

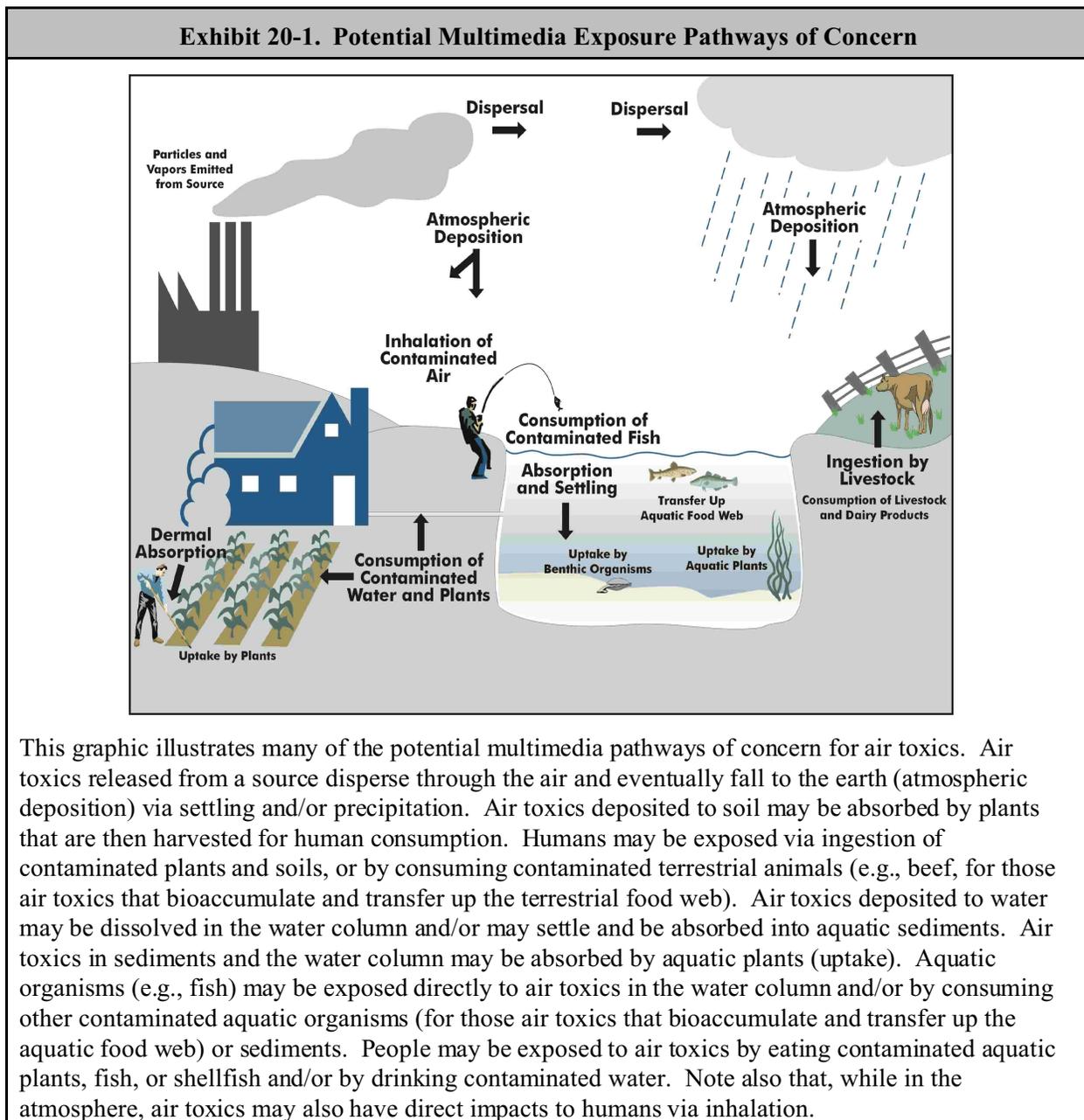
Chapter 20 Exposure Metrics for Multimedia Assessment

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20.1 Introduction

This chapter concludes the exposure assessment component of the multipathway risk assessment by describing how to develop estimates of intake (i.e., the metric of exposure) for the ingestion pathways selected for analysis. Estimates of chemical intake via the inhalation pathway were presented in Chapter 11. Exhibits 14-2 and 20-1 provide an overview of the potential multimedia exposure pathways by which air toxics that persist and potentially bioaccumulate may reach ecological and human receptors, respectively. Determination of chemical intake via the ingestion exposure route combines the estimates of chemical of potential concern (COPC) levels in food items and drinking water (discussed in Chapter 7) with estimates of consumption rates (food, water), exposure frequency and duration, averaging time, and body weight to derive estimates of the **chemical intake rate** (expressed generally as mg/kg-day).⁽¹⁾



This graphic illustrates many of the potential multimedia pathways of concern for air toxics. Air toxics released from a source disperse through the air and eventually fall to the earth (atmospheric deposition) via settling and/or precipitation. Air toxics deposited to soil may be absorbed by plants that are then harvested for human consumption. Humans may be exposed via ingestion of contaminated plants and soils, or by consuming contaminated terrestrial animals (e.g., beef, for those air toxics that bioaccumulate and transfer up the terrestrial food web). Air toxics deposited to water may be dissolved in the water column and/or may settle and be absorbed into aquatic sediments. Air toxics in sediments and the water column may be absorbed by aquatic plants (uptake). Aquatic organisms (e.g., fish) may be exposed directly to air toxics in the water column and/or by consuming other contaminated aquatic organisms (for those air toxics that bioaccumulate and transfer up the aquatic food web) or sediments. People may be exposed to air toxics by eating contaminated aquatic plants, fish, or shellfish and/or by drinking contaminated water. Note also that, while in the atmosphere, air toxics may also have direct impacts to humans via inhalation.

