3.0 Human Health Risk Assessment

EPA uses risk assessment to characterize the potential cancer risks and non-cancer hazards for individuals exposed to contaminants in environmental media. A systematic framework for risk assessment was first outlined by the National Academy of Sciences (NAS, 1983). Building upon this foundation, EPA has developed risk assessment guidance (e.g., USEPA, 1984, USEPA, 1989; USEPA, 1995) that consists of the following components:

- *Data Collection and Analysis* - involves gathering data to define the nature and extent of contamination in the environmental media of concern.
- *Exposure Assessment* - characterizes how people may be exposed to environmental contaminants and estimates the magnitude of these exposures.
- *Toxicity Assessment* - examines the types of adverse health effects associated with chemical exposure, and the relationship of the magnitude of exposure and the health response.
- *Risk Characterization* - estimates the potential for adverse health effects (both cancer risk and non-cancer hazards) by integrating the information on toxicity and exposure.

The data collection and analysis step for this study have been previously discussed in Section 1. Section 2 provides information on contaminant levels in fish tissues. Section 4 (Exposure Assessment) describes how these contaminant levels are used with other exposure information (e.g. how much fish people eat) to estimate the magnitude of exposure for people consuming fish from the Columbia River Basin. Section 5 (Toxicity Assessment) provides the toxicity information that is used with the exposure estimates to characterize cancer risks and non-cancer hazards in Section 6 (Risk Characterization).

4.0 Exposure Assessment

The objective of this exposure assessment is to estimate the amount of contamination that a person may be exposed to from eating fish caught as a part of this study.

4.1 Identification of Exposed Populations

The potentially exposed populations for this risk assessment include (1) individuals within the general public, and (2) CRITFC's member tribes.

As previously discussed in Section 1 of this report, the basis for the design of this fish study was the fish consumption survey conducted by CRITFC (CRITFC, 1994), which targeted members of the Nez Perce, Umatilla, Yakama, and Warm Springs Tribes (Appendix A). The CRITFC study is the only comprehensive survey of fish consumption that has been conducted for the Columbia Basin and was used to develop tribal fish ingestion rates for this risk assessment.

Three other recent fish consumption surveys have been conducted in the Columbia River Basin: in the middle Willamette River (EVS, 1998), lower Willamette River (Adolfson Associates, Inc., 1996), and in Lake Roosevelt (WDOH, 1997). These three studies are limited in scope and focused on specific regions or populations within the Columbia River Basin. Therefore, the data from them was not used to develop fish ingestion rates for this risk assessment. However, these three surveys as well as the CRITFC survey are discussed in Section 4.5 (Fish Ingestion Rates) because all the surveys illustrate the point that fish consumption practices can vary greatly depending upon the age, gender, cultural practices, and/or socioeconomic status of the anglers surveyed. These variations can include the types and amounts of fish eaten, the frequencies of meals, the portions of the fish that are eaten, and the preparation methods (USEPA, 1998a).

4.2 Exposure Pathway

An exposure pathway describes the course a chemical or physical agent takes from the source to the exposed individual. A complete description of an exposure pathway involves four elements: 1) a source and mechanism of chemical release, 2) movement of the chemical through the environment resulting in contamination of environmental media, 3) a point of potential human contact with these contaminated media (referred to as the exposure point), and 4) an exposure route, such as ingestion, at the point of contact with these media (USEPA, 1989). While several different exposure pathways could conceivably result in human exposure to chemical contaminants within the Columbia River Basin, this risk assessment evaluates only part of one pathway - exposure from consumption of fish. Data on contaminant levels in fish were gathered and potential exposures through fish consumption estimated, but the source of these contaminants and their subsequent movement through the environment into fish were not evaluated.

4.3 Quantification Of Exposure

To characterize the risk from consuming fish, an estimate of the amount of contaminant ingested from eating fish must be estimated. This exposure is estimated using Equation 4-1:

(Equation 4-1)

$$ADD = \frac{C \times CF \times IR \times EF \times ED}{BW \times AT}$$

where:

ADD	=	Average daily dose of a specific chemical (mg/kg-day)
C	=	Chemical concentrations in fish tissue (mg/kg)
CF	=	Conversion factor (kg/g)
IR	=	Ingestion (consumption) rate (g/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Averaging time for exposure duration (days)

As can be seen from this equation, an individual's exposure (average daily dose) depends upon several factors including: the concentrations of contaminants in fish; the amount of fish eaten; how often and how long fish are eaten; and body weight. Because this exposure occurs over time, the total exposure is divided by a time period of interest (the averaging time) to obtain an average exposure rate per unit time. When this average rate is expressed as a function of body weight, the resulting exposure rate is referred to as the average daily dose (ADD) expressed in milligrams of a chemical taken into the body per kilogram body weight per day (mg/kg/day).

As can be seen from Equation 4-1, one individual's exposure may differ from another's because of differences in these exposure factors. Thus, in a population of fish consumers, a wide range of individual exposures would be expected, from those individuals who have little exposure (e.g., because they don't eat much fish and/or eat fish that have low contaminant concentrations) to those who have high exposure (e.g., because they eat highly contaminated fish and/or eat large amounts of fish). For this risk assessment, several of the exposure factors (fish ingestion rate, exposure duration, and body weight) were varied to estimate a possible range in exposures among individual fish consumers (adults and children). For example, the use of average exposure factors in Equation 4-1 is expected to result in a daily dose that is more representative of the average exposure in a population while the use of a mixture of average and high-end exposure factors is more representative of those members of the population who have higher exposures. The selection of these exposure parameters was made to ensure that, at a minimum, cancer risks and non-cancer health impacts for those individuals with more average exposures as well as those with much higher exposures are calculated.

For this risk assessment, exposures were estimated for adults and children for both the general public and CRITFC's member tribes. The exposure values selected for estimating exposure with Equation 4-1 are shown in Table 4-1 (non-cancer) and Table 4-2 (cancer) and are discussed in more detail in Sections 4.4 through 4.9. The same tissue chemical concentrations are used to

estimate exposure for all of the populations, for cancer and non-cancer endpoints. However, other exposure parameters differ. For example, cancer risks are estimated for lifetime exposures only. Therefore, only exposure parameters for adults are included in Table 4-2. Four different fish ingestion rates were used for adults (for estimating both cancer risks and non-cancer hazards) and four for children (for estimating non-cancer hazards). These rates were based on two surveys discussed in Section 4.5. The body weights used for each population correspond to the age of the person for which consumption data was obtained in the two fish consumption surveys. For adults for both cancer and non-cancer endpoints, a 70 kilogram body weight is used. However, data were collected on children of different ages in the two surveys (children less than 15 years of age for the survey used for the general public and children less than 6 years of age for the survey used for CRITFC's member tribes), so the body weights also differ.

Table 4-1. Exposure parameters used to calculate average daily dose for assessing noncarcinogenic health effects for potentially exposed populations

		Potentially Exposed Population			
		General Public		CRITFC's member tribes	
Exposure Parameter	**Abbreviation**	**AFC**	**HFC**	**AFC**	**HFC**
Tissue chemical concentration	C	Average	Average	Average	Average
Ingestion rate of fish tissue (g/day)	IR				
Adults		7.5[a]	142.4[b]	63.2[c]	389[d]
Children <15		2.83[a]	77.95[b]	–	–
Children <6		–	–	24.8[c]	162[d]
Exposure frequency (days/yr)	EF	365	365	365	365
Exposure duration (yrs)	ED				
Adults		30[e]/70[f]	30[e]/70[f]	30[e]/70[f]	30[e]/70[f]
Children <15		15	15	–	–
Children <6		–	–	6	6
Body weight (kg)	BW				
Adults		70[g]	70[g]	70[g]	70[g]
Children <15		30[h]	30[h]	–	–
Children <6		–	–	15[i]	15[i]
Averaging time (days)	AT				
Adults		10,950/ 25,550	10,950/ 25,550	10,950/ 25,550	10,950/ 25,550
Children <15		5,475	5,475	–	–
Children <6		–	–	2,190	2,190

AFC - average fish consumption ; HFC - high fish consumption

[a] Mean U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA, 2000b).

[b] 99th percentile U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA ,2000b).

[c] Mean consumption rate for fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin (CRITFC, 1994)

[d] 99th percentile consumption rate for fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin (CRITFC, 1994).

[e] 90th percentile length of time an individual stays at one residence (USEPA, 1997b)

[f] Average life expectancy of the general public (USEPA, 1989).

[g] Average body weight for adults (male and female) in the general public (USEPA, 1989).

[h] Average body weight for children of both sexes of age 6 months to 15 years in the general public (USEPA, 1997c). Corresponds to ingestion rate data for children taken from USEPA 2000b.

[i] Average body weight for children of both sexes frm the age of 6 months through 5 years in the general public (USEPA, 1997c). Corresponds to ingestion rate data for children in CRITFC, 1994.

Table 4-2. Exposure parameters used to calculate average daily dose for assessing carcinogenic risks for potentially exposed populations.

		Potentially Exposed Population			
		General Public		CRITFC's member tribes	
Exposure Parameter	**Abbreviation**	**AFC**	**HFC**	**AFC**	**HFC**
Tissue chemical concentration	C	Average	Average	Average	Average
Ingestion rate of fish tissue (g/day)	IR				
Adults		7.5[a]	142.4[b]	63.2[c]	389[d]
Exposure frequency (days/yr)	EF	365	365	365	365
Exposure duration (yrs)	ED				
Adults		30[e]/70[f]	30[e]/70[f]	30[e]/70[f]	30[e]/70[f]
Body weight (kg)	BW				
Adults		70[g]	70[g]	70[g]	70[g]
Averaging time (days)	AT	25,550	25,550	25,550	25,55

AFC - average fish consumption ; HFC - high fish consumption

[a] Mean U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA, 2000b).

