessment (e.g., 70-year exposure duration (for cancer) and the high-end values for key variates in the driving pathways) make it unlikely that the risks are underestimated for the general population. If the results indicate that a facility's estimated cancer risk and noncancer hazard are below the level of regulatory concern, further analysis may not be warranted. If the results are above a regulatory level of concern, the risk assessor may want to proceed with further analysis as described in Tier-2, or use a more resource-intensive stochastic modeling effort described in Tier-3 and Tier-4. 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 site-specific information to develop point-estimates that are more appropriate to use in the site-specific HRA than the recommended point-estimates. In Tier-3, a stochastic approach to exposure assessment is taken using the exposure factor distributions presented in the Part IV TSD and in Chapter 5. The Part IV TSD exposure factor distributions apply only to a residential receptor and are used only for the determination of cancer risk. Tier-4 is also a stochastic approach but allows for utilization of site-specific distributions if they are justifiable and more appropriate for the site under evaluation than those recommended in this document.

Tier-3 and Tier-4 analyses show a distribution of cancer risk indicating the percent of the population exposed to various levels of risk. This type of analysis provides an illustration of population risk. The results from this type of analysis can also be used to show what percentage of the population would be protected with various risk management options.

OEHHA is not recommending a stochastic approach (Tier-3) for worker exposure, or noncancer inhalation chronic evaluations. A Tier-2 evaluation could be used for off-site worker risk assessments. There is only a Tier-1 option for determining acute noncancer risks since calculating the hazard quotient only involves the acute REL and short-term maximum ground level air concentrations. In addition, no exposure duration adjustment should be made for noncancer assessments.

D. Multipathway Cancer Risk Methodology

In order to characterize total substance risk for a single multipathway substance the inhalation risk is calculated by multiplying the inhalation dose (mg/kg-day) times the inhalation cancer potency factor to give the inhalation cancer risk (Section 8.2.1). Using Tier-1, the dermal and oral dose from each relevant exposure pathway is multiplied times the substance-specific oral potency factor to give the oral (noninhalation) cancer risk (see Sections 8.2.4 and 8.2.5). The inhalation cancer risk and oral cancer risk are then summed to give the multipathway cancer risk for that substance. Many facilities will emit multiple carcinogenic substances. If multiple substances are emitted, the cancer risk from each of the individual substances (including multipathway and volatile, inhalation-only substances) is summed to give the (total) multipathway cancer risk for the entire facility at the receptor location.

Cancer risks from different substances are treated additively in the Hot Spots Program in part because many carcinogens act through the common mechanism of DNA damage. However, this assumption fails to take into account the limited information on substance interactions. However, the overall uncertainty in the cancer potency factors and the variability in the human population is probably far greater than the uncertainty from the assumption of additivity. In addition, cancers are life threatening serious diseases so it is not unreasonable to consider total additive risk. Therefore, 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 substance dependent and can be dose dependent. All three types of interactions have been demonstrated scientifically.

8.2.6 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 results of the stochastic risk assessment is a distribution of risk which accounts for some of the variability in cancer risk that results from natural variability in exposure, such as breathing rates or water intake. The cancer risk distribution for inhalation cancer risk, for example, is generated by multiplying random values from the breathing rate distribution times 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. Distributions are only available for some of the exposure variates and none are currently recommended for the fate and transport algorithms. As more data become available for exposure variates and fate and transport variates, OEHHA will expand the number of distributions in our model to better capture the variability in exposure and risk.

The HARP software will perform an HRA using either OEHHA or user-provided data distributions using a Monte Carlo analysis and include the statistics on the distributions. The 70-year

exposure duration should be used as the basis for public notification and risk reduction audits and plans. If an assessor would prefer to evaluate 9 or 30-year exposure durations, then a cancer risk distribution for 9 or 30-year exposure duration would be presented in addition to the 70-year exposure duration. An adult's analysis would use the 30 and 70-year data distributions. If a stochastic analysis is performed for a child, then the child's (9-year) distribution must be used. A stochastic approach for acute and chronic health impacts and worker (MEIW) exposures are not currently recommended. 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*.

8.3 Risk Characterization for Noncarcinogens

Noncancer impacts are determined for acute (inhalation) exposure and for both inhalation and oral chronic exposure. Estimates of health impacts for noncancer endpoints are expressed as a hazard quotient (for individual substances) or a hazard index (for multiple substances). In addition, all hazard quotients (HQ) and hazard indices (HI) must be determined by target organ system. An HQ of one 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 hazard index above 1 is not necessarily indicative of health impacts due to the application of uncertainty factors in deriving the Reference Exposure Levels. There are limitations to this method of assessing cumulative noncancer chronic 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.

