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**COLUMBIA RIVER BASIN  
FISH CONTAMINANT SURVEY  
1996-1998**

**U.S. Environmental Protection Agency**  
Region 10  
Seattle, Washington 98101



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## CONTRIBUTORS

### EPA Region 10

Patricia Cirone  
Dana Davoli  
Duane Karna  
Robert Melton  
Rick Poeton  
Marc Stifelman  
Dave Terpening  
Michael Watson  
Peggy Knight  
Katherine Adams  
Roy Araki  
Isa Chamberlain  
Randy Cummings  
Gerald Dodo  
Stephanie Le  
Kathy Parker  
Steve Reimer  
Bob Rieck  
Tony Morris  
Tobi Braverman

Steve Ellis  
MCS Environmental, Inc

EVS Consultants  
Seattle, Washington

TetraTech Consultants  
Bellevue, Washington



## COLUMBIA RIVER BASIN FISH TISSUE CONTAMINANT STUDY WORKGROUP

Anne Watanabe  
Columbia River Intertribal Fish Commission  
(formerly)

Paul Lumley III  
Columbia River Intertribal Fish Commission

Catriona (Cat) Black  
Columbia River Intertribal Fish Commission

Barbara Harper  
Yakama Nation (formerly)

Lynn Hatcher  
Yakama Nation

Chris Walsh  
Yakama Nation Health Center

Silas Whitman  
Nez Perce Tribe (formerly)

Rick Eichsteadt  
Nez Perce Tribe

Nancy Hoefs  
Nez Perce Fisheries

Patti Howard  
Nez Perce Tribe (formerly)

Gary James  
Confederated Tribes of the Umatilla Indian  
Reservation

Stuart Harris  
Confederated Tribes of the Umatilla Indian  
Reservation

Patty O'Toole  
Confederated Tribes of the Warm Springs  
Reservation

## LIST OF ABBREVIATIONS AND ACRONYMS

ADD	average daily dose of a specific chemical (mg/kg-day)
AFC	average fish consumption
ALM	EPA Adult Lead Model
AT	averaging time for exposure duration (days)
ATSDR	Agency for Toxic Substances and Disease Registry
AVE	average
BCF	bioconcentration factor
BEIR	Biological Effects of Ionizing Radiation
BEST	Biomonitoring of Environmental Status and Trends
BKSF	biokinetic slope factor
BW	body weight
C	chemical concentration in fish tissue
CDC	Centers for Disease Control
CF	conversion factor
CSFII	Continuing Survey of Food Intake by Individuals
CSFs	cancer slope factors
CRITFC	Columbia River Intertribal Fish Commission
DDE	1,1-dichloro-2,2- <i>bis</i> ( <i>p</i> -chlorophenyl)ethylene
DDT	1,1,1-trichloro-2,2- <i>bis</i> ( <i>p</i> -chlorophenyl)ethane
DDD	1,1-dichloro-2,2- <i>bis</i> ( <i>p</i> -chlorophenyl)ethane
DDMU	1,1- <i>bis</i> ( <i>p</i> -chlorophenyl)2 chloro-ethylene
DF	detection frequency
DMA	dimethylarsenic
EF	exposure frequency (days/year)
ED	Exposure duration (years)
ECR <sub>new</sub>	Excess cancer risk for the new exposure duration
ECR <sub>70</sub>	Excess cancer risk estimate for a lifetime exposure duration of 70 years
ED <sub>new</sub>	Individual exposure duration in years
ED <sub>70</sub>	Default lifetime exposure duration of 70 years
EPA	Environmental Protection Agency
FS	fillet with skin
FW	fillet without skin
GC/AED	Gas Chromatograph/Atomic Emission Detector
GSD	Geometric Standard Deviation
GPS	global positioning system
HEAST	Health Effects Assessment Summary Tables
HFC	high fish consumption
HI	hazard index
HQ	hazard quotient
IEUBK	EPA integrated exposure uptake biokinetic model
IR	ingestion rate
LLD	lower limit of detection
LOAEL	lowest observed adverse effect level

MAX	maximum
MDC	minimum detectable concentration
MF	modifying factor
MIN	minimum
MMA	monomethylarsenic
NA	not applicable
NAERL	National Air and Radiation Environmental Laboratory
NCEA	National Center for Environmental Assessment
NCBP	National Contaminant Biomonitoring Program
NCRP	National Council on Radiation Protection and Measurements
ND	not detected
NOAEL	no observable adverse effect level
NS	not sampled
OCDD	Octachlorodibenzo- <i>p</i> -dioxin
OERR	EPA Office of Emergency and Remedial Response
PAHs	polycyclic aromatic hydrocarbons
PCBs	polychlorinated biphenyls
PSAMP	Puget Sound Ambient Monitoring Program
RfD	reference dose
RPFs	relative potency factors
2,3,7,8-TCDD	2,3,7,8-tetrachlorodibenzo- <i>p</i> -dioxin
2,3,7,8 TCDF	2,3,7,8 tetrachlorodibenzo- <i>p</i> -furan
TEC	toxicity equivalence concentration
TEF	toxicity equivalence factor
TRW	EPA Technical Review Workgroup for Lead
UF	uncertainty factors
WB	whole body
USEPA	U.S. Environmental Protection Agency
USGS	United States Geological Survey

Bq                      one radioactive disintegration  
per second

<b>Units</b>	
ng/kg	nanograms per kilogram (ppt)
µg/dl	micrograms per deciliter
µg/kg	micrograms per kilogram (ppb)
g/day	grams per day
mg/kg	milligram per kilogram (ppm)
kg	kilogram
kg/g	kilogram per gram

mg/kg-day            milligram per kilogram-day



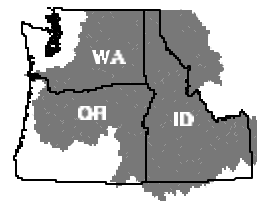
## EXECUTIVE SUMMARY

### Introduction

This report presents the results of an assessment of chemical pollutants in fish and the potential risks from consuming these fish. The fish were collected throughout the Columbia River Basin in Washington, Oregon, and Idaho.

After reviewing the results of the U.S. Environmental Protection Agency (USEPA. 1992a) 1989 national survey of pollutants in fish in the United States, EPA became concerned about the potential health threat to Native Americans who consume fish from the Columbia River Basin. The Columbia River Intertribal Fish Commission (CRITFC) and its member tribes (Warm Springs Tribe, Yakama Nation, Umatilla Confederated Tribes, Nez Perce Tribe) were also concerned for tribal members who consume more fish than non-Indians.

Map of Columbia River Basin



In order to evaluate the likelihood that tribal people may be exposed to high levels of contaminants in fish tissue EPA, CRITFC and its member tribes, designed a study in two phases. The first phase was a fish consumption survey which was conducted by the staff of CRITFC and its member tribes. The fish consumption survey was completed in 1994 (CRITFC 1994). The conclusions of the tribal survey were:

“The rates of tribal members’ consumption across gender, age groups, persons who live on- vs. off-reservation, fish consumers only, seasons, nursing mothers, fishers, and non-fishers range from 6 to 11 times higher than the national estimate used by USEPA.”(quote from CRITFC, 1994, Page 59)

The results of the fish consumption survey accentuated the need to complete an assessment of chemicals in the fish being consumed by CRITFC’s member tribes.

In 1994, EPA and CRITFC’s member tribes initiated the second phase of the study which was a survey of contaminants in fish tissue in the Columbia River Basin and the subject of this report. The contaminant survey was designed by a multi-agency group including CRITFC, Washington Departments of Ecology and Health, Oregon Departments of Environmental Quality and Health, the Confederated Tribes of Warm Springs, the Yakama Nation, the Umatilla Confederated Tribes, the Nez Perce Tribe, U.S. Geological Survey, and U.S. Fish and Wildlife Service. Sample collection took place between 1996 and 1998 with the help of CRITFC’s member tribes and staff of federal and state agencies. Chemical analyses were completed in 1999. The analyses were done by EPA and commercial laboratories.

While the study was initiated because of concern for Native American tribes, the results are

important to all people who consume fish from the Columbia River Basin. This study provided EPA with information to determine:

- 1) if fish were contaminated with toxic chemicals,
- 2) the difference in chemical concentrations among fish species and study sites, and
- 3) the potential human health risks due to consumption of fish from the Columbia River Basin.

The results of this survey provided information on those chemicals which were most likely to be accumulated in fish tissue and therefore posed the greatest potential risks to people. These are the chemicals for which regulatory strategies need to be defined to reduce these chemicals in our environment.

This study was *not* designed to evaluate:

- 1) health of past or future generations of people who consume fish from the Columbia River Basin,
- 2) rates of disease in tribal communities,
- 3) specific sources of chemicals,
- 4) multiple exposures to chemicals from air, water, and soil,
- 5) food other than fish, and
- 6) risks for a specific tribe or individual.

It is our hope that the results of this survey will be used by CRITFC's member tribes as well as others to more completely evaluate and protect the quality of the fishery resource.

### **Study Design**

This study was designed to estimate risks for a specific group of people (CRITFC's member tribes). Therefore, the sample location, fish species, tissue type, and chemicals were not randomly selected. Collection sites were selected because they were important to characterizing risks to CRITFC's member tribes. Chemicals were chosen because they were identified in other fish tissue surveys of the Columbia River Basin as well as being found throughout the environment.

This type of sampling is biased with unequal sample sizes and predetermined sample locations rather random. This bias is to be expected when attempting to provide information for

individuals or groups based on their preferences. The results of this survey should not be extrapolated to any other fish or fish from other locations.

A total of 281 samples of fish and fish eggs were collected from the Columbia River Basin. The fish species included five anadromous species (Pacific lamprey, smelt, coho salmon, fall and spring chinook salmon, steelhead) and six resident species (largescale sucker, bridgelip sucker, mountain whitefish, rainbow trout, white sturgeon, walleye). Four types of samples were collected: whole-body with scales, fillet with skin and scales, fillet without skin (white sturgeon only), and eggs. The fillets were all with skin except for the white sturgeon. The armor-like skin of the white sturgeon is considered too tough for ingestion. All the samples were composites of individual fish, except white sturgeon. The white sturgeon were analyzed as single fish instead of composites because of their large size. The number of fish in a composite varied with species, location, and tissue type. Eleven samples of eggs were collected from steelhead and salmon. Due to availability of fish, limitation in time and funds, certain species were not sampled as frequently as others. In particular, the bridgelip sucker, coho salmon, and eulachon were collected at only one location. Pacific lamprey and walleye were collected at only two locations. The type of tissue tested (whole body, fillet, egg) varied with species and sample location.

Three replicate samples for each fish type were collected from a total of 24 study sites. These sites were located on 16 rivers and creeks, including, Hood River, Little White Salmon River, Wind River, Fifteen Mile Creek, Wenatchee River, Willamette River, Deschutes River, Umatilla River, Thomas Creek, Meacham Creek, Klickitat River, Yakima River, Snake River, Clearwater River, Looking Glass Creek, and the mainstream Columbia River. Different species were collected from each site depending upon the fishing practices of CRITFC's member tribes. Despite these many variables, general trends in the monitoring of pollutants in these various species and tissues were evident.

The fish tissues were analyzed for 132 chemicals including 26 pesticides, 18 metals, 7 PCB Aroclors, 13 dioxin-like PCBs, 7 dioxin congeners, 10 furan congeners, and 51 miscellaneous organic chemicals. Of these 132 chemicals, 92 were detected. The most frequently detected chemicals in fish tissue were 14 metals, DDT and its structural analogs (DDD, DDE), chlordane and related compounds (*cis*-chlordane, *trans*-chlordane, *cis*-nonachlor, *trans*-nonachlor, and oxychlordane), PCBs (Aroclors<sup>1</sup> and dioxin-like PCBs), and chlorinated dioxin and furans.

## Results

The fish tissue chemical concentrations were evaluated for each study site and for the whole basin. The results of the study showed that all species of fish had some levels of toxic chemicals in their tissues and in the eggs of chinook and coho salmon and steelhead. The fish tissue chemical concentrations were variable within fish (duplicate fillets), across tissue type (whole body and fillet), across species, and study sites. However, the chemical residues exhibited some

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<sup>1</sup>Aroclors = commercial formulation of mixtures of PCB congeners; Aroclors 1242, 1254, and 1260 were the only aroclors detected in fish tissue in our study

trends in distribution across species and locations. The concentration of organic chemicals in the salmonids (chinook and coho salmon, rainbow and steelhead trout) and eulachon were lower than any other species. The concentrations of organic chemicals in three species (white sturgeon, mountain whitefish, largescale sucker) and Pacific lamprey were higher than any other species. The concentrations of metals were more variable, with maximum levels of occurring in different species.

Of the 132 chemicals analyzed in this study, DDE, Aroclors, zinc, and aluminum were detected in the highest concentration in most of the fish tissues sampled throughout the basin. The basin-wide average concentrations for the organic chemicals (DDE, Aroclors, chlorinated dioxins and furans) ranged from non-detectable in the anadromous fish species to the highest levels in resident species. DDE, the most commonly found pesticide in fish tissue from our study, ranged from a basin-wide average of 11 ppb<sup>2</sup> in whole body eulachon to 620 ppb in whole body white sturgeon. The sum of Aroclors ranged from non-detectable in eulachon to 190 ppb in mountain whitefish fillets. sturgeon. Chlorinated dioxins and furans were found at low concentrations in fish species. The basin-wide average concentration of the sum of chlorinated dioxins and furans ranged from 0.0001 ppb in the walleye, largescale sucker, coho, and steelhead fillets, fall chinook salmon (whole body, fillet, egg) and steelhead eggs to 0.03 ppb in whole body white sturgeon.

The concentration of metals did not show a distinct difference between anadromous and resident fish species. The basin-wide average concentrations of arsenic ranged from non-detectable in rainbow trout fillet to 890 ppb in whole body eulachon. Mercury ranged from non-detectable levels in Pacific lamprey fillets and whole body eulachon to 240 ppb in largescale sucker.

The distribution across stations was variable although fish collected from the Hanford Reach of the Columbia River and the Yakima River tended to have higher concentrations of organic chemicals than other study sites.

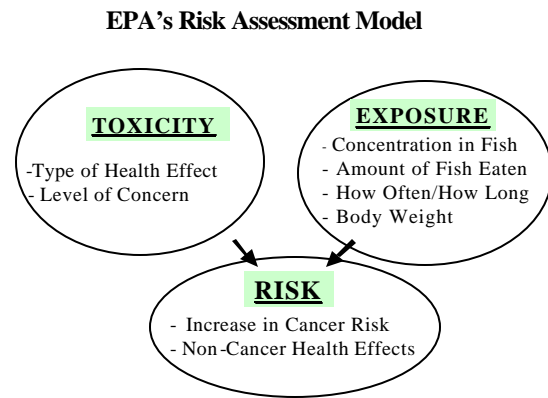
The chemical concentrations in fish species measured in this study were generally lower than levels reported in the literature from the early 1970's and similar to levels reported in the late 1980's to the present. The literature included studies from the Columbia River Basin as well as other water bodies in the United States.

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<sup>2</sup>ppb = parts per billion = µg/kg



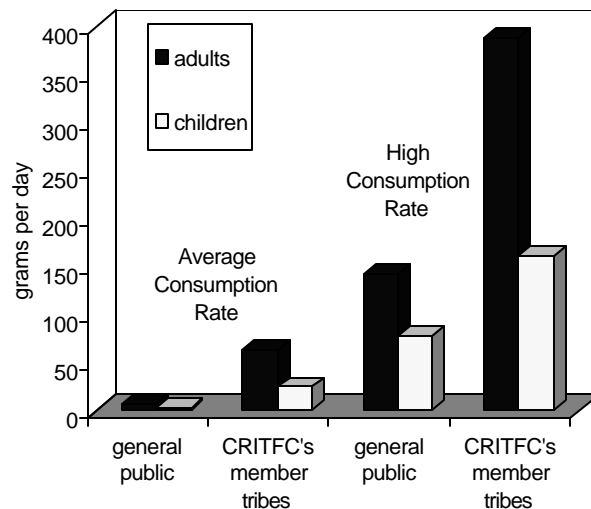
EPA uses a risk model to characterize the possible health effects associated with chemical exposure. For this model, toxicity information is combined with estimates of exposure to characterize cancer risks and non-cancer health effects. Toxicity information (*reference doses and cancer slope factors*) used in this study was obtained from USEPA databases.



The EPA method to estimate exposure to chemicals in fish depends upon the chemical concentration in the fish tissue, the amount and types of fish eaten, how long and how often fish is eaten, and the body weight of the person eating the fish. For this assessment, exposures to chemicals were estimated for both adults and children of CRITFC's member tribes and the general population. In addition to estimating exposure for each site, exposures were also estimated for the basin wide average of fish tissue. In estimating these exposures, it was assumed that a person eats the same type of fish for their lifetime.

Different fish ingestion rates were used for the general public and for CRITFC's member tribes. Fish consumption rates for CRITFC's member tribes were based upon data from the CRITFC fish consumption survey (CRITFC, 1994) while those for the general public were based upon EPA analysis of national fish consumption rates (USEPA, 2000b).

**Average and high (99<sup>th</sup> percentile) fish consumption rates for CRITFC's member tribes and the general public.**



In conducting a risk assessment, EPA evaluates the potential for developing non-cancer health effects such as immunological, reproductive, developmental, or nervous system disorders and for increased cancer risk. Different methods are used to estimate non-cancer health effects and cancer risks.

For non-cancer health effects, EPA assumes that a threshold of exposure exists below which

health effects are unlikely. To estimate non-cancer health effects, the estimated lifetime average daily dose of a chemical is compared to its *reference dose (RfD)*. The reference dose represents an estimate of a daily exposure level that is likely to be without deleterious effects in a lifetime. The ratio of the exposure level in humans to the *reference dose* is called a hazard quotient. To account for the fact that fish contained multiple chemicals, the hazard quotients for the chemicals which cause similar health effects were added to calculate a single hazard index for each type of health effect. For exposures resulting in hazard indices equal to or less than one, health impacts are unlikely. Generally, the higher hazard index is above one, the greater the level of concern for health effects.

For cancer, EPA assumes that any exposure to a carcinogen may increase the probability of getting cancer. Thus, the risk from exposure to a carcinogen is estimated as the increase in the probability or chance of developing cancer over a lifetime as a result of exposure to that chemical (e.g. an increased chance of 1 in 10,000). Cancer risks, which are calculated for adults only, are estimated by multiplying the lifetime average daily intake of a chemical by its *cancer slope factor*. The estimated cancer risk from exposure to a mixture of carcinogens is estimated by adding the cancer risks for each chemical in a mixture. The cancer risk estimates which are based on EPA's methodology are considered to be upper-bound estimates of risk or the most health-protective estimate. Due to our uncertainty in understanding the biological mechanisms which cause cancer, the true risks may in fact be substantially lower than the number estimated with EPA's risk assessment model.

In interpreting cancer risks, different federal and state agencies often have different levels of concern for cancer risks based upon their laws and regulations. EPA has not defined a level of concern for cancer. However, regulatory actions are often taken when the probability of risk of cancer is within the range of 1 in 1,000,000 to 1 in 10,000. Risk managers make their decisions regarding which level within this range is a concern depending on the circumstances of the particular exposure(s). A level of concern for cancer risk has not been defined for this risk assessment.

Using EPA's risk assessment models, hazard indices and cancer risks were estimated for people who consume resident and anadromous fish from the whole Columbia River Basin and from each study site in the basin. For adults, hazard indices and cancer risks were lowest for the general public at the average ingestion rate and highest for CRITFC's member tribes at the high ingestion rate. For adults in the general public with an average fish ingestion rate of about a meal<sup>3</sup> per month (7.5 g/day), hazard indices were less than 1 and cancer risks were less than 1 in 10,000 except for a few of the more highly contaminated samples of mountain whitefish and white sturgeon. For adults in CRITFC's member tribes, at the highest fish ingestion rate at about 48 meals<sup>1</sup> per month (389 g/day), hazard indices were greater than 1 for several species at some sites. Hazard indices (less than or equal to 8 at most sites) and cancer risks (7 in 10,000 to 2 in 1,000) were lowest for salmon, steelhead, eulachon and rainbow trout and highest (hazard indices greater than 100 and cancer risks up to 2 in 100 at some sites) for mountain whitefish and white sturgeon.

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<sup>3</sup>Meal = eight ounces of fish

For the general public, the hazard indices for children at the average fish ingestion rate were less for adults (0.9) at the average ingestion rate; the hazard indices for children at the high ingestion rate were 1.3 times greater than those for adults at the high ingestion rate. For CRITFC's member tribes, the hazard indices for children at the average and high ingestion rates were 1.9 times greater than those for adults in CRITFC's member tribes at the average and high ingestion rates, respectively.

For both resident and anadromous species, the major contributors to the hazard indices were PCBs (Aroclors) and mercury. DDT and its structural analogs were also important contributors for some resident species. The chemicals and or chemical classes that contributed the most to cancer risk for most of the resident fish were PCBs (Aroclors and dioxin-like PCBs), chlorinated dioxins and furans, and a limited number of pesticides. For most of the anadromous fish, the chemicals that contributed the most to cancer risk were PCBs (Aroclors and dioxin-like PCBs), chlorinated dioxins and furans, and arsenic.

In estimating hazard indices and cancer risks for people who eat a certain fish species, it is assumed that they eat only that type of fish for their lifetime. However, many people eat a variety of fish over a lifetime. Hazard indices and cancer risks were also estimated using a hypothetical multiple species diet. This hypothetical multiple species diet was based upon information from the CRITFC fish consumption study (CRITFC, 1994). The hazard indices and cancer risks for the multiple species diet were lower than those for most contaminated species of fish and greater than those for some of the least contaminated species. The risks for eating one type of fish may be an over or underestimate of the risks for consumers of a multiple-species diet depending upon the types of fish and concentration of chemicals in the fish which make up the diet.

The risk assessment model for assessing exposure to lead is different from other chemicals. Lead risk is based on a bio-kinetic model which includes all routes of exposure (ingestion of food, soil, water, and inhalation of dust). Based on EPA's risk assessment model, the lead concentrations in Columbia River Basin fish tissues were estimated to be unlikely to cause a human blood lead level greater than 10 µg/dl. The blood lead level of 10 µg/dl is the national level of concern for young children and fetuses (CDC, 1991).

In addition to the survey of the basin for the 131 chemicals, a special study of radionuclides was completed for a limited number of samples. White sturgeon were collected from the Hanford Reach of the Columbia River, artificial ponds on the Hanford Reservation, and from the upper Snake River and analyzed for radionuclides. The levels of radionuclides in fish tissue from Hanford Reach of the Columbia River and the ponds on the Hanford Reservation were similar to levels in fish from the Snake River. Cancer risks were estimated for consumption of fish which were contaminated with radionuclides. These risks estimates were not combined with the potential risks from other chemicals at these study sites. The potential cancer risks from consuming fish collected from Hanford Reach and the artificial ponds on the Hanford Reservation were similar to cancer risks in fish collected from the upper Snake River.

## Conclusions

The concentration of toxic chemicals found in fish from the Columbia River Basin may be a risk to the health of people who eat them depending on:

- 1) the toxicity of the chemicals,
- 2) the concentration in the fish,
- 3) the species and tissue type of the fish, and
- 3) how much and how often fish is consumed

The chemicals which contribute the most to the hazard indices and cancer risks are the persistent bioaccumulative chemicals (PCBs, DDE, chlorinated dioxins and furans) as well as some naturally occurring chemicals (arsenic, mercury). Some pollutants persist in the food chain largely due to past practices in the United States and global dispersion from outside North America. Although some of these chemicals are no longer allowed to be used in the United States, a survey of the literature indicates that these chemical residues continue to accumulate in a variety of foods including fish. Human activities can alter the distribution of the naturally occurring metals (e.g. mining, fuel combustion) and thus increase the likelihood of exposure to toxic levels of these chemicals through inhalation or ingestion of food and water.

Many of the chemical residues in fish identified in this study are not unlike levels found in fish from other studies in comparable aquatic environments in North America. The concern raised in the Columbia River Basin also gives rise to a much broader issue for water bodies throughout the United States. The results of this study, therefore, have implications not only for tribal members but also the general public.

While contaminants remain in fish, it is useful for people to consider ways to still derive beneficial effects of eating fish, while

### **Recommendations for eating fish**

EPA recommends that people follow the general advice provided by the health departments for preparing and cooking fish;

**\*Remove fat and skin before cooking**

**\*While cooking, allow fat and oil to drain**

These preparation and cooking methods should help to reduce exposures to PCBs, DDTs, dioxins, and furans, and other organics which accumulate in the fatty tissues of fish.

**Note:** It is also important to consider the health benefits of eating fish. While fish accumulate chemicals from the environment they are also an excellent source of protein that is low in saturated fats, rich in vitamin D and omega-3 fatty acids, as well as other nutrients.

at the same time reducing exposure to these chemicals. Fish are a good source of protein, low in saturated fats, and contain oils which may prevent coronary heart disease. Risks can be reduced by decreasing the amount of fish consumed, by preparing and cooking fish to reduce contaminant levels, or by selecting fish species which tend to have lower concentrations of contaminants.

The results of this study confirm the need for regulatory agencies to continue to pursue rigorous controls on environmental pollutants and to continue to significantly reduce those pollutants which have been dispersed into our ecosystems. Reducing dietary exposure through cooking or by eating a variety of fish will not eliminate these chemicals from the environment. Elimination of many of the man-made chemicals from the environment will take decades to centuries. Regulatory limits for new waste streams and clean up of existing sources of chemical wastes can help to reduce exposure. The exposure to naturally occurring chemicals can be reduced through better management of our natural resources.

There are many uncertainties in this risk assessment which could result in alternate estimates of risk. These uncertainties include our limited knowledge of the mechanisms which cause disease, the variability of contaminants in fish and fish ingestion rates, and the effects of food preparation. The uncertainties in our estimates may increase or decrease the risk estimates reported in this study.



## **1.0 Introduction**

### **1.1 Report Organization**

This report presents the results of an assessment of chemicals in fish and the risk estimates from consuming these fish based on data analysis and conclusions reached by EPA. It is organized into five volumes.

The study results are presented in 10 sections in Volume 1. Sections 1 and 2 describe the study background, methods, and the chemical concentrations in fish tissues. Sections 3,4, and 5 describe risk assessment methods. The risk characterization is presented in Section 6 for all chemicals except lead and radionuclides. Lead and radionuclide risk characterizations are presented in sections 7, and 8, respectively. The fish tissue residues from this study are compared to other fish contaminant studies as well as other food types in Section 9. Uncertainties in this study are presented in Section 10. The discussion of uncertainty includes all aspects of the risk assessment as well as the sections on fish tissue concentrations (Section 2) and the comparisons with other studies (Section 9). The uncertainty section contains additional calculations to show how the characterization of cancer risk and non-cancer hazards would change if different values had been used to estimate exposure or to characterize toxicity. Finally, conclusions for this study are discussed in Section 11.

Volume 2 provides all the chemical data from the results of the study, as well as sex, length and weight of the fish, and other descriptive data on fish collection. Volume 3 is the Field Operations Manager sampler's notebook(s) which provides a record for the collection of samples. Volume 4 is the Quality Assurance Report which includes a review of the field activities, sample preparation, laboratory measurements, quality assurance procedures, system audits, corrective actions, and the data quality assessment. The appendices to this volume contain all the project data including information about the field sampling locations. Volume 5 is the Quality Assurance Project Plan which was prepared in 1996. The Quality Assurance Project Plan contains the documentation for the study design, objectives, methods, and quality control procedures.

### **1.2 Study Background**

After reviewing the results of the EPA 1989 national survey of pollutants in fish (USEPA, 1992a), EPA became concerned about the potential health threat to Native Americans who consume large amounts of fish from the Columbia River Basin. The cause for concern for native peoples in the Columbia River Basin was also raised by the Columbia River Intertribal Fish Commission (CRITFC) and its member tribes<sup>4</sup>.

In order to evaluate the likelihood that tribal people may be exposed to high levels of

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<sup>4</sup>All references to "tribes" in this report are only applicable to CRITFC's member tribes: Confederated Tribes of Warm Springs, Yakama Nation, Umatilla Confederated Tribes, Nez Perce Tribe. They are collectively referred to as CRITFC's member tribes.

contaminants in fish tissue EPA, CRITFC and its member tribes designed a study in two phases. The first phase of this study was a fish consumption survey which was completed in 1994 by CRITFC (CRITFC, 1994). The results of this survey documented the importance of fish in the diet and culture of CRITFC's member tribes. The types and amounts of fish that were eaten by the four CRITFC's member tribes were identified. The primary fish that were consumed by CRITFC's member tribes were salmon and trout. The survey also demonstrated that the average daily fish consumption for adults (63.2 g/day) of CRITFC's member tribes was much higher than the national average for adults (6.5 g/day)<sup>5</sup>. This survey accentuated the need to complete a survey of contaminants in fish tissue to provide information on the quality of the fish being consumed by CRITFC's member tribes.

The plans for the fish contaminant survey began with the formation of a multi-agency task force with representatives from EPA, CRITFC, the Yakama Nation, the Umatilla Confederated Tribes, the Nez Perce Tribe, the Warm Springs Tribe, the Washington Departments of Ecology and of Health, the Oregon Departments of Environmental Quality and Health, the US Geological Survey (USGS), and the US Fish and Wildlife Service. A Memorandum of Agreement signed by EPA and CRITFC in 1996 established the basis for the continued interaction of the EPA staff and tribal members to complete the contaminant survey. With the help of members of CRITFC's member tribes as well as state and federal fish hatchery personnel, sample collection took place between 1996 and 1998. Chemical analyses were completed in 1999. The analyses were done by EPA and commercial laboratories.

This study was designed to estimate risks for a specific group of people (CRITFC's member tribes). The CRITFC fish consumption survey combined information from all the member tribes into a single distribution, therefore, the risk estimates in this study do not represent the risks of any specific tribe.

The types of fish, tissue types, and sampling locations were selected by the CRITFC's member tribes. Fish collection locations were selected because they were important to characterizing risks to CRITFC's member tribes. Chemicals were chosen because they were identified in other fish tissue surveys of the Columbia River Basin as well as being common contaminants found in the environment.

This type of sampling is biased with unequal sample sizes and predetermined sample locations rather random. This bias is to be expected when attempting to provide information for individuals or groups based on their preferences. The results of this survey should not be extrapolated to any other fish or fish from other locations.

The exposure assumptions used to estimate risk for CRITFC's member tribes were also predetermined from CRITFC fish consumption survey (CRITFC, 1994). While the study was designed to assess fish which were known to be important to CRITFC's member tribes, it was

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<sup>5</sup>The average fish ingestion used by the EPA in risk assessments for the general public was changed from 6.5 g/day to 7.5 g/day in 2000 (USEPA 2000a)



assumed that other people would be concerned about the contaminant levels in fish from the Columbia River Basin. This decision to estimate risks for the general public was determined after the chemical analyses were completed. Thus, the consumption patterns used this assessment for the general public were not specific to people who eat fish from the Columbia River Basin. However, the risk estimates provide a point of departure for discussions of levels of contamination in the fish from this river basin.

The objectives of this study of chemical residues in the fish from the Columbia River Basin were to determine:

- 1) if fish were contaminated with toxic chemicals,
- 2) the difference in chemical concentrations among fish species and study sites, and
- 3) the potential human health risk due to consumption of fish from the Columbia River Basin.

This contaminant survey also provided information on those chemicals which were most likely to be accumulated in fish tissue and therefore pose the greatest risks to people.

### **1.3 Study Area**

The Columbia River Basin dominates more than a dozen ecological regions as it flows 1,950 km from its source, Columbia Lake, located near the crest of the Rocky Mountains in British Columbia, to the Pacific Ocean. The Columbia River drains an area of about 670,800 km<sup>2</sup> of which about fifteen percent is in Canada. Eleven major tributaries enter the river: Cowlitz, Lewis, Willamette, Deschutes, Snake, Yakima, Spokane, Pend Oreille, Wenatchee, Okanagan, and Kootenay Rivers (Lang and Carriker, 1999). The study was confined to the Columbia Basin below Grand Coulee to the north, the Clearwater River to the east, just below Bonneville Dam to the west and the Willamette River to the south(Figure 1-1).

### **1.4 Sampling Locations**

One hundred and two fishing locations were identified by the Yakama, Nez Perce, Umatilla, and Warm Springs tribal biologists. Due to resource constraints, all of these sampling locations could not be sampled. The study design (Volume 5) presents in detail the process that was used to reduce the number of sampling locations. Initially fishing locations that represented greater than 40% of each CRITFC's member tribes' fishing use for resident and anadromous fish species were identified. The number of fishing locations was further reduced by selecting sampling locations at the base of a watershed to represent the entire watershed (98, 30,101, 96) and limiting the number of sampling locations on the mainstream Columbia River to each of the dam reaches (6, 7,8,9,14). Additional sampling locations (48,49) were added because they were near local pollution sources. Sample location 49 on the Yakima River was also important for rainbow trout spawning (personal communication CRITFC's member tribes). Other sampling locations (3, 21,21b, 62,63) were selected because of the concern for a particular fish species.