Chapter 7 described two general approaches for deriving the exposure concentration (EC) for an inhalation risk assessment: (1) use of ambient air concentrations as a surrogate for the EC, and (2) exposure modeling that combines estimates of ambient air concentrations with information about the population of interest, including the types of people present (e.g., ethnicity, age, sex), time spent in different microenvironments, and microenvironment concentrations. **The first approach (i.e., use of ambient concentrations in abiotic media such as soil, water, or sediments) generally is not used for multipathway air toxics risk assessments. Instead, a multipathway exposure assessment must involve some type of exposure modeling** (e.g., at a minimum simple scenarios to characterize persons who are exposed and the amount and duration of their contact with the abiotic and biotic media).

Note that EPA has derived some human health screening-level concentration benchmarks for surface water and soil (i.e., the Office of Water's Ambient Water Quality Criteria for the Protection of Human Health,⁽²⁾ and the Superfund Program's soil screening levels⁽³⁾). However, these human health benchmarks are based on specific scenarios (e.g., how much water a person drinks each day, how much they weigh) that were selected to meet different programmatic goals and statutory requirements. Therefore, the scenarios on which these benchmarks are based may not be appropriate for a specific air toxics risk assessment.

The way a chemical enters the body and eventually reaches the target organ is a complex process (see box below). For most chemicals, however, it is not necessary to quantify anything beyond the chemical **intake rate**, because the dose-response value (e.g., Reference Dose [RfD] or Cancer Slope Factor [CSF]) is also based only on the amount of chemical ingested and not the amount of chemical that has been absorbed into the bloodstream.

Exposure and Intake via Ingestion

The process of a chemical entering the body can be described in two steps: **exposure** (contact), followed by **entry** (crossing the boundary). **Intake** involves physically moving the chemical in question through an opening in the outer boundary (usually the mouth), typically via eating or drinking. Normally the chemical is contained in a medium that comes into contact with the body, such as food or water, and the concentration of the chemical at this point of contact is called the **exposure concentration**. The estimate of how much of the chemical enters into the body is based on how much of the carrier medium enters the body. The **chemical intake rate** is the amount of chemical crossing the outer boundary per unit time, and is the product of the exposure concentration times the ingestion rate. **Ingestion rate** is the amount of the carrier medium crossing the boundary per unit time, such as the number of kilograms of food ingested/day or liters of water consumed/day. Ingestion rates typically are not constant over time (they can vary over time and among individuals) and are usually given (for deterministic analyses) as an average intake rate over some period of time. In addition, the intake rates are usually normalized to body weight. Thus, a common intake rate would take the form of milligrams of pollutant ingested per kilogram of body weight per day (or mg/kg-d). A different ingestion rate would be developed for each type of person in the population under study. For example, one intake rate could be developed to represent the average adult (male and female) while a separate intake rate could be developed to represent children between the ages of birth to four years old.

The remainder of this chapter focuses on how to quantify ingestion exposure (intake) for multipathway air toxics risk assessments. The corresponding chapter for inhalation analyses

(Chapter 11) discusses how to evaluate uncertainty in the exposure assessment and how to present the exposure assessment results; this applies to all exposure evaluations (i.e., inhalation and ingestion).

20.2 Generic Equation for Dietary Intake

Equation 20-1 is the generic equation used to calculate dietary chemical intake:⁽⁴⁾

$$I = \frac{EC \times CR}{BW} \times \frac{EF \times ED}{AT} \quad \text{(Equation 20-1)}$$

where

I = Chemical intake rate, or the amount of pollutant ingested per unit time per body weight (mass), expressed in units of mg/kg-day. For evaluating exposure to noncarcinogens, the intake is referred to as Average Daily Dose (*ADD*); for evaluating exposure to carcinogenic compounds, the intake is referred to as Lifetime Average Daily Dose (*LADD*).

Chemical-related variable:

EC = Exposure concentration of the chemical in the medium of concern for the time period being analyzed, expressed in units of mg/kg for soil and food or mg/L for surface water or beverages (including milk).

Variables that describe the exposed population (also termed “intake variables”):

CR = Consumption rate, the amount of contaminated medium consumed per unit of time or event (e.g., kg/day for soil and L/day for water).

EF = Exposure frequency (number of days exposed per year).

ED = Exposure duration (number of years exposed).

BW = Average body weight of the receptor over the exposure period (kg).

Assessment-determined variable:

AT = Averaging time, the period over which exposure is averaged (days). For carcinogens, the averaging time is 25,550 days, based on an assumed lifetime exposure of 70 years; for noncarcinogens, averaging time equals *ED* (years) multiplied by 365 days per year.

The values of some exposure factors depend on site conditions as well as the characteristics of the potentially exposed population (e.g., child vs. adult). Because of differences in physiology and behavior, exposures among children are expected to be different than exposures among adults. For example, body weight and consumption rate differ for children and adults. For the evaluation of non-carcinogenic effects, intakes for children generally are estimated separately (often for ages 0-6) than for adults (often from ages 6-beyond). For the evaluation of carcinogenic effects, intake estimates are averaged over the assumed lifetime (70 years).

