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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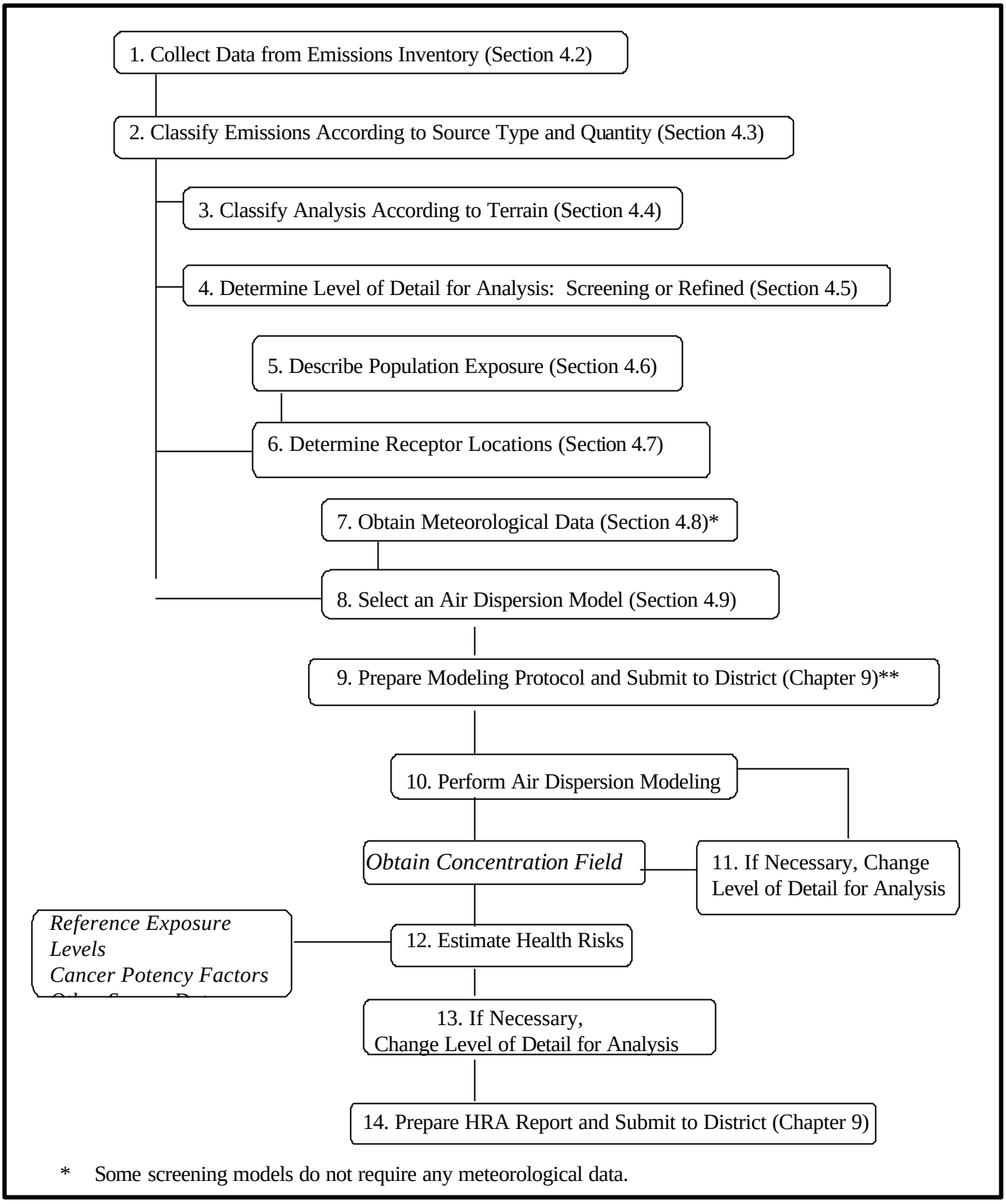
Figure 1. Overview of the Air Dispersion Modeling Process.



* 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*

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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.

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

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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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Appendix A

Air Toxics Hot Spots Program

List Of Substances*

*The List of Substances presented in Appendix A is periodically updated by the California Air Resources Board. The last update was July 1, 1997.

Appendix A-I

Substances For Which

Emissions Must Be Quantified

July 1, 1997

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APPENDIX A-I
Substances For Which Emissions Must Be Quantified

Emittent ID Other (Note [1]) Notes(s)	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Applicable Degree of Accuracy (lb/yr) (Note [5])	Source List(s) (Note [6])
75070	Acetaldehyde		c	20.	1 2 3 4
60355	Acetamide		c	2.	1 2 3 4
75058	Acetonitrile	06/91		200.	1 2
98862	Acetophenone	06/91		100.	1 2
53963	2-Acetylaminofluorene [PAH-Derivative, POM]		c	100.	1 2 4 5
107028	Acrolein			0.05	1 2
79061	Acrylamide		c	0.01	1 2 3 4
79107	Acrylic acid	06/91		5.	1 2
107131	Acrylonitrile		c	0.1	1 2 3 4 5
107051	Allyl chloride		c	5.	1 2 4
7429905	Aluminum	06/91		100.	1
1344281	Aluminum oxide (fibrous forms)	06/91		100.	7
117793	2-Aminoanthraquinone [PAH-Derivative, POM]		c	5.	1 2 4 5
92671	4-Aminobiphenyl [POM]		c	100.	1 2 3 4 5
61825	Amitrole		c	0.1	3 4 5
7664417	Ammonia			200.	1 2
6484522	Ammonium nitrate	06/91		100.	1
7783202	Ammonium sulfate	06/91		100.	1
62533	Aniline	09/90	c	5.	1 2 4
90040	o-Anisidine		c	100.	1 2 3 4 5
-	Anthracene [PAH, POM], (see PAH)				
7440360	Antimony	06/91		1.	7
*	Antimony compounds	06/91		1.	1 2
[7]					
	including but not limited to:				
1309644	Antimony trioxide	09/90	c	1.	1 2 3 4
[7]					
7440382	Arsenic		c	0.01	1 2 3 4 5
1016	Arsenic compounds (inorganic)		c	0.01	1 2 3 4 5
[7]					
	including but not limited to:				
7784421	Arsine			0.01	1 2 7
[7]					
1017	Arsenic compounds (other than inorganic)	06/91		0.1	1
[7]					
7440393	Barium	06/91		1.	7
*	Barium compounds	06/91		1.	1
[7]					
-	Benz[a]anthracene [PAH, POM], (see PAH)				
71432	Benzene		c	2.	1 2 3 4 5

92875	Benzidine (and its salts) [POM]		c	0.0001	1 2 3 4 5	
1020	Benzidine-based dyes [POM]		c	0.0001	1 2 3	
	including but not limited to:					
1937377	Direct Black 38 [PAH-Derivative, POM]		c	0.0001	1 2 4 5	
2602462	Direct Blue 6 [PAH-Derivative, POM]		c	0.0001	1 2 4 5	
16071866	Direct Brown 95 (technical grade) [POM]	09/89	c	0.0001	1 2 4	
-	Benzo[a]pyrene [PAH, POM], (see PAH)					
-	Benzo[b]fluoranthene [PAH, POM], (see PAH)					
271896	Benzofuran	06/91	c	100.	4	

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Emittent ID Other (Note [1]) Notes(s)	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Applicable Degree of Accuracy (lb/yr) (Note [5])	Source List(s) (Note [6])
98077	Benzoic trichloride {Benzotrichloride}		c	10.	1 2 4 5
-	Benzo[j]fluoranthene [PAH, POM], (see PAH)				
-	Benzo[k]fluoranthene [PAH, POM], (see PAH)				
98884	Benzoyl chloride	06/91		100.	1
94360	Benzoyl peroxide	06/91		100.	7
100447	Benzyl chloride		c	50.	1 2 4
7440417	Beryllium		c	0.001	1 2 3 4 5
*	Beryllium compounds	09/89	c	0.001	1 2 3 4 5
[7]					
92524	Biphenyl [POM]	06/91		0.5	1 2
111444	Bis(2-chloroethyl) ether {DCEE}	09/89	c	0.05	1 2 4
542881	Bis(chloromethyl) ether		c	0.001	1 2 3 4 5
103231	Bis(2-ethylhexyl) adipate	06/91		100.	1
7726956	Bromine			0.5	2
*	Bromine compounds (inorganic)			100.	1 2
[7]					
	including but not limited to:				
7758012	Potassium bromate			0.1	1 3 4
[7]					
75252	Bromoform	06/91		100.	1 2 4
106990	1,3-Butadiene		c	0.1	1 2 3 4 5
141322	Butyl acrylate	06/91		100.	1
71363	n-Butyl alcohol	06/91		100.	1
78922	sec-Butyl alcohol	06/91		100.	1
75650	tert-Butyl alcohol	06/91		100.	1
85687	Butyl benzyl phthalate	06/91		100.	1
7440439	Cadmium		c	0.01	1 2 3 4 5
*	Cadmium compounds		c	0.01	1 2 3 4 5
[7]					
156627	Calcium cyanamide	06/91		100.	1 2
105602	Caprolactam	06/91		100.	1 2
2425061	Captafol	09/89	c	100.	4
133062	Captan	09/90	c	100.	1 2 4
63252	Carbaryl [PAH-Derivative, POM]	06/91		100.	1 2
1050	Carbon black extracts		c	2.	1 3 4
75150	Carbon disulfide	09/89		200.	1 2 4
56235	Carbon tetrachloride		c	1.	1 2 3 4 5
463581	Carbonyl sulfide	06/91		100.	1 2
1055	Carrageenan (degraded)		c	100.	3 4
120809	Catechol	06/91		100.	1 2

133904	Chloramben	06/91		100.	1	2						
57749	Chlordane	09/89	c	10.	1	2		4				
108171262	Chlorinated paraffins (average chain length, C12; approximately 60% chlorine by weight)	09/89	c	2.					3	4	5	
7782505	Chlorine			0.5	1	2						
10049044	Chlorine dioxide	06/91		1.	1							
79118	Chloroacetic acid	06/91		100.	1	2						
532274	2-Chloroacetophenone	06/91		0.1	1	2						
106478	p-Chloroaniline	07/96		100.				4				7

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Substances For Which Emissions Must Be Quantified

Emittent ID Other (Note [1]) Notes(s)	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Applicable Degree of Accuracy (lb/yr) (Note [5])	Source List(s) (Note [6])
1058	Chlorobenzenes	06/91		100.	1
	including but not limited to:				
108907	Chlorobenzene			200.	1 2
25321226	Dichlorobenzenes (mixed isomers)	06/91		100.	1 7
	including:				
95501	1,2-Dichlorobenzene	06/91		200.	1 7
541731	1,3-Dichlorobenzene	06/91		100.	1 7
106467	p-Dichlorobenzene {1,4-Dichlorobenzene}		c	5.	1 2 3 5
120821	1,2,4-Trichlorobenzene	06/91		200.	1 2
510156	Chlorobenzilate [POM] {Ethyl-4,4'-dichlorobenzilate}	09/90	c	100.	1 2 4
67663	Chloroform		c	10.	1 2 3 4 5
107302	Chloromethyl methyl ether (technical grade)		c	100.	1 2 4 5
1060	Chlorophenols		c	100.	1 3
	including but not limited to:				
120832	2,4-Dichlorophenol	06/91	c	100.	1 7
87865	Pentachlorophenol	09/90	c	10.	1 2 4
58902	2,3,4,6-Tetrachlorophenol	07/96	c	100.	1 7
95954	2,4,5-Trichlorophenol	06/91	c	100.	1 2
88062	2,4,6-Trichlorophenol		c	2.	1 2 4
95830	4-Chloro-o-phenylenediamine		c	10.	3 4 5
76062	Chloropicrin			2.	7
126998	Chloroprene			5.	1 2
95692	p-Chloro-o-toluidine		c	0.5	3 4
7440473	Chromium	06/91		0.001	7
*	Chromium compounds (other than hexavalent) [7]	06/91		0.001	1 2
18540299	Chromium, hexavalent (and compounds) [7]		c	0.0001	1 2 3 4 5
	including but not limited to:				
10294403	Barium chromate [7]	06/91	c	0.001	1 2 5
13765190	Calcium chromate [7]	06/91	c	0.001	1 2 5
1333820	Chromium trioxide [7]	06/91	c	0.0001	1 2 5
7758976	Lead chromate [7]	06/91	c	0.001	1 2 5

10588019	Sodium dichromate	06/91	c	0.0001	1 2 5	[7]
7789062	Strontium chromate	06/91	c	0.001	1 2 5	[7]
-	Chrysene [PAH, POM], (see PAH)					
7440484	Cobalt	06/91		0.5		7
*	Cobalt compounds	06/91		0.5	1 2	[7]
1066	Coke oven emissions		c	0.05	1 2 3 4 5	
7440508	Copper			0.1	2	
*	Copper compounds	09/89		0.1	1 2	[7]
1070	Creosotes		c	0.05	1 3 4	
120718	p-Cresidine		c	1.	3 4 5	
1319773	Cresols (mixtures of) {Cresylic acid} including:			5.	1 2	
108394	m-Cresol	06/91		5.	1 2	
95487	o-Cresol	06/91		5.	1 2	
106445	p-Cresol	06/91		5.	1 2	
4170303	Crotonaldehyde	07/96	c	50.		7

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98828	Cumene	06/91		200.	1 2	
80159	Cumene hydroperoxide	06/91		100.	1	
135206	Cupferron		c	0.5	4 5	
1073	Cyanide compounds	06/91		0.05	1 2	[8]
	including but not limited to:					
74908	Hydrocyanic acid			10.	2	
110827	Cyclohexane	06/91		200.	1	
108930	Cyclohexanol	07/96		200.	7	
66819	Cycloheximide			2.	6	
1163195	Decabromodiphenyl oxide [POM]	06/91		100.	1 2	
1075	Dialkylnitrosamines			0.001	1	
	including but not limited to:					
924163	N-Nitrosodi-n-butylamine		c	0.0001	1 3 4 5	
1116547	N-Nitrosodiethanolamine		c	100.	1 3 4 5	
55185	N-Nitrosodiethylamine		c	0.001	1 3 4 5	
62759	N-Nitrosodimethylamine		c	0.01	1 2 3 4 5	
621647	N-Nitrosodi-n-propylamine		c	0.01	1 3 4 5	
10595956	N-Nitrosomethylethylamine		c	0.001	1 3 4	
615054	2,4-Diaminoanisole		c	5.	3 4	
1078	Diaminotoluenes (mixed isomers)	09/90	c	100.	1 4	
	including but not limited to:					
95807	2,4-Diaminotoluene {2,4-Toluenediamine}		c	0.05	1 2 3 4 5	
334883	Diazomethane	06/91	c	5.	1 2	
226368	Dibenz[a,h]acridine [POM]		c	0.5	1 2 3 4 5	
224420	Dibenz[a,j]acridine [POM]		c	0.5	1 2 3 4 5	
-	Dibenz[a,h]anthracene [PAH, POM], (see PAH)					
194592	7H-Dibenzo[c,g]carbazole		c	0.05	1 2 3 4 5	
-	Dibenzo[a,e]pyrene [PAH, POM], (see PAH)					
-	Dibenzo[a,h]pyrene [PAH, POM], (see PAH)					
-	Dibenzo[a,i]pyrene [PAH, POM], (see PAH)					
-	Dibenzo[a,l]pyrene [PAH, POM], (see PAH)					
132649	Dibenzofuran [POM]	06/91		100.	1 2	
-	Dibenzofurans (chlorinated) (see Polychlorinated dibenzofurans) [POM]					
96128	1,2-Dibromo-3-chloropropane {DBCP}		c	0.01	1 2 3 4 5	
96139	2,3-Dibromo-1-propanol	07/96	c	50.	4	
84742	Dibutyl phthalate	06/91		100.	1 2	
-	p-Dichlorobenzene {1,4-Dichlorobenzene} (see Chlorobenzenes)					

91941	3,3'-Dichlorobenzidine [POM]		c	0.1	1 2 3 4 5
72559	Dichlorodiphenyldichloroethylene {DDE} [POM]	09/89	c	100.	1 2 4
75343	1,1-Dichloroethane {Ethylidene dichloride}	09/90	c	20.	1 2 4
94757	Dichlorophenoxyacetic acid, salts and esters {2,4-D}	06/91		100.	1 2
78875	1,2-Dichloropropane {Propylene dichloride}	09/90	c	20.	1 2 4
542756	1,3-Dichloropropene		c	10.	1 2 3 4 5
62737	Dichlorovos {DDVP}	09/89	c	0.5	1 2 4
115322	Dicofol [POM]	06/91		100.	1 2

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- - [9]	Diesel engine exhaust	09/90	c		1 3 4
9901 [9]	Diesel engine exhaust, particulate matter	09/90	c	10.	1 3 4
9902 [9]	Diesel engine exhaust, total organic gas	09/90	c	10.	1 3 4
#	Diesel fuel (marine)	06/91	c		
111422	Diethanolamine	06/91		20.	1 2
117817	Di(2-ethylhexyl) phthalate {DEHP}		c	20.	1 2 3 4 5
64675	Diethyl sulfate		c	100.	1 2 3 4 5
119904	3,3'-Dimethoxybenzidine [POM]		c	100.	1 2 3 4 5
60117	4-Dimethylaminoazobenzene [POM]		c	0.01	1 2 3 4 5
121697	N,N-Dimethylaniline	06/91		200.	1 2
57976	7,12-Dimethylbenz[a]anthracene [PAH-Derivative, POM]	09/90	c	0.0001	1 2 4
119937	3,3'-Dimethylbenzidine {o-Tolidine} [POM]		c	10.	1 2 3 4 5
79447	Dimethyl carbamoyl chloride		c	100.	1 2 3 4 5
68122	Dimethyl formamide	09/90	c	100.	1 2 3
57147	1,1-Dimethylhydrazine		c	0.1	1 2 3 4 5
131113	Dimethyl phthalate	06/91		50.	1 2
77781	Dimethyl sulfate		c	0.01	1 2 3 4 5
534521	4,6-Dinitro-o-cresol (and salts)	06/91		100.	1 2
51285	2,4-Dinitrophenol	06/91		100.	1 2
42397648	1,6-Dinitropyrene [PAH-Derivative, POM]	06/91	c	0.001	1 2 3 4
42397659	1,8-Dinitropyrene [PAH-Derivative, POM]	06/91	c	0.05	1 2 3 4
25321146	Dinitrotoluenes (mixed isomers) including but not limited to:	06/91		100.	7
121142	2,4-Dinitrotoluene	09/89	c	0.5	1 2 4
606202	2,6-Dinitrotoluene	06/91		100.	7
123911	1,4-Dioxane		c	5.	1 2 3 4 5
-	Dioxins (Chlorinated dibenzodioxins) (see Polychlorinated dibenzo-p-dioxins) [POM]				
630933	Diphenylhydantoin [POM]		c	100.	1 2 4
122667	1,2-Diphenylhydrazine {Hydrazobenzene} [POM]		c	100.	1 2 4 5
1090	Environmental Tobacco Smoke		c	2.	1 3 4
106898	Epichlorohydrin		c	2.	1 2 3 4 5
106887	1,2-Epoxybutane	06/91		100.	1 2
1091	Epoxy resins	09/89		100.	6
140885	Ethyl acrylate		c	200.	1 2 3 4 5
100414	Ethyl benzene	06/91		200.	1 2

75003	Ethyl chloride {Chloroethane}			200.	1 2 4
-	Ethyl-4,4'-dichlorobenzilate (see Chlorobenzilate)				
74851	Ethylene	06/91		200.	7
106934	Ethylene dibromide {1,2-Dibromoethane}		c	0.5	1 3 4 5 6
107062	Ethylene dichloride {1,2-Dichloroethane}		c	2.	1 2 3 4 5
107211	Ethylene glycol	06/91		200.	1 2
151564	Ethyleneimine {Aziridine}	06/91		100.	1 2
75218	Ethylene oxide		c	0.5	1 2 3 4 5 6
96457	Ethylene thiourea		c	2.	1 2 3 4 5

APPENDIX A-I
Substances For Which Emissions Must Be Quantified

Emittent ID Other (Note [1]) Notes(s)	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Applicable Degree of Accuracy (lb/yr) (Note [5])	Source List(s) (Note [6])
1101	Fluorides and compounds	09/89		100.	2
	including but not limited to:				
7664393	Hydrogen fluoride			50.	1 2 7
1103 [10]	Fluorocarbons (brominated)			200.	6
1104 [10]	Fluorocarbons (chlorinated)			200.	1 6
	including but not limited to:				
76131	Chlorinated fluorocarbon {CFC-113} {1,1,2-Trichloro-1,2,2-trifluoroethane}			200.	1 2 6
75456	Chlorodifluoromethane {Freon 22}	07/96		200.	1 6 7
75434	Dichlorofluoromethane {Freon 12}	07/96		200.	1 6 7
75694	Trichlorofluoromethane {Freon 11}	07/96		200.	1 6 7
50000	Formaldehyde		c	5.	1 2 3 4 5 6
110009	Furan	07/96	c	5.	4
- - [9]	Gasoline engine exhaust	09/90	c		3
	including but not limited to:				
- - [9]	Gasoline engine exhaust (condensates & extracts)	06/91	c		4
9910 [9]	Gasoline engine exhaust, particulate matter	09/90	c	100.	3 4
9911 [9]	Gasoline engine exhaust, total organic gas	09/90	c	100.	3 4
1110 [11]	Gasoline vapors		c	200.	1 2 3 4
111308	Glutaraldehyde			0.1	1 6
1115	Glycol ethers and their acetates			100.	1 2 6
	including but not limited to:				
111466	Diethylene glycol	09/90		100.	1 6
111966	Diethylene glycol dimethyl ether	09/90		100.	1 2 6
112345	Diethylene glycol monobutyl ether	09/90		100.	1 2 6
111900	Diethylene glycol monoethyl ether	09/90		100.	1 2 6
111773	Diethylene glycol monomethyl ether	09/90		100.	1 2 6
25265718	Dipropylene glycol	09/90		100.	1 6
34590948	Dipropylene glycol monomethyl ether	09/90		100.	1 6
629141	Ethylene glycol diethyl ether	09/90		100.	1 2 6
110714	Ethylene glycol dimethyl ether	09/90		100.	1 2 6
111762	Ethylene glycol monobutyl ether	09/90		200.	1 2 6

110805	Ethylene glycol monoethyl ether	09/89		50.	1	2				6				
111159	Ethylene glycol monoethyl ether acetate	09/90		100.	1	2				6				
109864	Ethylene glycol monomethyl ether	09/89		10.	1	2				6				
110496	Ethylene glycol monomethyl ether acetate	09/90		200.	1	2				6				
2807309	Ethylene glycol monopropyl ether	09/90		100.	1	2				6				
107982	Propylene glycol monomethyl ether	09/90		200.	1					6				
108656	Propylene glycol monomethyl ether acetate	09/90		100.	1					6				
112492	Triethylene glycol dimethyl ether	09/90		100.	1	2				6				
76448	Heptachlor	09/89	c	100.	1	2		4						
118741	Hexachlorobenzene		c	0.1	1	2	3		5					
87683	Hexachlorobutadiene	06/91		0.1	1	2								
1120	Hexachlorocyclohexanes(mixed or technical grade) including but not limited to:		c	0.05	1		3	4	5					
319846	alpha-Hexachlorocyclohexane	07/96	c	0.1	1		3	4	5		7			
319857	beta-Hexachlorocyclohexane	07/96	c	0.1	1		3	4	5		7			
58899	Lindane {gamma-Hexachlorocyclohexane}	09/90	c	0.1	1	2		4						

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77474	Hexachlorocyclopentadiene			2.	1 2
67721	Hexachloroethane	09/90	c	200.	1 2 4
680319	Hexamethylphosphoramide		c	100.	1 2 3 4 5
110543	Hexane	06/91		200.	1 2
302012	Hydrazine		c	0.01	1 2 3 4 5
7647010	Hydrochloric acid			20.	1 2
-	Hydrocyanic acid (see Cyanide compounds)				
7783064	Hydrogen sulfide			5.	1 2
123319	Hydroquinone	06/91		100.	1 2
-	Indeno[1,2,3-cd]pyrene [PAH, POM], (see PAH)				
13463406	Iron pentacarbonyl	07/96		5.	7
1125	Isocyanates			0.05	6
	including but not limited to:				
822060	Hexamethylene-1,6-diisocyanate	06/91		0.05	1 2
101688	Methylene diphenyl diisocyanate {MDI} [POM]	06/91		0.1	1 2
624839	Methyl isocyanate			1.	1 2
-	Toluene-2,4-diisocyanate (see Toluene diisocyanates)				
-	Toluene-2,6-diisocyanate (see Toluene diisocyanates)				
78591	Isophorone	06/91		200.	1 2
78795	Isoprene, except from vegetative emission sources	07/96	c	200.	3
67630	Isopropyl alcohol	06/91		200.	1
80057	4,4'-Isopropylidenediphenol [POM]	06/91		100.	1 2
7439921	Lead		c	0.5	1 4 6
1128 [7]	Lead compounds (inorganic)		c	0.5	1 3
	including but not limited to:				
301042 [7] [12]	Lead acetate		c	1.	1 2 4 5
-	Lead chromate (see Chromium, hexavalent)				
7446277 [7]	Lead phosphate		c	2.	1 4 5
1335326 [7] [12]	Lead subacetate	09/90	c	2.	1 2 4
1129 [7]	Lead compounds (other than inorganic)	06/91		5.	1 2
108316	Maleic anhydride			0.5	1 2
7439965	Manganese			0.1	1 2

*	Manganese compounds	09/89		0.1	1 2
[7]					
7439976	Mercury			1.	1 2 4 6
*	Mercury compounds	09/89		1.	1 2 4
[7]					
	including but not limited to:				
7487947	Mercuric chloride			1.	2
[7]					
593748	Methyl mercury {Dimethylmercury}			1.	2
[7]					
67561	Methanol			200.	1 2
72435	Methoxychlor [POM]	06/91		100.	1 2
75558	2-Methylaziridine {1,2-Propyleneimine}		c	100.	1 2 3 4
74839	Methyl bromide {Bromomethane}			20.	1 2 6
74873	Methyl chloride {Chloromethane}	06/91		20.	1 2
71556	Methyl chloroform {1,1,1-Trichloroethane}			200.	1 2 6
56495	3-Methylcholanthrene [PAH-Derivative, POM]	09/90	c	0.001	1 2 4
3697243	5-Methylchrysene [PAH-Derivative, POM]		c	0.05	1 2 3 4 5

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101144	4,4'-Methylene bis(2-chloroaniline) {MOCA} [POM]		c	0.1	1 2 3 4 5	
75092	Methylene chloride {Dichloromethane}		c	50.	1 2 3 4 5 6	
101779	4,4'-Methylenedianiline (and its dichloride) [POM]		c	0.1	1 2 3 4 5	
78933	Methyl ethyl ketone {2-Butanone}	06/91		200.	1 2	
60344	Methyl hydrazine	06/91		100.	1 2	
74884	Methyl iodide {Iodomethane}		c	100.	1 2 4 5	
108101	Methyl isobutyl ketone {Hexone}	06/91		20.	1 2	
75865	2-Methyllactonitrile {Acetone cyanohydrin}	07/96		50.	7	
80626	Methyl methacrylate			200.	1 2 6	
109068	2-Methylpyridine	07/96		100.	7	
1634044	Methyl tert-butyl ether	06/91		200.	1 2	
90948	Michler's ketone [POM]		c	0.1	1 2 4 5	
1136	Mineral fibers (fine, manmade) (fine mineral fibers which are manmade and are airborne particles of a respirable size greater than 5 microns in length, less than or equal to 3.5 microns in diameter, with a length to diameter ratio of 3:1)	06/91	c	100.	1 2 7	
	including but not limited to:					
1056	Ceramic fibers	09/89	c	100.	1 2 3 4	
1111	Glasswool fibers	09/89	c	100.	1 2 3 4	
1168	Rockwool fibers	09/89	c	100.	1 2 3	
1181	Slagwool fibers	09/89	c	100.	1 2 3	
1135	Mineral fibers (other than manmade)			100.	2 7	
	including but not limited to:					
1332214	Asbestos		c	0.0001	1 2 3 4 5	
12510428	Erionite		c	100.	2 3 4	
1190	Talc containing asbestiform fibers		c	100.	2 3 4	
1313275	Molybdenum trioxide	06/91		100.	1	
-	Naphthalene [PAH, POM], (see PAH)					
7440020	Nickel		c	0.1	1 2 3 4 5	
*	Nickel compounds		c	1.	1 2 3 4 5	[7]
	including but not limited to:					
373024	Nickel acetate	06/91	c	0.1	1 2 5	[7]
3333393	Nickel carbonate	06/91	c	0.1	1 2 5	[7]
13463393	Nickel carbonyl		c	0.1	1 2 4 5	[7]

12054487	Nickel hydroxide	06/91	c	0.1	1 2 5 [7]
1271289	Nickelocene	06/91	c	0.1	1 2 5 [7]
1313991	Nickel oxide	06/91	c	0.1	1 2 5 [7]
12035722	Nickel subsulfide		c	0.1	1 2 4 5 [7]
1146	Nickel refinery dust from the pyrometallurgical process	09/89	c	0.1	4
7697372	Nitric acid	06/91		50.	1
139139	Nitrilotriacetic acid		c	100.	1 4 5
98953	Nitrobenzene			0.5	1 2
92933	4-Nitrobiphenyl [POM]	09/89	c	100.	1 2 4
7496028	6-Nitrochrysene [PAH-Derivative, POM]	06/91	c	0.001	1 2 3 4
607578	2-Nitrofluorene [PAH-Derivative, POM]	06/91	c	5.	1 2 3 4

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302705	Nitrogen mustard N-oxide		c	0.05	3 4
100027	4-Nitrophenol	06/91		100.	1 2
79469	2-Nitropropane		c	0.01	1 2 3 4 5
5522430	1-Nitropyrene [PAH-Derivative, POM]	06/91	c	0.5	1 2 3 4
156105	p-Nitrosodiphenylamine [POM]		c	5.	1 2 4 5
684935	N-Nitroso-N-methylurea		c	100.	1 2 4 5
59892	N-Nitrosomorpholine		c	0.01	1 2 3 4 5
100754	N-Nitrosopiperidine		c	200.	3 4 5
930552	N-Nitrosopyrrolidine		c	0.05	3 4 5
- - [13]	PAHs (Polycyclic aromatic hydrocarbons) [POM]				1 2
	including but not limited to:				
1151	PAHs, total, w/o individ. components reported			50.	1 2
1150	PAHs, total, with individ. components also reported			50.	1 2
83329	Acenaphthene [PAH, POM]	07/96		50.	1
208968	Acenaphthylene [PAH, POM]	07/96		50.	1
120127	Anthracene [PAH, POM]	06/91		50.	1 2 7
56553	Benz[a]anthracene [PAH, POM]		c	0.5	1 2 3 4 5
50328	Benzo[a]pyrene [PAH, POM]		c	0.05	1 2 3 4 5
205992	Benzo[b]fluoranthene [PAH, POM]		c	0.5	1 2 3 4 5
192972	Benzo[e]pyrene [PAH, POM]	07/96		0.5	1
191242	Benzo[g,h,i]perylene [PAH, POM]	07/96		0.5	1
205823	Benzo[j]fluoranthene [PAH, POM]		c	0.5	1 2 3 4 5
207089	Benzo[k]fluoranthene [PAH, POM]		c	0.5	1 2 3 4 5
218019	Chrysene [PAH, POM]	09/90	c	5.	1 2 4
53703	Dibenz[a,h]anthracene [PAH, POM]		c	0.1	1 2 3 4 5
192654	Dibenzo[a,e]pyrene [PAH, POM]		c	0.05	1 2 3 4 5
189640	Dibenzo[a,h]pyrene [PAH, POM]		c	0.001	1 2 3 4 5
189559	Dibenzo[a,i]pyrene [PAH, POM]		c	0.001	1 2 3 4 5
191300	Dibenzo[a,l]pyrene [PAH, POM]		c	0.001	1 2 3 4 5
206440	Fluoranthene [PAH, POM]	07/96		0.5	1
86737	Fluorene [PAH, POM]	07/96		0.5	1
193395	Indeno[1,2,3-cd]pyrene [PAH, POM]		c	0.5	1 2 3 4 5
91576	2-Methyl naphthalene [PAH, POM]	07/96		50.	1
91203	Naphthalene [PAH, POM]			50.	1 2
198550	Perylene [PAH, POM]	07/96		0.5	1
85018	Phenanthrene [PAH, POM]	07/96		0.5	1
129000	Pyrene [PAH, POM]	07/96		0.5	1

#	PAH-Derivatives (Polycyclic aromatic hydrocarbon derivatives) [POM] (including but not limited to those substances listed in Appendix A with the bracketed designation [PAH-Derivative, POM]) [14]	06/91			
56382	Parathion	06/91		100.	1 2
1336363	PCBs (Polychlorinated biphenyls) [POM]		c	0.01	1 2 3 4 5 6
82688	Pentachloronitrobenzene {Quintobenzene}	06/91		100.	1 2
79210	Peracetic acid	06/91		100.	1
127184	Perchloroethylene {Tetrachloroethene}		c	5.	1 2 3 4 5 6

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108952	Phenol			200.	1 2
106503	p-Phenylenediamine	06/91		100.	1 2
90437	2-Phenylphenol [POM]	06/91		100.	1 2
75445	Phosgene			2.	1 2
7723140	Phosphorus			0.1	1 2
- -	Phosphorus compounds:	09/89			2
7803512	Phosphine			0.01	1 2 7
7664382	Phosphoric acid	09/89		50.	1 2
10025873	Phosphorus oxychloride	09/89		0.1	2
10026138	Phosphorus pentachloride	09/89		0.1	2
1314563	Phosphorus pentoxide	09/89		0.1	2
7719122	Phosphorus trichloride	09/89		0.1	2
126738	Tributyl phosphate	09/89		100.	2
78400	Triethyl phosphine	09/89		100.	2
512561	Trimethyl phosphate	09/89		100.	2
78308	Triorthocresyl phosphate [POM]	09/89		0.5	1 2
115866	Triphenyl phosphate [POM]	09/89		100.	1 2
101020	Triphenyl phosphite [POM]	09/89		100.	1 2
85449	Phthalic anhydride			0.01	1 2
- -	Polychlorinated dibenzo-p-dioxins {PCDDs or Dioxins} [POM] including but not limited to:		c		1 2
1086	Dioxins, total, w/o individ. isomers reported {PCDDs}		c	0.00002	1 2
1085	Dioxins, total, with individ. isomers also reported {PCDDs}		c	0.00002	1 2
1746016	2,3,7,8-Tetrachlorodibenzo-p-dioxin {TCDD} [POM]		c	0.000001	1 2 3 4 5
40321764	1,2,3,7,8-Pentachlorodibenzo-p-dioxin [POM]		c	0.000001	1 2
39227286	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin [POM]		c	0.000001	1 2 4
57653857	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin [POM]		c	0.000001	1 2
19408743	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin [POM]		c	0.000001	1 2
35822469	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin [POM]		c	0.000001	1 2
3268879	1,2,3,4,5,6,7,8-Octachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1 2
41903575	Total Tetrachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1 2
36088229	Total Pentachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1 2
34465468	Total Hexachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1 2
37871004	Total Heptachlorodibenzo-p-dioxin [POM]	07/96	c	0.000001	1 2