[b] 99th percentile U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA ,2000b).

[c] Mean consumption rate for fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin (CRITFC, 1994)

[d] 99th percentile consumption rate for fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin (CRITFC, 1994).

[e] 90th percentile length of time an individual stays at one residence (USEPA, 1997b)

[f] Average life expectancy of the general public (USEPA, 1989).

[g] Average body weight for adults (male and female) in the general public (USEPA, 1989).

4.4 Exposure Point Concentrations (Chemical Concentrations in Fish)

The exposure point concentrations for this risk assessment are the average chemical concentrations in uncooked fish tissue. Exposure point concentrations for fish tissue or shellfish are commonly based on average concentrations (USEPA, 1989). The average concentrations are assumed to be representative of the chemical concentrations to which fish consumers would most likely be exposed over the long exposure durations being used in this risk assessment.

Ideally, the concentrations used as the exposure point concentrations for an individual should represent the average chemical concentrations in fish found at study sites where fish are collected for consumption during the exposure duration. Fishing study site preferences within the Columbia River Basin are available for members of the Nez Perce, Umatilla, Yakama, and Warm Springs Tribes (CRITFC, 1994); these preferences were used in designing the sampling plan for this study. However, similar information is not available for the general public. To try and maximize the information conveyed in this risk assessment and allow individuals to assess their own risks based on their fishing practices, the data for each fish species were pooled by (1) study

site - all replicate samples for a given fish species and tissue type collected at a study site were averaged to produce a "study site" average and (2) basin-wide all samples for a given fish species and tissue type collected in the Columbia River Basin during this study were averaged to calculate the "basin-wide" averages. The calculation of these study site and basin-wide averages were previously discussed in Section 1.

4.5 Fish Ingestion Rates

4.5.1 Fish Ingestion Rates for the General Population

Three fish consumption surveys were completed in the Columbia River Basin: two for the Willamette River, Oregon and one for Lake Roosevelt, Washington (EVS, 1998; Adolfson Associates, Inc., 1996; WDOH, 1997). A brief description of these surveys is presented in this section. Although these three surveys do not provide fish ingestion rates that can be used for this risk assessment, they do provide useful information on the species of fish consumed in different parts of the basin and on the parts of the fish that are eaten.

In 1998, EVS Environment Consultants (EVS, 1998) conducted a qualitative fish consumption survey for a 45-mile stretch of the Willamette River extending downstream from Wheatland Ferry to the Willamette Falls near Oregon City, Oregon. Information on fish consumption was obtained by conducting phone interviews with individuals representing various community centers, fishing guide services, ethnic associations, fishing-related government agencies and businesses. The survey indicated that anglers are consuming bullhead, carp, sucker, bass, northern pikeminnow, crappie, bluegill, trout, white sturgeon, lamprey, salmon, and steelhead from this section of the Willamette River. All respondents indicated that muscle tissue was the most commonly consumed portion of the fish, although some respondents indicated that the skin, eggs, eyes, and the entire fish were being consumed (EVS, 1998).

In 1995, Adolfson Associates (Adolfson Associates, Inc., 1996) conducted a fish consumption survey by interviewing anglers along the Columbia Slough and Sauvie Island at the mouth of the Willamette River, Oregon This survey found that Caucasians made up the majority of individuals consuming fish from these locations. The ethnic descent of Columbia Slough anglers was 47% Caucasians of eastern European descent, 22% Hispanic, 19% African American, 8% Caucasian (excluding eastern Europeans), and 3% Asian. The most commonly caught fish was carp, followed by yellow perch and banded sculpin. The ethnic descent of Sauvie Island anglers was 67% Caucasian (excluding eastern Europeans), 16% Asian, 8% African American, and 2% Hispanic. The most commonly caught fish was yellow perch, followed by brown bullhead, northern pikeminnow, starry flounder, and white sturgeon. Anglers from both locations indicated the most commonly consumed portion of fish was muscle tissue.

In 1994, the Washington State Department of Health (WDOH, 1997), in cooperation with the Spokane Tribe of Indians, conducted a fish consumption survey of anglers fishing within Lake Roosevelt, Washington, a 151-mile stretch of water extending upstream from the Grand Coulee Dam on the Columbia River to the United States-Canada border. Fish consumption data were collected using a survey form and from creel surveys. The majority of anglers surveyed consisted

of individuals who repeatedly fish from Lake Roosevelt. Surveyed anglers were mainly male (90%), Caucasian (97%), and over fifty years of age (60%). The most frequently consumed species were rainbow trout, followed by walleye, kokanee, and bass. The average annual number of fish meals consumed by respondents was 42 meals per year. Assuming a typical meal size of 8 ounces, this average consumption rate corresponds to a daily fish consumption rate of 26 g/day. Fillets were the primary portion of the fish consumed; few anglers consumed fish skin, eggs, or fish head.

Because these three studies provide only a limited amount of information on fish consumption rates for the general public within the Columbia River Basin, a recent EPA fish consumption report (USEPA, 2000b) was used to select the fish consumption rates for this risk assessment that may be representative of adults and children within the general public that consume average and high amounts of fish. The fish consumption rates reported by EPA are based on data collected from the combined 1994, 1995, and 1996 Continuing Survey of Food Intakes by Individuals (CSFII), conducted annually in all 50 states by the United States Department of Agriculture. The CSFII was conducted by interviewing over 15,000 respondents according to a stratified design that accounted for geographic location, degree of urbanization, and socioeconomics. Eligibility for the survey was limited to households with gross incomes at or less than 130% of the federal poverty guidelines. The mean daily average per capita (fish consumers and non-consumers) fish consumption rates of freshwater and estuarine fish (uncooked) reported by EPA (USEPA, 2000b) for adults (7.5 g/day) and children (14 years of age and younger, 2.83 g/day) were selected to be representative of average fish consumption by the general public within the Columbia River Basin. The 99th percentile per capita fish consumption rates of freshwater and estuarine fish (uncooked) reported by EPA (USEPA, 2000b) for adults (142.4 g/day) and children (14 years of age and younger, 77.95 g/day) were selected to be representative of high fish consumption by the general public within the Columbia River Basin.

4.5.2 Fish Ingestion Rates for CRITFC's Member Tribes

During 1991-1992, CRITFC conducted a comprehensive survey of fish consumption by members of the Nez Perce, Umatilla, Yakama, and Warm Springs Tribes that possess fishing rights to harvest anadromous fish and resident fish species originating in streams and lakes flowing throughout the Columbia River Basin (CRITFC, 1994). The survey data were collected by interviewing a total of 513 adult tribal members. Information obtained in this survey included age-specific fish consumption rates, the fish species and parts of the fish consumed, and the methods used to prepare the fish for consumption. Salmon and steelhead were consumed by the largest number of adult respondents followed by trout, lamprey and smelt. The survey determined that the average consumption rate of fish by adults and children (5 years of age and younger) who consume fish was 63.2 g/day and 24.8 g/day, respectively. The 99th percentile fish consumption rates of adults and children (5 years of age and younger) who consume fish was 389 g/day and 162 g/day, respectively. The average and 99th percentile fish consumption rates were selected as representative values for average and high fish consumption by CRITFC's member tribes.

The fish consumption survey conducted by CRITFC (1994) showed that fish consumption by

CRITFC's member tribes is considerably higher than that of the general public. The average and 99th percentile fish consumption rates for adults in CRITFC's member tribes are higher by factors of 8.4 and 2.7, respectively, than the corresponding per capita fish consumption rates reported for the general public by EPA (USEPA, 2000b). It should be noted that Harris and Harper (1997) have suggested that a fish consumption rate of 540 g/day represents a reasonable subsistence fish consumption rate for CRITFC's member tribes who pursue a traditional lifestyle. The value of 540 g/day was based on the authors' review of several non-subsistence Native American studies, two subsistence studies, and personal interviews (by the authors or others) of members of the Umatilla and Yakama Tribes. This value of 540 g/day is 1.4 times the 99th percentile fish consumption rate reported by CRITFC (1994) which is used as the high-end consumption rate for CRITFC's member tribes in this risk assessment.

Some individuals may find it difficult to assess their fish consumption in terms of grams per day. Two other common ways to present this information is in terms of 8-ounce fish meals over some period of time or in terms of pounds per year. An 8-ounce meal size is the value recommended by EPA (USEPA, 2000a) for fish meals. This meal size was also the most commonly selected (48.5%) serving size for adult fish meals based on the CRITFC (1994) survey of its member tribes.

Table 4-3 shows the fish consumption rates used in this risk assessment expressed in different units.

Table 4-3. Fish consumption rates expressed in alternative units.

	Consumption Rate Units		
Target Population	**g/day**	**8-oz Meals**	**Lbs/yr**
General public - average fish consumption			
Adults	7.5[a]	12 meals/year	6.0
Children <15	2.83[a]	5 meals/year	2.3
General public - high fish consumption			
Adults	142.4[b]	19 meals/month	114.6
Children <15	77.95[b]	11 meals/month	62.7
CRITFC's member tribes - average fish consumption			
Adults	63.2[c]	2 meals/week	50.8
Children <6	24.8[c]	40 meals/year	20.0
CRITFC's member tribes - high fish consumption			
Adults	389[d]	12 meals/week	313
Children <6	162[d]	5 meals/week	131

[a] Mean U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA, 2000b).
[b] 99th percentile U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA , 2000b).
[c] Mean consumption rate for fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin (CRITFC, 1994)
[d] 99th percentile consumption rate for fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin (CRITFC, 1994).

