Chapter 7
Characterizing Risk and Hazard

What's Covered in Chapter 7:

PLEASE NOTE: for the purposes of this guidance, "we" refers to the U.S. EPA OSW.

The HHRAP is written for the benefit of a varied audience, including risk assessors, regulators, risk managers, and community relations personnel. However, the "you" to which we speak in this chapter is the performer of a risk assessment: the person (or persons) who will actually put the recommended methods into practice.

The final step of a risk assessment is risk characterization. This involves combining the exposure quantities generated in Chapter 6, and the toxicity benchmarks available in the HHRAP companion database, to calculate the excess lifetime cancer risks (risk) and noncancer hazards (hazard) for each of the pathways and receptors identified in Chapter 4. Risks (and hazards) are then summed for each receptor, across all applicable exposure pathways, to obtain an estimate of total individual risk and hazard. Risk characterization also involves documenting the uncertainties and limitations associated with the rick assessment, as described in Chapter 8.

It is important that risk characterization exhibit the core values of transparency, clarity, consistency, and reasonableness (please see the related EPA Information Quality Guidelines recommendations as discussed in Chapter 1, page 1-11).

Risk from exposure to combustor emissions is the probability that a human receptor will develop ***cancer***, based on a unique set of exposure, model, and toxicity assumptions. We recommend using the slope or unit risk factor in risk assessments to estimate the probability of an individual developing cancer as a

result of exposure to a particular level of a COPC. For example, a risk of 1×10^{-5} is interpreted to mean that an individual has up to a one in 100,000 chance of developing cancer during their lifetime from the exposure being evaluated. In contrast, ***hazard*** is the potential for developing ***noncancer*** health effects as a result of exposure to COPCs. A hazard is not a probability but, rather, a comparison (calculated as a ratio) of a receptor's potential exposure relative to a standard exposure level (*RfD* or *RfC*). The standard exposure level is calculated over a similar exposure period and is estimated to pose no appreciable likelihood of adverse health effects to potential receptors, including special populations (U.S. EPA 1989e).

Risks and hazards here are typically characterized for single scenarios, and are referred to as individual risks and hazards (U.S. EPA 1989e; 1994g; NC DEHNR 1997). Individual risk and hazard descriptors are intended to convey information about the potential risks to individuals potentially impacted by emissions from a facility burning hazardous waste. A risk assessment developed following the procedures described in Chapters 2 through 8 and Appendixes B and C will provide

- quantitative and qualitative estimates of risk and hazard associated with exposure to COPCs;
- estimates of blood levels associated with exposure to lead;
- evaluation of infant exposure via breast milk to COPCs with appropriate biotransfer factors[1], and
- evaluation of acute risk and hazard resulting from direct inhalation.

If a permitting authority feels that you need to consider calculating population risks, we recommend following the applicable methods described in the U.S. EPA NCEA document, *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* (U.S. EPA 1998c).

Standard rules for rounding apply which will commonly lead to an answer of one significant figure in both risk and hazard estimates. For presentation purposes, hazard quotients (and hazard indices) and

[1] Currently 2,3,7,8-TCDD TEQ and dioxin-like PCBs are the only COPCs with biotransfer factors for the breastmilk pathway. However, appropriate biotransfer factors for other chemicals may become available and thus provide the information needed to include them in this pathway evaluation. We suggest consulting Chapter 9 (Breastmilk Pathway) of U.S. EPA (1998c).

cancer risk estimates are usually reported as one significant figure. We recommend rounding only the final reported results, not the intermediate calculations.

INFORMATION RECOMMENDED FOR RISK ASSESSMENT REPORT

- Indicate the scope of the risk assessment (match the level of effort to the scope)
- Summarize the major risk conclusions.
- Identify key issues (a key issue is critical to properly evaluate the conclusions). For example, was surrogate or measured emissions data used.
- Describe clearly the methods used to determine risk (provide qualitative narration of the quantitative results).
- Summarize the overall strengths and major uncertainties.