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- -	Polychlorinated dibenzofurans {PCDFs or Dibenzofurans} [POM] including but not limited to:		c		1 2	
1080	Dibenzofurans (Polychlorinated dibenzofurans) {PCDFs} [POM]		c	0.00002	1 2	
51207319	2,3,7,8-Tetrachlorodibenzofuran [POM]		c	0.000001	1 2	
57117416	1,2,3,7,8-Pentachlorodibenzofuran [POM]		c	0.000001	1 2	
57117314	2,3,4,7,8-Pentachlorodibenzofuran [POM]		c	0.000001	1 2	
70648269	1,2,3,4,7,8-Hexachlorodibenzofuran [POM]		c	0.000001	1 2	
57117449	1,2,3,6,7,8-Hexachlorodibenzofuran [POM]		c	0.000001	1 2	
72918219	1,2,3,7,8,9-Hexachlorodibenzofuran [POM]		c	0.000001	1 2	
60851345	2,3,4,6,7,8-Hexachlorodibenzofuran [POM]		c	0.000001	1 2	
67562394	1,2,3,4,6,7,8-Heptachlorodibenzofuran [POM]		c	0.000001	1 2	
55673897	1,2,3,4,7,8,9-Heptachlorodibenzofuran [POM]		c	0.000001	1 2	
39001020	1,2,3,4,5,6,7,8-Octachlorodibenzofuran [POM]	07/96	c	0.000001	1 2	
55722275	Total Tetrachlorodibenzofuran [POM]	07/96	c	0.000001	1 2	
30402154	Total Pentachlorodibenzofuran [POM]	07/96	c	0.000001	1 2	
55684941	Total Hexachlorodibenzofuran [POM]	07/96	c	0.000001	1 2	
38998753	Total Heptachlorodibenzofuran [POM]	07/96	c	0.000001	1 2	
#	POM (Polycyclic organic matter) (including but not limited to those substances listed in Appendix A with the bracketed designation of [POM], [PAH, POM], or [PAH-Derivative, POM])	09/89			1 2	[15]
1120714	1,3-Propane sultone		c	0.05	1 2 3 4 5	
57578	beta-Propiolactone		c	10.	1 2 3 4 5	
123386	Propionaldehyde	06/91		200.	1 2	
114261	Propoxur {Baygon}	06/91		100.	1 2	
115071	Propylene			200.	1 2	
75569	Propylene oxide		c	10.	1 2 3 4 5	
-	1,2-Propyleneimine (see 2-Methylaziridine)					
110861	Pyridine	06/91		100.		7
91225	Quinoline	06/91		100.	1 2	
106514	Quinone	06/91		100.	1 2	
1165	Radionuclides including but not limited to:		c	100.	1 2 4	[16]
24267569	Iodine-131	09/89	c	100.	1 2 4	
1166	Radon and its decay products	09/89	c	100.	1 4	

50555	Reserpine [POM]		c	100.	1	2		4	5		
#	Residual (heavy) fuel oils	06/91	c								
7782492	Selenium			0.5		2					
*	Selenium compounds			0.5	1	2					
[7]											
	including but not limited to:										
7446346	Selenium sulfide	09/90	c	0.1		2		4	5		
[7]											
1175	Silica, crystalline		c	0.1	1		3	4			
7440224	Silver	06/91		2.							7
*	Silver compounds	06/91		2.	1						
[7]											

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1310732	Sodium hydroxide			2.	1 2
100425	Styrene		c	100.	1 2 3 6
96093	Styrene oxide		c	100.	1 2 3 4
7664939	Sulfuric acid	06/91		2.	1
100210	Terephthalic acid	06/91		100.	1
79345	1,1,2,2-Tetrachloroethane	09/90	c	1.	1 2 4
7440280	Thallium	06/91		100.	7
*	Thallium compounds	06/91		100.	7
[7]					
62555	Thioacetamide		c	0.01	3 4 5
62566	Thiourea		c	0.1	1 3 4 5
7550450	Titanium tetrachloride	06/91		100.	1 2
108883	Toluene			200.	1 2 4 6
-	2,4-Toluenediamine (see 2,4-Diaminotoluene)				
1204	Toluene diisocyanates including but not limited to:	06/91	c	0.1	1 3
584849	Toluene-2,4-diisocyanate		c	0.1	1 2 3 5
91087	Toluene-2,6-diisocyanate		c	0.1	1 2 3 5
95534	o-Toluidine		c	10.	1 2 3 4 5
8001352	Toxaphene {Polychlorinated camphenes}		c	100.	1 2 3 4 5
79005	1,1,2-Trichloroethane {Vinyl trichloride}	06/91	c	50.	1 2 4
-	1,1,1-Trichloroethane (see Methyl chloroform)				
79016	Trichloroethylene		c	20.	1 2 4
-	2,4,6-Trichlorophenol (see Chlorophenols)				
96184	1,2,3-Trichloropropane	07/96	c	200.	3 4 7
121448	Triethylamine	06/91		20.	1 2
1582098	Trifluralin	06/91		100.	1 2
95636	1,2,4-Trimethylbenzene	06/91		5.	1
540841	2,2,4-Trimethylpentane	06/91		100.	1 2
51796	Urethane {Ethyl carbamate}		c	0.1	1 2 3 4 5
7440622	Vanadium (fume or dust)	06/91		10.	7
[17]					
108054	Vinyl acetate	06/91		200.	1 2
593602	Vinyl bromide		c	20.	1 2 3 4
75014	Vinyl chloride		c	0.5	1 2 3 4 5
100403	4-Vinylcyclohexene	07/96	c	5.	3
75025	Vinyl fluoride	07/96	c	200.	3
75354	Vinylidene chloride			20.	1 2
1206	Wood preservatives (containing arsenic and chromate)	09/89		100.	6

1210	Xylenes (mixed xylenes)		200.	1 2	6
	including:				
108383	m-Xylene	06/91	200.	1 2	
95476	o-Xylene	06/91	200.	1 2	
106423	p-Xylene	06/91	200.	1 2	
7440666	Zinc		2.	2	
*	Zinc compounds	09/89	2.	1 2	
[7]					
	including but not limited to:				
1314132	Zinc oxide		2.	2	
[7]					

Appendix A-II

Substances For Which Production, Use,

Or Other Presence Must Be Reported

July 1, 1997

APPENDIX A-II
Substances For Which Production, Use, Or Other Presence Must Be Reported

Emittent ID (Note [1])	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Source List(s) (Note [6])	Other Notes(s)
26148685	A-alpha-C {2-Amino-9H-pyrido[2,3-b]indole}	09/89	c	3 4	[18]
34256821	Acetochlor	09/89	c	4	
62476599	Acifluorfen [POM]	09/90	c	1 2 4	
3688537	AF-2		c	3 4	
1000	Aflatoxins		c	3 4 5	
15972608	Alachlor	09/89	c	4	
309002	Aldrin	09/89	c	4	
107186	Allyl alcohol	06/91		7	
60093	p-Aminoazobenzene {4-Aminoazobenzene} [POM]		c	1 2 3 4	
97563	o-Aminoazotoluene [POM]		c	1 2 3 4 5	
6109973	3-Amino-9-ethylcarbazole hydrochloride [POM]	09/89	c	1 2 4 5	
125848	Aminoglutethimide	09/90		4	
82280	1-Amino-2-methylanthraquinone [PAH-Derivative, POM]		c	1 2 4 5	
68006837	2-Amino-3-methyl-9H-pyrido(2,3-b) indole {MeA-alpha-C}	09/89	c	3 4	
712685	2-Amino-5-(5-nitro-2-furyl)-1,3,4-thiadiazole		c	3 4	
-	2-Amino-9H-pyrido(2,3-b)indole (see A-alpha-C)				
134292	o-Anisidine hydrochloride		c	4 5	
104949	p-Anisidine	06/91		7	
140578	Aramite		c	3 4	
492808	Auramine [POM]		c	1 2 3 4 5	
446866	Azathioprine		c	3 4 5	
103333	Azobenzene [POM]	09/90	c	1 2 4	
98873	Benzal chloride	06/91		7	
55210	Benzamide	06/91		7	
1694093	Benzyl violet 4B [POM]		c	1 2 3 4	
1025	Betel quid with tobacco		c	3 4	
494031	N-N-Bis(2-chloroethyl)-2-naphthylamine {Chlornaphazine} [PAH-Derivative, POM]		c	1 2 3 4 5	
108601	Bis(2-chloro-1-methylethyl) ether	06/91		7	
1030	Bitumens, extracts of steam-refined and air-refined bitumens		c	3 4	
1035	Bleomycins		c	3	
75274	Bromodichloromethane	09/90	c	4	
1689845	Bromoxynil	06/91		4	
25013165	Butylated hydroxyanisole {BHA}		c	3 4	
123728	Butyraldehyde	06/91		7	
3068880	beta-Butyrolactone		c	3 4	
630080	Carbon monoxide	09/89		4	
143500	Chlordecone {Kepone}		c	3 4	
6164983	Chlordimeform	09/89	c	4	
115286	Chlorendic acid	09/89	c	3 4 5	
124481	Chlorodibromomethane	09/90	c	4	

563473	3-Chloro-2-methylpropene	09/89	c		4 5
1065	Chlorophenoxy herbicides		c	3	
1897456	Chlorothalonil	09/89	c		4
1059	p-Chloro-o-toluidine (strong acid salts)	06/91	c	3	

APPENDIX A-II
Substances For Which Production, Use, Or Other Presence Must Be Reported

Emittent ID (Note [1])	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Source List(s) (Note [6])	Other Notes(s)
4680788	C. I. Acid Green 3 [POM]	06/91		1 2 7	
569642	C. I. Basic Green 4 [POM]	06/91		1 2 7	
989388	C. I. Basic Red 1 [POM]	06/91		1 2 7	
569619	C. I. Basic Red 9 monohydrochloride [POM]	09/89	c	1 2 4 5	
2832408	C. I. Disperse Yellow 3 [POM] (NOTE: "C. I." means "color index")	06/91		1 2 7	
87296	Cinnamyl anthranilate [POM]	09/89	c	1 2 4 5	
6358538	Citrus Red No. 2 [POM]		c	1 2 3 4	
8007452	Coal tars	09/89	c	3 4 5	
21725462	Cyanazine	09/90		4	
14901087	Cycasin		c	3 4	
13121705	Cyhexatin	09/89		4	
3468631	D and C Orange No. 17 [PAH-Derivative, POM]	09/90	c	1 2 4	
81889	D and C Red No. 19 [POM]	09/90	c	1 2 4	
2092560	D and C Red No. 8 [PAH-Derivative, POM]	06/91	c	1 2 4	
5160021	D and C Red No. 9 [PAH-Derivative, POM]	09/90	c	1 2 4	
1596845	Daminozide	09/90	c	4	
50293	DDT {1,1,1-Trichloro-2,2-bis(p-chlorophenyl)ethane} [POM]		c	1 2 3 4 5	
613354	N,N'-Diacetylbenzidine [POM]		c	1 2 3 4	
2303164	Diallate	06/91		7	
39156417	2,4-Diaminoanisole sulfate		c	4 5	
101804	4,4'-Diaminodiphenyl ether [POM]		c	1 2 3 4 5	
764410	1,4-Dichloro-2-butene	09/90	c	4	
28434868	3,3'-Dichloro-4,4'-diaminodiphenyl ether [POM]	09/89	c	1 2 3 4	
72548	Dichlorodiphenyldichloroethane {DDD} [POM]	09/89	c	1 2 4	
540590	1,2-Dichloroethylene	06/91		7	
78886	2,3-Dichloropropene	06/91		7	
60571	Dieldrin	09/89	c	4	
1464535	Diepoxybutane		c	3 4 5	
1615801	1,2-Diethylhydrazine		c	3 4	
84662	Diethyl phthalate	06/91		7	
101906	Diglycidyl resorcinol ether {DGRE}		c	3 4 5	
94586	Dihydrosafrole		c	3 4	
20325400	3,3'-Dimethoxybenzidine dihydrochloride [POM]	06/91	c	1 2 4	
55738540	trans-2-[(Dimethylamino)methylimino]-5-[2-(5-nitro-2-furyl)vinyl-1,3,4-oxadiazol		c	3 4	
540738	1,2-Dimethylhydrazine		c	3 4	
105679	2,4-Dimethylphenol {2,4-Xylenol}	06/91		7	
513371	Dimethylvinylchloride {DMVC}	09/89	c	4 5	
25154545	Dinitrobenzenes (mixtures of) including:	09/90		4 7	
99650	m-Dinitrobenzene	06/91		7	
528290	o-Dinitrobenzene	06/91		7	

100254	p-Dinitrobenzene	06/91		7
39300453	Dinocap	09/90	4	
88857	Dinoseb	09/89	4	
117840	n-Dioctyl phthalate	06/91		7

APPENDIX A-II
Substances For Which Production, Use, Or Other Presence Must Be Reported

Emittent ID (Note [1])	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Source List(s) (Note [6])	Other Notes(s)
2475458	Disperse Blue 1 [PAH-Derivative, POM]	06/91	c	1 2 3 4	
541413	Ethyl chloroformate	06/91		7	
62500	Ethyl methanesulfonate		c	3 4	
2164172	Fluometuron	06/91		7	
133073	Folpet	09/89	c	4	
3570750	2-(2-Formylhydrazino)-4-(5-nitro-2-furyl)thiazole		c	3 4	
60568050	Furmecyclox	09/90	c	4	
67730114	Glu-P-1 {2-Amino-6-methyldipyrido[1,2-a:3',2'-d]imidazole}		c	3 4	
67730103	Glu-P-2 {2-Aminodipyrido[1,2-a:3',2'-d]imidazole}		c	3 4	
765344	Glycidaldehyde		c	3 4	
556525	Glycidol	09/90	c	4	
16568028	Gyromitrin {Acetaldehyde methylformylhydrazone}		c	4	
2784943	HC Blue 1	09/89	c	4 5	
1024573	Heptachlor epoxide	09/89	c	4	
1335871	Hexachloronaphthalene [PAH-Derivative, POM]	06/91		1 2 7	
10034932	Hydrazine sulfate		c	4 5	
76180966	IQ {2-Amino-3-methylimidazo[4,5-f]quinoline}		c	3 4	
78842	Isobutyraldehyde	06/91		7	
120581	Isosafrole	09/90	c	4	
4759482	Isotretinoin			4	
77501634	Lactofen [POM]	09/89	c	1 2 4	
1131	Lubricant base oils and derived products, specifically vacuum distillates, acid treated oils, aromatic oils, mildly solvent-refined oils, mildly hydrotreated-oils and used engine oils.	09/89	c	3 4 5	
8018017	Mancozeb	09/90	c	4	
12427382	Maneb	09/90	c	4	
59052	Methotrexate	09/89		4	
96333	Methyl acrylate	06/91		7	
590965	Methylazoxymethanol	09/90	c	4	
592621	Methylazoxymethanol acetate	09/89	c	3 4	
101611	4,4'-Methylene bis (N,N-dimethyl) benzenamine [POM]		c	1 2 4 5	
838880	4,4'-Methylene bis(2-methylaniline) [POM]	09/89	c	1 2 3 4	
74953	Methylene bromide	06/91		7	
66273	Methyl methanesulfonate		c	3 4	
129157	2-Methyl-1-nitroanthraquinone (uncertain purity) [PAH-Derivative, POM]		c	1 2 3 4	
70257	N-Methyl-N'-nitro-N-nitrosoguanidine		c	3 4	
-	N-Methyl-N-nitrosourethane (see N-Nitroso-N-methylurethane)				
924425	N-Methyloacrylamide	09/90	c	4	

9006422	Metiram	09/90			4
1140	Mineral oils (untreated and mildly treated oils; and those used in occupations such as mulespinning, metal machining, and jute processing).		c	3	4 5

APPENDIX A-II
Substances For Which Production, Use, Or Other Presence Must Be Reported

Emittent ID (Note [1])	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Source List(s) (Note [6])	Other Notes(s)
2385855	Mirex		c	3 4 5	
315220	Monocrotaline		c	3 4	
505602	Mustard gas {Sulfur mustard}		c	3 4 5	
134327	1-Naphthylamine [PAH-Derivative, POM]	09/90	c	1 2 4	
91598	2-Naphthylamine [PAH-Derivative, POM]		c	1 2 3 4 5	
54115	Nicotine	09/90		4	
1148	Nitrilotriacetic acid (salts) including but not limited to:	06/91	c	3	
18662538	Nitrilotriacetic acid, trisodium salt monohydrate	06/91	c	4	
602879	5-Nitroacenaphthene [PAH-Derivative, POM]		c	1 2 3 4	
99592	5-Nitro-o-anisidine		c	4 5	
1836755	Nitrofen (technical grade)		c	3 4 5	
51752	Nitrogen mustard {Mechlorethamine}		c	3 4 5	
55867	Nitrogen mustard hydrochloride	09/89	c	4 5	
55630	Nitroglycerin	06/91			7
88755	2-Nitrophenol	06/91			7
57835924	4-Nitropyrene [PAH-Derivative, POM]	06/91	c	1 2 3 4	
86306	N-Nitrosodiphenylamine [POM]	09/89	c	1 2 4	
759739	N-Nitroso-N-ethylurea		c	4 5	
60153493	3-(N-Nitrosomethylamino)propionitrile	09/89	c	3 4	
64091914	4-(N-Nitrosomethylamino)-1-(3-pyridyl)-1-butanone {NNK}	09/89	c	3 4	
615532	N-Nitroso-N-methylurethane {N-Methyl-N-nitrosourethane}		c	3 4	
4549400	N-Nitrosomethylvinylamine		c	3 4 5	
16543558	N-Nitrosonornicotine		c	3 4 5	
13256229	N-Nitrososarcosine		c	3 4 5	
303479	Ochratoxin A [POM]	09/90	c	1 2 4	
2234131	Octachloronaphthalene [PAH-Derivative, POM]	06/91		1 2	7
2646175	Oil Orange SS [PAH-Derivative, POM]		c	1 2 3 4	
20816120	Osmium tetroxide	06/91			7
794934	Panfuran S {Dihydroxymethylfuratrizine}		c	3 4	
122601	Phenyl glycidyl ether	09/90	c	3 4	
57410	Phenytoin [POM]		c	1 2 3 4 5	
88891	Picric acid	06/91			7
1155	Polybrominated biphenyls {PBBs} [POM]		c	1 2 3 4 5	
53973981	Polygeenan	09/89	c	4	
3761533	Ponceau MX [PAH-Derivative, POM]		c	1 2 3 4	
3564098	Ponceau 3R [PAH-Derivative, POM]		c	1 2 3 4	
36791045	Ribavirin	09/90		4	
94597	Safrole		c	3 4 5	
1180	Shale oils		c	3 4	
132274	Sodium o-phenylphenate [POM]		c	1 2 3 4	

128449	Sodium saccharin	09/89	c		4
1185	Soots		c	3	4
10048132	Sterigmatocystin [POM]		c	1 2 3	4

APPENDIX A-II

Substances For Which Production, Use, Or Other Presence Must Be Reported

Emittent ID (Note [1])	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Source List(s) (Note [6])	Other Notes(s)
95067	Sulfallate		c	3 4 5	
5216251	p-alpha,alpha,alpha-Tetrachlorotoluene	09/90	c	4	
961115	Tetrachlorvinphos	06/91		7	
509148	Tetranitromethane	09/90	c	4	
139651	4,4'-Thiodianiline [POM]		c	1 2 3 4	
1314201	Thorium dioxide		c	4 5	
1200	Tobacco products, smokeless		c	3 4	
1205	alpha-chlorinated Toluenes		c	3	
636215	o-Toluidine hydrochloride		c	4 5	
106490	p-Toluidine	09/90	c	4	
52686	Trichlorfon	06/91		7	
68768	Tris(aziridinyl)-p-benzoquinone {Triaziquone}	09/90	c	4	
52244	Tris(1-aziridinyl) phosphine sulfide {Thiotepa}		c	3 4 5	
126727	Tris(2,3-dibromopropyl)phosphate	09/89	c	4	
62450060	Trp-P-1 {3-Amino-1,4-dimethyl-5H-pyrido[4,3-b]indole}		c	3 4	
62450071	Trp-P-2 {3-Amino-1-methyl-5H-pyrido[4,3-b]indole}		c	3 4	
72571	Trypan blue [PAH-Derivative, POM]		c	1 2 3 4	
106876	4-Vinyl-1-cyclohexene diepoxide {Vinyl cyclohexene dioxide}	09/90	c	4	
81812	Warfarin [POM]			1 2 4	
87627	2,6-Xylidene	06/91		4	
12122677	Zineb	09/90	c	4	

Appendix A-III

Substances Which Need Not Be Reported

Unless Manufactured By The Facility

July 1, 1997

The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments. September 2002.

APPENDIX A-III
Substances Which Need Not Be Reported Unless Manufactured By the Facility

Emittent ID (Note [1])	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Source List(s) (Note [6])	Other Notes(s)
546883	Acetohydroxamic acid	09/90		4	
50760	Actinomycin D	09/90	c	4	
23214928	Adriamycin [PAH-Derivative, POM]		c	1 2 3 4 5	
28981977	Alprazolam [POM]	09/90		1 2 4	
39831555	Amikacin sulfate	09/90		4	
54626	Aminopterin			4	
1005	Analgesic mixtures containing phenacetin		c	3 4 5	
1010	Androgenic (anabolic) steriods		c	3 4	
	including but not limited to:				
58184	Methyltestosterone	09/90		4	
434071	Oxymetholone		c	4 5	
58220	Testosterone and its esters	09/89		4	
	including but not limited to:				
315377	Testosterone enanthate	09/90		4	
50782	Aspirin	06/91		4	
115026	Azaserine		c	3 4	
5411223	Benzphetamine hydrochloride [POM]	09/90		1 2 4	
154938	Bischloroethyl nitrosourea		c	3 4	
55981	1,4-Butanediol dimethanesulfonate {Busulfen/ Myleran}		c	3 4 5	
41575944	Carboplatin	09/90		4	
474259	Chenodiol	09/90		4	
305033	Chlorambucil		c	3 4 5	
56757	Chloramphenicol		c	3 4	
1620219	Chlorcyclizine hydrochloride [POM]			1 2 4	
13010474	1-(2-Chloroethyl)-3-cyclohexyl-1-nitrosourea {CCNU}		c	3 4 5	
13909096	1-(2-Chloroethyl)-3-(4-methylcyclohexyl)-1-nitrosourea {Methyl CCNU}		c	3	
15663271	Cisplatin		c	3 4	
50419	Clomiphene citrate [POM]	09/90		1 2 4	
50180	Cyclophosphamide		c	3 4	
147944	Cytarabine	09/89		4	
4342034	Dacarbazine		c	3 4 5	
17230885	Danazol	09/90		4	
20830813	Daunomycin [PAH-Derivative, POM]		c	1 2 3 4	
23541506	Daunorubicin hydrochloride [PAH-Derivative, POM]	09/90		1 2 4	
84173	Dienestrol [POM]	09/90	c	1 2 4	
564250	Doxycycline	09/90		4	
379793	Ergotamine tartrate [POM]	09/90		1 2 4	
1095	Estrogens, non-steroidal		c	3 5	
	including but not limited to:				
56531	Diethylstilbestrol [POM]		c	1 2 3 4 5	
1100	Estrogens, steroidal		c	3 5	

	including but not limited to:			
1068	Conjugated estrogens	09/90	c	4
50282	Estradiol 17 beta		c	4 5
53167	Estrone		c	4 5

APPENDIX A-III
Substances Which Need Not Be Reported Unless Manufactured By the Facility

Emittent ID (Note [1])	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Source List(s) (Note [6])	Other Notes(s)
57636	Ethinyl estradiol		c	4 5	
72333	Mestranol		c	3 4 5	
33419420	Etoposide [POM]	09/90		2	
54350480	Etretinate			4	
51218	Fluorouracil	09/89		4	
76437	Fluoxymesterone	09/90		4	
13311847	Flutamide	09/90		4	
67458	Furazolidone	09/90	c	4	
126078	Griseofulvin		c	3 4	
23092173	Halazepam [POM]	09/90		1 2 4	
3778732	Ifosfamide	09/90		4	
9004664	Iron dextran complex		c	3 4 5	
303344	Lasiocarpine	09/89	c	3 4	
554132	Lithium carbonate	06/91		4	
919164	Lithium citrate	06/91		4	
846491	Lorazepam [POM]	09/90		1 2 4	
595335	Megestrol acetate	06/91		4	
148823	Melphalan		c	3 4 5	
9002680	Menotropins	09/90		4	
6112761	Mercaptopurine	09/90		4	
531760	Merphalan	09/89	c	4	
3963959	Methacycline hydrochloride	06/91		4	
60560	Methimazole	09/90		4	
15475566	Methotrexate sodium	09/90		4	
484208	5-Methoxypsoralen		c	3	
56042	Methylthiouracil		c	3 4	
443481	Metronidazole		c	3 4 5	
59467968	Midazolam hydrochloride [POM]	09/90		1 2 4	
62015398	Misoprostol	09/90		4	
50077	Mitomycin C		c	3 4	
70476823	Mitoxantrone hydrochloride [PAH-Derivative, POM]	09/90		1 2 4	
139913	5-(Morpholinomethyl)-3-[(5-nitrofurfurylidene)amino]-2-oxazolidinone		c	3 4	
86220420	Nafarelin acetate [PAH-Derivative, POM]	09/90		1 2 4	
3771195	Nafenopin [POM]		c	1 2 3 4	
1405103	Neomycin sulfate	09/90		4	
56391572	Netilmicin sulfate	09/90		4	
61574	Niridazole		c	3 4	
67209	Nitrofurantoin	06/91	c	4	
59870	Nitrofurazone	09/90	c	4	
555840	1-[(5-Nitrofurfurylidene)amino]-2-imidazolidinone		c	3 4	
531828	N-[4-(5-Nitro-2-furyl)-2-thiazolyl]acetamide		c	3 4	
6533002	Norgestrel	09/90		4	
79572	Oxytetracycline	06/91		4	

115673	Paramethadione	09/90	4
52675	Penicillamine	06/91	4

APPENDIX A-III
Substances Which Need Not Be Reported Unless Manufactured By the Facility

Emittent ID (Note [1])	Substance Name (Note [2])	Add Date (Note [3])	Carcinogen (Note [4])	Source List(s) (Note [6])	Other Notes(s)
57330	Pentobarbital sodium	09/90		4	
63989	Phenacemide	09/90		4	
62442	Phenacetin		c	3 4 5	
94780	Phenazopyridine hydrochloride		c	3 4 5	
3546109	Phenesterin	09/89	c	4 5	
50066	Phenobarbital		c	3 4	
59961	Phenoxybenzamine [POM]	09/89	c	1 2 4	
63923	Phenoxybenzamine hydrochloride [POM]	09/90	c	1 2 3 4 5	
54911	Pipobroman	09/90		4	
18378897	Plicamycin [PAH-Derivative, POM]	09/90		1 2 4	
366701	Procarbazine hydrochloride		c	3 4 5	
57830	Progesterone		c	3 4 5	
1160	Progestins		c	3	
	including but not limited to:				
71589	Medroxyprogesterone acetate		c	3 4	
68224	Norethisterone		c	4 5	
51525	Propylthiouracil		c	3 4 5	
302794	all-trans-Retinoic acid	09/89		4	
1167	Retinol/retinyl esters	09/89	c	4	
81072	Saccharin		c	3 4 5	
3810740	Streptomycin sulfate	06/91		4	
18883664	Streptozotocin		c	3 4 5	
54965241	Tamoxifen citrate [POM]	09/90		1 2 4	
846504	Temazepam [POM]	09/90		1 2 4	
64755	Tetracycline hydrochloride	06/91		4	
50351	Thalidomide			4	
154427	Thioguanine	09/90		4	
49842071	Tobramycin sulfate	09/90		4	
299752	Treosulfan		c	3 4	
28911015	Triazolam [POM]	09/90		1 2 4	
13647353	Trilostane	09/90		4	
127480	Trimethadione	06/91		4	
66751	Uracil mustard		c	3 4	
26995915	Urofollitropin	09/90		4	
99661	Valproate			4	
143679	Vinblastine sulfate [POM]	09/90		1 2 4	
2068782	Vincristine sulfate [POM]	09/90		1 2 4	

NOTES TO APPENDIX A

Note **Text of Note**

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[1] Emittent ID (the emittent identification number) is the Chemical Abstract Service (CAS) number where available, or an ARB-assigned 4-digit emittent ID code.

A dash ("-") is shown for the Emittent ID for substances which are alphabetized under a group header or synonym elsewhere on the list. Refer to the cross reference indicated in parenthesis, "( )".

A double dash ("- -") is shown for the Emittent ID to indicate that the entry is a non-reportable group header for the substances immediately following it.

An asterisk ("*") is shown for the Emittent ID to indicate that the emissions of unspecified metal compounds shall be reported as the metal atom equivalent. See Note [7].

A pound sign ("#") is shown for the Emittent ID to indicate that the individual, component listed substances must be reported for this mixture or group.

[2] Individual substances listed under a group heading must be reported individually. Other, unspecified substances in the group must be summed and reported using the emittent ID of the group heading.

The square bracket designation, "[ ]", indicates that the substance is a component of the chemical group heading(s) within the brackets.

The braces designation, "{ }", indicates a synonym for the substance listed.

[3] The date the Board approved addition of the substance to the original list. The original list was approved by the Board in July 1988.

[4] The letter "c" indicates that for purposes of this section the substance shall be treated as a human carcinogen or potential human carcinogen.

[5] Applicable degree of accuracy (in lbs/year except where noted). Radionuclides must be reported in Curie units, and the accuracy must be considered accordingly. Refer to Section VII.E. and Appendix B.

Note	Text of Note

[6] Substances are required to be included on the Hot Spots list based on the following lists cited in Health & Safety Code Section 44321:

1 = California Air Resources Board (44321(c));
2 = Environmental Protection Agency (44321(e));
3 = International Agency for Research on Cancer; (44321(a); Labor Code section 6382(b)(1))
4 = Governor's List of Carcinogens and Reproductive Toxicants; (44321(b); HSC Section 25249.8)
5 = National Toxicology Program (44321(a));
6 = Hazard Evaluation System and Information Service (44321(d))
7 = Added pursuant to HSC Section 44321 (f).

[7] Emissions of unspecified metal compounds shall be reported as the amount of the metal atom equivalent, using the metal emittent identification number for the metal itself (or the emittent identification number indicated on the table, such as for reporting inorganic versus other-than-inorganic arsenic compounds).

For unspecified metal compounds which contain two or more listed metals (e.g., zinc chromate), each component metal shall be reported as the amount of the appropriate metal atom equivalent (i.e., the zinc portion of the weight as zinc equivalent and the chromate portion as hexavalent chromium equivalent).

For specific, individually listed metal compounds (e.g., Lead chromate), emissions shall be reported for the compound (as pounds of whole compound), using the emittent identification number for that compound.

[8] Compounds of the form "X-CN", where formal dissociation can occur. Report as the amount of Cyanide equivalent in the compound using an emittent identification code of 1073.

[9] Emissions of these mixtures shall be reported as emissions of total particulate matter and total organic gas, using the following emittent identification numbers:

9901 Diesel exhaust, particulate matter
9902 Diesel exhaust, total organic gas
9910 Gasoline exhaust, particulate matter
9911 Gasoline exhaust, total organic gas

Individually listed substances from diesel and gasoline exhaust must also be reported.

Note	Text of Note
[10]	The emittent identification number 1105 has been discontinued for all facilities reporting for the first time and for all updates. Use the listed replacement emittent identification codes 1103 and 1104.
[11]	Emissions of the individual, component listed substances must be reported in addition to the total gasoline vapors emissions.
[12]	These lead compounds are listed here so that the inorganic lead fraction will be quantified and reported if these individual compounds cannot be quantified.
[13]	PAH: (Polycyclic Aromatic Hydrocarbon) - An organic compound consisting of a fused ring structure containing at least two (2) benzene rings, and which may also contain additional fused rings not restricted exclusively to hexagonal rings. The structure does not include any heteroatoms or substituent groups. The structure includes only carbon and hydrogen. PAHs are a subgroup of POM and have a boiling point of greater than or equal to 100 C.
[14]	PAH-DERIVATIVE: (Polycyclic Aromatic Hydrocarbon Derivative) - An organic compound consisting of a fused ring structure containing at least two (2) benzene rings, and which may also contain additional fused rings not restricted exc lusively to hexagonal rings. The fused ring structure does not contain heteroatoms. The structure does contain one or more substituent groups. PAH-Derivatives are a subgroup of POM and have a boiling point of greater than or equal to 100 C.
[15]	POM: (Polycyclic Organic Matter) - Includes organic compounds with more than one benzene ring, and which have a boiling point of greater than or equal to 100 C.
[16]	Radionuclides and other radioactive substances shall be reported in units of Curies per year (for annual average emissions) and in units of milliCuries per hour (for maximum hourly emissions).
[17]	Emissions of Vanadium (fume or dust) shall be reported as the amount of the vanadium atom equivalent, using the identification number 7440622.
[18]	The emittent identification number 1001 has been replaced with the CAS number 26148685.

Appendix B

Health And Safety Code Related to the

Air Toxics Hot Spots Program

Appendix B

Health And Safety Code Related To The Air Toxics Hot Spots Program[1]

PART 6. AIR TOXICS "HOT SPOTS" INFORMATION AND ASSESSMENT
(Part 6 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384. Note: Sections 44380 and 44384 became operative Jan. 1, 1988.)

CHAPTER 1. LEGISLATIVE FINDINGS AND DEFINITIONS
(Chapter 1 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44300. This part shall be known and may be cited as the Air Toxics "Hot Spots" Information and Assessment Act of 1987. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44301. The Legislature finds and declares all of the following:

(a) In the wake of recent publicity surrounding planned and unplanned releases of toxic chemicals into the atmosphere, the public has become increasingly concerned about toxics in the air.

(b) The Congressional Research Service of the Library of Congress has concluded that 75 percent of the United States population lives in proximity to at least one facility that manufactures chemicals. An incomplete 1985 survey of large chemical companies conducted by the Congressional Research Service documented that nearly every chemical plant studied routinely releases into the surrounding air significant levels of substances proven to be or potentially hazardous to public health.

(c) Generalized emissions inventories compiled by air pollution control districts and air quality management districts in California confirm the findings of the Congressional Research Service survey as well as reveal that many other facilities and businesses which do not actually manufacture chemicals do use hazardous substances in sufficient quantities to expose, or in a manner that exposes, surrounding populations to toxic air releases.

(d) These releases may create localized concentrations or air toxics "hot spots" where emissions from specific sources may expose individuals and population groups to elevated risks of adverse health effects, including, but not limited to, cancer and contribute to the cumulative health risks of emissions from other sources in the area. In some cases where large populations may not be significantly affected by adverse health risks, individuals may be exposed to significant risks.

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[1] *AB564 Passed in the 1996 legislative session. The text will be added when the code is revised.*

(e) Little data is currently available to accurately assess the amounts, types, and health impacts of routine toxic chemical releases into the air. As a result, there exists significant uncertainty about the amounts of potentially hazardous air pollutants which are released, the location of those releases, and the concentrations to which the public is exposed.
(f) The State of California has begun to implement a long-term program to identify, assess, and control ambient levels of hazardous air pollutants, but additional legislation is needed to provide for the collection and evaluation of information concerning the amounts, exposures, and short- and long-term health effects of hazardous substances regularly released to the surrounding atmosphere from specific sources of hazardous releases.
(g) In order to more effectively implement control strategies for those materials posing an unacceptable risk to the public health, additional information on the sources of potentially hazardous air pollutants is necessary.
(h) It is in the public interest to ascertain and measure the amounts and types of hazardous releases and potentially hazardous releases from specific sources that may be exposing people to those releases, and to assess the health risks to those who are exposed. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44302. The definitions set forth in this chapter govern the construction of this part. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44303. "Air release" or "release" means any activity that may cause the issuance of air contaminants, including the actual or potential spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing of a substance into the ambient air and that results from the routine operation of a facility or that is predictable, including, but not limited to, continuous and intermittent releases and predictable process upsets or leaks. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44304. "Facility" means every structure, appurtenance, installation, and improvement on land which is associated with a source of air releases or potential air releases of a hazardous material. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44306. "Health risk assessment" means a detailed comprehensive analysis prepared pursuant to Section 44361 to evaluate and predict the dispersion of hazardous substances in the environment and the potential for exposure of human populations and to assess and quantify both the individual and population wide health risks associated with those levels of exposure. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44307. "Operator" means the person who owns or operates a facility or part of a facility. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44308. "Plan" means the emissions inventory plan which meets the conditions specified in Section 44342. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44309. "Report" means the emissions inventory report specified in Section 44341. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

CHAPTER 2. FACILITIES SUBJECT TO THIS PART

(Chapter 2 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44320. This part applies to the following:

(a) Any facility which manufactures, formulates, uses, or releases any of the substances listed pursuant to Section 44321 or any other substance which reacts to form a substance listed in Section 44321 and which releases or has the potential to release total organic gases, particulates, or oxides of nitrogen or sulfur in the amounts specified in Section 44322.

(b) Except as provided in Section 44323, any facility which is listed in any current toxics use or toxics air emission survey, inventory, or report released or compiled by a district. A district may, with the concurrence of the state board, waive the application of this part pursuant to this subdivision for any facility which the district determines will not release any substance listed pursuant to Section 44321 due to a shutdown or a process change. (Amended by Stats. 1989, Ch. 1254, Sec. 7). References at the time of publication (see page iii): Regulations: 17, CCR, sections 90700-90703, 90704, 93303, 93306

44321. For the purposes of Section 44320, the state board shall compile and maintain a list of substances that contains, but is not limited to, all of the following:

(a) Substances identified by reference in paragraph (1) of subdivision (b) of Section 6382 of the Labor Code and substances placed on the list prepared by the National Toxicology Program issued by the United States Secretary of Health and Human Services pursuant to paragraph (4) of Section 262 of Public Law 95-622 of 1978. For the purposes of this subdivision, the state board may remove from the list any substance which meets both of the following criteria:
   (1) No evidence exists that it has been detected in air.
   (2) The substance is not manufactured or used in California, or, if manufactured or used in California, because of the physical or chemical characteristics of the substance or the manner in which it is manufactured or used, there is no possibility that it will become airborne.

