Chapter 7
Risk and Hazard Characterization

What's Covered in Chapter 7:

- Individual Risk and Hazard Estimation
- Quantitative Estimation of Cancer Risk
- Quantitative Estimation of Noncancer Effects
- Target Levels
- Acute Exposure Resulting from Direct Inhalation

Risk characterization must exhibit the core values of transparency, clarity, consistency, and reasonableness. The final step of a risk assessment is the calculation of the upper-bound excess lifetime cancer risks (risk) and noncarcinogenic hazards (hazard) for each of the pathways and receptors identified in Chapter 4. Risks and hazards are then summed for specific receptors, across all applicable exposure pathways, to obtain an estimate of total individual risk and hazard for specific receptors.

Risk from exposure to combustion emissions is the probability that a receptor will develop cancer, based on a unique set of exposure, model, and toxicity assumptions. The slope factor is used in risk assessments to estimate and upper bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen. For example, a risk of 1×10^{-5} is interpreted to mean that an individual has no more than, and likely less than, a one in 100,000 chance of developing cancer from the exposure being evaluated. In contrast, hazard is quantified as the potential for developing noncarcinogenic health effects as a result of exposure to COPCs, averaged over an exposure period. A hazard is not a probability but, rather, a measure (calculated as a ratio) of the magnitude 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 are typically characterized

for a single receptor and are referred to as individual risks and hazards (U.S. EPA 1989e; 1994g; NC DEHNR 1997).

At least one U.S. EPA guidance document, concerning the characterization of risks and hazards associated with combustion facilities, suggests that population risks and hazards should be calculated in addition to individual risks (U.S. EPA 1993h). Population risk is defined as the aggregate risk of the exposed population; it takes into account the risk associated with various exposure scenarios and the number of individuals represented by each exposure scenario. Therefore, U.S. EPA OSW recommends that the risk assessment address only the individual risks and hazards; calculation of population risks and hazards is not required. However, if a permitting authority feels that site-specific conditions indicate calculation of population risks should be considered, U.S. EPA OSW recommends following the methodologies described in U.S. EPA NCEA document, *Methodology for Assessing Health Risks Associated with Multiple Exposure Pathways to Combustor Emissions* (In Press).

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 ESTIMATION OF INDIVIDUAL RISK AND HAZARD

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 by following the procedures described in Chapters 2 through 6 and Appendixes B and C will provide (1) quantitative and qualitative estimates of risk and hazard associated with exposure to COPCs, (2) estimates of health effects associated with exposure to lead, (3) evaluation of infant exposure to 2,3,7,8-TCDD TEQ present in breast milk, and (4) evaluation of acute exposure resulting from direct inhalation.

7.2 QUANTITATIVE ESTIMATION OF CANCER RISK

As described above, for carcinogenic chemicals, 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). These risks are calculated as follows:

$$Cancer\ Risk = LADD \cdot CSF \qquad \text{Equation 7-1}$$

where

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

Within a specific exposure pathway, receptors may be exposed to more than one COPC. The total risk associated with exposure to all COPCs through a single exposure pathway is estimated as follows (U.S. EPA 1989e):

$$Cancer\ Risk_T = \sum_i Cancer\ Risk_i \qquad \text{Equation 7-2}$$

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

At particular exposure scenario locations, receptors may be exposed through a number of exposure pathways (see Table 4-1). Risks from multiple exposure pathways should be summed for a given receptor specific to each recommended exposure scenario. That is, risks should be summed across the receptor-exposure pathway combinations, which are identified in Table 4-1. In the context of risk assessments which evaluate the emissions from hazardous waste combustion units, the risks from all RCRA regulated

combustion units that are permitted, have interim status, or expected to be constructed, should be summed for each receptor. For fugitive emissions from storage and handling of hazardous, the risk associated with fugitive emissions should be added to the risks from the combustion unit for each receptor at each exposure scenario location. For example, if a facility operates both an incinerator and a boiler that burn hazardous waste, then the risks from both types of units should be summed across all the units for each receptor. The total risk posed to a receptor is the sum of total risks from each individual exposure pathway expressed as follows:

$$Total\ \ Cancer\ \ Risk\ = \sum CancerRisk_T \qquad \text{Equation 7-3}$$

where

Total Cancer Risk	=	Total cancer risk from multiple exposure pathways
$Cancer\ Risk_T$	=	Total cancer risk for a specific exposure pathway

Equations used to calculate dose and risk levels are presented in Appendix C. Appendix A-3 presents oral and inhalation slope factors (*CSF*) for many potential COPCs. However, for each risk assessment, the IRIS and HEAST databases should be checked for updated values. If toxicity values for COPCs not identified in Appendix A-3 are included in the risk assessment, *CSF*s for these compounds can be obtained from the following sources, listed in the preferred order: (1) U.S. EPA's IRIS (U.S. EPA 1996a) and (2) U.S. EPA HEAST (U.S. EPA 1994b).

In the assessment of carcinogenic risk from COPCs, U.S. EPA-derived or reviewed health benchmarks (*CSFs*, *URFs*, and *Inhalation CSFs*) are recommended. However, for numerous compounds, a complete set of inhalation and oral EPA-derived health benchmarks are not available. In such cases, the health benchmarks presented in Appendix A-3 were calculated based on available U.S. EPA-derived benchmarks values.

