Chapter 6
Quantifying Exposure

What's Covered in Chapter 6:

This chapter describes the factors to evaluate in quantifying the exposure received under each of the recommended exposure scenarios described in Chapter 4. Calculating COPC-specific exposure rates for each exposure pathway involves some or all of the following, depending upon the medium being assessed:

- the estimated COPC media concentrations calculated in Chapter 5,
- consumption rates of the medium,
- receptor body weight, and
- the frequency and duration of exposure.

We recommend repeating the appropriate calculation for each COPC and for each exposure pathway included in an exposure scenario, to generate multiple exposure concentration estimates, as recommended in the EPA information quality guidelines (see Chapter 1, page 1-11). We present recommended exposure pathway-specific equations in Appendix C. The following sections describe a general exposure rate calculation and the exposure pathway-specific variables that may affect this calculation. Acute exposure resulting from direct inhalation is discussed as a separate issue in Chapter 7, Section 7.4.

> ***PLEASE NOTE***: for the purposes of this guidance, "we" refers to the U.S. EPA OSW.
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> 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 is the performer of a risk assessment: the person (or persons) who will actually put the recommended methods into practice.

6.1 INHALATION EXPOSURE PATHWAYS

We recommend using COPC air concentrations calculated using the equation in Table B-5-1 to represent air concentrations for estimating exposure via inhalation by all exposure scenarios in the risk assessment (see Table 4-1).

Direct inhalation of vapors and particulate emissions from combustion sources is a potential pathway of exposure. Chapter 2 presented various variables and conditions that affect the rate, type, and quantity of combustor emissions. Chapter 3 presented the air dispersion and deposition modeling techniques we recommend using to estimate airborne concentrations of vapors and particulates in the assessment area.

As a result of normal respiration, receptors in the assessment area could be exposed to COPCs in vapor, particle, and particle-bound phases. Examples of factors that affect exposure from vapor and particulate inhalation include vapor and particulate COPC concentrations, particle size, and length of exposure.

Exposure can occur over a period of time. To calculate an average exposure per unit of time (Exposure Concentration, or *EC*), we recommend dividing the total exposure by the time period. We generally recommend using total COPC air concentrations (C_a, estimated using the equation in Table B-5-1) when estimating *EC* values. Estimating *EC*s doesn't involve or require adjustment for respiration rates, as those are inherent to inhalation toxicity factors. Sections 6.4 through 6.6 discuss exposure time-related parameters, and Appendix C Tables C-2-1 and C-2-2 further discuss estimating *EC*.

We consider it appropriate to estimate noncarcinogenic hazards and carcinogenic risks associated with direct inhalation exposure by combining *EC*s with inhalation toxicity factors (reference concentrations [RfCs] or unit risk factors [URFs]). These toxicity factors are developed for all human populations, including sensitive subpopulations (including children) who might be exposed to continuous concentrations over a lifetime. Inhalation risk parameters and inhalation pharmacokinetics are largely chemical-specific and a "one size fits all" approach to convert the standard RfC or URF parameters into scenario-specific toxicity values may not be appropriate. We therefore generally recommend that a single (lifetime) inhalation risk be predicted for each receptor identified in Chapter 4, Table 4-1.

Inhalation exposure concentrations for vapors and particles (arising from outdoor sources) can be influenced by the relative amount of time that a receptor spends indoors. Although vapors entering buildings and residences as a result of air exchange are likely to remain airborne and, therefore may be inhaled, particulates entering these same buildings are more likely to settle out and not be inhaled. However, for the purpose of the risk assessment, we recommend assuming that both vapor and particulates are inhaled throughout the day, both indoors and outdoors.

6.1.1 Soil Inhalation Resulting from Dust Resuspension

We don't typically recommend evaluating the soil inhalation of resuspended dust exposure pathway. However, site-specific exposure setting characteristics might support evaluating it (e.g. arid, windy climates). This section therefore discusses exposure to soil resulting from dust resuspension.

Inhalation of soil resulting from dust resuspension could be an issue for site-specific exposure scenario locations at which there is little vegetative cover. Wind erosion could resuspend pollutants in contaminated soil as particles in the air. As dust is resuspended, receptors could inhale the pollutant particles (direct inhalation of particulate matter is addressed in Section 6.1). The amount resuspended depends on the:

- moisture content of the soil,
- fraction of vegetation cover,
- wind velocity,
- soil particle size,
- pollutant concentration in the soil, and
- size of the contaminated area.