(b) Carcinogens and reproductive toxins referenced in or compiled pursuant to Section 25249.8, except those which meet both of the criteria identified in subdivision (a).

(c) The candidate list of potential toxic air contaminants and the list of designated toxic air contaminants prepared by the state board pursuant to Article 2 (commencing with Section 39660) of Chapter 3.5 of Part 2, including, but not limited to, all substances currently under review and scheduled or nominated for review and substances identified and listed for which health effects information is limited.

(d) Substances for which an information or hazard alert has been issued by the repository of current data established pursuant to Section 147.2 of the Labor Code.
(e) Substances reviewed, under review, or scheduled for review as air toxics or potential air toxics by the Office of Air Quality Planning and Standards of the Environmental Protection Agency, including substances evaluated in all of the following categories or their equivalent: preliminary health and source screening, detailed assessment, intent to list, decision not to regulate, listed, standard proposed, and standard promulgated.
(f) Any additional substances recognized by the state board as presenting a chronic or acute threat to public health when present in the ambient air, including, but not limited to, any neurotoxins or chronic respiratory toxins not included within subdivision (a), (b), (c), (d), or (e). (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44322. This part applies to facilities specified in subdivision (a) of Section 44320 in accordance with the following schedule:
(a) For those facilities that release, or have the potential to release, 25 tons per year or greater of total organic gases, particulates, or oxides of nitrogen or sulfur, this part becomes effective on July 1, 1988.
(b) For those facilities that release, or have the potential to release, more than 10 but less than 25 tons per year of total organic gases, particulates, or oxides of nitrogen or sulfur, this part becomes effective July 1, 1989.
(c) For those facilities that release, or have the potential to release, less than 10 tons per year of total organic gases, particulates, or oxides of nitrogen or sulfur, the state board shall, on or before July 1, 1990, prepare and submit a report to the Legislature identifying the classes of those facilities to be included in this part and specifying a timetable for their inclusion. (Amended by Stats. 1989, Ch. 1254, Sec. 8.)

44323. A district may prepare an industrywide emissions inventory and health risk assessment for facilities specified in subdivision (b) of Section 44320 and subdivisions (a) and (b) of Section 44322, and shall prepare an industrywide emissions inventory for the facilities specified in subdivision (c) of Section 44322, in compliance with this part for any class of facilities that the district finds and determines meets all of the following conditions:
(a) All facilities in the class fall within one four-digit Standard Industrial Classification Code.
(b) Individual compliance with this part would impose severe economic hardships on the majority of the facilities within the class.
(c) The majority of the class is composed of small businesses.
(d) Releases from individual facilities in the class can easily and generically be characterized and calculated. (Amended by Stats. 1989, Ch. 1254, Sec. 9.)

44324. This part does not apply to any facility where economic poisons are employed in their pesticidal use, unless that facility was subject to district permit requirements on or before August 1,1987. As used in this section, "pesticidal use" does not include the manufacture or formulation of pesticides. (Added by Stats. 1981, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44325. Any solid waste disposal facility in compliance with Section 41805.5 is in compliance with the emissions inventory requirements of this part. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

CHAPTER 3. AIR TOXICS EMISSION INVENTORIES
(Chapter 3 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44340. (a) The operator of each facility subject to this part shall prepare and submit to the district a proposed comprehensive emissions inventory plan in accordance with the criteria and guidelines adopted by the state board pursuant to Section 44342.

(b) The proposed plan shall be submitted to the district on or before August 1, 1989, except that, for any facility to which subdivision (b) of Section 44322 applies, the proposed plan shall be submitted to the district on or before August 1,1990. The district shall approve, modify, and approve as modified, or return for revision and resubmission, the plan within 120 days of receipt.

(c) The district shall not approve a plan unless all of the following conditions are met:

(1) The plan meets the requirements established by the state board pursuant to Section 44342.

(2) The plan is designed to produce, from the list compiled and maintained pursuant to Section 44321, a comprehensive characterization of the full range of hazardous materials that are released, or that may be released, to the surrounding air from the facility. Air release data shall be collected at, or calculated for, the primary locations of actual and potential release for each hazardous material. Data shall be collected or calculated for all continuous, intermittent, and predictable air releases.

(3) The measurement technologies and estimation methods proposed provide state-of-the-art effectiveness and are sufficient to produce a true representation of the types and quantities of air releases from the facility.

(4) Source testing or other measurement techniques are employed wherever necessary to verify emission estimates, as determined by the state board and to the extent technologically feasible. All testing devices shall be appropriately located, as determined by the state board.

(5) Data are collected or calculated for the relevant exposure rate or rates of each hazardous material according to its characteristic toxicity and for the emission rate necessary to ensure a characterization of risk associated with exposure to releases of the hazardous material that meets the requirements of Section 44361. The source of all emissions shall be displayed or described. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44341. Within 180 days after approval of a plan by the district, the operator shall implement the plan and prepare and submit a report to the district in accordance with the plan. The district shall transmit all monitoring data contained in the approved report to the state board. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44342. The state board shall, on or before May 1, 1989, in consultation with the districts, develop criteria and guidelines for site-specific air toxics emissions inventory plans which shall be designed to comply with the conditions specified in Section 44340 and which shall include at least all of the following:

(a) For each class of facility, a designation of the hazardous materials for which emissions are to be quantified and an identification of the likely source types within that class of facility. The hazardous materials for quantification shall be chosen from among, and may include all or part of, the list specified in Section 44321.

(b) Requirements for a facility diagram identifying each actual or potential discrete emission point and the general locations where fugitive emissions may occur. The facility diagram shall include any nonpermitted and nonprocess sources of emissions and shall provide the necessary data to identify emission characteristics. An existing facility diagram which meets the requirements of this section may be submitted.

(c) Requirements for source testing and measurement. The guidelines may specify appropriate uses of estimation techniques including, but not limited to, emissions factors, modeling, mass balance analysis, and projections, except that source testing shall be required wherever necessary to verify emission estimates to the extent technologically feasible. The guidelines shall specify conditions and locations where source testing, fence-line monitoring, or other measurement techniques are to be required and the frequency of that testing and measurement.

(d) Appropriate testing methods, equipment, and procedures, including quality assurance criteria.

(e) Specifications for acceptable emissions factors, including, but not limited to, those which are acceptable for substantially similar facilities or equipment, and specification of procedures for other estimation techniques and for the appropriate use of available data.

(f) Specification of the reporting period required for each hazardous material for which emissions will be inventoried.

(g) Specifications for the collection of useful data to identify toxic air contaminants pursuant to Article 2 (commencing with Section 39660) of Chapter 3.5 of Part 2.

(h) Standardized format for preparation of reports and presentation of data.

(i) A program to coordinate and eliminate any possible overlap between the requirements of this chapter and the requirements of Section 313 of the Superfund Amendment and Reauthorization Act of 1986 (Public Law 99-499). The state board shall design the guidelines and criteria to ensure that, in collecting data to be used for emissions inventories, actual measurement is utilized whenever necessary to verify the accuracy of emission estimates, to the extent technologically feasible. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44343. The district shall review the reports submitted pursuant to Section 44341 and shall, within 90 days, review each report, obtain corrections and clarifications of the data, and notify the Office of Environmental Health Hazard Assessment, the Department of Industrial Relations, and the city or county health department of its findings and determinations as a result of its review of the report. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384. Amended by Governor's Reorganization Plan No. 1 of 1991, §142.)

44344. Except as provided in Section 44391, emissions inventories developed pursuant to this chapter shall be updated every four years, in accordance with the procedures established by the state board. Those updates shall take into consideration improvements in measurement techniques and advancing knowledge concerning the types and toxicity of hazardous material released or potentially released. (Amended by Stats. 1993, Ch. 1041, Sec. 1. Effective January 1, 1994.)

44344.3.

(a) A facility shall be granted an exemption by a district from further compliance with this part after meeting all of the following criteria:
    (1) The facility was required to comply with this part only as a result of its particulate matter emissions.
    (2) The facility has participated in, utilized data derived from, or is eligible to utilize data derived from, approved pooled source testing.
    (3) The facility has submitted an emissions inventory plan and report that was subsequently accepted and approved.
    (4) The facility has been designated by the district as a low priority facility under the guidelines set forth pursuant to this part for facility prioritization, and facility emissions do not present a significant health risk as specified in subdivision (b) of Section 44362.
    (5) The facility handles, processes, stores, or distributes bulk agricultural commodities or handles, feeds, or rears livestock. (b) Subdivision (a) does not apply to a facility that, because of information provided pursuant to Section 44344.7, is reclassified as an intermediate or high priority facility by the district.
(c) The operator of a facility that has been granted an exemption pursuant to this section shall biennially submit a statement to the district for the district's review, with a copy of the most recent emissions inventory for the facility, indicating that, except as to matters for which an emissions inventory update has been or will be submitted pursuant to Section 44344.7, there has been no significant change in facility operations or activities. The district shall not impose any fee upon the operator with regard to the submission of the statement. (Added by Stats. 1993, Ch. 1037, Sec. 1. Effective January 1, 1994.)

44344.5. The operator of any new facility that previously has not been subject to this part shall prepare and submit an emissions inventory plan and report. (Added by Stats. 1993, Ch. 1037, Sec. 2. Effective January 1, 1994.)

44344.7. The operator of a facility exempted pursuant to subdivision (a) of Section 44344.3 shall submit an emissions inventory update for those sources and substances for which a change in activities or operations has occurred, as follows:

(a) The facility emits a newly listed substance.
(b) A sensitive receptor has been established or constructed on or after January 1, 1994, within 500 meters of the facility.
(c) The facility emits a substance for which the potency factor has increased.

(d) The facility has begun emission of a listed substance not included in the previous emissions inventory. (Added by Stats. 1993, Ch. 1037, Sec. 3. Effective January 1, 1994.)

44345. (a) On or before July 1, 1989, the state board shall develop a program to compile and make available to other state and local public agencies and the public all data collected pursuant to this chapter.

(b) In addition, the state board, on or before March 1, 1990, shall compile, by district, emissions inventory data for mobile sources and area sources not subject to district permit requirements, and data on natural source emissions, and shall incorporate these data into data compiled and released pursuant to this chapter. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44346. (a) If an operator believes that any information required in the facility diagram specified pursuant to subdivision (b) of Section 44342 involves the release of a trade secret, the operator shall nevertheless make the disclosure to the district, and shall notify the district in writing of that belief in the report.

(b) Subject to this section, the district shall protect from disclosure any trade secret designated as such by the operator, if that trade secret is not a public record.

(c) Upon receipt of a request for the release of information to the public which includes information which the operator has notified the district is a trade secret and which is not a public record, the following procedure applies:

(1) The district shall notify the operator of the request in writing by certified mail, return receipt requested.

(2) The district shall release the information to the public, but not earlier than 30 days after the date of mailing the notice of the request for information, unless, prior to the expiration of the 30-day period, the operator obtains an action in an appropriate court for a declaratory judgment that the information is subject to protection under this section or for a preliminary injunction prohibiting disclosure of the information to the public and promptly notifies the district of that action.

(d) This section does not permit an operator to refuse to disclose the information required pursuant to this part to the district.

(e) Any information determined by a court to be a trade secret, and not a public record pursuant to this section, shall not be disclosed to anyone except an officer or employee of the district, the state, or the United States, in connection with the official duties of that officer or employee under any law for the protection of health, or to contractors with the district or the state and its employees if, in the opinion of the district or the state, disclosure is necessary and required for the satisfactory performance of a contract, for performance of work, or to protect the health and safety of the employees of the contractor.

(f) Any officer or employee of the district or former officer or employee who, by virtue of that employment or official position, has possession of, or has access to, any trade secret subject to this section, and who, knowing that disclosure of the information to the general public is prohibited by this section, knowingly and willfully discloses the information in any manner to any person not entitled to receive it is guilty of a

misdemeanor. Any contractor of the district and any employee of the contractor, who has been furnished information as authorized by this section, shall be considered an employee of the district for purposes of this section.

(g) Information certified by appropriate officials of the United States as necessary to be kept secret for national defense purposes shall be accorded the full protections against disclosure as specified by those officials or in accordance with the laws of the United States

(h) As used in this section, "trade secret" and "public record" have the meanings and protections given to them by Section 6254.7 of the Government Code and Section 1060 of the Evidence Code. All information collected pursuant to this chapter, except for data used to calculate emissions data required in the facility diagram, shall be considered "air pollution emission data," for the purposes of this section. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

CHAPTER 4. RISK ASSESSMENT

(Chapter 4 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44360. (a) Within 90 days of completion of the review of all emissions inventory data for facilities specified in subdivision (a) of Section 44322, but not later than December 1,1990, the district shall, based on examination of the emissions inventory data and in consultation with the state board and the State Department of Health Services, prioritize and then categorize those facilities for the purposes of health risk assessment. The district shall designate high, intermediate, and low priority categories and shall include each facility within the appropriate category based on its individual priority. In establishing priorities pursuant to this section, the district shall consider the potency, toxicity, quantity, and volume of hazardous materials released from the facility, the proximity of the facility to potential receptors, including, but not limited to, hospitals, schools, day care centers, worksites, and residences, and any other factors that the district finds and determines may indicate that the facility may pose a significant risk to receptors. The district shall hold a public hearing prior to the final establishment of priorities and categories pursuant to this section.

(b) (1) Within 150 days of the designation of priorities and categories pursuant to subdivision (a), the operator of every facility that has been included within the highest priority category shall prepare and submit to the district a health risk assessment pursuant to Section 44361. The district may, at its discretion, grant a 30-day extension for submittal of the health risk assessment.

(2) Health risk assessments required by this chapter shall be prepared in accordance with guidelines established by the Office of Environmental Health Hazard Assessment. The office shall prepare draft guidelines which shall be circulated to the public and the regulated community and shall adopt risk assessment guidelines after consulting with the state board and the Risk Assessment Committee of the California Air Pollution Control Officers Association and after conducting at least two public workshops, one in the northern and one in the southern part of the state. The adoption of the guidelines is not subject to

Chapter 3.5 (commencing with Section 11340) of Part 1 of Division 3 of Title 2 of the Government Code. The scientific review panel established pursuant to Section 39670 shall evaluate the guidelines adopted under this paragraph and shall recommend changes and additional criteria to reflect new scientific data or empirical studies.

(3) The guidelines established pursuant to paragraph (2) shall impose only those requirements on facilities subject to this subdivision that are necessary to ensure that a required risk assessment is accurate and complete and shall specify the type of site-specific factors that districts may take into account in determining when a single health risk assessment may be allowed under subdivision (d). The guidelines shall, in addition, allow the operator of a facility, at the operator's option, and to the extent that valid and reliable data are available, to include for consideration by the district in the health risk assessment any or all of the following supplemental information:

(a) Information concerning the scientific basis for selecting risk parameter values that are different than those required by the guidelines and the likelihood distributions that result when alternative values are used.

(b) Data from dispersion models, microenvironment characteristics, and population distributions that may be used to estimate maximum actual exposure.

(c) Risk expressions that show the likelihood that any given risk estimate is the correct risk value.

(d) A description of the incremental reductions in risk that occur when exposure is reduced.

(4) To ensure consistency in the use of the supplemental information authorized by subparagraphs (A), (B), (C),and (D) of paragraph (3),the guidelines established pursuant to paragraph (2) shall include guidance for use by the districts in considering the supplemental information when it is included in the health risk assessment. (c) Upon submission of emissions inventory data for facilities specified in subdivisions (b) and (c) of Section 44322, the district shall designate facilities for inclusion within the highest priority category, as appropriate, and any facility so designated shall be subject to subdivision (b). In addition, the district may require the operator of any facility to prepare and submit health risk assessments, in accordance with the priorities developed pursuant to subdivision (a).

(e) The district shall, except where site specific factors may affect the results, allow the use of a single health risk assessment for two or more substantially identical facilities operated by the same person.

(f) Nothing contained in this section, Section 44380.5, or Chapter 6 (commencing with Section 44390) shall be interpreted as requiring a facility operator to prepare a new or revised health risk assessment using the guidelines established pursuant to paragraph (2) of subdivision (a) of this section if the facility operator is required by the district to begin the preparation of a health risk assessment before those guidelines are established. (Amended by Stats. 1992, Ch. 1162, Sec. 1. Effective January 1, 1993.)

44361. (a) Each health risk assessment shall be submitted to the district. The district shall make he health risk assessment available for public review, upon request. After preliminary review of the emissions impact and modeling data, the district shall submit the health risk assessment to the Office of Environmental Health Hazard Assessment for review and, within 180 days of receiving the health risk assessment, the office shall submit to the district its comments on the data and findings relating to health effects. The district shall consult with the state board as necessary to adequately evaluate the emissions impact and modeling data contained within the risk assessment.

(b) For the purposes of complying with this section, the Office of Environmental Health Hazard Assessment may select a qualified independent contractor to review the data and findings relating to health effects. The office shall not select an independent contractor to review a specific health risk assessment who may have a conflict of interest with regard to the review of that health risk assessment. Any review by an independent contractor shall comply with the following requirements:
   (1) Be performed in a manner consistent with guidelines provided by the office.
   (2) Be reviewed by the office for accuracy and completeness.
   (3) Be submitted by the office to the district in accordance with this section.

(c) The district shall reimburse the Office of Environmental Health Hazard Assessment or the qualified independent contractor designated by the office pursuant to subdivision (b), within 45 days of its request, for its actual costs incurred in reviewing a health risk assessment pursuant to this section.

(d) If a district requests the Office of Environmental Health Hazard Assessment to consult with the district concerning any requirement of this part, the district shall reimburse the office, within 45 days of its request, for the costs incurred in the consultation.

(e) Upon designation of the high priority facilities, as specified in subdivision (a) of Section 44360, the Office of Environmental Health Hazard Assessment shall evaluate the staffing requirements of this section and may submit recommendations to the Legislature, as appropriate, concerning the maximum number of health risk assessments to be reviewed each year pursuant to this section. (Added by Stats. 1987, Ch. 1252, Sec. l. Operative July 1, 1988, pursuant to Section

44384. Amended by Governor's Reorganization Plan No. l of l991, §144.)

44362. (a) Taking the comments of the Office of Environmental Health Hazard Assessment into account, the district shall approve or return for revision and resubmission and then approve, the health risk assessment within 180 days of receipt. If the health risk assessment has not been revised and resubmitted within 60 days of the district's request of the operator to do so, the district may modify the health risk assessment and approve it as modified.

(b) Upon approval of the health risk assessment, the operator of the facility shall provide notice to all exposed persons regarding the results of the health risk assessment prepared pursuant to Section 44361 if, in the judgment of the district, the health risk assessment indicates there is a significant health risk associated with emissions from the facility. If notice is required under this subdivision, the notice shall include only

information concerning significant health risks attributable to the specific facility for which the notice is required. Any notice shall be made in accordance with procedures specified by the district. (Added by Stats. 1981, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384. Amended by Governor's Reorganization Plan No. 1 of 1991, 145.)

44363. (a) Commencing July 1, 1991, each district shall prepare and publish an annual report which does all of the following:
(1) Describes the priorities and categories designated pursuant to Section 44360 and summarizes the results and progress of the health risk assessment program undertaken pursuant to this part.
(2) Ranks and identifies facilities according to the degree of cancer risk posed both to individuals and to the exposed population.
(3) Identifies facilities which expose individuals or populations to any noncancer health risks.
(4) Describes the status of the development of control measures to reduce emissions of toxic air contaminants, if any.
(b) The district shall disseminate the annual report to county boards of supervisors, city councils, and local health officers and the district board shall hold one or more public hearings to present the report and discuss its content and significance. (Added by Stats. 1987, Ch. 1252, Sec. l. Operative July 1, 1988, pursuant to Section 44384.)

44364. The state board shall utilize the reports and assessments developed pursuant to this part for the purposes of identifying, establishing priorities for, and controlling toxic air contaminants pursuant to Chapter 3.5 (commencing with Section 39650) of Part 2. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44365. (a) If the state board finds and determines that a district's actions pursuant to this part do not meet the requirements of this part, the state board may exercise the authority of the district pursuant to this part to approve emissions inventory plans and require the preparation of health risk assessments.
(b) This part does not prevent any district from establishing more stringent criteria and requirements than are specified in this part for approval of emissions inventories and requiring the preparation and submission of health risk assessments. Nothing in this part limits the authority of a district under any other provision of law to assess and regulate releases of hazardous substances. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44366. (a) In order to verify the accuracy of any information submitted by facilities pursuant to this part, a district or the state board may proceed in accordance with Section 41510. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

CHAPTER 5. FEES AND REGULATIONS
(Chapter 5 added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44380. (a) The state board shall adopt a regulation which does all of the following:

(1) Sets forth the amount of revenue which the district must collect to recover the reasonable anticipated cost which will be incurred by the state board and the Office of Environmental Health Hazard Assessment to implement and administer this part.

(2) Requires each district to adopt a fee schedule which recovers the costs of the district and which assesses a fee upon the operator of every facility subject to this part. A district may request the state board to adopt a fee schedule for the district if the district's program costs are approved by the district board and transmitted to the state board by April 1 of the year in which the request is made.

(3) Requires any district that has an approved toxics emissions inventory compiled pursuant to this part by August 1 of the preceding year to adopt a fee schedule, as described in paragraph (2), which imposes on facility operators fees which are, to the maximum extent practicable, proportionate to the extent of the releases identified in the toxics emissions inventory and the level of priority assigned to that source by the district pursuant to Section 44360.

(b) Commencing August 1, 1992, and annually thereafter, the state board shall review and may amend the fee regulation.

(c) The district shall notify each person who is subject to the fee of the obligation to pay the fee. If a person fails to pay the fee within 60 days after receipt of this notice, the district, unless otherwise provided by district rules, shall require the person to pay an additional administrative civil penalty. The district shall fix the penalty at not more than 100 percent of the assessed fee, but in an amount sufficient in its determination, to pay the district's additional expenses incurred by the person's noncompliance. If a person fails to pay the fee within 120 days after receipt of this notice, the district may initiate permit revocation proceedings. If any permit is revoked, it shall be reinstated only upon full payment of the overdue fee plus any late penalty, and a reinstatement fee to cover administrative costs of reinstating the permit.

(d) Each district shall collect the fees assessed pursuant to subdivision (a). After deducting the costs to the district to implement and administer this part, the district shall transmit the remainder to the Controller for deposit in the Air Toxics Inventory and Assessment Account, which is hereby created in the General Fund. The money in the account is available, upon appropriation by the Legislature, to the state board and the Office of Environmental Health Hazard Assessment for the purposes of administering this part. (Amended by Stats. 1992, Ch. 375, Sec. 1. Effective January 1, 1993.)

44380.1. A facility shall be granted an exemption by a district from paying a fee in accordance with Section 44380 if all of the following criteria are met:

(a) The facility primarily handles, processes, stores, or distributes bulk agricultural commodities or handles, feeds, or rears livestock.

(b) The facility was required to comply with this part only as a result of its particulate matter emissions.

(c) The fee schedule adopted by the district or the state board for these types of facilities is not solely based on toxic emissions weighted for potency or toxicity. (Added by Stats. 1993, Ch. 1037, Sec. 4. Effective January 1, 1994.)

44380.5. In addition to the fee assessed pursuant to Section 44380, a supplemental fee may be assessed by the district, the state board, or the Office of Environmental Health Hazard Assessment upon the operator of a facility that, at the operator's option, includes supplemental information authorized by paragraph (3) of subdivision (b) of Section 44360 in a health risk assessment, if the review of that supplemental information substantially increases the costs of reviewing the health risk assessment by the district, the state board, or the office. The supplemental fee shall be set by the state board in the regulation required by subdivision (a) of Section 44380 and shall be set in an amount sufficient to cover the direct costs to review the information supplied by an operator pursuant to paragraph (3) of subdivision (b) of Section 44360. (Added by Stats. 1992, Ch. 1162, Sec. 2. Effective January 1, 1993.)

44381. (a) Any person who fails to submit any information, reports, or statements required by this part, or who fails to comply with this part or with any permit, rule, regulation, or requirement issued or adopted pursuant to this part, is subject to a civil penalty of not less than five hundred dollars ($500) or more than ten thousand dollars ($10,000) for each day that the information, report, or statement is not submitted, or that the violation continues.

(b) Any person who knowingly submits any false statement or representation in any application, report, statement, or other document filed, maintained, or used for the purposes of compliance with this part is subject to a civil penalty of not less than one thousand dollars ($1,000) or more than twenty-five thousand dollars ($25,000) per day for each day that the information remains uncorrected. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1988, pursuant to Section 44384.)

44382. Every district shall, by regulation, adopt the requirements of this part as a condition of every permit issued pursuant to Chapter 4 (commencing with Section 42300) of Part 4 for all new and modified facilities. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative July 1, 1988, pursuant to Section 44384.)

44384. Except for Section 44380 and this section, all provisions of this part shall become operative on July 1, 1988. (Added by Stats. 1987, Ch. 1252, Sec. 1. Operative January 1, 1988, by its own provisions.)

CHAPTER 6. FACILITY TOXIC AIR CONTAMINANT RISK REDUCTION AUDIT AND PLAN

(Chapter 6 added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

44390. For purposes of this chapter, the following definitions apply:

(a) "Airborne toxic risk reduction measure" or "ATRRM" means those in-plant changes in production processes or feedstocks that reduce or eliminate toxic air emissions subject to this part. ATRRM's may include:

(1) Feedstock modification.

(2) Product reformulations.
(3) Production system modifications.
(4) System enclosure, emissions control, capture, or conversion.
(5) Operational standards and practices modification.

(b) Airborne toxic risk reduction measures do not include measures that will increase risk from exposure to the chemical in another media or that increase the risk to workers or consumers.

(c) "Airborne toxic risk reduction audit and plan" or "audit and plan" means the audit and plan specified in Section 44392. (Added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

44391. (a) Whenever a health risk assessment approved pursuant to Chapter 4 (commencing with Section 44360) indicates, in the judgment of the district, that there is a significant risk associated with the emissions from a facility, the facility operator shall conduct an airborne toxic risk reduction audit and develop a plan to implement airborne toxic risk reduction measures that will result in the reduction of emissions from the facility to a level below the significant risk level within five years of the date the plan is submitted to the district. The facility operator shall implement measures set forth in the plan in accordance with this chapter.

(b) The period to implement the plan required by subdivision (a) may be shortened by the district if it finds that it is technically feasible and economically practicable to implement the plan to reduce emissions below the significant risk level more quickly or if it finds that the emissions from the facility pose an unreasonable health risk.

(c) A district may lengthen the period to implement the plan required by subdivision (a) by up to an additional five years if it finds that a period longer than five years will not result in an unreasonable risk to public health and that requiring implementation of the plan within five years places an unreasonable economic burden on the facility operator or is not technically feasible.

(d) (1) The state board and districts shall provide assistance to smaller businesses that have inadequate technical and financial resources for obtaining information, assessing risk reduction methods, and developing and applying risk reduction techniques.

(2) Risk reduction audits and plans for any industry subject to this chapter which is comprised mainly of small businesses using substantially similar technology may be completed by a self-conducted audit and checklist developed by the state board. The state board, in coordination with the districts, shall provide a copy of the audit and checklist to small businesses within those industries to assist them to meet the requirements of this chapter.

(e) The audit and plan shall contain all the information required by Section 44392.

(f) The plan shall be submitted to the district, within six months of a district's determination of significant risk, for review of completeness. Operators of facilities that have been notified prior to January 1, 1993, that there is a significant risk associated with emissions from the facility shall submit the plan by July 1, 1993. The district's review of completeness shall include a substantive analysis of the emission reduction measures included in the plan, and the ability of those measures to achieve

emission reduction goals as quickly as feasible as provided in subdivisions (a) and (b).

(g) The district shall find the audit and plan to be satisfactory within three months if it meets the requirements of this chapter, including, but not limited to, subdivision (f). If the district determines that the audit and plan does not meet those requirements, the district shall remand the audit and plan to the facility specifying the deficiencies identified by the district. A facility operator shall submit a revised audit and plan addressing the deficiencies identified by the district within 90 days of receipt of a deficiency notice.

(h) Progress on the emission reductions achieved by the plan shall be reported to the district in emissions inventory updates. Emissions inventory updates shall be prepared as required by the audit and plan found to be satisfactory by the district pursuant to subdivision (g).

(i) If new information becomes available after the initial risk reduction audit and plan, on air toxics risks posed by a facility, or emission reduction technologies that may be used by a facility that would significantly impact risks to exposed persons, the district may require the plan to be updated and resubmitted to the district.

(j) This section does not authorize the emission of a toxic air contaminant in violation of an airborne toxic control measure adopted pursuant to Chapter 3.5 (commencing with Section 39650) or in violation of Section 41700. (Amended by Stats. 1993, Ch. 1041, Sec. 2. Effective January 1, 1994.)

44392. A facility operator subject to this chapter shall conduct an airborne toxic risk reduction audit and develop a plan which shall include at a minimum all of the following:

(a) The name and location of the facility.

(b) The SIC code for the facility.

(c) The chemical name and the generic classification of the chemical.

(d) An evaluation of the ATRRM's available to the operator.

(e) The specification of, and rationale for, the ATRRMs that will be implemented by the operator. The audit and plan shall document the rationale for rejecting ATRRMs that are identified as infeasible or too costly.

(f) A schedule for implementing the ATRRMs. The schedule shall meet the time requirements of subdivision (a) of Section 44391 or the time period for implementing the plan set by the district pursuant to subdivision (b) or (c) of Section 44391, whichever is applicable.

(g) The audit and plan shall be reviewed and certified as meeting this chapter by an engineer who is registered as a professional engineer pursuant to Section 6762 of the Business and Professions Code, by an individual who is responsible for the processes and operations of the site, or by an environmental assessor registered pursuant to Section 25570.3. (Added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

44393. The plan prepared pursuant to Section 44391 shall not be considered to be the equivalent of a pollution prevention program or a source reduction program, except insofar as the audit and plan elements are consistent with source reduction, as defined in Section 25244.14, or

subsequent statutory definitions of pollution prevention. (Added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

44394. Any facility operator who does not submit a complete airborne toxic risk reduction audit and plan or fails to implement the measures set forth in the plan as set forth in this chapter is subject to the civil penalty specified in subdivision (a) of Section 44381, and any facility operator who, in connection with the audit or plan, knowingly submits any false statement or representation is subject to the civil penalty specified in subdivision (b) of Section 44381. (Added by Stats. 1992, Ch. 1162, Sec. 3. Effective January 1, 1993.)

Appendix C

Asbestos Quantity Conversion Factors

Appendix C

Asbestos Quantity Conversion Factors

A. "PCM" versus "TEM"

Two main analytical methods have been used for the analysis of asbestos samples: phase contrast microscopy (PCM), the primary method used historically to analyze asbestos samples, and transmission electron microscopy (TEM), the current state-of-the-art method.

PCM analysis has been preferred in the past over TEM because it can be done more quickly and it is less expensive. One major limitation of PCM analysis, however, especially in outdoor environments, is that the analyst cannot distinguish asbestos from non-asbestos fibers, such as cellulose, talc, or gypsum. Also, PCM cannot detect fibers that have a diameter of about 0.3 microns or less, which could substantially underestimate the asbestos fiber concentrations. These limitations make PCM impractical for the analysis of ambient asbestos samples.

Transmission electron microscopy (TEM) is the preferred analytical method for outdoor asbestos samples because of its ability to detect small fibers (greater than or equal to 0.0002 microns in diameter) and to distinguish between asbestos fibers and non-asbestos fibers. The term "TEM structures" is often used to describe asbestos fibers detected by this method. TEM is the method recommended by the Office of Environmental Health Hazard Assessment (OEHHA). TEM measurements cannot be directly related to the risk potency factors, however, because the studies upon which OEHHA's risk assessment was based used the less expensive PCM analysis. The TEM measurements must be converted to PCM-equivalent units, using the following equation (ARB, 1990):

$$1\,\text{PCM fiber} = 320\,\text{TEM structures}$$

B. Asbestos Inhalation Cancer Potency Factor

The unit risk factor for asbestos fibers is 1.9×10^{-4} in units of $(100\ \text{PCM fibers/m}^3)^{-1}$ and the unit risk factor is 6.3×10^{-2} in units of $(\mu\text{g/m}^3)^{-1}$. The unit risk factor is based on epidemiological studies in which PCM fiber measurements were used. These unit risk factors are listed in Chapter 7 and in the Asbestos Toxic Air Contaminant (TAC) identification document (CDHS, 1986) and in OEHHA, 1999b. These asbestos cancer potency factors are for mesothelioma. Since these cancer potency factors are in units of concentration or dose, complications arise when the emitted asbestos quantities are reported in mass units (pounds/year and maximum pounds/hour) for the Air Toxics Hot Spots Program (Hot Spots).

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

The TAC inhalation cancer potency factor has been converted from mass to concentration using a factor of 0.003 µg asbestos = 100 asbestos PCM fibers. This conversion has been derived from information published by the United States Environmental Protection Agency (U.S. EPA) (U.S. EPA, 1986). The number of asbestos PCM fibers associated with a given mass of asbestos can vary appreciably. Also, U.S. EPA has stated that this conversion factor is the geometric mean of measured relationships between optical fiber counts and mass airborne chrysotile in several published studies, that the range of the conversion factor between the different studies is large (0.0005 - 0.015 µg asbestos/100 asbestos PCM fibers), and that the factor carries with it an appreciable uncertainty.

The current recommendation for Hot Spots risk assessments uses a default breathing rate of 393 L/day-kg body weight for a 70 year exposure duration. A dose is calculated from the ground level concentration using the following equation:

$$X\ (\mu g/m^3) \times 393\ L/day\text{-}kg\ body\ weight \times 10^{-6} = dose\ (mg/kg\text{-}day)$$

The 10^{-6} term converts the L in the breathing rate to m^3 and the µg in the air concentration term to mg.

In order to obtain cancer risk the dose is subsequently multiplied times the cancer potency factor as follows:

Dose (mg/kg-body weight) x cancer potency factor (mg/kg-body weight) = Cancer risk (unitless)

For risk communication purposes cancer risk may be converted into chances per million of developing cancer. This terminology is often more clearly understood by the public than cancer risk.

Cancer risk x (1×10^6) = chances per million of developing cancer

The cancer potency factor $(mg/kg\ body\ weight)^{-1}$ may be calculated from the fiber cancer potency factor using the relationship of 0.003 µg = 100 fibers PCM, 70 kg body weight, 20 m^3 breathed per day, and a factor of 1000 to convert µg asbestos into mg:

$$1.9\times10^{-4}\left(100\ PCM\ \ fibers/m^3\right)^{-1}\times\frac{70kg}{20m^3}\times\frac{1000}{0.003mg/100\ fibers}=2.2\times10^{+2}\left(mg/kg\ body\ weight\right)^{-1}$$

The ISCST3 air dispersion modeling program estimates concentrations in units of $\mu g/m^3$ based on emission estimates in lb/yr. If the ground level concentrations are derived from PCM fiber measurements, then no additional uncertainty is introduced by the conversion to µg using the factor of 0.003. This is because the factor is effectively cancelled out by its use to derive the cancer potency factor in $(mg/kg\ body\ weight)^{-1}$. There is a slight rounding error that may be introduced.

References

ARB, 1990. Proposed Control Measure for Asbestos-Containing Serpentine Rock in Surfacing Applications, Technical Support Document, Air Resources Board, February 1990.

CDHS, (1986) California Department of Health Services (CDHS) 1986. Report to the Air Resources Board on Asbestos. Part B. Health Effects of Asbestos. Epidemiological Studies Section, Berkeley, CA.

OEHHA. (1999b). Air Toxics Hot Spots Program Risk Assessment Guidelines. Part II. Technical Support Document for Describing Available Cancer Potency Factors. Available online at http://www.oehha.ca.gov

USEPA, 1986. Airborne Asbestos Health Assessment Update. EPA/600/8-84/003F, Office of Health and Environmental Assessment, Washington, DC.

Appendix D

Risk Assessment Procedures to Evaluate Particulate Emissions from Diesel-Fueled Engines

Appendix D

Risk Assessment Procedures to Evaluate Particulate Emissions from Diesel-Fueled Engines

A. Introduction

The objective of this appendix is to discuss procedures for estimating potential cancer and noncancer health risk from exposure to particulate matter (PM) emissions from diesel-fueled engines (diesel exhaust). It will also clarify the requirements and recommendations for acute noncancer and multipathway cancer and chronic risk assessment for diesel PM. In addition to the notification and risk reduction requirements under the Hot Spots Program, this appendix should facilitate the use of the *Risk Reduction Plan to Reduce Particulate Matter Emissions from Diesel-Fueled Engines and Vehicles* (ARB, 2000) (Diesel Guidelines). The Diesel Guidelines were developed by the Air Resources Board (ARB) with assistance from the Office of Environmental Health Hazard Assessment (OEHHA) in October 2000. The Diesel Guidelines are intended to assist local Air Pollution Control and Air Quality Management Districts (Districts) and sources of diesel PM emissions in making consistent risk management decisions.