If relevant information is not available from these sources, the applicant should contact the appropriate permitting authority, which may be able to assist in developing the necessary toxicity values. For example, Minimum Risk Values published by the Agency for Toxic Substances and Disease Registry (ASTDR) may be used at the discretion of the permitting authority.

7.3 QUANTITATIVE ESTIMATION OF POTENTIAL FOR NONCANCER EFFECTS

Standard risk assessment models assume that noncarcinogenic effects, exhibit a threshold; that is, there is a level of exposure below which no adverse effects will be observed (U.S. EPA 1989e). The potential for noncarcinogenic health effects resulting from exposure to a chemical is generally assessed by (1) comparing an exposure estimate (see Chapter 6) to an *RfD* for oral exposures, and (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 specific exposure duration. 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 1989e).

The exposure durations assumed for the exposure pathways identified in Table 4-1 range from subchronic to chronic in relative length. However, chronic *RfDs* and *RfCs* should be used to evaluate all exposure pathways. In the absence of a chronic *RfD*, a subchronic *RfD* with an Uncertainty Factor (3 to 10) can be considered. The comparisons of exposure estimates and COPC-specific air concentrations to *RfD* and *RfC* values, described above, are known as hazard quotients (*HQ*), which are calculated as follows:

$$HQ = \frac{ADD}{RfD} \quad \text{or} \quad HQ = \frac{C_a}{RfC} \qquad \text{Equation 7-4}$$

where

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

It should be noted that each program office within U.S. EPA determines the 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). Generally, the more that the *HQ* value exceeds 1, the greater is the level of concern. 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). It should also be noted that background exposures may be an important consideration in setting safe levels. This is because non-cancer effects are generally modeled as thresholds. In specific cases, a permitting authority may elect to adjust the *HQ* downward to account for any exposure that individuals may have from other sources.

As with carcinogenic chemicals in a specific exposure pathway, a receptor may be exposed to multiple chemicals associated with noncarcinogenic health effects. The total noncarcinogenic hazard for each exposure pathway is calculated by following the procedures outlined in U.S. EPA (1986e) and U.S. EPA (1989e). Specifically, the total noncarcinogenic hazard attributable to exposure to all COPCs through a single exposure pathway is known as a hazard index (*HI*). Consistent with the procedure for addressing carcinogenic risks, the noncarcinogenic hazards from all RCRA regulated combustion units that are permitted, have interim status, or are expected to be constructed, should be summed for each receptor. Also, noncarcinogenic hazard from fugitive emissions sources, should also be included in the calculation of the *HI* for each receptor. The *HI* is calculated as follows:

$$HI = \sum_i HQ_i \qquad \text{Equation 7-5}$$

where

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

This summation methodology assumes that the health effects, of the various COPCs to which a receptor is exposed, are additive. Specifically, this methodology is a simplification of the *HI* concept because it does not directly consider the portal of entry associated with each exposure pathway or the often unique toxic endpoints and toxicity mechanisms of the various COPCs.

As discussed in Section 7.2 for carcinogenic risks, a receptor may be exposed to COPCs associated with noncarcinogenic health effects through more than one exposure pathway. For the purposes of the risk assessment, it is reasonable to estimate a receptor's total hazard as the sum of the *HI*s for each of the

exposure pathways identified in Table 4-1. Specifically, a receptor's total hazard is the sum of hazards from each individual exposure pathway, expressed as follows:

$$Total\ HI = \sum HI \qquad \text{Equation 7-6}$$

where

Total HI	=	Total hazard from multiple exposure pathways
HI	=	Total hazard for a specific exposure pathway

Consistent with U.S. EPA guidance (1989e), all total *HI*s exceeding the target hazard level are further evaluated. The total *HI* for an exposure pathway can exceed the target hazard level as a result of the presence of either (1) one or more COPCs with an *HQ* exceeding the target hazard level, or (2) the summation of several COPC-specific *HQ*s that are each less than the target hazard level. In the former case, the presence of at least one COPC-specific hazard greater than the target hazard level is interpreted as indicating the potential for noncarcinogenic health effects. In the latter case, a detailed analysis is required to determine whether the potential for noncarcinogenic health effects is accurately estimated by the total *HI*, because the toxicological effects associated with exposure to multiple chemicals, often through different exposure pathways, may not be additive; therefore, the total *HI* may overestimate the potential for noncarcinogenic health effects. To address this issue, COPC-specific hazards are summed according to major health effects and target organs or systems (U.S. EPA 1989e). It is especially important to consider any differences related to exposure route; this process is referred to as the segregation of the *HI*. Technically, segregation of the HI based only on target organs or systems is a simplification of HI. Ideally, the HI should be segregated considering also the often unique mechanisms of toxicity of the various compounds to which receptors may be exposed. However, segregating the HI based on mechanisms of toxicity is beyond a screening level or initial risk evaluation approach.

The highest segregated *HI* resulting from this process is considered. If the segregated *HI* exceeds the target hazard level, there is a potential for noncarcinogenic health effects. However, if the segregated *HI* is less than the target hazard level, the total *HI* of all COPC-specific results likely is too conservative, and noncarcinogenic health effects are not likely to result from exposure to COPCs.