Study of estimated exposures to deposited combustor emissions via dust resuspension indicates that dust resuspension by wind erosion is usually not a significant pathway (U.S. EPA 1998c). Methods have also been developed to assess the exposure to pollutants resuspended by wind erosion for landfills and Superfund sites (U.S. EPA 1985a; 1988b; 1994q). We recommend consulting these reference documents if you'll be evaluating this exposure pathway. Also, it may be useful to review the methods described in U.S. EPA (1998c).

6.2 INGESTION EXPOSURE PATHWAYS

Exposure can occur over a period of time. To calculate an average exposure per unit of time, we recommend dividing the total exposure by the time period. Express an average exposure in terms of body weight. Ingestion exposures quantified per the HHRAP are

- unitized for time and body weight,
- presented in units of milligrams per kilogram of body weight per day, and
- termed "intakes."

Equation 6-1 is a generic equation for calculating ingestion chemical intake (U.S. EPA 1989e):

$$I = \frac{C_{gen} \cdot CR \cdot EF \cdot ED}{BW \cdot AT} \qquad \text{Equation 6-1}$$

where

I	=	Intake—the amount of COPC at the exchange boundary (mg/kg/day); for evaluating exposure to noncarcinogenic COPCs, 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*)
C_{gen}	=	Generic COPC concentration in medium of concern (e.g., mg/kg for soil or mg/L for surface water; see Chapter 5)
CR	=	Consumption rate—the amount of contaminated medium consumed per unit of time or event (e.g., kg/day for soil or L/day for water)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
BW	=	Average body weight of the receptor over the exposure period (kg)
AT	=	Averaging time—the period over which exposure is averaged (days); for carcinogens, the averaging time is 25,550 days, based on a lifetime exposure of 70 years; for noncarcinogens, averaging time equals *ED* (years) multiplied by 365 days per year.

We recommend variations of Equation 6-1 to calculate pathway- and receptor-specific exposures to COPCs. We present the equations recommended for each exposure pathway in Appendix C. The variation of input variables is also described in Appendix C.

The exposures calculated using the HHRAP are intended to represent reasonable maximum exposure (RME) conditions, as further described in U.S. EPA (1989e). Studies of the compounding of conservatism in probabilistic risk assessments show that setting as few as two factors at RME levels or high end (e.g., near the 90th percentile), while setting the remaining variables at less protective typical, or "central tendency" values (e.g., near the 50th percentile) results in output insignificantly different from output generated using all input variables at an RME level (e.g., 99th percentile value) (Cullen 1994).

As described in Chapter 2 (Section 2.2.1), the estimated air concentrations and deposition rates are based on RME emissions from trial or risk burns. We recommend setting the following variables set at RME levels:

- the highest ISCST3 modeled air concentrations and deposition rates at chosen exposure scenario locations,
- the exposure frequency, and
- the exposure duration.

We generally recommend setting other exposure parameters (e.g. body weight) at average levels.

6.2.1 Body Weight

The choice of body weight to use in the risk characterization equations presented in Appendix C depends on the definition of the receptor at risk—which, in turn, depends on exposure and susceptibility to adverse effects. U.S. EPA (1990e) defines the body weight of the receptor as either adult weight (70 kilograms) or child weight (1 to 7 years; 17 kilograms) on the basis of data presented in Nelson et al. (1969). However, as in other Agency guidance (U.S. EPA 1991b; 1994r; 1994g), we recommend using a weight of 15 kilograms for the child (exposure duration of 6 years) in the risk assessment.

The daily intake for an exposure pathway is expressed as the dose per body weight. Because children have lower body weights than adults. Typical ingestion exposures per body weight, such as for soil, milk, and fruits & vegetables, can be substantially higher for children. This is the primary reason to evaluate the child resident scenario (U.S. EPA 1996g). However, using these two body weights may not account for significant differences between weights of infants and toddlers or weights of teenagers and adults. Please remember that, for the purposes of the risk assessment, the child scenario is defined by the average body weight, rather than the chronological age. Obviously, the weight of a child changes significantly over the first several years. We assume 15 kilograms is a realistic average estimate for an exposure duration of 6 years. 15 kilograms overestimates the weight of the child for the early years, and then underestimates it for the later years (U.S. EPA 1996g).

6.2.2 Food (Ingestion) Exposure Pathways

Plants and animals impacted by emission sources may take up emitted COPCs in the air or deposited COPCs in soil. Humans could then be exposed to COPCs via the food chain when they consume these plants and animals as food. We generally recommend determining human intake of COPCs based on the:

- types of foods consumed,
- amount of food consumed per day,
- concentration of COPCs in the food, and
- percentage of the diet contaminated by COPCs.