In advance of performing a health risk assessment (HRA), it is recommended that the District and the stationary source of diesel emissions reach a consensus on the HRA approach for estimating health impacts from diesel exhaust. See Chapter 9 for an outline of a modeling protocol.

B. Calculations/Risk Assessment Procedures

In August 1998, the ARB identified diesel exhaust as a toxic air contaminant (TAC) (ARB, 1998). In the identification report, OEHHA provided an inhalation noncancer chronic reference exposure level (REL) of 5 micrograms per cubic meter ($\mu g/m^3$) and a range of inhalation cancer potency factors of 1.3×10^{-4} to 2.4×10^{-3} $(\mu g/m^3)^{-1}$. The Scientific Review Panel on Toxic Air Contaminants recommended a "reasonable estimate" inhalation unit risk factor of 3.0×10^{-4} $(\mu g/m^3)^{-1}$. From the unit risk factor an inhalation cancer potency factor of 1.1 $(mg/kg\text{-}day)^{-1}$ may be calculated. These noncancer and cancer health factors were developed based on whole (gas and particulate matter) diesel exhaust. The surrogate for whole diesel exhaust is diesel PM. PM_{10} (particulate matter, ten microns or less in size) is the basis for the potential risk calculations.

Cancer

When conducting an HRA, the potential cancer risk from inhalation exposure to diesel PM will outweigh the potential noncancer health impacts. Therefore, inhalation cancer risk is required for every HRA. (The methods for calculating inhalation cancer risk can be found in Chapters 5, 7, and 8.) When comparing whole diesel exhaust to speciated diesel exhaust (e.g., PAHs, metals), potential cancer risk from inhalation exposure to whole diesel exhaust will

outweigh the multipathway cancer risk from the speciated components. For this reason, there will be few situations where an analysis of multipathway risk is necessary.

The District may elect to require a multipathway analysis if reliable data are available and the District decides that it is necessary. If the District elects to require a multipathway analysis, the components of the diesel exhaust will need to be speciated since there is not an oral cancer potency factor for diesel PM. It is recommended that the District be consulted on the procedures for conducting a multipathway analysis for diesel exhaust. The District may wish to use speciation data from the ARB. If so, a resource for speciation data is available on the ARB's website at www.arb.ca.gov/emisinv/speciate/speciate.htm.

If a multipathway analysis is required, the speciated data should be compared with the substances in Table 5.1. Any substances in the speciation profile that are listed in Table 5.1 and have an oral cancer potency factor in Table 7.1 should be included in the multipathway analysis. Potential multipathway cancer risks are estimated following the procedures in Chapters 5 and 8 of this document. These procedures require summing the potential cancer risk from each carcinogen to estimate the total facility cancer risk.

Noncancer Chronic

To determine noncancer chronic inhalation health impacts from exposure to diesel exhaust use the methods described in Chapters 6 and 8.

In most situations, noncancer health impacts from inhalation exposure to whole diesel exhaust will outweigh the noncancer multipathway health impacts to the speciated components of diesel exhaust. However, there may be situations when the multipathway impacts need to be investigated.

Therefore, the District may elect to require a multipathway analysis if reliable data is available and they feel it is necessary. If the District elects to require a multipathway analysis, the components of the diesel exhaust will need to be speciated since there is not an oral reference exposure level for diesel PM. A resource for speciation data at the ARB is identified above. It is recommended that the District be consulted on the procedures for conducting a multipathway analysis. If a multipathway analysis is required, the speciated data should be compared with the substances in Table 5.1. Any substances in the speciation profile that are listed in Table 5.1 and have an oral chronic REL in Table 6.3 should be included in the multipathway analysis. Potential multipathway chronic risks are estimated following the procedures in Chapters 5 and 8 of this document.

Noncancer Acute

As stated above, potential cancer risk is usually the driving health impact for diesel exhaust. However, there may be certain unusual situations where an evaluation of the acute health effects may be warranted. One possible situation is when a nearby receptor is located above the emission release point (e.g. on a hillside or in a multistory apartment building). Since there is no acute REL for diesel exhaust, the components of the exhaust will need to be speciated to determine the potential acute health impacts. It is recommended that the District be consulted on the procedures for conducting an acute analysis. If an acute analysis is required, the speciated

data should be compared with the substances in Table 6.1. Any substances in the speciation profile that are listed in Table 6.1 should be included in the acute analysis. A resource for speciation data at the ARB is identified above. Potential acute risks are estimated following the procedures in Chapters 6 and 8 of this document.

References:

ARB 1998. Air Resources Board, "Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant, Appendix III, Part A, Exposure Assessment," April 1998.

ARB 2000. Air Resources Board, "Risk Reduction Plan to Reduce Particulate Matter Emissions from Diesel-Fueled Engines and Vehicles," October 2000.

Appendix E

Toxicity Equivalency Factors for Polychlorinated Dibenzo-*p*-Dioxins Dibenzofurans And Polychlorinated Biphenyls

Appendix E

Toxicity Equivalency Factors for Polychlorinated Dibenzo-*p*-Dioxins Dibenzofurans and Polychlorinated Biphenyls

Introduction

Dioxins and furans vary considerably in their potency for causing both cancer and noncancer health impacts. A facility may choose to speciate dioxin and furan emissions in order to obtain a more accurate picture of the risks. A scheme, based on both cancer and noncancer toxicity studies, has been developed to relate the potency of various dioxin and furan congeners to the potency of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. A detailed explanation of the World Health Organization's 1997 Toxicity Equivalents Factor (WHO_{97}-TEF) (van den Berg, 1998) scheme, the latest scheme adopted by OEHHA, is available in OEHHA (2003).

The individually calculated inhalation or oral doses of each dioxin or furan congener may be multiplied times the oral or inhalation cancer potency for each individual congener listed in Table 7.1. In order to determine the inhalation chronic hazard index, the ground level concentration of each congener may be divided by the chronic REL for each congener in Table 6.2 and the hazard quotients may be summed to give the hazard index for dioxins and furans. The oral chronic hazard quotient may be calculated by determining the oral dose of each congener and dividing by the individual chronic oral REL for each congener. The oral hazard quotients may be summed to give the hazard quotient for oral noncancer dioxin risks and may then be added to the inhalation hazard index to give the combined inhalation and oral chronic hazard quotient for dioxins.

A second equivalent procedure may also be used to calculate the cancer risk of a mixture of dioxin and furan congeners. The concentration of each congener listed in Table E-1 is multiplied by the WHO_{97}-TEF for that congener. For example, for 1,2,3,4,7,8-hexachlorodibenzodioxin the concentration ($\mu g/m^3$) may be multiplied by 0.1 to give the concentration equivalent to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. 2,4,7,8-tetrachlorodibenzodioxin would be multiplied by zero indicating no cancer or noncancer toxicity. The 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalent concentrations may be summed and treated as if the total concentration were 2,3,7,8-tetrachlorodibenzo-*p*-dioxin for the purposes of calculating cancer and noncancer risks. Thus, the potency adjusted ground level concentration can be multiplied by the breathing rate to give dose (see equation 5.4.1), and then multiplied times the cancer potency factor for 2,3,7,8- tetrachlorodibenzo-*p*-dioxin (Table 7.1) to give cancer risk for the entire mixture. If a noncancer chronic hazard index needs to be calculated the potency adjusted ground level concentration can be divided by the chronic reference exposure level for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin to give a hazard index for the entire mixture. The TEF may be multiplied times the individual congener dose calculated for the inhalation and oral cancer risk calculation, and the oral chronic hazard index determination.

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

The most recent TEF scheme adopted by OEHHA includes TEF for individual PCB congeners (see Table E-1) (OEHHA, 2003). These are the congeners that have dioxin-like biological effects. The same procedures as described above may be used to calculate the concentration or dose of these congeners. Where data are available on individual PCB congeners emitted by a facility, then these TEFs are to be used. If Table E1 is used to adjust the dose or concentration of the individual PCB congeners, the 2,3,7,8-tetrachlorodibenzo-*p*-dioxin oral and inhalation cancer potency factors should be used to determine cancer risk. The chronic inhalation and oral REL for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin should be used to determine the noncancer chronic hazard index. If only total PCB data are available, then the PCB slope factors provided in Table 7.1 can be used for cancer risk determination.

Table E1. WHO/97 Toxic equivalency factors (TEFs)

Congener		TEF_{WHO-97}
PCDDs		
2,3,7,8-TCDD		1
1,2,3,7,8-PeCDD		1
1,2,3,4,7,8-HxCDD		0.1
1,2,3,7,8,9-HxCDD		0.1
1,2,3,6,7,8-HxCDD		0.1
1,2,3,4,6,7,8-HpCDD		0.01
1,2,3,4,6,7,8,9-OCDD		0.0001
PCDFs		
2,3,7,8-TCDF		0.1
1,2,3,7,8-PeCDF		0.05
2,3,4,7,8-PeCDF		0.5
1,2,3,4,7,8-HxCDF		0.1
1,2,3,7,8,9-HxCDF		0.1
1,2,3,6,7,8-HxCDF		0.1
2,3,4,6,7,8-HxCDF		0.1
1,2,3,4,6,7,8-HpCDF		0.01
1,2,3,4,7,8,9-HpCDF		0.01
1,2,3,4,6,7,8,9-OCDF		0.0001
PCBs (IUPAC #, Structure)		
77	3,3',4,4'-TCB	0.0001
81	3,4,4',5-TCB	0.0001
105	2,3,3',4,4'-PeCB	0.0001
114	2,3,4,4',5-PeCB	0.0005
118	2,3',4,4',5-PeCB	0.0001
123	2',3,4,4',5-PeCB	0.0001
126	3,3',4,4',5-PeCB	0.1
156	2,3,3',4,4',5-HxCB	0.0005
157	2,3,3',4,4',5'-HxCB	0.0005
167	2,3',4,4',5,5'-HxCB	0.00001
169	3,3',4,4',5,5'-HxCB	0.01
189	2,3,3',4,4',5,5'-HpCB	0.0001

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

References

OEHHA, 2003. Technical Support Document for Describing Available Cancer Potency Factors, Appendix A (revised August, 2003) : Use of the Revised Toxicity Equivalency Factor (TEF_{WHO-97}) Scheme for Estimating Toxicity of Mixtures of Dioxin-Like Chemicals, September 2003. Available at **www.oehha.ca.gov**.

van den Berg, M., Birnbaum, L., Bosveld, A. T. C., Brunstrom, B., Cook, P., Feeley, M., Giesy, J. P., Hanberg, A., Hasegawa, R., Kennedy, S. W., Kubiak, T., Larsen, J. C., Van Leeuwen, F. X. R., Liem, A. K. D., Nolt, C., Peterson, R. E., Poellinger, L., Safe, S., Schrenk, D., Tillitt, D., Tysklind, M., Younes, M., Waern, F., and Zacharewski, T. (1998). Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. Environ. Health Perspec. **V106**, 775-792.

Appendix F

Overview of the Lead Risk Assessment Procedures

Appendix F

Overview of the Lead Risk Assessment Procedures

I. Introduction

The objective of this appendix is to provide a method for estimating potential cancer and noncancer health effects due to airborne lead exposure. This appendix should facilitate the use of the *Risk Management Guidelines for New, Modified, and Existing Sources of Lead* (Lead RM Guidelines) (ARB, 2001) for analysis of lead exposure. The Lead RM Guidelines were developed by the Air Resources Board (ARB) with assistance from Office of Environmental Health Hazard Assessment (OEHHA) and Department of Health Services (DHS) in March 2001 to assist local air districts and sources of lead in making consistent risk management decisions for new, modified, and existing sources of lead.

In April 1997, the ARB identified inorganic lead as a toxic air contaminant (TAC) (ARB, 1997). Lead is unique among other TACs identified by ARB in several ways. First, infants and children are particularly susceptible to the health effects of lead, and the risk assessment is based on health effects in children. Second, the chronic noncancer effects are related to blood lead levels (BLLs) as opposed to ambient air concentrations. These BLLs reflect current and past exposure from a number of sources; air emissions may only be a small part of the total exposure. Third, based on recommendations of the OEHHA and the Scientific Review Panel on Toxic Air Contaminants (SRP), the ARB did not identify a threshold level for chronic noncancer health effects due to lead exposure. Threshold levels are levels below which no adverse health effects are expected to occur. Since acute or chronic Reference Exposure Levels (RELs) are based on threshold levels, none were developed for lead. Thus, a hazard index approach is not used for lead. Instead, air concentrations are compared to defined air lead levels associated with specified percentages of children with BLL $\geq$ 10 μg/dL. Acceptable risk is based on minimizing the number of children at or above a BLL of 10 μg/dL.

II. Methods for Estimation of Health Risk Effects

Methods for estimating site-specific noncancer and cancer potential health impacts from exposure to lead emissions are given in the Lead RM Guidelines. The noncancer health effects pose greater public health significance than the cancer health effects. Minimizing noncancer health effects of lead will therefore also minimize cancer health effects.

Chronic noncancer health risks are estimated based on neurodevelopmental health risks to children and would also be protective of adults. These health effects can be evaluated using a tiered approach based on blood lead level distribution in the population.

Potential multipathway cancer risks are estimated following the procedures in Chapters 5 and 8 of this document. These procedures require summing individual cancer risk from each carcinogen to estimate the total facility cancer risk.

In advance of performing a health risk assessment (HRA), it is recommended that the Air Pollution Control or Air Quality Management District (District) and the stationary source of lead air emissions reach a consensus on the HRA approach for estimating chronic noncancer and cancer health risks. See Chapter 9 for an outline of a modeling protocol.

A. Tiered Approach for Estimating Noncancer Risks due to Lead Exposure

The Lead Risk Management Guidelines provide three tiers of analysis to determine baseline BLL distributions for estimating risk. Although there is a simple risk management option provided in the Lead RM Guidelines, in a risk assessment for the Air Toxics Hot Spots program one of the following tiers must be used to report estimates of the percent of children estimated to be above 10 μg/dL blood lead. The tiered approach is based on an assessment of neurodevelopmental risk, with the BLL distribution in the population as the most significant factor. The BLL distribution consists of two components: 1) the baseline BLL distribution due to all sources of exposure; and 2) the exposure due to emissions from a facility.

Tier I is a default approach that requires minimal site-specific information on concentrations of lead in environmental media other than air. Tier I uses two default BLL distributions, one for a high exposure scenario and one for an average exposure scenario. The exposure scenario is determined using the median age of the homes in the census tract and the ratio of area income to the poverty level. The default baseline BLL distribution for each of the exposure scenarios is based on a review of neighborhood and community blood lead studies. The assessor determines the 30-day average lead concentration due to the facility averaged over the 1 square kilometer area centered on the Maximum Offsite Concentration (MOC). The percentage of children with BLLs greater than or equal to 10 micrograms per deciliter (≥ 10 μg/dL) is determined using Table F-1 (also found on page 17 in the Lead RM Guidelines), the air lead concentration, and the determined exposure scenario. The 10 μg/dL threshold level has been identified by the Centers for Disease Control and Prevention (CDC) as a level where potential health effects may occur. The public health goal of management practices should be to implement procedures/practices to prevent BLLs at or above this level. The estimated percentage of children with BLLs ≥ 10 μg/dL is then used with risk management levels given in Chapter III, Section D of the Lead RM Guidelines to assist in making risk management decisions.

Table F-1 Percentage of Children with Blood Lead Levels ≥ 10 μg/dl for Various Air Lead Concentrations at Two Exposure Scenarios

Air Lead Concentration in the Maximum Exposure Area (30-day average) [μg/m^3]	Percent ≥ 10 μg/dL	
	High Exposure Scenario	Average Exposure Scenario
Baseline*	5.1	1.2
0.02	5.4	1.4
0.06	6.1	1.7
0.10	6.8	2.2
0.20	8.9	3.4
0.25	9.8	4.1
0.50	15.9	8.9
0.75	22.4	15.4
1.0	29.1	23.0
1.5	42.5	39.0

* The baseline represents BLLs due to lead in soil, dust, water, food, and background air lead concentrations.

Tier II requires the development of site-specific baseline BLL distributions within the impacted population using site-specific estimates of lead levels in environmental media, including soil, dust, water, and/or food, using the U.S. EPA Integrated Exposure Uptake Biokinetic (IEUBK) model. The IEUBK model calculates the probability of an individual exceeding a specific BLL based on site-specific information. The aggregate of the individual BLLs is used to estimate the neurodevelopmental risk in the maximum exposure area. A detailed discussion of this tier is beyond the scope of this overview; see Appendix D in the Lead RM Guidelines for a discussion of the IEUBK model and its use.

Tier III involves actual blood lead sampling of the population impacted by the facility to define the baseline BLLs. In Tier III, the facility is responsible for conducting BLL testing to establish a site-specific BLL distribution. The Lead RM Guidelines recommend the neurodevelopmental risk be calculated as the probability of children in an affected exposure area having a BLL ≥ 10 μg/dL using the results of the blood lead sampling. It is highly unlikely that this option would be used due to the cost incurred and the fact that the sampled population must consent to the sampling and an appropriate sampling strategy must be developed to adequately characterize the blood lead levels of the impacted population.

For further information on the tiered approach using the Tier I, Tier II, or Tier III, please see Chapter II of the *ARB Risk Management Guidelines for New, Modified, and Existing Sources of Lead* (ARB, 2001). This document can be downloaded from the ARB web site at *http://www.arb.ca.gov/toxics/lead/lead.htm* or can be requested by calling (916) 323-4327.

B. Methods for Estimating Potential Cancer Risks due to Lead

While lead has a unique noncancer assessment methodology, the determination of potential multipathway cancer risk is the same as other carcinogens. Chapters 5, 7, and 8, and Appendices I and L provide all the needed information for calculating potential cancer risk. The health risk assessment should report the multipathway cancer risks from lead emissions.

Chapter III in the Lead RM Guidelines provides methods for determining risk management of lead exposure, using the results from the cancer risk calculation, and the local District's defined significance levels.

III. References

ARB, 1997. Proposed Identification Inorganic Lead as a Toxic Air Contaminant, Parts A, B, C. California Air Resources Board. April, 1997.

ARB, 2001. ARB Risk Management Guidelines for New, Modified, and Existing Sources of Lead. California Air Resources Board. March 2001

APPENDIX G

PAH Potency Factors and Selection of Potency Equivalency Factors (PEF) for PAHs Based on Benzo[*A*]Pyrene Potency

Appendix G

PAH Potency Factors and Selection of Potency Equivalency Factors (PEF) for PAHs based on Benzo(*a*)pyrene Potency

Benzo(a)pyrene (BaP) was chosen as the primary representative of the class of polycyclic aromatic hydrocarbons (PAHs) because of (1) the large amount of toxicological data available on BaP (versus the relatively incomplete database for other PAHs), (2) the availability of monitoring techniques for BaP, and (3) the significant exposure expected (and found). The Office of Environmental Health Hazard Assessment (OEHHA) has developed a Potency Equivalency Factor (PEF) procedure to assess the relative potencies of PAHs and PAH derivatives as a group. This procedure can address the impact of carcinogenic PAHs in ambient air since they are usually present together. This procedure was approved by the Scientific Review Panel (SRP) on Toxic Air Contaminants (TAC) as part of the Health Effects Assessment of Benzo(a)pyrene during the TAC identification process (OEHHA, 1993).

Due to the variety of data available on the carcinogenicity and mutagenicity of PAHs, an order of preference for the use of available data in assessing relative potency was developed. If a health effects evaluation and quantitative risk assessment leading to a cancer potency value had been conducted on a specific PAH, then those values were given the highest preference. Cancer potency values for PAHs developed by this process are shown in Table G-1.

Table G-1: Potencies of PAHs and derivatives[1]

Chemicals	Cancer potency factors $(mg/kg\text{-}day)^{-1}$	Unit risks $(\mu g/m^3)^{-1}$
benzo[*a*]pyrene	11.5	1.1×10^{-3}
dibenz[*a*,*h*]anthracene	4.1	1.2×10^{-4}
7,12-dimethylbenzanthracene	250	7.1×10^{-2}
3-methylcholanthrene	22	6.3×10^{-3}
5-nitroacenaphthene	0.13	3.7×10^{-5}

1. Source: (OEHHA 1993; Collins *et al*., 1998). It is assumed that unit risks for inhalation have the same relative activities as cancer potencies for oral intake.

If potency values have not been developed for specific compounds, a carcinogenic activity relative to BaP, rather than a true potency, can be developed. These relative activity values are referred to as Potency Equivalency Factors or PEFs. For air contaminants, the relative potency to BaP based on data from inhalation studies would be optimal. Otherwise, intrapulmonary or intratracheal administration studies would be most relevant, since such studies are in the target organ of interest. Next in order of

preference is information on activity by the oral route and skin painting. Intraperitoneal and subcutaneous administration rank at the bottom of the *in vivo* tests considered useful for PEF development because of their lack of relevance to environmental exposures. Next, in decreasing order of preference, are genotoxicity data, which exist for a large number of compounds. In many cases genotoxicity information is restricted to mutagenicity data. Finally, there are data on structure-activity relationships among PAH compounds. Structure-activity considerations may help identify a PAH as carcinogenic, but at this time have not been established as predictors of carcinogenic potency.

Using this order of preference, PEFs were derived for 21 PAHs and are presented in Table G-2 (OEHHA, 1993: Collins *et al*., 1998).

Table G-2. OEHHA PEF weighting scheme for PAHs and their resulting cancer potency values.

PAH or derivative	PEF	Unit Risk $(\mu g/m^3)^{-1}$	Inhalation Slope Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
benzo[*a*]pyrene (index compound)	**1.0**	**1.1E-3**	**3.9E+0**	**1.2E+1**
benz[*a*]anthracene	0.1	1.1E-4	3.9E-1	1.2E+0
benzo[b]fluoranthene	0.1	1.1E-4	3.9E-1	1.2E+0
benzo[*j*]fluoranthene	0.1	1.1E-4	3.9E-1	1.2E+0
benzo[k]fluoranthene	0.1	1.1E-4	3.9E-1	1.2E+0
dibenz[*a*,*j*]acridine	0.1	1.1E-4	3.9E-1	1.2E+0
dibenz[*a*,*h*]acridine	0.1	1.1E-4	3.9E-1	1.2E+0
7H-dibenzo[c,*g*]carbazole	1.0	1.1E-3	3.9E+0	1.2E+1
dibenzo[*a*,*e*]pyrene	1.0	1.1E-3	3.9E+0	1.2E+1
dibenzo[*a*,*h*]pyrene	10	1.1E-2	3.9E+1	1.2E+2
dibenzo[*a*,*i*]pyrene	10	1.1E-2	3.9E+1	1.2E+2
dibenzo[*a*,*l*]pyrene	10	1.1E-2	3.9E+1	1.2E+2
indeno[1,2,3-*cd*]pyrene	0.1	1.1E-4	3.9E-1	1.2E+0
5-methylchrysene	1.0	1.1E-3	3.9E+0	1.2E+1
1-nitropyrene	0.1	1.1E-4	3.9E-1	1.2E+0
4-nitropyrene	0.1	1.1E-4	3.9E-1	1.2E+0
1,6-dinitropyrene	10	1.1E-2	3.9E+1	1.2E+2
1,8-dinitropyrene	1.0	1.1E-3	3.9E+0	1.2E+1
6-nitrochrysene	10	1.1E-2	3.9E+1	1.2E+2
2-nitrofluorene	0.01	1.1E-5	3.9E-2	1.2E-1
chrysene	0.01	1.1E-5	3.9E-2	1.2E-1

1. Source: OEHHA (1993)

The cancer potency comparisons show that some PAHs are more potent than BaP, while other PAHs analyzed were less or much less potent. These comparisons indicated that considering all PAHs to be equivalent in potency to BaP would likely overestimate the cancer potency of a PAH mixture, but such an assumption would be health protective and likely to be helpful in a screening estimate of PAH risks (OEHHA, 1993). If one

assumes that PAHs are as carcinogenic as they are genotoxic, then their hazard relative to BaP would be dependent on their concentration in the environment. In light of the limited information available on other PAHs, BaP remains an important representative or surrogate for this group of air pollutants.

Detailed descriptions on the criteria used for developing individual PEFs can be found in (OEHHA, 1999b) Currently, OEHHA is undertaking a review of all recent literature pertaining to the carcinogenicity and mutagenicity of PAHs. New cancer potency values for PAHs may be developed if an adequate health effects evaluation and quantitative risk assessment can be performed. Also, some current PEFs may be modified based on new data. Any changes to the potency values and PEFs for PAHs will be reflected in the HARP program when they occur. It is incumbent on the risk assessor to access the most recent version of the HARP program to ensure that the most up-to-date PAH potency values are used.

References

Collins, J.F., Brown, J.P., Alexeeff, G.V., and Salmon, A.G. 1998. Potency equivalency factors for some polycyclic aromatic hydrocarbons and polycyclic aromatic hydrocarbon derivatives. Regul. Toxicol. Pharmacol. 28:45-54.

OEHHA, 1993. Benzo[*a*]pyrene as a Toxic Air Contaminant. Part B. Health Effects of Benzo[*a*]pyrene. Air Toxicology and Epidemiology Section, Berkeley, CA.

OEHHA, 1999b. The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II. Technical Support Document for Describing Available Cancer Potency Factors, Office of Environmental Health Hazard Assessment, April 1999.

Appendix H

Recommendations for Estimating Concentrations of Longer Averaging Periods from the Maximum One-Hour Concentration for Screening Purposes

Appendix H

Recommendations for Estimating Concentrations of Longer Averaging Periods from the Maximum One-Hour Concentration for Screening Purposes

A. Introduction

The U.S. Environmental Protection Agency (U.S. EPA) SCREEN3 air dispersion model is frequently used to estimate the maximum one-hour concentration downwind due to emissions from a point source to assess impacts from a source. The SCREEN3 model results (or ISCST3 with screening meteorological data), in conjunction with the U.S. EPA screening factors, are frequently used to estimate concentrations for longer averaging periods, such as the maximum annual average concentration. In addition, it is permissible to use the ISCST3 air dispersion model in a screening mode with identical meteorological conditions as used in the SCREEN3 model to superimpose results from multiple sources.

This method to assess short-term and long-term impacts may be used as a first-level screening indicator to determine if a more refined analysis is necessary. In the event that representative meteorological data are not available, the screening assessment may be the only computer modeling method available to assess source impacts.

In California, this standard procedure will generally bias concentrations towards over prediction in most cases when the source is a continuous release. However, in the case when a source is not continuous, these screening factors may not be biased towards over prediction. In this case, we recommend an alternative procedure for estimating screening value concentrations for longer averaging periods than one-hour for intermittent releases.

B. Current Procedures

The current screening factors used to estimate longer term averages (i.e., 3-hour, 8-hour, 24-hour, 30-day, and annual averages) from maximum one-hour concentrations in California are shown in Table H.1 and Figure H.1. The factors are U.S. EPA recommended values with the exception of the 30-day factor. The 30-day factor is an ARB recommended value (ARB, 1994). The maximum and minimum values are recommended limits to which one may diverge from the general (Rec.) case, (U.S. EPA, 1992). Diverging from the general case should only be done on a case by case basis with prior approval from the reviewing agency.

C. Non-Standard Averaging Periods with a Continuous Release

The following is the ARB recommendation for estimating screening concentrations for non-standard averaging periods that are not listed in Table H.1 or Figure H.1. Specifically, the recommendation is for estimating screening concentrations for 4-hour, 6-hour, and 7-hour averaging periods.

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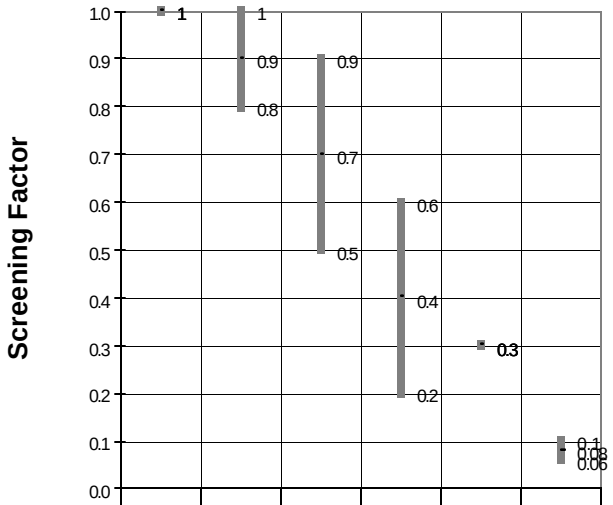
The current U.S. EPA screening factors applicable to standard averaging periods should be used for non-standard averaging periods. Specifically for the 4-hour, 6-hour, and 7-hour averaging periods, we recommend that the 3-hour screening factor of (0.9± 0.1) be used. The following illustrates the method to estimate a 6-hour average concentration from a continuous release from a single point source:

1. determine the maximum 1-hour concentration according to standard screening procedures ($C_{max1\text{-}hr}$),
2. scale the maximum 1-hour concentration by (0.9±0.1), and
3. the result is the maximum 6-hour concentration ($C_{max6\text{-}hr}=C_{max1\text{-}hr}$ *(0.9±0.1)).

In the case for the 6-hour and 7-hour average concentration estimates, the user may wish to take the lower bound of (0.9±0.1), or 0.8. For the 4-hour average estimate, we recommend the user to use the 3-hour factor as is, 0.9.

Figure H.1
Screening Factors to Convert
1-hr Max. to Longer Avg. Periods



	1-hr	3-hr	8-hr	24-hr	30-day	Annual
Max	1	1	0.9	0.6	0.3	0.1
Min	1	0.8	0.5	0.2	0.3	0.06
Rec	1	0.9	0.7	0.4	0.3	0.08

Averaging Period

Table H.1 Recommended Factors to Convert Maximum 1-hour Avg. Concentrations to Other Averaging Periods (U.S. EPA, 1992; 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

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Table H.2 summarizes these recommendations for the non-standard averaging periods.

Table H.2 Recommended Factors to Convert Maximum 1-hour Avg. Concentrations to Non-Standard Averaging Periods.

Averaging Time	Range	Typical Recommended
4 hours	0.8 - 1.0	0.9
6 hours	0.8 – 1.0	0.8
7 hours	0.8 – 1.0	0.8

D. Definitions

It is convenient to define the following terms relating to sources with respect to the duration of the release.

- Continuous Release – this is a release that is continuous over the duration of a year. An example of this type of release would be fugitive emissions from a 24-hour per day, 7-day per week operation or an operation that is nearly continuous.

- Intermittent Release – many emissions fall under this category. These are emission types that are not continuous over the year. Any operation that has normal business hours (e.g., 8 am to 6 pm) would fall into this category.

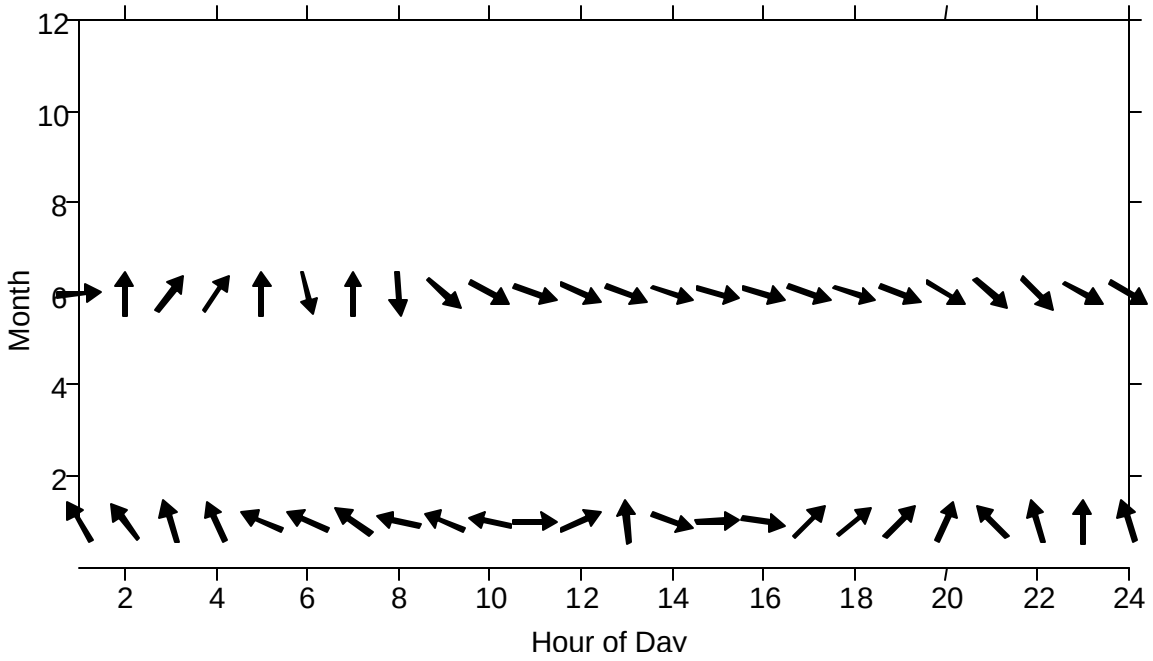
- Systematic Release – these are intermittent releases that occur at a specific time of the day. As an example, these type of releases can occur when a process requires clean out at the end of the work day. Thereby releasing emissions only at the end of the workday systematically. Systematic releases are similar to intermittent releases with a shorter duration during the normal operating schedule.

- Random Release – these are intermittent releases that can occur any time during the operating schedule. An example of this type of release would be of the type that depends on batch processing. For example, a brake shop may emit pollutants only when the brakes are cleaned which happens randomly throughout the normal business hours.

E. Screening Factors

The U.S. EPA screening factors, as shown in Table H.1, compensate for the effects of varying conditions of wind speed, wind direction, ambient temperature, atmospheric stability, and mixing height over longer averaging periods, even though it is not explicitly indicated in the U.S. EPA Guidance (U.S. EPA, 1992). Figure H.2 shows the variability in wind direction over a 24-hour period. The data are averaged for two seven-day periods from data collected at Los Angeles International Airport (LAX). Figure H.2 was compiled for data collected in 1989 for January 1 to January 7 and June1 through June 7, 1989. The ordinate in Figure H.2 shows the months of the year. Only two months are plotted. The abscissa shows the hour of the day.

Figure H.2
Hourly Wind Direction - Los Angeles
January (bottom – 1) and June (top - 6)



As seen in Figure H.2, the wind direction changes throughout all hours of the day. In addition, the wind direction for LAX, in the overnight and early morning hours, can vary from January to June. During the afternoon hours of 1400 – 1600, the wind direction is similar in both months of January and June.

The standard U.S. EPA screening factor to estimate the maximum 24-hour concentration from the maximum 1-hour concentration is 0.4, as seen in Table H.1. Figure H.2 shows that for 15 of 24 hours the wind blows from the west-northwest during June. A 24-hour screening factor could be 0.6 (0.6 ≈ 15hrs/24hrs) based on wind direction alone. This is consistent with the upper bound of the adjustment factors shown in Table H.1. Including the variability for wind speed, ambient temperature, and atmospheric stability could further reduce the estimated scaling factor of 0.6 closer towards the U.S. EPA recommended value of 0.4.

F. Intermittent Release

Support for the U.S. EPA screening factor is demonstrated for a continuous release (i.e., 24 hours per day) in the description above. It is important to be cautious when applying the U.S. EPA screening factors to an intermittent source for the purposes of estimating an annual average concentration (e.g., a business that may only emit during normal operating hours of 8 am to 6 pm).

Intermittent emissions, such as those from burning barrels, testing a standby diesel generator, or any normal business hour operation (e.g., 8am to 6pm Monday through Friday), could have the effect of eliminating some of the annual variability of meteorological conditions. For example, emissions only during the daytime could eliminate the variability of a drainage flow pattern in mountainous terrain. Guidance for estimating long-term averages for a screening approach and intermittent emissions is not available.

For a source located in the LAX meteorological domain, an emission pattern confined to the hours of 1400 to 1600 would eliminate any variability associated with the wind direction. In this case, estimating a 24-hour average with the U.S. EPA scaling factor of 0.4 would be incorrect.

In the event the emissions are intermittent but randomly distributed throughout the day, the scaling factor of 0.4 may be appropriate because the natural diurnal variability of meteorological conditions are concurrent with emissions. An additional pro-rating of the concentration, when estimating a 24-hour concentration, would be required to discount due to the intermittent nature of the emissions.

