

5.0 Toxicity assessment

The purpose of the toxicity assessment is to identify the types of health effects a COPC may potentially cause, and to define the relationship between the dose of a compound and the likelihood or magnitude of a health effect (response). Health effects are characterized by U.S. EPA as carcinogenic or noncarcinogenic. U.S. EPA defines dose-response relationships for oral and inhalation exposures. Combining the results of the dose-response assessment with information on the magnitude of potential human exposure (discussed in Section 6.0, Exposure Assessment) provides an estimate, usually very conservative, of potential health risk.

Both potentially carcinogenic and noncarcinogenic health effects were evaluated in the cumulative health risk assessment. The toxicity of each COPC is based on criteria developed by the U.S. EPA. Such criteria are referred to as dose-response values, and are derived for both inhalation and oral routes of exposure. The dose-response values derived by U.S. EPA for evaluation of potential carcinogenic health effects resulting from long-term exposure to COPC are called cancer slope factors (CSFs), and are expressed in units of $(\text{mg}/\text{kg}\cdot\text{day})^{-1}$. The dose-response values derived for evaluation of potential noncarcinogenic health effects resulting from long-term exposure to COPC are called reference doses (RfDs) and are expressed in units of $\text{mg}/\text{kg}\cdot\text{day}$. Sources of published dose-response values used in this cumulative health risk assessment include U.S. EPA's Integrated Risk Information System (IRIS) (U.S. EPA, 2004a), the National Center for Environmental Assessment (NCEA) and the Health Effects Assessment Summary Tables (U.S. EPA, 1997). Dose-response values developed by NCEA were generally obtained from U.S. EPA Region 9 Preliminary Remediation Goals (PRGs; U.S. EPA, 2004b). The dose-response information for COPCs evaluated in this health risk assessment are presented in Appendix C. Consistent with the HHRAP (U.S. EPA, 1998), if an oral dose-response value was available for a COPC but an inhalation dose-response value was not available, route-to-route extrapolation was assumed and the oral value was adopted for use in evaluating the inhalation exposure pathway.

5.1 Compound-specific dose-response values

Several compounds including PAHs, PCBs, dioxins, lead and mercury, do not have a single dose-response value available or require an additional modeling effort to evaluate their potential toxicity. The approaches taken in evaluating these compounds in the cumulative health risk assessment are described below.

5.1.1 Toxicity assessment for polynuclear aromatic hydrocarbons

PAHs are a class of over 100 related chemicals that are formed during the incomplete burning of organic matter (including, but not limited to, coal, oil, gas, garbage, tobacco or charbroiled meat). While 17 PAHs are routinely analyzed, U.S. EPA-derived dose-response values are available for only seven of them. These dose-response values include a CSF that has been developed only for benzo(a)pyrene, and RfDs that are available for six other PAHs. The following approach was taken in evaluating potential carcinogenic risk and noncarcinogenic hazard of PAHs in the cumulative health risk assessment.

A U.S. EPA-developed comparative potency approach (U.S. EPA, 1993) was used to evaluate potential carcinogenic risk associated with facility-related PAH emissions. U.S. EPA has classified seven PAHs as "probable human carcinogens (Class B2)". They include: benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, indeno(1,2,3-cd)pyrene, dibenz(a,h)anthracene, and chrysene. Benzo(a)pyrene (B(a)P) is one of the most potent of the PAHs that have been shown to be carcinogenic in animal experiments. A review of the scientific literature indicates that most of the other PAH are considerably less potent than B(a)P. U.S. EPA has developed an approach in which experimental studies on B(a)P and other PAH are used to derive toxicity equivalent factors (listed in Table 5-1) (U.S. EPA, 1993). In this health risk assessment the emission rate for each of the seven potentially carcinogenic PAH was multiplied by the corresponding toxicity equivalency factor (TEF), resulting in a B(a)P toxic equivalent. The TEF adjusted

emission rates for each carcinogenic PAH were summed to result in a total B(a)P-TEF emission rate. The CSFs for B(a)P were then applied directly to the estimate of potential human exposure to B(a)P-TEF in generating risk estimates. In modeling fate and transport of carcinogenic PAH in the environment, the chemical specific assumptions for B(a)P were used to predict fate and transport of total B(a)P-TEF.