The final sampling locations were located on 16 rivers and creeks and the mainstream Columbia (Figure 1-1, Table 1-1). The actual *sampling locations* were variable within a study reach because of the sampling techniques and/or mobility of fish species. To simplify the data analysis, similar *sampling locations* within a study reach were combined to yield one *study site*. The river miles for *sampling locations* are presented in Table 1-1. The latitude and longitude for each *sampling location* is presented in Volume II, Appendix A-2.



**Table 1-1. Description, study site, sampling location, and river mile for Columbia River Basin fish sampling 1996-1998. Some of the *sampling locations* (S. Location) are combined into a single site for this study (SS = *study site*). Fish species are also listed. RM = river mile**

<b>Waterbody</b>	<b>SS</b>	<b>S. Location</b>	<b>RM</b>	<b>Fish Species</b>
Columbia River below Bonneville Dam	3	3B	39-41	eulachon
Columbia River between Bonneville dam and Dalles dam	6	6C	154-155	white sturgeon
Columbia River between Dalles dam and John Day dam	7	7B,D 7A	203-207 197.5	walleye white sturgeon
Columbia River between John Day dam and McNary dam	8	8B,D,E,F,G,H,I	216-292	largescale sucker, white sturgeon, fall chinook salmon, steelhead trout
Columbia River below confluence with Snake River	9 L	9A,B,C,D	295-304	white sturgeon
Columbia River (Hanford Reach)	9 U	9 E,F,G, H, I, 9 N,O, P, Q	369-372 389-393	largescale sucker, white sturgeon mountain whitefish
Columbia River just below Priest Rapids Dam	14	14 hatchery	396	fall chinook salmon
Wind River	63	63 hatchery	18	spring chinook salmon
Little White Salmon River	62	62 hatchery	1	spring chinook salmon
Fifteen mile Creek	24	24	0.2-0.5	Pacific lamprey
Hood River	25	25	4	steelhead
Willamette Falls	21	21	26.6	Pacific lamprey
MF Willamette River	21B	21B-hatchery	203.6	spring chinook salmon
Deschutes River	98	98 A,B,C,D,E	55-59	mountain whitefish, rainbow trout, largescale sucker
Umatilla River at the mouth	30	30 30A , 30B	3 0-1	spring chinook salmon, coho salmon, fall chinook salmon largescale sucker, walleye,
Umatilla River upper river	101	101,101A	88.5-89.5	mountain whitefish, rainbow trout
Thomas Creek		101B	1.5-2.5	mountain whitefish, rainbow trout
Meacham Creek		101C	2-2.5	rainbow trout
Yakima River below Roza Dam	48	48 F, G  48 H, I, J	47.1  81-85	bridgelip sucker, largescale sucker, spring chinook salmon, fall chinook salmon, steelhead, mountain whitefish, spring chinook salmon, largescale sucker
Yakima River above Roza Dam	49	49	139-141	largescale sucker, rainbow trout
Klickitat River	56	56 56A hatchery 56 B, F	2.2 42.5 64-84	fall chinook salmon, steelhead spring chinook salmon rainbow trout
Snake River below Hell's Canyon Dams	13	13C,D,E,F	128-135	largescale sucker, white sturgeon
Snake River above Hell's Canyon Dams	93	93A hatchery	270	steelhead
Clearwater - Snake River	96	96 hatchery	40.5	steelhead
Looking Glass Creek - Grand Ronde	94	94 hatchery	0.1	spring chinook salmon
Icicle Creek - Wenatchee River	51	51 hatchery	2.8	spring chinook salmon

## 1.5 Fish Species

A total of 281 fish samples were collected including 132 whole body, 129 fillet, 11 egg, and 9 field duplicates (Table 1-2a,b). The fish species included anadromous fish species (Pacific lamprey, eulachon, coho salmon, fall and spring chinook salmon, steelhead) and resident fish species (largescale sucker, bridgelip sucker, mountain whitefish, rainbow trout, white sturgeon, walleye). These species were selected because of their importance to CRITFC's member tribes.

**Table 1-2a. Resident fish species collected from the Columbia River Basin, 1996 -1998. The sample location and identification number and number of replicates are given for each species.**

Fish species	Study Site	Replicates		Dup
		F	W	
White Sturgeon- <i>Acipenser transmontanus</i>	Columbia River - 6	3		1 fillet
16 single fillets without skin, BW = 9,525g - 34,927 g	Columbia River - 7	3		
8 single whole body, BW = 8,108g - 22,380 g	Columbia River - 8	3	3	
4 duplicates of single fish each	Columbia River - 9L	3	3	1 fillet
White sturgeon samples were individual fish.	Columbia River - 9U	1	2	1 fillet
	Snake River - 13	3		1 fillet
Rainbow Trout - <i>Oncorhynchus mykiss</i>	Deschutes River - 98	4	3	
7 fillet composites with skin; BW = 318g - 551 g	Umatilla River - 101		4	
Number in each composite = 7-11	Yakima River - 49	3	3	
12 whole body composites; BW = 47g - 475 g	Klickitat River - 56		2	
Number in each composite = 7 - 30				
Largescale Sucker - <i>Catostomus macrocheilus</i>	Columbia River - 8		2	
19 fillet composites with skin; BW = 809g- 1541 g	Columbia River - 9 U	3	3	
Number in each composite = 4 - 12	Umatilla River - 30	4	3	
23 whole body composites ; BW = 395g - 1,764 g	Deschutes River - 98	3	3	
Number in each composite = 5 - 12	Yakima River - 48	3	6	
	Yakima -River - 49	3	3	
	Snake River - 13	3	3	
Bridgelip sucker - <i>Catostomus columbianus</i>	Yakima River - 48		3	
3 whole body composites; BW = 588g - 637g;				
Number in each composite = 7				
Walleye - <i>Stizostedion vitreum</i>	Columbia River - 7		2	
3 fillet composites with skin; BW = 822g - 850g	Umatilla River - 30	3	1	
Number in each composite = 8				
3 whole body composites; BW = 749g - 1503g				
Number in each composite = 4 - 8				
Mountain Whitefish - <i>Prosopium williamsoni</i>	Columbia River - 9U	3	3	
12 fillet composites with skin; BW = 247g - 517g	Deschutes River - 98	3	3	1 fillet
Number in each composite = 9 - 35	Umatilla River - 101	3	3	
12 whole body composites; BW = 247g - 428 g	Yakima River - 48	3	3	
Number in each composite = 9 - 35				
1 duplicate composite				

BW = Body weight; F= fillet WB = whole body ; Dup = duplicate

**Table 1-2b. Anadromous fish species collected from the Columbia River Basin, 1996 -1998. The sample location and identification number are given for each species. The number of replicates for each tissue type are listed after the location.**

Fish Species	Study Site	Replicates			Dup
		F	WB	Egg	
Coho salmon - <i>Oncorhynchus kisutch</i> 3 fillet with skin composites; BW = 3,647g -3,960g Number in each composite = 6 3 whole body composite; BW = 2,855g - 3,455g Number in each composite = 4	Umatilla River 30	3	3	3	
Fall chinook salmon - <i>Oncorhynchus tshawytscha</i> 15 fillet composites with skin; BW = 3,790g - 10,970g Number in each composite = 4 15 whole body composites; BW = 4,160g - 8,623g Number in each composite = 6 1 egg composite ; 2 duplicate fillet composites	Columbia River - 8 Columbia River - 14* Umatilla River - 30 Yakima River - 48 Klickitat River - 56	3 3 3 3 3	3 3 3 3 3	1	1 fillet    1 fillet
Spring chinook salmon - <i>Oncorhynchus tshawytscha</i> 24 fillet composites with skin; BW = 4536g - 9373g Number in each composite = 3 - 5 24 whole body composites; BW = 4,292g - 7,058g Number in each composite = 5 6 egg composites; 1 duplicate composite	Little White Salmon River - 62* Wind River - 63** MF Willamette River - 21B** Umatilla River - 30 Yakima River - 48 Klickitat River - 56* Icicle Creek - 51* Grand Ronde River - 94*	3 3 3 3 3 3 3	3 3 3 3 3 3 3	3	      1 fillet
Steelhead - <i>Oncorhynchus mykiss</i> 21 fillet composite with skin; BW = 1,784g - 5,537g Number in each composite = 3 - 4 21 whole body composite; BW = 1,633g - 6,440g Number in each composite = 3 - 8 1 egg composite sample; 1 duplicate composite	Columbia River- 8 Hood River - 25 Yakima River - 48 Klickitat River - 56 Snake River - 93* Clearwater River - 96*	6 3 3 3 3 3	6 3 3 3 3 3	1	     1 fillet
Pacific Lamprey - <i>Lampetra tridentata</i> 3 fillet composites with skin; BW = 364g - 430g Number in each composite = 20 9 whole body composites; BW = 334g - 463g Number in each composite = 10 - 20	Fifteen mile Creek - 24 Willamette Falls - 21		3 6		
Eulachon - <i>Thaleichthys pacificus</i> 3 whole body composites BW = 37g; Number in composite = 144	Columbia River - 3		3		

\* Fish taken from hatchery Dup = duplicate; F= fillet; WB = whole body BW = average body weight of the fish in a composite

With the exception of walleye, all these fish are cold water native species which are stressed by alteration of their natural habitat (Netboy, 1980; Dietrich, 1995; Close, et. al., 1995; Musick, et. al., 2000; DeVore, et. al., 1995; Beamesderfer, et. al.,1995; Coon ,1978; Lepla, 1994). Walleye were introduced to the Columbia River Basin from the late 1800s to the early and mid 1900s and are well established in some of the reservoirs (e.g., the John Day Reservoir).

In order to estimate risks for the general public, it was assumed that these species were also consumed by other people in the basin. While there were no comprehensive surveys of fish

consumption by the general public in the Columbia River Basin at the time of this study, there have been surveys in the Middle Fork Willamette River (EVS, 1998), lower Willamette River (Adolfson Associates, Inc., 1996), and Lake Roosevelt (WDOH, 1997). The types of fish identified (Table 1-3) in these surveys include some of the same types listed in the CRITFC consumption survey (CRITFC, 1994).

**Table 1-3. Recent surveys of types of fish consumed by the general public in the Columbia River Basin.**

	<b>EVS 1998</b>	<b>Adolfson Associates</b>	<b>WDOH 1997</b>
<b>Location</b>	Middle Willamette	Lower Willamette	Lake Roosevelt
<b>Tissue Type</b>	primarily muscle some skin, eggs, eyes	muscle	fillets primarily some skin, eggs, fish heads
<b>Fish Type</b>	bullhead carp sucker bass northern pikeminnow crappie bluegill trout white sturgeon lamprey salmon steelhead	yellow perch brown bullhead northern pikeminnow starry flounder white sturgeon	rainbow trout walleye bass

## 1.6 Sampling Methods

Sampling methods (Volume 4, Appendix A) for fish included: electrofishing, hand collection, hatchery collection, trapping at dams, dip netting, fish traps, and gill netting. The preferred method was dependent on the conditions at the sampling location, selected species, and legal constraints. A global positioning system (GPS) was used to identify the latitude and longitude for each sampling location (Volume 4, Appendix A).

After retrieval from sampling devices, each fish was identified to the species level by personnel familiar with the taxonomy of the fish in the Columbia River Basin. The length and weight were then measured for each fish to ensure that they met the size class as defined in the Quality Assurance Project Plan (Volume 5). The length and weight data are provided in Volume 2, Appendix A.

Four types of samples were collected: whole-body with scales, fillet with skin and scales, fillet without skin, and eggs. The white sturgeon is the only species where fillet without skin was collected. The armor-like skin of the white sturgeon was considered too tough for ingestion. Whole-body samples were selected to maximize the chances of measuring detectable levels of contaminants of concern and because data presented in the consumption study showed that CRITFC's member tribes may consume several fish parts in addition to the fillet (CRITFC, 1994). Eggs from spring chinook salmon, fall chinook salmon, and steelhead were measured because consumption data show that their eggs were widely consumed by CRITFC's member

tribes. The fish were not scaled as recommended in the EPA guidance (USEPA, 1998a). Based on conversations with CRITFC's member tribes, it was assumed that people consume the whole body or fillet with scales intact.

The Columbia River Basin is very large and the number of samples which could be analyzed was relatively small. Due to limited resources, composites were analyzed (with the exception of white sturgeon) instead of individual fish as being a better estimate of the average concentrations of chemicals from a study site. The number of fish in each composite are listed in Volume II, Appendix A-2. It is assumed that by compositing, the error in representativeness would be reduced. However, by using an average of individual fish the true variability in individual fish tissue samples was lost. Thus, the actual residues in individual fish from the Columbia River Basin may be higher or lower than the concentrations reported in this study. Due to the size and difficulty of homogenization, composites were not taken for white sturgeon. Instead, individual fish were sampled and analyzed from each sampling location. Since this study was designed for fish consumption and people eat what they collect, random samples of fish were selected for each composite rather than predetermined age or gender.

An attempt was made to collect three replicate samples for each fish type from each study site to estimate variability between study sites. However, this was not always possible due to availability of fish and problems with sampling gear. The final number of replicates for each fish species and tissue type are listed in Table 1-2 a,b. To reduce differences due to sampling error, replicate samples were collected at the same time and study site.

## **1.7 Chemical Analysis**

The homogenization of samples, the lipid analysis, and chemical analysis of chlorinated dioxins and furans, and dioxin-like PCB congeners were conducted by AXYS Laboratory in Victoria, Canada. The remaining analyses were performed by the EPA Region 10 laboratory at Manchester, WA. Laboratory analytical protocols specified for this study are referenced in Volumes 4 and 5.

Chemical analysis of the fish tissue was completed in 1999. The fish samples were analyzed for 132 different chemicals (Tables 1-4 a,b,c,d,e,f,g), including the following classes: semi-vocatives, chlorinated dioxins and furans, dioxin-like PCB congeners, Aroclors, pesticides and selected trace metals<sup>6</sup>.

Of the 132 compounds analyzed, 40 were not detected (Tables 1-4 a,b,c,d,e,f,g). The individual chemical analyses of fish tissue samples are presented in Volume 2, and summarized in Volume 1, App D.

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<sup>6</sup> "Metals", as used in this report, also refers to metalloids or semi-metals. Antimony, selenium, boron, and arsenic are in the metalloid groups.



**Table 1-4a. 51 semi-volatile chemicals analyzed.**

<b>22 detected</b>	<b>29 not detected</b>
1,2-Diphenylhydrazine	Nitrobenzene
2,6-Dinitrotoluene	1,2-Dichlorobenzene
Acenaphthene	1,3-Dichlorobenzene
Acenaphthylene	1,4-Dichlorobenzene
Anthracene	1,2,4-Trichlorobenzene
Benz-a-anthracene	2,4-Dinitrotoluene
Benzo-a-pyrene	2-Chloronaphthalene
Benzo-b-fluoranthene	4-Bromophenyl-phenylether
Benzo-k-fluoranthene	4-Chlorophenyl-phenylether
Chrysene	bis(2-Chloroisopropyl)ether
Dibenz[a,h]anthracene	Hexachlorobutadiene
Fluoranthene	Hexachloroethane
Fluorene	Dibenzofuran
Indeno(1,2,3-cd)pyrene	2-Chlorophenol
Pyrene	4-Chloro-3-methylphenol
Phenanthrene	2,4-Dichlorophenol
Benzo(g,h,i)perylene	2,4-Dimethylphenol
Naphthalene	2,4,5-Trichlorophenol
1-Methyl-naphthalene	2,3,4,6-Tetrachlorophenol
2-Methyl-naphthalene	2,4,6-Trichlorophenol
Phenol	Pentachlorophenol
Retene	4-Chloroguaiacol
	3,4-Dichloroguaiacol
	4,5-Dichloroguaiacol
	4,6-Dichloroguaiacol
	3,4,5-Trichloroguaiacol
	3,4,6-Trichloroguaiacol
	4,5,6-Trichloroguaiacol
	Tetrachloroguaiacol

**Table 1-4b. 26 pesticides analyzed.**

<b>21 Detected</b>	<b>5 Not Detected</b>
Aldrin	gamma-Chlordane
cis-Chlordane	Heptachlor
gamma-Chlordane	Delta-HCH
oxy-Chlordane	Beta-HCH
cis-Nonachlor	Toxaphene
trans-Nonachlor	
alpha-Chlordane	
o,p' DDT	
p,p' DDT	
o,p' DDE	
p,p' DDE	
o,p' DDE	
p,p' DDE	
DDMU	
Endosulfan Sulfate	
Hexachlorobenzene	
Heptachlor Epoxide	
Alpha BHC	
Gamma-BHC (Lindane)	
Mirex	
Pentachloroanisole	

**Table 1-4c. 18 Metals analyzed.**

<b>16 detected</b>	<b>2 not detected</b>
Aluminum	Lead
Arsenic	Manganese
Barium	Mercury
Beryllium	Nickel
Cadmium	Selenium
Chromium	Thallium
Cobalt	Vanadium
Copper	Zinc
	Antimony
	Silver

**Table 1-4d. 7 Aroclors analyzed**

<b>3 detected</b>	<b>4 not detected</b>
Aroclor 1242	Aroclor 1016
Aroclor 1254	Aroclor 1221
Aroclor 1260	Aroclor 1232
	Aroclor 1248

**Table 1-4e. 13 Dioxin-like PCB congeners analyzed. All Detected**

PCB 77	PCB 157
PCB 105	PCB 167
PCB 114	PCB 169
PCB 118	PCB 170*
PCB 123	PCB 180*
PCB 126	PCB 189
PCB 156	

**Table 1-4f. 7 chlorinated dioxins analyzed. All Detected**

2,3,7,8-TCDD
1,2,3,7,8-PeCDD
1,2,3,4,7,8-HxCDD
1,2,3,6,7,8-HxCDD
1,2,3,7,8,9-HxCDD
1,2,3,4,6,7,8-HpCDD
OCDD

**Table 1-4g. 10 chlorinated furans analyzed. All Detected**

2,3,7,8-TCDF
1,2,3,7,8-PeCDF
2,3,4,7,8-PeCDF
1,2,3,4,7,8-HxCDF
1,2,3,6,7,8-HxCDF
1,2,3,7,8,9-HxCDF
2,3,4,6,7,8-HxCDF
1,2,3,4,6,7,8-HpCDF
1,2,3,4,7,8,9-HpCDF
OCDF

### 1.7.1 PCB analysis

Two methods were used for measuring PCB congeners: 1) congener analysis, and 2) Aroclor analysis. PCB congeners are a group of synthetic organic chemicals that contain 209 individual chlorinated biphenyl compounds. Each molecule of a PCB congener has 10 positions in its ringed structure which can be occupied by a chlorine atom. The placement and number of chlorine atoms into these positions determine the physical and chemical properties and the toxicological significance of the specific PCB congener molecule in question. Each unique arrangement is called a “PCB congener”. The congeners which have chlorine atoms substituted in the “para” and “meta” positions acquire a structure which is similar to chlorinated dioxins and furans.

In the congener method only those congeners (Table 1-4e) which are believed to have the same toxicological mechanisms as 2,3,7,8 tetrachlordibenzodioxin (2,3,7,8-TCDD) were measured. Of the 209 possible PCB congeners 13 were analyzed. Of these 13 congeners only 11 were considered in the risk assessment. Two of the congeners (PCB 180 and PCB 170) were included because they were in the original EPA chemical method for measuring dioxin-like PCB congeners. However, subsequent methods do not include these congeners because there was “insufficient evidence on *in vivo* toxicity” to establish toxicity factors for these congeners (Van den Berg, et al., 1998). Although PCB 81 is considered to have the same toxicological mechanism as 2,3,7,8-TCDD, EPA Method 1668 (USEPA, 1997a) did not list it as a target compound. Therefore, it was not included in this study.

Commercially available PCB congener mixtures are known in the United States by their industrial trade name, “Aroclor”. The last two digits indicate the percentage of chlorine in the compound (i.e., 42% for Aroclor 1242 and 54% for Aroclor 1254). Each Aroclor mixture is further identifiable by a specific number; i.e., “Aroclor 1242”. The “12” portion of this designation refers to the fact that the molecule contains 12 carbon atoms (bound together in two six-sided phenyl rings; e.g., a “biphenyl”). The Aroclor analysis is the most common method for measuring total PCBs.

### 1.7.2 Mercury and Arsenic analysis

Mercury and arsenic occur in organic and inorganic forms. In this study, the chemical analyses were as total mercury and total arsenic. The fish tissue concentrations that are discussed in Section 2 and Section 9 are based on the measured total mercury and total arsenic. For the purposes of the risk assessment, the total mercury concentrations were assumed to be all methylmercury. Arsenic fish tissue concentrations was assumed to be 10% inorganic arsenic in the anadromous fish tissue and 1% inorganic arsenic in the resident fish tissue.

### 1.7.3 Total Chlordane and Total DDT

The pesticides chlordane and DDT include a series of respective metabolites which are assumed to act in the same manner with respect to human exposure and toxicity. For this study, all forms of chlordane (*cis*-chlordane, *trans*-chlordane, *cis*-nonachlor, *trans*-nonachlor, and oxychlordane)

were summed as total chlordane to estimate tissue concentrations and risk estimates.

1,1,1-trichloro-2,2-*bis*(p-chlorophenyl)ethane (DDT) and its structural analogs and breakdown products: 1,1-dichloro-2,2-*bis*(p-chlorophenyl)ethylene (DDE), and 1,1-dichloro-2,2-*bis*(p-chlorophenyl)ethane (DDD) are organo-chlorine pesticides. DDT, DDE, and DDD also have two isomers: the para (p,p) and ortho- para isomers (o,p). The p,p' and o,p' isomers of each DDT structural analog (DDT, DDD, DDE) were combined into three concentration terms (DDT, DDD, DDE) for fish tissue concentrations, and for the estimate of carcinogenic risks. All the DDT structural analogs (p,p'-DDD, o,p'-DDD, o,p'-DDE, p,p'-DDE, o,p'-DDT, p,p'-DDT) were summed into a single concentration (total DDT) term to estimate non-carcinogenic risks.

Although, 1,1-*bis*(p-chlorophenyl)2 chloro-ethylene (DDMU) is another structural analog or breakdown of DDT it is not believed to exhibit the same toxicity as the other structural analogs. Therefore it was not included in the sum of DDT for fish tissue concentrations and for the risk assessment.

#### **1.7.4. Lead Risk Characterization**

Lead is not included in the risk characterization sections for other chemicals. The methods for assessing risks from exposure to lead are unique due to the ubiquitous nature of lead exposure and the reliance upon blood lead concentrations to describe lead exposure, toxicity, and risks. Human health risk assessment methods for lead also differ from other types of risk assessment because they integrate all potential sources of exposure to predict a blood lead level.

#### **1.7.5 Data Quality Validation of Chemical Analyses**

A total of 93 data validation reports (Volume 4, Appendix B) were prepared detailing the quality of project data. Data quality assessment involved the following determinations:

- 1) whether the data met the assumptions under which the data quality objectives described in Volume 5 were developed, and
- 2) whether the total error in the data was small enough to allow the decision maker to use the data.

No data were rejected in this study.

Nine field duplicate samples consisting of the opposite fillets of the same species and same type of sample were collected to estimate the error in sample preparation and analysis (see Table 1-2a-b for list of field duplicates). The range in duplicate concentrations is discussed in Section 10.

All the chemicals analyzed in fish tissue were within the requirements of the quality assurance limits. In the quality assurance review of the chemical data, certain chemical concentrations were qualified with a "J". The "J" qualifier designates a concentration which is estimated. Therefore, the analytical methodology suggests that the "J" qualified measurement may be

inaccurate. We chose to use these data in this study without conditions. No data were rejected.

### **1.7.6 Detection limits**

The detection limits for chemicals were determined by performing a risk-based screening analysis of tissue contaminant data collected within the Columbia River Basin during the last ten years (1984-1994). The screening methods and quantitation limits are described in Volume 5. The analytical methods were chosen to provide detection or quantitation limits which were as low as possible within the constraints of available methods and resources.

The detection limits varied for each sample and each chemical. The concentrations of chemicals which are found at the detection limit could be treated as a zero; alternately they could also be equal to the detection limit or somewhere in between. For this study we assumed that the concentration of a particular chemical was one half of the detection limit. For comparison, the tissue chemical concentrations are presented in Appendix E assuming the concentration for a particular chemical equals 1) zero, 2) the detection limit, or 3)  $\frac{1}{2}$  the detection limit

The following rules were used when calculating average chemical concentrations in fish tissue:

- 1) If a chemical was not detected in any sample for a given fish species and sample type, it was assumed to not be present and was not evaluated.
- 2) If a chemical was detected at least once in samples for a given fish species and sample type, a concentration equal to one-half the detection limit was assumed for values reported as not detected when calculating the average chemical concentration.
- 3) The paired duplicate sample concentration for a fish at a site was averaged to obtain one concentration for that fish at that site. In cases where one duplicate was reported as a measured concentration and the paired duplicate as a non-detected concentration, the measured concentration and one-half the detection limit for the non-detected value were averaged to obtain a single estimate of concentration. In cases where both duplicate samples were not detected, one-half the detection limit for each sample was used as the mean chemical concentration.

### **1.7.7 Statistical Data Summaries**

All fish residue data are presented on a wet weight basis. All the data for each sample are included in Volume II, Appendix C. The summary statistics (average, minimum, maximum, and standard deviation) for each site and the basin are included in Volume 1, Appendix D.

The following statistical summaries include the non-detect rules described in Section 1.7.6. The data for each fish species were pooled and average chemical concentrations were calculated by site and by basin:

- 1) Site averages—All replicate samples for a given fish species and tissue type collected

at a given site were pooled to obtain an estimate of the average chemical concentration at each site.

2) Basin averages—All samples for a given fish species and tissue type collected during this study were pooled to obtain an estimate of the average chemical concentration within the basin.

## 1.8 Lipid Analysis

Most of the organic chemicals measured in this study were lipid soluble to a significant extent. The lipid content of all samples was analyzed as a measure of the likelihood of bioaccumulation of these types of organic chemicals. The percent lipid for each sample is given in Volume 4, Appendix A. The lipid normalized tissue concentrations are included in Volume 2, Appendix A.

Chemical residues were normalized to lipid using the following formula:

$$(Equation 1-1) \quad ug \text{ chemical} / kg \text{ lipid} = (ug \text{ chemical}/kg \text{ tissue} \times 100) \div \text{percent lipid}$$

For example if wet weight concentration = 40 ug DDT/kg and the percent lipid = 5%  
 $(40 \mu\text{g}/\text{kg} \times 100) \div 5 = 800 \text{ ug DDT}/\text{kg lipid}$

The lipid normalized data were not used in the risk assessment.

## 1.9 Special Studies

Three additional studies were added after the original study was initiated:

- 1) fish tissue chemical concentrations in channel catfish and smallmouth bass,
- 2) exploratory study of acid-labile pesticide analysis using Gas Chromatograph/Atomic Emission Detector (GC/AED) methods for a limited number of samples, and
- 3) radionuclide analysis for fish possibly exposed to potential releases from the Hanford Nuclear Facility.

### 1.9.1 Channel Catfish and Smallmouth Bass

Due to interest in comparing the results of this study with other Columbia River Basin surveys, two additional species (channel catfish and smallmouth bass) were added to the initial study when additional resources became available (Table 1-5).

**Table 1-5. Sampling study sites and numbers of replicates for survey of chemicals in tissues of smallmouth bass and channel catfish collected in the Columbia River Basin, 1996-1998.**

Species	Study site	Replicates	
		FS	WB
Channel Catfish - <i>Ictalurus punctatus</i>	Columbia River - 8	2	3
5 fillet with skin composites; BW = 1,236g - 2,555g Number in each composite = 2	Yakima River - 48	3	3
6 whole body composites; BW = 734g - 1,135g Number in each composite = 5 - 6			
Smallmouth Bass - <i>Micropterus dolomieu</i>	Yakima River -48	3	3
3 fillet with skin composites; BW = 1,413g - 1463g Number in each composite = 3			
3 whole body composites; BW = 1,313g - 1,487g Number in each composite = 3			

FS = fillet with skin; WB = Whole body BW= average body weight of fish in a composite

Since these were not species which were consumed in large amounts by CRITFC's member tribes, the assessment of chemicals in these fish were not included in the discussion of fish tissue concentrations in Section 2 or in the risk assessment (Sections 3-8). The results of chemical analyses in these fish are discussed in Section 9.

### 1.9.2 Acid-Labile Pesticides

In addition to the basic set of chemical analyses, EPA Region 10's laboratory measured 76 acid labile pesticides using advanced EPA Gas Chromatography/Atomic Emission Detection (GC/AED) method 8085 (Volume 5, Table 12). Of the 76 acid-labile pesticides measured only 17 were detected (Table 1-6). Method 8085 is applicable to the screening of semi-volatile organohalide, organophosphorus, organonitrogen, and organosulfur pesticides that are amenable to gas chromatography.

The chemical analytical results are included in Appendix L. Risk estimates were not completed for the acid labile pesticides. These analyses were done to ascertain only the presence or absence of these chemicals. A description of these chemicals is included in the toxicity profiles (Appendix C).

**Table 1-6. AED pesticides detected in fish tissue from the Columbia River Basin, 1996-1998.**

Atrazine	DACTHAL-DCPA	Endosulfan II	Pentabromodiphenyl ether
Bromacil	Dichlorobenzophenone	Endosulfan Sulfate	Propargite
Chlorpyrifos	Dieldrin	Hexabromodiphenyl ether	Tetrabromodiphenyl ether
Chlorpyrifos-methyl	Endosulfan I	Pendimethalin	Triallate
			Trifluralin

### 1.9.3 Radionuclide analyses

Due to the possibility of radionuclide contamination of fish in the mainstream Columbia River a subset of fish samples was selected for radionuclide analysis. These samples were collected in the mainstream Columbia River (sites 7, 8, 9L, 9U) and cooling ponds (K ponds) on the Hanford Reservation (Table 1-7). Additional samples were collected from the Snake River (Study Site 13)

as a background or reference sample for the samples collected at or in the vicinity of the Hanford Nuclear Facility.

**Table 1-7. Radionuclide fish tissue samples including study site, species, and number of replicates from the Columbia River Basin, 1996-1998.**

Study Site	Fish species	Replicates*		Duplicate
		F	WB	
Columbia River 7	white sturgeon	3		
Columbia River 8	white sturgeon	3	3	
	channel catfish	1	3	
	largescale sucker		2	
Columbia River 9 lower (L)	white sturgeon	3	3	1 whole body
Columbia River 9 upper (U)	white sturgeon	2	2	2 fillet
	mountain whitefish	3	3	1 whole body
	largescale sucker	3	3	
Hanford Reservation cooling ponds - 9K	white sturgeon		3	
Snake River 13	white sturgeon	3		1 fillet

\* each replicate was a composite of 4-35 fish except white sturgeon which were single fish; Fillets were with skin, except white sturgeon which were fillets without skin; F - fillet; WB = whole body;

Radionuclides ( Table 1-8) were measured by EPA National Air and Radiation Environmental Laboratory (NAERL) in Montgomery, Alabama, and a commercial laboratory (Barringer Laboratory) in Golden, Colorado.

**Table 1-8. The radionuclides analyzed in fish tissue collected in the Columbia River Basin 1996-1998.**

Uranium -234	Plutonium -239	Bismuth-214	Lead-212	Radon-224	Tellurium-208
Uranium-235+D	Strontium-90+D	Bismuth-212	Lead-214	Radon-226+D	Thorium-228+D
Uranium-238+D	Potassium-40	Cesium 137+D			

NAREL is a comprehensive environmental laboratory managed by the EPA Office of Radiation and Indoor Air. Among its responsibilities, NAREL conducts a national program for collecting and analyzing environmental samples from a network of monitoring stations for the analysis of radioactivity. This network has been used to track environmental releases of radioactivity from nuclear weapons tests and nuclear accidents.

Quality assurance requirements for the 45 samples (see Volume 4, Appendix A, Table A-1) selected for radionuclide measurements are described in the Quality Assurance Project Plan.. The radionuclide data are reported in Volume 1, Appendix K.

The radionuclide fish tissue measurements and risk assessment are discussed in Section 8. Radionuclides were not included with the other chemicals because radionuclides were not analyzed in all fish tissues. Although the method used to assess cancer risk from exposure to radionuclides is similar to that for other chemicals in this risk assessment, there are some unique aspects for radionuclides (e.g., analytical issues, estimation of risk coefficients) that make a separate discussion of them advantageous.

## 2.0 Fish Tissue Chemical Concentrations

In this section fish tissue chemical residues measured in this study are discussed. The fish tissue and egg samples were all composites with the exception of the white sturgeon which were individual fish. The concentrations discussed in this section include the rules for non-detected chemicals described in Section 1.7.6. In reviewing the results of this study the species were evaluated in two groups: 1) resident fish species (white sturgeon, mountain whitefish, walleye, bridgelip sucker, largescale sucker, rainbow trout) and the anadromous fish species (coho salmon, spring and fall chinook salmon, steelhead, pacific lamprey, eulachon). The resident fish species spend their life cycle in the Columbia River and its tributaries. Their exposure and uptake of chemicals will occur in fresh water in the vicinity of the locations where they were collected. The anadromous species spend most of their life cycle in open ocean. They reproduce in fresh water, but feed at sea. Therefore, their uptake of chemicals is likely to occur at sea rather than at the site where they were collected.