We recommend the following steps to estimate a screening based estimate of annual average concentrations from intermittent emissions.

1. Estimate the maximum one-hour concentration ($c_{1\text{-hr}}$) based on the SCREEN3 model approach (or similar, e.g., ISCST3 with screening meteorological data) for possible meteorological conditions consistent with the operating conditions and the actual hourly emission rate. It is acceptable to estimate downwind concentrations using all meteorological combinations available to SCREEN3. However, it is possible to be selective for the choices of meteorological conditions and still be conservative. For example, daytime only emissions need not be evaluated for nighttime stable atmospheric conditions (Pasquill-Gifford classes A through D are unstable and neutral atmospheric conditions applicable during the day. Classes D through F are neutral and stable atmospheric conditions applicable during the night.)

2. Estimate the concentration for the longest averaging period applicable based on the length of time of the systematic or randomly distributed emissions and the factors in Table H.1. For example, the longest averaging period concentration that may be estimated with the U.S. EPA scaling factors is an 8-hour concentration ($c_{8\text{-hr}}$) for emissions that are systematically released for 12 hours. Scaling factors between 8-hours and 12-hours are not available. In the case of the 8-hour concentration, the U.S. EPA screening factor of 0.7 ± 0.2 to estimate the maximum 8-hour concentration is appropriate.

   The U.S. EPA Screening Guidance allows for deviation from the suggested conversion factor on a case-by-case basis. We recommend the lower end of the range for the conversion factor (i.e., 0.5 for the 8-hour average) when estimating an annual average concentration. This is because variability associated with seasonal differences in wind speed, wind direction, and atmospheric stability would not be addressed otherwise. As seen in Figure H.2, there are seasonal differences in the wind direction.

For example, if X is the length of time of systematic or randomly distributed emissions, the following scalars can apply.

- $X \leq 2$ hrs; Scalar = 1.0 to estimate a 1-hour average
- 3 hrs $\leq X \leq$ 7 hrs; Scalar = 0.8 to estimate a 3-hour average
- 8 hrs $\leq X \leq$ 20 hrs; Scalar = 0.5 to estimate an 8-hour average (the selection of 20 hours is arbitrary)
- 21 hrs $\leq X \leq$ 24 hrs; this may be a continuous release, use standard screening procedures.

3. Estimate the annual average concentration (c_{annual}) by assuming the longer averaging period estimated above is persistent for the entire year. In the above example the 8-hour concentration is assumed to be persistent for an entire year to estimate an annual average concentration (i.e., the annual average concentration is assumed to be equal to the 8-hour concentration).

   In addition, the annual average concentration should be pro-rated over the final averaging period based on the pro-rated emissions (i.e., the calculation should include the fact that for some hours over the year, the emission rate is zero).

   For example, if Y is the number of operating hours in the year (e.g., Y = X * 365), the following may apply.
   $(c_{annual}) = (c_{1\text{-}hr})$ (Scalar) (Y/8760hrs/yr)

4. The hourly emission rate should be calculated based on the assumed operating schedule in the steps above. An example for a facility operating Y hours per year follows.
   $(q_{hourly}) = (Q_{yearly})/(Y$ hrs/yr)

5. The annual average concentration (or ground level concentration GLC) can be estimated as follows.
   $GLC = (c_{annual})\ (q_{hourly})$
   $= (c_{1\text{-}hr})$(Scalar) (Yhrs/8760hrs) $(Q_{yearly})/(Y$ hrs/yr)
   $= (c_{1\text{-}hr})$(Scalar) $(Q_{yearly})/(8760$ hrs/yr)

Practically speaking, the above five steps condense down to determining three values. The first value is the maximum 1-hour concentration. The second value is the Scalar (either 1.0, 0.8, or 0.5). And the third value is the hourly emission rate estimated by uniformly distributed over the entire year (8760 hours). The operating hours per year drops out of the calculations for an annual average concentration provided the emissions are based on an annual inventory (See step 5).

In the event that the acute averaging period is required and the emissions are based on an annual inventory, then the annual operating hours are required.

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

Below are four examples using the steps as outlined above. In each case, the annual average concentration is the desired value for use in risk assessment calculations. A fifth example is also included to demonstrate the need for the operating hours per year for an acute analysis when the inventory is provided on an annual basis.

Example 1 - Fugitive Gasoline Station Emissions

Emissions are **continuous** for 24 hours per day and 365 days per year.

1. Estimate the maximum 1-hour concentration with the Screen3 model (or similar screening modeling approach), $c_{1\text{-}hr}$.

2. Estimate the annual average concentration, $c_{annual,}$ with the U.S. EPA screening factor of 0.08.
   $(c_{annual}) = (c_{1\text{-}hr})(0.08)$

3. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 24 hours per day and 365 days per year (8760 hours per year).
   $(q_{hourly}) = (Q_{yearly})/(8760\ hrs/yr)$

4. The annual average concentration (or ground level concentration GLC) can be estimated as follows.
   $GLC = (c_{annual})\ (q_{hourly})$
   $GLC = (c_{1\text{-}hr})(0.08)\ (Q_{yearly})/(8760\ hrs/yr)$

Example 2 - Dry Cleaner Emissions

Emissions are **intermittent** over the year but **systematic** for 10 hours per day, 5 days per week and 50 weeks per year.

1. Estimate the maximum 1-hour concentration with the Screen3 model (or similar screening modeling approach), $c_{1\text{-}hr}$.

2. Estimate the maximum 8-hour average concentration, $c_{8\text{-}hr,}$ with the U.S. EPA screening factor of 0.7 ± 0.2 as the longest averaging period of continuous release. The averaging period would need to be less than 10 hours. Use the lower range of the screening factor, 0.5, because the annual average is the final product and variability due to seasonal differences are not accounted for otherwise.
   $(c_{8\text{-}hr}) = (c_{1\text{-}hr})(0.5)$

3. Assume the worst-case 8-hour concentration is persistent throughout the year and pro-rate the concentration based on emissions over the year. For this dry cleaner, there are 2500 hours of operating condition emissions. Therefore the annual average is calculated as follows.
   $(c_{annual}) = (c_{8\text{-}hr})\ (2500hrs/8760hrs)$
   $= (c_{1\text{-}hr})(0.5)\ (2500hrs/8760hrs)$

4. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 2500 hours per year.
   $(q_{hourly}) = (Q_{yearly})/(2500\ hrs/yr)$

5. The annual average concentration (or ground level concentration GLC) can be estimated as follows.
   $GLC = (c_{annual})\ (q_{hourly})$
   $= (c_{1\text{-}hr})(0.5)\ (2500hrs/8760hrs)\ (Q_{yearly})/(2500\ hrs/yr)$
   $= (c_{1\text{-}hr})(0.5)\ (Q_{yearly})/(8760\ hrs/yr)$

Example 3 - Burning Barrel Emissions

Emissions are **intermittent** over the year and **random** during daylight hours for two hours per burn, two burns per week, and 52 weeks per year.

1. Estimate the maximum 1-hour concentration with the Screen3 model (or similar screening modeling approach), $c_{1\text{-}hr}$. Meteorological combinations may be restricted to daytime conditions for this screening analysis. Pasquill-Gifford stability classes A, B, C, and D are unstable and neutral conditions for daytime conditions.

2. Estimate the maximum 8-hour average concentration, $c_{8\text{-}hr,}$ with the U.S. EPA screening factor of 0.7 ± 0.2 as the longest averaging period where the emissions have the potential to be randomly distributed. Depending on the day of the year and latitude of the emissions, the daylight hours can vary. For this example, we assume the daylight hours can be as short as 10 hours per day to as long as 14 hours per day. Since the emissions are randomly distributed throughout the daylight hours, the longest averaging period we can scale with U.S. EPA scaling factors is a 10 hour average. In this case, the averaging period becomes the 8-hour average and the scaling factor becomes 0.7 ± 0.2. Again since this is for an annual average, we use the lower end of the range, 0.5.
   $(c_{8\text{-}hr}) = (c_{1\text{-}hr})(0.5)$

3. Assume the worst-case 8-hour concentration is persistent throughout the year and pro-rate the concentration based on the emissions over the year. For the burning barrels there are 208 hours of operating condition emissions (208 hrs = (2hrs/burn)(2burns/wk)(52wk/yr)). Therefore the annual average concentration is calculated as follows.
   $(c_{annual}) = (c_{8\text{-}hr})\ (208hrs/8760hrs)$
   $= (c_{1\text{-}hr})(0.5)\ (208hrs/8760hrs)$

4. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 208 hours per year.
   $(q_{hourly}) = (Q_{yearly})/(208\ hrs/yr)$

5. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

$GLC = (c_{annual})\ (q_{hourly})$
$= (c_{1\text{-}hr})(0.5)\ (208hrs/8760hrs)\ (Q_{yearly})/(208\ hrs/yr)$
$= (c_{1\text{-}hr})(0.5)\ (Q_{yearly})/(8760\ hrs/yr)$

Example 4 - Standby Diesel Engine Testing

Emissions are **intermittent** over the year and **systematic** for two hours per week and 50 weeks per year. The engine testing is conducted at 2 pm on Fridays.

1. Estimate the maximum 1-hour concentration with the Screen3 model (or similar screening modeling approach), $c_{1\text{-}hr}$. Meteorological combinations may be restricted to daytime conditions in this screening analysis because the engine test is conducted at 2 pm. Pasquill-Gifford stability classes A, B, C, and D are unstable and neutral conditions for daytime conditions.

2. In this case, the emission schedule is systematically fixed over a two hour period. Therefore, the longest averaging period which is applicable for the U.S. EPA screening factors is one-hour because a two-hour conversion factor is not available. Therefore, we assume the maximum 1-hour concentration is persistent for the entire year. We still prorate the concentration based on the emissions. There are 100 hours of engine testing per year. Therefore the annual average concentration becomes.
$(c_{annual}) = (c_{1\text{-}hr})\ (100hrs/8760hrs)$

3. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 100 hours per year.
$(q_{hourly}) = (Q_{yearly})/(100\ hrs/yr)$

4. The annual average concentration (or ground level concentration GLC) can be estimated as follows.
$GLC = (c_{annual})\ (q_{hourly})$
$= (c_{1\text{-}hr})\ (100hrs/8760hrs)\ (Q_{yearly})/(100\ hrs/yr)$
$= (c_{1\text{-}hr})\ (Q_{yearly})/(8760\ hrs/yr)$

Below is an example using the steps above to estimate an acute concentration longer than a 1-hour averaging period. This case is similar to Example 3 above with the exception of the averaging period.

Example 5 - Burning Barrel Emissions – Acute REL

Emissions are **intermittent** over the year and **random** during daylight hours for two **continuous** hours per burn, two burns per week, and 52 weeks per year. The arsenic acute REL is for a 4-hour averaging period. The steps below are used to estimate the acute concentration, 4-hour REL, for arsenic.

1. Estimate the maximum 1-hour concentration with the Screen3 model (or similar screening modeling approach), $c_{1\text{-}hr}$. Meteorological combinations may be restricted

to daytime conditions for this screening analysis. Pasquill-Gifford stability classes A, B, C, and D are unstable and neutral conditions for daytime conditions.

2. The maximum 1-hour concentration is used as is without screening adjustment factors listed in Tables H.1 or H.2. The emissions are **continuous** through a 2-hour event within a 4-hour window. The adjustments in Table H.2 would only be used if the emissions were continuous for a 4-hour event or **randomly** distributed through a 4-hour event.

3. Assume the worst-case 1-hour concentration is persistent for the 4-hour averaging period and pro-rate the concentration based on the emissions over the 4-hour window. For the burning barrels there are 2 hours of operating condition emissions (2hrs/burn). Therefore the 4-hour average concentration is calculated as follows.
   $(c_{4\text{-hr}}) = (c_{1\text{-hr}})$ (2hrs/4hrs)

4. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 208 hours per year (208 hrs = (2hrs/burn)(2burns/wk)(52wk/yr)).
   $(q_{hourly}) = (Q_{yearly})/(208$ hrs/yr)

5. The 4-hr average concentration (or ground level concentration $GLC_{4\text{-hr}}$) can be estimated as follows.
   $GLC_{4\text{-hr}} = (c_{4\text{-hr}})\ (q_{hourly})$
   $= (c_{1\text{-hr}})$ (2hrs/4hrs) $(Q_{yearly})/(208$ hrs/yr)

   This step of Example 5 differs from the previous Examples because the number of operating hours per year does not drop out of the calculation as seen above.

The above methods were used in a recent modeling evaluation for emissions from a burning barrel (example 3 above) (ARB, 2002). Table H.3, below, shows results from the modeling evaluation. Shown in Table H.3 are the maximum annual average concentration based on the screening approach outlined above as well as a refined approach with site specific meteorological data from four locations, Alturas, Bishop, San Benito, and Escondido. As seen in Table H.3, the screening evaluation as described in the example overestimates the values calculated based on the refined analysis. This is the desired outcome of a screening approach.

Table H.3
Maximum Annual Average Concentration (c/q)
Above Ambient Conditions - Burning Barrel Emissions

Met. City	Alturas	Bishop	San Benito	Escondido	*SCREENING*
D (m)	(mg/m^3)/(g/s)	(mg/m^3)/(g/s)	(mg/m^3)/(g/s)	(mg/m^3)/(g/s)	*(mg/m^3)/(g/s)*
20	44.	61.	85.	110.	*590.*
50	12.	16.	22.	30.	*230.*
100	4.	5.	7.	9.	*85.*

Notes: (a) Annual χ/q is based on 208 hours of emissions at 1 g/s.
(b) χ/q is the concentration in $\mu g/m^3$ based on an hourly emission rate of 1 g/s.

G. Implementation

The approach outlined above has been implemented in the HARP program. Appendix J provides example output files from the Hot Spot Analysis and Reporting Program (HARP). The HARP software has been developed by a contractor through consultation with OEHHA, Air Resources Board (ARB), and District representatives. The HARP software is the recommended model for calculating and presenting HRA results for the Hot Spots Program. Information on obtaining the HARP software can be found on the ARB's web site at www.arb.ca.gov. Note, since the HARP software is a tool that uses the methods specified in this document, the software will be available after these guidelines have undergone public and peer review, been endorsed by the state's Scientific Review Panel (SRP) on Toxic Air Contaminants, and adopted by OEHHA.

References

ARB (1994). ARB memorandum dated 4/11/94 from A. Ranzieri to J. Brooks on the subject, "One-hour to Thirty-day Average Screening Factor."

ARB (2002). Staff Report: Initial Statement of Reasons for the Proposed Airborne Toxic Control Measure to Reduce Emissions of Toxic Air Contaminants from Outdoor Residential Waste Burning, January 2002. California Air Resources Board.

U.S. EPA (1992). Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised, October 1992, EPA-454/R-92-019. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Appendix I

Calculation Examples for

Estimating Potential Health Impacts

Appendix I

Calculation Examples for Estimating Potential Health Impacts

This appendix provides three example calculations to illustrate the procedures to estimate potential health impacts from a facility. The examples provided are intended to assist the risk assessor in understanding the steps associated with conducting the final step of risk assessment, risk characterization. The three examples provided in this appendix evaluate the inhalation cancer risk, the noncancer acute hazard quotient (HQ) and hazard index (HI), and the multipathway (inhalation and oral) noncancer chronic HQ and HI for seven compounds. Specific requirements for health risk assessment (HRA) under the Hot Spots Program are presented in Chapter 8. The HARP software will perform the calculations that are presented here and required in Chapters 8 and 9. See the ARB's website at *www.arb.ca.gov* for more information on HARP.

A. Sample Calculation for Inhalation Cancer Health Risk Assessment

The following example illustrates the steps for calculating cancer risk at the maximum exposed individual resident (MEIR) using the high-end point-estimate for the inhalation exposure pathway. This example does not cover the steps for completing a noninhalation or multipathway HRA. Algorithms to estimate point-estimate and stochastic multipathway exposure can be found in Chapter 5. For simplicity, it is recommended that the risk assessor use HARP to conduct a multipathway risk assessment or stochastic risk assessment.

Step one - Determine the annual average concentration at the MEIR and inhalation cancer potency factor for each emitted compound.

The risk assessor would obtain the annual average concentrations from the air dispersion modeling results. This step has been completed for this example. Table I.1 presents the annual average concentrations at our hypothetical facility. In addition, Table I.1 also presents inhalation cancer potency factors for each substance, which also can be found in Chapter 7 and Appendix L. Note that where no inhalation cancer potency has been developed for a substance, the tables in this example have been annotated with dashes, since it will not be possible to conduct a quantitative risk assessment for these compounds. As previously stated, this example does not take into account multipathway effects for the compounds listed in Table I.1. It is recommended that the risk assessor use HARP for conducting such an analysis.

Table I.1 Annual Average Concentrations at the MEIR and Inhalation Cancer Potency Factors

Substance	Annual Average Concentrations ($\mu g/m^3$)	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$
Ammonia	--	--
Arsenic	0.0015	12
Benzene	5	0.10
Chlorine	--	--
Chlorobenzene	--	--
2,3,7,8-TCDD (dioxin)	0.000004	130,000
Nickel	0.02	0.91

Step two - Determine the inhalation dose for each compound.

Once you have determined the annual average concentration for the emitted substance, the equation below is used to calculate the inhalation dose for each substance. This equation is listed in Section 5.4.1 of this document, and is also listed in the *Air Toxics Hot Spots Risk Assessment Guidelines*; *Part IV; Exposure Assessment and Stochastic Analysis Technical Support Document (OEHHA, 2000b)* (Part IV TSD).

$$\text{dose} - \text{inh} = \frac{(\text{Cair})(\text{DBR})(\text{A})(\text{EF})(\text{ED})(1 \times 10^{-6})}{\text{AT}}$$

Where:

dose-inh	=	Dose through inhalation (mg/kg/d)
$1x10^{-6}$	=	Micrograms to milligrams conversion (10^{-3} mg/µg), liters to cubic meters conversion (10^{-3} m^3/l)
Cair	=	Concentration in air ($\mu g/m^3$)
DBR	=	Daily breathing rate (L/kg body weight-day or L/kg-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 days for 70 year cancer risk)

A summary of the exposure point-estimates and data distributions for use in risk assessment can be found in Chapter 5 of this document. For more detail on point-estimates and data distributions see the Part IV TSD. The recommended default values presented in Table I.2 can be used when site-specific information is not available.

Table I.2 Recommended Default Values

Variable	Recommended Default Value
EF	350 days/year
ED	9; 30; or 70 years
AT	70 years (25,550 days)
DBR (used in this example) 30 and 70 year-exposure	271 (mean); 393 (95th percentile) L/kg body weight – day (For other DBRs see Table 5.4, Chapter 5)
A	1 (currently used for all substances included in the Hot Spots program)

The following equation shows the calculation for the inhalation dose of arsenic by using the annual average concentration for arsenic (Table I.1) and the recommended default values in Table I.2. Note that the high-end (95th percentile) 70-year daily breathing rate of 393 liters/kg - day was used in this example.

$$\text{arsenic}\ (\text{dose}-\text{inh}) = \frac{\left(\frac{0.0015\,mg}{m^3}\right)\left(\frac{393\,liters}{kg-day}\right)(1)\left(\frac{350\ days}{year}\right)(70\ years)\left(\frac{1\text{x}10^{-3}\ mg}{1mg}\right)\left(\frac{1\text{x}10^{-3}\ m^3}{liters}\right)}{25{,}550\ days}$$

$$\text{arsenic}\ (\text{dose}-\text{inh}) = 5.7\times10^{-7}\,mg/kg-day$$

This calculation would be repeated for each substance under evaluation using their respective annual average concentrations. For our hypothetical facility, we have calculated each inhalation dose for each substance. Table I.3 shows the results from our analysis.

Table I.3 Calculated Doses for Substances

Compound	Calculated Dose
Ammonia	--
Arsenic	5.7 x 10^{-7}
Benzene	1.9 x 10^{-3}
Chlorine	--
Chlorobenzene	--
2,3,7,8-TCDD (dioxin)	1.5 x 10^{-9}
Nickel	7.5 x 10^{-6}

Step three – Determine potential inhalation cancer risk for the MEIR.

Once you have calculated the inhalation dose, multiply the dose by the inhalation cancer potency factor as shown below. Use a factor of $1x10^6$ to express cancer risk in chances per million.

$$\left(\text{Inhalation Dose } \frac{mg}{kg-day}\right)\left(\text{Cancer Potency } \frac{kg-day}{mg}\right)\left(1 \times 10^6\right) = \text{Cancer Risk (chances per million)}$$

For our hypothetical facility, the equation below shows the calculation for the inhalation cancer risk of arsenic. For this example, the inhalation cancer potency factor for arsenic is 12 $(mg/kg\text{-}d)^{-1}$ taken from Table I.1.

$$\left(5.7 \times 10^{-7} \frac{mg}{kg-day}\right)\left(12 \frac{kg-day}{mg}\right)\left(1 \times 10^6\right) = 6.8 \text{ chances per million}$$

Use the substance-specific inhalation dose and inhalation cancer potency factor to determine the cancer risk for each compound by repeating this step. Finally, sum the individual substance cancer risks to give you the total facility (inhalation) cancer risk. Table I.4 shows the individual substance and total facility potential (inhalation) cancer risk. In this example, our hypothetical facility poses a (inhalation) cancer risk of 399 chances per million at the MEIR. Note, although not presented here, a facility emitting arsenic or dioxins should also evaluate cancer risk from noninhalation exposure pathways.

Table I.4 Hypothetical Facility Inhalation Cancer Risk

Compound	Cancer risk (per million)
Ammonia	--
Arsenic	6.8
Benzene	190
Chlorine	--
Chlorobenzene	--
2,3,7,8-TCDD (dioxin)	195
Nickel	6.8
Total Facility Inhalation Cancer Risk	**399**

While this example illustrates the steps used to calculate cancer risk using the inhalation dose algorithm, steps one through three can also be used to calculate noninhalation cancer risk and ultimately multipathway (inhalation and noninhalation pathway) cancer risk. To determine noninhalation cancer risk, an assessor should use the appropriate exposure pathway algorithm presented in Chapter 5. For example, equation 5.4.3.1.A (Chapter 5) would be used to determine

dose for the soil ingestion pathway. Once the assessor has determined the ingestion dose, the cancer risk for that pathway is calculated using the substance-specific oral slope factor. Oral slope factors can be found in Appendix L and Chapter 7. To calculate multipathway cancer risk, the cancer risks for all substances and exposure pathways are summed. See Chapter 8 for further discussion.

B. Sample Calculation of Noncancer Acute Hazard Indices

Risk characterization for noncancer health impacts are expressed as a hazard quotient (for individual substances) or a hazard index (for multiple substances). In addition, all hazard quotients (HQ) and hazard indices (HI) must be determined by target organ system. The example below illustrates the approach for calculating a noncancer acute HQ and HI at the MEIR. As discussed in Chapter 8, the following example is provided to assist the risk assessor in understanding how to calculate an acute HQ and HI. Using HARP, both the acute HQ and HI will be automatically calculated at each receptor. No exposure duration adjustment should be made for noncancer assessments. Specific requirements for risk assessment under the Hot Spots Program can be found in Chapters 8 and 9.

Step one - Determine the 1-hour maximum concentrations at the MEIR and acute reference exposure levels (RELs) for each emitted substance.

The risk assessor would obtain the 1-hour maximum (or 4, 6, or 7-hour, if required) concentrations from the air dispersion modeling results. This step has been completed for this example. Table I.5 presents the maximum 1, 4, 6, or 7-hour concentrations, target organ systems, and acute RELs for seven substances. Note that where an acute REL has not been developed for a substance, the tables in this example have been annotated with dashes. In this

Table I.5 Concentrations, Acute RELs, and Target Organ System(s) for Substances at the MEIR

Substance	1, 4, 6 or 7-hour Maximum Concentration ($\mu g/m^3$)	Acute REL ($\mu g/m^3$)	Target Organ System(s)
Ammonia	1900	3200	Respiratory system; Eye
Arsenic	0.03	0.19	Reproductive/developmental
Benzene	20	1300	Reproductive/developmental; Immune system; Hematologic system
Chlorine	40	210	Respiratory system; Eye
Chlorobenzene	--	--	--
2,3,7,8-TCDD (dioxin)	--	--	--
Nickel	1.8	6	Respiratory system; Immune system

example, chlorobenzene and 2,3,7,8-TCDD (dioxin) do not have acute REL values. The acute RELs and their corresponding target organ system(s) can be found in Table 6.1 (Chapter 6) and also in Appendix L.

Step two - Determine the hazard quotient for each compound.

The hazard quotients for each compound are calculated by taking the acute maximum 1, 4, 6, or 7-hour concentration and dividing by the substance-specific acute REL. The following equation shows how to calculate the hazard quotient for ammonia.

$$\text{Acute Hazard Quotient} = \frac{\left(\begin{matrix}Maximum\ 1,\ 4,\ 6,\ or\ 7-hr\\ Concentration\end{matrix}\right)}{\left(\begin{matrix}Acute\\ REL\end{matrix}\right)} \Rightarrow \text{Acute Hazard Quotient}_{(\text{ammonia})} = \frac{\left(1900\ mg/m^3\right)}{\left(3200\ mg/m^3\right)} = 0.6$$

Step three – Determine the HI for all emitted substances.

The acute HI is calculated by summing each hazard quotient for each substance by target organ system(s). For example, add the HQs for all substances that impact the respiratory system, then repeat this step for the next target organ system (e.g., reproductive/developmental system). This step is repeated until all target organs (for the substances emitted) are individually totaled. See Table 6.1 for target organ system information. Note, never add together the HQs or HIs for different target organ systems (e.g., do not add the impacts for the respiratory system to the reproductive/developmental system). Table I.6 shows individual hazard quotients for each substance and total hazard index. {Bob, adding benzene (6-hr) and arsenic (4-hr) below OK?]

Table I.6 Individual Hazard Quotients and Total Hazard Index

Substance	Immune System	Reproductive/ Developmental	Hematologic System	Respiratory System	Eye
Ammonia				0.6	0.6
Arsenic		0.2			
Benzene	0.02	0.02	0.02		
Chlorine				0.2	0.2
Chlorobenzene					
2,3,7,8-TCDD (dioxin)					
Nickel	0.3			0.3	
Total Hazard Index	**0.32**	**0.22**	**0.02**	**1.1**	**0.8**

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

In this example, an HQ of one was not equaled or exceeded for any individual substance. However, an HI (the sum of the hazard quotients for each target organ) of one was exceeded for the respiratory system. Exceeding a hazard index of one may indicate that there is the potential for adverse acute health impacts at this receptor location. Therefore, there is increased concern that exposed individuals may experience respiratory system irritation, particularly among sensitive individuals. The District and OEHHA should be consulted when a hazard index exceeds one (see Section 8.3).

C. Sample Calculation of Noncancer Chronic Hazard Indices

The example below illustrates the approach for calculating a noncancer chronic HQ and HI at the MEIR. An HQ expresses the noncancer health impacts for an individual substance and an HI expresses the potential impacts for multiple substances. As discussed in Chapter 8, the following example is provided to assist the risk assessor in understanding the calculation of a chronic HQ and HI. Using the HARP software, both the chronic HQ and HI will be automatically calculated at each receptor. No exposure duration adjustment (e.g., 9/70) should be made for noncancer assessments. Specific requirements for risk assessment under the Hot Spots Program can be found in Chapters 8 and 9.

Step one - Determine the annual average concentrations at the MEIR and inhalation and oral chronic RELs for each emitted substance.

The risk assessor would obtain the substance-specific annual average concentrations from the air dispersion modeling results. This step has been completed for this example. Table I.7 presents the annual average concentrations, target organ systems, and chronic RELs for seven substances. All of the substances have a chronic REL value associated with them. In addition, arsenic, dioxins, and nickel are multipathway substances; therefore, oral and dermal exposure must be included as potential exposure pathways. The chronic RELs and their corresponding target organ system(s) can be found in Tables 6.2 and 6.3 (Chapter 6) and also in Appendix L.

Table I.7 Annual Average Concentrations, Chronic RELs, and Target Organ Systems for Substances at the MEIR.

Substance	Annual Average Conc. ($\mu g/m^3$)	Chronic REI (inhalation) ($\mu g/m^3$)	Target Organ System(s) (inhalation)	Chronic Oral REL (mg/kg-day)	Target Organ System(s) (oral/dermal)
Ammonia	160	200	Respiratory System	-	-
Arsenic	0.0015	0.03	Development; Cardiovascular System; Nervous System	0.0003	Cardiovascular system; skin
Benzene	5	60	Hematopoietic System; Development; Nervous System	-	-
Chlorine	0.08	0.2	Respiratory System	-	-
Chlorobenzene	20	1000	Alimentary System; Kidney; Reproductive System	-	-
2,3,7,8-TCDD (dioxin)	0.000004	0.00004	Alimentary System (Liver); Reproductive System; Development; Endocrine System; Respiratory System; Hematopoietic System	0.00000001 (10 pg/kg-day)	Alimentary System (Liver); Reproductive System; Development; Endocrine System; Respiratory System; Hematopoietic System
Nickel	0.02	0.05	Respiratory System; Hematopoietic System	0.05	Alimentary System

Step two – Determine the inhalation chronic hazard quotient for each substance.

For inhalation exposure, the individual hazard quotients for each substance are calculated by taking the annual average concentration and dividing by its corresponding chronic inhalation REL. Using the information contained in Table I.7, the equation below is used to calculate the inhalation hazard quotient for arsenic.

$$\text{Chronic Hazard Quotient} = \frac{\left(\begin{matrix}\text{Annual avg.}\\ \text{Concentration}\end{matrix}\right)}{\left(\begin{matrix}\text{Chronic}\\ \text{REL}\end{matrix}\right)} \Rightarrow \text{Chronic Hazard Quotient}_{(arsenic)} = \frac{\left(0.0015\ \mu g/m^3\right)}{\left(0.03\ \mu g/m^3\right)} = 0.05$$

Step three – Determine the noninhalation hazard quotient for each substance.

For the substances that are subject to deposition, noninhalation (i.e., oral and dermal) exposure pathways need to be considered in the chronic hazard quotient evaluation. The point-estimates and algorithms for calculating the oral dose for all of the applicable exposure

pathways and receptors (e.g., workers or residents) are explained in Chapter 5. Note, the HARP software uses the appropriate information and performs all the steps discussed in these examples.

As discussed in Sections 8.2.5 and 8.3, Tier-1 of the tiered approach to risk assessment states that the high-end point-estimates are used for the two dominant noninhalation exposure pathways and the non-dominant exposure pathways use the average point-estimates to determine the dose and chronic health impacts at a residential receptor. To determine which exposure pathways are the two dominant ones, high-end point-estimates are used for all applicable exposure pathways to see which two pathways provide the highest impacts for each substance. Once the two dominant noninhalation pathways are determined for each substance, the doses for the remaining noninhalation exposure pathway for that substance are recalculated using the average point-estimates. The 70-year exposure duration point-estimates are used for residential receptors and the worker (single) point-estimates are used for the maximum exposed worker (see Chapter 5). No exposure duration adjustment (e.g., 9/70) should be made for noncancer assessments.

This example shows how to combine the impacts from multiple exposure pathways to obtain an oral (noninhalation) hazard quotient for a single substance. For each substance, the impacts for a specific exposure pathway are assessed by dividing the oral dose (derived from the annual average concentration) in milligrams per kilogram-day (mg/kg-day) by the oral chronic REL, expressed in units of (mg/kg-day) (Table 6.3). The next equation shows the HQ calculation for arsenic through the soil ingestion (SI) exposure pathway.

Note, prior to this point in this calculation, we are assuming several steps have taken place. These steps include: 1) the completion of air dispersion modeling to obtain the ground-level annual-average air concentration; 2) identification of the existing exposure pathways at the receptor location; 3) calculation of the concentration in the exposure media (e.g., for soil - Equation 5.3.2.A); 4) determination of the dominant noninhalation exposure pathway(s) for the substance; and 5) the calculation of the substance-specific dose for that exposure pathway (e.g., Equation 5.4.3.1.A is used to calculate the dose from soil ingestion). See Chapter 5 for the algorithms for calculating the oral dose for all of the applicable exposure pathways and receptors. For this example, the calculated dose for arsenic from soil ingestion is assumed to be 0.000015 mg/kg-day.

$$\text{Chronic Oral Hazard Quotient} = \frac{SI\ dose}{\left(\frac{Chronic}{Oral\ REL}\right)} \Rightarrow \text{Chronic Oral Hazard Quotient}_{(SI\ arsenic)} = \frac{(0.000015\ mg/kg-day)}{(0.0003\ mg/kg-day)} = 0.05$$

For each substance, this step is repeated for each applicable noninhalation exposure pathway. As illustrated below, the (total) oral HQ for a substance is calculated by summing the HQs for all applicable exposure pathways. In this example, the chronic oral HQ is assumed to equal 0.1.

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$$\text{Chronic Oral Hazard Quotient*}_{(\text{arsenic})} = [HQ_{(SI)} + HQ_{(D)} + HQ_{(DW)} + HQ_{(MI)} + HQ_{(FI)} + HQ_{(HV)}]$$

$$\text{Chronic Oral Hazard Quotient*}_{(\text{arsenic})} = 0.1$$

* Noninhalation pathways:

SI = soil ingestion	FI = fisher-caught fish
DW = drinking water	HV= homegrown vegetables
D = dermal absorption	BM= breast milk (not applicable for arsenic exposure)
MI = meat, milk & egg	

Step four – Determine the chronic HI

The chronic HI is calculated by summing each hazard quotient (inhalation and noninhalation) for each substance by the target organ system(s). For example, add the HQs for all substances that impact the respiratory system, then repeat this step for the next target organ

Table I.8 Substance-Specific Inhalation and Noninhalation Hazard Quotients and the Hazard Index by Target Organ System

Substance	Respiratory System	Hematopoietic System	Alimentary System	Endocrine System	Development	Reproductive System	Kidney	Nervous System	Cardiovascular System	Skin
Ammonia	0.8									
Arsenic					***0.05(i)***			***0.05(i)***	***0.05(i)*** ***0.1(ni)***	***0.1(ni)***
Benzene		0.08			0.08			0.08		
Chlorine	0.04									
Chlorobenzene			0.02			0.02	0.02			
2,3,7,8-TCDD (dioxin)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)				
Nickel	0.4(i)	0.4(i)	0.1(ni)							
Hazard Index	**1.54**	**0.78**	**0.32**	**0.3**	**0.43**	**0.32**	**0.02**	**0.13**	**0.15**	**0.1**

i = inhalation pathway contribution
ni = noninhalation pathway contribution

system (e.g., cardiovascular system). This step is repeated until all target organs (for the substances emitted) are individually totaled. See Tables 6.2 and 6.3 for target organ system information. Note, never add together the HQs or HIs for different target organ systems (e.g., do not add the impacts for the respiratory system to the cardiovascular system). No exposure duration adjustment (e.g., 9/70) should be made for noncancer assessments. Table I.8 shows individual hazard quotients (inhalation and noninhalation) for each substance and the hazard index by target organ system. In this table, arsenic is highlighted in bold to identify how the information calculated above is presented and used.

In this example, an HQ of one was not equaled or exceeded for any individual substance. However, an HI (the sum of the hazard quotients for each target organ) of one was exceeded for the respiratory system. Exceeding a hazard index of one may indicate that there is the potential for adverse chronic health impacts at this receptor location. Therefore, there is increased concern that exposed individuals may experience respiratory system irritation or injury, particularly among sensitive individuals. The District and OEHHA should be consulted when a hazard index exceeds one (see Section 8.3).

Appendix J

Glossary Of Acronyms and Definition of Selected Terms

Glossary of Acronyms and Definitions of Selected Terms

Acute Exposure: One or a series of short-term exposures generally lasting less than 24 hours.

Acute Health Effects: A health effect that occurs over a relatively short period of time (e.g., minutes or hours). The term is used to describe brief exposures and effects which appear promptly after exposure.

Adverse Health Effect: A health effect from exposure to air contaminants that may range from relatively mild temporary conditions, such as eye or throat irritation, shortness of breath, or headaches, to permanent and serious conditions, such as birth defects, cancer or damage to lungs, nerves, liver, heart, or other organs.

AERMOD: a proposed (by U.S. EPA) steady-state, plume-based air dispersion model for estimating near-field impacts from a variety of industrial source types (designed to provide reasonable concentration estimates over a wide range of conditions with minimal discontinuities, to be easily implemented with reasonable input requirements and computer resource needs, to be based on up-to-date science that captures the essential physical processes while remaining simple, and to be easily revised as the science evolves). To the extent practicable, the structure of the input or the control file for AERMOD is the same as that for ISCST3.

Air Dispersion Modeling: Algorithms, usually performed with a computer, that relate a mass emission rate, source configuration, and meteorological information to calculate ambient air concentrations.