7.1 QUANTITATIVELY ESTIMATING CANCER RISK

As described above, risk estimates represent the incremental probability that an individual will develop cancer over a lifetime as a result of a specific exposure to a carcinogenic chemical (U.S. EPA 1989e). We recommend calculating these risks as follows:

Inhalation Cancer Risk

$$Cancer\ Risk = EC \times URF \qquad \text{Equation 7-1}$$

where

EC = Exposure concentration ($\mu g/m^3$) [see Chapter 6]
URF = Unit risk factor $(\mu g/m^3)^{-1}$

Ingestion Cancer Risk

$$Cancer\ Risk = LADD \times CSF \qquad \text{Equation 7-2}$$

where

$LADD$ = Lifetime average daily dose (mg/kg-day)
CSF = Cancer slope factor $(mg/kg\text{-}day)^{-1}$

PLEASE NOTE: In the *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (U.S. EPA 2005g), the Agency recommends estimating inhalation and ingestion cancer risk slightly differently for carcinogens the Agency determines to cause cancer by a mutagenic Mode of Action (MOA, as defined in *Guidelines for Carcinogen Risk Assessment* [U.S. EPA 2005f]). Unfortunately, we haven't completed our recommendations for how to implement the guidelines set out in U.S. EPA (2005f; g). We recommend periodically checking the EPA hazardous waste combustion web site (http://www.epa.gov/epaoswer/hazwaste/combust.htm) for updates on our recommendations.

It's possible for receptors to be exposed to multiple COPCs within a individual exposure pathway. We recommend estimating the total risk associated with exposure to all COPCs through a single exposure pathway as follows (U.S. EPA 1989e):

$$Cancer\ Risk_T = \sum_i Cancer\ Risk_i \qquad \text{Equation 7-3}$$

where

$Cancer\ Risk_T$	=	Total cancer risk for a specific exposure pathway
$Cancer\ Risk_i$	=	Cancer risk for COPC i for a specific exposure pathway

Receptors might be exposed through a number of exposure pathways (see Table 4-1). We consider it appropriate to sum risks from multiple exposure pathways for a given receptor. The cumulative risk posed to a receptor is the sum of total risks from each individual exposure pathway. Express the cumulative risk as follows:

$$Cumulative\ Cancer\ Risk = \sum Cancer\ Risk_T \qquad \text{Equation 7-4}$$

where

Cumulative Cancer Risk=		Cumulative cancer risk from multiple exposure pathways
$Cancer\ Risk_T$	=	Cumulative cancer risk for exposure pathway T

In addition to multiple pathways, a receptor might be exposed to emissions from multiple sources (See Chapter 2 for additional discussion on emission sources). In addition to emission source-specific risk/hazard estimates (see Chapter 3 regarding source-specific modeling), we recommend summing the risks from all modeled sources for each receptor at each exposure scenario location. For example, if a facility operates an incinerator and a boiler that both burn hazardous waste, sum the risks from both units for each receptor. For fugitive emissions from storage and handling of hazardous waste, add the risk

associated with fugitive emissions to the risks from the combustion unit for each receptor at each exposure scenario location.

We present the equations we recommend to estimate dose and risk levels in Appendix C. The HHRAP companion database presents inhalation *URF*s and oral *CSF*s for many potential COPCs. However, for each risk assessment, we recommend checking the hierarchy of toxicity benchmark and slope factor resources listed in Appendix A-2, Section A2.6 (Human Health Benchmarks) for updated values. We suggest using the same hierarchy to acquire toxicity values for COPCs not identified in Appendix A-2.

In the assessment of carcinogenic risk from COPCs, we recommend U.S. EPA-derived or reviewed health benchmarks (*URFs* and *CSFs*). However, for numerous compounds, a complete set of inhalation and oral EPA-derived health benchmarks are not available. In such cases, we calculated the health benchmarks presented in the companion database based on available U.S. EPA-derived benchmark values.