20.3 Estimating Exposure Concentrations

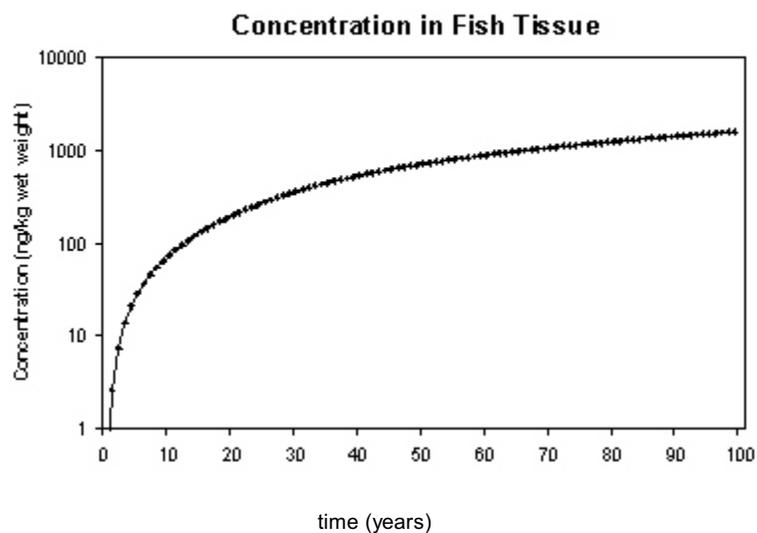
The exposure concentration for a chemical is calculated separately for each food item and environmental medium of concern. The value of these variables may be determined by modeling (Chapter 18), monitoring (Chapter 19), or a combination of both. The specific algorithms for determining these concentrations will depend on the specific models and/or sampling and analysis techniques used. For example, EPA has developed methodologies for estimating EC values in soil, water, sediment, and various food items for releases from hazardous waste combustion facilities (see Appendix L).⁽⁵⁾

For ingestion pathways, the specific media concentration values obtained from a multimedia modeling simulation for use in deriving exposure concentrations depends on several important decisions made during problem formulation, including:

- Choice of modeling duration for a model run;
- Choice of the year or years of the model run on which to base the EC; and
- Choice of a specific ED.

Exhibit 20-2 presents several different examples relevant to different purposes/objectives for an assessment.

Exhibit 20-2. Example Decisions in Assessing Exposures Resulting From Distribution of Air Toxics into Other Media



In this hypothetical example, a modeling analysis was used to predict the concentrations of a persistent bioaccumulative hazardous air pollutant (PB-HAP) in fish tissue during a 100-year emissions scenario (annual average was estimated each year and is plotted here using a logarithmic scale). As discussed below, the exposure scenario assessed will reflect several key choices including:

- (1) choice of modeling duration for model run;
- (2) choice of year or years of model run on which to base EC (i.e., the model outputs); and
- (3) choice of specific ED.

Exhibit 20-2 (continued)

The modeling duration is a separate decision from the ED and is not related to the average human lifespan.

Note that in this example, the analyst assumed that the starting concentration was zero (i.e., the tissue concentrations reflect only the sources being modeled). Some multimedia models (e.g., TRIM.FaTE) can start with an initial concentration.

Modeling Duration. The analyst can choose to run a multimedia model for any period of time. Duration will usually be chosen to reflect the expected duration of emissions from the source(s) being evaluated or, perhaps, that duration expected in order to reach steady-state conditions. A common duration is 30 or 40 years (e.g., the expected lifespan of many facilities or processes). For this example, a 100-year duration was selected.

Selection of Model Outputs. Usually the modeling duration will have been chosen with consideration of the model outputs on which the exposure scenario is to be based and the exposure duration. Some common examples follow:

- **Year of maximum concentration.** Screening-level analyses often use the maximum concentration reached during the modeling period which, for a constant emissions scenario, will usually be the final year of the modeling simulation. For this example (see figure), it would be the 100th year (at such time as the fish concentration was approximately 2,000 ng/kg).
 - **Exposure Duration.** With use of the maximum model result, the analysis presumes no change in fish concentration over the exposure duration (i.e., in this example EC = 2,000 ng/kg throughout the exposure period).
- **Initial years of simulation.** In this case the exposure being assessed is that beginning with initiation of emissions and extending through the duration selected for assessment.
 - **30-year Exposure Duration.** In this case, the analyst is basing the exposure duration near the 95 percentile of how long people live in the same home.⁽⁶⁾ If the analyst chose to examine changing concentrations over time, the ECs would vary, reflecting the concentration outputs from the first 30 years of the modeling duration.
 - **70-year Exposure Duration.** In this case, the analyst is using a lifetime exposure assumption. The exposure scenario then may be based on the model outputs from the first 70 years of the modeling duration.
- **Last years of simulation.** In this case, the exposure being assessed is that which occurs during the ending years of the simulation, with the number of years involved equal to the exposure duration selected for assessment.
 - **30 -year Exposure Duration.** For this ED, the ECs would vary reflecting the predicted concentrations from the last 30 years of the model simulation.
 - **70-year Exposure Duration.** In this case, the analyst is using a lifetime exposure assumption. The exposure scenario may then employ varying ECs reflecting the predicted concentrations from the last 70 years of the model simulation.