As discussed in Section 1 of this report, a small number of egg samples were collected for some

of the anadromous fish species. There are no studies for the Columbia River Basin with quantitative ingestion rates for eggs. Therefore, a risk characterization for eggs was not included in the Risk Characterization Section (Section 6) of this report. However, an example risk characterization for eggs is presented in the Uncertainty Section (Section 10). This example for eggs is very uncertain but serves as a useful comparison to the results for fish tissue.

4.6 Exposure Frequency

An exposure frequency of 365 days per year was assumed for calculation of the average daily dose. While not all fish species analyzed for this risk assessment can be collected by anglers throughout the year, an exposure frequency of 365 days per year was assumed for all fish species since anglers might catch and freeze fish for later consumption or receive fish for consumption from other anglers.

4.7 Exposure Duration

The exposure duration is the length of time over which exposure occurs at the concentrations and ingestion rates specified by the other parameters in Equation 4-1. Specific information on the length of time over which the general public or CRITFC's member tribes may be consuming fish from the Columbia River Basin is not available. Therefore estimates of exposure duration were made for this risk assessment.

4.7.1 Adults

Two exposure durations, 30 years and 70 years, were assumed for calculations of the adult average daily intake in this risk assessment. Thirty years is the national 90th percentile length of time that an individual stays at one residence (USEPA, 1997b). This value is recommended by EPA (USEPA, 1989) as a reasonable maximum exposure duration when assessing the potential health risks for a residential exposure scenario.

A 70-year exposure duration was selected to assess the potential health risk of a lifetime exposure to chemicals detected in fish tissue. The average life expectancy of the general population in the United States is 72 years for males and 79 years for females (USEPA, 1997c). EPA (USEPA, 1997c) suggests that 75 years is an appropriate value to reflect the average life expectancy of the general population. A value of 70 years was selected as a lifetime exposure duration in this risk assessment because this value has been commonly used in other regional human health risk assessments of fish consumption (e.g., Tetra Tech, 1996; EVS, 2000) to represent the exposure duration for those individuals (e.g., tribal members) who fish from one area their entire life. In addition, since a 70-year lifetime is used to derive cancer slope factors (USEPA, 2000c), the use of 70 years avoids the necessity of having to adjust the cancer slope factors used in this risk assessment.

4.7.2 Children

An exposure duration of 15 years was used to estimate the average daily dose for children in the general public. This exposure duration was selected for children because it corresponds to the age range for which the fish consumption rate data were developed for children in the CSFII Survey (USEPA, 2000b).

An exposure duration of 6 years was used to estimate the average daily dose of children for CRITFC's member tribes. This exposure duration was selected because it corresponds to the age range for which fish consumption data were reported by CRITFC (1994) for children up to 6 years of age.

4.8 Body Weight

The value for body weight in Equation 4-1 is the average body weight over the exposure period. Information on the body weights of the individuals reported in the CRITFC consumption survey (CRITFC, 1994) and the CSFII consumption survey (USEPA, 2000b) were not available, therefore data from the studies, discussed in the following sections, were used.

4.8.1 Adults

Existing EPA guidance (USEPA, 1989, USEPA, 2000a) recommends the use of a body weight of 70 kg (kilograms) to calculate adult exposures. A 70 kg adult body weight is assumed for the derivation of cancer slope factors in IRIS. However, a more recent survey data of the population in the United States suggests that a body weight of 71.8 kg may be more appropriate for adults (USEPA, 1997c).

For this risk assessment, a 70 kg body weight was assumed for adults because its use is consistent with EPA risk assessment guidance (USEPA, 2000f), it avoids the necessity of having to adjust cancer slope factors to accommodate the 71.8 kg average body weight, and allows for comparisons with other regional human health risk assessments of fish consumption that also used 70 kg as the adult body weight.

4.8.2 Children

A body weight of 30 kg was used to calculate the average daily dose of children in the general public. This body weight corresponds to the average weight of female and male children ages 6 months through age 14 (USEPA, 1997c). Six months through the age of age 14 is the age group for which fish consumption data were collected in the CSFII Survey.

A body weight of 15 kg was used to calculate the average daily dose of children for the Columbia River Basin tribes. This body weight corresponds to the average weight of female and male children ages 6 months through age 5 (USEPA, 1997c). Six months through age 5 years is the age group for which fish consumption data were collected in the CRITFC fish consumption survey.

4.9 Averaging Time

As discussed earlier, exposure to contaminants in fish occurs over time. Therefore the total exposure is divided by the time period of interest (the averaging time) to obtain an average exposure rate per unit time. When this average rate is expressed as a function of body weight, the resulting exposure rate is referred to as the average daily dose (ADD) expressed in milligrams of a chemical taken into the body per kilogram body weight per day (mg/kg/day).

The averaging time selected depends upon the type of toxic effect being assessed. When evaluating exposures to non-cancer effects, exposures (dose) are calculated by averaging dose over the period of exposure (for this risk assessment - 30 or 70 years for adults; 6 or 15 years for children). Since the averaging time (AT) is always the same as the time period over which exposure occurs for non-cancer effects, exposure duration (ED), the exposure (dose) in mg/kg/day is the same for both exposure durations within a target populations (e.g. the same for both 30 and 70 years exposure duration for general public adults).

For evaluating cancer risks for adults, exposures are calculated by prorating the total dose over a lifetime (70 years). The exposures calculated for cancer risk assuming 30 or 70 years exposure duration are different from each other because the averaging time is always a lifetime or 25,550 days, but the exposure durations assumed for this report for adults are either 30 (10,950 days) or 70 years (25,550 days). Thus, in this report, cancer risks for both exposure durations (30 and 70 years) are presented.

4.10 Multiple-Species Diet Exposures

The cancer risk and non-cancer hazards that are discussed in most of Section 6 assume that people eat only one species of fish. For example, for estimating the cancer risk from consuming white sturgeon, it is assumed that the adults in the general public, with high fish consumption (142.4 g/day), consume 142.4 grams a day of white sturgeon for either 30 years or 70 years.

However, it is likely that many individuals consume more than one species of fish from the Columbia River Basin. When an individual consumes multiple fish species, additional exposure information is needed on the relative amounts of different species in that individual's diet to obtain an estimate of the individual's potential overall health risk. Because fish consumption practices, including the types and amounts of fish eaten, can vary greatly among individuals, within populations because of differences in age, gender, cultural practices, and/or socioeconomic status, it is difficult to generalize about the potential risk of an individual diet that includes the consumption of multiple species. This section includes the methods and the assumptions used in the example of a multiple-species diet. This example is intended to assist individuals to use the data for individual fish species presented in this report to estimate their own risks when consuming multiple species.

The example selected to illustrate the risk associated with consuming multiple species is based on information obtained during the 1991-1992 survey of fish consumption by members of the Nez Perce, Umatilla, Yakama, and Warm Springs Tribes (CRITFC, 1994). The survey included 513

adult participants. The percentage of these adults that consumed 10 fish species were also presented in this survey (CRITFC, 1994; Table 17). These percentages are included in this section in Table 4-4, column A. To simplify the calculations, the responses from the CRITFC survey for fall chinook salmon, spring chinook salmon, coho salmon, and steelhead were combined into one category, salmon. To estimate the hypothetical diet, it was assumed that the data in the CRITFC survey on percentages of adults consuming different fish species could be used to estimate the percent that each fish species contributes to the hypothetical diet. Table 4-4, Column B, shows the percentage of the diet assumed for each fish species. Each species value in Column B was calculated by dividing the percentage of each fish species consumed (based on the CRITFC study and shown in Column A) by the sum of the percentages for all species in Column A. For example, the value of 27.7% shown for salmon in Table 4-4 (Column B) was obtained by dividing the percentage of adults that consume salmon (92.4 in Column A) by the sum of the percentages of consumption for all species (333.5 in Column A) and multiplying the result by 100 to express the fraction as a percentage:

(*Equation 4-2*)

$$\textit{Percent of diet composed of salmon} = \frac{\textit{percentage of adults that consume salmon}}{\textit{sum of the percentages for all species}} \times 100$$

$$27.7\% = \frac{92.4}{333.5} \times 100$$

In Table 4-4, a consumption rate of 63.2 g/day (the average ingestion rate reported for adults in CRITFC's member tribes (CRITFC, 1994), is used along with the percentages of fish in the hypothetical diet to calculate the consumption rates for each species in the hypothetical multiple diet of an adult in CRITFC's member tribes with average fish consumption. Consumption rates for each species were calculated by multiplying 63.2 g/day by the percentage assumed in the hypothetical diet for that species. For example, the consumption rate of 17.5 g/day shown for salmon in Table 4-4 (Column C) was obtained by multiplying the total average consumption rate (63.2 g/day) for adults in CRITFC's member tribes by the percent that salmon was calculated to represent (27.7%) in this multiple-species diet.

(Equation 4-3)

$$\textit{Consumption rate for salmon (g/day)} = \textit{Percent of hypothetical diet composed of salmon} \times \textit{Average adult ingestion rate for all species}$$

$$17.5 \text{ g/day} = 27.7\% \times 63.2 \text{ g/day}$$

This multiple-species diet methodology was used to estimate exposure and to calculate cancer risks and non-cancer hazards for adults in the general public and CRITFC member tribes in Section 6.2.5 for both the average and high fish ingestion rates. The hypothetical diet of multiple-species based on the CRITFC fish consumption study was used for all of the adult populations.

The exposure due to ingestion of each species in the hypothetical diet was calculated by using the same exposure parameters described for adults in Tables 4-1 and 4-2 except that the fish consumption rates for the multiple-species diet scenario replaced those in the tables. For the adults in CRITFC's member tribes with an average fish consumption rate, those ingestion rates in Table 4-4 (Column C) were used. For the other 3 adult populations assessed (high fish consumption rates for adults in CRITFC's member tribes; average and high fish consumption rates for general public adults), species specific consumption rates were calculated using the multiple diet method just described but using total fish consumption rates for that population and the hypothetical multiple-species diet shown in Table 4-4. Exposure for the hypothetical mixed diet is the sum of all of the exposures calculated for each of the eight species that had ingestion rates calculated in Table 4-4.