Air District: The Air Pollution Control and Air Quality Management Districts are the political bodies responsible for managing air quality on a regional or county basis. California is currently divided into 35 air districts.

Air monitoring: The periodic or continuous sampling and analysis of air pollutants in ambient air or from individual pollutant sources.

Air Toxics Hot Spots Act Emission Inventory Reports: Documents that contain information regarding emission sources, emitted substances, emission rates and release parameters, prepared under the Emission Inventory Criteria and Guidelines (also referred to as "Inventory Reports").

Air Toxics Hot Spots Information and Assessment Act of 1987 (AB 2588): (Health and Safety Code, Section 44300-44394) - A state law which established the "Hot Spots" Program to develop a statewide inventory of site-specific air toxic emissions, to assess the risk to public health from exposure to these emissions, to notify the public of any significant health risks and to reduce emissions below the significant risk levels.

Algorithm: a set of rules for solving a problem in a finite number of steps

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California Air Resources Board (ARB): The State's lead air quality management agency consisting of an eleven-member board appointed by the Governor. The ARB is responsible for attainment and maintenance of the state and federal air quality standards, and is fully responsible for motor vehicle pollution control. It oversees county and regional air pollution management programs.

Asthma: A chronic inflammatory disorder of the lungs characterized by wheezing, breathlessness, chest tightness, and cough.

Atmospheric half-life: The time required for the concentration of a pollutant or reactant to fall to one-half of its initial value.

Benchmark Dose: That dose derived from linear regression of one or more dose-response curves associated with a specific response rate (such as 1, 5, or 10%) in the test population. This is the starting dose to which uncertainty factors are applied to determine a reference exposure level (REL) using the benchmark dose approach.

Urban Block Groups (BGs): A geographical unit smaller than a census tract used for reporting census data. BGs contain roughly 1,100 persons.

Bioaccumulation: the concentration of a substance in a body or part of a body or other living tissue in a concentration higher than that of the surrounding environment

Bioconcentrate: The process of increasing contaminant concentration in biota up the food chain as contaminants are ingested and concentrated in tissues of organisms higher up in the chain.

Cancer burden: The estimated number of theoretical cancer cases in a defined population resulting from lifetime exposure to pollutants emitted from a facility.

Cancer potency factor (CPF): The theoretical upper bound probability of extra cancer cases occurring in an exposed population assuming a lifetime exposure to the chemical when the chemical dose is expressed in exposure units of milligrams/kilogram-day (mg/kg-d).

California Air Pollution Control Officers Association (CAPCOA): A non-profit association of the air pollution control officers from all 35 air quality districts throughout California. CAPCOA was formed in 1975 to promote clean air and to provide a forum for sharing knowledge, experience, and information among the air quality regulatory agencies around the state.

Cal/EPA: In July 1991, the California Environmental Protection Agency (Cal/EPA) was created to coordinate the State's environmental quality programs and assure that there is a cabinet level voice for environmental protection.

Chemical Abstract Services Registry Number (CAS): The Chemical Abstracts Service Registry Number (CAS) is a numeric designation assigned by the American Chemical Society's Chemical Abstracts Service and uniquely identifies a specific chemical compound. This entry allows one to conclusively identify a material regardless of the name or naming system used.

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CCR: California Code of Regulations

CERCLA: Comprehensive Environmental Response, Compensation and Liability Act (Superfund), a federal regulation providing direction and financial support for the clean-up of major hazardous waste sites

Centroid Locations: The location at which calculated ambient concentration is assumed to represent the entire subarea, typically the geometric centroid of an area, but possibly the population-weighted centroid of the area.

Census Tract: A physical area used by the U.S. Census Bureau to compile population and other statistical data.

Chronic Exposure: Long-term exposure, usually lasting one year to a lifetime.

Chronic Health Effect: An adverse non-cancer health effect that develops and persists (e.g., months or years) over time after long-term exposure to a substance

Criteria Air Pollutant: a pollutant or precursor to a pollutant for which the U.S. Environmental Protection Agency or the Air Resources Board has established an Ambient Air Quality Standard (AAQS). Examples include ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, lead, and PM_{10} and $PM_{2.5}$.

Default: A value used when specific information that applies to a specific situation is not available.

Developmental toxicity: Adverse effects on the developing organism that may result from exposure prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism. Major manifestations of developmental toxicity include: death of the developing organism; induction of structural birth defects; altered growth; and functional deficiency.

Dilution factor (c/Q): a site-specific quantity defined as a ratio of the ground level concentration in $\mu g/m^3$ to the mass emission rate in g/s and represented by c/Q.

Dose: A calculated amount of a substance estimated to be received by the subject, whether human or animal, as a result of exposure. Doses are generally expressed in terms of amount of chemical per unit body weight; typical units are mg/kg-day.

Dose-response assessment: The process of characterizing the relationship between the exposure to an agent and the incidence of an adverse health effect in exposed populations.

DTSC: California Department of Toxic Substances Control

ED: Rural Enumeration District. A geographical unit smaller than a census tract used to report census data. EDs contain roughly 1,100 persons.

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Emission Inventory Criteria and Guidelines: Regulation and Report adopted by the California Air Resources Board specifying criteria and procedures for the preparation of Air Toxics Hot Spots Act Emission Inventory Reports (Title 17, California Code or Regulations, Sections 93300-93300.5)

Endpoint: An observable or measurable biological or biochemical event including cancer used as an index of the effect of a chemical on a cell, tissue, organ, organism, etc.

Epidemiology: The study of the occurrence and distribution of a disease or physiological condition in human populations and of the factors that influence this distribution.

Exposure: Contact of an organism with a chemical, physical, or biological agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, digestive tract) and available for absorption.

Exposure Pathway: A route of exposure by which xenobiotics enter the human body, (e.g., inhalation, ingestion, dermal absorption).

Fugitive Dust: Dust particles that are introduced into the air through certain activities such as soil cultivation, or vehicles operating on open fields or dirt roadways. A subset of fugitive emissions.

Fugitive Emissions: Emissions not caught by a capture system which are often due to equipment leaks, evaporative processes, and windblown disturbances.

Gaussian Model: An air dispersion model based on the assumption that the time-averaged concentration of a species emitted from a point source has a Gaussian distribution about the mean centerline.

Genotoxic: having an adverse effect on the genetic material (DNA) resulting in a mutation or in chromosome damage

GLC: Estimated ground level concentration, usually for a specified averaging time (e.g., annual average, 1 hour, etc.)

Hot Spots Analysis and Reporting Program (HARP): A single integrated software package designed to promote statewide consistency, efficiency, and cost-effective implementation of health risk assessments and the Hot Spots Program. The HARP software package consists of three modules that include: 1) the Emissions Inventory Database Module, 2) the Air Dispersion Modeling Module, and 3) the Risk Analysis and Mapping Module.

Health Risk Assessment (HRA): the name of a computer program developed by the ARB, the OEHHA, and the University of California which was designed to aid in the computation of risk in the Hot Spots program

HSC: Health and Safety Code of the State of California

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Haber's Law: The product of the concentration (C) and time of exposure (t) required to produce a specific physiologic effect is equal to a constant level or severity of response (K), or C * t = K

Hazard identification: The process of determining whether exposure to an agent can cause an increase in the incidence of an adverse health effect including cancer

Health Risk Assessment: A health risk assessment (HRA) is an evaluation or report that a risk assessor (e.g., Air Resources Board, district, consultant, or facility operator) develops to describe the potential a person or population may have of developing adverse health effects from exposure to a facility's emissions. Some health effects that are evaluated could include cancer, developmental effects, or respiratory illness. The pathways that can be included in an HRA depend on the toxic air pollutants that a person (receptor) may be exposed to, and can include breathing, the ingestion of soil, water, crops, fish, meat, milk, and eggs, and dermal exposure.

Health Risk Guidance Value (HRGV): A numerical value with which to compare an exposure level in order to determine the probability of occurrence of an adverse health effect. In the Hot Spots program the toxicity criteria or toxicity values are known as Reference Exposure Levels (RELs) for noncancer effects and as inhalation unit risk factors and cancer potency values for cancer effects.

Hazard Index (HI): The sum of individual acute or chronic hazard quotients (HQs) for each substance affecting a particular toxicological endpoint.

Hazard Quotient (HQ): The estimated ground level concentration divided by the reference exposure level for a single substance and a particular endpoint. For an acute HQ the one hour maximum concentration is divided by the acute Reference Exposure Level for the substance. For a chronic HQ, the annual concentration is divided by the chronic Reference Exposure level.

Hot Spot: A location where emissions from specific sources may expose individuals and population groups to elevated risks of adverse health effects, including but not limited to cancer, and contribute to the cumulative health risks of emissions from other sources in the area.

Individual Excess Cancer Risk: The theoretical probability of an individual person developing cancer as a result of lifetime exposure to carcinogenic substances. The Individual Excess Cancer Risk is calculated by summing the potential cancer risks due to both inhalation and noninhalation routes of exposure.

Inhalation (Breathing) Rate: The amount of air inhaled in a specified time period (e.g., per minute, per hour, per day, etc.); also called breathing rate and ventilation rate. This is an example of a variate.

Inhalation unit risk factor: The theoretical upper bound probability of extra cancer cases occurring in the exposed population assuming a lifetime exposure to the chemical when the air concentration is expressed in exposure units of per microgram/cubic meter $(\mu g/m^3)^{-1}$.

Initiator carcinogen: A substance which causes the first stage of carcinogenesis, the conversion of a normal cell to a neoplastic cell. Initiation is considered to be a rapid, irreversible change often involving a change in the DNA caused by the initiator.

Interspecies: Between different species.

Intraspecies: Within the same species.

Industrial Source Complex Dispersion model (ISC3): Air modeling software that incorporates three previous programs into a single program. These are the short-term model (ISCST), the long term model (ISCLT), and the complex terrain model (COMPLEX).

Isopleth: A line on a map connecting points of equal value (e.g., risk, concentration, etc.).

Lowest-observed adverse effect level (LOAEL): The lowest dose or exposure level of a chemical in a study at which there is a statistically or biologically significant increase in the frequency or severity of an adverse effect in the exposed population as compared with an appropriate, unexposed control group.

Margin of safety: The ratio of the no-observed-adverse-effect level (NOAEL) to the estimated human exposure.

Mean: The arithmetic average.

MEI: Maximum exposed individual (theoretical)

MEIR: Maximum exposed individual resident (actual)

MEIW: Maximum exposed individual worker (actual)

Meteorology: The science that deals with the phenomena of the atmosphere especially weather and weather conditions. In the area of air dispersion modeling, *meteorology* is used to refer to climatological data needed to run an air dispersion model including: wind speed, wind direction, stability class and ambient temperature.

Milligram: One one-thousandth (10^{-3}) of a gram.

Molecular formula: The formula which identifies the atoms and the number of each kind in the molecules of a compound. Elements in the molecular formula are listed according to the Hill convention (C, H, then other elements in alphabetical order).

Molecular weight: The sum of the atomic weights of the atoms in a molecule. For example, methane (CH_4) is 16.043, the atomic weights being carbon = 12.011, hydrogen = 1.008.

Monte Carlo simulation: Application of random sampling to obtain an approximate value of an expression.

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Multipathway substance: A substance or chemical that once airborne from an emission source can, under environmental conditions, be taken into a human receptor by inhalation and by other exposure routes such as after deposition on skin or after ingestion of soil contaminated by the emission

No Observed Adverse Effect Level (NOAEL): The highest experimental dose at which there is no statistically or biologically significant increase in frequency or severity of adverse health effects in the exposed population compared with an appropriate, unexposed population. Effects may be produced at this level, but they are not considered to be adverse. Substances are generally considered to not have a NOAEL for the cancer endpoint.

Noncarcinogenic Effects: Noncancer health effects which may include birth defects, organ damage, morbidity, and death.

Office of Environmental Health Hazard Assessment (OEHHA): An office within the California Environmental Protection Agency that is responsible for evaluating chemicals for adverse health impacts and establishing safe exposure levels. OEHHA also assists in performing health risk assessments and developing risk assessment procedures for air quality management purposes.

PM_{10}, $PM_{2.5}$: PM_{10} is particulate matter less than 10 μm in diameter; $PM_{2.5}$ is particulate matter less than 2.5 μm in diameter.

PMI: Off-site point of maximum impact. A location, with or without people currently present, at which the total cancer risk, or the total noncancer risk, has the highest numerical value.

Point Estimate: A single value estimate for a given variate

Potency: Essentially the relative effectiveness, or risk, of a standard amount of a substance to cause a toxic response.

Potency Slope: Used to calculate the probability or risk of cancer associated with an estimated exposure, based on the assumption in cancer risk assessments that risk is directly proportional to dose and that there is no threshold for carcinogenesis. It is the slope of the dose-response curve estimated at low exposures.

Proposition 65: Safe Drinking Water and Toxic Enforcement Act of 1986, also known as Proposition 65. This Act is codified in California Health and Safety Code Section 25249.5, et seq. No person in the course of doing business shall knowingly discharge or release a chemical known to the state to cause cancer or reproductive toxicity into water or into land where such chemical passes or probably will pass into any source of drinking water, without first giving clear and reasonable warning to such individual.

Resource Conservation and Recovery Act (RCRA) of 1976: A federal law regulating disposal of hazardous waste

Receptor: A location with or without people present at which the ground level concentration of an emitted chemical can be estimated

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Refined Models: Air dispersion models designed to provide more representative concentration estimates than screening models taking into account actual meteorological conditions.

Reference Concentration (RfC): An estimate, derived by the U.S. EPA (with an uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population, (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime of exposure. The RfC is derived from a no or lowest observed adverse effect level from human or animal exposures, to which uncertainty or "safety" factors are applied.

Reference Dose (RfD): An estimate delivered by the U.S. EPA (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subpopulations) that is likely to be without deleterious effects during a lifetime. The RfD is reported in units of mg of substance/kg body weight/day for oral exposures.

Reference exposure level (REL): expressed in units of $\mu g/m^3$ for inhalation exposures and of mg/kg-d for noninhalation exposures. The REL is an exposure level at or below which no noncancer adverse health effect is anticipated to occur in a human population exposed for a specific duration. An REL is virtually the same as the terms Reference Concentration (RfC) for inhalation or Reference Dose (RFD) used by U.S. EPA, only it may be for varying amounts of time rather than lifetime only. It has been given a different name so that the values estimated by the State Office of Environmental Health Hazard Assessment can easily be distinguished from those developed by the U.S. EPA. RELs are used to evaluate toxicity endpoints other than cancer.

Reproductive toxicity: Harmful effects on fertility, gestation, or offspring, caused by exposure of either parent to a substance.

Risk: The (characterization of the) probability of potentially adverse effects to human health, in this instance from the exposure to environmental hazards.

Risk Assessment: The characterization (in the present context) of the probability of potentially adverse health effects to people from exposure to environmental chemical hazards.

Risk Management: An evaluation of the need for and feasibility of reducing risk. It includes consideration of magnitude of risk, available control technologies, and economic feasibility.

Risk Management and Prevention Program (RMPP): A program administered by the Office of Emergency Services (OES) and local agencies to reduce the frequency and severity of accidental releases of toxic materials

Scientific Review Panel on Toxic Air Contaminants or SRP: A nine-member panel appointed to advise the Air Resources Board and the Department of Pesticide Regulation in their evaluation of the adverse health effects toxicity of substances being evaluated as Toxic Air Contaminants.

Screening Models: Dispersion models used to provide a maximum concentration that is likely to overestimate public exposure.

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Sensitive Receptor: A location such as a hospital or daycare center where the human occupants are considered to be more sensitive to pollutants than "average".

Severity Level: The (acute) discomfort or mild effect level; the concentration of an airborne substance at or below which exposure for one hour may result in some odors, tastes, visual cues, and sensations but which will cause no adverse health effects in essentially all of the population. Exposure to concentrations above this level, depending on the chemical, may result in minor health effects, such as mild eye and respiratory irritation, skin irritation, minor histologic effects, and headaches.

Severity Level II: The (acute) disability or serious effect level. Exposure for one hour to an airborne substance above this level may lead individuals to seek assistance. Exposures above this level, depending on the chemical, may result in serious health effects such as severe eye irritation, severe respiratory irritation, bronchospasm, shortness of breath, disorientation, blurred vision, vomiting, cardiac arrhythmia and adverse outcomes of an existing or subsequent pregnancy.

Stationary source: A non-mobile source of air pollutants which can be either a point or area source.

Stochastic: A process that involves random variation

Synergism: A pharmacologic or toxicologic interaction in which the combined effect of two or more chemicals is greater than the sum of the effects of each chemical alone.

Subcensus Tract: Smaller population unit within a census tract.

Surrogate: As used in this document refers to a single substance category used to represent a family of related chemical compounds, e.g., gasoline vapors or POM (polycyclic organic matter) in place of benzo(a)pyrene.

Threshold, Nonthreshold: A threshold dose is the minimally effective dose of any chemical that is observed to produce a response (e.g., enzyme change, liver toxicity, death). For most toxic effects, except carcinogenesis, there appear to be threshold doses. Nonthreshold substances are those substances, including nearly all carcinogens, that are known or assumed to have some risk of response at any dose above zero.

Toxic air contaminant (TAC): As defined by California Health and Safety Code, Section 39655 (a): an air pollutant which may cause or contribute to an increase in mortality or in serious illness, or which may pose a present or potential hazard to human health. Substances, which have been identified by the United States Environmental Protection Agency as hazardous air pollutants (e.g. benzene, asbestos), shall be identified by the Board as toxic air contaminants.

Toxicology: The multidisciplinary study of toxicants, their harmful effects on biological systems, and the conditions under which these harmful effects occur. The mechanisms of action, detection, and treatment of the conditions produced by toxicants are studied.

Uncertainty: True uncertainty is that which is not known about a factor that influences its value.

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URF: See inhalation unit risk factor

UTM Coordinates: Universal Transfer Mercator Coordinates. Coordinates used to define a specific location by means of two values (i.e., easting and northing coordinates).

United States Environmental Protection Agency (U.S. EPA): The Federal agency charged with setting policy and guidelines, carrying out legal mandates, for the protection, and national interests in environmental resources.

Vapor: The gaseous phase of liquids or solids at atmospheric temperature and pressure.

Vapor Pressure: The pressure exerted by a chemical vapor in equilibrium with its liquid or solid phase at any given temperature, used to calculate the rate of evaporation of a substance.

Variability: Ability to have different numerical values of a parameter, such as height or weight

Variate: A variable quantity associated with a probability distribution (e.g. inhalation rate)

Volatile: Chemicals that rapidly pass off from the liquid state in the form of vapors.

Xenobiotic: A toxic agent; a relatively small (MW<1000), non-nutritive chemical that is foreign to the species being studied

Zone of impact: The area in the vicinity of the facility in which an individual is exposed to a specified cancer risk, usually one in a million or greater.

Appendix K

HRA Forms and Maps Used With Air Dispersion Modeling

- Example of Census Tract Map

- Example of 7.5 minute Series Map

- Examples of Tables for Emissions Reporting

Figure 1
Census Tracts, View 1

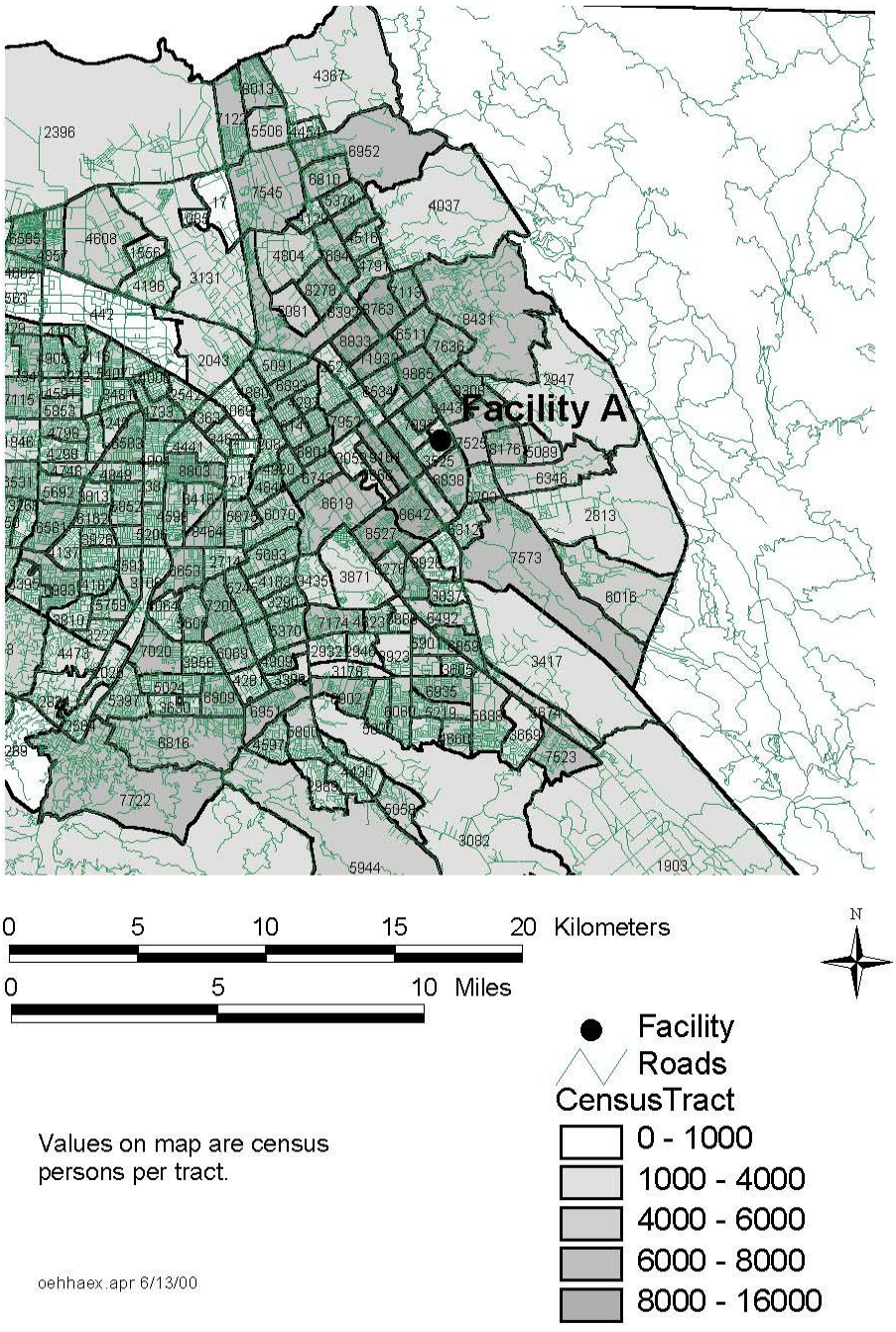


Figure 2
Census Tracts, View 2

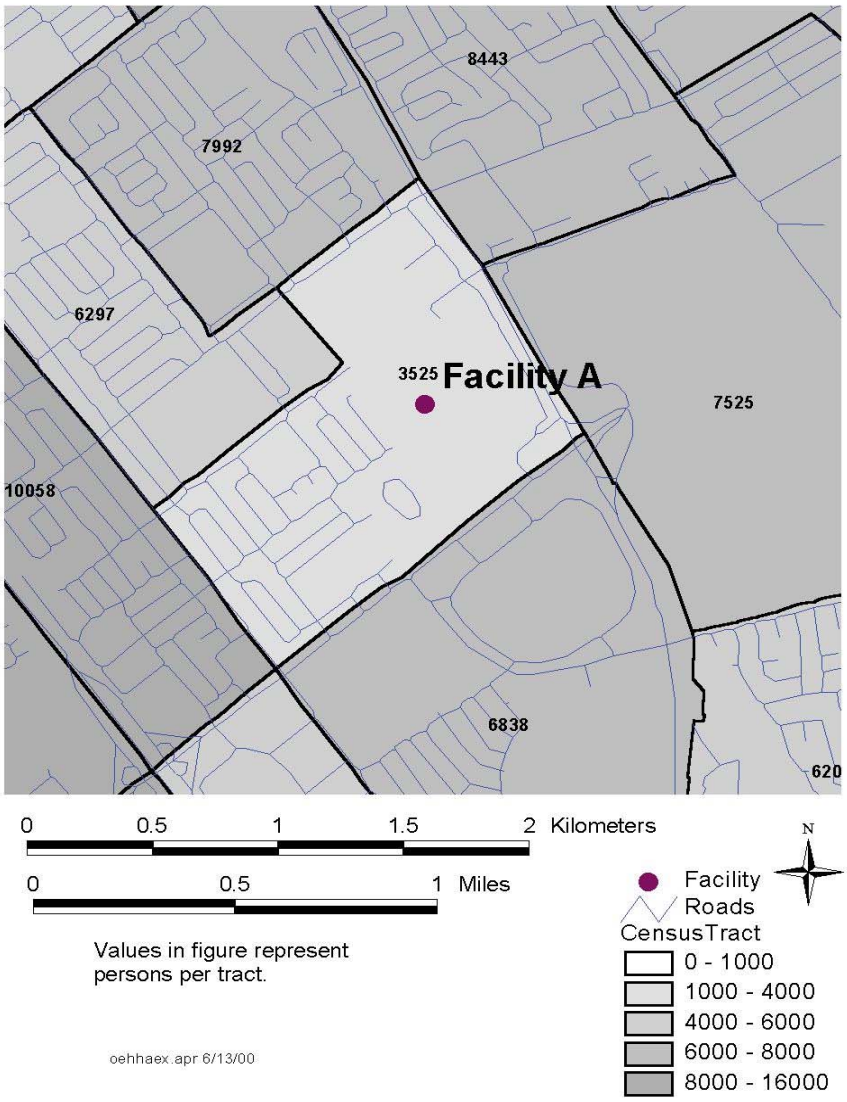
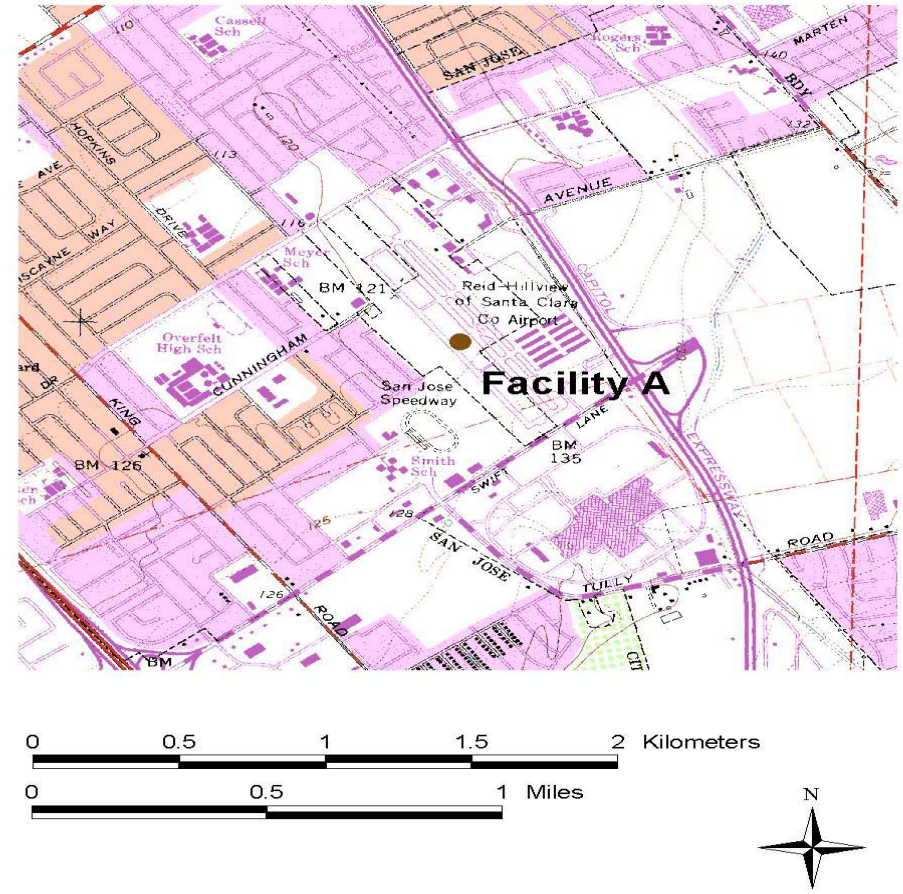


Figure 4
USGS 7.5 Minute Topographic Map



oehhaex.apr 6/13/00

HEALTH RISK ASSESSMENT

EMISSION RATE BY SUBSTANCE AND SOURCE RAG-001

FACILITY NAME / FACILITY ADDRESS / SITE ID#:

SOURCE ID No.	SOURCE NAME	SUBSTANCE NAME	CAS No.	1-HOUR MAXIMUM (lb/hr)	1-HOUR MAXIMUM (g/s)	ANNUAL AVERAGE (lb/yr)	ANNUAL AVERAGE (g/s)

HEALTH RISK ASSESSMENT

EMISSION RATE BY SUBSTANCE – TOTALS – RAG-002

FACILITY NAME / FACILITY ADDRESS / SITE ID#

SUBSTANCE NAME	CAS No.	1-HOUR MAXIMUM (lb/hr)	1-HOUR MAXIMUM (g/s)	ANNUAL AVERAGE (lb/yr)	ANNUAL AVERAGE (g/s)

HEALTH RISK ASSESSMENT

SOURCE PARAMETERS – STACKS – RAG-003

FACILITY NAME / FACILITY ADDRESS / SITE ID#

SOURCE ID No.	STACK NAME	UTM Easting	UTM Northing	HEIGHT (m)	DIAMETER (m)	TEMP. (F)	TEMP. (K)	FLOW RATE (ACFM)	EXIT VEL. (m/s)

HEALTH RISK ASSESSMENT

SOURCE OPERATING HOURS – RAG-004

FACILITY NAME / FACILITY ADDRESS / SITE ID#

SOURCE ID No.	STACK NAME	AVERAGE OPERATING HOURS		MAXIMUM OPERATING HOURS	
		(hr/day)	(days/year)	(hr/day)	(days/year)

Appendix L

OEHHA/ARB Approved Health Values
for Use in Hot Spot Facility Risk Assessments

Purpose of the Appendix L Tables:

The purpose of the following reference tables is to provide a quick list of all health values that have been approved by the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) for use in facility health risk assessments conducted for the AB 2588 Air Toxics Hot Spots Program. The OEHHA has developed and adopted new risk assessment guidelines that update and replace the California Air Pollution Control Officers Association's (CAPCOA) *Air Toxics "Hot Spots" Program Revised 1992 Risk Assessment Guidelines, October 1993*.

The following tables list the OEHHA adopted inhalation and oral cancer slope factors, noncancer acute Reference Exposure Levels (RELs), and inhalation and oral noncancer chronic RELs. In addition, these tables list the substances in Appendix A-I *(Substances For Which Emissions Must Be Quantified*) and Appendix F (*Criteria For Inputs For Risk Assessment Using Screening Air Dispersion Modeling*) of the ARB's *Hot Spots Emission Inventory Criteria and Guidelines (EICG).* OEHHA is still in the process of adopting new noncancer chronic RELs. Therefore, new health values will periodically be added to, or deleted from, these tables. Users of these tables are advised to monitor the OEHHA website (*www.oehha.ca.gov*) for any updates to the health values.

Substances written in *italics* do not have explicit OEHHA approved health values, but are included in this table to clarify applicability of OEHHA adopted heath effects values to individual or grouped substances listed in the *Hot Spots Emission Inventory Criteria and Guidelines*, Appendix A-I list of *"Substances For Which Emissions Must Be Quantified."*

The "Date Value Reviewed" column lists the date that the health value was last reviewed by OEHHA and the Scientific Review Panel, and/or approved for use in the AB 2588 Air Toxics Hot Spots Program. This information is useful to tell where the number came from. If the health value is unchanged since it was first approved for use in the Hot Spots Program, then the date that the value was first approved for use by CAPCOA is listed within the brackets [ ].

- April 1999 is listed for the cancer potency values and noncancer acute RELs, which have been adopted by the OEHHA as part of the AB 2588 "Hot Spots" Risk Assessment Guidelines.
- February 2000, April 2000, January 2001, and December 2001 are listed for the first set of 22, the second set of 16, the third set of 22, and the fourth set of 12 noncancer chronic RELs, respectively.
- October 2000 is listed for the oral chronic RELs and oral cancer slope factors. 1996 is listed for the U.S. EPA Reference Concentrations. Dates of 1990-1992 and 1996 are listed for CAPCOA chronic RELs that may eventually be dropped or replaced.
- For the substances identified as Toxic Air Contaminants, the Air Resources Board hearing date is listed. The dates for acetaldehyde, benzo[a]pyrene, and methyl tertiary-butyl ether represent the dates the values were approved by the Scientific Review Panel.