Note: When using varying exposure concentrations for the exposure scenario, other variables included in the calculation of ingestion exposure estimates (pollutant intake, mg/kg-day) for the population(s) of interest may also vary. For example, if the exposure scenario includes exposure for cohorts aging from birth - 30 years, other exposure factors (e.g., body weight, consumption rate) will also vary over time.

20.4 Calculating Intake Variable Values

Each intake variable in Equation 20-1 (e.g., consumption rate, body weight) has a range of potential values. Intake variable values for a given pathway may be selected so that the combination of all intake variables results in an estimate for an individual at the “high-end” of potential exposure levels. Alternatively, the intake variables may be selected to represent a “central tendency” individual expected to receive an average exposure. In doing this, the assessor needs to avoid combinations of parameter values that are inconsistent (e.g., low body weight used in combination with high dietary intake rates), and must keep in mind the ultimate objective of being within the distribution of actual expected exposures and doses, and not beyond it. Commonly, both the central tendency and high end intakes are quantified. In some cases, the distribution of intake rates in the population may be described using probabilistic risk assessment methods (discussed in Part VI).

EPA recommends values for intake variables for the U.S. population in the *Exposure Factors Handbook*,⁽⁷⁾ the *Child-Specific Exposure Factors Handbook*,⁽⁸⁾ and the *Consolidated Human Activity Database*.^{(9)(a)} EPA also recently published draft guidance on selecting the appropriate age groups for assessing childhood exposures.⁽¹⁰⁾ Note, however, that there are likely to be differences between recommended default, and regional and site-specific, exposure parameter values. This may be especially true for consumption rate (see below).

For **central tendency** estimates, risk assessors commonly set all of the exposure factors in the Equation 20-1 at central tendency values. If only limited information on the distribution of the exposure or dose factors is available, risk assessors commonly approach the **high-end** estimates by identifying the most sensitive variables and using high-end values for a subset of these variables, leaving others at their central values. As mentioned earlier, the assessor needs to avoid combinations of parameter values that are inconsistent (e.g., low body weight with high dietary intake rates) and must keep in mind the ultimate objective of being within the distribution of actual expected exposures and doses.

Maximizing all variables will in virtually all cases result in an estimate that is above the actual values seen in the population. When the principal parameters of the dose equation (e.g., concentration [appropriately integrated over time], intake rate, and duration) are broken out into sub-components, it may be necessary to use maximum values for more than two of these sub-component parameters, depending on a sensitivity analysis.

For **probabilistic analyses**, values for exposure factors are commonly allowed to vary according to specific assumed distributions of potential values.

Note that the high-end intake estimate is a plausible estimate of intake for those persons at the upper end of the exposure distribution. This descriptor is intended to estimate the exposures that are expected to occur in small but definable high-end segments of the subject population

^aNCEA recently published a new compilation of consumption data from the 1994-1996 CSFII. This data updates CSFII data in the 1997 Exposure Factors Handbook.
See: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=56610>.

(but not higher than the highest person in the population), but may not be appropriate for estimating exposure for the population as a whole.⁽¹⁾

20.4.1 Consumption Rate

Consumption rate is the amount of contaminated food or medium consumed per event or unit of time (e.g., amount of fish consumed per meal or per day). The consumption rate is multiplied by a fraction of the total dietary intake for this type of food or medium, representing the amount consumed from the study area. The specific fraction applied depends on the analysis.

- For screening-level analyses, it is common to assume that the person obtains 100 percent of the food type from the study area (e.g., farm, water body) being evaluated. This assumption also might be used for a subsistence-type receptor (e.g., a local fish consumer who only eats fish caught from the study area).
- For higher tiers of analyses, it is common to assume that the person obtains some of the food type from the study area (i.e., the contaminated fraction) and some of the food type from other sources (e.g., at the grocery store). This latter fraction generally is assumed to be uncontaminated by the source(s) under assessment. Thus, if a person is assumed to eat ½ pound of fish per day, but only 25 percent is caught within the study area, the assumed consumption of contaminated fish would be 1/8 pound per day.

The following pathway-specific considerations are important for estimating consumption rate.

- **Food Ingestion.** Plants and animals may accumulate COPCs that were deposited onto soil or water. Humans may be exposed to these compounds via the food chain when they consume these plants (and animals that consume these plants) as a food source. Human intake of COPCs is quantified on the basis of the concentration of COPC in the food (Section 20.3) and:
 - The types of foods consumed, which vary with age (e.g., children and adults often eat different things), geographical region, and sociocultural factors (e.g., ethnicity, cultural factors);
 - The amount of food consumed per day, which can vary with age, sex, and geographic region, and also within these categories;
 - The fraction of the diet contaminated by COPCs (which can vary by food type); and
 - The effect of food preparation techniques on concentrations of COPCs in the food itself.
- **Soil Ingestion.** Children and adults may receive direct exposure to COPCs in soil when they consume soil that has adhered to their hands (called incidental soil ingestion). Factors that influence exposure by soil ingestion include concentration of the COPC in soil, the rate of soil ingestion during the time of exposure, and the length of time spent in the vicinity of contaminated soil. Soil ingestion rates in children are based on studies that measure the quantities of nonabsorbable tracer minerals in the feces of young children. Ingestion rates for adults are based on assumptions about exposed surface area and frequency of hand-to-mouth transfer. Indoor dust and outdoor soil may both contribute to the total daily incidental ingestion of soil (indoor dust is partially made up of outdoor soil that has been tracked inside).