There is only one approach to calculating the acute HI because the calculation is based on the highest short-term ground level air concentrations and the acute Reference Exposure Level. Likewise the chronic inhalation HI calculation is performed using the annual average ground level concentration and the chronic REL. Therefore no Tier-2, Tier-3 or Tier-4 options are available for acute or chronic noncancer inhalation hazard evaluation. However, there may be cases in which site specific fate and transport variates or exposure variates may be more appropriate to determine dose (mg/kg-day) for the noninhalation chronic HI; therefore, in some cases a Tier-2 evaluation may be appropriate for the noninhalation pathways.

Generally, the inhalation pathway is the largest contributor to the total dose. However, there are situations where a noninhalation pathway of exposure contributes substantially to a noncancer chronic HI. In these cases, the high-end point-estimate of dose is appropriate to use for the three dominant pathways and the average point-estimate for the non-dominant pathways. Dominant pathways are defined as the three pathways that contribute the most to the total hazard quotient for a chemical noncancer HI result when using high-end point-estimates for all the exposure pathways under

consideration. Typically inhalation would be one of these three pathways. In addition, no exposure duration adjustment (e.g., 9/70 or 30/70) should be made for noncancer assessments. See the Part IV TSD for a detailed discussion of the tiered approach, or Section 8.2.5 for a short overview of each tier.

Information contained in the following locations is needed to evaluate noncancer health impacts. Chapter 4 describes air dispersion modeling and both Chapter 6 and Appendix L list all the needed dose-response information. Appendix I presents sample calculations for determining chronic multipathway noncancer HQs and HIs and acute (inhalation) HQs and HIs. Chapter 9 provides an outline of information required for risk characterization. The HARP software is the recommended model for calculating and presenting HRA results for the Hot Spots Program. Information on obtaining the HARP software can be found under the Air Toxics Program on the ARB's web site at *www.arb.ca.gov*.

A. 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 method for calculating the health impact from both acute and chronic exposure (respiratory endpoint) is the standard HI approach (see Sections 8.3.1 and 8.3.4). 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 both the chronic and acute HI calculations, 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 both the acute and chronic assessments for the respiratory endpoint are ozone, nitrogen dioxide, sulfur dioxide, sulfates, and hydrogen sulfide.

8.3.1 Noncancer Chronic Inhalation Health Impacts

All substances in the Hot Spots Program must be evaluated through the inhalation pathway. Noncancer chronic inhalation health impacts are calculated by dividing the substance-specific annual average air concentration in micrograms per cubic meter ($\mu g/m^3$) by the chronic inhalation REL ($\mu g/m^3$) (Table 6.2). An 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. If this calculation is performed for a single substance, then it is called the hazard quotient (HQ). The following equation illustrates how to calculate the HQ for chronic inhalation exposure.

$$\text{Hazard Quotient} = \frac{\text{Annual Average Concentration } (mg/m^3)}{\text{Chronic Reference Exposure Level } (mg/m^3)}$$

The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments. July 2003.

The risk characterization of cumulative noncancer chronic health impacts from the emissions of multiple substances by the inhalation route is accomplished by determining the HI. The HI is calculated by summing the HQs from all of the substances that affect the same organ system. Note, do not add the HQs or HIs for different target organs together (e.g., do not add the impacts for the eye to the cardiovascular system). Table 6.2 and Appendix L have a list of the organ systems affected by each substance. No exposure duration adjustment (e.g., 9/70) should be made for noncancer assessments. The following equation illustrates how to calculate the HI for chronic exposure for the eye (target organ) from two substances. See Appendix I for an example calculation.

$$\text{Hazard Index } (HI_{eye}) = HQ_{\text{substance 1(eye)}} + HQ_{\text{substance 2(eye)}}$$