There were not equal numbers of samples of fish species or tissue types (Table 1-2a,b). In particular, the bridgelip sucker, coho salmon and eulachon were each collected at only one location; Pacific lamprey and walleye at only two locations. Thus the data reported for these species were not indicative of concentrations throughout the basin. Bridgelip sucker and eulachon were only collected as whole body fish tissue. Bridgelip sucker were collected opportunistically at this particular site. However, they were not part of the original study design. The eulachon were small fish. Therefore, it was necessary to collect 144 individual fish for each composite to obtain enough tissue for analysis. It was also impractical to attempt to fillet these fish. Therefore only whole body samples were collected. Despite these many variables, general trends in the monitoring of pollutants in these various species and tissues were evident.

The method for combining duplicate samples in this study was to average the duplicates. Thus, the two measurements would be treated as one number for the purposes of this assessment. The non-detects were included in the data summaries at  $\frac{1}{2}$  their detection limits. The actual detection limit is noted on the tables and in the text with a symbol for less than (<). See Sections 1.7.6 and 1.7.7 for a detailed description of these methods.

The basin-wide and study site specific average chemical concentrations reported in this section were used as the exposure concentrations in the estimation of risks discussed in Section 6.

### 2.1 Percent Lipid

The egg samples from the chinook salmon, and steelhead, had the highest percent lipid of all the fish tissue samples (Figure 2-1). The whole body and fillet tissues of Pacific lamprey and spring chinook salmon, and the whole body eulachon had higher percent lipid than the whole body or fillet tissues of any other species. Coho salmon, rainbow trout, walleye fillets, and largescale sucker had the lowest percent lipid.

With the exception of the walleye samples there was not a large difference in lipid content of whole body and fillet samples. The average whole body walleye samples contained 8% lipid as



compared to the 1.5% from the walleye filets. The technique used to fillet the samples was to keep as much of the skin and associated fatty tissue (lipid) intact. Thus, the chance of finding a clear differentiation between fillet and whole body was not preserved.

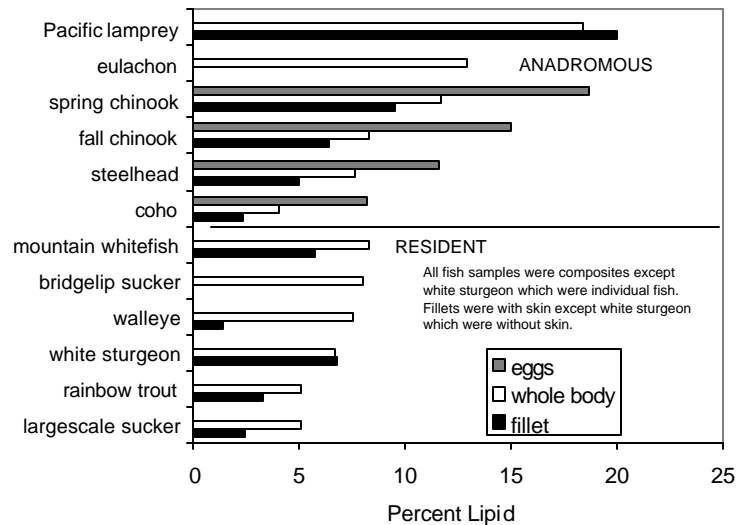


Figure 2-1. Basin-wide average percent lipid in fish collected from the Columbia River Basin. Study sites are described in Table 1-1. Sample numbers for each species are listed in Table 1-2.a,b

## 2.2 Semi-Volatile Chemicals

The semi-volatile chemicals include the guaicol, ethers, phenols, and polynuclear aromatic hydrocarbons (PAH). The number of samples with detectable levels of the semi-volatile chemicals was quite low (Table 2-1a,b). The guaicol and ethers were not detected in any sample. There were no semi-volatile chemicals detected in the fall chinook salmon or coho salmon tissue samples. The phenols were detected in only one white sturgeon sample from the main-stem Columbia River (study site 8). Many of these semi-volatile chemicals were not detected because they were not in the fish tissue, the detection limits were too high, or the chemicals may have been metabolized or otherwise degraded to chemicals which were not included in this survey.

The average concentrations for the PAHs were quite similar across species and chemicals. Of the PAHs, 2-methyl naphthalene (Table 2-1a,b) had the highest detection frequency. Pyrene was found at the highest concentrations of all the PAHs (450 ppb) in a rainbow trout collected from the upper Yakima River (study site 49). The largescale sucker was the fish species with the most frequent detection of PAHs. This may be due to the large number of largescale sucker samples rather than some unique exposure.

**Table 2-1a. Basin-wide composite concentrations\* of semi-volatile chemicals detected in resident fish species**

Species/Chemical	T	N	F	µg/kg		Species/Chemical	T	N	F	µg/kg	
				Max	Ave					Max	Ave
<b>bridgelip sucker</b>						<b>rainbow trout</b>					
1,2-Diphenylhydrazine	WB	3	1	14	7	Anthracene	WB	12	1	27	5
Naphthalene, 1-methyl-	WB	3	1	10	5	Fluoranthene	WB	12	1	53	12
Naphthalene, 2-methyl-	WB	3	3	20	16	Naphthalene, 2-methyl-	FS	7	3	11	5
<b>largescale sucker</b>						Naphthalene, 2-methyl-	WB	12	1	27	6
1,2-Diphenylhydrazine	WB	23	1	120	12	phenanthrene	WB	12	1	50	9
9H-Fluorene	WB	23	1	26	5	Pyrene	WB	12	1	450	46
Acenaphthene	WB	23	1	53	11	Retene	WB	12	1	53	12
Acenaphthylene	WB	23	2	26	5	<b>walleye</b>					
Benzo(a)anthracene	FS	19	1	24	5	Naphthalene, 1-methyl-	WB	3	1	10	6
Benzo(a)pyrene	FS	19	1	24	5	Naphthalene, 2-methyl-	FS	3	2	10	6
Benzo(g,h,i)perylene	FS	19	1	47	10	Naphthalene, 2-methyl-	WB	3	1	16	9
Benzo[b]Fluoranthene	FS	19	1	24	5	<b>white sturgeon</b>					
Benzo[k]fluoranthene	FS	19	1	24	5	Naphthalene, 1-methyl-	FW	16	1	15	4
Chrysene	FS	19	1	24	5	Naphthalene, 2-methyl-	FW	16	1	25	5
Dibenz[a,h]anthracene	FS	19	1	47	10	Phenol	WB	8	1	530	230
Indeno(1,2,3-cd)pyrene	FS	19	1	47	10	<b>mountain whitefish</b>					
Naphthalene	WB	23	1	67	12	2,6-Dinitrotoluene	WB	12	1	40	16
Naphthalene, 1-methyl-	WB	23	2	26	5	Acenaphthene	WB	12	1	31	9
Naphthalene, 2-methyl-	FS	19	2	24	5	Naphthalene, 2-methyl-	WB	12	3	10	5
Naphthalene, 2-methyl-	WB	23	7	26	8						
Phenanthrene	WB	23	1	95	7						
Pyrene	WB	23	2	53	10						
Retene	WB	23	2	200	16						

**Table 2-1b. Basin-wide composite concentrations\* of semi-volatile chemicals detected in anadromous fish species from the Columbia River Basin, 1996-1998.**

Fish Species	T	N	F	µg/kg	
				Max	Ave
<b>eulachon</b>					
9H-Fluorene	WB	3	1	170	56
Naphthalene, 2- methyl	WB	3	1	11	6
Phenanthrene	WB	3	1	170	60
<b>Pacific lamprey</b>					
Fluoranthene	WB	9	1	50	14
Naphthalene, 1- methyl	WB	9	4	25	12
Naphthalene, 2- methyl	FS	3	1	77	42
Naphthalene, 2- methyl	WB	9	4	44	22
Phenanthrene	WB	9	3	25	10
<b>spring chinook salmon</b>					
Acenaphthene	WB	24	1	81	13
Naphthalene, 2-methyl	FS	24	4	29	6
Naphthalene, 2-methyl	WB	24	5	40	8
Pyrene	WB	24	2	120	18
<b>steelhead</b>					
1,2-Diphenylhydrazine	FS	21	1	100	7
1,2-Diphenylhydrazine	WB	21	1	26	6
2,4-Dinitrotoluene	FS	21	2	48	9
2,4-Dinitrotoluene	WB	21	1	52	12
Benzo(a)pyrene	FS	21	1	24	5

\*All samples were composites except white sturgeon which were individual fish;

T= tissue type; N= number of samples; F= detection frequency; FS = fillet with skin; FW= fillet without skin; WB = whole body;

Ave= average; Max = Maximum

## 2.3 Pesticides

Of the 26 pesticides that were analyzed the most frequently observed pesticides were hexachlorobenzene, mirex, pentachloroanisole, chlordane and related compounds, and the DDT series of structural analogs (DDT,DDE,DDD).

The basin-wide average concentrations of all pesticide residues were compared across fish species. With the exception of rainbow trout and walleye fillets, the average pesticide residue levels in the resident fish species were higher than in the anadromous fish species (Figure 2-2). The average concentrations of total pesticide residues were highest in white sturgeon (Figure 2-2).

Of the anadromous fish species, Pacific lamprey had the highest basin-wide average concentrations of total pesticides. Pacific lamprey also had the highest lipid content of any anadromous fish species (Figure 2-1). The concentrations of pesticides in the Pacific lamprey may have been due to this high lipid content. However, egg samples which had high lipid concentrations (Figure 2-1) did not have high pesticide concentrations as one would expect for lipophilic compounds.

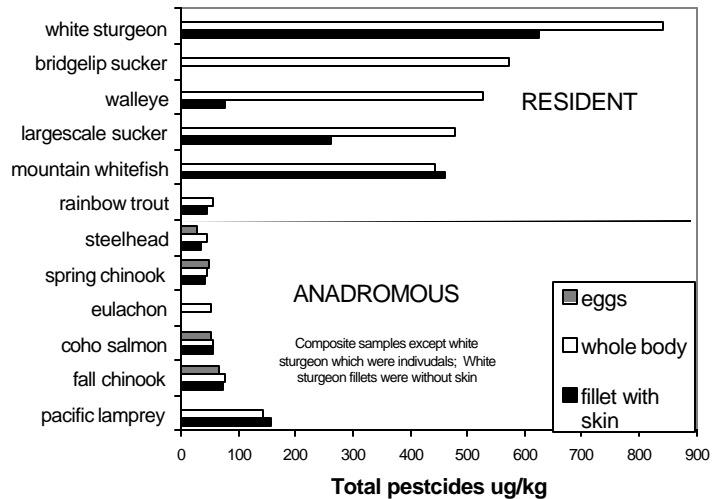


Figure 2-2. Basin-wide average concentrations of total pesticides in composite fish tissue collected from Columbia River Basin. Study sites are described in Table 1-1. Sample numbers are given in Table 1-2a,b.

### 2.3.1 DDMU, Hexachlorobenzene, Aldrin, Pentachloroanisole, and Mirex

DDMU, Aldrin, pentachloroanisole, and mirex were detected infrequently. The highest concentration (40  $\mu\text{g}/\text{kg}$ ) of DDMU was in fish tissue from largescale sucker and mountain whitefish. Aldrin was detected in only 2 species: mountain whitefish and white sturgeon (Table 2-2a). The maximum concentration (6  $\mu\text{g}/\text{kg}$ ) of aldrin occurred in mountain whitefish from the Hanford Reach of the Columbia River (study site 9U). The maximum concentration of pentachloroanisole occurred in largescale sucker (5  $\mu\text{g}/\text{kg}$ ). Mirex was only detected 9 times in all the fish tissue from this study. The maximum concentration of mirex (13  $\mu\text{g}/\text{kg}$ ) was detected in mountain whitefish. Hexachlorobenzene was detected over 100 times; most frequently in white sturgeon, spring and fall chinook salmon, and steelhead (Table 2-2a,b). The maximum concentration of hexachlorobenzene (19  $\mu\text{g}/\text{kg}$ ) occurred in white sturgeon (Table 2-2a).

**Table 2.2a. Basin-wide concentrations of pesticides in resident fish tissue from the Columbia River Basin, 1996-1998.**

Species/Chemicals	T	N	F	µg/kg		Species/Chemicals	T	N	F	µg/kg	
				Max	Ave					Max	Ave
<b>bridgelip sucker</b>						<b>white sturgeon</b>					
Endosulfan Sulfate	WB	3	3	5.4	4.6	Hexachlorobenzene	WB	8	7	19.0	9.3
<b>largescale sucker</b>						Hexachlorobenzene					
Pentachloroanisole	WB	23	4	5.0	1.1	Hexachlorobenzene	FW	16	16	13.0	5.5
Pentachloroanisole	FS	19	2	2.6	1.0	Heptachlor Epoxide	FW	16	1	2.0	1.0
Mirex	WB	23	3	5.0	1.2	DDMU	WB	8	6	16.0	7.8
Mirex	FS	19	1	2.6	1.1	Alpha-Chlordene	FW	16	1	2.4	1.0
Hexachlorobenzene	WB	23	4	5.0	1.3	Aldrin	WB	8	4	2.0	1.1
Endosulfan Sulfate	WB	23	2	6.5	1.5	Aldrin	FW	16	4	2.0	1.0
Endosulfan Sulfate	FS	19	3	2.6	1.3	<b>walleye</b>					
DDMU	WB	23	13	40.0	8.8	Mirex	WB	3	2	4.1	2.8
DDMU	FS	19	8	19.0	4.5	Hexachlorobenzene	WB	3	2	3.8	2.3
<b>mountain whitefish</b>						DDMU					
Pentachloroanisole	WB	12	3	3.0	1.3	rainbow trout	WB	2	2	8.3	8.1
Pentachloroanisole	FS	12	2	2.4	1.1	<b>rainbow trout</b>					
Mirex	FS	12	3	13.0	2.9	Pentachloroanisole	WB	12	2	5.4	1.1
Mirex	WB	12	3	6.0	2.1						
Hexachlorobenzene	WB	12	6	3.0	1.4						
Hexachlorobenzene	FS	12	3	2.4	1.0						
DDMU	FS	12	6	40.0	14.0						
DDMU	WB	12	6	31.0	13.9						
Alpha-BHC	WB	12	3	3.0	1.2						
Aldrin	FS	12	1	6.0	1.4						
Aldrin	WB	12	3	3.0	1.3						

\* All fish samples were composites except white sturgeon which were individual fish. T= tissue type; N = number of samples; F= detection frequency; Max = maximum; Ave = average; FS= fillet with skin; FW = fillet without skin; WB = whole body

**Table 2.2b. Basin-wide concentrations of pesticides in anadromous fish tissue from the Columbia River Basin, 1996-1998. All anadromous fish samples were composites.**

Species/Chemicals	Tissue Type	N	F	µg/kg	
				Max	Ave
<b>coho salmon</b>					
Hexachlorobenzene	WB	3	3	1.2	1.2
<b>fall chinook salmon</b>					
Hexachlorobenzene	WB	15	1	4.5	3.0
Hexachlorobenzene	FS	15	1	3.4	2.1
DDMU	WB	15	2	2.4	1.1
DDMU	FS	15	2	2.0	1.0
<b>spring chinook salmon</b>					
Pentachloroanisole	WB	24	6	4.2	1.1
Pentachloroanisole	FS	24	1	3.8	1.1
Hexachlorobenzene	WB	24	1	3.8	2.3
Hexachlorobenzene	FS	24	1	3.5	2.1
DDMU	WB	24	2	4.2	1.2
DDMU	FS	24	2	3.8	1.1
<b>steelhead</b>					
Hexachlorobenzene	WB	21	2	3.2	2.2
Hexachlorobenzene	FS	21	1	2.8	1.6
DDMU	WB	21	9	2.4	1.3
Endosulfan Sulfate	WB	21	3	2.1	1.0
Heptachlor Epoxide	WB	21	3	2.1	1.0
Pentachloroanisole	WB	21	2	2.1	1.0
Endosulfan Sulfate	FS	21	3	2.1	1.0
DDMU	FS	21	5	2.0	1.1
<b>pacific lamprey</b>					
Hexachlorobenzene	WB	9	6	11.0	6.3
Hexachlorobenzene	FS	3	3	8.0	7.6
DDMU	WB	9	6	6.9	3.9
DDMU	FS	3	3	5.6	4.5
Pentachloroanisole	WB	9	6	3.6	1.4
Pentachloroanisole	FS	3	3	1.7	1.6

T= tissue type; N = number of samples; F= detection frequency; Max = maximum; Ave = average; FS= fillet with skin; FW = fillet without skin; WB = whole body

### 2.3.2 Total Chlordane

Total chlordane is a mixture of several chemically related compounds (oxy-chlordane, gamma, beta and alpha chlordane, *cis* and *trans* nonachlor).

The fillet or whole body samples of bridgelip sucker, rainbow trout, eulachon, and coho salmon had no detectable concentrations of any of the chlordane compounds. The highest concentrations of total chlordane were in egg samples from the spring chinook salmon and the fillet and whole body Pacific lamprey.

The total chlordane concentrations in the whole body fish tissue samples were generally equal to or greater than the fillet samples with the exception of the Pacific lamprey where the fillet samples were slightly higher than the whole body samples (Table 2-3). The walleye samples had the most variation between whole body and fillet.

**Table 2-3 . Basin-wide average concentrations of total chlordane (oxy-chlordane, gamma, beta and alpha chlordane, *cis* and *trans* nonachlor) in fish from the Columbia River Basin, 1996-1998.**

Resident species	Fillet with skin		Whole body		Eggs	
	<i>N</i>	µg/kg	<i>N</i>	µg/kg	<i>N</i>	µg/kg
white sturgeon*	16	23	8	29		
walleye	3	6	3	20		
mountain whitefish	12	11	12	12		
largescale sucker	19	6	23	8		
rainbow trout	7	<5	12	<7		
bridgelip sucker	NS		3	<8		
<b>Anadromous species</b>						
Pacific lamprey	3	43	9	33		
eulachon	NS	NS	3	<10		
spring chinook salmon	24	7	24	8	6	66
fall chinook salmon	15	7	15	8	1	15
steelhead	21	6	21	7	1	15
coho salmon	3	<5	3	<5	3	33

\* white sturgeon were single fish and fillets without skin

*N* = number of samples; NS= not sampled; Ave = average; < = chemicals not detected

### 2.3.3 Total DDT

Total DDT is the sum of the DDT structural analogs and breakdown products: p,p' and o,p' DDT, p,p' and o,p' DDD, and p,p' and o,p' DDE. DDMU is also a breakdown product of DDT which is not believed to exhibit the same toxicity as the other breakdown products. Therefore it was not included in the total DDT concentrations for fish tissue concentrations.

The concentrations of total DDT (Table 2-4) in the salmonids (chinook, coho, rainbow, and steelhead) and eulachon were much lower than in white sturgeon, largescale sucker, whole body walleye, and mountain whitefish. The Pacific lamprey DDT concentrations were higher than the salmonids but 3 to 8 times lower than the resident species. White sturgeon had the highest concentrations followed by bridgelip sucker. This is the same pattern observed with the total pesticides (Figure 2-2). The concentration of total DDT in walleye fillet was much less than in the whole body, similar to the distribution seen with total chlordane.

The concentrations in egg samples were much lower than the fish tissue of the white sturgeon, bridgelip and largescale suckers, whole body walleye, and mountain whitefish. The concentrations in egg samples from steelhead were higher than the other egg samples and fish tissues of the anadromous species and rainbow trout.

**Table 2-4. Basin-wide average concentrations of total DDT (DDT, DDE, DDD) in composite fish tissue samples from the Columbia River Basin, 1996-1998.**

Resident Species	Fillet with skin		Whole body		Eggs	
	N	µg/kg	N	µg/kg	N	µg/kg
white sturgeon*	16	578	8	787		
bridgelip sucker	NS	NS	3	529		
walleye	3	59	3	489		
largescale sucker	19	241	23	450		
mountain whitefish	12	424	12	405		
rainbow trout**	7	29	12	38		
<b>Anadromous Species</b>						
pacific lamprey	3	95	9	90		
coho salmon***	3	41	3	42	3	39
steelhead***	21	21	21	27	1	14
spring chinook salmon	24	22	24	27	6	24
fall chinook salmon****	15	21	15	25	1	14
eulachon****	NS	NS	3	21		

N= number of samples; NS = not sampled \* white sturgeon were individual fish and fillets without skin; \*\* p,p'-DDE and p,p'-DDT were the only isomers detected; \*\*\* p,p'-DDD and p,p'-DDE were the only isomers detected; \*\*\*\*p,p'-DDE was the only isomer detected

DDT found in the environment gradually degrades to DDE. Because of it is ubiquitous, lipophilic, and persistent, DDE can be a useful surrogate in comparing fish species and study sites in terms of estimating general trends of “relative loading” from persistent and agriculturally derived organochlorines. p,p'DDE was the pesticide measured at the highest concentrations of all the DDT structural analogs in fish tissues from this study (Figure 2-3).

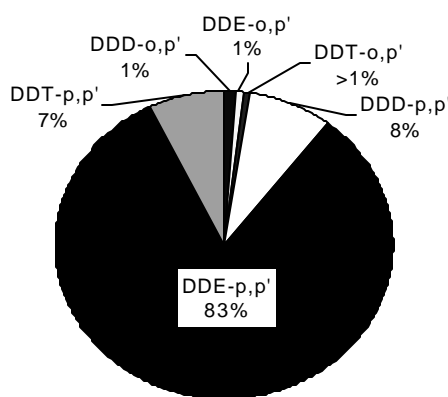


Figure 2-3. Percent contribution of DDT structural analogs to total DDT concentration in whole body largescale sucker. Basin-wide average of 23 fish tissue samples.

With the exception of walleye and rainbow trout fillet samples, the maximum concentrations of p,p'-DDE were higher in the resident fish species than the anadromous fish species (Table 2-5). The maximum concentrations were measured in the white sturgeon fillet (1400 µg/kg) and whole body largescale sucker (1300 µg/kg). The maximum concentration in the anadromous fish species was in the whole body Pacific lamprey (77 µg/kg).

**Table 2-5. Basin-wide average and maximum concentrations of p,p'DDE in composite samples of fish from the Columbia River Basin, 1996-1998.**

	Fillet With Skin				Whole Body				Egg			
			µg/kg				µg/kg				µg/kg	
	N	F	range	Ave	N	F	range	Ave	N	F	range	Ave
<b>Resident Species</b>												
white sturgeon*	16	16	100-1400	470	8	8	400-1100	620				
largescale sucker	19	19	14-740	200	23	23	28-1300	370				
mountain whitefish	12	12	8-910	360	12	12	13-770	340				
walleye	3	3	44-52	47	3	3	350-440	410				
rainbow trout	7	7	4-54	22	12	12	3-84	29				
bridgelip	NS		NS	NS	3	3	310-560	400				
<b>Anadromous Species</b>												
Pacific lamprey	3	3	46-55	50	9	9	35-77	53				
fall chinook salmon	15	15	4-26	12	15	15	5-53	15	1	1	6.6	
coho salmon	3	3	29-35	33	3	3	31-37	35	3	3	31-33	32
steelhead	21	21	5-28	11	21	21	5-33	15	1	1	6.5	
spring chinook salmon	24	24	6-18	12	24	24	11-22	15	6	6	10-16	12
eulachon	NS		NS	NS	3	3	10-11	11				

NS = not sampled; N = number of samples; F = detection frequency; Ave = average \*White sturgeon samples were single fish and fillets without skin

The chemical concentrations in replicate fish tissue samples were compared across study sites for white sturgeon, largescale sucker, and mountain whitefish (Figure 2-4).

The concentrations across study sites were extremely variable for the three fish species. The highest concentrations of p,p'DDE observed in white sturgeon were from the Hanford Reach of the Columbia River (study site 9U; Figure 2-4a). These samples were duplicate fillets from opposite sides of the same fish. The duplicate sample concentrations were similar (1300 µg/kg and 1400 µg/kg). The concentrations of p,p'DDE in the two whole body samples from this site were much lower: 540 µg/kg and 640 µg/kg. The size of the fish from which the fillets (34,927g) were collected was greater than the two whole body fish samples (-10,000 and 20,000g). This may account for the difference in p,p'DDE concentrations between the whole body and fillets at study site 9U. The fillet samples from study site 9U were quite different than the other sites on the main-stem Columbia and Snake Rivers where white sturgeon were sampled. The duplicate samples from the lower Columbia River (study site 9L; 590 µg/kg, 630 µg/kg), main-stem Columbia River (study site 6; 410 µg/kg, 590 µg/kg) and the Snake River (380 µg/kg, 420 µg/kg) were similar to each other.

The maximum concentration (1300 µg/kg) for the whole body largescale sucker was from the Yakima River below Roza Dam (study site 48; Figure 2-4b). The concentrations of p,p'DDE in whole body largescale sucker from this site ranged from 390 to 1300 µg/kg while the fillets ranged from 430- 680 µg/kg. The largescale sucker composite samples from this study site (48) included 6 replicates. The number of replicates of the largescale suckers may have accounted for the range in concentrations.

Mountain whitefish p,p'DDE concentrations were lower than the white sturgeon and largescale sucker (Figure 2-4c). The highest concentrations occurred in the Hanford Reach of the Columbia River (study site 9U) and Yakima River (study site 48) similar to the largescale sucker and white sturgeon. The p,p'DDE fish tissue concentrations in the Deschutes and Umatilla River sites were



much lower than those in the Columbia or Yakima Rivers. The concentrations of p,p' DDE in duplicate fillet samples from the Deschutes River were similar (6.6 µg/kg and 9.4 µg/kg) to each other.

**LEGEND**  
 FW = fillet without skin  
 FS = fillet with skin  
 WB = whole body

Study sites are listed by number and name and described in Table 1-1.  
 Concentration points on graphs include each duplicate and chemicals at their

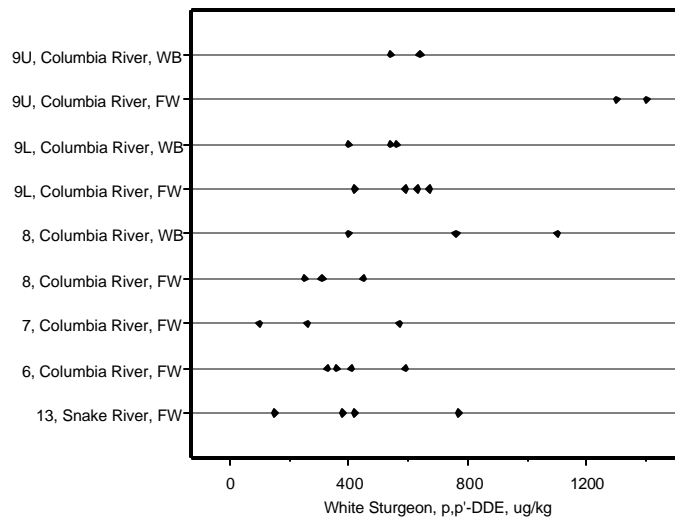


Figure 2-4a. Study site specific concentrations of p,p' DDE in white sturgeon individual fish tissue samples in the Columbia River Basin. Duplicate fillets were collected from study sites 9U, 9L, 6, and 13.

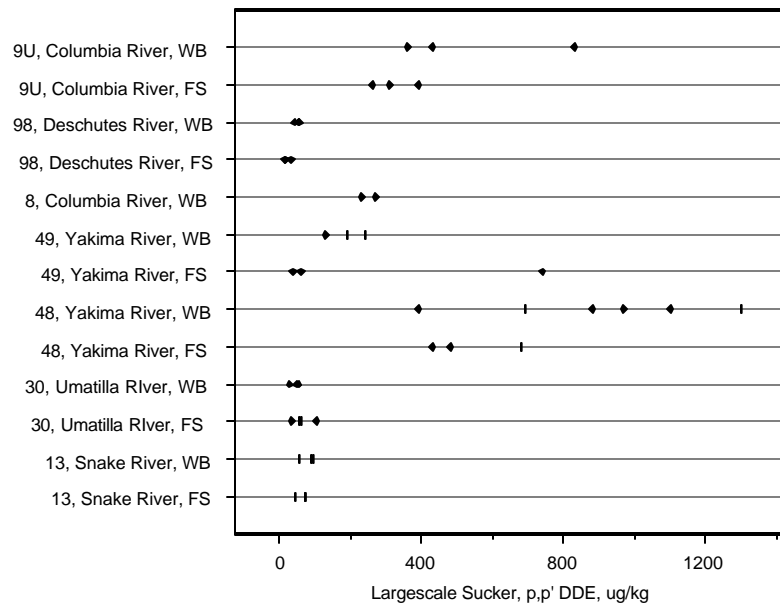


Figure 2-4b. Study site specific concentrations of p,p DDE in largescale sucker composite fish tissue samples from the Columbia River Basin.

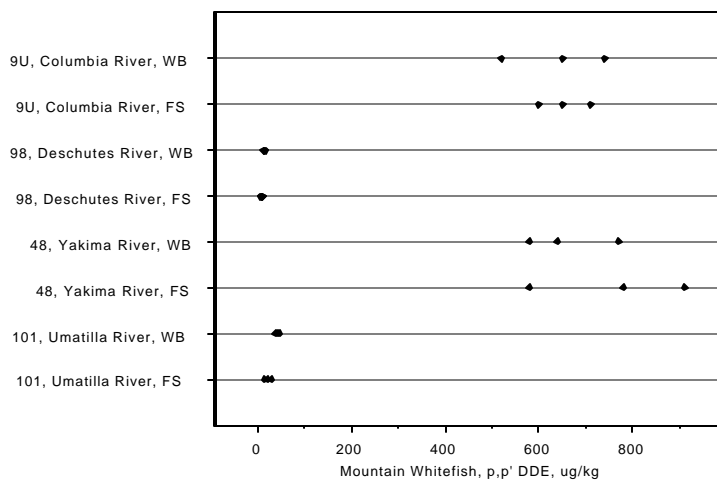


Figure 2-4c. Study site specific concentrations of p,p DDE in mountain whitefish composite fish tissue samples from the Columbia River Basin. Study site 98 includes duplicate fillet samples.

## 2.4 Aroclors

Of the seven Aroclors analyzed in this study (Aroclors: 1016,1221,1232,1248,1242,1254,1260) Aroclor 1016, Aroclor 1221, Aroclor 1232, and Aroclor 1248 never detected (Table 1-4d). The most frequently observed Aroclors were 1254 and 1260. Aroclor 1242 was only detected in the mountain whitefish samples.

The white sturgeon, mountain whitefish, whole body walleye, and Pacific lamprey had the highest concentrations of Aroclors (Table 2-6). The whole body concentrations of Aroclors in the walleye were higher than the concentrations in fillets. There were no Aroclors detected in the eulachon. The concentrations in the egg samples were similar to the anadromous fish fillet and whole body samples and less than the levels all the resident fish species except rainbow trout.

**Table 2-6. Basin-wide average concentrations of total Aroclors (1242, 1254,1260) detected\* in composite fish tissue samples from the Columbia River Basin.**

Resident Species	Fillet with skin		Whole body		Eggs	
	N	µg/kg	N	µg/kg	N	µg/kg
white sturgeon**	16	120	8	173		
walleye	3	30	3	135		
mountain whitefish	12	190	12	123		
largescale sucker	19	52	23	78		
bridgelip sucker	NS	NS	3	70		
rainbow trout	7	33	12	32		
<b>Anadromous Species</b>						
pacific lamprey	3	106	9	114		
eulachon	NS	NS	3	<57		
spring chinook salmon	24	38	24	40	6	43
fall chinook salmon	15	37	15	40	1	31
coho salmon	3	35	3	38	3	34
steelhead	21	34	21	37	1	35

< = detection limit N= number of samples: NS= not sampled.\

\*Aroclor 1242 was only detected in mountain whitefish; aroclors 1016, 1221, 1232, and 1248 were not detected in any fish or egg samples

\*\*White sturgeon samples are individual fish and fillets without skin

Aroclors 1254 and 1260 were compared across study sites for white sturgeon (Figure 2-5a,b), largescale sucker (Figure 2-6 a,b), and mountain whitefish (Figure 2-7 a,b).

The maximum concentration for Aroclor 1254 was in the mountain whitefish (930 µg/kg) fillet sample from the Hanford Reach of the Columbia River (study site 9U; Figure 2-7a). The white sturgeon fillet samples from the Hanford Reach of the Columbia River (study site 9U) had the highest concentration (200 µg/kg) of Aroclor 1260 for all species and all sites (Figure 2-5b).

Aroclor 1254 and 1260 were quite similar in white sturgeon samples (Figure 2-5a,b). The highest concentrations for both Aroclors occurred in the fillet samples from the Hanford Reach of the Columbia River (study site 9U). Aroclor 1254 concentrations in the duplicate fillet samples from study site 9U were 170 µg/kg and 210 µg/kg. The whole body concentrations from this study site

were much lower (65 µg/kg in both samples). Aroclor 1260 concentrations were 190 µg/kg and 210 µg/kg in the duplicate fillets from study site 9U and 65 µg/kg in the whole body samples. The differences in sizes of the fillet and whole body fish (discussed in Section 2.3.3) from study site 9U, may account for the difference in PCB concentrations in the fillet and whole body samples.

The next highest Aroclor 1254 concentrations were from the main-stem Columbia River (study site 6) where the duplicate concentrations were quite different (47µg/kg and 160 µg/kg;

Figure 2-5a). The percent lipid (4.8%) of the duplicate with the higher Aroclor 1254 concentration was higher than percent lipid (3.1%) in the opposite fillet. Thus, the lipid may account for the difference in tissue levels. However, the concentration of Aroclor 1260 in the duplicate fillets from this site were similar (43 µg/kg and 40 µg/kg) to each other (Figure 2-5b).