Chapter 6 describes procedures for determining the concentration of COPCs in food. It also considers the variations in exposure resulting from food preparation methods and type of food item (e.g., aboveground versus belowground - i.e. root - vegetables). Other variables, described below, may also significantly affect exposure estimates.

6.2.2.1 Types of Foods Consumed

The types of foods consumed will affect exposure, because different plant and animal tissues take up different COPCs, and take them up at different rates. The COPC concentrations a receptor is exposed to will then vary with the types of food in the diet. Therefore, it is important to determine COPC concentrations in food according to the type of food. The types of food consumed might also vary with the age of the receptor, geographical region, and socio-cultural factors.

6.2.2.2 Food Consumption Rate

Consumption rate is the amount of contaminated medium (soil, food) consumed per unit of time or event. The soil at an exposure location are inherent to the location. Food consumed at an exposure location, however, may or may not have originated there. The HHRAP assumes that only food produced at the exposure location is contaminated by emissions from the facility being assessed. Food not produced at the point of exposure is not assumed to be contaminated, and is irrelevant to the assessment. Therefore, the consumption rates we recommend in the HHRAP (see Table 6-1 and Appendix C) are for food that is both produced and consumed at the exposure location (i.e. at home).

> ***Please Note***: these rates do not represent the entire dietary intake of the individual, but only that portion of the diet produced at home. For example, the beef consumption rate represents only the amount of beef consumed each day which was raised on the farm property.

TABLE 6-1

MEAN CONSUMPTION RATES[a] FOR RECOMMENDED EXPOSURE SCENARIOS (number of servings per week)

Contaminated food	Exposure Scenario					
	Farmer [b]	Farmer Child[b]	Resident	Resident Child	Fisher	Fisher Child
Produce (8 oz servings)	2.8	1.4	2.3	1.2	2.3	1.2
Beef (1/4 lb servings)	5.3	0.7	N/A	N/A	N/A	N/A
Milk (8 oz servings)	29.5	10.5	N/A	N/A	N/A	N/A
Chicken (1/4 lb servings)	2.8	0.4	N/A	N/A	N/A	N/A
Eggs (number[c])	4.3	0.7	N/A	N/A	N/A	N/A
Pork (1/4 lb servings)	2.4	0.4	N/A	N/A	N/A	N/A
Fish (1/4 lb servings)	N/A	N/A	N/A	N/A	5.4	0.8

Notes:
[a] Values derived from the U.S. EPA Exposure Factors Handbook (1997).
[b] Values based on consumption rates of a 154 lb adult and a 33 lb child.
[c] Values based on an assumed egg weight of 3.0 ounces.

As described in Section 6.2, exposures calculated using HHRAP methods are intended to represent RME conditions. Accordingly, the HHRAP recommends default values for exposure parameters that will result in RME estimates. However, there are likely to be differences between recommended default, and regional and site-specific exposure parameter values. This may be especially true for the consumption rates (a general term for intake rates and inhalation rates). In addition to estimates using the recommended default parameter values, you can refine the your risk assessment by including supplemental calculations using regional- or site-specific parameter values. We recommend doing this only if you document the regional- or site-specific parameter values in the risk assessment report. We recommend providing these supplemental calculations in addition to, and not instead of estimates based on recommended default exposure parameter values. This will help standardize risk assessment methods, thereby aiding the ability to compare outputs from different risk assessments. The following subsections describe exposure pathway-specific considerations regarding consumption rate.

This section gives some of the pertinent history of consumption rates. It also describes the series of steps we followed to derive the recommended consumption rates found in Table 6-1. Site-specific conditions might exist such that you need to derive an alternative, or additional consumption rate. For example, you may need a consumption rate for an additional exposure scenario (e.g. consumption of deer meat for a hunter scenario). If you need to calculate your own consumption rate(s), we recommend using the process described below. For transparency and clarity, we recommend identifying all consumption rates used in the risk assessment. We also recommend clearly identifying and discussing alterations to recommended default rates, and additional consumption rates, with the permitting authority prior to use.

The amount of daily food consumption varies with age, sex, body weight, and geographic region, and it also varies within these categories. U.S. EPA (1998c) recommended using values from the 1997 *Exposure Factors Handbook* (EFH) (U.S. EPA 1997b) to complete the risk assessment process. The EFH used the 1987-1988 USDA Food Consumption Survey to represent consumption rates for urban and suburban areas. However, if site-specific information indicates that the population is in a more rural or agricultural area, U.S. EPA (1990e) recommended using the 1966-67 USDA Food Consumption Survey to represent the consumption rates of a more agrarian population.