8.3.2 Noncancer Chronic Health Impacts from the Oral Route

Risk characterization for chronic health effects from exposure via the oral route is also conducted using the hazard index approach. The hazard quotient is obtained by dividing the oral dose (derived from the annual average concentration) in milligrams per kilogram-day (mg/kg-day) by the oral chronic REL, expressed in units of (mg/kg-day) (Table 6.3). 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 high-end point-estimates are used for all exposure pathways to determine which exposure pathways are dominant. Once the dominant exposure pathways are decided, the assessor uses the high-end point-estimates for the two dominant noninhalation pathways and the average point estimates for the rest of the non-dominant exposure pathways to determine the dose and chronic health impacts at the residential receptor. The 70-year exposure duration point-estimates are used for residential receptors and the worker (single) point-estimates are used for the MEIW in this calculation. No exposure duration adjustment (e.g., 9/70) should be made for noncancer assessments. The oral HQ is calculated by dividing the oral dose by the oral chronic REL. The significance of oral HQs greater or less than one are the same as explained for the chronic inhalation chronic HQ in Section 8.3.1. The following equation illustrates how to calculate the HQ for chronic noninhalation exposure.
To estimate the hazard index from noninhalation exposures when multiple pollutants impact the same target organ, the oral HQ's are summed (See Section 8.3.3 below).

$$\text{Hazard Quotient}_{\text{oral}} = \frac{\text{Exposure Pathway Dose (mg/kg - day)}}{\text{Chronic (oral) Reference Exposure Level (mg/kg - day)}}$$

8.3.3 Evaluation of Chronic Noncancer Multipathway Hazard Quotients and Hazard Indices

To determine multipathway chronic noncancer health impacts, it is necessary to calculate the total hazard index from both inhalation and noninhalation exposures. First, the inhalation HQ is calculated (Section 8.3.1). Second, if the substance has an oral REL, then the oral HQ is calculated (see Sections 8.2.5, 8.3, and 8.3.2). For a residential receptor, the oral HQ is calculated using the 70-year high-end point-estimates for the two dominant noninhalation pathways and the average point-estimates for the rest of the pertinent exposure pathways. If a worker is under evaluation, then the worker single point-estimates are used for the soil and dermal pathways. The third step is to add the HQs together for each exposure pathway to give the substance's total multipathway HQ by target organ. If there is only one substance, then the multipathway HQ is the same as the HI.

- If there are multiple substances emitted, then the fourth step is to total the HQs for all the individual substances by each target organ. For example, add the HQs for all substances that impact the respiratory system, then repeat this step for the next target organ system (e.g., cardiovascular system). This step is repeated until all target organs (for the substances emitted) are individually totaled. These impacts by target organ are now referred to as the HI. Note, do not add the HQs or HIs for different target organ together (e.g., do not add the impacts for the respiratory system to the cardiovascular system). No exposure duration adjustment (e.g., 9/70) should be made for noncancer assessments. See Appendix I for an example calculation.

- For respiratory irritants, do not add in an oral contribution to the HI for the respiratory system for chemicals with both inhalation and oral RELs.

8.3.4 Noncancer Acute Health Impacts

Risk characterization for acute health effects uses the same principles (HQ, for an individual substance, and HI, for multiple substances) as the chronic noncancer inhalation methodology (see Section 8.3.1). All acute substances are evaluated through the inhalation pathway only.

- Noncancer acute health impacts are calculated by dividing the substance-specific short-term maximum concentration in micrograms per cubic meter ($\mu g/m^3$) by the acute REL (also in units of $\mu g/m^3$) (Table 6.1) for each substance. If this calculation is performed for a single substance, then it is called the HQ. The HQ should be applied to all appropriate target organs for a given substance.

- If multiple substances are emitted, then the next step is to total the individual substance's HQs by each target organ. For example, add the HQs for all substances that impact the respiratory system, then repeat this step for the next target organ system. This step is repeated until all target organs (for the substances emitted) are individually totaled. These impacts by target organ are now referred to as the HI. Note, do not add the

HQs or HIs for different target organs together (e.g., do not add the impacts for the respiratory system to immune system).

There are no oral acute RELs since it is anticipated that health effects from such a brief exposure via the oral route would be insignificant relative to the inhalation route. No exposure duration adjustment should be made for noncancer assessments. See Appendix I for an example calculation. HQs calculated using one, four, six, and seven hour exposure duration RELs may be added together for calculation of an acute HI. This would only occur in evaluating reproductive and developmental toxicants, since all other endpoints have only one hour acute RELs.