Table 4-4. Description of the methodology used to calculate exposure for a multiple-species diet.

Species	A Percentage of Adults that Consume Species	B Percentage of Hypothetical Diet	C Consumption Rate[c] (grams/day)
Salmon[a]	92.4	27.7	17.5
Rainbow trout	70.2	21.0	13.3
Mountain whitefish	22.8	6.8	4.3
Smelt	52.1	15.6	9.9
Pacific lamprey	54.2	16.3	10.3
Walleye	9.3	2.8	1.8
White sturgeon	24.8	7.4	4.7
Sucker	7.7	2.3	1.5
Totals	333.5[b]	100.0	63.2

[a] This category includes spring chinook salmon, fall chinook salmon, steelhead and coho salmon.
[b] Although shad and pikeminnow were included in the CRITFC fish consumption survey (CRITFC ,1994), this total does not include values for these species because these two species were not sampled in this study.
[c] a consumption rate of 63.2 g/day (the average ingestion rate reported for adults in CRITFC's member tribes (CRITFC, 1994), is used along with the percentages of fish in the hypothetical diet to calculate the consumption rates for each species

5.0 Toxicity Assessment

The toxicity assessment for a chemical is done in two steps. The first step, hazard identification, summarizes and weighs the available evidence regarding a chemical's potential to cause adverse health effects, such as cancer, birth defects, or organ damage. The second step, dose-response evaluation, provides an estimate of the relationship between the extent of exposure to the contaminant and the likelihood of these adverse effects occurring. As part of the dose-response assessment, toxicity values - reference doses (RfD) and cancer slope factors (CSFs) - are derived. These toxicity factors are used with the exposures calculated using methods described in Section 4 to estimate cancer risks and non-cancer hazards.

For most environmental contaminants of concern, EPA has already performed the toxicity evaluation and has made the results available in databases. For the risk characterization in this section, all of the toxicity information, including the reference doses and cancer slope factors, was obtained from three EPA toxicity databases. Information was preferentially obtained from IRIS (USEPA, 2000c). If data were not available in IRIS, they were obtained from the fiscal year 1997 Health Effects Assessment Summary Tables (HEAST) (USEPA, 1997d), and finally, from the EPA National Center for Environmental Assessment (NCEA).

A toxicity value has not been developed for all chemicals analyzed in this study. Chemicals currently without toxicity values are listed in Table 5-1. The potential health risks associated with exposure to these chemicals were not evaluated.

Table 5-1. Chemicals without oral reference doses and cancer slope factors. (Source: IRIS, NCEA, USEPA, 2000c; USEPA, 1997d)

Acenaphthylene	1-methyl-Naphthalene
alpha-Chlordene	2-methyl-Naphthalene
Benzo(ghi)perylene	4-Bromophenyl-Phenylether
DDMU	4-Chloroguaiacol
delta-HCC	4-Chlorophenyl-Phenylether
Dibenzofuran	3,4-Dichloroguaiacol
gamma-Chlordene	4-Chloro-3-methylphenol
Pentachloroanisole	4,5-Dichloroguaiacol
Phenanthrene	4,6-Dichloroguaiacol
Retene	3,4,5-Trichloroguaiacol
Tetrachloroguaiacol	3,4,6-Trichloroguaiacol
	4,5,6-Trichloroguaiacol

Of the 23 chemicals listed in Table 5-1, only two, 2-methyl naphthalene and pentachloroanisole, were detected in fish at greater than a 10% frequency. Table 1-4 in Section 1 shows both the detected and non-detected chemicals in this study. It should also be noted that although lead does not have toxicity values (RfD, CSF), lead toxicity is well characterized and is discussed in detail in Section 7.

The remainder of this section is divided into three parts. First, the methods used to assess toxicity data and develop reference doses for non-cancer effects are summarized in Section 5.1. Next, the methods used to assess carcinogenicity data and develop cancer slope factors are summarized in

Section 5.2. Finally, those chemicals for which unique assumptions and/or methods were used to estimate the study site and basin-wide averages due to toxicological considerations are discussed in Section 5.3.

5.1 Summary of Toxicity Assessment for Non-Cancer Health Effects

Summaries of the available toxicity information (e.g., results of animal tests and/or human occupational studies) for each chemical are provided in IRIS, HEAST or by NCEA. For those chemicals that were analyzed for in fish in this study and that have toxicity values, a summary of the types of non-cancer effects caused by that chemical is provided in Table 5-2.

In Table 5-2, the effects that can potentially result from exposure to each of these chemicals are designated with a check or a closed circle. For most chemicals, there is more than one type of non-cancer health effect (e.g., effects on metabolism, effects on the immune system) that can result from exposure to that chemical. The number of effects seen and the severity of a given effect depend upon the level of exposure to that chemical, with both the number and severity of effects usually increasing as exposure increases.

The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of the daily exposure to the human population, including sensitive sub-populations, that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 2000c). To derive the RfD, all available studies are first reviewed. If adequate human data are available, this information is used as the basis of the RfD. Otherwise, animal studies are the basis of the RfD. If several animal studies are available, the study on the most sensitive species (the species showing the toxic effect at the lowest dose) is selected as the critical study for the basis of the RfD. The effect associated with the lowest dose which resulted in an observed adverse effect is referred to as the "critical toxic effect". After the critical study and critical toxic effect have been selected, the experimental exposure level at which no adverse effect is demonstrated (the no-observable-adverse-effect-level) for that effect is then defined. The no-observable-adverse-effect-level is used as the basis for deriving the RfD and is in part based upon the assumption that if the critical toxic effect is prevented then all toxic effects will be prevented. For example, for total Aroclors, the RfD was based upon a rhesus monkey study. This study was designated as the critical study and the RfD is based on the critical toxic effects on the immune system that were found in the study. For some chemicals (e.g., methyl mercury), the RfD may be based on more than one critical toxic effect (central nervous system and developmental/reproductive effects). Table 5-2 also contains information on critical health endpoints used to derive the RfD as well as other adverse health effects.

To develop the RfD, the no-observable-adverse-effect-level, or the lowest-observed-adverse-effect-level if no-observable-adverse-effect-level can be determined from the studies, is divided by uncertainty factors and a modifying factor. These factors, which usually consist of multiples of 10 or lower, are applied to account for the different areas of uncertainty and variability that are inherent in the toxicological data. They include:

- An uncertainty factor to account for variations in the sensitivity of the general population. This factor is intended to protect sensitive subpopulations (e.g., the elderly and children).

- An uncertainty factor to extrapolate from animals to humans when animal data is used.

- An uncertainty factor to account for the uncertainty if only a lowest-observed-adverse-effect-level instead of a no-observable-adverse-effect-level is available.

- An uncertainty factor if data from only short term rather than lifetime studies are available.

- A modifying factor to account for additional uncertainties not already addressed (e.g., if there is a lack of data on reproductive or developmental effects in the experimental data).

For each chemical with non-cancer effects, Table 5-3 presents the oral reference dose for that chemical, the confidence in the reference dose, the uncertainty factors and the modifying factor associated with the reference dose, and the toxic effect from the critical study that the reference dose was based upon. For many chemicals, both oral and inhalation reference doses have been developed and are included in EPA toxicity databases. However, because the exposures assessed in this study result from ingestion of fish, only oral reference doses were used.

TABLE 5-2. CHEMICALS CONTRIBUTING TO NON-CANCER HAZARD INDICES (WITH TOXIC EFFECTS OF EACH CHEMICAL DENOTED BY U AND Ž)

GROUP	ANALYTE	Metabolism	Blood and blood formation	Immune system	Cardiovascular	Kidney	Liver	Central Nervous System	Reproduction/development	Gastrointestinal or intestinal lesions	Argyria	Thyroid	Other	Adrenal gland	Clinical signs	Selenosis	Hyperpigmentation/keratosis	No clear critical toxicity endpoint
Metals	Aluminum							(U)										
	Antimony	U			Ž													
	Arsenic				U	Ž		Ž									U	
	Barium				(U)	(U)												
	Beryllium									U								
	Cadmium					U		Ž										
	Chromium (VI)									(U)								
	Cobalt		(U)															
	Copper																	(U)
	Manganese							U										
	Mercury							U	U									
	Nickel	U											Ž					
	Selenium		Ž				U	Ž	Ž							U		
	Silver					Ž	Ž				U							
	Thallium	Ž			Ž	Ž	U	Ž					Ž					
	Vanadium																	(U)
	Zinc	U																
Semivolatiles	2-Chloronaphthalene						U						Ž					
	2,4-Dinitrotoluene		U				U	U										
	2,6-Dinitrotoluene		U			U	U	U										
	1,2,4-Trichlorobenzene													U				
	Acenaphthene						U											
	Anthracene																	(U)
	Benzene, 1,2-dichloro-																	(U)
	Benzene, 1,3-dichloro-						(U)					(U)						

TABLE 5-2. CHEMICALS CONTRIBUTING TO NON-CANCER HAZARD INDICES (WITH TOXIC EFFECTS OF EACH CHEMICAL DENOTED BY U AND Ž)