The 1997 *Exposure Factors Handbook* (EFH) (U.S. EPA 1997b) performed an analysis of the 1987-1988 USDA National Food Consumption Survey (NFCS). The NFCS collects information over a 7-day period on the socioeconomic and demographic characteristics of households, and the types, values, and sources of foods consumed. The following information was taken from the survey:

- whether or not the food product was used in the house that week;
- whether or not the food product used that week was home produced;
- the quantity (mass, such as pounds or kilograms) of food consumed (home produced or not) in the house that week;
- the number, age, and body weight of individuals in the household; and
- the number of weekly meals consumed by each family member.

All households were surveyed about the same food types, and consumption rates were averaged over the entire survey population - to calculate what are known in the EFH as the "Per Capita" rates. In addition, EPA calculated consumption rates for "consumers only:" a rate for only those households which consumed a particular food stuff during the week the survey was taken. In addition to total consumption (i.e. for the entire population of consumers), rates were broken out according to various criteria, such as age of consumer, geographic region, and level of urbanization. Survey participants were also asked if they operated a farm or ranch, raised animals, or had a home garden, and consumption rates were also broken out for these sub-groups. We recommend using food consumption rate information (ingestion rates) from the EFH; specifically, the section regarding home produced food items.

Chapter 13 of the EFH (Intake Rates for Various Home Produced Food Items) lists consumer only consumption rates of home produced food. For example, Table 13-65 (Consumer Only Intake of Home grown Root Vegetables (g/kg-day)) lists a mean consumption rate of 1.16 g/kg-day for all households that consumed root vegetables they produced themselves. From these households, the EFH also breaks out consumption rates specific to households who farm (e.g. a mean of 2.63 g/kg-day). These farm-specific rates represent the amount of food that farm families produced themselves that was consumed during the week the survey was taken. We recommend using these consumption rates to estimate exposures to the Farmer and Farmer Child scenarios.

The recommended farm-specific consumption rates, as listed in the EFH, are averaged across all family members, regardless of age or body weight. They also do not consider losses during preparation and cooking of the food. Additional work is needed, therefore, to acquire scenario-appropriate actual consumption rates:

As mentioned above, the EFH provides consumption rates for the entire population of consumers - the consumer only rate. Depending on data availability, the consumer only rate is also broken out into various age-specific demographics, covering 0-1 year, 1-2 years, 3-5 years, 6-11 years, 12-19 years, 20-39 years, and 40-69 years. We suggest combining the consumer only rate, age-appropriate demographic rates, and the sub-population rate (e.g. for a farm family) to derive a scenario-specific consumption rate (e.g. for an adult Farmer) using equation 6-2:

$$CR_{scen} = CR_{subpop} \times \frac{TWA_{gen}}{CR_{gen}}$$ Equation 6-2

where:

CR_{scen}	= Consumption rate for scenario
CR_{subpop}	= Consumption rate of subpopulation
TWA_{gen}	= Time-weighted average of age-appropriate subset of consumer only population
CR_{gen}	= Consumption rate for consumer only population

Sufficient age-specific data were not always available. For example, there was insufficient data for the EFH to provide a consumer only poultry consumption rate for the 12-19 age group. In this event we suggest again using Equation 6-2, to combine the total consumer only population rate, the age-specific Per Capita rate, and the total Per Capita rate (for poultry, Per Capita values are found in EFH Table 11-11), to generate an age-specific consumer only rate.

See the example derivation below for a demonstration of how we recommend generating scenario-specific consumption rates. To derive consumption rates for the Farmer and Farmer Child exposure scenarios, we recommend using the home produced, consumers only consumption rates of households who farm, for home produced beef, pork, chicken, milk, eggs, vegetables, and fruits.

Consumption rates of households who farm are not necessarily the most appropriate rates for households who don't farm. For certain food types, the EFH also breaks out consumption rates of home-produced foods specific to those households who garden. This combination of subpopulation (i.e. gardeners) and food source (i.e. home produced) is the closest option available in the EFH to the residential and fishing scenarios we recommend in the HHRAP. Consumption rates for this subpopulation are available for the food types included in the produce-related exposure pathways we recommend (i.e. protected produce, exposed produce, and belowground vegetables). We recommend using these rates to generate produce-related consumption rates for the Resident, Resident Child, Fisher, and Fisher Child exposure scenarios.

The EFH provides information to account for cooking and post-cooking losses for food products which are home produced. These data are summarized in Table 6-2. See the example derivation below for a demonstration of how to use cooking loss data in deriving consumption rates.