The HARP software incorporates two procedures for determining an acute HI. Both procedures use the calculations for HQ and HI described above. These two procedures make a difference when a facility has two or more separated emission points or for HRAs involving multiple facilities. The first procedure is a more simplistic approach (consistent with previous CAPCOA HRA methods) where the maximum concentrations from each emission source are superimposed to impact receptors at the same time, irrespective of wind direction and/ or atmospheric stability. This procedure is a simple, health protective approach to assess acute impacts. The second procedure is more refined than the first and improves on previous HRA methods. This second procedure takes into account meteorology and relative source positions by superimposing results from multiple sources with concurrent wind direction and atmospheric conditions, thereby computing a more refined maximum impact by hour at each receptor. This refined HI procedure may decrease the concentrations at many receptor locations when compared to the simplistic approach, but should not underestimate potential health impacts (i.e., HQs or HIs). This dual procedure approach is another way the new HRA guidelines are building flexibility into the HRA methods.

8.4 Population-Level Risk Estimates

8.4.1 Carcinogenic Risk

There are basically two ways to provide population-level risk estimates, namely cancer burden estimates and estimates of the number of people exposed at specific cancer risk levels.

1. The cancer burden is calculated by multiplying the number of people exposed (census information) by the cancer risk at either the MEIR or the population centroid of each census block. The result of this calculation is an estimate of the number of cancer cases expected from a 70-year exposure to current estimated facility emissions.

2. 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 the cancer burden calculation used by some Districts in the past. The new approach provides a much easier way 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. Such information can be useful in risk management decisions. The level of detail required for the population 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. Some suggested approaches and methods for handling the breakdown of population and performance of a screening or refined population exposure analyses are provided in Section 4.6.

The population estimates should be based on the latest available census results. The population of the census block may be assumed to be equally distributed over the census block, unless for some reason more refined information is available. The population in census blocks cut by two or more risk isopleths can thus be apportioned based on the area in each isopleth. The isopleths needed should be drawn using the smallest practical grid size. The Districts may ask facilities to use the new procedure or the cancer burden approach. The District or reviewing authority should be consulted before beginning the population exposure estimates and, as results are generated, further consultation may be necessary.

A fundamental first step in estimating the number of people at risk from facility emissions is to define the zones of impact (see Section 4.6.1). This zone is commonly defined as the area within the isopleth surrounding the facility where receptors have a multipathway cancer risk greater than 10^{-6}. 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. See Table 8.3 for an example of data summarizing population exposure estimates for cancer risk.

Table 8.3 Example of Estimates of Population Risk

Estimated Number of Persons Exposed	Cancer Risk [N] (chances per million)
X	1 to 10
Y	10 to 100
Z	>100

(N) Column would be titled to reflect acute or chronic noncancer 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. 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*. Chapter 9 provides an outline that specifies the content and recommended format of HRA results.

8.4.2 Population Estimates of Noncancer Health Impacts

The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments. July 2003.

A noncancer chronic and acute population estimate of the number of people exposed to acute 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 8.4.1 (for the population exposure estimate for cancer risk) should be used in the chronic and acute population estimates. Population estimates for acute and chronic health impacts should be presented separately and in a format consistent with Table 8.3.

9. Summary of the Requirements for a Modeling Protocol and a Health Risk Assessment Report

The purpose of this chapter is to clarify the type of information that is expected to be included in modeling protocols and health risk assessments (HRAs). These outlines are intended to promote transparent, consistent presentation and efficient review of these products. It is possible that protocols and HRAs that do not include all the information presented in these outlines may be considered deficient by the reviewing authority. We recommend that persons preparing these products consult with the local Air Pollution Control or Air Quality Management District (District) to determine if the District has modeling or HRA guidelines that supercede these outlines. 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 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.**

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.

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 maximum one-hour emissions (pounds/hour and grams/second)*.

- 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.

III. Models / Modeling Assumptions

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

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

- 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.

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

V. Deposition

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

VI. Receptors

- Identify the method that will be used to determine the location of sensitive receptors, the point of maximum impact (PMI), and the maximum exposed individual residential (MEIR) and worker (MEIW) receptors (e.g., fine receptor spacing of 20 meters at the fenceline 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 the method that will be used to evaluate potential cancer risk in the vicinity of the facility for purposes of calculating cancer burden or population impact estimates. Clarify the same information for the presentation of noncancer 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.

- Identify if sensitive receptors are present and which receptors will be evaluated in the HRA.

VII. Maps

- 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 and chronic zones of impact (e.g., 0.5, 1.0, etc.).

9.2 Outline for a Health Risk Assessment Report

The purpose of this section is to provide an outline to assist with the preparation and review of heath risk assessments (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.

I. Table of Contents

- Section headings with page numbers indicated.
- Tables and figures with page numbers indicated.
- Appendices with page numbers indicated.