If relevant information is not available from these sources, we recommend contacting the appropriate permitting authority, which may be able to assist in developing the necessary toxicity values. For example, Minimum Risk Levels published by the Agency for Toxic Substances and Disease Registry (ATSDR) might be applicable.

7.2 QUANTITATIVELY ESTIMATING NONCANCER HAZARD

Standard risk assessment models assume that, for most chemicals with noncancer effects, the noncancer effects exhibit a threshold response[2],. That is, there is a level of exposure below which no adverse effects will be observed (U.S. EPA 1989e). The default approaches used by USEPA to assess the potential for health effects associated with a nonlinear or threshold relationship with exposure as set out in U.S. EPA (2002; 2005f) involve:

1. Comparing an estimate of ingested exposure (see Chapter 6) to an *RfD* for oral exposures; and

[2] Some chemicals don't demonstrate a threshold response. Lead and ozone are two examples of chemicals with noncancer effects that don't have a threshold below which no adverse effects are observed.

2. Comparing an estimated chemical-specific air concentration to the *RfC* for direct inhalation exposures.

An *RfD* is a daily oral intake rate that is estimated to pose no appreciable risk of adverse health effects, even to sensitive populations, over a 70-year lifetime. Similarly, an *RfC* is an estimated daily concentration of a chemical in air, the exposure to which over a specific exposure duration poses no appreciable risk of adverse health effects, even to sensitive populations (U.S. EPA 2002).

The exposure durations assumed for the exposure pathways identified in Table 4-1 range from subchronic to chronic in relative length. However, we consider it appropriate to use chronic *RfDs* and *RfCs* to evaluate all recommended exposure pathways. The comparisons of oral and inhalation exposure estimates to *RfD* and *RfC* values, described above, are known as hazard quotients (*HQ*), which are calculated as follows:

$$HQ = \frac{ADD}{RfD} \; or \; HQ = \frac{EC}{RfC} \qquad \text{Equation 7-5}$$

where

HQ	=	Hazard quotient (unitless)
ADD	=	Average daily dose (mg/kg-day)
RfD	=	Reference dose (mg/kg-day)
EC	=	Exposure air concentration (mg/m^3)
RfC	=	Reference concentration (mg/m^3)

Please note that each program office within U.S. EPA determines what *HQ* level poses a concern to exposed individuals. For example, Superfund has determined that an *HQ* of less than or equal to 1 is considered health-protective (U.S. EPA 1989e). However, because *RfDs* and *RfCs* do not have equal accuracy or precision, and are not based on the same severity of effect, the level of concern does not increase linearly as an *HQ* approaches and exceeds 1 (U.S. EPA 1989e). In addition, noncancer estimates only identify the exposure level below which adverse effects are unlikely; an RfD or RfC does not say anything about incremental risk for higher exposures (U.S. EPA 1998c).

Also note that background exposures may be an important consideration in setting *HQ* levels of concern. This is because you generally model noncancer effects as thresholds, and biologic systems (including human receptors) do not distinguish between exposures from regulated versus non-regulated sources. In

certain cases, a permitting authority may elect to adjust the assessed facility-specific *HQ* downward, to account for any exposure that individuals may have from non-assessed sources.

As with carcinogenic chemicals, a receptor might be exposed to multiple chemicals associated with noncancer health effects. We recommend calculating the total chronic hazard for each exposure pathway by following the procedures outlined in U.S. EPA (1986e; 1989e; and 2000e). Specifically, the total chronic hazard attributable to exposure to all COPCs through a single exposure pathway is known as a hazard index (*HI*). The *HI* is calculated as follows:

$$HI = \sum_i HQ_i \qquad \text{Equation 7-6}$$

where

HI	=	Hazard index for a specific exposure pathway
HQ_i	=	Hazard quotient for COPC *i*

This method assumes that the health effects of the various COPCs are additive. This method is a simplification of the *HI* concept because it doesn't, at this stage, directly consider the portal of entry associated with each exposure pathway (i.e. inhalation, or ingestion). This method also doesn't consider the often unique toxic endpoints and toxicity mechanisms of the various COPCs.