TABLE 6-2

COOKING-RELATED WEIGHT LOSSES
FOR VARIOUS HOME-PRODUCED FOODS

Percent Weight Losses from Preparation of Various Meats (SOURCE: EFH Table 13-5)		
Meat Type	Mean Net Cooking Loss (%)	Mean Net Post Cooking Loss (%)
Beef	27	24
Pork	28	36
Chicken	32	31
Milk	N/A	N/A
Eggs	N/A	N/A
Fish	30	11
Percent Weight Losses from Preparation of Various Produce (SOURCE: EFH Tables 13-6 and 13-7)		
Produce Type	Mean Paring or Preparation Loss (%)	Moisture Content (%)
Protected Fruits	29	87
Protected Vegetables	23	82
Exposed Fruit	21	85
Exposed Vegetables	16	90

Example Consumption Rate Derivation: Homegrown Poultry consumption for the Farmer

The Farmer scenario includes an average consumption rate during a 70-year lifetime, from age 7 to 70. Table 13-55 of the EFH lists the following Farmer-related mean consumption rates of home produced poultry (g/kg-day):

Total population	1.57	
broken out by age group:		
ages 6-11	ND	(5 years)
ages 12-19	ND	(8 years)
ages 20-39	1.17	(20 years)
ages 40-69	1.51	(30 years)
households who farm	1.54	

Please note that though the EFH demographic subset includes years 6-11, the Farmer scenario begins at age 7, so only 5 years of that consumption rate are used.

In addition, Table 11-11 (Mean Meat Intakes Per Individual in a Day, by Sex and Age (g/day as consumed) for 1987-1988) lists the following Per Capita consumption rates of poultry (among others):

All individuals	26
broken out by age group:	
ages 6-11	27
ages 12-19	27

Using the 12-19 age group as an example, equation 6-2 is used to calculate age-specific consumer only rates as follows:

$$CR_{ages12\text{-}19} = 1.57 * 27/26 = 1.63 \text{ g/kg-day}$$

A time weighted average for an adult Farmer =

$$TWA_a = \frac{(1.63 \times 5) + (1.63 \times 8) + (1.17 \times 20) + (1.51 \times 30)}{5 + 8 + 20 + 30} = 1.43$$

And, using Equation 6-2:

$$CR_{Farmer} = 1.54 * 1.43/1.57 = 1.40 \text{ g/kg-day}$$

Table 13-5 of the EFH lists cooking and post-cooking losses for poultry of 31% and 32%, respectively.

Therefore:

$$1.40 * (1\text{-}0.31)*(1\text{-}0.32) = 0.66$$

Poultry consumption rate for the Farmer = 0.66 g/kg-day

Additional consumption rate information is presented in Appendix C as follows: Table C-1-2 (produce); Table C-1-3 (beef, milk, pork, chicken, and eggs); and Table C-1-4 (fish).

6.2.2.3 Percentage of Contaminated Food

The percentage of food consumed by an individual which is home-grown will affect exposure, because the HHRAP assumes that only the portion of an individual's dietary intake which is home-grown is impacted by facility emissions.

We recommend assuming that all food produced at the exposure location - i.e. the farm for the farming scenarios, and the home garden for the residential and fishing scenarios - is impacted by facility emissions. Only that portion of the diet produced at home (and therefore exposed to facility emissions) is of consequence in the risk assessment. As detailed in Section 6.2.2.2, the consumption rates we recommend represent only the home-produced portion of the diet. Therefore, by using consumption rates specific to home produced foods, we consider it reasonable to assume that 100% of those home produced foods are contaminated.

6.2.3 Soil (Ingestion) Exposure Pathway

Soil ingestion, dermal exposure to soil, and inhalation of resuspended dust are potential soil exposure pathways. For the purpose of RCRA combustion permitting decisions, we recommend considering soil ingestion. However, we currently only recommend evaluating dermal exposure to soil (see Section 6.3) and inhalation of resuspended dust (see Section 6.1.1) if site-specific exposure setting characteristics support evaluating these exposure pathways. Based on air dispersion modeling and deposition of COPCs, emission concentrations in soil will vary with distance from the source. It's possible to determine potential routes of exposure by evaluating the way in which the soils in the area are used. Soil used for farming or recreation will be involved in pathways of human exposure that differ from those of soil on roadways or in urban areas.

Children and adults are exposed to COPCs in soil when they consume contaminated soil that has adhered to their hands. Factors that influence exposure by soil ingestion include soil concentration, 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 measured the quantities of non-absorbable 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 activity. Indoor dust and outdoor soil may both contribute to the total daily ingestion. Exposure levels are also influenced by the amount of time that the individual spends in the vicinity of soil exposed to deposition of emitted pollutants.