The Aroclor concentrations in the duplicate fillets for Snake River (study site 13) and for the lower Columbia River (study site 9L) were similar to each other (Figure 2-5a,b).

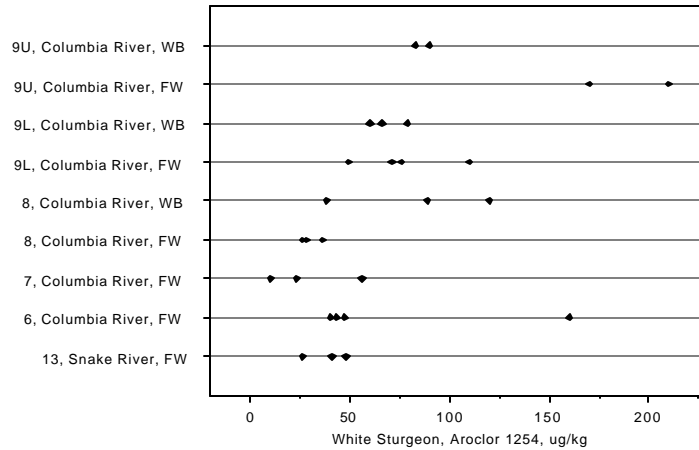


Figure 2-5a. Study site concentrations of Aroclor 1254 in white sturgeon individual fish tissue samples from the Columbia River Basin.

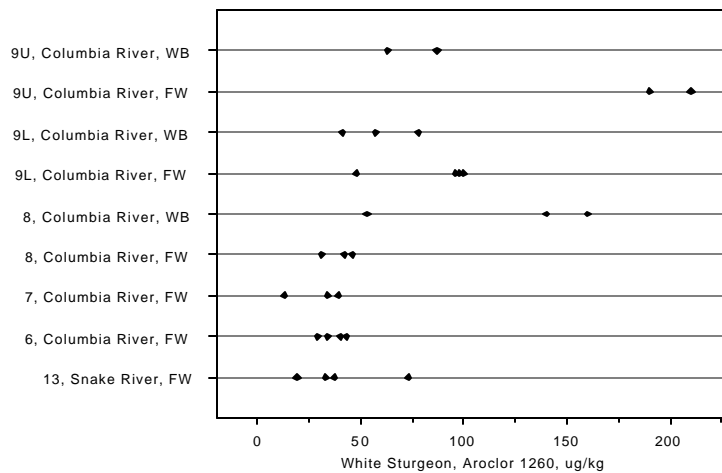


Figure 2-5b. Study site specific concentrations of Aroclor 1260 in white sturgeon individual fish tissue samples from the Columbia River Basin.

**LEGEND**

FW = fillet without skin  
 WB = whole body  
 Study sites are listed by number and name and described in Table 1-1.  
 Study sites 9u, 9L 6, and 13 include duplicate fillet samples.  
 Concentration points on graphs include duplicate fillets and chemicals at their detection limits.

The concentrations of Aroclor 1254 and 1260 were variable in largescale sucker. Aroclor 1254 ranged from <18 µg/kg in the fillet composite from the Umatilla River to 65 µg/kg in the whole body sample from the Hanford Reach of the Columbia River (study site 9U; Figure 2-6a).

Aroclor 1260 concentrations ranged from <19 µg/kg in the Snake River (study site 13) and Deschutes River (study site 98) to 100 µg/kg in several whole body samples from the Hanford Reach of the Columbia River (study site 9U) and the Yakima River (study site 48) (Figure 2-6b).

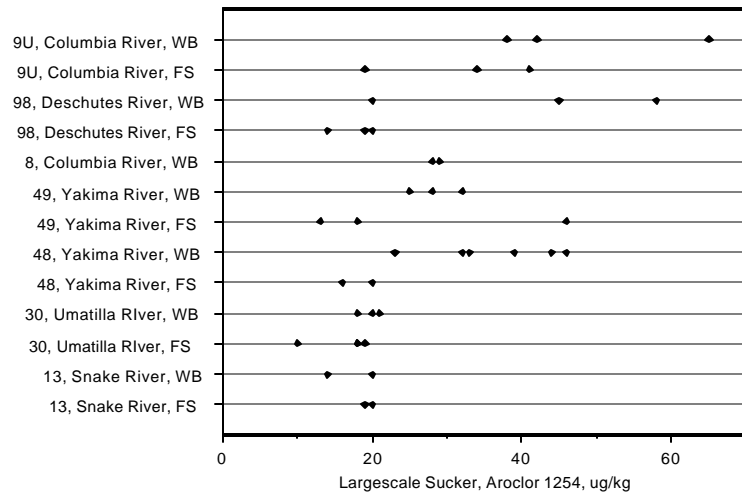


Figure 2-6a. Concentration of Aroclor 1254 in largescale sucker composite fish tissue samples from the Columbia River Basin.

**LEGEND**  
 FS = fillet with skin  
 WB = whole body  
 Study sites are listed by number and name and described in Table 1-1. Concentration points on graphs include chemicals at their detection limits.

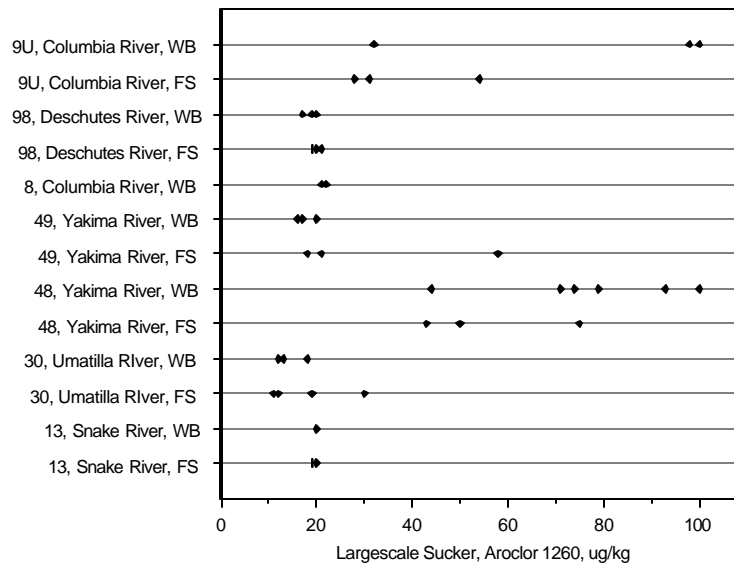


Figure 2-6b. Concentration of Aroclor 1260 in largescale sucker composite fish tissue samples from the Columbia River Basin.

In the mountain whitefish samples Aroclor concentrations from the Deschutes and the Umatilla River sites were low with <math><17 \mu\text{g}/\text{kg}</math> for Aroclor 1254 in the Umatilla River and <math><16 \mu\text{g}/\text{kg}</math> for Aroclor 1260 in the Deschutes River (Figure 2-7a,b). The duplicate fillet samples from the Deschutes River were equal or similar to each other. The maximum Aroclor 1254 concentration of 930  $\mu\text{g}/\text{kg}$  in the fillet fish tissue from the Hanford Reach of the Columbia River was much higher than the other fillet and whole body samples from this study site(Figure 2-7a). The three fillet samples from this study site had the same number of fish per composite (35), approximately the same weight (448-515g), length (352-369 mm) and percent lipid (7.9-7.7%). Thus, there was nothing in the fish size or lipid content which could account for the differences in concentrations.

The maximum Aroclor 1260 in the mountain whitefish fillet (190  $\mu\text{g}/\text{kg}$ ) was from the Yakima River (study site 48; Figure 2-7b).

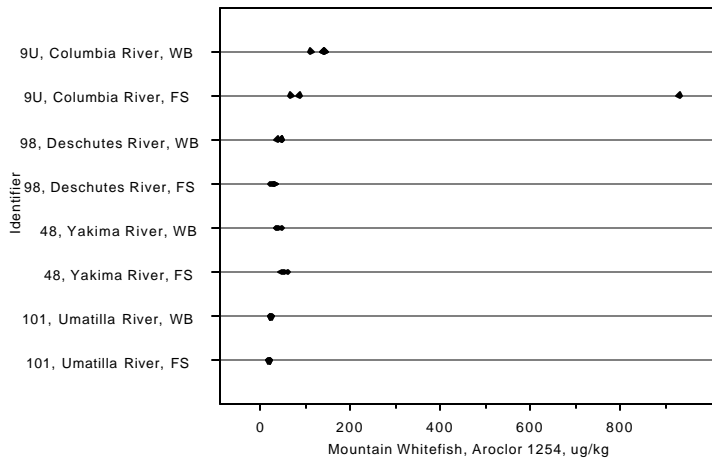


Figure 2-7a. Concentration of Aroclor 1254 in mountain whitefish composite fish tissue samples from the Columbia River Basin.

**LEGEND**  
 FS = fillet with skin  
 WB = whole body  
 Study sites are listed by number and name and described in Table 1-1  
 Study site 98 includes duplicate fillet samples.  
 Concentration points on graphs include duplicate fillets and chemicals on their detection limits. .

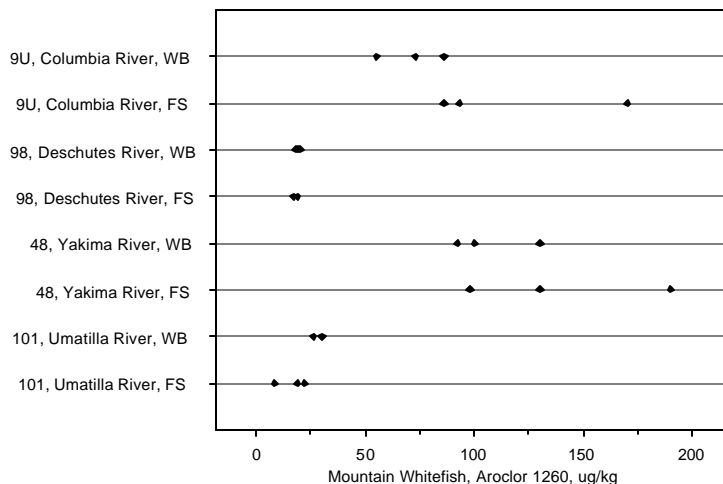


Figure 2-7b. Concentration of Aroclor 1260 in mountain whitefish composite fish tissue samples from the Columbia River Basin.

## 2.5 Dioxin-Like PCB congeners

When compared across all fish species, mountain whitefish fillet had the highest average concentration (25 µg/kg) of dioxin-like PCB congeners followed by the whole body walleye (11.7 µg/kg, Table 2-7).

There was considerable difference between the whole body walleye samples and the fillets. This was similar to the pattern observed in the walleye for DDT, chlordane, and Aroclors. This may be related to the amount of lipid in the whole body sample since dioxin-like PCB congeners are also lipid soluble similar to the pesticides.

The concentrations of dioxin-like PCB congeners (Table 2-7) in the egg samples from the anadromous fish were similar to the fillet and whole body samples of the coho salmon, eulachon, spring and fall chinook salmon, and steelhead.

**Table 2-7. Basin-wide average concentrations of the sum of dioxin-like PCB congeners in composite fish samples from the Columbia River Basin, 1996-1998.**

Resident Species	Fillet With		Whole Body		Eggs	
	N	µg/kg	N	µg/kg	N	µg/kg
		ave		ave		ave
mountain whitefish	12	25.0	12	10.2		
walleye	3	1.2	3	11.7		
white sturgeon*	16	6.5	8	10.0		
largescale sucker	19	3.1	23	5.1		
bridgelip sucker	NS		3	2.3		
rainbow trout	7	2.0	12	1.6		
<b>Anadromous species</b>						
Pacific Lamprey	3	5.5	9	5.5		
coho salmon	3	1.3	3	1.3	3	1.2
steelhead	21	1.0	21	1.1	1	0.6
fall chinook salmon	15	0.9	15	1.0	1	0.4
spring chinook salmon	24	0.8	24	1.0	6	0.8
eulachon	NS		3	0.5		

N= number of samples; NS = not sampled. \* white sturgeon were individual fish; fillets without skin

The concentrations of dioxin-like PCB congeners 118 and 105 were the major contributors to the total dioxin-like PCB congeners (Figure 2-8a,b) for resident and anadromous fish species. PCB congeners 126, 169, and 189 each contributed less than 1% to the total dioxin-like PCB congeners in mountain whitefish (Figure 2-8a) and spring chinook (Figure 2-8b). PCB 126, the most toxic dioxin-like PCB congener, was at quite low concentrations with a range of 0.0006-0.096 µg/kg in mountain whitefish fillets and 0.00081- 0.028 µg/kg in whole body. PCB 126 was not detected in 5 of the 12 samples in mountain whitefish. The range of PCB 126 concentrations in spring chinook was 0.00081-0.0046 µg/kg in fillets and 0.00052-0.0047 µg/kg in whole body. Of the 24 samples of spring chinook, 7 fillet and 8 whole body samples were not detectable.

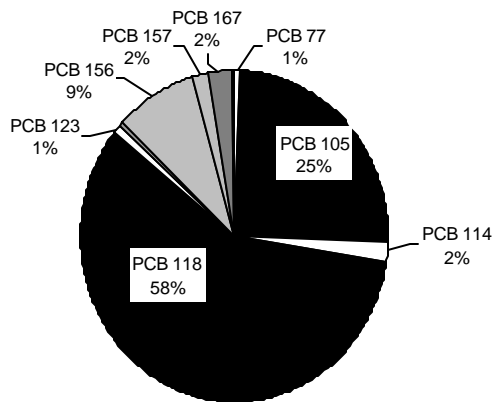


Figure 2-8a. Percent contribution of dioxin-like PCB congeners in mountain whitefish composite fillet samples from the Columbia River Basin.

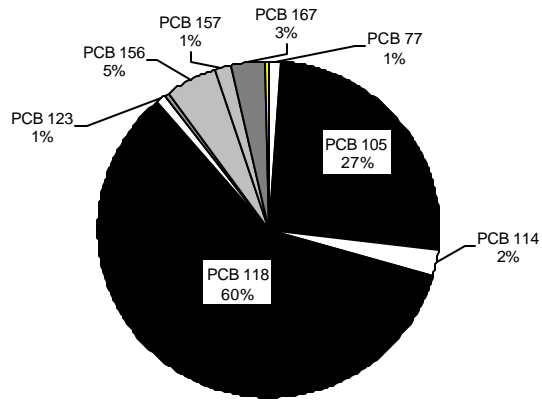


Figure 2-8b. Percent contribution of dioxin-like PCB congeners in spring chinook salmon composite fillet samples from the Columbia River Basin.

The concentrations of dioxin-like PCB congeners (Figure 2-9) were compared across study sites for white sturgeon and mountain whitefish. The average concentrations in mountain whitefish and white sturgeon fillets from the Hanford Reach of the Columbia River (study site 9U) were the highest of all the stations sampled. The levels in the lower Columbia River (study site 9L), Deschutes River, and Umatilla River were lower. The concentrations of dioxin-like PCB congeners in the white sturgeon and mountain whitefish (Figure 2-9) were consistent with the Aroclor tissue residues (Figure 2-5, 2-6, and 2-7). The white sturgeon fillet from the Hanford Reach of the Columbia River was an average of two fillets from the same fish.

The mountain whitefish were an average of three replicate composite samples with 35 fish per composite. The variability of dioxin-like PCB congener concentrations in the mountain whitefish fillets was similar to the distribution of Aroclors (Table 2-6). The mountain whitefish fillet from the Hanford Reach of the Columbia River (study site 9U) had a higher concentration (186 µg/kg) of dioxin-like PCB congeners than other replicates from that site (29µg/kg, 36 µg/kg).



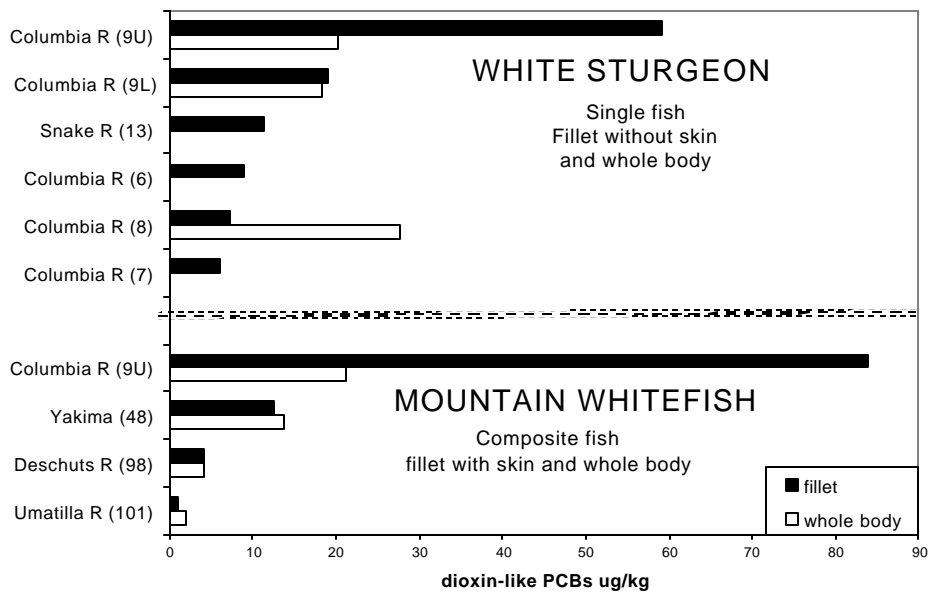


Figure 2-9. Study site average dioxin-like PCB congeners in white sturgeon and mountain whitefish samples from the Columbia River Basin. Study sites are described in Table 1-1. Sample numbers are listed in Table 1-2a,b.

The dioxin-like PCB congeners were highly correlated with Aroclors in whole body samples of fish tissue (Figure 2-10). The coefficient of determination ( $R^2$ ) for these two variables was 0.94. The coefficient of determination is a measure of the degree of association of two variables. It can range from zero to 1, with 1 being a perfect association (Sokal and Rohlf 1981). The two variables are not dependent upon each other, it is simply that they are both effects of a common cause (Sokal and Rohlf, 1981). It is also evident from this graph that the white sturgeon, walleye, and mountain whitefish had the highest average concentrations of dioxin-like PCB congeners and Aroclors.

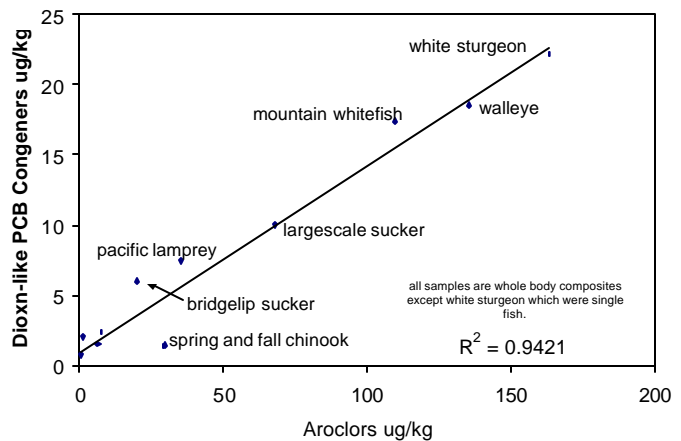


Figure 2-10. Correlation of basin-wide average concentrations of Aroclors 1242,1254,1260 (x axis) with dioxins like PCB congeners (y axis).

## 2.6 Chlorinated Dioxins and Furans

The average concentrations of chlorinated dioxins and furans in white sturgeon were higher than the all other fish by an order-of-magnitude (Table 2-8). The next highest average concentration was in the mountain whitefish. Coho salmon had the highest average concentrations of chlorinated dioxins and furans for the anadromous fish species although the levels were an order

of magnitude lower than the highest white sturgeon concentrations measured in this study. The egg samples from the steelhead and fall chinook were lower than the fillet or whole body fish tissues of all species. The egg samples from the coho salmon were higher than the other egg samples, as well as the fish tissue of spring and fall chinook salmon, steelhead, largescale sucker, and rainbow trout.

**Table 2-8. Basin-wide average concentrations of the sum of chlorinated dioxins and furans in composite fish samples from the Columbia River Basin, 1996-1998.**

Resident Species	Fillet with skin		Whole body		Eggs	
	N	µg/kg	N	µg/kg	N	µg/kg
white sturgeon*	16	0.020	8	0.030		
walleye	3	0.001	3	0.007		
mountain whitefish	12	0.006	12	0.006		
bridgelip sucker	NS	NS	3	0.003		
largescale sucker	19	0.001	23	0.002		
rainbow trout	7	0.002	12	0.002		
<b>Anadromous Species</b>						
eulachon	NS	NS	3	0.004		
pacific lamprey	3	0.003	9	0.004		
spring chinook salmon	24	0.002	24	0.002	6	0.002
steelhead	21	0.001	21	0.002	1	0.0008
fall chinook salmon	15	0.001	15	0.001	1	0.0009
coho salmon	3	0.001	3	0.008	3	0.003

N = number of samples; NS = not sampled . \*white sturgeon were individual fish; fillets without skin

Chlorinated dioxins and furans concentrations were compared across study sites for mountain whitefish, white sturgeon, and largescale sucker (Figure 2-11). The largescale sucker samples were quite low compared to the mountain whitefish and the white sturgeon. The largescale sucker concentrations of chlorinated dioxins and furans (Figure 2-11), similar to the Aroclors (Figure 2-6a,b), were much lower than the levels observed in mountain whitefish or white sturgeon. However, the largescale sucker p,p'DDE concentrations (Figure 2-4b) were equal to the levels found in white sturgeon and mountain whitefish.

The total chlorinated dioxins and furans were highest in the white sturgeon fillet from the lower Columbia River (study site 9L, Figure 2-11). The distribution of dioxins and furans in white sturgeon across sites was different than the p,p' DDE (Figure 2-4a) and Aroclor (Figure 2-5a,b) fish tissue residue distribution. The p,p' DDE and Aroclor levels were higher in the Hanford Reach (study site 9U) and study sites 6 and 8 in the Columbia River.

The mountain whitefish chlorinated dioxins and furans concentrations were highest in the Hanford Reach of the Columbia River followed by the concentrations in the Yakima River (Figure 2- 11). This distribution was similar to the p,p' DDE (Figure 2-4c) and Aroclor 1260 levels (Figure 2-7b).

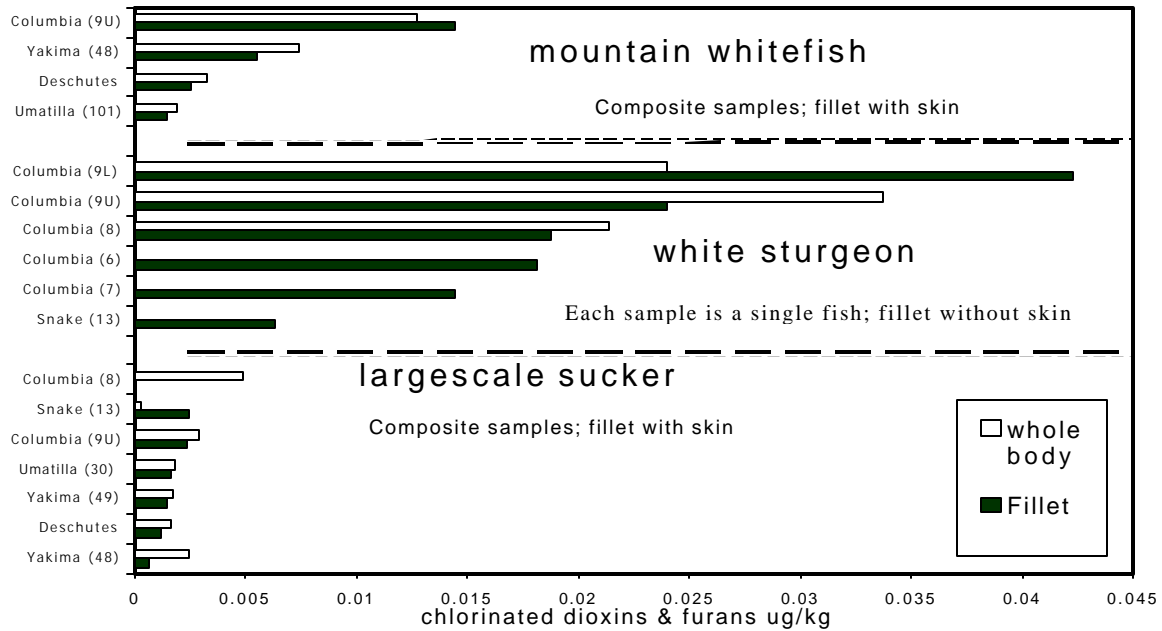


Figure 2-11. Study site average concentrations of chlorinated dioxins and furans in mountain whitefish, white sturgeon, and largescale sucker from study sites in the Columbia River Basin. Study sites are described in Table 1-1). The number of samples are listed in Table 1-2.

2,3,7,8-TCDD, the most commonly studied chlorinated dioxin was generally found at the lowest concentrations in all the samples. The most frequently detected and the highest concentrations of chlorinated dioxins and furans in fish tissue from this study were 2,3,7,8-TCDF and OCDD (Figure 2-12).

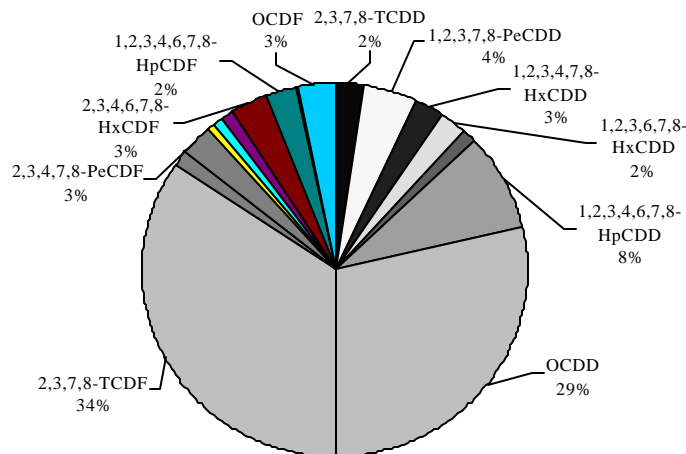


Figure 2-12. Percent contribution of each chlorinated dioxin and furan in largescale sucker. Basin-wide average of 23 composite whole body fish tissue samples. Only those congeners which exceed 1% of total chlorinated dioxin and furan concentrations are shown on the figure.

The maximum concentration of 2,3,7,8-TCDF was in the white sturgeon (Table 2-9). The fish species tended to cluster into three groups:

- 1) < 0.001 µg/kg = all the egg samples; walleye fillets, rainbow trout, spring chinook salmon fillets, steelhead, coho salmon, eulachon,
- 2) > 0.001 to < 0.010 µg/kg = largescale sucker, whole body walleye, bridgelip sucker, Pacific lamprey, fall chinook salmon, and whole body spring chinook salmon, and
- 3) > 0.010 µg/kg = white sturgeon and mountain whitefish.

**Table 2-9a. Basin-wide concentrations of 2,3,7,8-TCDF in composite samples of fish tissue from the Columbia River Basin, 1996-1998.**

	Fillet				Whole Body			
			µg/kg				µg/kg	
	N	F	range	Ave	N	F	range	Ave
<b>Resident species</b>								
white sturgeon*	16	16	0.0025 - 0.054	0.017	8	8	0.008 - 0.047	0.021
mountain whitefish	12	12	0.00014 - 0.014	0.0045	12	12	0.0002 - 0.012	0.0044
largescale sucker	19	18	<0.0001 - 0.0015	0.0004	23	23	0.0008 - 0.0036	0.0009
walleye	3	3	0.0006 - 0.0008	0.0007	3	3	0.0038 - 0.0055	0.0046
rainbow trout	7	7	0.0001 - 0.0003	0.0002	12	11	0.0004 - 0.0005	0.0002
bridgelip sucker	NS				3	3	0.0008 - 0.001	0.001
<b>Anadromous species</b>								
Pacific lamprey	3	3	0.0012 - 0.0017	0.0014	9	9	0.0011 - 0.0032	0.0020
fall chinook salmon	15	14	<0.0003 - 0.0014	0.0007	15	15	0.0004 - 0.0014	0.0008
spring chinook salmon	24	24	0.0004 - 0.0007	0.0006	24	24	0.0006 - 0.0011	0.0007
eulachon	NS				3	3	0.0006 - 0.0008	0.0007
steelhead	21	21	0.0002 - 0.0007	0.0004	21	21	0.0003 - 0.0006	0.0004
coho salmon	3	3	0.0004 - 0.0005	0.0005	3	3	0.0004 - 0.0005	0.0004

N = number of samples; F = detection frequency; NS = not sampled; < = detection limit

\*white sturgeon were individual fish and fillets without skin

**Table 2-9b. Basin-wide concentrations of 2,3,7,8-TCDF in composite samples of eggs from anadromous fish species in the Columbia River Basin, 1996-1998.**

	Egg			
			µg/kg	
	N	F	range	Ave
fall chinook salmon	1	1	0.00043	
spring chinook salmon	6	6	0.0004 - 0.0007	0.0005
steelhead	1	1	0.0002	
coho salmon	3	3	0.0003 - 0.0007	0.0005

N = number of samples; F = detection frequency

## 2.7 Toxicity Equivalence Concentrations of Chlorinated Dioxins and Furans, and Dioxin-Like PCB congeners

Chlorinated dioxins and furans are found in the environment together with other structurally-related chlorinated chemicals, such as some of the various dioxin-like PCB congeners. Therefore, people and other organisms are generally exposed to mixtures of these structurally similar compounds, rather than to a single chlorinated dioxin or furan, or dioxin-like PCB congener.

In order to estimate risks for exposure to dioxin-like chemicals (Table 1-4e,f,g) a method was developed to estimate a toxicity equivalence concentration (Van den Berg et al., 1998). In this methodology the toxicity equivalence factor for 2,3,7,8-TCDD is equal to 1; all other dioxin, furan, and dioxin-like PCB congeners are calculated as some relative percent of 1. The toxicity equivalence factors (Table 2-10) were derived by a panel of experts using careful scientific judgment after considering all available relative potency data (Van den Berg et al., 1998). Dioxin-like congener-specific toxicity equivalence factors (Table 2-10) are used to convert individual dioxin-like congener concentrations to 2,3,7,8-TCDD equivalents.

**Table 2-10. Toxicity Equivalence Factors (TEF) for dioxin-like PCB congeners, dioxins, and furans (from Van den Berg et al., 1998).**

PCBs	TEF	Dioxins	TEF	Furans	TEF
PCB 126	0.1	2,3,7,8-TCDD	1	2,3,4,7,8-PeCDF	0.5
PCB 169	0.01	1,2,3,7,8-PeCDD	1	2,3,7,8-TCDF	0.1
PCB 157	0.0005	1,2,3,4,7,8-HxCDD	0.1	1,2,3,4,7,8-HxCDF	0.1
PCB 156	0.0005	1,2,3,6,7,8-HxCDD	0.1	1,2,3,6,7,8-HxCDF	0.1
PCB 114	0.0005	1,2,3,7,8,9-HxCDD	0.1	1,2,3,7,8,9-HxCDF	0.1
PCB 77	0.0001	1,2,3,4,6,7,8-HpCDD	0.01	2,3,4,6,7,8-HxCDF	0.1
PCB 189	0.0001	OCDD	0.0001	1,2,3,7,8-PeCDF	0.05
PCB 123	0.0001			1,2,3,4,6,7,8-HpCDF	0.01
PCB 118	0.0001			1,2,3,4,6,7,8,9-HpCDD	0.01
PCB 105	0.0001			OCDF	0.0001
PCB 167	0.00001				

The toxicity equivalence concentration is the product of the toxicity equivalence factor multiplied by the concentration for an individual dioxin-like congener as shown in Equation 2-1:

$$\text{Equation 2-1)} \quad \text{TEC} = (\text{TEF}_i \times [\text{congener fish tissue concentration}]_i)$$

TEF = Toxicity equivalence factor

TEC = toxicity equivalence concentration

The toxicity equivalence concentrations for each dioxin, furan, and dioxin-like PCB congener are then summed to determine the total toxicity equivalence concentration.

The mountain whitefish fillet sample had the highest toxicity equivalence concentration (0.0063 µg/kg) followed by the white sturgeon (Table 2-11). The primary contributors to the mountain whitefish toxicity equivalence concentration were 2,3,7,8-TCDF and dioxin-like PCB congeners (118,126,156). The primary contributor to the high white sturgeon toxicity equivalence concentration was 2,3,7,8-TCDF and dioxin-like PCB congeners (105,118,156). The

Pacific lamprey had the highest concentration of toxicity equivalence concentrations of all the anadromous species. The concentrations 2,3,7,8 TCDF (Table 2-9), dioxinlike PCBs (Table 2-7) Aroclors (Table 2-6, and total pesticides (Figure 2-2) were also higher in Pacific lamprey than in any of the anadromous species.