As discussed in Section 7.1 for carcinogenic risks, a receptor might be exposed to COPCs associated with noncancer health effects through more than one exposure pathway, and from multiple emissions sources. We recommend estimating the noncancer hazards from each modeled source (including fugitive emissions) separately, as well as all sources summed for each receptor. We consider it reasonable to estimate a receptor's total hazard as the sum of the *HI*s for each of the exposure pathways chosen for the receptor. Specifically, a receptor's cumulative hazard is the sum of hazards from each individual exposure pathway, expressed as follows:

$$\textit{Cumulative HI} = \sum HI \qquad \text{Equation 7-4}$$

where

Cumulative HI	=	Cumulative hazard index from all scenario-specific exposure pathways
HI	=	Hazard index for a specific exposure pathway

As in U.S. EPA (1989e), we recommend further evaluating a cumulative *HI* which exceeds the target hazard level. A cumulative *HI* can exceed the target hazard level due to either

- One or more COPCs with an *HQ* exceeding the target hazard level, or
- The summation of several COPC-specific *HQ*s that are each less than the target hazard level.

In the former case, you can interpret the presence of at least one COPC-specific hazard greater than the target hazard level as indicating the potential for noncancer health effects. In the latter case, you need to perform a detailed analysis to determine whether the potential for noncancer health effects is accurately estimated by the total *HI*. This is because the toxicological effects associated with exposure to multiple chemicals, often through different exposure pathways, may not be additive. The total *HI* might therefore overestimate the potential for noncancer health effects.

To address this issue, we recommend summing the COPC-specific hazards according to toxicological similarity (e.g. the same target organs or systems) (U.S. EPA 2000e). This process is referred to as segregating the *HI*. It is especially important to consider any differences related to exposure route. If any segregated *HI* exceeds the target hazard level, noncancer health effects cannot be ruled out. However, if all segregated *HI*s are less than the target hazard level, noncancer health effects are not likely to result from exposure to the COPCs included in the *HI*.

Summing all *HI*s
As stated above, estimating a single *HI* encompassing all *HI*s across all exposure pathways is a simplification of the *HI* concept. However, it may save valuable resources: if the single *HI* is not above the target hazard level, then no further segregation would be necessary. We recommend this as a first step, and going to the expense of segregating *HI*s only if the single *HI* falls above the target hazard level.

Technically, segregating the *HI* based only on target organs or systems is a simplification of *HI*. Ideally, the *HI* would also be segregated according to the often unique mechanisms of toxicity of the COPCs. However, segregating the *HI* based on mechanisms of toxicity is beyond a screening level or initial risk evaluation approach (U.S. EPA 2000e).

The HHRAP companion database includes information on target organs and systems that are affected by each COPC. The database also presents *RfDs* and *RfCs* for these same COPCs. If you include COPCs not identified in Appendix A-2 (and therefore not in the companion database) in the risk assessment, we recommend obtaining *RfDs* and *RfCs* for these compounds using the hierarchy of toxicity benchmark and

slope factor resources listed in Appendix A-2, Section A2.6 (Human Health Benchmarks). If relevant information is not available from these sources, we recommend working with the permitting authority to contact the U.S. EPA National Center for Environmental Assessment (NCEA) office in Cincinnati, Ohio. NCEA personnel may be able to assist in developing the necessary toxicity values.

In the assessment of noncancer risk from COPCs, we recommend U.S. EPA-derived or reviewed *RfD*s and *RfC*s. However, for numerous compounds, a complete set of inhalation and oral health benchmarks is not available. If such was the case for COPCs listed in Appendix A-2, we calculated the health benchmarks presented based on available U.S. EPA-derived benchmarks values. For instance, if the *oral RfD* (mg/kg/day) was available and the *RfC* (mg/m^3) was not, we calculated the *RfC* by multiplying the *RfD* by an average human inhalation rate of 20 m^3/day and dividing by the average human body weight of 70 kg. This conversion is called a route-to-route extrapolation, which assumes that the toxicity of the given compound is equivalent over all routes of exposure.