In addition, some young children—referred to as “pica” children—may intentionally eat soil. As discussed in U.S. EPA (1989f), the typical medical and scientific use of the term “pica” refers to the ingestion of non-food items, such as soil, chalk, and crayons. Such behavior is considered a temporary behavior and a normal part of child 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 (U.S. EPA 1989f). Agency risk assessment documents don’t identify a default “pica” soil ingestion rate (U.S. EPA 1989e; 1989f ; 1991b). Pica behavior is not generally included as part of risk assessments.

If available information indicates that there are children exhibiting pica behavior in the assessment area, and you determine that these children represent a special subpopulation potentially receiving significant exposure (see Chapter 4), it may be prudent to include these children in the risk assessment. We recommend making this evaluation on a case-by-case basis based on site-specific exposure setting characterization.

6.2.4 Water (Ingestion) Exposure Pathways

Evaluating HHRAP water exposure pathways involves estimating COPC concentrations in drinking water from surface water bodies or collected precipitation (e.g., cisterns). Contaminants moving through the water pathways also influence COPC concentrations in fish. Various models are available to estimate daily exposures of individuals using these water sources for various purposes, such as fishing and drinking water.

We recommend using site-specific information to determine which water exposure pathways to evaluate in the risk assessment. Whether it's collected precipitation, or from a surface water body such as a lake, farm pond, or city reservoir, the way in which water is used will suggest possible exposure pathways. For example, using a surface water body as a drinking water source will introduce water ingestion as a possible exposure pathway. Commercial and/or recreational fishing, with subsequent use of fish and shellfish as a food source, make the food chain an important route of exposure for communities having a surface water body in the vicinity of a combustor.

U.S. EPA (1998c) recommended varying the water input variables to determine a range of exposures. An individual that fishes and obtains drinking water from the same water source could represent an average exposure scenario. A worst-case possibility might involve a person who (1) uses drinking water from a cistern that collects precipitation, and (2) fishes in a small farm pond.

Because ground-level concentrations of COPCs generally decrease with distance from the source, important factors in determining the water concentration include:

- the location of the precipitation-collection apparatus,
- surface water body onto which emitted COPCs are deposited, and
- the COPC soil concentration (which affects runoff and leachate concentrations).

In addition, the location and size of the watershed will affect the concentration of COPCs suspended in runoff.

6.2.4.1 Ingestion of Drinking Water from Surface Water Sources

For evaluating a surface water body as a drinking water source, exposure is affected by the COPC concentration in the water, the daily amount of water ingested, and the length of time that the receptor spends in the area serviced by that water supply system. The equations we recommend for estimating the COPC concentration in a surface water body are discussed in Chapter 5 and Appendix B. These equations also consider contributions of COPC loading from the surrounding watershed. We recommend using the water consumption rates specified in U.S. EPA (1997b) and described in Appendix C.

As in previous Agency guidance (U.S. EPA 1998c), we recommend typically assuming that treatment processes for drinking water do not alter dissolved COPC concentrations.

6.2.4.2 Ingestion of Drinking Water from Ground Water Sources

For the purpose of RCRA combustion permitting decisions, we don't typically recommend evaluating exposure from ground water sources used as drinking water. Study of this pathway for combustor emissions indicates that this isn't a significant exposure pathway (U.S. EPA 1998c). However, COPCs may - because of special site-specific characteristics - infiltrate into ground water, resulting in COPC exposure via ingestion when ground water is used as drinking water. This could be because of extremely shallow aquifers being tapped for drinking water, or a karst environment in which the local surface water significantly affects the quality of ground water used as a drinking water source. The method developed to calculate risks from the ground water pathway was originally intended for use in evaluating impacts of the land disposal of various wastes (U.S. EPA 1998c; 1994q; 2003). We recommend consulting these reference documents if you intend to evaluate this exposure pathway.

6.2.4.3 Ingestion of Fish

You may find the fish ingestion rates specified in U.S. EPA (1997b) and further described in HHRAP Appendix C useful for evaluating the fish ingestion pathway. Factors that affect human exposure by ingestion of fish from a surface water body affected by combustion unit emissions include:

- COPC concentrations in the sediment and water column,
- the types of fish and shellfish consumed,
- the ingestion rates for the various fish and shellfish groups, and
- the percent of dietary fish caught in the surface water body affected by the combustor.

The types of fish consumed will affect exposure, because different types of fish and shellfish take up COPCs at different rates. For example, fatty fish tend to accumulate organic COPCs 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 vary greatly, depending on geographic region and social or cultural factors. For example, populations such as Indian tribes, American & Pacific Islanders, and some immigrant groups are known to have high local fish consumption rates. Because 100 percent of a receptor's dietary fish may not originate from the surface water body near the combustion facility, the percentage of locally caught fish is also a variable for exposure.