GROUP	ANALYTE	Metabolism	Blood and blood formation	Immune system	Cardiovascular	Kidney	Liver	Central Nervous System	Reproduction/development	Gastrointestinal or intestinal lesions	Argyria	Thyroid	Other	Adrenal gland	Clinical signs	Selenosis	Hyperpigmentation/keratosis	No clear critical toxicity endpoint
	Benzene, 1,4-dichloro-						(U)		(U)									
	bis(2-Chloroisopropyl)ether		U															
	Fluoranthene		U			U	U											
	9H-Fluorene		U															
	Hexachloroethane					U	Ž		Ž									
	Hexachlorobutadiene					U												
	Naphthalene	U																
	Nitrobenzene		U			U	U							U				
	Pyrene					U												
Guaiacols/ Phenols	2-Chlorophenol						Ž		U									
	2,3,4,6-Tetrachlorophenol						U											
	2,4-Dichlorophenol			U			Ž	Ž										
	2,4-Dimethylphenol		U					Ž							U			
	2,4,5-Trichlorophenol					U	U											
	Pentachlorophenol	Ž				U	U	Ž										
	Phenol	Ž				Ž			U									
Pesticides	Aldrin						U	Ž										
	Chlordane (total)	Ž					U	Ž										
	DDT [a]						U	Ž	Ž									
	Endosulfan sulfate	U			U	U		Ž	Ž									
	Heptachlor						U	Ž					Ž					
	Heptachlor epoxide						U	Ž	Ž									
	Hexachlorobenzene						U	Ž					Ž					
	gamma-HCH					U	U	Ž										
	Mirex				Ž	Ž	U	Ž				U						

TABLE 5-2. CHEMICALS CONTRIBUTING TO NON-CANCER HAZARD INDICES (WITH TOXIC EFFECTS OF EACH CHEMICAL DENOTED BY U AND Ž)

GROUP	ANALYTE	Metabolism	Blood and blood formation	Immune system	Cardiovascular	Kidney	Liver	Central Nervous System	Reproduction/development	Gastrointestinal or intestinal lesions	Argyria	Thyroid	Other	Adrenal gland	Clinical signs	Selenosis	Hyperpigmentation/keratosis	No clear critical toxicity endpoint
PCBs	Total Aroclors [b]			**U**			**Ž**	**Ž**	**Ž**									

U - Chronic oral reference dose for this chemical is based on this health endpoint (critical effect). All chemicals with a **U** for a given health endpoint were summed to obtain an estimate of the hazard index.

(**U**) - Chronic oral reference dose has been developed for this chemical but the critical effect used is not clear. Although hazard quotients were calculated for these chemicals and summed into the total hazard index, these chemicals were not summed into endpoint-specific hazard indices.

Ž - Other observed health endpoints

[a] Comprised of DDE, DDD, and DDT.

[b] For each species, total Aroclors is the sum of detected Aroclors, which includes at least one of the following: Aroclor 1242, Aroclor 1254, and Aroclor 1260.

Table 5-3. Oral reference doses (RfDs) used in this assessment, including the level of confidence in the RfD, uncertainty factors (UF) and modifying factor (MF) used to develop the RfD, and the toxic effect(s) from the critical study that the RfD was based upon.

Chemical	Oral RfD (mg/kg-day)	Confidence	UF/MF	Critical Effect	Source
1,2,4-Trichlorobenzene	1.0×10^{-2}	Medium	1000/1	Increased adrenal weight	USEPA, 2000c
2,3,4,6- Tetrachlorophenol	3.0×10^{-2}	Medium	1000/1	Increased liver weights and centrilobular hypertrophy	USEPA, 2000c
2,4,5-Trichlorophenol	1.0×10^{-1}	Low	1000/1	Liver and kidney pathology	USEPA, 2000c
2-Chloronaphthalene	8.0×10^{-2}	Low	3000/1	Dyspnea, abnormal appearance, liver enlargement	USEPA, 2000c
2-Chlorophenol	5.0×10^{-3}	Low	1000/1	Reproductive effects	USEPA, 2000c
2,4-Dichlorophenol	3.0×10^{-3}	Low	100/1	Decreased delayed hypersensitivity response	USEPA, 2000c
2,4-Dimethylphenol	2.0×10^{-2}	Low	3000/1	Clinical signs (lethargy, prostration, and ataxia) and hematological changes	USEPA, 2000c
2,4-Dinitrotoluene	2.0×10^{-3}	High	100/1	Neurotoxicity, Heinz bodies and biliary tract hyperplasia	USEPA, 2000c
2,6-Dinitrotoluene	1.0×10^{-3}	-	3000	Mortality, neurotoxicity, Heinz bodies effects, methemoglobinemia, bile duct hyperplasia, and kidney histopathology	USEPA 1997e
Acenaphthene	6.0×10^{-2}	Low	3000/1	Hepatotoxicity	USEPA, 2000c
Aldrin	3.0×10^{-5}	Medium	1000/1	Liver toxicity	USEPA, 2000c
Aluminum	1.0	–	–	Minimal neurotoxicity	NCEA
Anthracene	3.0×10^{-1}	Low	3000/1	No treatment-related specific toxicological endpoints observed in mice at the doses administered in laboratory studies	USEPA, 2000c
Antimony	4.0×10^{-4}	Low	1000/1	Longevity, blood glucose, cholesterol	USEPA, 2000c
Total Aroclor [a]	2.0×10^{-5}	Medium	300/1	Ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger- and toenails; decreased antibody (IgG and IgM) response to sheep erythrocytes	USEPA, 2000c
Arsenic, inorganic [b]	3.0×10^{-4}	Medium	3/1	Hyperpigmentation/keratosis and possible vascular complications	USEPA, 2000c
Barium	7.0×10^{-2}	Medium	3/1	Hypertension and kidney effects	USEPA, 2000c
Benzene, 1,2-dichloro-	9.0×10^{-2}	Low	1000/1	None identified	USEPA, 2000c
Benzene, 1,3-dichloro-	9.0×10^{-4}	–	–	No identified critical toxicological endpoint	NCEA
Benzene, 1,4-dichloro-	3.0×10^{-2}	–	–	Liver and reproductive effects	NCEA
Beryllium	2.0×10^{-3}	Low to Medium	300/1	Small intestinal lesions	USEPA, 2000c
bis(2-Chloroisopropyl)ether	4.0×10^{-2}	Low	1000/1	Decrease in hemoglobin and possible erythrocyte destruction	USEPA, 2000c
Cadmium	1.0×10^{-3}	High	10/1	Significant proteinuria	USEPA, 2000c
Chlordane (total) [c]	5.0×10^{-4}	Medium	300/1	Hepatic necrosis	USEPA, 2000c
Chromium (VI)	3.0×10^{-3}	Low	300/3	Gastrointestinal effects	USEPA, 2000c
Cobalt	6.0×10^{-2}	–	–	Polycytemia - too many red blood cells	NCEA
Copper	3.7×10^{-2}	–	–	Unspecified	USEPA 1997e
DDT [d]	5.0×10^{-4}	Medium	100/1	Liver lesions	USEPA, 2000c

Table 5-3. Oral reference doses (RfDs) used in this assessment, including the level of confidence in the RfD, uncertainty factors (UF) and modifying factor (MF) used to develop the RfD, and the toxic effect(s) from the critical study that the RfD was based upon.

Chemical	Oral RfD (mg/kg-day)	Confidence	UF/MF	Critical Effect	Source
Endosulfan sulfate	6.0×10^{-3}	Medium	100/1	Reduced body wt. gain, increased incidence of marked progressive glomerulonephrosis in males	USEPA, 2000c
Fluoranthene	4.0×10^{-2}	Low	3000/1	Nephropathy, increased liver weights, hematological alterations, and clinical effects	USEPA, 2000c
Fluorene	4.0×10^{-2}	Low	3000/1	Decreased red blood cell, packed cell volume and hemoglobin	USEPA, 2000c
gamma-HCH (Lindane)	3.0×10^{-4}	Medium	1000/1	Liver and kidney toxicity	USEPA, 2000c
Heptachlor	5.0×10^{-4}	Low	300/1	Liver weight increases in males	USEPA, 2000c
Heptachlor epoxide	1.3×10^{-5}	Low	1000/1	Increased liver-to-body weight ratio in both males and females	USEPA, 2000c
Hexachlorobenzene	8.0×10^{-4}	Medium	100/1	Liver effects	USEPA, 2000c
Hexachlorobutadiene	2.0×10^{-4}	–	1000	Renal tube regeneration	USEPA 1997e
Hexachloroethane	1.0×10^{-3}	Medium	1000/1	Atrophy and degeneration of the renal tubules	USEPA, 2000c
Manganese	1.4×10^{-1}	–	1/1	CNS effects	USEPA, 2000c
Methylmercury [e]	1.0×10^{-4}	Medium	10/1	Developmental neurological abnormalities in human infants	USEPA, 2000c
Mirex	2.0×10^{-4}	High	300/1	Liver cytomegaly, fatty metamorphosis, angiectasis; thyroid cystic follicles	USEPA, 2000c
Naphthalene	2.0×10^{-2}	Low	3000/1	Decreased average terminal body weight in males	USEPA, 2000c
Nickel, soluble salts	2.0×10^{-2}	Medium	300/1	Decreased body and organ weights	USEPA, 2000c
Nitrobenzene	5.0×10^{-4}	Low	10,000/1	Hematologic, adrenal, renal and hepatic lesions	USEPA, 2000c
Pentachlorophenol	3.0×10^{-2}	Medium	100/1	Liver and kidney pathology	USEPA, 2000c
Phenol	6.0×10^{-1}	Low	100/1	Reduced fetal body weight	USEPA, 2000c
Pyrene	3.0×10^{-2}	Low	3000/1	Kidney effects (renal tubular pathology, decreased kidney weights)	USEPA, 2000c
Selenium	5.0×10^{-3}	High	3/1	Clinical selenosis, liver dysfunction	USEPA, 2000c
Silver	5.0×10^{-3}	Low	3/1	Argyria	USEPA, 2000c
Thallium [f]	9.0×10^{-5}	Low	3000/1	Increased levels of SGOT[g] and LDH[h]	USEPA, 2000c
Vanadium	7.0×10^{-3}	–	100	Unspecified	USEPA, 2000c
Zinc	3.0×10^{-1}	Medium	3/1	47% decrease in erythrocyte superoxide dismutase (ESOD) concentration in adult females after 10 weeks of zinc exposure	USEPA, 2000c

[a] For each fish species, total Aroclors is the sum of detected Aroclors, which includes at least one of the following: Aroclor 1242, Aroclor 1254, and Aroclor 1260. The toxicity value for Aroclor 1254 was used.