**Table 2-11. Basin-wide average concentrations of the toxicity equivalence concentrations for composite fish samples from the Columbia River Basin, 1996-1998.**

Resident Species	Fillet		Whole body		Anadromous Species	Fillet		Whole body	
	N	µg/kg	N	µg/kg		N	µg/kg	N	µg/kg
white sturgeon*	16	0.0043	8	0.0051	Pacific lamprey	3	0.0027	9	0.0035
walleye	3	0.00049	3	0.0036	spring chinook salmon	24	0.0006	24	0.0009
mountain whitefish	12	0.0063	12	0.0033	steelhead	21	0.0.0009	21	0.0009
largescale sucker	19	0.0009	23	0.0016	eulachon	NS		3	0.0007
bridgelip sucker	NS		3	0.0013	coho salmon	3	0.0.0004	3	0.0006
rainbow trout	7	0.0008	12	0.0009	fall chinook salmon	15	0.0.0004	15	0.0005

N = number of samples; NS = not sampled.; \*white sturgeon were individual fish and fillets without skin

## 2.8 Metals

Of the sixteen metals analyzed, antimony and silver were not detected. Thallium was only detected once in a mountain whitefish. Unlike the organic chemicals the high metal concentrations did not appear to be associated with certain species or locations.

The percent contribution of each of the metals to the sum of metals was compared in fillet samples of largescale sucker (Figure 2-13a) and spring chinook salmon (Figure 2-13b). While there was considerable variability in the percent contribution in fish tissue, zinc and aluminum were found at the highest concentrations in all species (Figures 2-13a,b). Arsenic was generally higher in the anadromous fish species than in the resident fish species.

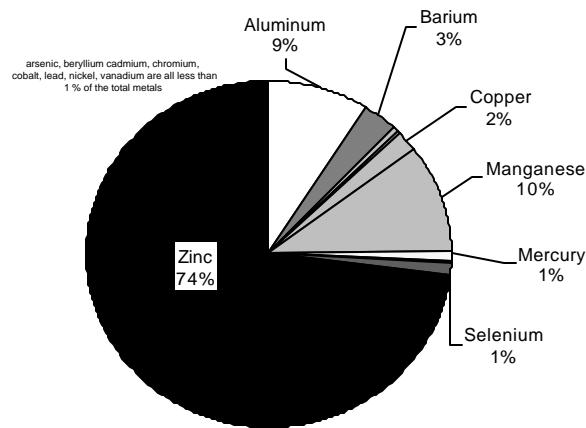


Figure 2-13a. Basin-wide average percent of individual metals in largescale sucker fillets. N= 23.

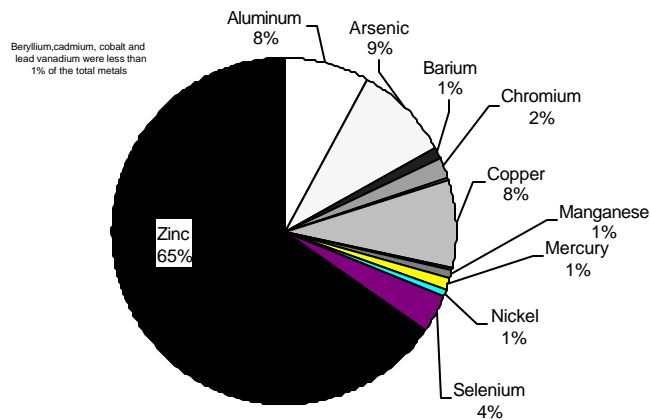


Figure 2-13b. Basin-wide percent of individual metals in spring chinook salmon fillets. N=24.

Basin-wide concentrations of metals were compared across species (Table 2-12, 2-13, 2-14). The maximum concentrations of individual metals (Table 2-12) were generally higher in the whole body fish samples with the exception of arsenic, copper, mercury, selenium, and zinc. Arsenic and mercury were higher in fillet samples while copper, selenium, and zinc were higher in the egg samples from the anadromous fish. The maximum concentrations of barium, cadmium, and manganese were in whole body largescale sucker samples from the Hanford Reach of the Columbia River (study site 9U). The maximum concentrations of chromium and cobalt were measured in the whole body white sturgeon from the main-stem Columbia River (study site 8).

**Table 2-12. Basin-wide maximum concentrations \* of metals in composite fish tissues measured in the Columbian River Basin, 1996 -1998.**

Chemical	Species	N	Tissue type	µg/kg	Study Site**
Aluminum	Largescale sucker	2	WB	190000	Columbia River (8)
Arsenic	Steelhead	3	FS	1500	Hood River (25)
Barium	Largescale sucker	3	WB	4700	Columbia River (9U)
Cadmium	Largescale sucker	3	WB	250	Columbia River (9U)
Chromium	White sturgeon	3	WB	1000	Columbia River (8)
Copper	Steelhead	1	Egg	18000	Snake River (96)
Copper	Fall chinook	3	WB	14000	Columbia River (14)
Cobalt	White sturgeon	3	WB	420	Columbia River (8)
Lead	Fall chinook	3	WB	1200	Columbia River (14)
Manganese	Largescale sucker	3	WB	21000	Columbia River (9U)
Mercury	Spring chinooksalmon	3	FS	510	Klickitat River (56)
Nickel	Steelhead	3	WB	17000	Klickitat River (56)
Selenium	Spring chinooksalmon	3	egg	5500	Umatilla River (30)
Selenium	White sturgeon	1	FW	2700	Columbia River (9U)
Vanadium	Rainbow trout	4	WB	770	Umatilla River (101)
Zinc	Steelhead	1	egg	76000	Snake River (96)
Zinc	Mountain whitefish	3	WB	40000	Deschutes (98)

\*All samples were composites except white sturgeon which were individual fish.; \*\*study site name with study site number in parentheses  
N = number of samples; FS = fillet with skin; FW = fillet without skin; WB = whole body.

Mercury was not detected in any anadromous egg sample (Table 2-13). The concentrations of copper, manganese, selenium and zinc were higher in the egg samples than any of the anadromous fish tissue samples (Table 2-12;Table 2-14).

**Table 2-13. Basin-wide average concentrations of metals in samples of eggs from anadromous fish collected in the Columbia River Basin, 1996-1998. Barium and beryllium were not detected in any egg samples.**

Chemical	fall chinook salmon	spring chinook salmon	coho salmon	steelhead
Number of samples	1	6	3	1
Concentration (µg/kg)				
Aluminum	500	950	850	4500
Arsenic	240	460	330	25
Cadmium	<4	35	<4	34
Chromium	<100	100	<100	220
Cobalt	35	43	12	170
Copper	5800	6200	4500	18000
Lead	<10	14	<10	41
Manganese	960	1500	700	2200
Mercury	<50	<79	<100	<43
Nickel	54	78	84	520
Selenium	2400	4200	1200	4500
Vanadium	19	13	28	110
Zinc	36000	43000	31000	76000

< = detection limit

Largescale sucker had the highest basin-wide average concentrations (Table 2-14) of aluminum (69,000 µg/kg), barium (2,300 µg/kg), manganese (14,000 µg/kg), mercury (240 µg/kg), and vanadium (310 µg/kg). White sturgeon had the highest basin-wide average concentrations of beryllium (8 µg/kg), chromium (360 µg/kg), cobalt (260 µg/kg), and selenium (1,100 µg/kg).

The basin-wide average whole body concentrations of cadmium, chromium, cobalt, copper, lead, manganese, nickel, vanadium, and zinc were higher than the fillet concentrations (Table 2-14). This may be due to the concentrations of these chemicals in the internal organs, bones, and skin of the fish. Selenium was generally higher in the whole body fish tissue with the exception of the white sturgeon. The concentrations of barium and aluminum were higher in the whole body tissue of resident fish species. In the anadromous fish species the whole body aluminum and barium concentrations were equal to or less than the fillet.



**Table 2-14. Basin-wide average concentrations of metals in composite samples of fish from the Columbia River Basin, 1996-1998.**

Chemical	Tissue Type	fall	spring	coho	Pacific		largescale	*white	mountain	rainbow		bridgelip	
		chinook salmon	chinook salmon	salmon	steelhead	lamprey	eulachon	sucker	sturgeon	whitefish	walleve	trout	sucker
N-FS		15	24	3	21	3	NS	19	16	12	3	7	NS
N-WB		15	24	3	21	9	3	23	8	12	3	12	3
		µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg
Aluminum	FS	630	790	<1000	1200	500		2400	3800	2600	2500	1100	
Aluminum	WB	510	610	<1000	550	1200	8800	69000	48100	11100	2400	27000	37000
Arsenic	FS	810	850	540	560	310		70	300	100	360	<50	
Arsenic	WB	860	830	500	580	260	890	160	370	140	490	120	280
Barium	FS	130	100	160	220	100		800	250	280	240	390	
Barium	WB	110	110	140	220	100	180	2300	1900	700	670	1200	2000
Beryllium	FS	2	2	2	2	2		3	2	2	2	5	
Beryllium	WB	2	2	2	3	2	2	5	8	2	2	3	5
Cadmium	FS	<4	10	<4	6	24		5	2	7	<4	2	
Cadmium	WB	6	120	22	57	110	9	55	42	28	7	12	29
Chromium	FS	71	180	140	81	80		120	65	130	90	70	
Chromium	WB	100	210	130	140	100	<100	310	360	120	110	93	180
Cobalt	FS	47	21	120	57	33		65	27	51	8	28	
Cobalt	WB	140	110	120	150	96	7	170	260	110	56	88	96
Copper	FS	640	790	1700	720	1200		550	250	620	570	500	
Copper	WB	3400	1400	1300	3200	4500	940	1400	990	1200	2500	1800	1200
Lead	FS	7	14	81	8	<10		29	8	15	<10	<10	
Lead	WB	220	21	15	45	16	500	170	120	35	190	26	54
Manganese	FS	87	90	190	150	380		2700	260	840	370	450	
Manganese	WB	320	370	500	460	390	500	14000	2700	3400	950	3200	18000
Mercury	FS	84	100	120	120	<110		240	150	80	180	77	
Mercury	WB	77	64	100	100	120	<35	130	140	67	180	73	32
Nickel	FS	75	63	54	44	15		110	56	76	260	59	
Nickel	WB	130	270	1200	900	110	50	1100	410	280	260	330	400
Selenium	FS	330	350	290	330	430		260	1100	510	390	220	
Selenium	WB	470	530	360	650	580	290	310	650	960	470	360	280
Vanadium	FS	6	5	7	14	10		11	9	29	5	17	29
Vanadium	WB	24	17	38	66	40	17	310	220	160	14	190	190
Zinc	FS	6700	6300	7100	7900	20000		20000	3800	15000	8700	12000	
Zinc	WB	27000	25000	30000	22000	22000	14000	23000	8200	27500	14000	29000	20000

\* white sturgeon were single fish; fillets were without skin N= Number of samples; FS = fillet with skin; WB = whole body; < = detection limit

### 2.8.1 Arsenic

Arsenic and mercury are discussed in detail in this report because of their contribution to risk. They are often primary components of risk because of their toxicity as well as their ubiquitous distribution in the environment as natural minerals in soil and from mining activities, smelting (arsenic) and fossil fuel burning (mercury).

With the exception of Pacific lamprey, anadromous fish had higher arsenic concentrations than resident fish (Table 2-14). The whole body concentrations of arsenic were uniformly higher than the fillet concentrations in the resident fish species (Table 2-14). However, there was no consistent pattern in the whole body versus fillet arsenic concentrations in the anadromous fish species (Table 2-14). Pacific lamprey had the lowest arsenic concentrations of all the anadromous species, which was the inverse of the relationship for organic chemicals, where Pacific lamprey had the highest concentrations. The average concentrations (240 - 460  $\mu\text{g}/\text{kg}$ ) of arsenic in the egg samples (Table 2-14) was similar to the whole body and fillet fish tissue concentrations (70-860  $\mu\text{g}/\text{kg}$ ) except for the steelhead eggs (25  $\mu\text{g}/\text{kg}$ ) and rainbow trout fillets (<50) which had the lowest concentrations of all the samples.

Arsenic concentrations were compared across sites for white sturgeon (2-14a) largescale sucker (Figures 2-14b), mountain whitefish (2-14c), spring chinook (2-15a) and steelhead (2-15b)

White sturgeon arsenic concentrations were generally consistent within sites but with considerable variability across sites (Figure 2-14a). For instance, the concentration in whole body samples ranged from 240  $\mu\text{g}/\text{kg}$  in the white sturgeon from the Hanford Reach of the Columbia River (study site 9U) to 660  $\mu\text{g}/\text{kg}$  in the white sturgeon from the main-stem Columbia River (study site 8). The fillet samples ranged from 150  $\mu\text{g}/\text{kg}$  in the Snake River (study site 13) to 640  $\mu\text{g}/\text{kg}$  in the fillet sample from main-stem Columbia River (study site 7). The maximum concentration occurred in the whole body sample from the main-stem Columbia River (660  $\mu\text{g}/\text{kg}$ ; study site 8). The arsenic concentrations in the duplicate fillets were equal or similar to each other.

The highest arsenic concentrations of largescale sucker were measured in whole body and fillet samples from the main-stem Columbia River (200-320  $\mu\text{g}/\text{kg}$ ; study sites 9U, 8) and the whole body samples from the Snake River (study site 13; 200-270  $\mu\text{g}/\text{kg}$ ; Figure 2-14b). The lower concentrations ranged from 50-150  $\mu\text{g}/\text{kg}$  in whole body and fillet fish tissues from the Deschutes, Yakima, Umatilla Rivers and the fillet fish tissues from Snake River (Figure 2-14b).

Mountain whitefish arsenic concentrations ranged from 100 to 140  $\mu\text{g}/\text{kg}$  with the maximum at 180  $\mu\text{g}/\text{kg}$  in the whole body sample from the Umatilla River (Figure 2-14c). The lowest concentrations were measured in the Deschutes River fillet samples. There was some variability between fillet and whole body with the whole body samples being higher than the fillet samples from Umatilla River and Deschutes River. The arsenic concentrations in the duplicate fillets from the Deschutes River were similar to each other.

The concentrations of arsenic in spring chinook salmon showed no consistent trend within

stations or across stations (Figure 2-15a). The highest concentrations were in the whole body (1200 µg/kg) and fillet (1100 µg/kg) from the Little White Salmon River and the whole body (1100 µg/kg) and fillet (1200 µg/kg) from the Middle Fork of the Willamette River. The arsenic concentrations in the duplicate fillet samples from Looking Glass Creek (study site 94) were similar (777 µg/kg, 783 µg/kg) to each other.

The maximum concentration (1500 µg/kg) of arsenic in all the fish samples was in the fillet sample from the Hood River (Table 1-12 and Figure 2-15b). The maximum whole body concentration from the Hood River was 1200 µg/kg. However there was considerable variability in the replicates for this site with most whole body and fillet samples at about 430 µg/kg. The samples from the other sites were between 290 and 800 µg/kg (Figure 2-15b). The duplicate fillet samples from the Clearwater River were not the same (480 µg/kg, 582 µg/kg) with the higher concentration (582 µg/kg) falling outside the range of the other samples from this site but lower than the maximum observed in the Hood River.

**LEGEND**  
 FW = fillet without skin  
 FS = fillet with skin  
 WB = whole body  
 Study sites are listed by number and name and described in Table 1-1  
 Concentration points on the graphs include duplicate fillets and chemicals at their detection limits.

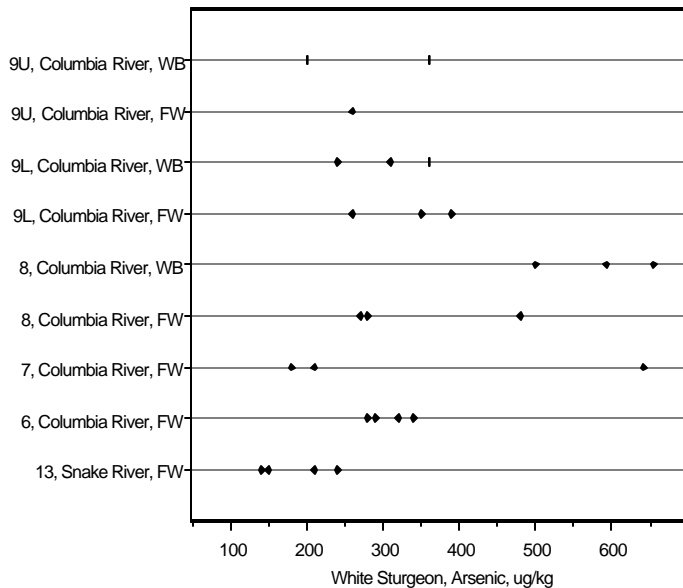


Figure 2-14a. Site specific concentrations of arsenic in white sturgeon individual fish tissue samples from the Columbia River Basin. Study sites 9U, 9L, 6, and 13 include duplicate fillet samples.

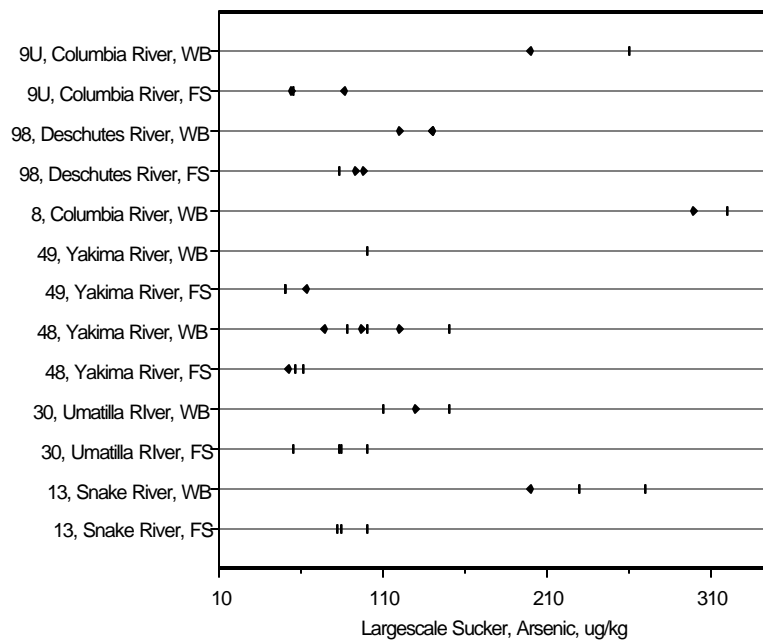


Figure 2-14b. Site specific concentration of arsenic in largescale sucker composite fish tissue samples from the Columbia River Basin.

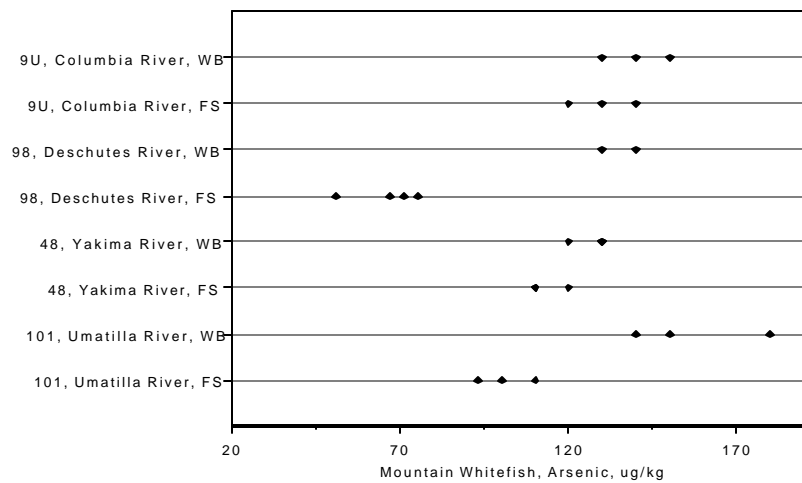


Figure 2-14c. Site specific concentration of arsenic in mountain whitefish composite fish tissue samples from the Columbia River Basin. Study site 98 includes duplicate fillet samples.

**LEGEND**

FS = fillet with skin  
 WB = whole body  
 Study sites are listed by number and name and described in Table 1-1.  
 Concentration points on graphs include duplicate fillets and chemicals at their detection limits.

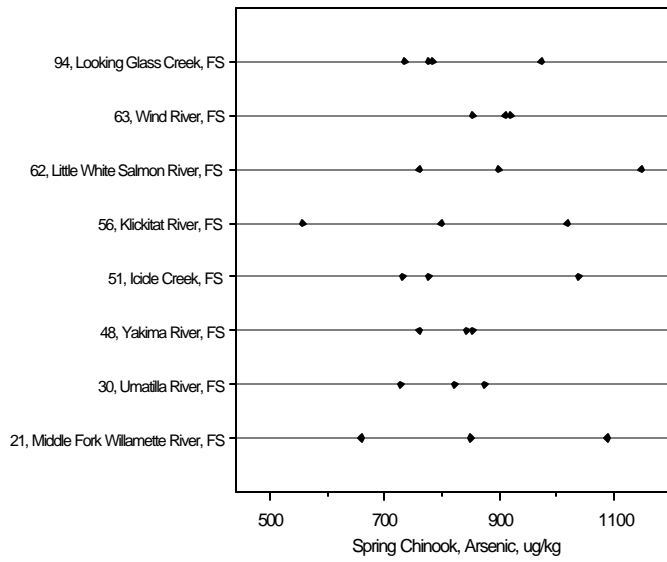


Figure 2-15a. Study site concentrations of arsenic in spring chinook composite samples from the Columbia River Basin. Study site 94 includes duplicate fillet samples.

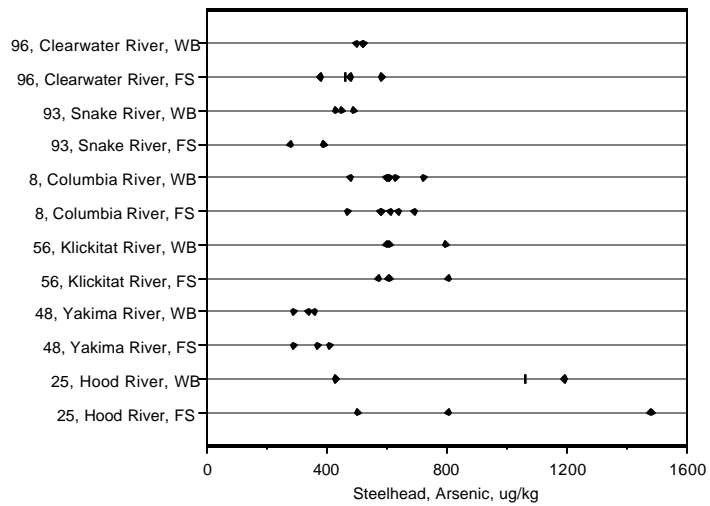


Figure 2-15b. Site specific concentrations of arsenic in steelhead composite fish tissue samples from the Columbia River Basin. Study site 96 includes duplicate fillet samples.

## 2.8.2 Mercury

The mercury levels in fish samples were extremely variable. The maximum concentration of mercury (510 µg/kg) was in the fillet sample of spring chinook salmon from the Klickitat River (Table 2-12).

There was no consistent pattern in mercury concentrations between whole body and fillet samples in the basin-wide average concentrations (Table 2-14). The average concentrations in fillet samples ranged from <91 µg/kg in the Pacific lamprey to 240 µg/kg in the largescale sucker. The whole body average concentrations ranged from <35 µg/kg in the eulachon to 180 µg/kg in the walleye.

Mercury concentrations were compared across study sites for white sturgeon, largescale sucker, mountain whitefish, spring chinook salmon, and steelhead (Figures 2-16a,b,c and 2-17a,b).

The maximum concentration (617 µg/kg) for white sturgeon was measured in the duplicate fillet from the Snake River (Figure 2-16a). The mercury concentrations in duplicate fillets from the Snake River were quite different from each other (617 µg/kg, 353 µg/kg) and the whole body samples (100 µg/kg) from this site. Since, the duplicate fillets from the same fish were averaged (430 µg/kg) in the data-set for this report, the maximum level of mercury for this study was reported as 510 µg/kg for spring chinook (Table 2-12). The concentrations in the duplicate fillets from study sites 9L, 6, and 13 were similar to each other.

The largescale sucker mercury concentrations were extremely variable across and within study sites. There was no distinct maximum although the fillet samples for the Umatilla and Snake Rivers were higher than the whole body samples from these study sites.

The mountain whitefish mercury concentrations were also variable. The maximum concentrations occurred in the Yakima, and Deschutes Rivers, although there was no difference in average concentrations. The duplicate fillets from the Deschutes River were equal to each other (71 µg/kg).

The concentrations of mercury in spring chinook salmon samples were at or near non-detectable levels, with the exception of the fillet samples from the Klickitat River, where the maximum concentration (510 µg/kg) was measured. This fillet sample also appeared to be an outlier for spring chinook salmon within this site and across all sites. The duplicate fillets from Looking Glass Creek were equal to each other (100 µg/kg).

The maximum concentration (420 µg/kg) was a single whole body sample from the Clearwater River. Except for the whole body sample from the Clearwater River, Steelhead mercury concentrations were all less than 180 µg/kg, with most samples in the 50-110 µg/kg range. The duplicate fillets from the Clearwater River were equal to each other.

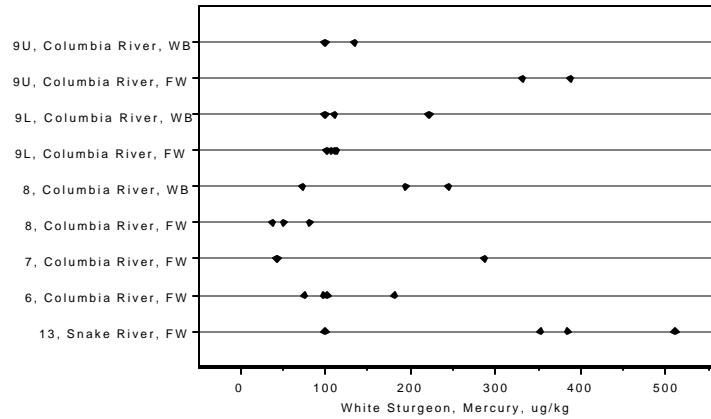


Figure 2-16a. Site specific concentrations of mercury in white sturgeon fish tissue samples from the Columbia River Basin. Study sites 9U, 9L, 13, and 6 include duplicate fillet samples.

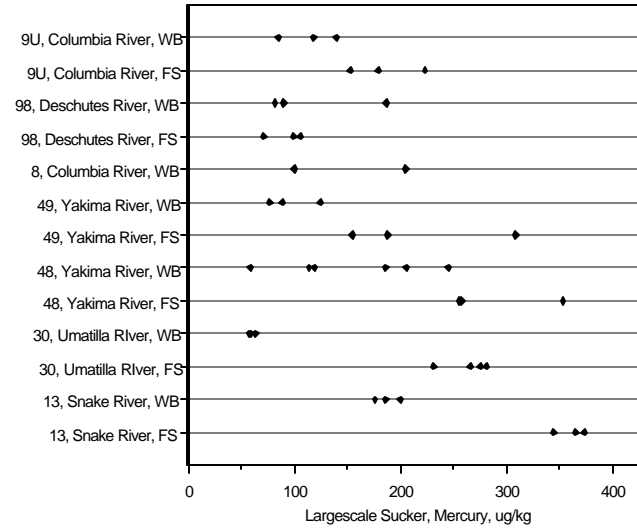


Figure 2-16b. Site specific concentrations of mercury in largescale sucker composite fish tissue samples from the Columbia River Basin.

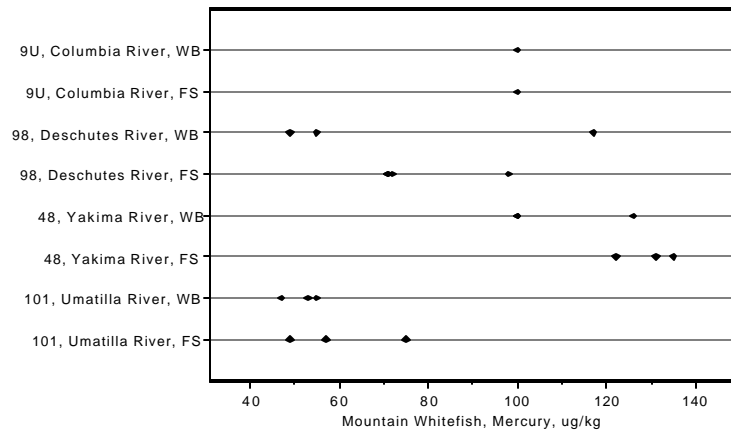


Figure 2-16c. Site specific concentrations of mercury in mountain whitefish composite fish tissue samples from the Columbia River Basin. Study site 98 includes duplicate fillet samples.

**LEGEND**

FW = fillet without skin  
 FS = fillet with skin  
 WB = whole body

Data points represent composite samples of fish tissue except white sturgeon which are individual fish. Study sites are listed by name and number and described in Table 1-1.

Concentration points on graphs include duplicate fillets and chemicals at their detection limits.

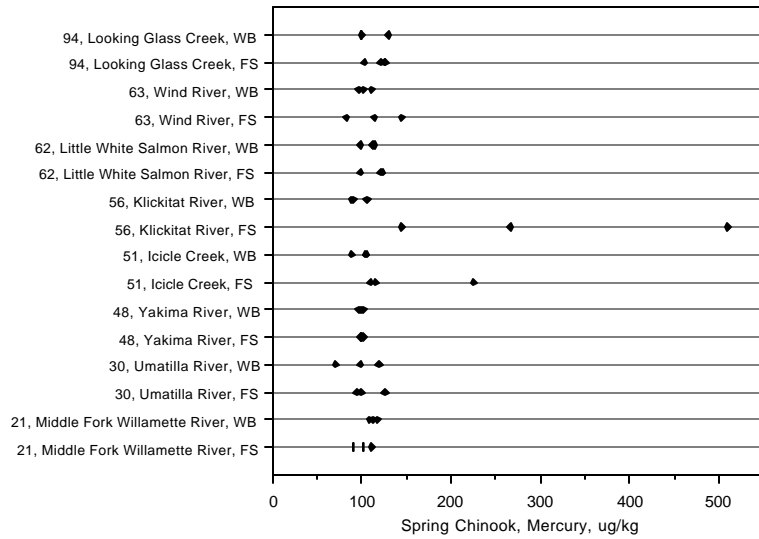


Figure 2-17a. Site specific concentrations of mercury in spring chinook salmon composite fish tissue samples from the Columbia River Basin. Study site 94 includes duplicate fillet samples.

**LEGEND**  
 FS = fillet with skin  
 WB = whole body  
 Study sites are listed by name and number and described in Table  
 .Concentration points on graphs include duplicate fillets and chemicals at their detection limits.

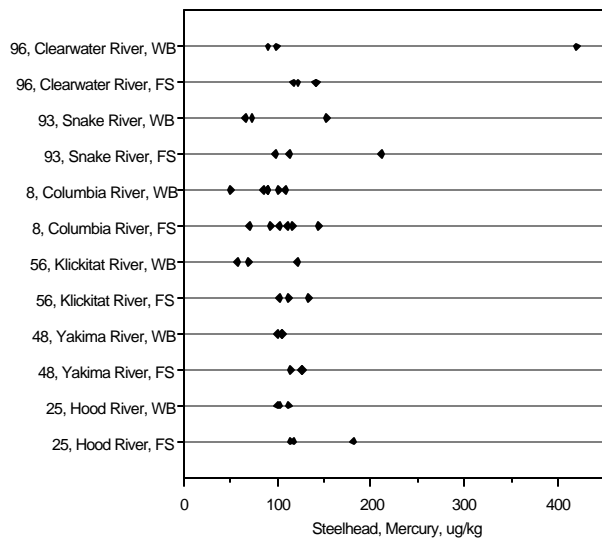


Figure 2-17b. Site specific concentrations of mercury in steelhead composite fish tissue samples from the Columbia River Basin. Study site 96 includes duplicate fillet samples.



### 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:

$$(Equation\ 4-1) \quad ADD = \frac{C \times CF \times IR \times EF \times ED}{BW \times AT}$$

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**

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

<sup>a</sup> Mean U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA, 2000b).

<sup>b</sup> 99th percentile U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA, 2000b).

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

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

<sup>e</sup> 90th percentile length of time an individual stays at one residence (USEPA, 1997b)

<sup>f</sup> Average life expectancy of the general public (USEPA, 1989).

<sup>g</sup> Average body weight for adults (male and female) in the general public (USEPA, 1989).

<sup>h</sup> 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.

<sup>i</sup> Average body weight for children of both sexes from 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.**

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

AFC - average fish consumption ; HFC - high fish consumption

<sup>a</sup> Mean U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA, 2000b).

<sup>b</sup> 99th percentile U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA, 2000b).

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

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

<sup>e</sup> 90th percentile length of time an individual stays at one residence (USEPA, 1997b)

<sup>f</sup> Average life expectancy of the general public (USEPA, 1989).

<sup>g</sup> 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 socioeconomic status. 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 99<sup>th</sup> 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 99<sup>th</sup> 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 99<sup>th</sup> 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 99<sup>th</sup> 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 99<sup>th</sup> 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.

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

<sup>a</sup> Mean U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA, 2000b).

<sup>b</sup> 99th percentile U.S. per capita consumption rate of uncooked freshwater and estuarine fish (USEPA, 2000b).