In addition to potential carcinogenic effects, all PAH (total PAH) may also have associated noncarcinogenic effects. These effects were evaluated using the RfD for pyrene. This is a conservative approach, because pyrene has one of the lowest RfDs of all the noncarcinogenic PAHs, and its use leads to a high estimate of noncarcinogenic hazard for every compound represented. Fate and transport of total PAH in the environment was also evaluated using chemical-specific assumptions for pyrene.

5.1.2 Toxicity assessment for polychlorinated biphenyls

U.S. EPA has developed a range of CSFs to evaluate potential exposures to PCBs (U.S. EPA, 2001). The upper-bound range is 0.07 to 2 (mg/kg-day)⁻¹ and the central range is 0.04 to 1 (mg/kg-day)⁻¹. The specific CSF used depends on the type of environmental exposure and amount of chlorination on the PCB compounds. The highest CSF of 2 (mg/kg-day)⁻¹ was used to evaluate potential carcinogenic health risks associated with PCBs through all of the potential exposure pathways evaluated in the cumulative health risk assessment. This is a conservative approach, because the CSF used (2 (mg/kg-day)⁻¹) is the highest available for PCBs and its use leads to a high estimate of potential carcinogenic risk for every PCB compound represented and every exposure pathway evaluated.

In addition to carcinogenic effects, the potential noncarcinogenic effects of PCBs were also evaluated. U.S. EPA has developed oral RfDs of 7×10^{-5} mg/kg-day and 2×10^{-5} mg/kg-day for two commercial PCB mixtures, Aroclor-1016 and Aroclor-1254 respectively (U.S. EPA, 2004a). The RfD for Aroclor-1254 was used to evaluate the potential noncarcinogenic effects associated with potential exposure to facility-related PCB emissions.

Chemical-specific parameters for Aroclor-1254 were also used in predicting fate and transport of total PCBs in the environment.

5.1.3 Toxicity assessment for dioxins/furans

The concentrations of emitted dibenzodioxins and dibenzofurans are expressed as 2,3,7,8-TCDD toxic equivalents (TCDD-TE). U.S. EPA has developed toxicity equivalence factors (TEFs) that relate the toxicity of each dibenzodioxin and dibenzofuran congener to that of 2,3,7,8-TCDD, which is the most toxic congener of the set (U.S. EPA, 1989). The TEFs are listed in Table 5-2. In this health risk assessment the emission rate for each of the 17 2,3,7,8-dibenzodioxin and 2,3,7,8-dibenzofuran congeners was multiplied by the corresponding TEF, resulting in a 2,3,7,8-TCDD toxic equivalent (TEQ) emission rate for that congener. The TEF adjusted emission rates for each dibenzodioxin and dibenzofuran congener were summed to result in a total TCDD-TEQ emission rate. The CSF for 2,3,7,8-TCDD was used to evaluate potential carcinogenic health effects associated with the total TCDD-TEQ emissions from the RRF. U.S. EPA has not developed noncarcinogenic toxicity values for dioxins and/or furans.

5.1.4 Toxicity assessment for lead

U.S. EPA has not derived RfDs for lead due to uncertainties about the health effects and dose-response associated with exposures to lead. Based on findings that neurobehavioral effects in young children occur at exposure levels below those that have caused cancer in laboratory animals, an Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children has been developed by USEPA (U.S. EPA, 1994b). Young children represent the most sensitive receptor for potential lead exposures. In the latest U.S. EPA guidance for combustion risk assessment (U.S. EPA, 1998 and 1999), the IEUBK model is recommended for use in evaluating potential risk to human health associated with combustion facility emissions of lead.

Several recent combustor facility risk studies have yielded extremely low incremental concentrations of lead in the modeled environmental media. Those concentrations are often so low that their media concentrations can not be input into the publicly available version of the IEUBK model (due to threshold format restrictions). As a conservative approach, the U.S. EPA benchmark (U.S. EPA, 1994c) of less than 5 percent of children having blood lead concentrations exceeding 10 ug/dL has been used by the agency to calculate proportional concentrations that were used in the MRA.