[b] Total arsenic was measured. Inorganic arsenic was assumed to represent 10% of the total arsenic concentration (see Section 5.3.3).

[c] Chlordane (total) is the sum of cis-chlordane, cis-nonachlor, oxychlordane, trans-chlordane, and trans-nonachlor.

[d] Toxicity value for p,p'-DDT used.

[e] Reported as mercury in data set.

[f] Toxicity value based on thallium nitrate.

[g] Serum glutamic oxaloacetic transaminase.

[h] LDH-lactate dehydrogenase.

5.2 Summary of Toxicity Assessment for Cancer

In the hazard identification step for cancer, summaries of the available toxicity information (e.g., results of animal tests and/or human occupational studies) on a chemical are reviewed. For cancer, this review is done to determine if that chemical is likely to cause cancer in humans. Based upon this evaluation, a chemical is classified into one of five weight-of-evidence classes that have been developed by EPA. These classes, shown in Table 5-4, define the potential for a chemical to cause cancer in humans.

Table 5-4. EPA weight-of-evidence classifications for carcinogens. (USEPA, 2000c).

Weight-of-Evidence Classification	Category
A	Human carcinogen
B	Probable human carcinogen
C	Possible human carcinogen
D	Not classifiable as a human carcinogen
E	Evidence of noncarcinogenicity in humans

In the second part of the toxicity assessment, the dose-response assessment, the toxicity values (CSFs) used to estimate cancer risk are developed. Based upon the manner in which some chemicals are thought to cause cancer, no exposure is thought to be without risk. Therefore, in evaluating cancer risks, a "safe" level of exposure cannot be estimated. To develop toxicity values for carcinogens, mathematical models are used to extrapolate from high levels of exposure where effects have been seen in animal studies or human studies to the lower exposures expected for human contact in the environment. The result of this extrapolation is a dose-response line whose slope is known as the cancer slope factor.

Table 5-5 shows the cancer slope factors for the 23 chemicals evaluated for cancer in this risk assessment. Because of the method used to develop these cancer slope factors, they are considered to be a plausible upper-bound estimate of the cancer potency of a chemical. By using these upper-bound estimates for the cancer slope factors, there is reasonable confidence that the actual cancer risks will not exceed the estimated risks calculated with these slope factors and may actually be lower. Table 5-5 also includes the weight-of-evidence classification for each carcinogen, the type of tumor that the cancer slope factor was based upon, and the source of this information. As previously discussed with reference doses, for many chemicals, both oral and inhalation cancer slope factors have been developed and are included in EPA toxicity databases. However, because the exposures assessed in this study result from ingestion of fish, only oral cancer slope factors were used.

Table 5-5. Oral cancer slope factors with their weight of evidence classification with the type(s) of tumor the cancer slope factor is based upon.

Chemical	Cancer Slope Factor (kg-d/mg)	Weight of Evidence	Tumor type	Source
2,3,7,8-TCDD	1.5×10^{5}	B2	Respiratory system and liver tumors	USEPA, 1997d
1,2-Diphenylhydrazine	8.0	B2	Hepatocellular carcinomas and neoplastic liver nodules	USEPA, 2000c
2,4,6-Trichlorophenol	1.1×10^{-2}	B2	Leukemia	USEPA, 2000c
Aldrin	1.7×10^{1}	B2	Liver carcinoma	USEPA , 2000c
alpha-HCH (alpha-BHC)	6.3	B2	Liver tumors	USEPA, 2000c
Adjusted Aroclors [a]	2.0	B2	Hepatocellular carcinomas	USEPA,1996
Arsenic, inorganic	1.5	A	Skin cancer, internal organs (liver, kidney, lung, bladder)	USEPA, 2000c
1,4-dichlorobenzene	2.40×10^{-2}	C	Liver tumors	USEPA, 1997d
Benzo(a)pyrene	7.3	B2	Forestomach, squamous cell papillomas and carcinomas	USEPA, 2000c
beta-HCH (beta-BHC)	1.8	C	Benign liver tumors	USEPA, 2000c
bis(2-Chloroisopropyl)ether	7.0×10^{-2}	C	Liver and lung tumors	USEPA, 1997d
Chlordane (total)[b]	3.5×10^{-1}	B2	Non-Hodgkin''s lymphoma and liver tumors	USEPA, 2000c
DDD (total)[c]	2.4×10^{-1}	B2	Lung, liver, and thyroid tumors	USEPA, 2000c
DDE (total)[c]	3.4×10^{-1}	B2	Liver and thyroid tumors	USEPA, 2000c
DDT (total)[c]	3.4×10^{-1}	B2	Liver	USEPA, 2000c
gamma-HCH (Lindane)	1.3	B2-C	Liver tumors	USEPA, 1997d
Heptachlor	4.5	B2	Hepatic nodules and hepatocellular carcinomas	USEPA, 2000c
Heptachlor epoxide	9.1	B2	Liver carcinoma	USEPA, 2000c
Hexachlorobenzene	1.6	B2	Liver, thyroid, kidney tumors	USEPA, 2000c
Hexachlorobutadiene	7.8×10^{-2}	C	Renal tubular adenomas and adenocarcinomas	USEPA, 2000c
Hexachloroethane	1.4×10^{-2}	C	Hepatocellular carcinomas	USEPA, 2000c
Pentachlorophenol	1.2×10^{-1}	B2	Hepatocellular adenoma/carcinoma, pheochromocytoma/malignant pheochromocytoma, hemangiosarcoma/hemangioma	USEPA, 2000c
Toxaphene	1.1	B2	Hepatocellular carcinoma and neoplastic nodules	USEPA, 2000c

[a] For each fish species, adjusted Aroclors is the sum of detected Aroclors less the sum of detected PCB congeners. Detected Aroclors included at least one of the following: Aroclor 1242, Aroclor 1254, and Aroclor 1260.

[b] Chlordane (total) is the sum of alpha-chlordane, cis-nonachlor, gamma-chlordane, oxychlordane, and trans-nonachlor.

[c] Slope factor for DDD (total), DDE (total), and DDT (total) based on the p,p' isomers.

5.3 Special Assumptions and Methods Used For Selected Chemicals

The average study site and basin fish contaminant levels for some of the chemicals in this risk characterization were calculated using unique assumptions. The need for these assumptions results from the lack of non-cancer toxicity values (reference doses) for each of the isomers of chlordane; for DDE and DDD; and for Aroclors 1242 and 1260 (Section 5.3.1); special methods for calculating cancer risks for chlorinated dioxins/furans, Aroclors and dioxin-like PCB congeners, and PAHs (Section 5.3.2); and the differential toxicity among arsenic species (Section 5.3.3).

5.3.1 Non-Cancer Toxicity Values for Chlordanes, DDT/DDE/DDD, and Aroclors

For non-cancer effects for chlordanes, DDT/DDE/DDD, and Aroclors, the average fish contaminant levels were calculated as summed quantities of individual chemicals in the class of chemicals. This summation methodology was applied to these three classes of chemicals because toxicity values were not available for all individual chemicals in these three classes and these chemicals were commonly detected in fish tissue. Use of this methodology assumes that the mechanisms of action for all of the chemicals in a class of chemicals are the same.

- Total chlordane was calculated as the sum of *cis*-chlordane, *trans*-chlordane, *cis*-nonachlor, *trans*-nonachlor, and oxychlordane. Non-cancer health effects for total chlordane were based on the reference dose for technical chlordane (USEPA, 2000c). Technical chlordane is not a single chemical, but is a mixture of several closely related chemicals, which consist of some of the various chlordane isomers and metabolites, including: cis-chlordane, trans-chlordane, cis-nonachlor, trans-nonachlor, and chlordenes, and other compounds.

- Total DDT was calculated by summing the ortho-para and para-para isomers of DDT, DDD, and DDE. IRIS contains a reference dose for DDT, but there are no specific reference doses for DDE or DDD. However, because the structures and toxicities of DDD and DDE closely resemble that of DDT (see Toxicity Profiles in Appendix B), for purposes of this risk characterization, it was assumed that they (and their various ortho- and para-isomers) have the same reference dose as DDT.

- Although PCB congeners were analyzed using two different methods: 1) Aroclors and 2) individual PCB congeners, non-cancer health effects were estimated only for Aroclors as EPA has not established an oral reference dose for individual PCBs congeners (USEPA, 2000c). Three Aroclors were detected in fish tissues, depending on the particular fish species, study site, and tissue type: Aroclor 1242, Aroclor 1254, and Aroclor 1260. The types and amounts of specific PCB congeners (each of which have their individual associated toxicity) differ in these three Aroclor mixtures. Only one of the Aroclors detected in this study has an oral reference dose, Aroclor 1254. Therefore, to provide a health protective estimate of non-cancer health impacts, the oral reference dose for Aroclor 1254 was also used for Aroclor 1242 and Aroclor 1260.