II. Executive Summary

- Name of the facility including the complete address.
- Facility identifier number (consult the District).
- Description of facility operations and a list identifying emitted substances including table of maximum 1-hour and annual average emissions.
- Provide a brief definition of acute, chronic, and cancer health impacts and multipathway substances.
- Text presenting overview of dispersion modeling and exposure assessment.
- Text defining dose-response assessment for cancer and noncancer health impacts and a table showing target organ systems by substance for noncancer impacts.

- Summary of results, including:
    - 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 the following information, potential cancer risk should be presented for a 70-year, Tier–1 analysis. Results of other exposure assumptions or tier evaluations can be presented, but must be clearly labeled. For the Hot Spots Program, the 70-year exposure duration should be used as the basis for public notification and risk reduction audits and plans.

- Text presenting an overview of the (total) potential multipathway cancer risk 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, and PMI.

- Provide a map of 70-year lifetime cancer risk zone of impact, if applicable.

- Text presenting an overview of the acute and chronic noncancer hazard quotients or the (total) hazard indices for the PMI, MEIR, MEIW, and sensitive receptors. Include separate statements (for acute 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 and chronic exposures.

- Identify any subpopulations (e.g., subsistence fishers) 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.

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 and chronic health impacts. In addition, identify any substances that present a potential 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 should be reported in the risk assessment. In addition, doses calculated by pathway of exposure for each substance should be included in this section. The District may have specific requirements regarding format and content (see Section 4.13). **Sample calculations may need to be provided (in an appendix) for each step to indicate how the reported emissions data were used, if software other than HARP is used. The educated reader should be able to reproduce the risk assessment without the need for clarification. The location of any information that is presented in appendices, on electronic media, or attached documents that supports information presented in this section, must be clearly identified by title and page number in this section's text and in the document's table of contents.**

1. Information on the Facility and its Surroundings

- Report the following information regarding the facility and its surroundings:
  - Facility name
  - Facility identifier number (consult the District).
  - Location (use actual UTM coordinates and street address)
  - Land use type (see Section 4.4)
    - Local topography.
    - Facility plot plan identifying†
      - emission source locations
      - property line
      - horizontal scale
      - building heights and dimensions
      - complex terrain if applicable

- Description of the site/route dependent exposure pathways. Provide a summary of the site-specific inputs used for each 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.

2. Source and Emission Inventory Information†

Source Description and Release Parameters

- Report the following information for each source in table format:
  - Source identification number used by the facility
  - Source name
  - Source location using actual UTM coordinates (m)
  - Source base elevation (m)
  - Source height (m)
  - Source dimensions (e.g., stack diameter, building dimensions, area size) (m)
  - Exhaust gas exit velocity (m/s)
  - Exhaust gas volumetric flow rate (ACFM)
  - Exhaust gas exit temperature (K)

(See Appendix K for an example.)

Source Operating Schedule

- The operating schedule for each source should be reported in table form including the following information:
  - Number of operating hours per day and per year (e.g., 0800-1700, 2700 hr/yr)
  - Number of operating days per week (e.g., Mon-Sat)
  - Number of operating days or weeks per year (e.g., 52 wk/yr excluding major holidays)

(See Appendix K for an example**.)**

Emission Control Equipment and Efficiency

- Report emission control equipment and efficiency by source and by substance. The description should be brief.

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 (see Appendix K):
  - Source name
  - Source identification number
  - Substance name and CAS number (Emittent ID from Inventory Guidelines)
  - Annual average emissions for each substance (lb/yr & g/s)*
  - Maximum one-hour emissions for each substance (lb/hr & g/s)*

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

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 (see Appendix K):
  - Substance name and CAS number (Emittent ID from Inventory Guidelines)
  - Annual average emissions for each substance (lb/yr & g/s)
  - Maximum one-hour emissions for each substance (lb/hr & g/s)

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.

3. Meteorological Data

- The HRA should indicate the source and time period of the meteorological data used. Include the meteorological data (electronically) with the HRA.

- Include proper justification for using this data including information regarding appropriateness and quality assurance/quality control.

- Identify any gaps in the data; if gaps exist, describe how the data gaps are filled.
- Provide a wind rose for a minimum of the entire time period of the meteorological data used, and time period coincident with operating schedule. (Other wind roses may be useful as well, such as a stability rose or a day/night wind rose.)