In addition, some young children – referred to as “pica” children – may intentionally eat soil. The typical medical and scientific use of the term “pica” refers to the ingestion of nonfood items, such as soil, chalk, and crayons.⁽¹⁰⁾ Such behavior is considered a temporary part of a child’s development. For risk assessment purposes, pica is typically defined as “an abnormally high soil ingestion rate” and is believed to be uncommon in the general population. If available information indicates that there are children exhibiting pica behavior in the assessment area, it may be appropriate to include these children as a separate group in the exposure assessment. EPA’s *Exposure Factors Handbook* provides quantitative data on soil ingestion rates related to pica.⁽¹¹⁾

Inhalation of soil resulting from dust resuspension by wind erosion generally is not a significant pathway of concern for air toxics.⁽⁵⁾ However, it may be an issue for locations at which there is little vegetative cover. Methodologies have been developed to assess the exposure to pollutants resuspended by wind erosion for landfills and Superfund sites.⁽¹²⁾ The exposure estimate from resuspended soil would depend on moisture content of the soil, fraction of vegetation cover, wind velocity, soil particle size, COPC concentration in the soil, and size of the contaminated area.

Depth of Contaminated Soils: A Key Variable

When exposures to COPCs in soils are modeled for human health risk assessment, an important factor affecting the exposure estimate is the depth of contaminated soils used to calculate soil concentrations. The same deposition rate will result in different soil concentrations depending on how deeply the COPCs are assumed to mix or migrate into the soil. Mixing depth also may affect exposure estimates via specific pathways. For example, in calculations of exposures resulting from uptake through plant roots, the average concentration of COPCs over the depth of the plant root determines plant uptake. However, calculations that assess soil ingestion through hand-to-mouth activity commonly focus on only the top few centimeters of soil.

COPCs deposited onto undisturbed soils generally are assumed to remain in the shallow, upper soil layer. However, COPCs deposited onto soil surfaces may be moved into lower soil profiles by tilling, whether manually in a garden or mechanically in a large field. Other factors such as soil disturbance by domestic animals (e.g., cattle in an enclosure) also may need to be considered. Some chemicals are also highly soluble in water and may be carried deeper into soil along with infiltrating rainwater. The key questions to ask therefore include:

- Are soils tilled, or is it reasonable to assume they are undisturbed?
- If soils are tilled, what mixing depth is reasonable to assume?
- What other factors might affect how deeply COPCs will be moved into soils?

EPA guidance and other references⁽⁵⁾⁽¹³⁾ provide a more detailed discussion of depth of contaminated soils, along with recommended values.

- **Ingestion of Drinking Water.** In air toxics assessments, assessors only evaluate the ingestion of drinking water when an affected surface water body or collected precipitation (e.g., a cistern) is used as a drinking water source.^(b) Important factors affecting the concentration of COPCs in a surface water body include the location of the surface water body or precipitation collection apparatus relative to emissions sources; concentrations of COPCs in and characteristics of the soils (which affects runoff and leachate concentrations); and the size and location of the watershed. For drinking water, the exposure estimate is affected by:
 - The concentration of the COPC in the water;
 - The daily amount of drinkable water ingested; and
 - The fraction of time that the individual spends in the area serviced by that water supply system. (Note that for screening level analyses, 100% of drinking water may be presumed to come from the contaminated source.)

Note that in estimated exposures associated with drinking water supplies, risk assessors commonly assume that the drinking water undergoes at least a minimum level of treatment to remove solids (i.e., particles in the water which are PB-HAPs or onto which PB-HAPs may be absorbed). Therefore, the risk assessment commonly focuses on the dissolved concentrations of PB-HAPs in drinking water sources.

Groundwater as a Source of Drinking Water

If site-specific circumstances suggest that groundwater may represent a potential concern (e.g., the presence of extremely shallow aquifers used for drinking water purposes or a karst environment in which the local surface water significantly affects the quality of ground water used as a drinking water source), the TRIM.FaTE library includes a groundwater compartment that can be used to assess the groundwater pathway. EPA's *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities*⁽¹⁴⁾ and *Draft Technical Background Document for Soil Screening Guidance*⁽¹⁵⁾ discuss the methods for evaluating the groundwater pathway.

- **Ingestion of Fish.** Factors that affect human exposure by ingestion of fish from a surface water body include:
 - Sediment and water COPC concentrations;
 - The types of fish and shellfish consumed;
 - The portion of fish eaten (e.g., fillet only, fillet plus skin, whole body);
 - The effect of food preparation techniques on concentrations of COPCs in the fish;
 - Ingestion rates for the various fish and shellfish groups; and
 - The fraction of dietary fish caught in the surface water body or bodies being evaluated. (Note that for screening level analyses, 100 percent of fish/shellfish is presumed to come from the contaminated water body.)

^bNote that ingestion of contaminated groundwater generally is not a significant pathway of concern for air toxics risk assessments because most air toxics that persist and may bioaccumulate tend to get bound up in soil and, therefore, tend not to move readily into groundwater. However, if the groundwater pathway were a concern for a specific study, it would be evaluated in generally the same way as the ingestion of surface water pathway (i.e., as a drinking water source; however, depending on the circumstances, groundwater may or may not be treated to remove particles prior to consumption).