Based on the IEUBK model, the target soil lead concentration is 400 mg/kg. The U.S. EPA incorporates a margin of safety by assuming that only 25% of the allowable threshold lead level would be assigned to a facility. That leads to a target soil concentration of 100 mg/kg. Similarly, U.S. EPA has derived a target ambient air concentration for lead of 0.2 ug/m³. This value assumes that the target equals 25% of the quarterly average air concentration of 1.5 ug/m³ specified by the National Ambient Air Quality Standards (NAAQS) adjusted on an annual basis to 0.9 ug/m³.

5.1.5 Toxicity assessment for mercury

Mercury may be present in the environment in different forms that may have different potential to cause impacts to human health. For example, different RfDs are available for divalent mercury (mercuric chloride) and methylmercury (3×10^{-4} mg/kg-day and 1×10^{-4} mg/kg-day, respectively). The modeling of mercury in the environment has changed significantly in recent years and is discussed in Appendix D. The most recent U.S. EPA combustion risk assessment guidance (U.S. EPA, 1998 and 1999) will be followed for the evaluation of mercury. However, based on recent observations of mass-balance exceedances in the fate and transport modeling of mercury (Smith and Garcia, 2001) modifications were made to U.S. EPA default parameters to prevent mass-balance problems. These modifications are also discussed in Appendix D. U.S. EPA combustion risk assessment guidance (U.S. EPA, 1998 and 1999) recommends that a small fraction of mercury in soils, plants, and water be assumed to be methylmercury and the rest be treated as mercuric chloride. For the fish pathway, U.S. EPA recommends that all mercury in fish be evaluated as methylmercury.

5.2 Health benchmarks for short term exposure

As currently required by U.S. EPA, risks due to short-term inhalation exposure (such as respiratory or irritant health effects), in addition to the more commonly evaluated chronic risks to human health discussed above, were evaluated in this health risk assessment. A screening level evaluation of short-term health effects was conducted by comparing predicted maximal short-term air concentrations against applicable guidelines. Although there are many uncertainties associated with the underlying data, these guidelines still represent health benchmark levels that are appropriate for making risk management decisions. The short-term ambient air concentration guidelines utilized in this health risk assessment, summarized by hierarchical preference, include: 1) level 1 acute inhalation exposure guidelines (AEGL-1) (U.S. EPA, 2004c; NAC, 1997); 2) level 1 emergency response planning guidelines (ERPG-1) (AIHA, 1996; DOE, 2004); 3) level 1 acute reference exposure levels (AREL-1) (CalEPA, 2000); 4) level 1 temporary emergency exposure limits (TEEL-1) (DOE, 2004).

Table 5-1
Benzo(a)pyrene Toxicity Equivalency Factors (BAP-TEFs)

Compound	Published EPA TEF ^a
BENZO(A)PYRENE	1.0
BENZO(A)ANTHRACENE	0.1
BENZO(B)FLUORANTHENE	0.1
BENZO(K)FLUORANTHENE	0.01
CHRYSENE	0.001
DIBENZ(A,H)ANTHRACENE	1.0
INDENO(1,2,3-CD)PYRENE	0.1
Notes: ^a - From Table 8, U.S. EPA, Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons EPA/600/R93/089, July, 1993.	

Table 5-2
2,3,7,8-TCDD Toxic Equivalents (TCDD-TE)

Compound	2,3,7,8-TCDD Toxic Equivalent
1,2,3,4,6,7,8-HpCDD	0.010
1,2,3,4,6,7,8-HpCDF	0.010
1,2,3,4,7,8-HxCDD	0.100
1,2,3,4,7,8-HxCDF	0.100
1,2,3,4,7,8,9-HpCDF	0.010
1,2,3,6,7,8-HxCDD	0.100
1,2,3,6,7,8-HxCDF	0.100
1,2,3,7,8-PeCDD	0.500
1,2,3,7,8-PeCDF	0.050
1,2,3,7,8,9-HxCDD	0.100
1,2,3,7,8,9-HxCDF	0.100
2,3,4,6,7,8-HxCDF	0.100
2,3,4,7,8-PeCDF	0.500
2,3,7,8-TCDD	1.000
2,3,7,8-TCDF	0.100
OCDD	0.001
OCDF	0.001
Notes: From: U.S. EPA, 1989. Interim Procedures for Estimating Risk Associated with Mixtures of Chlorinated Dibenzo-p-dioxins and Dibenzofurans. EPA/652/3-89/016.	