Route-to-route extrapolation introduces additional uncertainty into the risk assessment, and there isn't Agency consensus regarding the appropriateness of its use. This method assumes that the qualitative data supporting the benchmark value for a certain route also applies to the route in question. For example, if an *RfD* is available and you calculate the *RfC* from that value, you are assuming that the toxicity seen following oral exposure will be equivalent to toxicity following inhalation exposure. This assumption could overestimate or underestimate the toxicity of the given compound following inhalation exposure.

Because of the degree of uncertainty involved in using toxicity benchmark values calculated based on route-to-route extrapolation, we recommend using route-to-route extrapolations for organic compounds (but not inorganic), and revisiting the appropriateness of applying this extrapolation for individual chemicals if they are found to be risk drivers. An example might include using route-to-route extrapolations as the first step in a screening risk assessment, then expending resources evaluating the appropriateness of only those extrapolations associated with risk drivers. Including this further evaluation (a qualitative assessment of the toxicity information available for the compound and exposure route) in the Uncertainty section of the risk assessment report will enable the risk manager to make an informed decision concerning the validity of values calculated based on route-to-route extrapolation.

7.3 TARGET LEVELS

Target levels are risk management-based and set by the permitting authority. Target values are not a discrete indicator of observed adverse effect. If a risk estimate falls below target levels, a regulatory authority may, without further investigation, conclude that a proposed action does not present an unacceptable risk. A risk estimate that exceeds these targets, however, would not, in and of itself, necessarily indicate that the proposed action is not safe or that it presents an unacceptable risk. Rather, a risk estimate that exceeds a target value triggers further careful consideration of the underlying scientific basis for the calculation.

7.4 ESTIMATING ACUTE EXPOSURE FROM DIRECT INHALATION

In addition to long-term chronic effects, we recommend considering short-term or acute effects from direct inhalation of vapor phase and particle phase COPCs. Short-term emissions don't typically have a significant impact through the indirect exposure pathways (as compared to impacts from long-term emissions). Therefore, we recommend evaluating acute effects only through the short-term (maximum 1-hour) inhalation of vapors and particulates exposure pathway of the acute risk scenario. We give our recommendations for where and when to evaluate the acute risk scenario in Sections 4.2 and 4.3.
In order to establish acute inhalation exposure criteria (AIEC), we needed to identify and evaluate

1. Existing guidelines for acute inhalation exposure; and
2. Existing approaches for developing acute inhalation exposure levels.

Existing approaches are composed of hierarchical guidelines for acute inhalation exposure, ranked in order of applicability and technical basis, and all being protective of the general public.

> ***Please Note***: hierarchical approaches are needed because criteria values are COPC-specific, and no single organization or method has developed acute criteria values or benchmarks for all of the potential COPCs.

7.4.1 Existing Hierarchical Approaches for Acute Inhalation Exposure

Existing guidelines or criteria for evaluating acute inhalation exposure have been or are being developed by several organizations in the United States, including:

- Agency for Toxic Substances and Disease Registry (ATSDR 1997);
- American Conference of Governmental Industrial Hygienists (ACGIH 1996);
- American Industrial Hygiene Association (AIHA 1997);
- California Environmental Protection Agency (Cal/EPA) (Cal/EPA 1999);
- National Advisory committee (NAC 1997); and
- National Institute of Occupational Safety and Health (NIOSH 1994);
- National Research Council Committee on Toxicology (NRC COT 1986; U.S. EPA 1987b);
- Occupational Safety and Health Administration (NIOSH 1994);
- U.S. Department of Energy, Subcommittee on Consequence Assessment and Protective Actions (SCAPA) (SCAPA 2001a; 2001b).
- U.S. EPA (U.S. EPA 1987b);

Acute inhalation exposure guidelines and criteria are

- Designed to protect a variety of exposure groups, including occupational workers, military personnel, and the general public,
- Based on varying exposure durations up to 24 hours in length, and
- Intended to protect against a variety of toxicity endpoints ranging from discomfort or mild adverse health effects to serious, debilitating, and potentially life-threatening effects, up to and including death.