The types of fish consumed will affect exposure because different types of fish and shellfish accumulate COPCs at different rates. For example, fatty fish tend to accumulate lipophilic organic compounds more readily than lean fish. The amount of fish consumed also affects exposure because people who eat large amounts of fish will tend to have higher exposures. Fish consumption rates and the parts of the fish that are consumed can vary greatly, depending on geographic region and social or cultural factors. Also, because all of a person's dietary fish may not originate from the surface water body near the source of the PB-HAP, the fraction of locally caught fish is also a variable for exposure.

20.4.2 Exposure Frequency

The specific exposure frequency will depend on how the exposure analysis is set up. For example, a scenario-based analysis would specify one or more exposure frequencies for each defined scenario. A typical screening-level exposure frequency is 350 days per year; this number is based on the assumption that all people spend a minimum of two weeks at a location other than the exposure scenario location selected for analysis (e.g., on vacation).⁽¹⁾⁽⁵⁾ However, many activities vary on a weekly and/or seasonal basis. For example, recreational fishing is more likely to occur on weekends than on weekdays, and most areas in the U.S. have limited fishing and hunting seasons.

20.4.3 Exposure Duration

Exposure duration is the length of time over which exposure occurs (e.g., a lifetime or a particular residence time). As noted in Section 20.3 above, choice of ED will depend on many factors, including the purpose of the assessment or risk management decision, the tier of analysis, and the particular effect(s) of concern. There are no universally established ED values for risk assessments because different EDs may be appropriate in different situations. Some commonly used EDs include:

- Lifetime (70 years) – generally used for screening-level analyses;
- High-end number of years a person resides in a single location (about 30 years);
- Median number of years a person resides in a single location (about 9-10 years); and
- Seven years (ten percent of an assumed lifetime) – sometimes used for noncancer effects.

Although a source may remain in the same location for more than 70 years, and a person may have a lifetime of exposure to emissions from that source, U.S. Bureau of the Census data on population mobility indicate that many Americans do not always remain in the same area for their assumed 70-year lifetime.⁽¹⁶⁾ An estimate of the number of years that a person is likely to spend in one area can be derived from information about mobility rate and median time in a residence.

Analysts may use long EDs when conducting simple screening analyses performed to determine if more complex analyses are necessary. The rationale for use of such EDs is that if risks are not of concern when the exposure duration is long, then they would not be of concern given other, shorter, exposure durations. (Typically analysts also make other conservative or “health-protective” assumptions when conducting this type of screening analysis.) Analysts may use specific EDs particular to the legal framework for the assessment. For example, the residual risk section of the Clean Air Act (CAA) references an Agency rulemaking for which one prominent

risk metric considered a 70-year exposure duration (see CAA section 112(f)(2) and 54 *Federal Register* 38044).

The type of risk metric being derived also influences the consideration of exposure duration. For example, when the analyst wants to describe central tendency risk based on a deterministic analysis, s/he typically will use mean or median exposure assumptions to calculate risk.^(c) Similarly, when the analyst wants to describe high-end risk based on a deterministic analysis, s/he may use high-end exposure assumptions or a combination of central tendency and high-end exposure assumptions that provide a reasonable estimate of the individual risk for those persons at the upper end of the risk distribution. As explained in EPA's *Policy on Risk Characterization*:⁽¹⁷⁾ "Conceptually, high-end exposure means exposure above about the 90th percentile of the population distribution, but not higher than the individual in the population who has the highest exposure."^(d) When the analyst wants to conduct a probabilistic analysis of risk, s/he typically will use or develop a distribution of exposure durations from the available data (e.g., see EPA's *Exposure Factors Handbook, Part III*; Tables 15-164, 15-166, 15-167, and 15-168).⁽¹⁰⁾

The areal extent of the impacted area(s) may also be a consideration. If a source of concern occurs in the majority of communities, then it is possible that individuals may be exposed to the source for a longer period of time than one might predict using standard estimates of exposure duration. In this case, the analyst might assume that even though an individual changes residence, the individual still would be exposed to the source of concern, and thus the individual's exposure duration would be greater than typically anticipated. Such an analysis must consider whether the concentration of the pollutant at the multiple locations of exposure would be equivalent. Because location-specific parameters such as meteorological conditions, distance from the source, and the presence of certain pathways of exposure (e.g., surface water, home-grown produce) may vary considerably by geographic area, the analyst likely will have to estimate exposure concentrations for each geographic location or community of interest. Similarly, if a single source impacts a large geographic area, then it is possible that national estimates of population mobility will not adequately capture an individual's potential duration of exposure. That is, an individual may move from one point of exposure associated with a particular source to another point of exposure associated with that same source. For example, data indicate 29 percent of home buyers move less than five miles to a new home (Table 15-171 in EPA's *Exposure Factors Handbook, Part III*)⁽¹⁰⁾. Similar to the caution expressed above, the concentrations of pollutants within an area impacted by a single source may vary considerably. The analysis should reasonably account for such situations.

^cThe central tendency estimate of adult exposure duration commonly used in risk assessments is 9 years (Section 15.4.3 and Table 15-174 in EPA's *Exposure Factors Handbook, Part III*).⁽¹⁰⁾ This estimate is a median value based on national residential occupancy data for the general population. This estimate may not be appropriate in certain situations, such as when population-specific data exist or when the analyst is evaluating a specific sub-population that is expected to differ from the general population (e.g., farm families).

^dAs described in Section 20.4, estimation of high-end exposure will sometimes involve setting exposure duration at its high-end value. The high-end estimate of adult exposure duration typically used in risk assessments is 30 years (Section 15.4.3 and Table 15-174 in EPA's *Exposure Factors Handbook, Part III*),⁽¹⁰⁾ although this may vary for specific sub-populations.