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
ACETALDEHYDE	75-07-0			9.0E+00	5/93			1.0E-02	4/99 [5/93]			1
ACETAMIDE	60-35-5							7.0E-02	4/99			1
ACROLEIN	107-02-8	1.9E-01	4/99	6.0E-02	1/01							--
ACRYLAMIDE	79-06-1							4.5E+00	4/99 [7/90]			1
ACRYLIC ACID	79-10-7	6.0E+03	4/99									--
ACRYLONITRILE	107-13-1			5.0E+00	12/01			1.0E+00	4/99 [1/91]			1
ALLYL CHLORIDE	107-05-1							2.1E-02	4/99			1
2-AMINOANTHRAQUINONE	117-79-3							3.3E-02	4/99			1
AMMONIA	7664-41-7	3.2E+03	4/99	2.0E+02	2/00							--
ANILINE	62-53-3							5.7E-03	4/99			1
Antimony Compounds	*7440-36-0*											--
ANTIMONY TRIOXIDE	1309-64-4											--
ARSENIC AND COMPOUNDS (INORGANIC) TAC ♣	7440-38-2 1016 [1015]	1.9E-01 AveP	4/99	3.0E-02	1/01	3.0E-04	10/00	1.2E+01 TAC	7/90	1.5E+00	10/00	1
ARSINE	7784-42-1	1.6E+02	4/99									--
ASBESTOS TAC ¤	1332-21-4							1.9E-04 TAC¤	3/86			333.33 ¤
BENZENE TAC	71-43-2	1.3E+03 AveP	4/99	6.0E+01	2/00			1.0E-01 TAC	1/85			1
BENZIDINE (AND ITS SALTS) *values also apply to:*	92-87-5							5.0E+02	4/99 [1/91]			1
Benzidine based dyes	*1020*							*5.0E+02*	*4/99 [1/91]*			*1*
Direct Black 38	*1937-37-7*							*5.0E+02*	*4/99 [1/91]*			*1*
Direct Blue 6	*2602-46-2*							*5.0E+02*	*4/99 [1/91]*			*1*
Direct Brown 95 (technical grade)	*16071-86-6*							*5.0E+02*	*4/99 [1/91]*			*1*
BENZYL CHLORIDE	100-44-7	4E+02	4/99					1.7E-01	4/99			1
BERYLLIUM AND COMPOUNDS ♣	7440-41-7 [1021]			7.0E-03	12/01	2.0E-03	12/01	8.4E+00	4/99 [7/90]			1
BIS(2-CHLOROETHYL)ETHER (Dichloroethyl ether)	111-44-4							2.5E+00	4/99			1
BIS(CHLOROMETHYL)ETHER	542-88-1							4.6E+01	4/99 [1/91]			1
1,3-BUTADIENE TAC	106-99-0			2.0E+01	1/01			6.0E-01 TAC	7/92			1

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
CADMIUM AND COMPOUNDS TAC ♣	7440-43-9 [1045]			2.0E-02	1/01	5.0E-04	10/00	1.5E+01 TAC	1/87			1
CARBON DISULFIDE	75-15-0	6.2E+03 AveP	4/99	8.0E+02 RfC								--
CARBON MONOXIDE	630-08-0	2.3E+04	4/99									--
CARBON TETRACHLORIDE TAC (Tetrachloromethane)	56-23-5	1.9E+03 AveP	4/99	4.0E+01	1/01			1.5E-01 TAC	9/87			1
CHLORINATED PARAFFINS	108171-26-2							8.9E-02	4/99			1
CHLORINE	7782-50-5	2.1E+02	4/99	2.0E-01	2/00							--
CHLORINE DIOXIDE	10049-04-4			6.0E-01	1/01							--
4-CHLORO-O-PHENYLENEDIAMINE	95-83-0							1.6E-02	4/99			1
CHLOROBENZENE	108-90-7			1.0E+03	1/01							--
CHLORODIFLUOROMETHANE ... (see Fluorocarbons)												
CHLOROFORM TAC	67-66-3	1.5E+02 AveP	4/99	3.0E+02	4/00			1.9E-02 TAC	12/90			1
Chlorophenols	*1060*											--
PENTACHLOROPHENOL	87-86-5							1.8E-02	4/99			1
2,4,6-TRICHLOROPHENOL	88-06-2							7.0E-02	4/99 [1/91]			1
CHLOROPICRIN	76-06-2	2.9E+01	4/99	4.0E-01	12/01							--
CHLOROPRENE	126-99-8											--
p-CHLORO-o-TOLUIDINE	95-69-2							2.7E-01	4/99			1
CHROMIUM 6+ TAC ♣ *values also apply to:*	18540-29-9			2.0E-01	1/01	2.0E-02	10/00	5.1E+02 TAC	1/86			1
Barium chromate♣	*10294-40-3*			*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*5.1E+02* TAC	*1/86*			*0.2053*
Calcium chromate♣	*13765-19-0*			*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*5.1E+02* TAC	*1/86*			*0.3332*
Lead chromate♣	*7758-97-6*			*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*5.1E+02* TAC	*1/86*			*0.1609*
Sodium dichromate♣	*10588-01-9*			*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*5.1E+02* TAC	*1/86*			*0.397*
Strontium chromate♣	*7789-06-2*			*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*5.1E+02* TAC	*1/86*			*0.2554*
CHROMIUM TRIOXIDE ♣ (as chromic acid mist)	1333-82-0			2.0E-03	1/01	2.0E-02	10/00	5.1E+02 TAC	1/86			0.52
COPPER AND COMPOUNDS	7440-50-8 [1067]	1.0E+02	4/99									--
p-CRESIDINE	120-71-8							1.5E-01	4/99			1
CRESOLS (mixtures of)	1319-77-3			6.0E+02	1/01							--
m-CRESOL	108-39-4			6.0E+02	1/01							--

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
o-CRESOL	95-48-7			6.0E+02	1/01							--
p-CRESOL	106-44-5			6.0E+02	1/01							--
CUPFERRON	135-20-6							2.2E-01	4/99			1
Cyanide Compounds (inorganic)	*57-12-5 1073*	*3.4E+02*	*4/99*	*9.0E+00*	*4/00*							--
HYDROGEN CYANIDE (Hydrocyanic acid)	74-90-8	3.4E+02	4/99	9.0E+00	4/00							--
2,4-DIAMINOANISOLE	615-05-4							2.3E-02	4/99			1
2,4-DIAMINOTOLUENE	95-80-7							4.0E+00	4/99			1
1,2-DIBROMO-3-CHLOROPROPANE (DBCP)	96-12-8							7.0E+00	4/99 [1/92]			1
p-DICHLOROBENZENE	106-46-7			8.0E+02	1/01			4.0E-02	4/99 [1/91]			1
3,3-DICHLOROBENZIDINE	91-94-1							1.2E+00	4/99 [1/91]			1
1,1,-DICHLOROETHANE (Ethylidene dichloride)	75-34-3							5.7E-03	4/99			1
1,1-DICHLOROETHYLENE … (see Vinylidene Chloride)												
DI(2-ETHYLHEXYL)PHTHALATE (DEHP)	117-81-7							8.4E-03	4/99 [1/92]	8.4E-03	10/00	1
DIESEL EXHAUST … (see Particulate Emissions from Diesel-Fueled Engines)												
DIETHANOLAMINE	111-42-2			3.0E+00	12/01							--
DIMETHYLAMINE	124-40-3											--
p-DIMETHYLAMINOAZOBENZENE	60-11-7							4.6E+00	4/99			1
N,N-DIMETHYL FORMAMIDE	68-12-2			8.0E+01	1/01							--
2,4-DINITROTOLUENE	121-14-2							3.1E-01	4/99			1
1,4-DIOXANE (1,4-Diethylene dioxide)	123-91-1	3.0E+03	4/99	3.0E+03	4/00			2.7E-02	4/99 [1/91]			1
EPICHLOROHYDRIN (1-Chloro-2,3-epoxypropane)	106-89-8	1.3E+03	4/99	3.0E+00	1/01			8.0E-02	4/99 [1/92]			1
1,2-EPOXYBUTANE	106-88-7			2.0E+01	1/01							--
ETHYL ACRYLATE	140-88-5											--
ETHYL BENZENE	100-41-4			2.0E+03	2/00							--
ETHYL CHLORIDE (Chloroethane)	75-00-3			3.0E+04	4/00							--
ETHYLENE DIBROMIDE TAC (1,2-Dibromoethane)	106-93-4			8.0E-01	12/01			2.5E-01 TAC	7/85			1

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS

Substance	Chemical Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg-d)^{-1}$	Date Value Reviewed [Added]	Oral Slope Factor $(mg/kg-d)^{-1}$	Date Value Reviewed [Added]	MAF
ETHYLENE DICHLORIDE TAC (1,2-Dichloroethane)	107-06-2			4.0E+02	1/01			7.2E-02 TAC	9/85			1
ETHYLENE GLYCOL	107-21-1			4.0E+02	4/00							--
ETHYLENE GLYCOL BUTYL ETHER … (see Glycol ethers)												
ETHYLENE OXIDE TAC (1,2-Epoxyethane)	75-21-8			3.0E+01	1/01			3.1E-01 TAC	11/87			1
ETHYLENE THIOUREA	96-45-7							4.5E-02	4/99			1
Fluorides	*1101*	*2.4E+02*	*4/99*	*1.3E+01*	*8/03*	4.0E-2	8/03					--
HYDROGEN FLUORIDE (Hydrofluoric acid)	7664-39-3	2.4E+02	4/99	1.4E+01	8/031	4.0E-2						--
FORMALDEHYDE TAC	50-00-0	9.4E+01	4/99	3.0E+00	2/00			2.1E-02 TAC	3/92			1
GASOLINE VAPORS	1110											--
GLUTARALDEHYDE	111-30-8			8.0E-02	1/01							--
GLYCOL ETHERS	1115											
ETHYLENE GLYCOL MONOBUTYL ETHER – EGBE	111-76-2	1.4E+04	4/99									--
ETHYLENE GLYCOL MONOETHYL ETHER – EGEE	110-80-5	3.7E+02 AveP	4/99[1/92]	7.0E+01	2/00							--
ETHYLENE GLYCOL MONOETHYL ETHER ACETATE – EGEEA	111-15-9	1.4E+02 AveP	4/99	3.0E+02	2/00							--
ETHYLENE GLYCOL MONOMETHYL ETHER – EGME	109-86-4	9.3E+01 AveP	4/99	6.0E+01	2/00							--
ETHYLENE GLYCOL MONOMETHYL ETHER ACETATE – EGMEA	110-49-6			9.0E+01	2/00							--
HEXACHLOROBENZENE	118-74-1							1.8E+00	4/99 [1/91]			1
HEXACHLOROCYCLOHEXANES (mixed or technical grade)	608-73-1 1120							4.0E+00	4/99 [1/91]	4.0E+00	10/00 [1/92]	1
Alpha-HEXACHLOROCYCLOHEXANE	319-84-6							4.0E+00	4/99 [1/91]	4.0E+00	10/00 [1/92]	1
beta- HEXACHLOROCYCLOHEXANE	319-85-7							4.0E+00	4/99 [1/91]	4.0E+00	10/00 [1/92]	1
Gamma-HEXACHLOROCYCLOHEXANE (Lindane)	58-89-9							1.1E+00	4/99	1.1E+00	10/00	1

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
n-HEXANE	110-54-3			7.0E+03	4/00							--
HYDRAZINE	302-01-2			2.0E-01	1/01			1.7E+01	4/99 [7/90]			1
HYDROCHLORIC ACID (Hydrogen chloride)	7647-01-0	2.1E+03	4/99	9.0E+00	2/00							--
HYDROGEN BROMIDE … (see Bromine & Compounds)												
HYDROGEN CYANIDE … (see Cyanide & Compounds)												
HYDROGEN FLUORIDE … (see Fluorides)												
HYDROGEN SELENIDE … (see Selenium & Compounds)												
HYDROGEN SULFIDE	7783-06-4	4.2E+01	4/99[7/90]	1.0E+01	4/00							--
ISOPHORONE	78-59-1			2.0E+03	12/01							--
ISOPROPYL ALCOHOL (Isopropanol)	67-63-0	3.2E+03	4/99	7.0E+03	2/00							--
LEAD AND COMPOUNDS[TAC ✶ ♣] (inorganic) *values also apply to:*	7439-92-1 1128 [1130]							4.2E-02 TAC	4/97	8.5E-03	10/00	1
Lead acetate♣	*301-04-2*							*4.2E-02* TAC	*4/97*	*8.5E-03*	*10/00*	*0.637*
Lead phosphate♣	*7446-27-7*							*4.2E-02* TAC	*4/97*	*8.5E-03*	*10/00*	*0.7659*
Lead subacetate♣	*1335-32-6*							*4.2E-02* TAC	*4/97*	*8.5E-03*	*10/00*	*0.7696*
LINDANE ... (see gamma-Hexachlorocyclohexane)												
MALEIC ANHYDRIDE	108-31-6			7.0E-01	12/01							--
MANGANESE AND COMPOUNDS	7439-96-5 [1132]			2.0E-01	4/00							--
MERCURY AND COMPOUNDS (INORGANIC)	7439-97-6 [1133]	1.8E+00	4/99	9.0^{E}-02	2/00	3.0E-04	10/00 [1/92]					--
Mercuric chloride	*7487-94-7*	*1.8E+00*	*4/99*	*9.0E-02*	*2/00*	*3.0E-04*	*10/00 [1/92]*					--
MERCURY AND COMPOUNDS (ORGANIC) *values also apply to:*	N/A											
METHYL MERCURY	593-74-8											--
METHANOL	67-56-1	2.8E+04	4/99	4.0E+03	4/00							--
METHYL BROMIDE (Bromomethane)	74-83-9	3.9E+03	4/99	5.0E+00	2/00							--
METHYL tertiary-BUTYL ETHER	1634-04-4			8.0E+03	2/00			9.1E-04	11/99			1
METHYL CHLOROFORM (1,1,1-Trichloroethane)	71-55-6	6.8E+04	4/99	1.0E+03	2/00							--

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
METHYL ETHYL KETONE (2-Butanone)	78-93-3	1.3E+04	4/99									--
METHYL ISOCYANATE	624-83-9			1.0E+00	12/01							--
METHYL MERCURY ... (see Mercury & Compounds)												
METHYL METHACRYLATE	80-62-6											--
4,4'-METHYLENE BIS (2-CHLOROANILINE) (MOCA)	101-14-4							1.5E+00	4/99			1
METHYLENE CHLORIDE TAC (Dichloromethane)	75-09-2	1.4E+04	4/99	4.0E+02	2/00			3.5E-03 TAC	7/89			1
4,4'-METHYLENE DIANILINE (AND ITS DICHLORIDE)	101-77-9			2.0E+01	12/01			1.6E+00	4/99	1.6E+00	10/00	1
METHYLENE DIPHENYL ISOCYANATE	101-68-8			7.0E-01	1/01							--
MICHLER'S KETONE (4,4'-Bis(dimethylamino)benzophenone)	90-94-8							8.6E-01	4/99			1
N-NITROSO-n-BUTYLAMINE	924-16-3							1.1E+01	4/99 [1/92]			1
N-NITROSODI-n-PROPYLAMINE	621-64-7							7.0E+00	4/99 [1/91]			1
N-NITROSODIETHYLAMINE	55-18-5							3.6E+01	4/99 [1/91]			1
N-NITROSODIMETHYLAMINE	62-75-9							1.6E+01	4/99 [1/91]			1
N-NITROSODIPHENYLAMINE	86-30-6							9.0E-03	4/99			1
N-NITROSO-N-METHYLETHYLAMINE	10595-95-6							2.2E+01	4/99 [7/90]			1
N-NITROSOMORPHOLINE	59-89-2							6.7E+00	4/99 [7/92]			1
N-NITROSOPIPERIDINE	100-75-4							9.4E+00	4/99 [7/92]			1
N-NITROSOPYRROLIDINE	930-55-2							2.1E+00	4/99 [7/90]			1
NAPHTHALENE ... (see Polycyclic aromatic hydrocarbons)												
NICKEL AND COMPOUNDS TAC ♣ *values also apply to:*	7440-02-0 [1145]	6.0E+00	4/99	5.0E-02	2/00	5.0E-02	10/00	9.1E-01 TAC	8/91			1
Nickel acetate♣	*373-02-4*	*6.0E+00*	*4/99*	*5.0E-02*	*2/00*	*5.0E-02*	*10/00*	*9.1E-01* TAC	*8/91*			*0.3321*
Nickel carbonate♣	*3333-39-3*	*6.0E+00*	*4/99*	*5.0E-02*	*2/00*	*5.0E-02*	*10/00*	*9.1E-01* TAC	*8/91*			*0.4945*
Nickel carbonyl♣	*13463-39-3*	*6.0E+00*	*4/99*	*5.0E-02*	*2/00*	*5.0E-02*	*10/00*	*9.1E-01* TAC	*8/91*			*0.3438*
Nickel hydroxide♣	*12054-48-7*	*6.0E+00*	*4/99*	*5.0E-02*	*2/00*	*5.0E-02*	*10/00*	*9.1E-01* TAC	*8/91*			*0.6332*

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
Nickelocene♣	*1271-28-9*	*6.0E+00*	*4/99*	*5.0E-02*	*2/00*	*5.0E-02*	*10/00*	*9.1E-01* TAC	*8/91*			*0.4937*
NICKEL OXIDE♣	1313-99-1	6.0E+00	4/99	1.0E-01	2/00	5.0E-02	10/00	9.1E-01 TAC	8/91			0.7859
Nickel refinery dust from the pyrometallurgical process	*1146*	*6.0E+00*	*4/99*	*5.0E-02*	*2/00*	*5.0E-02*	*10/00*	*9.1E-01* TAC	*8/91*			*1*
Nickel subsulfide♣	*12035-72-2*	*6.0E+00*	*4/99*	*5.0E-02*	*2/00*	*5.0E-02*	*10/00*	*9.1E-01* TAC	*8/91*			*0.2443*
NITRIC ACID	7697-37-2	8.6E+01	4/99									--
NITROGEN DIOXIDE	10102-44-0	4.7E+02	4/99[1/92]									--
2-NITROPROPANE	79-46-9											--
p-NITROSODIPHENYLAMINE	156-10-5							2.2E-02	4/99			1
OZONE	10028-15-6	1.8E+02	4/99[1/92]									--
PARTICULATE EMISSIONS FROM DIESEL-FUELED ENGINES TAC ■	9901			5.0E+00 TAC	8/98			1.1E+00 TAC	8/98			1
PENTACHLOROPHENOL ... (see Chlorophenols)												
PERCHLOROETHYLENE TAC (Tetrachloroethylene)	127-18-4	2.0E+04	4/99	3.5E+01 TAC	10/91			2.1E-02 TAC	10/91			1
PHENOL	108-95-2	5.8E+03	4/99	2.0E+02	4/00							--
PHOSGENE	75-44-5	4.0E+00	4/99									--
PHOSPHINE	7803-51-2			8.0E-01	9/02							--
PHOSPHORIC ACID	7664-38-2			7.0E+00	2/00							--
PHTHALIC ANHYDRIDE	85-44-9			2.0E+01	1/01							--
PCB (POLYCHLORINATED BIPHENYLS-unspeciated mixture) [lowest risk] ★	1336-36-3							7.0E-02	2/02	7.0E-02	2/02	1
PCB (POLYCHLORINATED BIPHENYLS-unspeciated mixture) [low risk] ★	1336-36-3							4.0E-01	2/02	4.0E-01	2/02	1
PCB (POLYCHLORINATED BIPHENYLS - unspeciated mixture) [high risk] ★	1336-36-3							2.0E+00	2/02	2.0E+00	2/02	1
PCB (POLYCHLORINATED BIPHENYLS (speciated)∇												
3,3',4,4'-TETRACHLOROBIPHENYL (77)	35298-13-3			4.0E-01	8/03	1.0E –04	8/03	1.3E +01	8/03	1.3E +01	8/03	
3,4,4',5-TETRACHLOROBIPHENYL (81)	70362-50-4			4.0E-01	8/03	1.0E –04	8/03	1.3E +01	8/03	1.3E +01	8/03	
2,3,3',4,4'- PENTACHLOROBIPHENYL (105)	32598-14-4			4.0E-01	8/03	1.0E –04	8/03	1.3E +01	8/03	1.3E +01	8/03	
2,3,4,4'5- PENTACHLOROBIPHENYL (114)	74472-37-0			8.0E-02	8/03	2.0E –05	8/03	6.5E +01	8/03	6.5E +01	8/03	
2,3'4,4',5- PENTACHLOROBIPHENYL (118)	31508-00-6			4.0E-01	8/03	1.0E –04	8/03	1.3E +01	8/03	1.3E +01	8/03	

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg-d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
2',3,4,4',5- PENTACHLOROBIPHENYL (123)	65510-44-3			4.0E-01	8/03	1.0E –04	8/03	1.3E +01	8/03	1.3E +01	8/03	
3,3',4,4',5- PENTACHLOROBIPHENYL (126)	57465-28-8			4.0E-04	8/03	1.0E –07	8/03	1.3E +04	8/03	1.3E +04	8/03	
2,3,3',4,4',5-HEXACHLOROBIPHENYL (156)	38380-08-4			8.0E-02	8/03	2.0E –05	8/03	6.5E +01	8/03	6.5E +01	8/03	
2,3,3',4,4',5'-HEXACHLOROBIPHENYL (157)	69782-90-7			8.0E-02	8/03	2.0E –05	8/03	6.5E +01	8/03	6.5E +01	8/03	
2,3',4,4',5,5'-HEXACHLOROBIPHENYL (167)	52663-72-6			4.0E-00	8/03	1.0E –03	8/03	1.3E +00	8/03	1.3E +00	8/03	
3,3',4,4'5,5' - HEXACHLOROBIPHENYL (169)	32774-16-6			4.0E-03	8/03	1.0E –06	8/03	1.3E +03	8/03	1.3E +03	8/03	
2,3,3'4,4',5,5' - HEPTACHLOROBIPHENYL (189)	39635-31-9			4.0E-01	8/03	1.0 E-04	8/03	1.3E +01	8/03	1.3E +01	8/03	
POLYCHLORINATED DIBENZO-*P*-DIOXINS (PCDD) (AS 2,3,7,8-PCDD EQUIVALENT)[TAC] ●	1085 1086											
2,3,7,8-TETRACHLORODIBENZO-*P*-DIOXIN[TAC]	1746-01-6			4.0E-05	2/00	1.0E-08	10/00	1.3E+05 TAC	8/86	1.3E+05 TAC	8/86	1
1,2,3,7,8-PENTACHLORODIBENZO-*P*-DIOXIN	40321-76-4			8.0E-05	2/00	2.0E-08	10/00	1.3E+05	4/99	1.3E+05	10/00	1
1,2,3,4,7,8-HEXACHLORODIBENZO-*P*-DIOXIN	39227-28-6			4.0E-04	2/00	1.0E-07	10/00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,6,7,8-HEXACHLORODIBENZO-*P*-DIOXIN	57653-85-7			4.0E-04	2/00	1.0E-07	10/00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,7,8,9-HEXACHLORODIBENZO-*P*-DIOXIN	19408-74-3			4.0E-04	2/00	1.0E-07	10/00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,4,6,7,8-HEPTACHLORODIBENZO-*P*-DIOXIN	35822-46-9			4.0E-03	2/00	1.0E-06	10/00	1.3E+03	4/99	1.3E+03	10/00	1
1,2,3,4,6,7,8,9-OCTACHLORODIBENZO-*P*-DIOXIN	3268-87-9			4.0E-02	2/00	1.0E-05	10/00	1.3E+01	4/99	1.3E+01	10/00	1
POLYCHLORINATED DIBENZOFURANS (AS 2,3,7,8-PCDD EQUIVALENT) (PCDF) [TAC] ●	1080											
2,3,7,8-TETRACHLORODIBENZOFURAN	5120-73-19			4.0E-04	2/00	1.0E-07	10/00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,7,8-PENTACHLORODIBENZOFURAN	57117-41-6			8.0E-04	2/00	2.0E-07	10/00	6.5E+03	4/99	6.5E+03	10/00	1
2,3,4,7,8-PENTACHLORODIBENZOFURAN	57117-31-4			8.0E-05	2/00	2.0E-08	10/00	6.5E+04	4/99	6.5E+04	10/00	1
1,2,3,4,7,8-HEXACHLORODIBENZOFURAN	70648-26-9			4.0E-04	2/00	1.0E-07	10/00	1.3E+04	4/99	1.3E+04	10/00	1

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
1,2,3,6,7,8-HEXACHLORODIBENZOFURAN	57117-44-9			4.0E-04	2/00	1.0E-07	10/00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,7,8,9-HEXACHLORODIBENZOFURAN	72918-21-9			4.0E-04	2/00	1.0E-07	10/00	1.3E+04	4/99	1.3E+04	10/00	1
2,3,4,6,7,8-HEXACHLORODIBENZOFURAN	60851-34-5			4.0E-04	2/00	1.0E-07	10/00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,4,6,7,8-HEPTACHLORODIBENZOFURAN	67562-39-4			4.0E-03	2/00	1.0E-06	10/00	1.3E+03	4/99	1.3E+03	10/00	1
1,2,3,4,7,8,9-HEPTACHLORODIBENZOFURAN	55673-89-7			4.0E-03	2/00	1.0E-06	10/00	1.3E+03	4/99	1.3E+03	10/00	1
1,2,3,4,6,7,8,9-OCTACHLORODIBENZOFURAN	39001-02-0			4.0E-02	2/00	1.0E-05	10/00	1.3E+01	4/99	1.3E+01	10/00	1
POLYCYCLIC AROMATIC HYDROCARBON (PAH)	1150 1151											
BENZ(A)ANTHRACENE❖	56-55-3							3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
BENZO(A)PYRENE❖	50-32-8							3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
BENZO(B)FLUORANTHENE❖	205-99-2							3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
BENZO(J)FLUORANTHENE❖	205-82-3							3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
BENZO(K)FLUORANTHENE❖	207-08-9							3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
CHRYSENE❖	218-01-9							3.9E-02	4/99 [4/94]	1.2E-01	10/00 [4/94]	1
DIBENZ(A,H)ACRIDINE❖	226-36-8							3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
DIBENZ(A,H)ANTHRACENE❖	53-70-3							4.1E+00	4/99 [4/94]	4.1E+00	10/00 [4/94]	1
DIBENZ(A,J)ACRIDINE❖	224-42-0							3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
DIBENZO(A,E)PYRENE❖	192-65-4							3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
DIBENZO(A,H)PYRENE❖	189-64-0							3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
DIBENZO(A,I)PYRENE ❖	189-55-9							3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
DIBENZO(A,L)PYRENE ❖	191-30-0							3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
7H-DIBENZO(C,G)CARBAZOLE ❖	194-59-2							3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
7,12-DIMETHYLBENZ(A)ANTHRACENE ❖	57-97-6							2.5E+02	4/99 [4/94]	2.5E+02	10/00 [4/94]	1
1,6-DINITROPYRENE ❖	42397-64-8							3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
1,8-DINITROPYRENE ❖	42397-65-9							3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
INDENO(1,2,3-C,D)PYRENE ❖	193-39-5							3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
3-METHYLCHOLANTHRENE ❖	56-49-5							2.2E+01	4/99 [4/94]	2.2E+01	10/00 [4/94]	1
5-METHYLCHRYSENE ❖	3697-24-3							3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
NAPHTHALENE	91-20-3			9.0E+00	4/00							--
5-NITROACENAPHTHENE ❖	602-87-9							1.3E-01	4/99 [4/94]	1.3E-01	10/00 [4/94]	1
6-NITROCHRYSENE ❖	7496-02-8							3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
2-NITROFLUORENE ❖	607-57-8							3.9E-02	4/99 [4/94]	1.2E-01	10/00 [4/94]	1
1-NITROPYRENE ❖	5522-43-0							3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
4-NITROPYRENE ❖	57835-92-4							3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
POTASSIUM BROMATE.... ... (see Bromine & Compounds)												
1,3-PROPANE SULTONE	1120-71-4							2.4E+00	4/99			1
PROPYLENE (PROPENE)	115-07-1			3.0E+03	4/00							--
PROPYLENE GLYCOL MONOMETHYL ETHER	107-98-2			7.0E+03	2/00							--
PROPYLENE OXIDE	75-56-9	3.1E+03	4/99	3.0E+01	2/00			1.3E-02	4/99 [7/90]			1
SELENIUM AND COMPOUNDS	7782-49-2 [1170]			2.0E+01	12/01							--
HYDROGEN SELENIDE	7783-07-5	5.0E+00	4/99									--
Selenium sulfide	*7446-34-6*			*2.0E+01*	*12/01*							--
SODIUM HYDROXIDE	1310-73-2	8.0E+00	4/99	4.8E+00	7/90							--

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS

Substance	Chemical Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date Value Reviewed [Added]	MWAF
STYRENE	100-42-5	2.1E+04	4/99	9.0E+02	4/00							--
SULFATES	9960	1.2E+02	4/99	2.5E+01	1/92							--
SULFUR DIOXIDE	7446-09-5	6.6E+02	4/99[1/92]	6.6E+02	1/92							--
SULFURIC ACID AND OLEUM	7664-93-9	1.2E+02	4/99	*1.0E+00*	*12/01*							--
SULFURIC ACID	*7664-93-9*	*1.2E+02*	*4/99*	1.0E+00	12/01							--
SULFUR TRIOXIDE	*7446-71-9*	*1.2E+02*	*4/99*									--
OLEUM	*8014-95-7*	*1.2E+02*	*4/99*	*1.0E+00*	*12/01*							--
1,1,2,2-TETRACHLOROETHANE	79-34-5							2.0E-01	4/99			1
TETRACHLOROPHENOLS ... (see Chlorophenols)												
2,4,5-TRICHLOROPHENOL ... (see Chlorophenols)												
2,4,6-TRICHLOROPHENOL ... (see Chlorophenols)												
THIOACETAMIDE	62-55-5							6.1E+00	4/99			1
TOLUENE	108-88-3	3.7E+04	4/99	3.0E+02	4/00							--
Toluene diisocyantates	*26471-62-5 1204*			*7.0E-02*	*1/01*			*3.9E-02*	*4/99*			*1*
TOLUENE-2,4-DIISOCYANATE	584-84-9			7.0E-02	1/01			3.9E-02	4/99			1
TOLUENE-2,6-DIISOCYANATE	91-08-7			7.0E-02	1/01			3.9E-02	4/99			1
1,1,2-TRICHLOROETHANE (Vinyl trichloride)	79-00-5							5.7E-02	4/99			1
TRICHLOROETHYLENE[TAC]	79-01-6			6.0E+02	4/00			7.0E-03 TAC	10/90			1
TRIETHYLAMINE	121-44-8	2.8E+03	4/99	2.0^{E}+02	9/02							--
URETHANE (Ethyl carbamate)	51-79-6							1.0E+00	4/99 [7/90]			1
Vanadium Compounds	*N/A*											
Vanadium (fume or dust)	*7440-62-2*	*3.0E+01*	*4/99*									--
VANADIUM PENTOXIDE	1314-62-1	3.0E+01	4/99									--
VINYL ACETATE	108-05-4			2.0E+02	12/01							--
VINYL CHLORIDE[TAC] (Chloroethylene)	75-01-4	1.8E+05	4/99					2.7E-01 TAC	12/90			1
VINYLIDENE CHLORIDE (1,1-Dichloroethylene)	75-35-4			7.0E+01	1/01							--

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ⊛

Substance ⊛	Chemical▼ Abstract Service Number (CAS)	Noncancer Effects						Cancer Risk				
		Acute Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Inhalation REL ($\mu g/m^3$)	Date ◆ Value Reviewed [Added]	Chronic Oral REL (mg/kg/d)	Date ◆ Value Reviewed [Added]	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date ◆ Value Reviewed [Added]	M♣ W A F
XYLENES (mixed isomers)	1330-20-7 1210	2.2E+04	4/99	7.0E+02	4/00							--
m-XYLENE	108-38-3	2.2E+04	4/99	7.0E+02	4/00							--
o-XYLENE	95-47-6	2.2E+04	4/99	7.0E+02	4/00							--
p-XYLENE	106-42-3	2.2E+04	4/99	7.0E+02	4/00							--

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ❁

Purpose:	The purpose of this reference table is to provide a quick list of all health values that have been approved by the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) for use in facility health risk assessments conducted for the AB 2588 Air Toxics Hot Spots Program. The OEHHA has developed and adopted new risk assessment guidelines that update and replace the California Air Pollution Control Officers Association's (CAPCOA) *Air Toxics "Hot Spots" Program Revised 1992 Risk Assessment Guidelines, October 1993.* The OEHHA has adopted five technical support documents for these guidelines. This table lists the OEHHA adopted inhalation and oral cancer slope factors, noncancer acute Reference Exposure Levels (RELs), and inhalation and oral noncancer chronic RELs. In addition, it lists the substances in Appendix A-I *(Substances For Which Emissions Must Be Quantified)* and Appendix F (*Criteria For Inputs For Risk Assessment Using Screening Air Dispersion Modeling*) of the ARB's *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG)*. OEHHA is still in the process of adopting new noncancer chronic RELs. Therefore, new health values will periodically be added to, or deleted from, this table. Users of this table are advised to monitor the OEHHA website (*www.oehha.ca.gov*) for any updates to the health values.
❁	Substances written in *italics* do not have explicit OEHHA approved health values, but are included in this table to clarify applicability of OEHHA adopted heath effects values to individual or grouped substances listed in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines*, Appendix A-I list of *"Substances For Which Emissions Must Be Quantified".*
▼	Chemical Abstract Service Number (CAS): For chemical groupings and mixtures where a CAS number is not applicable, the 4-digit code used in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG) Report* is listed. The 4-digit codes enclosed in brackets [ ] are codes that have been phased out, but may still appear on previously reported Hot Spots emissions. For information on the origin and use of the 4-digit code, see the EICG report.
◆	Date Value Reviewed [Added]: These columns list the date that the health value was last reviewed by OEHHA and the Scientific Review Panel, and/or approved for use in the AB 2588 Air Toxics Hot Spots Program. If the health value is unchanged since it was first approved for use in the Hot Spots Program, then the date that the value was first approved for use by CAPCOA is listed within the brackets [ ]. • April 1999 is listed for the cancer potency values and noncancer acute RELs, which have been adopted by the OEHHA as part of the AB 2588 "Hot Spot" Risk Assessment Guidelines. • February 2000, April 2000, January 2001, and December 2001 are listed for the first set of 22, the second set of 16, the third set of 22, and the fourth set of 12 noncancer chronic RELs, respectively. • October 2000 is listed for the oral chronic RELs and oral cancer slope factors. 1996 is listed for the U.S. EPA Reference Concentrations. Dates of 1990-1992 and 1996 are listed for CAPCOA chronic RELs, which may eventually be dropped or replaced. • For the substances identified as Toxic Air Contaminants, the Air Resources Board hearing date is listed. The dates for acetaldehyde, benzo[a]pyrene, and methyl tertiary-butyl ether represent the dates the values were approved by the Scientific Review Panel.
♣	Molecular Weight Adjustment Factor: Molecular weight adjustment factors (MWAF) are only to be used when a toxic metal has a cancer potency factor. For most of the Hot Spots toxic metals, the OEHHA cancer potency factor applies to the weight of the toxic metal atom contained in the overall compound. Some of the Hot Spots compounds contain various elements along with the toxic metal atom (e.g., "Nickel hydroxide", CAS number 12054-48-7, has a formula of H_2NiO_2). Therefore, an adjustment to the reported pounds of the overall compound is needed before applying the OEHHA cancer potency factor for "Nickel and compounds" to such a compound. This ensures that the cancer potency factor is applied only to the fraction of the overall weight of the emissions that are associated with health effects of the metal. In other cases, the Hot Spots metals are already reported as the metal atom equivalent (e.g., CAS 7440-02-0, "Nickel"), and these cases do not use any further molecular weight adjustment. (Refer to Note [7] in Appendix A, List of Substances in the EICG Report for further information on how the emissions of various Hot Spots metal compounds are reported.) The appropriate molecular weight adjustment factors (MWAF) to be used along with the OEHHA cancer potency factors for Hot Spots metals can be found in the MWAF column of this table. A double dash (--) was entered into the column if the substance does not currently have a cancer potency factor. So, for example, assume 100 pounds of "Nickel hydroxide" emissions are reported under CAS number 12054-48-7. To get the Nickel atom equivalent of these emissions, multiply by the listed MWAF (0.6332) for Nickel hydroxide: • 100 pounds x 0.6332 = 63.32 pounds of Nickel atom equivalent *This step should be completed prior to applying the OEHHA cancer potency factor for "Nickel and compounds" in a calculation for a prioritization score or risk assessment calculation.* (For more information see Chapter 4 and Appendix H of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part V; Technical Support Document; Guidance Manual for Preparation of Health Risk Assessments.*) Note: The value listed in the MWAF column for Asbestos is not a molecular weight adjustment. This is a conversion factor for adjusting mass to fibers or structures. See Appendix C of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part V; Technical Support Document; Guidance Manual for Preparation of Health Risk Assessments* for more information on Asbestos, or see the EICG report for reporting guidance. Also see the Asbestos footnote (designated by the symbol ⌑)
N/A	Not Applicable
Ñ	Values calculated using WHO TEF procedure in OEHHA, 2003
TAC	Toxic Air Contaminant: The Air Resources Board has identified this substance as a Toxic Air Contaminant.

APPENDIX L - TABLE 1
OEHHA/ARB APPROVED HEALTH VALUES FOR USE IN HOT SPOT FACILITY RISK ASSESSMENTS ❁

AveP	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 OEHHA's technical support document for *The Determination of Acute Reference Exposure Levels for Airborne Toxicants, March 1999*). Typically the RELs for the following substances are 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). 4-Hour: Arsenic and Inorganic Arsenic Compounds 6-Hour: Benzene, Carbon disulfide, Ethylene glycol monoethyl ether, Ethylene glycol monoethyl ether acetate, Ethylene glycol monomethyl ether 7-Hour: Carbon tetrachloride, Chloroform
¤	Asbestos: The units for the Inhalation Cancer Potency factor for asbestos are $(100 \text{ PCM fibers/m}^3)^{-1}$. A conversion factor of 100 fibers/0.003 µg can be multiplied by a receptor concentration of asbestos expressed in $\mu g/m^3$. Unless other information necessary to estimate the concentration (fibers/m^3) of asbestos at receptors of interest is available. A unit risk factor of $2.7E\ 10^{-6}$ $(\mu g/m^3)^{-1}$ and an inhalation cancer potency factor of $2.2E\ 10^{+2}$ $(\text{mg/kg BW} * \text{day})^{-1}$ are available. For more information on asbestos quantity conversion factors, see Appendix C of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II; Technical Support Document for Describing Available Cancer Potency Factors*, and Appendix C of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part V; Technical Support Document; Guidance Manual for Preparation of Health Risk Assessments*.
✳	Inorganic Lead: Inorganic Lead was identified by the Air Resources Board as a Toxic Air Contaminant in April 1997. Since information on noncancer health effects show no identified threshold, no Reference Exposure Level has been developed. The document, *Risk Management Guidelines for New, Modified, and Existing Sources of Lead, March 2001*, has been developed by ARB and OEHHA staff for assessing noncancer health impacts from sources of lead. See Appendix F of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part V; Technical Support Document; Guidance Manual for Preparation of Health Risk Assessments* for an overview of how to evaluate noncancer impacts from exposure to lead using these risk management guidelines.
❖	Polycyclic Aromatic Hydrocarbons (PAHs): These substances are PAH or PAH-derivatives that have OEHHA-developed Potency Equivalency Factors (PEFs) which were approved by the Scientific Review Panel in April 1994 (see ARB document entitled *Benzo [a]pyrene as a Toxic Air Contaminant*). PAH inhalation slope factors listed here have been adjusted by the PEFs. See Appendix G of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part V; Technical Support Document; Guidance Manual for Preparation of Health Risk Assessments* for more information.
★	Polychlorinated Biphenyls: (unspeciated mixtures) Lowest Risk: For use in cases where congeners with more than four chlorines comprise less than one-half percent of total polychlorinated biphenyls. High Risk: For use in cases where congeners with more than four chlorines do not comprise less than one-half percent of total polychlorinated biphenyls. The Low Risk: This number would not ordinarly be used in the Hot Spots program.
•	Polychlorinated Dibenzo-*p*-dioxins and Polychlorinated Dibenzofurans (also referred to as chlorinated dioxins and dibenzofurans): The OEHHA has adopted the World Health Organization 1997 (WHO_{97}) Toxicity Equivalency Factor) scheme for evaluating the cancer and noncancer risk due to exposure to samples containing speciated mixtures of polychlorinated dibenzo-*p*-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF). See Appendix A of OEHHA's *Technical Support Document For Describing Available Cancer Potency Factors* for more information about the scheme. See Appendix E of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part V; Technical Support Document; Guidance Manual for Preparation of Health Risk Assessments* for the methodology for calculating 2,3,7,8-equivalents for PCDD and PCDFs.
■	Particulate Emissions from Diesel-Fueled Engines: The inhalation cancer potency factor and chronic REL were derived from whole diesel exhaust and should be used only for impacts from the inhalation pathway. The inhalation impacts from speciated emissions from diesel-fueled engines are already accounted for in the inhalation cancer potency factor and REL. However, at the discretion of the risk assessor, speciated emissions from diesel-fueled engines may be used to estimate acute noncancer health impacts or the contribution to cancer risk or chronic noncancer health impacts for the non-inhalation exposure pathway. See Appendix D of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part V; Technical Support Document; Guidance Manual for Preparation of Health Risk Assessments* for more information.