- The HRA should indicate if the District required the use of a specified representative meteorological data set or the use of default meteorological conditions from SCREEN3. 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 model used, the level of detail (screening or refined analysis) and the rationale behind the selection.

- Table and text that specifies the following information for each air dispersion model used:
  - version number
  - selected options and parameters
  - receptor grid spacing

5. Air Dispersion Modeling Results

- All information used for the report must be presented in the HRA. Ideally, a summary of data used in the HRA will be on paper and all data and model input and output (e.g., the ISCST3 input file containing the regulatory options and emission parameters, receptor locations, meteorology, etc) files will be provided electronically (e.g., CD).

- For the PMI, MEIR, MEIW, and any sensitive receptors required by the District, include tables that summarize the annual average concentrations that are calculated for all the substances at each site. We recommend the use of tables to present the relative contribution of each emission point to the receptor concentration. (These tables should have clear reference to the computer model that generated the data. It should be made clear to any reader how data from the computer output was transferred to these tables).

- For the PMI, MEIR, MEIW, and any sensitive receptors required by the District, include tables that summarize the maximum one-hour; four, six, or seven-hour (for those substance with RELs based on those averaging periods); and 30-day average (lead only‡) concentrations. (These tables should have clear reference to the computer model that generated the data. It should be made clear to any reader how data from the computer output was transferred to these tables).

- If proprietary software is used, all algorithms and parameters should be included with the HRA in a clear, easy to use format.

C. Dose-Response

- Provide tables of the inhalation and oral RELs 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.

- Provide a table of target organ systems for each noncancer substance, including chronic inhalation, chronic oral (if applicable), and acute.

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 potential cancer risk for the PMI, MEIR and sensitive receptors of interest must be presented in the HRA's text, tables, and maps using a (lifetime) 70-year exposure period. MEIW location should use appropriate exposure periods. For the Hot Spots Program, the 70-year exposure duration should be used as the basis for residential public notification and risk reduction audits and plans. All HRAs must include the results of a Tier-1 exposure assessment (see Chapter 2 and 8, or Part IV 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 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.

- Table and text providing description of the PMI and MEIR for 9-and 30-year cancer risk.

- Table and text providing the location [UTM coordinates and the block/street address (e.g., north side of 3,000 block of Smith Street)] and description of any sensitive receptors that are 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.). 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; Part IV; Technical Support Document for Exposure Assessment and Stochastic Analysis (2000b)* (Part IV TSD), 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.

- 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).

- 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) 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. Note: chronic noncancer results should be shown with inhalation and oral contributions (shown separately) and for the combined (multipathway) impact.

- Identify any subpopulations (e.g., subsistence fishers) 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 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). Note that the HARP program contains a mapping feature.
    - 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 minimum exposure pathways (inhalation, soil ingestion, dermal exposure, and breast-milk consumption) and the zone of impact for all the applicable exposure pathways (minimum exposure pathways plus additional site/route specific 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.
- 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), 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 heath 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

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. The HARP program (input and output) files will include many of the items listed below.

- **Potential Appendix Topics (if not presented elsewhere in the HRA report):**
  - List of all receptors locations (UTM coordinates 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.
  - 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).

- **List of HARP files that meet the Submittal Requirements**
  - ISC workbook file with all ISC parameters (filename.ISC).
  - ISC input file generated by HARP when ISC is run (filename.INP)
  - ISC output file generated by HARP when ISC in run (filename.OUT)
  - ISC binary output file; holds χ/Q for data for each hour (filename.BIN)
  - List of error messages generated by ISC (filename.ERR)
  - Sources receptor file; contains list of sources and receptors for the ISC run; generated by HARP when you set up ISC (filename.SRC)
  - Point estimate risk values generated by HARP; this file is updated automatically each time you perform one of the point estimate risk analysis functions (filename.RSK)
  - Average and maximum χ/Q values for each source-receptor combination; generated by ISC (filename.XOQ)
  - Plot file generated by ISC (filename.PLT)

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- Representative meteorological data used for the facility air dispersion modeling (filename.MET)
- Site-specific parameters used for all receptor risk modeling (filename.SIT)
- Map file used to overlay facility and receptors (filename.DEB)

(†) Health and Safety Code (HSC) Section 44346 authorizes facility operators to designate certain Hot Spots information as trade secret. HSC Section 44361(a) requires Districts to make health risk assessments available for public review upon request. HSC 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.

(‡) Please see Appendix F or contact the Office of Environmental Health Hazard Assessment for information on calculating and presenting chronic lead results.

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