The persistence of the source-associated contamination may also be an important consideration in the exposure duration for ingestion pathway exposure assessment. For example, the analyst should not automatically assume that the exposure duration can be no greater than the operating life of the source. Persistent pollutants may remain in the environment (e.g., soils and sediments) for years after the primary source is discontinued. Nevertheless, in certain cases, once the source of exposure stops, the pollutant concentrations in the affected media may diminish. Particularly in more refined assessments, the exposure concentration may reflect any expected variations in media or food concentrations over time.

When evaluating the risk of noncancer health effects from ingestion exposures (i.e., calculating hazard quotients for ingestion exposures), we do not average pollutant dose over the lifetime of an individual as we do when calculating carcinogenic risk. Rather, when calculating hazard quotients for ingestion exposures, we average the dose over an averaging time equivalent to only the period of exposure (i.e., we calculate an average daily dose rather than a lifetime average daily dose). Consequently, the values for exposure duration and averaging time are the same, and mathematically cancel each other out. Nevertheless, when calculating average daily dose, the analyst must still consider exposure duration when selecting and computing food and media intakes for use in the dose equation. EPA typically considers exposures of seven years or greater as chronic exposures. Food and media intakes that represent time-weighted averages over a seven-year period are reasonable for evaluating chronic non-cancer health effects. **Durations as short as one year are also commonly used, particularly in screening assessments, and for childhood evaluations where intake on a per body weight basis may rapidly change from year to year.**

20.4.4 Body Weight

The choice of body weight for use in the exposure assessment depends on the definition of the population group at potential risk. Because children have lower body weights, typical ingestion exposures per unit of body weight, such as for soil, milk, and fruits, tend to be higher for children. If a lifetime exposure duration (or an exposure duration over the childhood and adult years) is being evaluated, it needs to be based on differing values for the different age groups. If a less than a lifetime exposure estimate is being evaluated, it is important to include the children's age group in the specific scenarios or cohorts used. EPA's *Exposure Factors Handbook*⁽⁶⁾ and *Child-Specific Exposure Factors Handbook*⁽⁷⁾ provide age-specific values for body weight and consumption rate per unit body weight.

20.5 Calculating Averaging Time Value

When evaluating exposure for the purposes of assessing hazard (vs. predicting cancer risk), **intakes** are calculated by averaging intakes over the period of exposure (i.e., subchronic or chronic durations) and result in average daily doses or ADDs for the duration of interest. For evaluation of cancer risks, potential dose is calculated as the average daily dose over a lifetime (i.e., chronic daily intakes, also called lifetime average daily doses or LADDs). The approach for carcinogens is based on the premise that risk is proportional to total lifetime dose (i.e., a high dose received over a short period of time is equivalent to a corresponding low dose spread over a lifetime).⁽¹⁸⁾ The basis for this approach becomes less strong as the exposures in question become more intense but less frequent, especially when there is evidence that the agent has shown age-related variations in carcinogenic potency, or a nonlinear dose-response relationship.

In some cases, therefore, it may be necessary to consult a toxicologist to assess the level of uncertainty associated with the exposure assessment for carcinogens.

Note that, even when the exposure of interest is a full lifetime, chronic hazards are generally calculated separately for chronic exposures to age groups that differ substantially with regard to pertinent exposure factors (e.g., ingestion rate or body weight) and are not combined (i.e., usually the oral route hazards calculated for children are not added to the hazards posed to adults to represent a “lifetime hazard”). Rather, both hazard quotients/indices are presented as chronic hazard metrics relevant to the two groups. When assessing carcinogenic risks for a lifetime exposure, on the other hand, cancer risk estimates are usually added across different age groups, since the risk received over discrete periods of time (e.g., as a child, as a young adult, as an older adult) are each considered to be fractions of the risk associated with a full lifetime of exposure. Note that in calculating LADDs, it is essential to account for differences in the values of different intake variables (e.g., body weight, consumption rate) at different ages.

20.6 Combining Exposure Estimates Across Pathways

A given population may receive exposure to an individual chemical from several different exposure pathways. For example, individuals may receive exposure via inhalation of the chemical in the air and via ingestion of surface water and fish that have become contaminated through deposition. The specific exposure scenarios or cohorts defined for the analysis may include more than one pathway. The corresponding intake variables used in the analysis may need to account for the number of pathways over which exposure will be combined. For example, to develop a high-end estimate for a scenario that includes inhalation, ingestion of soil, and ingestion of fish, it may be necessary to combine high-end exposure assumptions for all pathways. In other cases, it may be more appropriate combine high-end exposure assumptions for particular pathways with more central-tendency assumptions for others. Otherwise, the estimate may represent an extreme situation in which the simulated behavior is assumed to result in high exposures via all pathways.