Table last updated: August, 2003

APPENDIX L - TABLE 2 OEHHA/ARB ACUTE REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ❁

Substance ❁	Chemical▼ Abstract Service Number (CAS)	Acute REL ($\mu g/m^3$)	Date ◆ Value Reviewed	Target Organs									
				Alimentary Tract	Cardiovascular	Developmental	Eye	Hematologic	Immune	Nervous	Reproductive	Respiratory	Skin
ACROLEIN	107-02-8	1.9E-01	4/99				X					X	
ACRYLIC ACID	79-10-7	6.0E+03	4/99				X					X	
AMMONIA	7664-41-7	3.2E+03	4/99				X					X	
ARSENIC AND COMPOUNDS (INORGANIC) TAC	7440-38-2 1016 [1015]	1.9E-01 AveP	4/99			X					X		
ARSINE	7784-42-1	1.6E+02	4/99					X					
BENZENE TAC	71-43-2	1.3E+03 AveP	4/99			X		X	X		X		
BENZYL CHLORIDE	100-44-7	2.4E+02	4/99				X					X	
CARBON DISULFIDE	75-15-0	6.2E+03 AveP	4/99			X				X	X		
CARBON MONOXIDE	630-08-0	2.3E+04	4/99		X								
CARBON TETRACHLORIDE TAC (Tetrachloromethane)	56-23-5	1.9E+03 AveP	4/99	X		X				X	X		
CHLORINE	7782-50-5	2.1E+02	4/99				X					X	
CHLOROFORM TAC	67-66-3	1.5E+02 AveP	4/99			X				X	X		
CHLOROPICRIN	76-06-2	2.9E+01	4/99				X					X	
COPPER AND COMPOUNDS	7440-50-8 [1067]	1.0E+02	4/99									X	
Cyanide Compounds (inorganic)	*57-12-5 1073*	*3.4E+02*	*4/99*							✓			
HYDROGEN CYANIDE (Hydrocyanic acid)	74-90-8	3.4E+02	4/99							X			
1,4-DIOXANE ✣ (1,4-Diethylene dioxide)	123-91-1	3.0E+03	4/99				X					X	
EPICHLOROHYDRIN (1-Chloro-2,3-epoxypropane)	106-89-8	1.3E+03	4/99				X					X	
Fluorides and Compounds	*1101*	*2.4E+02*	*4/99*				✓					✓	
HYDROGEN FLUORIDE (Hydrofluoric acid)	7664-39-3	2.4E+02	4/99				X					X	
FORMALDEHYDE TAC	50-00-0	9.4E+01	4/99				X		X			X	
GLYCOL ETHERS	1115												
ETHYLENE GLYCOL BUTYL ETHER – EGBE	111-76-2	1.4E+04	4/99				X					X	
ETHYLENE GLYCOL ETHYL ETHER – EGEE	110-80-5	3.7E+02 AveP	4/99 [1/92]			X					X		
ETHYLENE GLYCOL ETHYL ETHER ACETATE - EGEEA	111-15-9	1.4E+02 AveP	4/99			X				X	X		
ETHYLENE GLYCOL METHYL ETHER – EGME	109-86-4	9.3E+01 AveP	4/99			X					X		

APPENDIX L - TABLE 2 OEHHA/ARB ACUTE REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Acute REL ($\mu g/m^3$)	Date ◆ Value Reviewed	Target Organs									
				Alimentary Tract	Cardiovascular	Developmental	Eye	Hematologic	Immune	Nervous	Reproductive	Respiratory	Skin
HYDROCHLORIC ACID (Hydrogen chloride)	7647-01-0	2.1E+03	4/99				X					X	
HYDROGEN CYANIDE (Hydrocyanic acid) ... (see Cyanide Compounds)													
HYDROGEN FLUORIDE (Hydrofluoric acid) ... (see Fluorides & Compounds)													
HYDROGEN SELENIDE ... (see Selenium & Compounds)													
HYDROGEN SULFIDE	7783-06-4	4.2E+01	4/99 [7/90]							X			
ISOPROPYL ALCOHOL (Isopropanol)	67-63-0	3.2E+03	4/99				X					X	
MERCURY AND COMPOUNDS (INORGANIC)	7439-97-6 [1133]	1.8E+00	4/99			X					X		
Mercuric chloride	*7487-94-7*	*1.8E+00*	*4/99*			✓					✓		
METHANOL	67-56-1	2.8E+04	4/99							X			
METHYL BROMIDE (Bromomethane)	74-83-9	3.9E+03	4/99			X				X	X	X	
METHYL CHLOROFORM (1,1,1-Trichloroethane)	71-55-6	6.8E+04	4/99							X			
METHYL ETHYL KETONE (2-Butanone)	78-93-3	1.3E+04	4/99				X					X	
METHYLENE CHLORIDE[TAC] (Dichloromethane)	75-09-2	1.4E+04	4/99							X			
NICKEL AND COMPOUNDS[TAC]	7440-02-0 [1145]	6.0E+00	4/99						X			X	
Nickel acetate,	*373-02-4*	*6.0E+00*	*4/99*						✓			✓	
Nickel carbonate	*3333-39-3*	*6.0E+00*	*4/99*						✓			✓	
Nickel carbonyl	*13463-39-3*	*6.0E+00*	*4/99*						✓			✓	
Nickel hydroxide	*12054-48-7*	*6.0E+00*	*4/99*						✓			✓	
Nickelocene	*1271-28-9*	*6.0E+00*	*4/99*						✓			✓	
NICKEL OXIDE	1313-99-1	6.0E+00	4/99						X			X	
Nickel refinery dust from the pyrometallurgical process	*1146*	*6.0E+00*	*4/99*						✓			✓	
Nickel subsulfide	*12035-72-2*	*6.0E+00*	*4/99*						✓			✓	
NITRIC ACID	7697-37-2	8.6E+01	4/99									X	
NITROGEN DIOXIDE	10102-44-0	4.7E+02	4/99 [1/92]									X	
OZONE	10028-15-6	1.8E+02	4/99 [1/92]				X					X	
PERCHLOROETHYLENE[TAC] (Tetrachloroethylene)	127-18-4	2.0E+04	4/99				X			X		X	
PHENOL	108-95-2	5.8E+03	4/99				X					X	

APPENDIX L - TABLE 2 OEHHA/ARB ACUTE REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ❁

Substance ❁	Chemical▼ Abstract Service Number (CAS)	Acute REL ($\mu g/m^3$)	Date ◆ Value Reviewed	Target Organs									
				Alimentary Tract	Cardiovascular	Developmental	Eye	Hematologic	Immune	Nervous	Reproductive	Respiratory	Skin
PHOSGENE	75-44-5	4.0E+00	4/99									X	
PROPYLENE OXIDE	75-56-9	3.1E+03	4/99			X	X				X	X	
Selenium and Compounds	*7782-49-2 [1170]*												
HYDROGEN SELENIDE	7783-07-5	5.0E+00	4/99				X					X	
SODIUM HYDROXIDE	1310-73-2	8.0E+00	4/99				X					X	X
STYRENE	100-42-5	2.1E+04	4/99				X					X	
SULFATES	9960	1.2E+02	4/99									X	
SULFUR DIOXIDE	7446-09-5	6.6E+02	4/99 [1/92]									X	
SULFURIC ACID AND OLEUM	7664-93-9	1.2E+02	4/99									X	
SULFURIC ACID	*7664-93-9*	*1.2E+02*	*4/99*									✓	
SULFUR TRIOXIDE	*7446-71-9*	*1.2E+02*	*4/99*									✓	
OLEUM	*8014-95-7*	*1.2E+02*	*4/99*									✓	
TOLUENE	108-88-3	3.7E+04	4/99			X	X			X	X	X	
TRIETHYLAMINE	121-44-8	2.8E+03	4/99				X			X			
Vanadium Compounds	*N/A*												
Vanadium (fume or dust)	*7440-62-2*	*3.0E+01*	*4/99*				✓					✓	
VANADIUM PENTOXIDE	1314-62-1	3.0E+01	4/99				X					X	
VINYL CHLORIDE[TAC] (Chloroethylene)	75-01-4	1.8E+05	4/99				X			X		X	
XYLENES (mixed isomers)	1330-20-7 1210	2.2E+04	4/99				X					X	
m-Xylene	108-38-3	2.2E+04	4/99				X					X	
o-Xylene	95-47-6	2.2E+04	4/99				X					X	
p-Xylene	106-42-3	2.2E+04	4/99				X					X	

APPENDIX L - TABLE 2 OEHHA/ARB ACUTE REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ❁

Purpose:	The purpose of this reference table is to provide a quick list of all health values that have been approved by the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) for use in facility health risk assessments conducted for the AB 2588 Air Toxics Hot Spots Program. The OEHHA has developed and adopted new risk assessment guidelines that update and replace the California Air Pollution Control Officers Association's (CAPCOA) *Air Toxics "Hot Spots" Program Revised 1992 Risk Assessment Guidelines, October 1993*. The OEHHA has adopted five technical support documents for these guidelines. This table lists the OEHHA adopted noncancer acute Reference Exposure Levels (RELs). In addition, it lists the substances in Appendix A-I *(Substances For Which Emissions Must Be Quantified*) and Appendix F (*Criteria For Inputs For Risk Assessment Using Screening Air Dispersion Modeling*) of the ARB's *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG)*. Users of this table are advised to monitor the OEHHA website (*www.oehha.ca.gov*) for any updates to the health values.
❁	Substances written in *italics* and with a ✓ do not have explicit OEHHA approved health values, but are included in this table to clarify applicability of OEHHA adopted health effects values to individual or grouped substances listed in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines*, Appendix A-I list of *"Substances For Which Emissions Must Be Quantified"*.
▼	Chemical Abstract Service Number (CAS): For chemical groupings and mixtures where a CAS number is not applicable, the 4-digit code used in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG) Report* is listed. The 4-digit codes enclosed in brackets [ ] are codes that have been phased out, but may still appear on previously reported Hot Spots emissions. For information on the origin and use of the 4-digit code, see the EICG report.
◆	Date Value Reviewed [Added]: This column lists the date that the health value was last reviewed by OEHHA and the Scientific Review Panel, and/or approved for use in the AB 2588 Air Toxics "Hot Spots" Program. If the health value is unchanged since it was first approved for use in the "Hot Spots" Program, then the date that the value was first approved for use by CAPCOA is listed within the brackets [ ]. • April 1999 is listed for the noncancer acute RELs which have been adopted by the OEHHA as part of the AB 2588 "Hot Spot" Risk Assessment Guidelines.
TAC	Toxic Air Contaminant: The Air Resources Board has identified this substance as a Toxic Air Contaminant.
AveP	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 OEHHA's technical support document for *The Determination of Acute Reference Exposure Levels for Airborne Toxicants, March 1999).* Typically the RELs for the following substances are 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). 4-Hour: Arsenic and Inorganic Arsenic Compounds 6-Hour: Benzene, Carbon disulfide, Ethylene glycol ethyl ether, Ethylene glycol ethyl ether acetate, Ethylene glycol methyl ether 7-Hour: Carbon tetrachloride, Chloroform

Table last updated: August 2003

APPENDIX L - TABLE 3 OEHHA/ARB CHRONIC INHALATION AND ORAL REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Chronic Inhalation REL (µg/m^3)	Chronic Oral REL	Date ◆ Value Reviewed [Added]	Target Organs												
					Alimentary	Bone	Cardiovascular	Developmental	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Reproductive	Respiratory	Skin
ACETALDEHYDE	75-07-0	9.0E+00		5/93												X	
ACROLEIN	107-02-8	6.0E-02		1/01						X						X	
ACRYLONITRILE	107-13-1	5.0E+00		12/01												X	
AMMONIA	7664-41-7	2.0E+02		2/00												X	
ARSENIC AND COMPOUNDS (INORGANIC) TAC	7440-38-2 1016 [1015]	3.0E-02		1/01			X	X						X			
			3.0^E-04	10/00			X										X
BENZENE TAC	71-43-2	6.0E+01		2/00				X			X			X			
BERYLLIUM AND COMPOUNDS	7440-41-7 [1021]	7.0E-03		12/01								X				X	
			2.0^E-03	12/01	X												
1,3-BUTADIENE TAC	106-99-0	2.0E+01		1/01											X		
CADMIUM AND COMPOUNDS T7AC	7440-43-9 [1045]	2.0E-02		1/01									X			X	
			5.0^E-04	10/00									X				
CARBON DISULFIDE	75-15-0	8.0E+02		11/01										X	X		
CARBON TETRACHLORIDE TAC (Tetrachloromethane)	56-23-5	4.0E+01		1/01	X			X						X			
CHLORINE	7782-50-5	2.0E-01		2/00												X	
CHLORINE DIOXIDE	10049-04-4	6.0E-01		1/01												X	
CHLOROBENZENE	108-90-7	1.0E+03		1/01	X								X		X		
CHLOROFORM TAC	67-66-3	3.0E+02		4/00	X			X					X				
CHLOROPICRIN	76-06-2	4.0E-01		12/01												X	
CHROMIUM 6+ TAC	18540-29-9	2.0E-01		1/01												X	
			2.0E-02	10/00							X						
Barium chromate	*10294-40-3*	*2.0E-01*		*1/01*												✓	
			2.0E-02	*10/00*							✓						
Calcium chromate	*13765-19-0*	*2.0E-01*		*1/01*												✓	
			2.0E-02	*10/00*							✓						
Lead chromate	*7758-97-6*	*2.0E-01*		*1/01*												✓	
			2.0E-02	*10/00*							✓						

APPENDIX L - TABLE 3 OEHHA/ARB CHRONIC INHALATION AND ORAL REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ❁

Substance ❁	Chemical▼ Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL	Date ◆ Value Reviewed [Added]	Target Organs												
					Alimentary	Bone	Cardiovascular	Developmental	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Reproductive	Respiratory	Skin
Sodium dichromate	*10588-01-9*	*2.0E-01*		*1/01*												✓	
			2.0E-02	*10/00*							✓						
Strontium chromate	*7789-06-2*	*2.0E-01*		*1/01*												✓	
			2.0E-02	*10/00*							✓						
CHROMIUM TRIOXIDE (as chromic acid mist)	1333-82-0	2.0E-03		1/01												**X**	
			2.0E-02	*10/00*							✓						
CRESOLS (mixtures of)	1319-77-3	6.0E+02		1/01										**X**			
m-CRESOL	108-39-4	6.0E+02		1/01										**X**			
o-CRESOL	95-48-7	6.0E+02		1/01										**X**			
p-CRESOL	106-44-5	6.0E+02		1/01										**X**			
Cyanide Compounds (inorganic)	*57-12-5 1073*	*9.0E+00*		*4/00*			✓		✓					✓			
HYDROGEN CYANIDE (Hydrocyanic acid)	74-90-8	9.0E+00		4/00			**X**		**X**					**X**			
p-DICHLOROBENZENE	106-46-7	8.0E+02		1/01	**X**								**X**	**X**		**X**	
1,1,-DICHLOROETHYLENE … (see Vinylidene Chloride)																	
DIESEL EXHAUST … (see Particulate Emissions from Diesel-Fueled Engines)																	
DIETHANOLAMINE	111-42-2	3.0E+00		12/01			**X**							**X**			
N,N-DIMETHYL FORMAMIDE	68-12-2	8.0E+01		1/01	**X**											**X**	
1,4-DIOXANE✣ (1,4-Diethylene dioxide)	123-91-1	3.0E+03		4/00	**X**		**X**						**X**				
EPICHLOROHYDRIN (1-Chloro-2,3-epoxypropane)	106-89-8	3.0E+00		1/01						**X**						**X**	
1,2-EPOXYBUTANE	106-88-7	2.0E+01		1/01			**X**									**X**	
ETHYL BENZENE	100-41-4	2.0E+03		2/00	**X**			**X**	**X**				**X**				
ETHYL CHLORIDE (Chlorethane)	75-00-3	3.0E+04		4/00	**X**			**X**									
ETHYLENE DIBROMIDE[TAC] (1,2-Dibromoethane)	106-93-4	8.0E-01		12/01											**X**		
ETHYLENE DICHLORIDE[TAC] (1,2-Dichloroethane)	107-06-2	4.0E+02		1/01	**X**												
ETHYLENE GLYCOL	107-21-1	4.0E+02		4/00				**X**					**X**			**X**	
ETHYLENE OXIDE[TAC] (1,2-Epoxyethane)	75-21-8	3.0E+01		1/01										**X**			

APPENDIX L - TABLE 3 OEHHA/ARB CHRONIC INHALATION AND ORAL REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL	Date ◆ Value Reviewed [Added]	Target Organs													
					Alimentary	Bone	Cardiovascular	Developmental	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Reproductive	Respiratory	Skin
Fluorides	1101	1.3E+1	4.0E-2	8/03		X				✓*						✓	✓*
HYDROGEN FLUORIDE (Hydrofluoric acid)	7664-39-3	1.4E+1	4.0E-2	8/03		X				X*						X	X*
FORMALDEHYDE TAC	50-00-0	3.0E+00		2/00						X						X	
GLUTARALDEHYDE	111-30-8	8.0E-02		1/01												X	
GLYCOL ETHERS	1115																
ETHYLENE GLYCOL ETHYL ETHER – EGEE	110-80-5	7.0E+01		2/00							X				X		
ETHYLENE GLYCOL ETHYL ETHER ACETATE - EGEEA	111-15-9	3.0E+02		2/00				X									
ETHYLENE GLYCOL METHYL ETHER – EGME	109-86-4	6.0E+01		2/00											X		
ETHYLENE GLYCOL METHYL ETHER ACETATE – EGMEA	110-49-6	9.0E+01		2/00											X		
n-HEXANE	110-54-3	7.0E+03		4/00										X			
HYDRAZINE	302-01-2	2.0E-01		1/01	X				X								
HYDROCHLORIC ACID (Hydrogen chloride)	7647-01-0	9.0E+00		2/00												X	
HYDROGEN CYANIDE (Hydrocyanic acid) (see Cyanide Compounds)																	
HYDROGEN FLUORIDE (Hydrofluoric acid) (see Fluorides & Compounds)																	
HYDROGEN SULFIDE	7783-06-4	1.0E+01		4/00												X	
ISOPHORONE	78-59-1	2.0E+03		12/01	X			X									
ISOPROPYL ALCOHOL (Isopropanol)	67-63-0	7.0E+03		2/00				X					X				
MALEIC ANHYDRIDE	108-31-6	7.0E-01		12/01												X	
MANGANESE AND COMPOUNDS	7439-96-5 [1132]	2.0E-01		4/00										X			
MERCURY AND COMPOUNDS (INORGANIC)	7439-97-6 [1133]	9.0E-02		2/00										X			
			3.0^{E}-04	10/00 [1/92]								X	X				
Mercuric chloride	*7487-94-7*	*9.0E-02*		*2/00*										✓			
			3.0E-04	*10/00 [1/92]*								✓	✓				
MERCURY AND COMPOUNDS (ORGANIC)	N/A																
METHANOL	67-56-1	4.0E+03		4/00				X									
METHYL BROMIDE (Bromomethane)	74-83-9	5.0E+00		2/00				X						X		X	
METHYL tertiary-BUTYL ETHER	1634-04-4	8.0E+03		2/00	X					X			X				

APPENDIX L - TABLE 3 OEHHA/ARB CHRONIC INHALATION AND ORAL REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ❁

Substance ❁	Chemical▼ Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL	Date ◆ Value Reviewed [Added]	Target Organs												
					Alimentary	Bone	Cardiovascular	Developmental	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Reproductive	Respiratory	Skin
METHYL CHLOROFORM (1,1,1-Trichloroethane)	71-55-6	1.0E+03		2/00										X			
METHYL ISOCYANATE	624-83-9	1.0E+00		12/01											X	X	
METHYLENE CHLORIDE[TAC] (Dichloromethane)	75-09-2	4.0E+02		2/00			X							X			
4,4'-METHYLENE DIANILINE (AND ITS DICHLORIDE)	101-77-9	2.0E+01		12/01	X					X							
METHYLENE DIPHENYL ISOCYANATE	101-68-8	7.0E-01		1/01												X	
NAPHTHALENE	91-20-3	9.0E+00		4/00												X	
NICKEL AND COMPOUNDS[TAC]	7440-02-0 [1145]	5.0E-02		2/00							X					X	
			5.0E-02	10/00	X												
Nickel acetate	*373-02-4*	*5.0E-02*		*2/00*							✓					✓	
			5.0E-02	*10/00*	✓												
Nickel carbonate	*3333-39-3*	*5.0E-02*		*2/00*							✓					✓	
			5.0E-02	*10/00*	✓												
Nickel carbonyl	*13463-39-3*	*5.0E-02*		*2/00*							✓					✓	
			5.0E-02	*10/00*	✓												
Nickel hydroxide	*12054-48-7*	*5.0E-02*		*2/00*							✓					✓	
			5.0E-02	*10/00*	✓												
Nickelocene	*1271-28-9*	*5.0E-02*		*2/00*							✓					✓	
			5.0E-02	*10/00*	✓												
NICKEL OXIDE	1313-99-1	1.0E-01		2/00							X					X	
			5.0E-02	10/00	X												
Nickel refinery dust from pyrometallurgical process	*1146*	*5.0E-02*		*2/00*							✓					✓	
			5.0E-02	*10/00*	✓												
Nickel subsulfide	*12035-72-2*	*5.0E-02*		*2/00*							✓					✓	
			5.0E-02	*10/00*	✓												
PCB (POLYCHLORINATED BIPHENYLS (speciated)∇																	
3,3',4,4'-TETRACHLOROBIPHENYL (77)	35298-13-3	4.0E-01		8/03	X			X	X		X				X	X	
			1.0E –04	8/03	X			X	X		X				X	X	
3,4,4',5-TETRACHLOROBIPHENYL (81)	70362-50-4	4.0E-01		8/03	X			X	X		X				X	X	
			1.0E –04	8/03	X			X	X		X				X	X	

APPENDIX L - TABLE 3 OEHHA/ARB CHRONIC INHALATION AND ORAL REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL	Date ◆ Value Reviewed [Added]	Target Organs												
					Alimentary	Bone	Cardiovascular	Developmental	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Reproductive	Respiratory	Skin
2,3,3',4,4' - PENTACHLOROBIPHENYL (105)	32598-14-4	4.0E-01		8/03	X			X	X		X				X	X	
			1.0E –04	8/03	X			X	X		X				X	X	
2,3,4,4'5 - PENTACHLOROBIPHENYL (114)	74472-37-0	8.0E-02		8/03	X			X	X		X				X	X	
			2.0E –05	8/03	X			X	X		X				X	X	
2,3'4,4',5- PENTACHLOROBIPHENYL (118)	31508-00-6	4.0E-01		8/03	X			X	X		X				X	X	
			1.0E –04	8/03	X			X	X		X				X	X	
2',3,4,4',5 - PENTACHLOROBIPHENYL (123)	65510-44-3	4.0E-01		8/03	X			X	X		X				X	X	
			1.0E –04	8/03	X			X	X		X				X	X	
3,3',4,4',5 - PENTACHLOROBIPHENYL (126)	57465-28-8	4.0E-04		8/03	X			X	X		X				X	X	
			1.0E –07	8/03	X			X	X		X				X	X	
2,3,3',4,4',5-HEXACHLOROBIPHENYL (156)	38380-08-4	8.0E-02		8/03	X			X	X		X				X	X	
			2.0E –05	8/03	X			X	X		X				X	X	
2,3,3',4,4',5'-HEXACHLOROBIPHENYL (157)	69782-90-7	8.0E-02		8/03	X			X	X		X				X	X	
			2.0E –05	8/03	X			X	X		X				X	X	
2,3',4,4',5,5'-HEXACHLOROBIPHENYL (167)	52663-72-6	4.0E-00		8/03	X			X	X		X				X	X	
			1.0E –03	8/03	X			X	X		X				X	X	
3,3',4,4'5,5' - HEXACHLOROBIPHENYL (169)	32774-16-6	4.0E-03		8/03	X			X	X		X				X	X	
			1.0E –06	8/03	X			X	X		X				X	X	
2,3,3'4,4',5,5' - HEPTACHLOROBIPHENYL (189)	39635-31-9	4.0E-01		8/03	X			X	X		X				X	X	
			1.0 E-04	8/03	X			X	X		X				X	X	
PARTICULATE EMISSIONS FROM DIESEL-FUELED ENGINES[TAC]■	9901	5.0E+00[TAC]		8/98												X	
PERCHLOROETHYLENE[TAC] (Tetrachloroethylene)	127-18-4	3.5E+01[TAC]		10/91	X								X				
PHENOL	108-95-2	2.0E+02		4/00	X		X						X	X			
PHOSPHINE	7803-51-2	8.0E-1		9/02	X									X		X	
PHOSPHORIC ACID	7664-38-2	7.0E+00		2/00												X	
PHTHALIC ANHYDRIDE	85-44-9	2.0E+01		1/01												X	
POLYCHLORINATED DIBENZO-P-DIOXINS (PCDD) (AS 2,3,7,8-EQUIV)[TAC]●	1085 1086																

APPENDIX L - TABLE 3 OEHHA/ARB CHRONIC INHALATION AND ORAL REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ❁

Substance ❁	Chemical▼ Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL	Date ◆ Value Reviewed [Added]	Target Organs												
					Alimentary	Bone	Cardiovascular	Developmental	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Reproductive	Respiratory	Skin
2,3,7,8-TETRACHLORODIBENZO-*P*-DIOXIN[TAC]	1746-01-6	4.0E-05		2/00	X			X	X		X				X	X	
			1.0E-08	10/00	X			X	X		X				X	X	
1,2,3,7,8-PENTACHLORODIBENZO-*P*-DIOXIN	40321-76-4	4.0E-05		2/00	X			X	X		X				X	X	
			1.0E-08	10/00	X			X	X		X				X	X	
1,2,3,4,7,8-HEXACHLORODIBENZO-*P*-DIOXIN	39227-28-6	4.0E-04		2/00	X			X	X		X				X	X	
			1.0E-07	10/00	X			X	X		X				X	X	
1,2,3,6,7,8-HEXACHLORODIBENZO-*P*-DIOXIN	57653-85-7	4.0E-04		2/00	X			X	X		X				X	X	
			1.0E-07	10/00	X			X	X		X				X	X	
1,2,3,7,8,9-HEXACHLORODIBENZO-*P*-DIOXIN	19408-74-3	4.0E-04		2/00	X			X	X		X				X	X	
			1.0E-07	10/00	X			X	X		X				X	X	
1,2,3,4,6,7,8-HEPTACHLORODIBENZO-*P*-DIOXIN	35822-46-9	4.0E-03		2/00	X			X	X		X				X	X	
			1.0E-06	10/00	X			X	X		X				X	X	
1,2,3,4,6,7,8,9-OCTACHLORODIBENZO-*P*-DIOXIN	3268-87-9	4.0E-01		2/00	X			X	X		X				X	X	
			1.0E-04	10/00	X			X	X		X				X	X	
POLYCHLORINATED DIBENZOFURANS (PCDF) (AS 2,3,7,8-EQUIV) [TAC] ●	1080																
2,3,7,8-TETRACHLORODIBENZOFURAN	5120-73-19	4.0E-04		2/00	X			X	X		X				X	X	
			1.0E-07	10/00	X			X	X		X				X	X	
1,2,3,7,8-PENTACHLORODIBENZOFURAN	57117-41-6	8.0E-04		2/00	X			X	X		X				X	X	
			2.0E-07	10/00	X			X	X		X				X	X	
2,3,4,7,8-PENTACHLORODIBENZOFURN	57117-31-4	8.0E-05		2/00	X			X	X		X				X	X	
			2.0E-08	10/00	X			X	X		X				X	X	
1,2,3,4,7,8-HEXACHLORODIBENZOFURAN	70648-26-9	4.0E-04		2/00	X			X	X		X				X	X	
			1.0E-07	10/00	X			X	X		X				X	X	
1,2,3,6,7,8-HEXACHLORODIBENZOFURAN	57117-44-9	4.0E-04		2/00	X			X	X		X				X	X	
			1.0E-07	10/00	X			X	X		X				X	X	
1,2,3,7,8,9-HEXACHLORODIBENZOFURAN	72918-21-9	4.0E-04		2/00	X			X	X		X				X	X	
			1.0E-07	10/00	X			X	X		X				X	X	
2,3,4,6,7,8-HEXACHLORODIBENZOFURAN	60851-34-5	4.0E-04		2/00	X			X	X		X				X	X	
			1.0E-07	10/00	X			X	X		X				X	X	

APPENDIX L - TABLE 3 OEHHA/ARB CHRONIC INHALATION AND ORAL REFERENCE EXPOSURE LEVELS AND TARGET ORGANS ◉

Substance ◉	Chemical▼ Abstract Service Number (CAS)	Chronic Inhalation REL ($\mu g/m^3$)	Chronic Oral REL	Date ◆ Value Reviewed [Added]	Target Organs												
					Alimentary	Bone	Cardiovascular	Developmental	Endocrine	Eye	Hematologic	Immune	Kidney	Nervous	Reproductive	Respiratory	Skin
1,2,3,4,6,7,8-HEPTACHLORODIBENZOFURAN	67562-39-4	4.0E-03		2/00	X			X	X		X				X	X	
			1.0E-06	10/00	X			X	X		X				X	X	
1,2,3,4,7,8,9-HEPTACHLORODIBENZOFURAN	55673-89-7	4.0E-03		2/00	X			X	X		X				X	X	
			1.0E-06	10/00	X			X	X		X				X	X	
1,2,3,4,6,7,8,9-OCTACHLORODIBENZOFURAN	39001-02-0	4.0E-01		2/00	X			X	X		X				X	X	
			1.0E-04	10/00	X			X	X		X				X	X	
PROPYLENE (PROPENE)	115-07-1	3.0E+03		4/00												X	
PROPYLENE GLYCOL MONOMETHYL ETHER	107-98-2	7.0E+03		2/00	X												
PROPYLENE OXIDE	75-56-9	3.0E+01		2/00												X	
SELENIUM AND COMPOUNDS (other than hydrogen selenide)	7782-49-2 [1170]	2.0E+01		12/01	X		X							X			
STYRENE	100-42-5	9.0E+02		4/00										X			
SULFURIC ACID	7664-93-9	1.0E+00		12/01												X	
Sulfuric Acid and Oleum	*7664-93-9*	*1.0E+00*		*12/01*												✓	
Sulfuric Trioxide	*7446-71-9*	*1.0E+00*		*12/01*												✓	
Oleum	*8014-95-7*	*1.0E+00*		*12/01*												✓	
TOLUENE	108-88-3	3.0E+02		4/00				X						X		X	
Toluene diisocyanates	*26471-62-5 1204*	*7.0E-02*		*1/01*												✓	
TOLUENE-2,4-DIISOCYANATE	584-84-9	7.0E-02		1/01												X	
TOLUENE-2,6-DIISOCYANATE	91-08-7	7.0E-02		1/01												X	
TRICHLOROETHYLENE[TAC]	79-01-6	6.0E+02		4/00						X				X			
TRIETHYLAMINE	121-44-8	2.0E+02		9/02						X		X				X	
VINYL ACETATE	108-05-4	2.0E+02		12/01												X	
VINYLIDENE CHLORIDE (1,1,-Dichloroethylene)	75-35-4	7.0E+01		1/01	X												
XYLENES (mixed isomers)	1330-20-7 1210	7.0E+02		4/00										X		X	
m-XYLENE	108-38-3	7.0E+02		4/00										X		X	
o-XYLENE	95-47-6	7.0E+02		4/00										X		X	
p-XYLENE	106-42-3	7.0E+02		4/00										X		X	

Purpose:	The purpose of this reference table is to provide a quick list of all health values that have been approved by the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) for use in facility health risk assessments conducted for the AB 2588 Air Toxics Hot Spots Program. The OEHHA has developed and adopted new risk assessment guidelines that update and replace the California Air Pollution Control Officers Association's (CAPCOA) *Air Toxics "Hot Spots" Program Revised 1992 Risk Assessment Guidelines, October 1993*. The OEHHA has adopted five technical support documents for these guidelines. This table lists the OEHHA adopted inhalation and oral noncancer chronic RELs. In addition, it lists the substances in Appendix A-I *(Substances For Which Emissions Must Be Quantified*) and Appendix F (*Criteria For Inputs For Risk Assessment Using Screening Air Dispersion Modeling*) of the ARB's *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG)*. OEHHA is still in the process of adopting new noncancer chronic RELs. Therefore, new health values will periodically be added to, or deleted from, this table. Users of this table are advised to monitor the OEHHA website (*www.oehha.ca.gov*) for any updates to the health values.
❁	Substances written in *italics* and with a ✓ do not have explicit OEHHA approved health values, but are included in this table to clarify applicability of OEHHA adopted heath effects values to individual or grouped substances listed in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines*, Appendix A-I list of *"Substances For Which Emissions Must Be Quantified"*.
▼	Chemical Abstract Service Number (CAS): For chemical groupings and mixtures where a CAS number is not applicable, the 4-digit code used in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG) Report* is listed. The 4-digit codes enclosed in brackets [ ] are codes that have been phased out, but may still appear on previously reported Hot Spots emissions. For information on the origin and use of the 4-digit code, see the EICG report.
◆	Date Value Reviewed [Added]: This column lists the date that the health value was last reviewed by OEHHA and the Scientific Review Panel, and/or approved for use in the AB 2588 Air Toxics "Hot Spots" Program. If the health value is unchanged since it was first approved for use in the "Hot Spots" Program, then the date that the value was first approved for use by CAPCOA is listed within the brackets [ ]. • February 2000, April 2000, January 2001, and December 2001 are listed for the first set of 22, the second set of 16, the third set of 22, and the fourth set of 12 noncancer chronic RELs, respectively. • October 2000 is listed for the oral chronic RELs. The chronic REL for carbon disulfide was adopted in May 2002. Chronic RELS for phosphine and triethylamine were adopted in September 2002. Chronic RELs for fluorides including hydrogen fluoride were adopted August 2003. • For the substances identified as Toxic Air Contaminants, the Air Resources Board hearing date is listed. The date for acetaldehyde represents the date the value was approved by the Scientific Review Panel.
TAC	Toxic Air Contaminant: The Air Resources Board has identified this substance as a Toxic Air Contaminant.
★	Polychlorinated Biphenyls: Chronic Oral: The chronic oral value is U.S. EPA's 1996 oral Reference Dose for Aroclor-1254.
•	Polychlorinated Dibenzo-*p*-dioxins and Polychlorinated Dibenzofurans (also referred to as chlorinated dioxins and dibenzofurans): The OEHHA has adopted the World Health Organization 1997 (WHO-$_{97}$) Toxicity Equivalency Factor scheme for evaluating the cancer risk due to exposure to samples containing mixtures of polychlorinated dibenzo-*p*-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) and determining cancer risks for a number of specific PCB congeners. See Appendix A of OEHHA's *Technical Support Document For Describing Available Cancer Potency Factors* for more information about the scheme. See Appendix E of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part V; Technical Support Document; Guidance Manual for Preparation of Health Risk Assessments* for the methodology for calculating 2,3,7,8-equivalents for PCDD, PCDFs and a number of specific PCB congeners.
■	Particulate Emissions from Diesel-Fueled Engines: The unit risk factor and chronic REL were derived from whole diesel exhaust and should be used only for impacts from the inhalation pathway. The inhalation impacts from speciated emissions from diesel-fueled engines are already accounted for in the unit risk factor and REL. However, at the discretion of the risk assessor, speciated emissions from diesel-fueled engines may be used to estimate acute noncancer health impacts or the contribution to cancer risk or chronic noncancer health impacts for the non-inhalation exposure pathway. See Appendix D of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part V; Technical Support Document; Guidance Manual for Preparation of Health Risk Assessments* for more information.

Table last updated: August 2003