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

<sup>d</sup> 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 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)

$$\begin{aligned} \text{Percent of diet composed} &= \frac{\text{percentage of adults that consume salmon}}{\text{sum of the percentages for all species}} \times 100 \\ \text{of salmon} & \\ 27.7\% &= \frac{92.4}{333.5} \times 100 \end{aligned}$$

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)

$$\begin{aligned} \text{Consumption rate for} &= \text{Percent of hypothetical diet} \times \text{Average adult ingestion} \\ \text{salmon} & \quad \text{composed of salmon} \quad \text{rate for all species} \\ \text{(g/day)} & \\ 17.5 \text{ g/day} &= 27.7\% \times 63.2 \text{ g/day} \end{aligned}$$

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.**

<b>Species</b>	<b>A Percentage of Adults that Consume Species</b>	<b>B Percentage of Hypothetical Diet</b>	<b>C Consumption Rate<sup>c</sup> (grams/day)</b>
Salmon <sup>a</sup>	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 <sup>b</sup>	100.0	63.2

<sup>a</sup> This category includes spring chinook salmon, fall chinook salmon, steelhead and coho salmon.

<sup>b</sup> 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.

<sup>c</sup> 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 Z)**

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			Z														
	Arsenic				U	Z		Z									U		
	Barium				(U)	(U)													
	Beryllium									U									
	Cadmium					U		Z											
	Chromium (VI)									(U)									
	Cobalt		(U)																
	Copper																		(U)
	Manganese								U										
	Mercury								U	U									
	Nickel	U											Z						
	Selenium			Z				U	Z	Z							U		
	Silver						Z	Z			U								
	Thallium	Z				Z	Z	U	Z					Z					
	Vanadium																		(U)
Zinc	U																		
Semivolatiles	2-Chloronaphthalene						U						Z						
	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 <sup>a</sup>						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 Z)**

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 <sup>b</sup>			U			Z	Z	Z										

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.

Z - Other observed health endpoints

<sup>a</sup> Comprised of DDE, DDD, and DDT.

<sup>b</sup> 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.**

<b>Chemical</b>	<b>Oral RfD (mg/kg-day)</b>	<b>Confidence</b>	<b>UF/MF</b>	<b>Critical Effect</b>	<b>Source</b>
1,2,4-Trichlorobenzene	1.0 x 10 <sup>-2</sup>	Medium	1000/1	Increased adrenal weight	USEPA, 2000c
2,3,4,6- Tetrachlorophenol	3.0 x 10 <sup>-2</sup>	Medium	1000/1	Increased liver weights and centrilobular hypertrophy	USEPA, 2000c
2,4,5-Trichlorophenol	1.0 x 10 <sup>-1</sup>	Low	1000/1	Liver and kidney pathology	USEPA, 2000c
2-Chloronaphthalene	8.0 x 10 <sup>-2</sup>	Low	3000/1	Dyspnea, abnormal appearance, liver enlargement	USEPA, 2000c
2-Chlorophenol	5.0 x 10 <sup>-3</sup>	Low	1000/1	Reproductive effects	USEPA, 2000c
2,4-Dichlorophenol	3.0 x 10 <sup>-3</sup>	Low	100/1	Decreased delayed hypersensitivity response	USEPA, 2000c
2,4-Dimethylphenol	2.0 x 10 <sup>-2</sup>	Low	3000/1	Clinical signs (lethargy, prostration, and ataxia) and hematological changes	USEPA, 2000c
2,4-Dinitrotoluene	2.0 x 10 <sup>-3</sup>	High	100/1	Neurotoxicity, Heinz bodies and biliary tract hyperplasia	USEPA, 2000c
2,6-Dinitrotoluene	1.0 x 10 <sup>-3</sup>	-	3000	Mortality, neurotoxicity, Heinz bodies effects, methemoglobinemia, bile duct hyperplasia, and kidney histopathology	USEPA 1997e
Acenaphthene	6.0 x 10 <sup>-2</sup>	Low	3000/1	Hepatotoxicity	USEPA, 2000c
Aldrin	3.0 x 10 <sup>-5</sup>	Medium	1000/1	Liver toxicity	USEPA, 2000c
Aluminum	1.0	-	-	Minimal neurotoxicity	NCEA
Anthracene	3.0 x 10 <sup>-1</sup>	Low	3000/1	No treatment-related specific toxicological endpoints observed in mice at the doses administered in laboratory studies	USEPA, 2000c
Antimony	4.0 x 10 <sup>-4</sup>	Low	1000/1	Longevity, blood glucose, cholesterol	USEPA, 2000c
Total Aroclor <sup>a</sup>	2.0 x 10 <sup>-5</sup>	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 <sup>b</sup>	3.0 x 10 <sup>-4</sup>	Medium	3/1	Hyperpigmentation/keratosis and possible vascular complications	USEPA, 2000c
Barium	7.0 x 10 <sup>-2</sup>	Medium	3/1	Hypertension and kidney effects	USEPA, 2000c
Benzene, 1,2-dichloro-	9.0 x 10 <sup>-2</sup>	Low	1000/1	None identified	USEPA, 2000c
Benzene, 1,3-dichloro-	9.0 x 10 <sup>-4</sup>	-	-	No identified critical toxicological endpoint	NCEA
Benzene, 1,4-dichloro-	3.0 x 10 <sup>-2</sup>	-	-	Liver and reproductive effects	NCEA
Beryllium	2.0x10 <sup>-3</sup>	Low to Medium	300/1	Small intestinal lesions	USEPA, 2000c
bis(2-Chloroisopropyl)ether	4.0 x 10 <sup>-2</sup>	Low	1000/1	Decrease in hemoglobin and possible erythrocyte destruction	USEPA, 2000c
Cadmium	1.0 x 10 <sup>-3</sup>	High	10/1	Significant proteinuria	USEPA, 2000c
Chlordane (total) <sup>c</sup>	5.0 x 10 <sup>-4</sup>	Medium	300/1	Hepatic necrosis	USEPA, 2000c
Chromium (VI)	3.0 x 10 <sup>-3</sup>	Low	300/3	Gastrointestinal effects	USEPA, 2000c
Cobalt	6.0 x 10 <sup>-2</sup>	-	-	Polycythemia - too many red blood cells	NCEA
Copper	3.7 x 10 <sup>-2</sup>	-	-	Unspecified	USEPA 1997e
DDT <sup>d</sup>	5.0 x 10 <sup>-4</sup>	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.**

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

<sup>a</sup> 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.

<sup>b</sup> Total arsenic was measured. Inorganic arsenic was assumed to represent 10% of the total arsenic concentration (see Section 5.3.3).

<sup>c</sup> Chlordane (total) is the sum of cis-chlordane, cis-nonachlor, oxychlordane, trans-chlordane, and trans-nonachlor.

<sup>d</sup> Toxicity value for p,p'-DDT used.

<sup>e</sup> Reported as mercury in data set.

<sup>f</sup> Toxicity value based on thallium nitrate.

<sup>g</sup> Serum glutamic oxaloacetic transaminase.

<sup>h</sup> 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).**

<b>Weight-of-Evidence Classification</b>	<b>Category</b>
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 x 10 <sup>5</sup>	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 x 10 <sup>2</sup>	B2	Leukemia	USEPA, 2000c
Aldrin	1.7 x 10 <sup>1</sup>	B2	Liver carcinoma	USEPA, 2000c
alpha-HCH (alpha-BHC)	6.3	B2	Liver tumors	USEPA, 2000c
Adjusted Aroclors <sup>a</sup>	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 x 10 <sup>2</sup>	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 x 10 <sup>2</sup>	C	Liver and lung tumors	USEPA, 1997d
Chlordane (total) <sup>b</sup>	3.5 x 10 <sup>-1</sup>	B2	Non-Hodgkin's lymphoma and liver tumors	USEPA, 2000c
DDD (total) <sup>c</sup>	2.4 x 10 <sup>-1</sup>	B2	Lung, liver, and thyroid tumors	USEPA, 2000c
DDE (total) <sup>c</sup>	3.4 x 10 <sup>-1</sup>	B2	Liver and thyroid tumors	USEPA, 2000c
DDT (total) <sup>c</sup>	3.4 x 10 <sup>-1</sup>	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 x 10 <sup>2</sup>	C	Renal tubular adenomas and adenocarcinomas	USEPA, 2000c
Hexachloroethane	1.4 x 10 <sup>2</sup>	C	Hepatocellular carcinomas	USEPA, 2000c
Pentachlorophenol	1.2 x 10 <sup>-1</sup>	B2	Hepatocellular adenoma/carcinoma, pheochromocytoma/malignant pheochromocytoma, hemangiosarcoma/hemangioma	USEPA, 2000c
Toxaphene	1.1	B2	Hepatocellular carcinoma and neoplastic nodules	USEPA, 2000c

<sup>a</sup>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.

<sup>b</sup>Chlordane (total) is the sum of alpha-chlordane, cis-nonachlor, gamma-chlordane, oxychlordane, and trans-nonachlor.

<sup>c</sup>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).**

<b>Chemical</b>	<b>Relative Potency Factors</b>
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.

$$(Equation 5-1) \text{ adjusted Aroclors} = \text{Mass of Aroclors} - \text{Mass of PCB congeners}$$

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, dimethylarsenic (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		Inorganic As		Percent Inorganic As		Average Percent Inorganic As
		(ug/kg WW)	Q	(ug/kg WW)	Q	Q	Q	
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% <sup>a</sup>
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% <sup>b</sup>
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

<sup>a</sup>for whole body carp; <sup>b</sup>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)

In this risk assessment, hazard quotients were first calculated for individual chemicals in each species at each study site and for the basin. These results are found in Appendices G1 and G2. However, because the fish collected for this study contain more than one contaminant, estimating non-cancer hazard by considering only one chemical at a time might significantly underestimate the non-cancer effects associated with simultaneous exposures to several chemicals. Therefore, to assess the overall potential for non-cancer hazards posed by multiple chemicals, the procedures recommended by EPA for dealing with mixtures were applied (USEPA, 1986a; USEPA, 1989).

EPA recommends that a total hazard index value first be calculated by summing all hazard quotients for individual chemicals regardless of the type of health effect that each chemical causes. This approach to assessing mixtures - adding the hazard quotients - is known as dose addition. Dose addition assumes that all compounds in a mixture have similar uptake, pharmacokinetics (absorption, distribution, and elimination in the body), and toxicological processes; and that dose-response curves of the components have similar shapes. Thus, calculating a total hazard index (adding all of the hazard quotients for all of the chemicals in a fish sample regardless of their health endpoint) has several uncertainties since it results in combining chemicals with reference doses that are based upon very different critical effects, levels of confidence, and uncertainty/modifying factors. Because the assumption of dose additivity is most properly applied to compounds that induce the same effect by the same mechanism of action, summing the hazard quotients for all chemicals to calculate a total hazard index could overestimate the potential for effects, and is therefore, only the first step in assessing non-cancer effects from a mixture.

If the total hazard index calculated is greater than one, EPA recommends that the hazard quotient values for chemicals with similar target organs or mechanisms of action (health endpoints) be summed to calculate a hazard index specific for each health endpoint (USEPA, 1986a). If an endpoint specific hazard index is greater than 1, unacceptable exposures may be occurring, and there may be concern for potential non-cancer effects. Generally, the greater the magnitude of the hazard index greater than 1, the greater the level of concern for non-cancer health effects.

For this risk assessment, both the total hazard index and endpoint specific hazard indices were calculated for each study site and for the basin. As previously discussed in Section 5, a total of seventeen non-cancer health endpoints were considered in developing endpoint specific hazard indices. Hazard indices are presented by species in Appendices O (resident fish species) and P (anadromous fish species). The non-cancer hazard discussion in this section (Section 6) further summarizes the information in these appendices, focusing on the range in total and endpoint specific hazard indices among the species and on the chemicals which contribute the most to non-cancer hazards.

### **6.1.2 Cancer Risk Assessment**

The potential cancer risk from exposure to a carcinogen is estimated as the incremental increase in the probability of an individual developing cancer over a lifetime as a result of exposure to that carcinogen (USEPA, 1989). The term “incremental” means the risk due to environmental chemical exposure above the background cancer risk experienced by all individuals in a course of

a lifetime. Approximately one out of every two American men and one out of every three American women will have some type of cancer during their lifetime (American Cancer Society, 2002). The risk characterization in this report estimates the cancer risk that may result from only one source - exposure to contaminants as a result of eating fish from the Columbia River Basin. Other cancer risks (i.e., “background” cancer risks) are not evaluated.

Under current risk assessment guidelines, EPA assumes that a threshold dose does not exist for carcinogens and that any dose can contribute to cancer risks (USEPA, 1986b). In other words, the risk of cancer is proportional to exposure and there is never a zero probability of cancer risk when exposure to a carcinogenic chemical occurs. Cancer risk probabilities were estimated by multiplying the estimated exposure level (average daily dose in mg/kg-day, discussed in Section 4) by the cancer slope factor (SF) for each chemical. The cancer slope factors used in this risk characterization were developed by EPA and are discussed in Section 5 and shown in Table 5-5. Cancer slope factors are expressed in units that are the reciprocal of those for exposure (i.e., (mg/kg-day)<sup>-1</sup>). The cancer risk calculated for a chemical using this method represents the upper-bound incremental cancer risk that an individual has of developing cancer in their lifetime due to exposure to that chemical.

(Equation 6-2)                       $Risk = ADD \times SF$

Where:

- Risk = Estimated chemical-specific individual excess lifetime cancer risk (probability; unit-less)
- ADD = Chemical-specific average daily dose (mg/kg-day)
- SF = Chemical-specific oral cancer slope factor (kg-day/mg)<sup>-1</sup>

The excess cancer risk estimates in this report are shown in scientific notation format. These values should be interpreted as the upper-bound estimates of the increased risk of developing cancer over a lifetime. For example, 1 X 10<sup>-6</sup> or 1E-06 (E=exponent of base 10) is the estimated upper-bound lifetime cancer risk of 1 in 1 million. Because these are upper-bound estimates, the true risks could be lower.

Because the fish collected for this study contain more than one carcinogen, estimating cancer risks by considering only one carcinogen at a time might significantly under-estimate the cancer risk associated with simultaneous exposures to several chemicals. Therefore, to assess the overall potential for cancer risks from exposure to multiple chemicals, the procedure recommended by EPA for dealing with mixtures were applied (USEPA, 1986a; USEPA, 1989).

EPA recommends that to assess the risk posed by simultaneous exposure to multiple carcinogenic chemicals, the excess cancer risk for all carcinogenic chemicals be summed to calculate a total cancer risk. This summing approach for carcinogens, also called response addition, assumes independence of action by the carcinogens in a mixture. The assumption in applying this method is that there are no synergistic or antagonistic interactions among the carcinogens in fish and that all chemicals produce the same effect, which in this case is cancer.

In interpreting cancer risks, different federal and state agencies often have different levels of concern for cancer risks based upon their laws and regulations. EPA has not defined a level of concern for cancer. However, regulatory actions are often taken when the risk of cancer exceeds a probability of 1 in 1,000,000 to 10,000 (i.e.,  $1 \times 10^{-6}$  to  $1 \times 10^{-4}$ ). A level of concern for cancer risk has not been defined for this risk assessment.

For this risk assessment, the cancer risks for each chemical for a given species and study site were calculated (Appendix I). The cancer risks for each chemical were then summed to calculate the total cancer risks for each study site and for the basin. Appendices O (resident fish species) and P (anadromous fish species) show these total cancer risks by species as well as the contaminants with risks equal to or greater than  $1 \times 10^{-5}$  for CRITFC's member tribal adults (average fish consumption, 70 years exposure duration). The cancer risk discussion in this section (Section 6) further summarizes the information in the Appendices focusing on the range in total cancer risk among the species and on the chemicals which contribute the most to cancer risks.

### **6.1.3 Chemicals Not Evaluated**

As previously discussed in Section 1 of this report, a total of 132 chemicals were selected for analyses in all fish in this study. Forty (30%) of these chemicals, including 29 semivolatiles, 5 pesticides, 4 Aroclors, and 2 metals, were never detected in the tissue of any fish samples at the detection limits achieved for this study (Table 1-4a-g). Twenty-three chemicals that were analyzed for did not have reference doses or cancer slope factors (see Section 5.0) so that cancer risks and non-cancer hazards using the methods described in Section 6.1.2 and 6.1.3 could not be estimated. A risk characterization was done for only the detected chemicals with toxicity values; a total of 82 chemicals.

### **6.1.4 Arsenic**

As was previously discussed in Section 5.3.3, the non-cancer hazards and cancer risks discussed in Section 6.2.1 and 6.2.2, respectively, and the results presented in the appendices assume that for all fish species (resident fish and anadromous fish) caught in this study, 10% of the total arsenic is inorganic arsenic. Section 6.2.6 includes risk characterization results (using basin-wide data) assuming the alternative assumption that inorganic arsenic is only 1% of total arsenic for anadromous fish species.

### **6.1.5 Sample Type**

In the CRITFC fish consumption study (CRITFC, 1994), respondents were asked to identify the fish parts they consume for each species. For most of the fish species sampled as a part of this study, the majority of the respondents said that they consume fish fillet with skin. However, a smaller proportion consumed other fish parts as well (head, eggs, bones and organs).

Information on the portions of fish that are consumed by the general public is not available. However, as previously discussed in the Exposure Section, respondents to the qualitative fish consumption survey conducted by EVS (EVS, 1998) for the Wheatland Ferry-Willamette Falls

Reach of the Willamette River, which is a part of the Columbia River Basin, indicated that all ethnic groups consume fillet tissue; however, other parts of the fish (eyes, eggs and skin) are also consumed as are whole body fish.

For this study, whole body samples as well as fillets were collected when possible, since the fish consumption surveys show that fillets as well as other body parts may be eaten. Both whole fish and fillet with skin samples were analyzed for all species except white sturgeon, bridgelip sucker, and eulachon. Sturgeon were analyzed as whole fish and fillet without skin (since it is unlikely that sturgeon skin is eaten). For bridgelip sucker and eulachon only whole body samples were collected.

Some of the risk characterization results summarized in Sections 6.2.1 and 6.2.2 are presented for fillet and whole body samples, and others only for fillet with skin samples (except for those species for which fillet with skin data were not available). However, non-cancer hazards and cancer risks were calculated for all samples collected and are included in the Appendices of this report. In addition, the impacts of sample type on the risk characterization results are discussed in more detail in Section 6.2.4, where the risk characterization results for whole body and fillet fish samples are compared using site specific data.

## **6.2 Risk Characterization Results**

A summary and discussion of the non-cancer hazards (for adults and children for both the general public and CRITFC's member tribes) and excess cancer risks (for adults for the general public and CRITFC's member tribes) are presented in this section. More detailed information on the risk characterization results are presented in Appendices G through J and Appendices M through P for each fish species and tissue type analyzed in this study, for both individual study sites and for the Columbia River Basin:

- Appendix G1: Hazard quotients for individual chemicals for adults
- Appendix G2: Hazard quotients for individual chemicals for children
- Appendix H1: Percent contribution from individual chemicals to the total hazard index
- Appendix H2: Percent contribution from individual chemicals to endpoint-specific hazard indices
- Appendix I1: Estimated cancer risks for individual chemicals for adults, assuming 30 years exposure
- Appendix I2: Estimated cancer risks for individual chemicals for adults, assuming 70 years exposure
- Appendix J: Percent contribution of individual chemicals to total estimated cancer risk
- Appendix M: Comparison of the total and endpoint specific hazard indices across sites for a CRITFC tribal child (high fish consumption rate).
- Appendix N: Cancer risks across a range of consumption rates, by site and species
- Appendix O: Summary of risk characterization results (hazard indices and estimated cancer risks) for resident species
- Appendix P: Summary of risk characterization results (hazard indices and estimated cancer risks) for anadromous species

## **6.2.1 Non-Cancer Hazard Evaluation**

### **6.2.1.1 Non-Cancer Hazard Evaluation for Resident Fish**

Six species of resident fish were sampled in the Columbia River Basin: bridgelip sucker, largescale sucker, mountain whitefish, white sturgeon, walleye, and rainbow trout. Because of the large amounts of data that are presented in the appendices on the risk characterization for these species, one species (white sturgeon) was chosen as an example species to be discussed in detail. Data for the other resident fish species will be summarized. Tables 6-1 and 6-2 are identical to Tables 4.1 and 4.2, respectively, in Appendix O for sturgeon.

As previously discussed in Section 1, white sturgeon were collected from six study sites in the Columbia River Basin: 5 study sites in the main-stem Columbia River (study sites 6, 7, 8, 9L, and 9U) and in the Snake River (study site 13). Chemical analyses were performed on two tissue types, fillet without skin and whole body.

Table 6-1 summarizes both the total and end-point specific hazard indices calculated for white sturgeon. Results are presented for each of the six study sites that white sturgeon were caught as well as for the basin.

**Table 6-1. Total hazard indices (HI) and endpoint specific hazard indices (at or greater than 1.0) for white sturgeon.**

Consumption Rate/ Tissue Type			Hazard Index						Basin Average
			Study site <sup>e</sup>						
			CR-6	CR-7	CR-8	CR-9L	CR-9U	SR-13	
<b>General Public - Adult<sup>a,b</sup></b>									
AFC	FW	Immune system	–	–	–	–	2.1	–	0.6
		<b>Total HI</b>	0.8	0.6	0.6	1.2	2.9	0.9	0.9
AFC	WB	Immune system	na	na	1.1	–	–	na	0.9
		<b>Total HI</b>	na	na	1.5	1.0	1.2	na	1.3
HFC	FW	Liver	2.3	2.1	2.2	4.0	7.7	2.5	3.1
		Central nervous system	2.4	2.2	1.0	2.2	7.3	6.2	3.1
		Immune system	9.9	5.9	7.1	16	40	7.9	11
		Reproduction/development	2.4	2.2	1.0	2.2	7.3	6.2	3.1
		<b>Total HI</b>	15	11	11	23	55	17	18
HFC	WB	Liver	na	na	4.0	3.2	3.8	na	3.8
		Central nervous system	na	na	3.5	2.7	1.9	na	2.8
		Immune system	na	na	20	13	16	na	17
		Reproduction/development	na	na	3.5	2.6	1.9	na	2.7
		<b>Total HI</b>	na	na	29	20	23	na	24
<b>General Public - Child<sup>a,b</sup></b>									
AFC	FW	Immune system	–	–	–	–	1.8	–	0.5
		<b>Total HI</b>	0.7	0.5	0.5	1.1	2.6	0.8	0.8
AFC	WB	<b>Total HI</b>	na	na	1.3	0.9	1.1	na	1.1
HFC	FW	Liver	2.9	2.6	2.8	5.1	9.8	3.2	4.0
		Central nervous system	3.1	2.9	1.3	2.8	9.4	7.9	4.0
		Immune system	13	7.6	9.1	21	51	10	14
		Reproduction/development	3.1	2.9	1.3	2.8	9.4	7.9	4.0
		<b>Total HI</b>	19	14	14	29	70	22	23
HFC	WB	Liver	na	na	5.1	4.1	4.9	na	4.9
		Central nervous system	na	na	4.5	3.4	2.4	na	3.9
		Immune system	na	na	26	16	21	na	22
		Reproduction/development	na	na	4.4	3.3	2.4	na	3.8
		<b>Total HI</b>	na	na	37	25	29	na	31
<b>CRITFC's Member Tribes - Adult<sup>c,d</sup></b>									
AFC	FW	Liver	1.0	–	–	1.8	3.4	1.1	1.4
		Central nervous system	1.1	–	–	–	3.3	2.8	1.4
		Immune system	4.4	2.6	3.1	7.2	18	3.5	5.0
		Reproduction/development	1.1	–	–	–	3.3	2.8	1.4
		<b>Total HI</b>	6.6	4.7	4.7	10	24	7.5	7.9
AFC	WB	Liver	na	na	1.8	1.4	1.7	na	1.7
		Central nervous system	na	na	1.6	1.2	–	na	1.2
		Immune system	na	na	9.0	5.7	7.3	na	7.4
		Reproduction/development	na	na	1.5	1.2	–	na	1.2
		<b>Total HI</b>	na	na	13	8.8	10	na	11
HFC	FW	Liver	6.2	5.6	6.1	11	21	6.8	8.5
		Central nervous system	6.6	6.1	2.8	6.0	20	17	8.5
		Immune system	27	16	19	44	108	22	31
		Reproduction/development	6.6	6.1	2.8	6.0	20	17	8.5
		Selenosis	–	1.3	1.5	2.0	–	–	1.2
		<b>Total HI</b>	40	29	29	62	150	46	49
HFC	WB	Liver	na	na	11	8.8	10	na	10

**Table 6-1. Total hazard indices (HI) and endpoint specific hazard indices (at or greater than 1.0) for white sturgeon.**

Consumption Rate/ Tissue Type			Hazard Index					Basin Average	
			Study site <sup>e</sup>						
			CR-6	CR-7	CR-8	CR-9L	CR-9U		SR-13
		Central nervous system	na	na	9.6	7.2	5.1	na	7.6
		Immune system	na	na	56	35	45	na	45
		Reproduction/development	na	na	9.5	7.1	5.1	na	7.5
		<b>Total HI</b>	na	na	79	54	62	na	66
<b>CRITFC's Member Tribes - Child<sup>a,d</sup></b>									
AFC	FW	Liver	1.8	1.7	1.8	3.2	6.2	2.0	2.5
		Central nervous system	2.0	1.8	–	1.8	6.0	5.1	2.5
		Immune system	8.0	4.8	5.8	13	32	6.4	9.2
		Reproduction/development	2.0	1.8	–	1.8	6.0	5.1	2.5
		<b>Total HI</b>	12	8.6	8.6	18	45	14	14
AFC	WB	Liver	na	na	3.2	2.6	3.1	na	3.1
		Central nervous system	na	na	2.9	2.2	1.5	na	2.5
		Immune system	na	na	17	10	13	na	14
		Reproduction/development	na	na	2.8	2.1	1.5	na	2.4
		<b>Total HI</b>	na	na	24	16	18	na	20
HFC	FW	Liver	12	11	12	21	41	13	16
		Cardiovascular	1.1	1.2	1.2	1.2	–	–	1.1
		Central nervous system	13	12	5.5	12	39	33	16
		Immune system	52	32	38	86	210	42	60
		Reproduction/development	13	12	5.5	12	39	33	16
		Hyperpigmentation/keratosis	1.1	1.2	1.2	1.2	–	–	1.1
		Selenosis	–	2.6	2.9	3.8	1.4	1.5	2.3
		<b>Total HI</b>	79	56	56	120	290	89	94
HFC	WB	Liver	na	na	21	17	20	na	20
		Cardiovascular	na	na	1.8	1.1	1.0	na	1.4
		Central nervous system	na	na	19	14	10	na	16
		Immune system	na	na	110	69	87	na	91
		Reproduction/development	na	na	18	14	9.9	na	16
		Hyperpigmentation/keratosis	na	na	1.8	1.1	1.0	na	1.4
		Selenosis	na	na	1.1	1.7	1.4	na	1.3
		Gastrointestinal	na	na	1.1	1.8	–	na	1.1
		<b>Total HI</b>	na	na	150	110	120	na	130

AFC = average fish consumption na =not applicable; sample type not analyzed at this study site

HFC = high fish consumption – = health endpoint <1.0 at that study site

Total HI = the sum of hazard quotients regardless of health endpoint FW - fillet without skin; WB - whole body

<sup>a</sup> AFC risk based on average U.S. per capita consumption rate of uncooked freshwater and estuarine fish for general public (adult) of 7.5 g/day, or 1 8-oz meal per month, and for general public (child) of 2.83 g/day, or 0.4 8-oz meal per month (USEPA, 2000b).

<sup>b</sup> HFC risk based on 99th percentile U.S. per capita consumption rate of uncooked freshwater and estuarine fish for general public of 142.4 g/day, or 19 8-oz meals per month, and for general public (child) of 77.95 g/day, or 11 8-oz meals per month (USEPA, 2000b).

<sup>c</sup> AFC risk based on average consumption rate for adult fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin of 63.2 g/day, or 9 8-oz meals per month, and for child fish consumers of 24.8 g/day, or 3 8-oz meals per month (CRITFC 1994).

<sup>d</sup> HFC risk based on 99th percentile consumption rate for adult fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin of 389 g/day, or 53 8-oz meals per month, and for child fish consumers of 162 g/day, or 22 8-oz meals per month (CRITFC 1994).

<sup>e</sup> Study sites are described in Table 1-1. CR = Columbia River ; SR = Snake River



For white sturgeon, the endpoints which had hazard indices greater than 1 for most of the populations were the immune system, liver, central nervous system, and reproduction/developmental, with the immune system endpoint having a higher hazard index than the other endpoints (Table 6-1). At the lowest (average) fish ingestion rates for the general public (average fish consumption, adults and children), only the immune endpoint exceeds a hazard index of 1 (high of 2.1). At the higher fish ingestion rates (e.g., the high ingestion rates for CRITFC's member tribal child), other endpoints with hazard indices greater than 1 begin to appear: liver, central nervous system, reproductive/developmental, cardiovascular, hyperpigmentation/keratosis, selenosis, and gastrointestinal.

Table 6-1 also shows that, as expected, the magnitude of both the end-point specific and total hazard indices increases proportionally to the estimated exposure for that population. For adults, the only differences in exposure for the four adult populations (general public, average and high fish consumption; CRITFC's member tribes, average and high fish consumption) are due to the different fish ingestion rates used. Thus, the hazard index increases proportionally to the fish ingestion rate. All other exposure parameters either remain constant for all four adult populations (fish contaminant levels, exposure frequency, body weight) or do not impact the exposure (exposure duration and averaging time) for the reasons discussed in Section 4.9 (Averaging Time). This direct relationship between the hazard index and the fish ingestion rates for adults is shown in Figure 6-1 and Table 6-2.

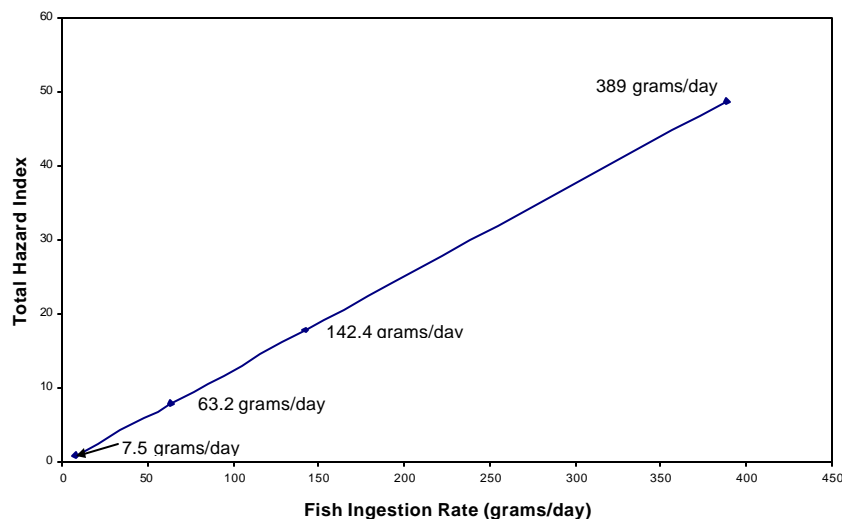


Figure 6-1. Total hazard index versus fish consumption rate for adults. White sturgeon, Columbia River Basin-wide average concentrations (fillet without skin).

**Table 6-2. Comparison of Estimated Total Hazard Indices Among Adult Populations.  
White sturgeon (whole body) from Columbia River, study site 8**

<b>Population</b>	<b>Ingestion rate (g/day)</b>	<b>Total hazard index</b>	<b>Approximate ratio of hazard index to that of general public adult with average fish consumption</b>
<b>General public</b>			
average fish consumption	7.5	1.5	1
high fish consumption	142.4	29	19
<b>CRITFC's member tribal</b>			
average fish consumption	63.2	13	9
high fish consumption	389	79	50

Table 6-2 shows the total hazard indices estimated for adults consuming sturgeon at Columbia River study site 8 (whole body samples) at each ingestion rate. Also shown is the ratio of the total hazard indices for CRITFC's member tribes (average and high fish consumption) and the general public (high fish consumption) to that for the general public, average fish consumption. The ingestion rate and exposure for adults is lowest at the average fish consumption rate for the general public and increases proportionally for the other populations as their ingestion rates increase. For example, the ingestion rate for the high fish consumers, general public, is about 19 times higher than that for the average fish consumer. Thus, the exposure estimated and the total hazard indices calculated for the general public, high fish consumer would be expected to be 19 times higher than those calculated for the general public, average fish consumer. This relationship also holds true for the endpoint specific hazard indices calculated for each study site and the basin. The hazard index for the immune system (Table 6-1) was about 1 at Columbia River study site 8 for the general public, average fish consumption (whole body fish) and 20 for the high fish consumption, general public - approximately a 20 fold difference (not exactly 19 fold as shown in the Table 6-2 due to rounding of hazard indices).

A similar comparison can be made for the populations of children assessed in this risk assessment. However, as discussed in Section 4.3, for children, exposures vary by ingestion rate as well as by body weight and exposure duration. This is because of the difference in the ages of the children in the two different fish consumption studies used to estimate fish ingestion rates for children (general public children versus CRITFC's member tribal children). Table 6-3 shows the ratio of hazard indices for three of the child populations (general public, high fish consumption; CRITFC's member tribes, average and high fish consumption) compared to that of the general public child with average fish consumption using data for the Columbia River (study site 8), whole body sturgeon. As can be seen from this table, the hazard indices estimated for CRITFC's member tribal children at the high ingestion rate were over 100 times those estimated for general public children at the average ingestion rate.