Two steps are required to determine whether intake estimates should be combined for a single scenario:

- **Identify reasonable exposure pathway combinations.** Identify exposure pathways that have the potential to expose the *same* individual, cohort, or subpopulation at the key exposure areas evaluated in the exposure assessment, making sure to consider *areas of highest exposure* for each pathway. For each pathway, the intake estimates have been developed for a particular exposure area and time period; they do not necessarily apply to other locations or time periods. Hence, if two pathways do not affect the same individual, cohort, or subpopulation, neither pathway’s exposure estimate affects the other, and exposures should not be combined.
- **Examine whether it is likely that the *same* individuals would consistently face a reasonable central tendency or high-end exposure by more than one pathway.** Once reasonable exposure pathway combinations have been identified, it is necessary to examine whether it is likely that the *same individuals* would *consistently* face central tendency or high-end exposure conditions. As noted in Section 20.4 above, the exposure estimate for each exposure pathway includes many conservative estimates. Also, some of the exposure

parameters are not completely predictable in space and/or time (e.g., the maximum downwind concentration may shift compass direction). For real-world situations in which contaminant concentrations vary over time and space, the same individual or cohort may or may not experience central-tendency or high-end exposure conditions for more than one pathway over the same period of time. Thus, it is important to clearly explain why the key assumptions chosen for more than one pathway for an individual, subpopulation, or cohort are set at central tendency and/or high-end exposure estimates. (Note that an important goal in the analysis of high-end receptors is to identify exposures that are in the high-end of the range - usually higher than the 90th percentile exposure - but not higher than the highest exposure in the population.)

20.7 Exposure Models

Exposure models have been developed that automate the calculation of chemical intake. They may simply calculate exposure for a set of individual scenarios, or they may draw upon activity pattern and/or dietary survey databases to characterize cohort exposure within a population. Three exposure models are described below.

California Total Exposure Model for Hazardous Waste Sites (CalTOX)

As described previously in Part II, Chapter 9, the California Environmental Protection Agency funded the development of the CalTOX program.⁽¹⁹⁾ CalTOX has been developed as a set of spreadsheet models and spreadsheet data sets to assist in assessing human exposures and defining soil clean-up levels at uncontrolled hazardous wastes sites. CalTOX addresses contaminated soils and the contamination of adjacent air, surface water, sediments, and ground water. The modeling components of CalTOX include exposure scenario models. The exposure models encompass twenty-three exposure pathways. The exposure assessment process consists of relating pollutant concentrations in the multimedia model compartments to pollutant concentrations in the media with which a human population has contact (e.g., personal air, tap water, foods, household dusts, soils). The temporal resolution is either daily for inhalation and dermal exposure or annual for ingestion. The aggregation period is variable, depending on the duration of residence at a single location. The spatial resolution and modeling domain are user-specified, but generally encompass some vicinity around the waste site of interest. Activity data, such as inhalation, ingestion, and dermal contact rates, are derived from EPA's *Exposure Factors Handbook*.⁽⁶⁾

TRIM.Expo

As discussed in Chapter 18, TRIM.Expo is the exposure component of the TRIM modeling system. The ingestion component of TRIM.Expo (TRIM.Expo_{Ingestion}) is designed to take input values from TRIM.FaTE, but may also be operated independently with inputs from measurement studies or alternative models. TRIM.Expo_{Ingestion} will employ a scenario-based approach, based on that used in the 3MRA modeling system, in its initial version. Information about the ingestion component of TRIM.Expo is available on EPA's Fate, Exposure and Risk Analysis (FERA) web site: <http://www.epa.gov/ttn/fera>.

Stochastic Human Exposure and Dose Simulation Model (SHEDS)

The Stochastic Human Exposure and Dose Simulation (SHEDS) Model⁽²⁰⁾ is a probabilistic, physically-based model that simulates aggregate exposure and dose for population cohorts and multimedia pollutants of interest. It is being developed by EPA's National Exposure Research Laboratory (<http://www.epa.gov/nerlpage/>). At present the model is applied to assess children's exposures to pesticides (SHEDS-Pesticides) and population exposures to particulate matter (SHEDS-PM).

SHEDS-Pesticides focuses on children's aggregate population exposure to pesticides. Activity data are selected from daily sequential time/location/activity diaries from surveys contained in EPA's Consolidated Human Activity Database (CHAD).⁽⁸⁾ For each individual, SHEDS-Pesticides constructs daily exposure and dose time profiles for the inhalation, dietary and non-dietary ingestion, and dermal contact exposure routes, and then aggregates the dose profiles across routes. A pharmacokinetic component has been incorporated to predict pollutant or metabolite concentrations in the blood compartment or eliminated urine. Exposure and dose metrics of interest (e.g., peak, time-averaged, time-integrated) are extracted from the individual's profiles. Two-stage Monte-Carlo sampling is applied to predict the range and distribution of aggregate doses within the specified population and identify the uncertainties associated with percentiles of interest.

SHEDS-Pesticides is currently being refined to characterize both aggregate and cumulative dose associated with human exposure (i.e., for both adults and children) to a variety of environmental pollutants in addition to pesticides. SHEDS-Pesticides will eventually be expanded to include source-to-concentration (i.e., fate and transport) models and more complete exposure-to-dose models (i.e., pharmacokinetic or dosimetric models).

SHEDS-PM estimates the population distribution of particulate matter (PM) exposure by sampling from distributions of ambient PM concentrations, distributions of emission strengths for indoor sources of PM (e.g., cigarette smoking and cooking), and distributions of mass-balance parameters (e.g., air exchange rate, penetration rate, deposition rate). A steady-state mass balance equation is used to calculate PM concentrations for the residential and other microenvironments. Additional model inputs include demographic and human activity pattern data from the National Human Activity Pattern Survey (NHAPS). Output from the SHEDS-PM model includes distributions of PM exposures in various microenvironments (e.g., in the home, in vehicles, outdoors) and the relative contributions of these various microenvironments to the total exposure.

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