Hierarchical approaches for establishing acute inhalation exposure levels protective of the general public have been developed by a variety of organizations and teams of organizations. These organizations include:

- U.S. Department of Defense (DoD 1996);
- U.S. Department of Energy (DoE) (SCAPA 1997a; WSRC 1998).
- U.S. EPA Region 3 (EPA 1996b);
- U.S. EPA Region 10 (U.S. EPA 1996a); and

- Federal Emergency Management Agency, Department of Transportation (DoT), and U.S. EPA (U.S. EPA 1993i).

The acute inhalation exposure guidelines developed by these organizations are generally quite heterogenous, developed to protect different subpopulations against different effects and apply to various exposure durations. All the hierarchical approaches listed above except the SCAPA approach needed to adjust the existing guidelines using safety factors (usually multiples of 10) to account for differences in exposure group, exposure duration, and toxicity endpoint, to arrive at acute inhalation exposure values applicable to the general public.

In contrast to the hierarchical approaches developed using safety factors, the DoE's Emergency Management Advisory Committee's SCAPA developed temporary emergency exposure limits (TEELs) based on tiered, formula-like statistical analyses between existing guidelines for acute inhalation exposure and AIHA emergency response planning guidelines (ERPG) (Craig et al. 1995; WSRC 1998). The methodology is described at http://www.orau.gov/emi/scapa/Method_for_deriving_TEELs.pdf and available on-line at http://www.atlintl.com/DOE/teels/teel/teel_pdf.html. Like ERPGs, TEELs are multiple-tiered, representing concentrations associated with no effects (TEEL-0), mild, transient effects (TEEL-1), irreversible or serious effects (TEEL-2), and potentially life-threatening (TEEL-3). DOE developed TEELs for situations where no other value is available. TEELs do not undergo peer review. For compounds for which TEEL values could not be developed using this approach, SCAPA developed a supplementary approach using available toxicity information, primarily (1) lethal dose and concentration median, and (2) lethal dose and concentration low values (DoE 1997a).

7.4.2 Our Recommended Hierarchical Approach

After reviewing the existing hierarchical approaches, we recommend the following approach. Because of the daily operations of most combustion units and the potential for upset conditions to sometimes occur during operations, we consider acute values that address intermittent exposures more appropriate and more protective than values that are based on the assumption that acute exposures will be one-time only. When available, we recommend using values from all of the sources that are based on one-hour exposures.

1. **Cal/EPA Acute RELs** – the concentration in air at or below which no adverse health effects are anticipated in the general population, including sensitive individuals, for a specified exposure period (Cal/EPA 1999)
(On-Line Address – http://www.oehha.ca.gov/air/pdf/acuterel.pdf)

2. **Acute inhalation exposure guidelines (AEGL-1)** – "the airborne concentration of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic nonsensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure." (NOAA 2001; U.S. EPA 2001a) (On-Line Address – http://www.epa.gov/oppt/aegl/chemlist.htm)

3. **Level 1 emergency planning guidelines (ERPG-1)** – "the maximum concentration in air below which it is believed nearly all individuals could be exposed for up to one hour without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor." (DoE 2001; SCAPA 2001b)
(On-Line Address – http://www.bnl.gov/scapa/scapawl.htm)

4. **Temporary emergency exposure limits (TEEL-1)** – "the maximum concentration in air below which it is believed nearly all individuals could be exposed without experiencing other than mild transient adverse health effects or perceiving a clearly defined odor." (DoE 2001; SCAPA 2001a) (On-Line Address – http://tis-hq.eh.doe.gov/web/Chem_Safety/teel.html)

5. **AEGL-2 values** – "the airborne concentration of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape." AEGL-2 values are to be used only if lower ERPG-1 or TEEL-1 values are not available. (NOAA 2001; U.S. EPA 2001a) (On-Line Address – http://www.epa.gov/oppt/aegl/chemlist.htm)