5.3.2 Cancer Toxicity for Chlorinated Dioxins/Furans, Dioxin-Like PCB congeners, and PAHs

The toxicity of the chlorinated dioxins/furans and dioxin-like PCB congeners were evaluated using toxicity equivalence factors recommended by WHO (Van den Berg et al., 1998). Table 2-10 (Section 2.7) listed the seventeen 2,3,7,8-substituted dioxin and furan congeners and 11 dioxin-like PCB congeners with 2,3,7,8-TCDD toxicity equivalence factor values. The toxicity equivalence factors were developed using careful scientific judgement after considering all available scientific data and are an order-of-magnitude estimate of the toxicity of these compounds relative to 2,3,7,8-TCDD.

Cancer risks from exposure to polycyclic aromatic hydrocarbons (PAHs) found in fish tissue in this study that are thought to be carcinogens were estimated from methods described in EPA guidance (USEPA, 1993). A cancer slope factor is available for one PAH only, benzo(a)pyrene. Relative potency factors have been developed for six PAHs (benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(ah)anthracene, indeno(1,2,3-cd) pyrene) relative to benzo(a)pyrene (see Table 5-6) (USEPA, 1993). These relative potency factors are used to convert the concentrations of the six PAHs into benzo(a)pyrene equivalent concentrations. As with the toxicity equivalence factors for chlorinated dioxins and furans and dioxin-like PCB congeners, these relative potency factors are order-of-magnitude estimates and, therefore, have inherent uncertainties. However, unlike the toxicity equivalence factors, these relative potency factors for the PAHs are to be considered as an "estimated order of potential potency" because they do not meet all of the guiding criteria for the toxicity equivalence method described by EPA for PCB mixtures (USEPA, 1991).

Table 5-6. Relative potency factors for PAHs (USEPA,1993).

Chemical	Relative Potency Factors
Benz(a)anthracene	0.1
Benzo(a)pyrene	1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Chrysene	0.001
Dibenz(ah)anthracene	1
Indeno(1,2,3-cd)pyrene	0.1

A methodology recommended by EPA for Aroclors was used to calculate cancer risk estimates for study site and basin-wide average fish concentrations (USEPA, 1996a). Because Aroclors consist of a mixture of both dioxin-like and non-dioxin-like congeners, calculating a cancer risk estimate for PCB congeners by summing the risk of both Aroclors and individual dioxin-like PCB congeners would overestimate cancer risk. To reduce this bias, the total Aroclor concentrations were "adjusted" by subtracting the total concentrations of dioxin-like congeners for each sample as shown in Equation 5-1.

(Equation 5-1) adjusted Aroclors = ∑Mass of Aroclors – ∑Mass of PCB congeners

The resulting adjusted Aroclor concentrations were used in association with a cancer slope factor for Aroclor mixtures to estimate the cancer risk associated with Aroclors detected in the fish samples (USEPA, 1996a). The cancer risk of dioxin-like PCB congeners was determined using the cancer slope factor for 2,3,7,8-TCDD and toxicity equivalence factors for PCB congeners. The cancer risks attributable to total PCBs were estimated by summing the risk estimates based on adjusted Aroclor concentrations and PCB congeners. While this method still likely overestimates the cancer risk of PCB congeners because the cancer slope factors developed for Aroclors include an unknown contribution from dioxin-like PCB congeners, the approach attempts to reduce the bias of double-counting the PCB risk (USEPA, 1996a).

5.3.3 Arsenic Toxicity

Arsenic exists in many chemical forms (chemical species), both organic and inorganic. These chemical species have varying toxicities ranging from practically non-toxic to very toxic. Organic arsenic species (those with carbon molecules bonded to the arsenic) are less toxic and the inorganic arsenic species (those in which the arsenic atom has a 3+ or 5+ charge and no carbon molecules; denoted as As^{3+} or As^{5+}, respectively) are more toxic. EPA considers inorganic arsenic to be a human carcinogen (see Table 5-5 for the oral CSF for inorganic arsenic). An oral RfD for the non-cancer health endpoints of inorganic arsenic has also been developed (see Table 5-3). EPA consensus toxicity values for organic arsenic species are not available at this time.

Fish contain both organic and inorganic arsenic species, with the organic arsenic species predominating. The organic arsenic species identified in fish include arsenobetaine, arsenocholine, arsenosugars, dimethyarsenic (DMA) and monomethylarsenic (MMA) For this risk assessment, fish tissue were analyzed for total (inorganic and organic) arsenic. Since toxicity values are only available for inorganic arsenic, to estimate the cancer risk and potential non-cancer health impacts from exposure to arsenic in this report, an estimate of the percentage of inorganic arsenic in fish had to be made. Of the many studies that have been done worldwide to measure the levels of arsenic in fish, several have included analyses of the various organic and inorganic species (ICF Kaiser, 1996). Most of these studies have been done with saltwater species and report inorganic arsenic levels in fish from zero to a few percent; however, some higher percentages of inorganic arsenic have also been found (e.g., 3.6% for herring, hairtail and saury, and 9.5% for shark). There are very few studies in which inorganic arsenic species have been determined in freshwater fish tissues (ICF Kaiser, 1996).

Inorganic arsenic results are available from two studies in fish from the Columbia River Basin - one in the Lower Columbia River Bi-State Water Quality Program (Tetra Tech, 1996) and a more recent one done on the Willamette River.

In the Lower Columbia River study (Tetra Tech, 1996), composites of fish were collected in 1995 from the mouth of the Columbia River to below the Bonneville Dam on the Columbia River (at River Mile 146) and analyzed for a large suite of chemicals, including inorganic arsenic. Sturgeon samples were skinned and analyzed as individual fish; all other fish were composites of fillets with skin. Table 5-7a shows a summary of the arsenic data from the six fish species collected as a part of this study (coho salmon, chinook salmon, sturgeon, sucker, carp and

steelhead). Analyses were done for total arsenic, inorganic arsenic, and the methylated species (MMA, DMA). The percent of inorganic arsenic and the percent of the sum of DMA and MMA were calculated and are also shown in the table.

The percent inorganic arsenic ranged from a low of 0.1% in two of the steelhead composites and one chinook composite (2 of the 3 values of 0.1% are based on non-detect values) to a high of 26.6% in a sucker composite (Table 5-7a). Within the same species the variation between different composite samples was large. For example, percent inorganic arsenic in the sucker composites ranged from 0.6% (based upon a nondetected value) to 26.6%. Individual sturgeon ranged from 1.9% to 18.2% . The average percent inorganic arsenic by species ranged from 0.5% in carp to 9.2% in sturgeon (Table 5-7c) with an overall arithmetic average for all composites of 6.5% (see Table 5-7b).

Average percent inorganic arsenic was also estimated for anadromous fish versus resident fish species (Table 5-7d). As can be seen from this table, the average percent inorganic arsenic in anadromous fish species is about 1% while that from resident fish species is about 9%.

Table 5-7a. Results of arsenic (As) analyses from Lower Columbia River Bi-State Water Quality Program (Source: Tetra Tech, 1996).

Species/Sample	Total As (ug/g WW)	Inorganic As (ug/g WW)	Q*	Percent Inorganic As	DMA & MMA (ug/g WW)	Q*	Percent DMA & MMA
Coho/HCMP1	0.415	0.001	UJ	0.2%	0.056		13.5%
Coho/HCMP2	0.344	0.007	J	2.0%	0.029		8.4%
Coho/HCMP3	0.361	0.001	UJ	0.3%	0.039		10.8%
Chinook/KCMP1	1.235	0.023	J	1.9%	0.038		3.1%
Chinook/KCMP2	0.884	0.001	UJ	0.1%	0.078		8.8%
Chinook/KCMP3	0.760	0.015	J	2.0%	0.034		4.5%
Sturgeon/SIND1	1.793	0.034		1.9%	0.038		2.1%
Sturgeon/SIND2	0.563	0.011		2.0%	0.023		4.1%
Sturgeon/SIND3	0.558	0.047		8.4%	0.019		3.4%
Sturgeon/SIND4	0.533	0.045		8.4%	0.013		2.4%
Sturgeon/SIND5	0.275	0.05		18.2%	0.007		2.5%
Sturgeon/SIND6	0.485	0.047		9.7%	0.009		1.9%
Sturgeon/SIND7	0.395	0.039		9.9%	0.01		2.5%
Sturgeon/SIND8	0.357	0.04		11.2%	0.003		0.8%
Sturgeon/SIND9	0.669	0.043		6.4%	0.01		1.5%
Sturgeon/SIND10	0.748	0.033		4.4%	0.13		17.4%
Sturgeon/SIND11	0.24	0.039		16.3%	0.009		3.8%
Sturgeon/SIND12	0.311	0.041		13.2%	0.01		3.2%
Sucker/LSCMP1-1	0.151	0.017		11.3%	0.007		4.6%
Sucker/LSCMP1-2	0.133	0.024		18.0%	0.004		3.0%
Sucker/LSCMP1-3	0.143	0.038		26.6%	0.007		4.9%
Sucker/LSCMP2-1	0.113	0.012		10.6%	0.004		3.5%
Sucker/LSCMP2-2	0.181	0.008		4.4%	0.007		3.9%
Sucker/LSCMP2-3	0.17	0.004		2.4%	0.011		6.5%
Sucker/LSCMP3-1	0.098	0.006		6.1%	0.001	U	1.0%
Sucker/LSCMP3-2	0.178	0.001	U	0.6%	0.011		6.2%
Sucker/LSCMP3-3	0.168	0.003		1.8%	0.007		4.2%
Carp/CCMP1	0.221	0.001		0.5%	0.02		9.0%
Steelhead/DCMP1	0.677	0.018		2.7%	0.021		3.1%
Steelhead/DCMP2	0.753	0.001		0.1%	0.033		4.4%
Steelhead/DCMP3	0.703	0.001	U	0.1%	0.031		4.4%

Table 5-7b. Mean concentrations of arsenic(As) in all fish species combined**

	Total As (ug/g WW	Inorganic As (ug/g WW)	Percent Inorganic As	DMA & MMA (ug/g WW)	Percent DMA & MMA
Arithmetic mean	0.47	0.02	6.5%	0.02	5.0%
Geometric mean	0.36	0.01	2.9%	0.01	3.9%

Table 5-7c. Arithmetic means of percent inorganic arsenic by species.**

Species	Mean
coho	0.9%
chinook	1.3%
sturgeon	9.2%
sucker	9.1%
carp	0.5%
steelhead	1.0%

Table 5-7d. Arithmetic means ** of percent inorganic arsenic - resident fish versus anadromous fish species.