**Table 6-3. Comparison of Estimated Total Hazard Indices Among Child Populations  
White sturgeon (whole body) from Columbia River, study site 8**

<b>Population</b>	<b>Ingestion rate (g/day)</b>	<b>Total hazard index</b>	<b>Ratio of hazard index to that of general public with average fish consumption</b>
<b>General public</b>			
average fish consumption	2.83	1.3	1
high fish consumption	77.95	37	28
<b>CRITFC's member tribal</b>			
average fish consumption	24.8	24	18
high fish consumption	162	150	115

A review of Table 6-1 also shows that for the general public at the average ingestion rate, the hazard indices for children were about 0.9 of those for adults; the hazard indices for general public children at the high ingestion rate were about 1.3 times those for general public adults, high ingestion rate. For example, the basin-wide total hazard index was 23 at the high fish consumption rate (77.95 grams/day) assumed for the general public child compared to 18 for the high fish consumption rate (142.2 grams/day) assumed for the general public adult. For CRITFC's member tribes, the hazard indices for children at the average and high fish ingestion rates were both about 2 times those for CRITFC's member tribal adults at the average and high ingestion rates, respectively.

The differences in hazard indices between adults and children as well as the differences among sites and at different fish ingestion rates is shown in Figures 6-2a-d. These figures show a comparison of the total hazard indices for sturgeon (fillet without skin) across sites for both adults and children at different fish ingestion rates (note that the scale of the Y axis increases from Figure 6-2a through Figure 6-2d). Figure 6-2a compares the total hazard indices for general public adults and children at the average fish ingestion rate. The hazard index varies by site with the Hanford Reach of the Columbia River (study site 9U) having the highest values (hazard indices of 2.9 for adults and 2.6 for children). At a given site, the total hazard index for a child is about 0.9 that of that for an adult at the average fish ingestion rate for the general public. Figure 6-2d compares the results for CRITFC tribal adults and children at the high ingestion rate. Again, the total hazard index varies across sites with the Hanford Reach of the Columbia River (study site 9U) having the highest values (hazard indices of 150 for adults and 290 for children). At a given site, the total hazard index for a child is about 2 times that for those of adults at the high fish ingestion rate for CRITFC tribal adults and children.

The chemicals which had hazard quotients at or greater than 1.0 (i.e., exposures for that chemical were greater than the reference dose) for sturgeon for most populations were total Aroclors, total DDT, and mercury (Table 6-4, same as Table O-4.2 in Appendix O). Selenium, arsenic, and chromium were generally greater than 1.0 only at the highest exposures (high fish consumption rates for CRITFC's member tribal adults and children). It is useful to compare the chemicals contributing the most to non-cancer hazard for sturgeon (Table 6-4) with the hazard indices for each endpoint (Table 6-1). Aroclors, which had the highest hazard quotients (Table 6-4) were also the only chemicals contributing to the endpoint of immunotoxicity. Thus the endpoint specific hazard indices for immunotoxicity were also the highest of all hazard indices (Table 6-1).

Mercury was the major contributor to the endpoints of central nervous system and reproduction/developmental, and DDT to the liver endpoint. Thus the hazard quotients calculated for Aroclors, mercury, and DDT (Table 6-4) were the major contributors to (and often equal or close to) the hazard indices for the endpoints of immunotoxicity, central nervous system and reproduction/development, and liver, respectively (Table 6-1). The hazard indices greater than 1.0 for the cardiovascular and hyperpigmentation endpoints (Table 6-1) were primarily a result of exposures greater than the reference dose for arsenic. Selenosis was a result of exposures greater than the reference dose for selenium, and gastrointestinal effects were a result of exposures greater than the reference dose for chromium.

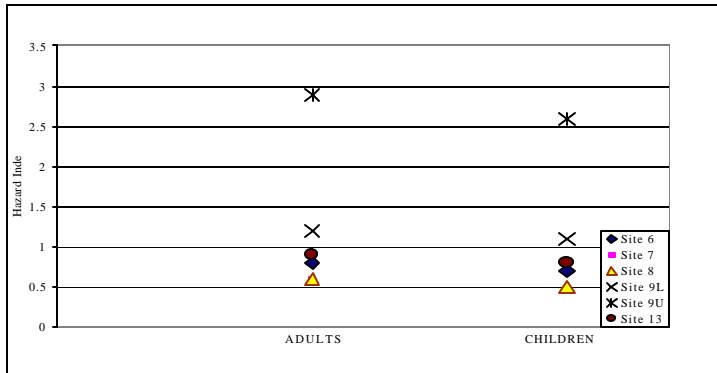


Figure 6-2a. Hazard indices for general public adults and children, average fish consumption rate of white sturgeon fillets. Note that hazard indices are the same at study site 7 and 13.

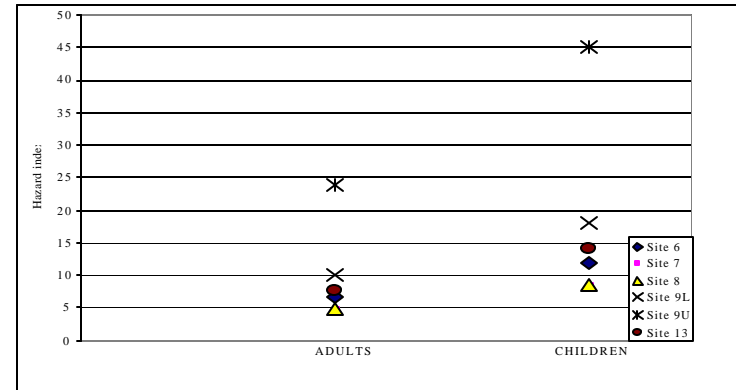


Figure 6-2b. Hazard indices for CRITFC's member tribal adults and children, average fish consumption rate for white sturgeon fillets. Note that hazard indices are the same at study sites 7 and 13.

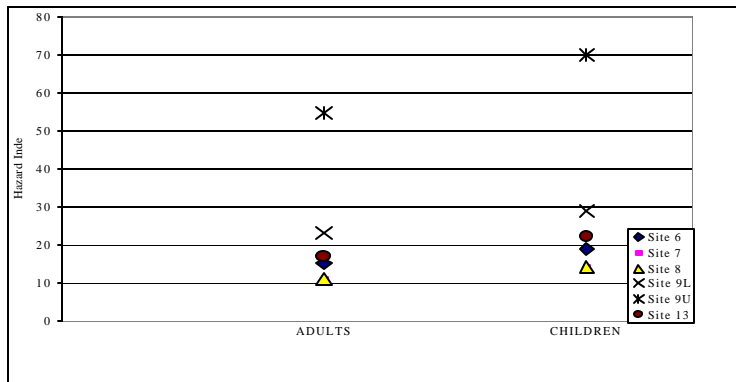


Figure 6-2c. Hazard indices for general public adults and children, high fish consumption rate of white sturgeon fillets. Note that hazard indices are the same for study sites 7 and 13.

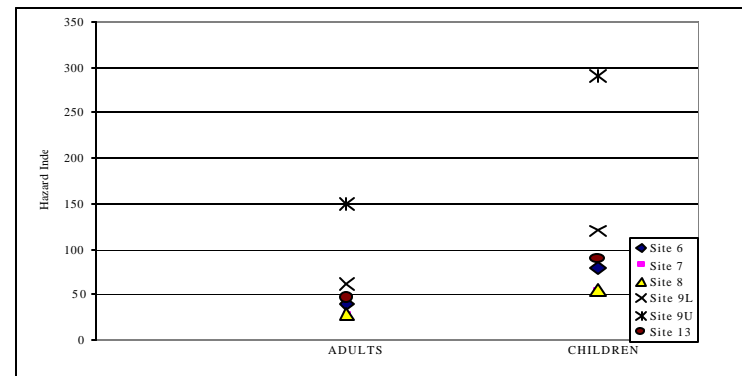


Figure 6-2d. Hazard indices for CRITFC's member tribal adults and children, high fish consumption rate of white sturgeon fillets. Note that hazard indices are the same at study sites 7 and 13.

It is important to point out that there are no reference doses available for dioxins, furans and dioxin-like PCB congeners. Therefore, hazard quotients could not be calculated for these classes of chemicals and their potential impact on the magnitude of non-cancer hazards (i.e., endpoint specific hazard indices and total hazard indices) could not be evaluated.

**Table 6-4. Chemicals having hazard quotients at or greater than 1.0 in white sturgeon.**

Tissue Type	Adults			Chemical	Children		
	Hazard Quotient		Study sites <sup>a</sup> with Values >1		Hazard Quotient		Study Sites <sup>a</sup> with Values >1
	AFC	HFC			AFC	HFC	
<b>General Public</b>							
<b>Fillet without skin</b>							
Total Aroclors	2.1	5.9-40	6 <sup>b</sup> ,7 <sup>b</sup> ,8 <sup>b</sup> ,9L <sup>b</sup> ,9U,13 <sup>b</sup>	Total Aroclors	1.8	7.6-51	6 <sup>b</sup> ,7 <sup>b</sup> ,8 <sup>b</sup> ,9L <sup>b</sup> ,9U,13 <sup>b</sup>
Total DDT	-	1.5-7.1	6,7,8,9L,9U,13	Total DDT	-	1.9-9.1	6,7,8,9L,9U,13
Mercury	-	1.0-7.3	6,7,8,9L,9U,13	Mercury	-	1.3-9.4	6,7,8,9L,9U,13
<b>Whole body</b>							
Total Aroclors	1.1	13-20	8,9L <sup>b</sup> ,9U <sup>b</sup>	Total Aroclors	-	17-26	8,9L,9U
Total DDT	-	2.6-3.7	8,9L,9U	Total DDT	-	3.4-4.7	8,9L,9U
Mercury	-	1.9-3.5	8,9L,9U	Mercury	-	2.4-4.4	8,9L,9U
<b>CRITFC's Tribal Members</b>							
<b>Fillet without skin</b>							
Total Aroclors	2.6-18	16-110	6 <sup>b</sup> ,7 <sup>b</sup> ,8 <sup>b</sup> ,9L,9U,13 <sup>b</sup>	Total Aroclors	4.8-32	32-210	6,7,8,9L,9U,13
Total DDT	1.3-3.2	4.1-20	6,7,8,9L,9U	Total DDT	1.2-5.8	8.0-38	6,7,8,9L,9U,13
Mercury	1.0-3.3	2.8-20	6,7,8 <sup>b</sup> ,9L <sup>b</sup> ,9U,13	Arsenic	-	1.1-1.2	6,7,8,9L
Selenium	-	1.3-2.0	7,8,9L	Mercury	1.8-6.0	5.5-39	6,7,8 <sup>b</sup> ,9L,9U,13
				Selenium	-	1.4-3.8	7,8,9L,9U,13
<b>Whole body</b>							
Total Aroclors	5.7-9.0	35-56	8,9L,9U	Total Aroclors	11-17	69-110	8,9L,9U
Total DDT	1.2-1.6	7.8-10	8,9L,9U	Total DDT	2.1-3.0	14-20	8,9L,9U
Mercury	1.2-1.5	5.1-9.5	8,9L,9U <sup>b</sup>	Arsenic	-	1.0-1.8	8,9L,9U
				Chromium	-	1.1-1.8	8,9L
				Mercury	1.5-2.8	9.9-19	8,9L,9U
				Selenium	-	1.1-1.7	8,9L,9U

AFC = average fish consumption; HFC = high fish consumption; - = <1; <sup>a</sup>study sites are described in Table 1-1. <sup>b</sup>HFC only

The summary of the results of the non-cancer hazard evaluation for the other resident fish species are shown in Appendix O by species. Summaries of the endpoint specific and total hazard indices and of the chemicals having hazard quotients at or greater than 1 are shown in Tables 1.1 and 1.2 (bridgeline sucker), 2.1 and 2.2 (largescale sucker), 3.1 and 3.2 (mountain whitefish), 4.1 and 4.2 (white sturgeon), 5.1 and 5.2 (walleye), and 6.1 and 6.2 (rainbow trout). A review of these tables shows that:

- The total hazard indices and endpoint specific hazard indices increase among the general public and CRITFC's member tribal populations as the exposures for that population increase;

- The endpoints which are more frequently greater than a hazard index of 1 are immune system (due to Aroclors), liver (due primarily to DDE for most species), and central nervous system and reproduction/developmental (due primarily to methyl mercury), with the immune system endpoint usually having a higher hazard index than the other endpoints. These hazard indices vary among sites for a given species and among species;
- At the lowest (average) fish ingestion rates for the general public (adults and children), the endpoint-specific hazard indices were at or less than 1 for all of the resident fish with the exception of sturgeon and whitefish at the Hanford Reach of the Columbia River (9U) where hazard indices for immunotoxicity were greater than 1 (high of 3 for whitefish).
- For the more highly exposed populations (e.g., at the high fish ingestion rates for CRITFC's member tribes), endpoint specific hazard indices for reproduction/development and central nervous system, immunotoxicity, and liver are greater than 1 at most sites for most species. For mountain whitefish and white sturgeon, hazard indices for the most contaminated study site (Columbia River, study site 9U) were greater than 100 for the immunotoxicity endpoint.
- At these highest ingestion rates for CRITFC's member tribal adults and children, other endpoints with hazard indices greater than 1 begin to appear for some species. These endpoints include cardiovascular and hyperpigmentation/keratosis, selenosis, gastrointestinal, kidney, and metabolism. These effects were primarily the result of exposures greater than the reference dose for arsenic; selenium; chromium; cadmium; and nickel and zinc, respectively. For walleye, thallium also contributes to the overall hazard index calculated for liver. The highest endpoint-specific hazard index for these endpoints was approximately 4.0.

Table 6-5 is a summary of the ranges in endpoint specific hazard indices across study sites for each resident fish species. Results are shown for both average and high fish consumption rates for the general public and CRITFC tribal member adults. Hazard indices are shown only for those endpoints that most frequently exceed a hazard index of 1 (reproduction/development and the central nervous system, immunotoxicity, and liver). It should be kept in mind that not all fish species were caught at the same sites and that sample numbers varied by species.

**Table 6-5 Summary of ranges in endpoint specific hazard indices across study sites for adults who consume resident fish from the Columbia River Basin.**

Non-cancer endpoints which most frequently exceed a hazard index of 1 for all species				
Species	N	Reproductive/ Developmental And Central Nervous System	Immunotoxicity	Liver
<b>General Public - Adult</b>				
<b>Average Fish Consumption</b>				
bridgelip sucker	3	<1	<1	<1
largescale sucker	19	<1	<1	<1
mountain whitefish	12	<1	<1 to 3	<1
white sturgeon	16	<1	<1 to 2	<1
walleye	3	<1	<1	<1
rainbow trout	7	<1	<1	<1
<b>High Fish Consumption</b>				
bridgelip sucker	3	<1	6	2
largescale sucker	19	2 to 7	1 to 8	<1 to 3
mountain whitefish	12	<1 to 3	1 to 50	<1 to 4
white sturgeon	16	1 to 7	6 to 40	2 to 8
walleye	3	4	1	1
rainbow trout	7	1 to 2	1 to 2	<1
<b>CRITFC's Member Tribal Adult</b>				
<b>Average Fish Consumption</b>				
bridgelip sucker	3	<1	3	1
largescale sucker	19	<1 to 3	<1 to 3	<1 to 1
mountain whitefish	12	<1 to 1	<1 to 22	<1 to 2
white sturgeon	16	<1 to 3	3 to 18	<1 to 3
walleye	3	2	<1	<1
rainbow trout	7	<1	<1	<1
<b>High Fish Consumption</b>				
bridgelip sucker	3	2	17	6
largescale sucker	19	5 to 20	<1 to 21	<1 to 7
mountain whitefish	12	<1 to 7	4 to 140	<1 to 11
white sturgeon	16	3 to 20	16 to 108	6 to 21
walleye	3	10	4	4
rainbow trout	7	4 to 5	3 to 4	<1

N = number of samples; all samples are fillet with skin except white sturgeon which is fillet without skin.  
Bridgelip sucker and eulachon are whole body samples.

Figure 6-3 summarizes the total basin-wide hazard indices for resident fish species using average and high fish consumption rates for the general public and CRITFC's member tribal adult populations. This figure shows that mountain whitefish and white sturgeon had the highest total basin-wide hazard indices, followed by sucker, walleye, and rainbow trout. It also shows that for all species, the total hazard indices are highest for CRITFC's member tribal adults at the high fish ingestion rates (389 g/day) followed by the general public adult, high ingestion rate (142.4 g/day); CRITFC's member tribal adults, average ingestion rate (63.2 g/day); and general public adult,



average ingestion rate (7.5 g/day).

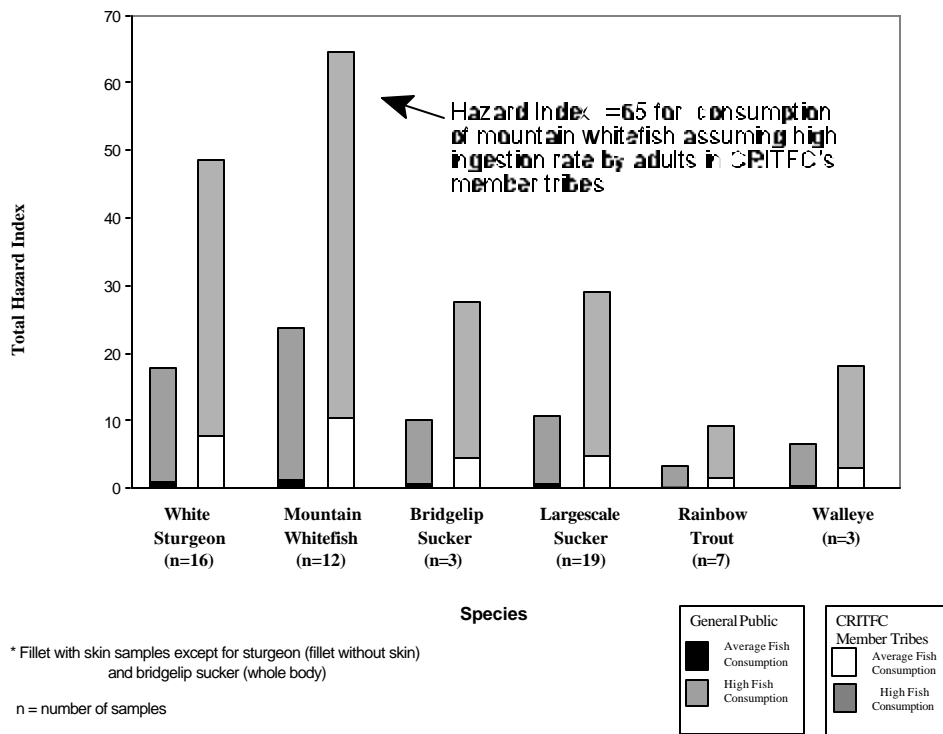


Figure 6-3. Adult total non-cancer hazard indices for resident fish species\* using basin-wide average data.

For a more detailed comparison of the total and endpoint specific hazard indices, see Appendix M, where hazard indices are compared for all resident species across study sites for CRITFC’s member tribal children with a high fish consumption rate (162 g/day or 5 meals per week).

The contribution from specific chemicals and classes of chemicals to the overall non-cancer hazard for resident fish species is shown in Table 6-6. These results were calculated using Columbia River Basin average concentrations for fillet without skin samples, except for those species where such sample types were not available (bridgelip sucker, whole body; white sturgeon, fillet without skin). The number of samples used to compute the basin-wide averages vary among species, and for some species represent only a few samples (e.g., 3 samples for walleye and bridgelip sucker). The results in Table 6-6, which are also depicted in the charts in Figures 6-4 through 6-9, show that the percent contribution of specific chemicals to the total hazard index differs among the resident fish species. For example, Aroclors contribute 83% to the total non-cancer hazard for mountain whitefish, but only 20% for walleye. Total DDT contribution to the total hazard index ranges from 3-21% among the species and methyl mercury from about 6-54%. Except for thallium for walleye (percent contribution of 14%), the only chemicals contributing greater than 5% to the non-cancer hazards for resident fish species are Aroclors, total DDT, and mercury.

**Table 6-6. Percent contribution of contaminant groups to total non-cancer hazards for resident fish species. Based on Columbia River Basin-wide averages.**

	<b>white sturgeon</b>	<b>bridgelip sucker</b>	<b>largescale sucker</b>	<b>mountain whitefish</b>	<b>walleye</b>	<b>rainbow trout</b>
<i>Tissue Type</i>	<i>FW</i>	<i>WB</i>	<i>FS</i>	<i>FS</i>	<i>FS</i>	<i>FS</i>
<i>Number of samples</i>	<i>16</i>	<i>3</i>	<i>19</i>	<i>12</i>	<i>3</i>	<i>7</i>
<b>Total metals</b>	<b>22</b>	<b>18</b>	<b>50</b>	<b>9</b>	<b>77</b>	<b>55</b>
Mercury	17	6	45	7	54	46
Arsenic	1	2	<1	<1	4	ND
Chromium	<1	1	1	<1	1	1
Manganese	<1	3	<1	<1	<1	<1
Selenium	2	1	1	1	2	3
Thallium	ND	ND	ND	ND	14	ND
Zinc	<1	1	1	<1	1	2
Other Metals	<1	4	1	<1	1	2
<b>Total Aroclors</b>	<b>63</b>	<b>60</b>	<b>40</b>	<b>83</b>	<b>20</b>	<b>42</b>
<b>Total Pesticides</b>	<b>15</b>	<b>21</b>	<b>10</b>	<b>8</b>	<b>3</b>	<b>3</b>
Total DDT	13	21	9	7	3	3
Other Pesticides	2	<1	<1	1	ND	ND

FW = fillet without skin; FS = fillet with skin; WB = whole body; ND = Not Detected

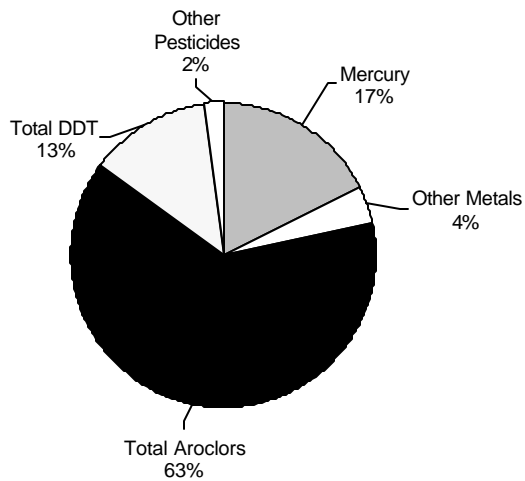


Figure 6-4. Percent contribution of basin-wide average chemical concentrations to non-cancer hazards from consumption of white sturgeon fillet without skin. Number of samples = 16.

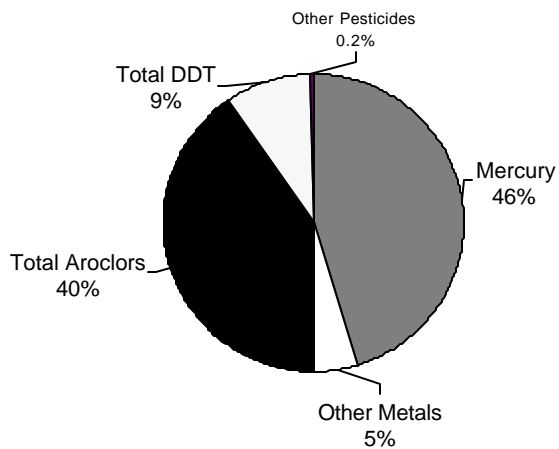


Figure 6-5. Percent contribution of basin-wide average chemical concentrations of non-cancer hazards from consumption of largescale sucker fillets with skin. Number of samples = 19.

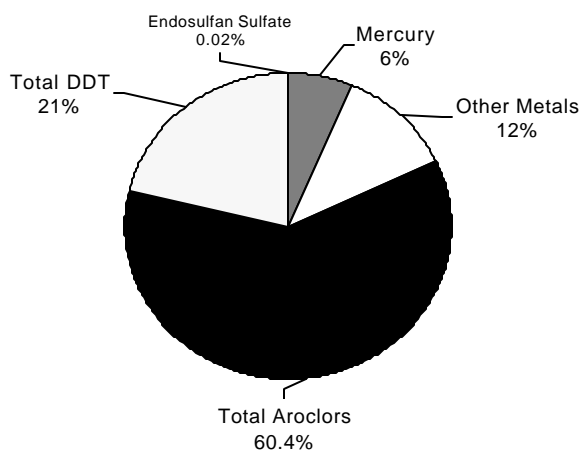


Figure 6-6. Percent contribution of basin-wide average chemical concentrations to non-cancer hazards from consumption of whole body bridgelip sucker. Number of samples = 3.

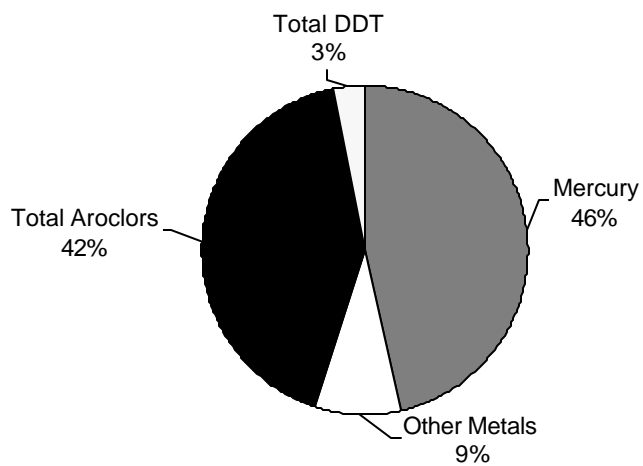


Figure 6-7. Percent contribution of basin-wide average chemical concentrations to non-cancer hazards from consumption of rainbow trout fillet with skin. Number of samples = 7.

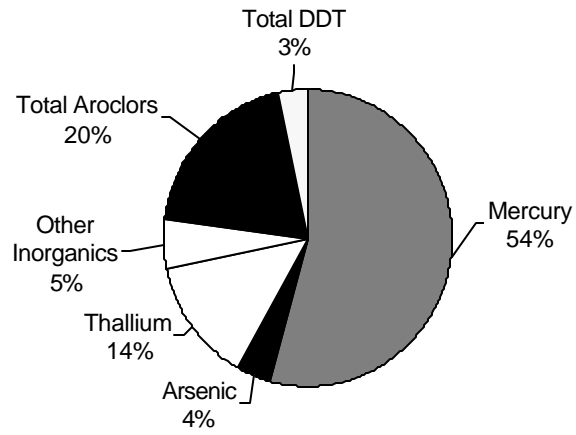


Figure 6-8. Percent contribution of basin-wide average chemical concentrations to non-cancer hazards from consumption of walleye fillet with skin. Number of samples = 3.

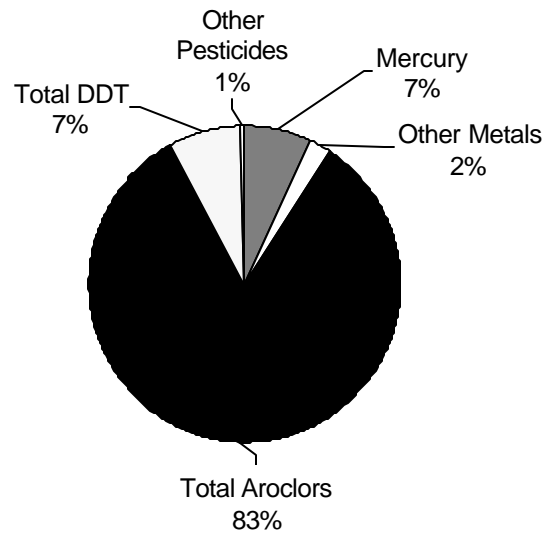


Figure 6-9. Percent contribution of basin-wide chemical concentrations to non-cancer hazards from consumption of mountain whitefish fillet with skin. Number of samples = 12.

### 6.2.1.2 Non-cancer Hazard Evaluation for Anadromous Fish

The anadromous fish sampled in the Columbia River Basin were coho salmon, fall chinook salmon, spring chinook salmon, steelhead, eulachon, and Pacific lamprey. The summary of the results of the non-cancer hazard evaluation for these anadromous fish species are shown in Appendix P by species. Summaries of the endpoint-specific and total hazard indices and of the chemicals having hazard quotients greater than 1 are shown in Tables 1.1 and 1.2 (coho salmon), 2.1 and 2.2 (fall chinook salmon), 3.1 and 3.2 (spring chinook salmon), 4.1 and 4.2 (steelhead), 5.1 and 5.2 (eulachon), and 6.1 and 6.2 (Pacific lamprey). As with the resident fish species, the values of the total hazard indices and endpoint-specific hazard indices increase among all of the populations as the exposure to that population increases.

Because the results for coho salmon, fall chinook, spring chinook, and steelhead were similar, they are summarized as a group. The results for eulachon and lamprey are discussed separately.

Tables 1.1 and 1.2 (coho salmon), 2.1 and 2.2 (fall chinook salmon), 3.1 and 3.2 (spring chinook salmon), and 4.1 and 4.2 (steelhead) show that:

- At the average fish ingestion rates for the general public, adults and children, the endpoint specific hazard indices were less than 1.0.
- The endpoints which had hazard indices greater than 1 most frequently for salmon and steelhead were immunotoxicity (due to Aroclors) and reproductive/developmental and central nervous system (due primarily to mercury). In general, the hazard indices for the immunotoxicity endpoint for salmon and steelhead were much lower and did not vary as much across study sites as those for the resident fish species with the highest contaminant levels (largescale sucker, mountain whitefish, and white sturgeon).
- As exposures increase, other endpoints with hazard indices greater than 1 begin to appear. These include: cardiovascular and hyperpigmentation/keratosis; metabolism; selenosis; gastrointestinal; and kidney, resulting primarily from exposures greater than the reference dose to arsenic; nickel and zinc; selenium; chromium; and cadmium, respectively. The highest hazard indices for these endpoints at the highest ingestion rates were at or less than 4. At these exposures, hazard indices for immunotoxicity, reproduction/development, and central nervous system are greater than 1 for most sites.

Pacific lamprey were collected at 2 study sites, Willamette Falls (study site 21) and Fifteen Mile Creek (study site 24). Pacific lamprey results were similar to those for salmon and steelhead in that, at the average fish ingestion rates for the general public, adults and children, the endpoint specific hazard indices never exceed 1.0. In examining endpoint specific hazard indices with increasing exposure, the immune system hazard index is exceeded first. The estimated endpoint specific hazard index for immunotoxicity, which is the largest contributor to the total hazard index for Pacific lamprey is due to exposures greater than the reference dose for Aroclors. At the same ingestion rates, the endpoint specific hazard indices for immunotoxicity were higher for lamprey than for salmon and steelhead.

Eulachon (smelt) were caught at only one study site, Columbia River study site 3, and analyzed as whole body samples. Two endpoint specific hazard indices were exceeded (cardiovascular and hyperpigmentation/keratosis) at the high fish consumption rates for CRITFC's member tribal adults (hazard index of 1.7) and children (hazard index of 3.2) (see Table 5.1). These exceedances were a result of arsenic exposures greater than the reference dose (Table 5.2).

Table 6-7 is a summary of the ranges in endpoint specific hazard indices across study sites for anadromous fish. Results are shown for both average and high fish consumption rates for the general public and CRITFC tribal member adults. Hazard indices are shown only for the three endpoints which frequently exceeded a hazard index of 1: reproduction/development and the central nervous system, immunotoxicity, and liver. It should be kept in mind that not all species were caught at the same study sites and that sample numbers varied by species.

Figure 6-10 shows the relative differences in total hazard indices in the Columbia River Basin for anadromous fish species using average and high fish consumption rates for general public adults and for CRITFC's member tribal adults. The total hazard index is highest for lamprey, followed by salmon and steelhead, which are in the same range, and then eulachon.

For a more detailed comparison of the total and endpoint specific hazard indices across study sites for anadromous fish species, see Appendix M. In this appendix, hazard indices are compared for the population with the highest exposure and non-cancer hazards - CRITFC's member tribal children with a high fish consumption rate (162 grams/day or about 5 meals per week).

**Table 6-7 Summary of ranges in endpoint specific hazard indices across study sites for adults who consume anadromous fish species from the Columbia River Basin.**

Non-cancer endpoints which most frequently exceed a hazard index of 1 for all species				
Species	N	Reproductive/ Developmental And Central Nervous System	Immunotoxicity	Liver
<b>General Public-</b>				
<b>Average Fish Consumption</b>				
coho salmon	3	<1	<1	<1
fall chinook salmon	15	<1	<1	<1
spring chinook salmon	24	<1	<1	<1
steelhead	21	<1	<1	<1
eulachon	3	<1	<1	<1
Pacific lamprey	3	<1	<1	<1
<b>High Fish Consumption</b>				
coho salmon	3	2	3	<1
fall chinook salmon	15	1 to 2	<1 to 3	<1
spring chinook salmon	24	<1 to 6	1 to 2	<1
steelhead	21	1 to 3	1 to 2	<1
eulachon	3	<1	<1	<1
Pacific lamprey	3	<1	9	<1
<b>CRITFC's Member Tribal</b>				
<b>Average Fish Consumption</b>				
coho salmon	3	1	1	<1
fall chinook salmon	15	<1 to 1	1	<1
spring chinook salmon	24	<1 to 3	<1	<1
steelhead	21	<1 to 1	<1 to 1	<1
eulachon	3	<1	<1	<1
Pacific lamprey	3	<1	4	<1
<b>High Fish Consumption</b>				
coho salmon	3	7	7	<1
fall chinook salmon	15	3 to 6	<1 to 8	<1
spring chinook salmon	24	<1 to 17	3 to 6	<1
steelhead	21	4 to 8	3 to 6	<1
eulachon	3	<1	<1	<1
Pacific lamprey	3	<1	24	2

N= number of samples; All samples are fillet with skin except white sturgeon which is fillet without skin. Bridgelip sucker and eulachon are whole body fish samples.