The hierarchy is presented in order of preference, from 1 (most preferred) to 5 (least preferred). We generally recommend the Acute Reference Exposure Levels (Acute RELs) developed by Cal/EPA (Cal/EPA 1999) as the first choice for acute inhalation values. If no acute REL value is available for a given COPC, you can work down the list in order. If no AEGL-1 value is available, but an AEGL-2 value is available, select the AEGL-2 as the AIEC only if it's a more protective value (lower in concentration) than an ERPG-1, or a TEEL-1 value if either of these values is available. If no acute values are available for a COPC, an acute value can be developed following the toxicity-based approach used by SCAPA (Tier 5) (DoE 1997a; WSRC 1998). The companion database provides a listing of AIECs compiled from values currently available following the hierarchical approach presented above.

Please note that the TEEL-1 values (SCAPA 2001a) are calculated assuming a 15-minute exposure period. As discussed in Section 3.10, for the purposes of this protocol, we recommend evaluating risks due to acute exposure based on the highest 1-hour average air concentrations. Therefore, the TEEL-1 values were extrapolated from a 15-minute to a 1-hour exposure basis using a modification to Haber's Rule developed by ten Berge et al (1986) and used by Cal/EPA to develop acute RELs (Cal/EPA 1999), as shown below.

$$C^n \not{\ell} T = K \qquad \text{Equation 7-8}$$

where

C	=	Concentration (mg/m^3)
n	=	Constant greater than zero (unitless)
T	=	Time of exposure (hour)
K	=	Constant level or severity of response (unitless)

Where available, chemical-specific values for the exponent *n* were used to make the extrapolations (Cal/EPA 1999). For chemicals for which a chemical-specific value of *n* was not available, extrapolations were made using a value of n = 1, as recommended by OEHHA, because the extrapolations were all based on an initial exposure period (15-minutes) of less than 1 hour duration (Cal/EPA 1999).

Using the modified form of Haber's Rule allows you to consider contributions by both concentration and time to the overall severity of effect. However, we highly recommend taking special care interpreting the extrapolated air concentrations, as they aren't absolutes. For example, chemical-specific values of the exponent *n* are sometimes based on a relatively limited set of dose-response data. Also, the majority of extrapolated TEEL-1 values were calculated using default exponent values and, therefore, are likely to be even less certain than exponent values based on limited data sets.

The EPA IRIS program is currently developing additional acute reference values that do not exclude intermittent exposures. When available, we recommend using those values (referred to as Acute Reference Concentrations [Acute RfCs]) as the first choice, with the Cal/EPA acute RELs second in the hierarchy.

7.4.3 Characterizing Potential Health Effects from Acute Exposure

We recommend characterizing the potential for adverse health effects from acute exposure to COPC-specific emissions by comparing the acute air concentration (C_{acute}) resulting from maximum

emissions over a 1-hour period to the COPC-specific AIEC (see Appendix C, Table C-4-1). This comparison is known as the acute hazard quotient (AHQ_{inh}). Chapter 3 discusses air dispersion modeling related to obtain 1-hour maximum values. Appendix B, Table B-6-1 describes how to calculate C_{acute}. We recommend using Equation 7-9 to calculate the AHQ_{inh}:

$$AHQ_{inh} = \frac{C_{acute} \cdot 0.001}{AIEC} \qquad \text{Equation 7-9}$$

where

AHQ_{inh}	=	Acute hazard quotient (unitless)
C_{acute}	=	Acute air concentration (:g/m^3)
0.001	=	Conversion factor (mg/:g)
AIEC	=	Acute inhalation exposure criteria (mg/m^3)

We recommend calculating acute hazard quotients at the selected acute exposure scenario locations (see Sections 4.2 and 4.3) for COPCs specific to emissions from each source, and from all facility sources combined. We recommend summing acute hazard quotients from individual chemicals (e.g. acid gases), if they have similar effects. Setting target levels for evaluating acute hazard quotients is a risk management decision made by the permitting authority.