Species	% Inorganic As
Anadromous only	1.0%
Resident only	9.1%

WW = wet weight; As = arsenic; MMA = momomethylarsenic; DMA = dimethylarsenic

*Q = data qualifiers; Blanks indicate data was not qualified; U = not detected; J= estimated;

**calculations based on Tetra Tech, 1996.

coho/HCMP=coho/coho composite; chinook/KCMP = chinook/chinook composite;

sturgeon/SIND = sturgeon/sturgeon individual; sucker/LSCMP = sucker/largescale sucker composite;

carp/CCMP= carp/carp composite; steelhead/DCMP = steelhead/steelhead composite

For the middle Willamette River study (EVS, 2000), composites of fish (largescale sucker, carp, smallmouth bass, and northern pikeminnow) were collected from a 45-mile section of the Willamette River extending from the Willamette Falls near Oregon City (River Mile 26.5) to Wheatland Ferry (River Mile 72). Total arsenic and inorganic arsenic concentrations were determined in each of the composite fish samples. These samples included composites of whole body, composites of fillet with skin, and composites of that portion of the fish remaining after removing fillets from both sides of the fish. A summary of the arsenic data for whole body and fillet with skin samples is shown in Table 5-8. Percent inorganic arsenic in the individual composites ranged from 2% (carp) to 13.3% (sucker). Only two species had multiple composite samples analyzed for the same body type, whole body for carp and fillet for smallmouth bass. The average percent of inorganic arsenic was 4.2% for the carp (range of 2 to 6.9% in the four whole body composites) and 3.8% for the smallmouth bass (2.7% (not detected) and 6.3% in two fillet composites).

Table 5-8. Summary of Willamette River, speciated arsenic data (EVS, 2000).

Composite	Tissue Type	Total As (ug/kg WW)	Q	Inorganic As (ug/kg WW)	Q	Percent Inorganic As	Q	Average Percent Inorganic As
Sucker/ Comp 1	F	0.08		0.004		5.0%		
Sucker/ Comp 12	WB	0.12		0.016		13.3%		
Carp/ Comp 3	WB	0.16		0.007		4.4%		
Carp/ Comp 4	WB	0.13		0.009		6.9%		
Carp/ Comp 5	WB	0.15		0.005		3.3%		
Carp/ Comp 14	WB	0.15		0.003		2.0%		4.2%[a]
Carp/ Comp 9	F	0.12		0.003	U	2.5%	U	
Bass/ Comp 6	F	0.11		0.003	U	2.7%	U	
Bass/ Comp 7	F	0.08		0.005		6.3%		3.8%[b]
Pikeminnow/ Comp 13	WB	0.05	U	0.003	U	6.0%	U	
Pikeminnow/ Comp 10	F	0.05	U	0.003	U	6.0%	U	

Comp = composite; F= fillet; WW= wet weight; WB = whole body
Q = data qualifier; U = not detected; blanks indicate that data was not qualified
[a]for whole body carp; [b]for bass fillet

Only two species, carp and sucker, were analyzed for inorganic arsenic and total arsenic in both the Lower Columbia River and Willamette River studies. For carp, one composite sample of fillet with skin was analyzed in each of the studies giving inorganic arsenic percentages of 2.5% (Willamette, based on a non-detected value) and 0.5% (Lower Columbia River). For sucker composites, the average for percent inorganic arsenic in the Lower Columbia River study (fillet with skin, 9 composites) is 9.1% compared to that for the one fillet sample from the Willamette of 5.0%. The range of values for the 9 sucker composites from the Lower Columbia River study is large (0.6% to 26.6%).

In deciding what value to assume for inorganic arsenic in fish in this assessment, consideration was given to the Lower Columbia River and Willamette River inorganic arsenic data cited in this study as well as to uncertainties related to 1) arsenic toxicity (i.e., from DMA) and 2) arsenic analyses in fish tissue:

(1) Arsenic toxicity - Because arsenobetaine and arsenocholine are readily absorbed from the human digestive tract and excreted in urine rapidly and unchanged, these arsenic species are considered virtually non-toxic. In contrast, arsenosugars are apparently metabolized in the human body to DMA which is then excreted in urine (Ma and Le, 1998). EPA has classified DMA as a category B2 carcinogen (probable human carcinogen based on sufficient animal but insufficient human evidence) based on tumors in rodents (USEPA, 2001). However, no EPA consensus toxicity values are available for DMA.

Although DMA may be toxic, no DMA data is available on the fish samples collected as a part of this Columbia River Basin study. In addition information on the concentrations of DMA in freshwater fish from other studies are limited. Concentrations of DMA and MMA, combined, are available from the Lower Columbia River Bi-State Water Quality Program (Tetra Tech, 1996) and are shown in Tables 5-7a and 5-7b. The percent of DMA and MMA combined ranged from 0.8% to 17.4% among the composites. The arithmetic mean for the combined levels of MMA and DMA among all six of the fish species analyzed was about 5% (Table 5-7b). However, the values for DMA alone are not available.

Thus, although DMA may be an arsenic species of concern in fish or of concern as a result of metabolism of arsenosugars, it is not possible to evaluate the potential impact on the risk characterization that this compound would have in this study.

(2) Analysis for arsenic in fish - the identity of the chemical species of arsenic in aquatic species is currently an area of active research and rapidly advancing knowledge. Existing analytical methods for the chemical speciation of arsenic have several limitations including, but not limited to, a lack of data on the efficiencies of recovery of arsenic species during analysis, the possible inter-conversion of arsenical species during extraction and analyses and the lack of native standard reference materials for use in determining accuracy, precision and reproducibility.

In the estimating non-cancer hazards and cancer risks from exposure to arsenic in fish tissue (Sections 6.2.1 and 6.2.2) it was assumed that 10% of total arsenic is inorganic arsenic. The value of 10% was chosen after considering:

1) the wide range found in percent inorganic arsenic among the freshwater samples of a given species in the Lower Columbia River and Willamette River studies,
2) the limited data base on concentrations of inorganic arsenic in freshwater fish,
3) the uncertainties in the toxicity and concentrations of DMA in fish, and
4) the uncertainties in the analytical techniques used for the chemical speciation of arsenic.

This value of 10% is expected to result in a health protective estimate of the potential health effects from arsenic in fish.

However, the inorganic arsenic data for anadromous fish species in the Lower Columbia River

study suggest that the assumption of a lower percentage (i.e., about 1%, see Table 5-8d) of inorganic arsenic in these anadromous fish species may also be appropriate. This is also consistent with the literature on saltwater species which show inorganic arsenic levels in the low percentages for most saltwater fish. Therefore, in Section 6.2.6 the analyses of cancer risk and non-cancer hazards were presented assuming that inorganic arsenic is only 1% of the total arsenic in anadromous fish species.

Using a range of assumptions for percent inorganic arsenic in anadromous fish species provides information on the potential uncertainties in the risk characterization.

6.0 Risk Characterization

Risk characterization is the final step in the risk assessment process. It combines the information from the Exposure Assessment (Section 4) and Toxicity Assessment (Section 5) to estimate non-cancer hazards and cancer risks. In addition, risk characterization addresses the uncertainties underlying the risk assessment process (Section 10, Uncertainty Evaluation). This risk characterization was prepared in accordance with the EPA guidance on risk characterization (USEPA, 1992b; USEPA, 1995).

The methodology used to quantify potential non-cancer health effects and cancer risks is described in Section 6.1. The estimated non-cancer health hazards are discussed in detail in Section 6.2.1. and the estimated cancer risks in Section 6.2.2. Cancer and non-cancer results are summarized in Section 6.2.3. In Section 6.2.4 the differences in cancer risks and non-cancer hazards are compared between whole body and fillet fish samples collected from each site in the Columbia River Basin. Section 6.2.5 discusses the results of the multiple-species diet calculation, and; Section 6.2.6 shows how assumptions of percent inorganic arsenic impact the risk characterization.

Non-cancer health hazards and cancer risk estimates are calculated separately and reported separately. Because EPA uses different methods to evaluate these endpoints, non-cancer and cancer estimates cannot be combined.

6.1 Risk Characterization Methodology

6.1.1 Non-Cancer Health Effects

For non-cancer health effects, it is assumed that there is an exposure threshold below which adverse effects are unlikely to occur. In this assessment, the evaluation of non-cancer health effects involved a comparison of average daily exposure to chemicals in fish tissue with the EPA reference doses discussed in Section 5. The reference dose is an estimate of the daily exposure to a chemical that is unlikely to cause toxic effects. Potential health hazards from non-cancer effects for a specific chemical are expressed as a hazard quotient (HQ), which is the ratio of the calculated exposure (Section 4) to the reference dose for that chemical.

Both the estimated average daily doses from consuming fish and the reference doses are expressed in units of amount (in milligrams) of a chemical ingested per kilogram of body weight per day (mg/kg-day) (USEPA, 1989):

(Equation 6-1)

$$HQ = \frac{ADD}{RfD}$$

Where:

HQ = Chemical-specific hazard quotient (unitless)
ADD = Average daily dose (mg/kg-day)
RfD = Chemical-specific oral reference dose (mg/kg-day)