6.3 DERMAL EXPOSURE PATHWAYS

6.3.1 Dermal Exposure to Soil

For the purpose of RCRA combustion permitting decisions, We don't typically recommend evaluating dermal exposure to COPCs through contact with soil. However, site-specific exposure setting characteristics may support evaluating this exposure pathway. Therefore, this section discusses dermal soil exposure.

Available data indicate that the contribution to overall risk from dermal exposure to soils impacted from hazardous waste combustion facilities is typically small relative to contributions resulting from exposures via the food chain (U.S. EPA 1995h; 1996g). For example, the risk assessment conducted for the Waste Technologies Industries, Inc., hazardous waste incinerator in East Liverpool, Ohio, indicated that—for an adult subsistence farmer in a subarea with high exposures—the risk resulting from soil ingestion and dermal contact was 50-fold less than the risk from any other exposure pathway and 300-fold less than the total estimated risk (U.S. EPA 1995h; 1996g).

Humans can be exposed to COPCs by absorption through the skin when it comes into contact with contaminated soil. Factors that affect dermal exposure include:

- exposed skin surface area;
- contact time;

- contact amount;
- amount of time spent near the combustion source; and
- fraction of COPCs absorbed through the skin.

In general, an increased dose of COPCs can potentially be absorbed through the skin as the surface area of the skin is increased. Surface area is affected by age and body weight; for example, children have less total surface area than adults. The amount of surface area available for exposure to soil is also affected by the amount of clothing worn. An adult working in the garden in long sleeves and pants will have a smaller exposed surface than an adult working in shorts and a short-sleeved shirt. For dermal exposure from soil, the exposed surface area affects the amount of soil that can adhere to exposed skin.

Contact time refers to the duration of time each day that skin is in contact with contaminated soil. As duration increases, so does the amount of COPCs that can be absorbed. Dermal exposure is also affected by the amount of time each day spent in the vicinity of the combustion source, where the soil may contain pollutants emitted from the combustion facility. Indoor dust and outdoor soil may both increase the daily contact. Consider seasonal exposure might also be appropriate, because regional climate will influence contact time.

The amount of COPCs that can be absorbed through the skin depends on the chemical properties of the COPC, properties of the soil matrix, and dermal pharmacokinetics. If a COPC can’t be readily absorbed through the skin, the daily intake of the COPC may be small even if other exposure characteristics (e.g. contact time) encourage absorption. However, if site-specific conditions suggest that dermal exposure to soil may contribute significantly to total soil-related exposures, we recommend considering the assessment methods described in U.S. EPA (2004d).

6.3.2 Dermal Exposure to Water

We don’t typically recommend evaluating the dermal water exposure pathway when assessing risk from hazardous waste combustor emissions. However, if the surface water body affected by combustor emissions is used frequently for recreational purposes such as swimming and boating, dermal absorption of contaminated water becomes another possible route for human exposure. Dermal exposure is affected by:

- the surface area of exposed skin,
- the COPC concentration in the water,
- the permeability of the skin to the COPC, and
- the length of time that the individual is in contact with the water.

6.4 EXPOSURE FREQUENCY

The HHRAP assumes that the receptors in each recommended exposure scenario are exposed to all of the scenario-specific exposure pathways 350 days per year (U.S. EPA 1989e; 1991b; 1991d). This assumption is based on the protective estimate that all receptors spend a maximum of 2 weeks away from the exposure scenario location selected in Section 4.3.

6.5 EXPOSURE DURATION

Exposure duration is the length of time that a receptor is exposed via a specific exposure pathway. A receptor is no longer exposed to COPCs via the direct inhalation exposure pathway after an emission source ceases operation. However, a receptor could be exposed via the indirect exposure pathways for as long as they remain in the assessment area. We recommend using default RME values to estimate exposure duration for specified receptors.

As in U.S. EPA (1998c), we recommend assuming that receptors are exposed to the long-term average COPC soil or water concentrations (and the subsequent COPC plant or animal concentrations) present in the environment or media following a period of time during which there were continuous hazardous waste combustor emissions. For existing facilities, U.S. EPA (1990e) assumed that this period of time can be represented by default time periods of 30, 60, or 100 years. U.S. EPA (1998c) simplified this to assume that the period could be ≥ 30 years. These values are based on the assumptions that the hazardous waste combustion unit or the emission source:

1. is already in place,

2. will continue to be used for the rest of its useful life (estimated to be 30 years), and

3. may be replaced when it reaches the end of its useful life (possibly as long as 60 or 100 years), because it is an integral part of the facility operations.

We consider these assumptions reasonable for a hazardous waste emission source, such as an industrial boiler burning a continuous stream of facility hazardous waste.

Although a combustor may remain in the same location for 100 years—and a person may have a lifetime of exposure to emissions from that combustor— data on population mobility (U.S. Bureau of the Census, 1986) indicate that many Americans do not remain in the same area for their 70-year lifetime. An estimate of the number of years that a person is likely to spend in one area, such as the vicinity of a combustion facility, can be derived from information about mobility rate and median time in a residence. In addition to the number of years at a particular location or residence, the amount of time spent at that location each day directly affects exposure. For example, children that attend day care or adults that work in a different location for part of the day may be exposed to higher or lower COPC levels.

The exposure duration values we recommend are presented in Table 6-3.

TABLE 6-3

EXPOSURE DURATION VALUES

Recommended Exposure Scenario Receptor	Value	Source
Child Resident	6 years	U.S. EPA 1990f; 1994r
Adult Resident	30 years	U.S. EPA 1990f; 1994r
Fisher	30 years	U.S. EPA 1990f; 1994r
Fisher Child	6 years	Assumed to be the same as the Child Resident
Farmer	40 years	U.S. EPA 1994l; 1994r
Farmer Child	6 years	Assumed to be the same as the Child Resident

6.6 AVERAGING TIME

For noncarcinogenic COPCs, we generally recommend using a value of exposure duration (years-as specified for each receptor in Section 6.4) x 365 days/year as the averaging time (U.S. EPA 1989e; 1991d). However, for carcinogenic COPCs—the effects of which may have long latency periods—the age of the receptor (i.e., child, adult, or elderly) influences the COPC exposure pathway, because the exposure duration and, therefore, the quantity of exposure, will vary. For carcinogenic COPCs, we recommend using an averaging time of 70 years.

We recommend evaluating carcinogenic exposures for different receptor ages separately, because the daily activities of these receptors (and body weights, as described in Section 6.6) vary, including:

- the amounts of food and water consumed;
- the types of food consumed; and
- the amount of exposed skin surface.

Health-based criteria, such as health advisories for drinking water, are also different for children and adults. As a result, for some exposure pathways, such as soil ingestion, children may have a greater quantifiable exposure and be at greater risk than adults. Some behaviors, such as mouthing of dirty objects or direct ingestion of soil, which could also contribute to exposure, are also much more prevalent in children than adults.

Because quantifying carcinogenic COPC exposure depends on the duration of exposure, the age of the receptor is important. For risk assessment purposes, the average human lifespan is generally considered to be 70 years. Childhood represents only about 10 percent of the lifespan (6 years) (U.S. EPA 1998c). In actual exposure scenarios, individuals may be exposed only during childhood or adulthood. In other cases, exposure may overlap these periods, such as a child who grows into adulthood and remains in the same geographical area. Based on the age of the receptor and information on the duration of exposure, U.S. EPA (1990e) recommended considering risk to three different receptors:

1. a child who grows to an adult and is exposed for his or her entire 70-year lifetime,

2. a child who grows to an adult and is exposed for only a part of his or her adulthood—a total of 30 years, and

3. an adult exposed for 16 years.

Because the effects of certain carcinogenic COPCs may have long latency periods—in some instances approaching the human lifespan—it may be appropriate to estimate daily intake by using the adult value for body weight and a longer averaging time. In cases where effects have a shorter latency period, U.S. EPA (1990e) recommended an averaging time period of less than 10 years. However, where children are known to be at special risk, it may be more appropriate to use this averaging time with a body weight value for toddlers, infants, or young children. For COPCs classified as carcinogens, we recommend using a longer averaging time and the adult body weight to calculate the risk resulting from air or water exposure.

It is significant that childhood is defined differently in the different references. U.S. EPA (1990e) defines childhood as being from 1 to 7 years old. As in this and other Agency guidance (U.S. EPA 1991b; 1994r; 1998c), we define childhood as having an exposure duration of 6 years. Please note that some of the data used for input into the various exposure scenario equations in Appendix C was not available for children, or was available for more restrictive age groups, such as 2-year-olds or 4- to 6-year-olds. In such cases, and as noted in Appendix C where such values are presented,

1. the available data were evaluated to ensure that the presented default values are sufficient for conducting a risk assessment, and

2. in cases in which the available data were not sufficient, reasonable interpolations of the available data were possible.

RECOMMENDED INFORMATION FOR THE RISK ASSESSMENT REPORT

- Identification of site-specific or alternate default media equations and/or inputs; including justification and full referencing
- Exposure calculations