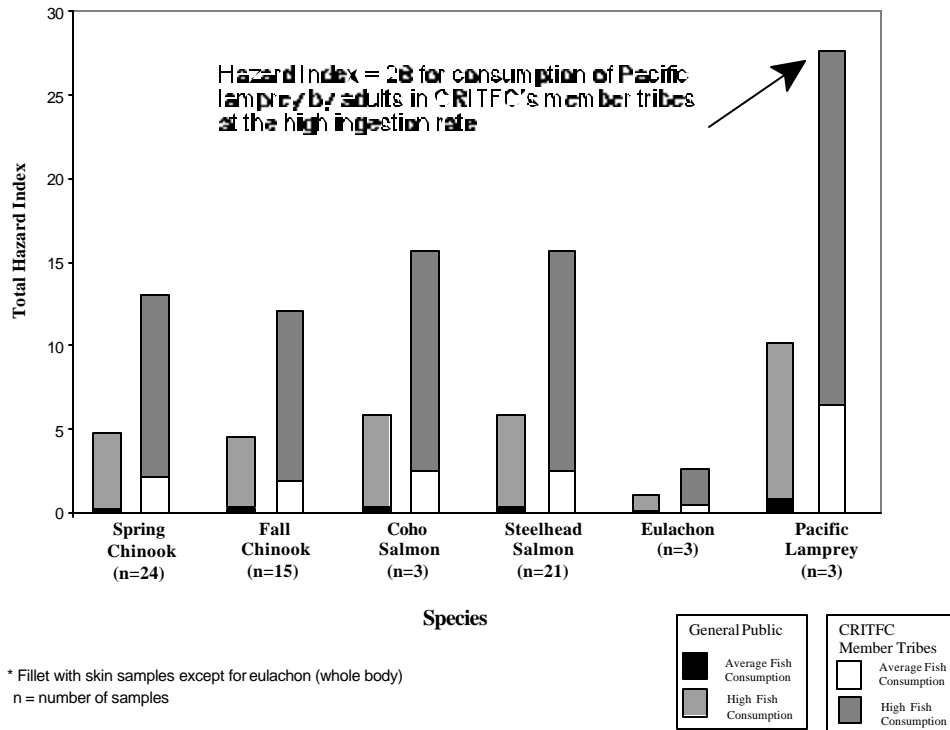


Figure 6.10 Adult total non-cancer indices for anadromous fish species\*. Average concentrations for the Columbia River Basin.

Table 6-8 and Figures 6-11 through 6-16 show the major chemicals contributing to the total hazard index for each anadromous fish species (shown for basin-wide data, fillet with skin for all species except eulachon which was whole body). Aroclors and mercury were the primary chemicals of concern for non-cancer hazards for anadromous fish species, followed by arsenic. For eulachon, arsenic was the major contributor to non-cancer hazard. For Pacific lamprey, Aroclors contributed almost 87% to the non-cancer health effects.

**Table 6-8. Percent contribution of contaminant groups to total non-cancer hazards for anadromous fish species. Based on Columbia River Basin-wide averages.**

	spring chinook	coho salmon	eulachon	fall chinook	Pacific lamprey	steelhead
<i>Number of samples</i>	24	3	3	15	3	21
<i>Tissue type</i>	FS	FS	WB	FS	FS	FS
<b>Total Metals</b>	<b>65</b>	<b>54</b>	<b>95</b>	<b>58</b>	<b>7</b>	<b>55</b>
Mercury	43	41	ND	39	ND	43
Aluminum	<1	ND	2	<1	ND	<1
Arsenic	12	6	62	12	2	7
Cadmium	<1	ND	2	ND	1	<1
Chromium	3	2	ND	1	1	1
Copper	1	2	5	1	1	1
Selenium	3	2	12	3	2	2
Zinc	1	1	9	1	1	1
Other Metals	2	<1	2	<1	<1	<1
<b>Total Aroclors</b>	<b>34</b>	<b>45</b>	<b>ND</b>	<b>40</b>	<b>87</b>	<b>43</b>
<b>Total Pesticides</b>	<b>2</b>	<b>1</b>	<b>4</b>	<b>2</b>	<b>6</b>	<b>2</b>
Chlordane (total)	<1	<1	ND	<1	2	<1
Total DDT	2	1	4	2	4	1
Hexachlorobenzene	<1	ND	ND	<1	<1	<1

FS = fillet with skin; FW = fillet without skin; WB = whole body; ND= not detected

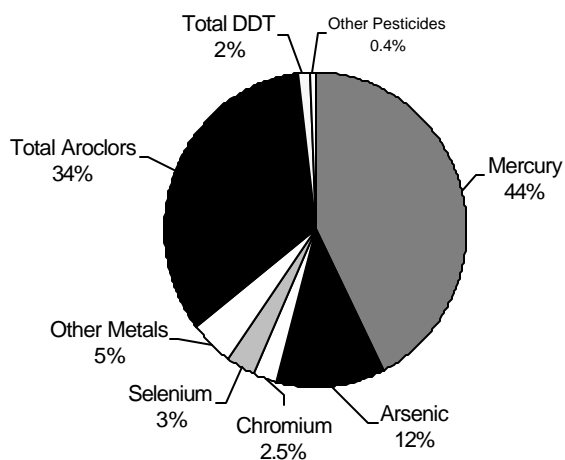


Figure 6-11. Percent contribution of basin-wide average chemical concentrations to non-cancer hazards from consumption of spring chinook fillet with skin. Number of samples = 24.

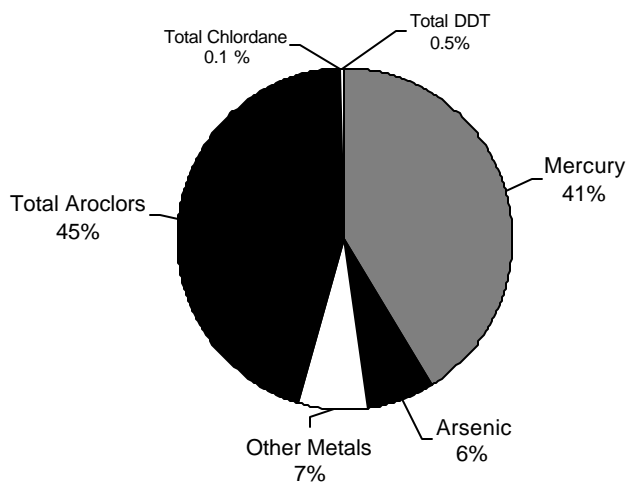


Figure 6-12. Percent contribution of basin-wide chemical concentrations to non-cancer hazards from consumption of coho salmon. Number of samples = 3.

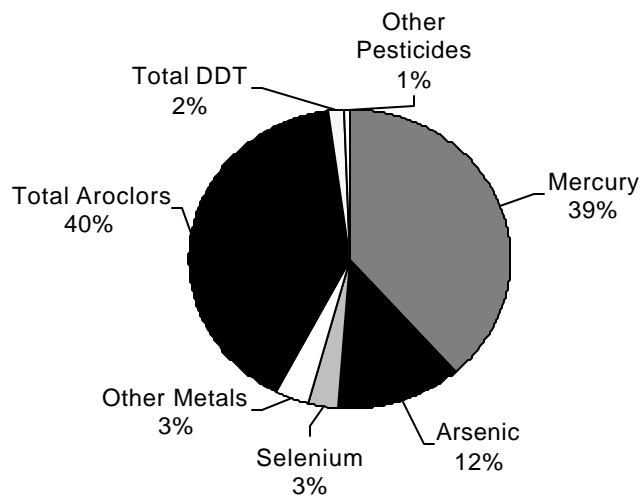


Figure 6-13. Percent contribution of basin-wide average chemical concentrations to non-cancer hazards from consumption of fall chinook fillet with skin. Number of samples = 15.

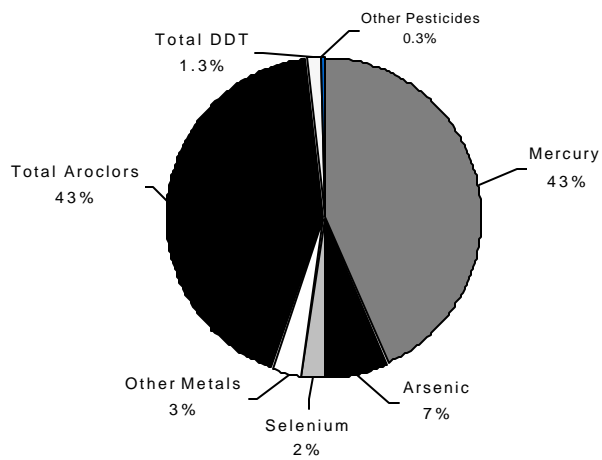


Figure 6-14. Percent contribution of basin-wide average chemical concentrations to non-cancer hazards from consumption of steelhead fillet with skin. Number of samples = 21.

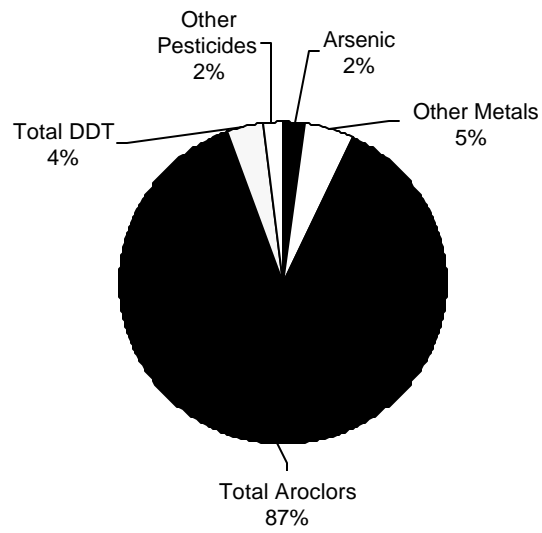


Figure 6-15. Percent contribution of basin-wide average chemical concentrations to non-cancer hazards from consumption of Pacific lamprey fillet with skin. Number of samples = 3.

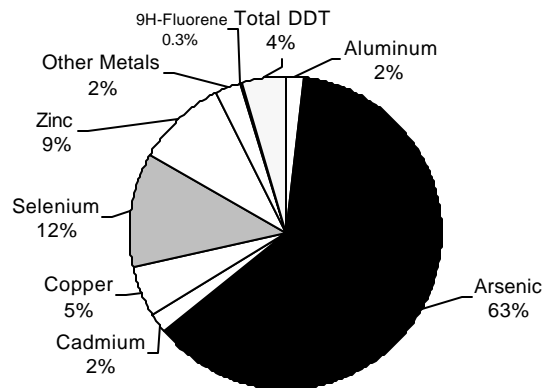


Figure 6-16. Percent contribution of basin-wide average chemical concentrations to non-cancer hazards from consumption of whole body eulachon. Number of samples = 3.

### 6.2.1.3 Comparisons Between Anadromous Fish and Resident Fish Species

A comparison of the total hazard indices, endpoint specific hazard indices, and chemicals with hazard quotients greater than 1.0 among all of the fish species (resident fish and anadromous fish) can be made using the summary tables in Appendices O and P. The conclusions from these comparisons, are limited by the fact that different species were caught at different study sites and that sample numbers and sample types for each species varied.

- The endpoint specific hazard indices that were greater than 1 the most often and that had the highest values for all of the resident fish species were immunotoxicity, central nervous system, reproduction/developmental, and liver, with immunotoxicity usually having the highest endpoint specific hazard index. For resident fish species, endpoint specific hazard indices were rarely greater than 1 for children and adults in the general population with an average fish ingestion rate. The exceptions to this were white sturgeon and mountain whitefish caught in the Hanford Reach of the Columbia River (study site 9U), where endpoint specific hazard indices were greater than 1 (high of 2.7) for the endpoint of immunotoxicity. This was due to exposures to Aroclor greater than its reference dose.
- For salmon and steelhead, three of these endpoints were also the ones that also had the highest hazard indices: immunotoxicity, central nervous system, and reproduction/developmental, with most endpoints specific hazard indices being within a small range among the three salmon and steelhead (the exception is for the Klickitat due to mercury levels in spring chinook). No endpoint specific hazard indices were greater than 1 for children or adults in the general population with an average fish ingestion rate.
- For Pacific lamprey fillet with skin, the major contributor to non-cancer hazards was due to immunotoxicity; for whole body lamprey, it was immunotoxicity as well as central nervous system and reproduction/development endpoints (due to higher levels of mercury in whole body samples of lamprey). There were no endpoint specific hazard indices greater than 1 for the general population (adults or children) with an average fish consumption rate.
- For eulachon, only the endpoints of cardiovascular and hyperpigmentation/keratosis had hazard indices greater than 1 and only at the highest exposures (CRITFC's member tribal adults and children, high fish consumption).

Hazard indices greater than 1 for specific endpoints were primarily a result of elevated hazard quotients for a few chemicals: total Aroclors (immunotoxicity), mercury (central nervous system, and reproduction/developmental), total DDTs (liver), and arsenic (cardiovascular and hyperpigmentation/keratosis). This can be seen in the figures previously discussed for resident fish species (Figures 6-4 to 6-9) and anadromous fish species (Figures 6-11 to 6-16).

Although similar endpoint specific hazard indices were exceeded for many of the fish species tested, the magnitude of both the endpoint specific and total hazard indices vary substantially

among the species. Table 6-9 shows a summary of the non-cancer results across all species at the high fish consumption rate for CRITFC's member tribal adults. All of the non-cancer endpoints that exceed 1.0 are shown for each species as are the range in total hazard indices across study sites and the total hazard index for the basin. For this table, fillet with skin data were used except for the species that had no fillet with skin samples (fillet without skin data for sturgeon and whole body for bridgelip sucker and eulachon).

**Table 6-9. Summary of endpoint specific hazard indices and total hazard indices (by study site and basin-wide) for CRITFC's tribal member adult, high fish consumption.**

Species	N	Sample type	Non-cancer endpoints						Range in study site total hazard indices	Total basin hazard index
			Central nervous system	Reproduction/developmental	Immuno-toxicity	Cardio-vascular	Liver	Hyperpig-mentation		
<b>Resident Species</b>										
Bridgelip sucker	3	WB	2	2	17	6	<1	<1	27	27*
Largescale	19	FS	5 - 20	5 - 20	<1 - 21	1 - 7	<1	<1	10 - 45	29
Mt. whitefish	12	FS	<1 - 7	<1 - 7	4 - 140	<1 -	<1	<1	9 - 150	65
White sturgeon	16	FW	3 - 20	3 - 20	16 - 108	6 - 21	<1	<1	29 - 150	49
Walleye	3	FS	10	10	4	4	<1	<1	18	18*
Rainbow trout	7	FS	4, 5	4, 5	3, 4	<1	<1	<1	8, 10	9
<b>Anadromous species</b>										
Coho salmon	3	FS	7	7	7	<1	<1	<1	16	16*
Fall chinook	15	FS	3 - 6	3 - 6	<1 - 8	<1	1 - 2	1 - 2	6 - 16	12
Spring chinook	24	FS	<1 - 17	<1 - 17	3 - 6	<1	2	2	6 - 24	13
Steelhead	21	FS	4 - 8	4 - 8	3 - 6	<1	1 - 2	1 - 2	9 - 15	16
Eulachon	3	WB	<1	<1	<1	<1	2	2	3	3*
Pacific lamprey	3	FS	<1	<1	24	2	<1	<1	28	28*

N= Number of samples; FW = fillet without skin; FS = fillet with skin, WB = whole body

\*Columbia River Basin index based on study site.

A review of Table 6-9 (reference to study site specific information can be found in the tables in Appendices O and P) suggests that:

- For *eulachon*, all of the endpoint specific hazard indices were equal to or less than 2. The endpoint specific hazard indices were at or less than 2 for *Pacific lamprey* with the exception of a value of 24 for immunotoxicity. This was due to exposures greater than the reference dose for Aroclors. Total basin-wide hazard indices were 3 and 28, respectively, for eulachon and lamprey.
- For the *salmon and steelhead*, all of the study site endpoint specific hazard indices were 8 or less, except for one study site/species (hazard index of 17 for spring chinook for reproduction/development and central nervous system due to mercury in the sample from the Klickitat River). The total basin-wide hazard indices range from 12 to 16 for salmon and steelhead.
- For two of the resident fish species, *walleye* and *rainbow trout*, the endpoint specific

hazard indices were at or less than 10. The endpoint specific hazard index for *bridgelip sucker* were less than 6, with the exception of immunotoxicity which had a value of 17. The total basin-wide hazard indices were 9, 18 and 27 for rainbow trout, walleye and bridgelip sucker, respectively.

- For *largescale sucker* the endpoint specific hazard indices for the central nervous system and reproductive/development range from 5 to 20 and for immunotoxicity from <1 to 21. The study site total hazard indices were from 10 to 45 with five of the six study site total hazard indices being greater than 20.
- The resident fish species, *mountain whitefish* and *sturgeon*, had the highest total study site hazard indices which ranged from 9 to 150 and 29 to 150, respectively. For the *whitefish*, total hazard indices were 9 (Umatilla), 13 (Deschutes), 72 (Yakima), and 150 (Hanford Reach of the Columbia, study site 9U)(see Table 3.1). The two highest values (72 for the Yakima and 150 for the Columbia at 9U) were due primarily to the high endpoint specific hazard indices for immunotoxicity (due to Aroclors) at these study sites. For *sturgeon*, all of the study site total hazard indices were greater than 20: hazard indices of 29 (Columbia at study sites 7 and 8); 40 (Columbia, study site 6); 46 (Snake, study site 13); 62 (Columbia, study site 9L); and 150 (Columbia, study site 9U)(see Table 4.1). The high values for sturgeon were also in large part also due to exposures greater than the reference dose for Aroclors resulting in high endpoint specific hazard indices for immunotoxicity. It is obvious from Table 6-9 that for these 2 species (whitefish and sturgeon), their high endpoint specific hazard indices for immunotoxicity (due to total Aroclors) at some study sites tend to distinguish them from the other species.

Figure 6-17 is a summary of the total hazard indices for each species for all four ingestion rates for adults (general public adult, average and high fish consumption; CRITFC's member tribal adult, average and high fish consumption). Basin-wide fillet with skin data were used for this figure, except for those species that had only whole body samples (bridgelip sucker and eulachon) or fillet without skin (sturgeon) data. As can be seen from this table, the total hazard indices vary by species with white sturgeon and mountain whitefish having the highest total hazard indices among the 12 fish sampled. Largescale sucker, lamprey, and bridgelip sucker had similar but lower total hazard indices followed by the salmon, steelhead, and walleye, then rainbow trout and eulachon.



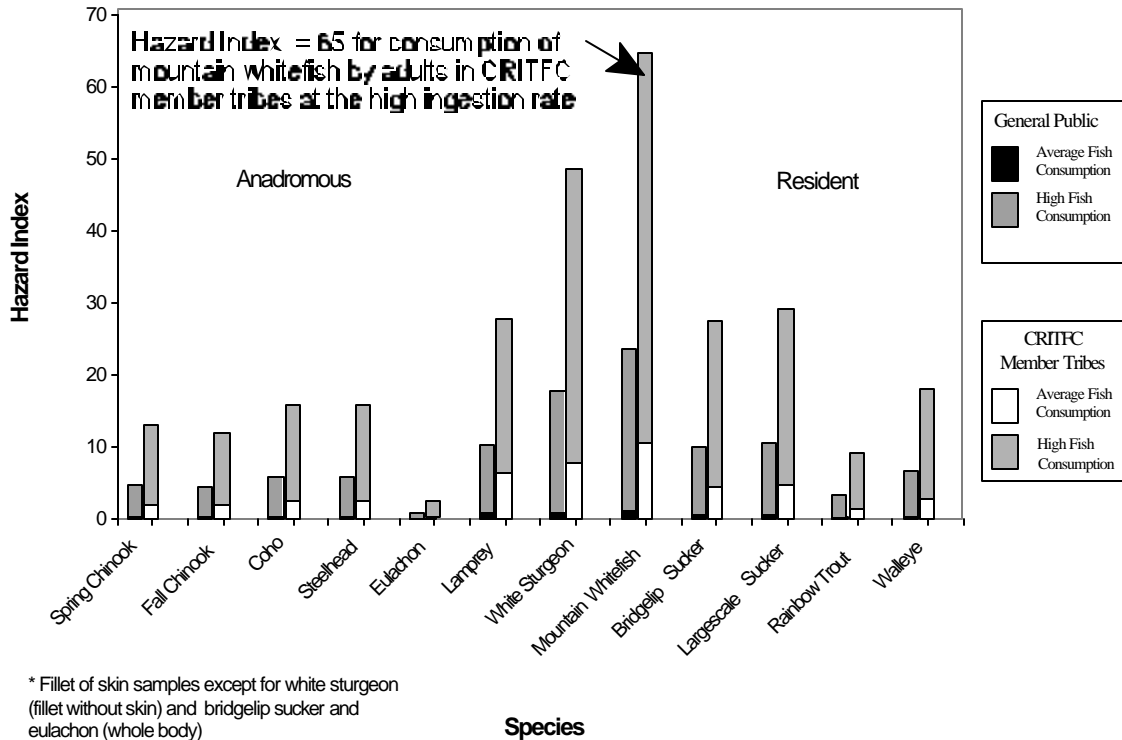


Figure 6-17. Adult total non-cancer hazard indices across all species\*. Columbia River Basin data.

As was previously discussed for white sturgeon (Figures 6-2a-d), the estimated hazard indices for children were different than those for adults. For the general public, the hazard indices for children at the average fish ingestion were about 0.9 of those for adults at the average ingestion rate; the hazard indices for children at the high ingestion rate were about 1.3 times those for adults at the high ingestion rate. For CRITFC’s member tribes, the hazard indices for children at the average and high ingestion rates were both about 1.9 times those for CRITFC’s member tribal adults at the average and high ingestion rates, respectively.

Appendix M contains a comparison of the total and endpoint specific hazard indices across sites (anadromous and resident fish species) for CRITFC’s member tribal children with a high ingestion rate. This was the population with the highest exposures and hazard indices.

### 6.2.2 Cancer Risk Evaluation

Because the incremental increase in cancer risks resulting from ingestion of fish was calculated for adults only, only four populations had cancer risk estimates: average and high fish consumption for both the general public adult and CRITFC’s member tribal adult. However, for

cancer risk, exposure duration does have an impact on the calculations. Therefore, risks were estimated for both 30 and 70 year exposure durations. This results in eight separate cancer risk calculations per study site and in the basin:

***Average Fish Consumption***

General public adult, 30 years	CRITFC's member tribal adult, 30 years
General public adult, 70 years	CRITFC's member tribal adult, 70 years

***High Fish Consumption***

General public adult, 30 years	CRITFC's member tribal adult, 30 years
General public adult, 70 years	CRITFC's member tribal adult, 70 years

The cancer risks calculated for each chemical for each study site are shown in Appendices I1 (general public and CRITFC's member tribal adults, 30 year exposure) and I2 (general public and CRITFC's member tribal adults, 70 year exposure). Appendix N shows the species specific cancer risks by study site over a range of fish ingestion rates. Appendices O and P, which were previously used for discussion of the non-cancer results, include summary results for the total cancer risk estimates by fish species and tissue type. Included in Appendices O and P are: (1) tables showing the total cancer risks by study site and basin for all 8 separate cancer risk calculations, and (2) tables showing the cancer risks by study site for those chemicals that were at or greater than a cancer risk of  $1 \times 10^{-5}$  for one population, CRITFC's member tribal adults, average fish consumption, 70 years exposure.

As with the non-cancer summary, a more detailed discussion of cancer risk will be done with one species, white sturgeon. This will be followed by a summary of the cancer risks for the rest of the resident fish species, the anadromous fish species, and finally, a summary across all species.

As previously discussed in Section 6.1.2, all of the cancer risks discussed in this risk characterization should be considered to be upper bound estimates of the increased risk of developing cancer as a result of fish consumption.

**6.2.2.1 Cancer Risk Evaluation for Resident Fish**

The potential cancer risks associated with consumption of fillet without skin and whole body white sturgeon were assessed by first calculating the risk for all detected chemicals with cancer slope factors (see Appendix I). These chemical specific risks in each sample were then summed to estimate the total cancer risk for a study site and for the basin. For sturgeon, these results are shown in Table 6-10.

**Table 6-10. Summary of total estimated cancer risks for white sturgeon.**

Consumption Rate/ Exposure Duration	Tissue Type	Total Excess Cancer Risk						Basin Average
		Study Site <sup>e</sup>						
		CR -6	CR -7	CR- 8	CR -9L	CR -9U	SR -13	
<b>General Public<sup>a,b</sup></b>								
AFC/30-yr	FW	4X10 <sup>-5</sup>	3X10 <sup>-5</sup>	4X10 <sup>-5</sup>	8X10 <sup>-5</sup>	1X10 <sup>-4</sup>	3X10 <sup>-5</sup>	5X10 <sup>-5</sup>
	WB	na	na	7X10 <sup>-5</sup>	6X10 <sup>-5</sup>	7X10 <sup>-5</sup>	na	7X10 <sup>-5</sup>
HFC/30-yr	FW	8X10 <sup>-4</sup>	6X10 <sup>-4</sup>	7X10 <sup>-4</sup>	1X10 <sup>-3</sup>	2X10 <sup>-3</sup>	6X10 <sup>-4</sup>	9X10 <sup>-4</sup>
	WB	na	na	1X10 <sup>-3</sup>	1X10 <sup>-3</sup>	1X10 <sup>-3</sup>	na	1X10 <sup>-3</sup>
AFC/70-yr	FW	9X10 <sup>-5</sup>	7X10 <sup>-5</sup>	8X10 <sup>-5</sup>	2X10 <sup>-4</sup>	3X10 <sup>-4</sup>	7X10 <sup>-5</sup>	1X10 <sup>-4</sup>
	WB	na	na	2X10 <sup>-4</sup>	1X10 <sup>-4</sup>	2X10 <sup>-4</sup>	na	2X10 <sup>-4</sup>
HFC/70-yr	FW	2X10 <sup>-3</sup>	1X10 <sup>-3</sup>	2X10 <sup>-3</sup>	3X10 <sup>-3</sup>	5X10 <sup>-3</sup>	1X10 <sup>-3</sup>	2X10 <sup>-3</sup>
	WB	na	na	3X10 <sup>-3</sup>	3X10 <sup>-3</sup>	3X10 <sup>-3</sup>	na	3X10 <sup>-3</sup>
<b>CRITFC's Tribal Member<sup>c,d</sup></b>								
AFC/30-yr	FW	3X10 <sup>-4</sup>	3X10 <sup>-4</sup>	3X10 <sup>-4</sup>	6X10 <sup>-4</sup>	1X10 <sup>-3</sup>	3X10 <sup>-4</sup>	4X10 <sup>-4</sup>
	WB	na	na	6X10 <sup>-4</sup>	5X10 <sup>-4</sup>	6X10 <sup>-4</sup>	na	6X10 <sup>-4</sup>
HFC/30-yr	FW	2X10 <sup>-3</sup>	2X10 <sup>-3</sup>	2X10 <sup>-3</sup>	4X10 <sup>-3</sup>	6X10 <sup>-3</sup>	2X10 <sup>-3</sup>	3X10 <sup>-3</sup>
	WB	na	na	4X10 <sup>-3</sup>	3X10 <sup>-3</sup>	4X10 <sup>-3</sup>	na	3X10 <sup>-3</sup>
AFC/70-yr	FW	8X10 <sup>-4</sup>	6X10 <sup>-4</sup>	7X10 <sup>-4</sup>	1X10 <sup>-3</sup>	2X10 <sup>-3</sup>	6X10 <sup>-4</sup>	1X10 <sup>-3</sup>
	WB	na	na	1X10 <sup>-3</sup>	1X10 <sup>-3</sup>	1X10 <sup>-3</sup>	na	1X10 <sup>-3</sup>
HFC/70-yr	FW	5X10 <sup>-3</sup>	4X10 <sup>-3</sup>	4X10 <sup>-3</sup>	9X10 <sup>-3</sup>	1X10 <sup>-2</sup>	4X10 <sup>-3</sup>	6X10 <sup>-3</sup>
	WB	na	na	9X10 <sup>-3</sup>	7X10 <sup>-3</sup>	8X10 <sup>-3</sup>	na	8X10 <sup>-3</sup>

AFC - average fish consumption HFC - high fish consumption FW - fillet without skin WB - whole body

na - not applicable; sample type not analyzed at this study site

<sup>a</sup>AFC risk based on average U.S. per capita consumption rate of uncooked freshwater and estuarine fish for general public of 7.5 g/day, or 1 8-oz meal per month (USEPA, 2000a).

<sup>b</sup>HFC risk based on 99th percentile U.S. per capita consumption rate of uncooked freshwater and estuarine fish for general public of 142.4 g/day, or 19 8-oz meals per month (USEPA, 2000a).

<sup>c</sup>AFC risk based on average consumption rate for fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin of 63.2 g/day, or 9 8-oz meals per month (CRITFC 1994).

<sup>d</sup>HFC risk based on 99th percentile consumption rate for fish consumers in the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin of 389 g/day, or 53 8-oz meals per month (CRITFC 1994).

<sup>e</sup> Study site descriptions are in Table 1.1. CR = Columbia River; SR = Snake River

As can be seen from Table 6-10, for white sturgeon the total excess cancer risks range from a low of 3 X 10<sup>-5</sup> in fillet without skin samples from the Columbia River (study site 7) and the Snake River (study site 13) assuming an average fish consumption rate and a 30 year exposure for the general population adult to a high of 1 X 10<sup>-2</sup> in fillet without skin samples from the Columbia (study site 9U) assuming a high fish consumption rate and a 70 year exposure duration for CRITFC's member tribal adults.

The estimated upper bound cancer risks differ by study site for sturgeon since contaminant levels vary by study site (Table 6-10). For example, for one exposure - CRITFC's member tribal adult, average fish consumption, 30 year exposure - the ingestion of sturgeon (fillet without skin) from

the Columbia River (study sites 6, 7 and 8) and the Snake River (study site 13) results in the same estimated cancer risk,  $3 \times 10^{-4}$ , while the risks estimated from consuming fish from the Columbia River, study site 9L ( $6 \times 10^{-4}$ ) and study site 9U ( $1 \times 10^{-3}$ ) were higher. This same difference was seen across all study sites (within a given sample type) for each of the exposure groups evaluated for cancer risk.

As previously discussed for non-cancer effects, the cancer risk at a given study site increases proportionally with increasing exposure. For cancer risks, exposures were lowest for the general public adult, average fish consumption, 30 years exposure and highest for CRITFC's member tribal adult, high fish consumption, 70 years exposure and depend both upon the exposure duration (30 or 70 year) and fish consumption rate. Table 6-11 shows the total cancer risks for all adult populations for white sturgeon (whole body) caught in the Columbia River at study site 8. Also shown are the ratios of the total cancer risks for the general public, average fish consumption at 30 years exposure to that of the other groups assessed in this risk assessment: CRITFC's member tribal adults with average and high fish consumption at both 30 and 70 years exposure; the general public adults with high fish consumption at 30 years exposure, and; the general public adults with average and high fish ingestion at 70 years exposure. As can be seen from this table, for whole body samples of sturgeon at Columbia River study site 8, the estimated upper bound cancer risk from eating fish was  $7 \times 10^{-5}$  for the general public, average fish consumption and 30 years exposure and  $1 \times 10^{-3}$  for the general public, high fish consumption and 30 years exposure. This was a difference of about 19 fold (when the rounding of the values in this table are accounted for). Likewise, the risks from eating sturgeon for the general public, average fish consumption and 70 years exposure was about 2 times higher than that for general public, average fish consumption and 30 years exposure.

Figure 6-18 shows the differences in cancer risks across sites for sturgeon (fillet without skin) for CRITFC member tribal adults and general public adults at the high fish consumption for both 30 and 70 year exposures. As can be seen, the cancer risks vary by site with the Hanford Reach of the Columbia River (site 9U) having the highest estimated risks.

**Table 6-11. Comparison of estimated total cancer risks among adult populations**

	<b>Fish ingestion rate (grams/day)</b>	<b>Exposure duration (years)</b>	<b>Total cancer risk for adults for white sturgeon at Columbia River, study site 8 (whole body samples)</b>	<b>Approximate ratio of estimated cancer risks to that of general public with average fish consumption, 30 years exposure</b>
General public	average (7.5)	30	$7 \times 10^{-5}$	1
General public	high (142.4)	30	$1 \times 10^{-3}$	19
CRITFC's member tribe	average (63.2)	30	$6 \times 10^{-4}$	8
CRITFC's member tribe	high (389)	30	$4 \times 10^{-3}$	52
General public	average (7.5)	70	$2 \times 10^{-4}$	2
General public	high (142.4)	70	$3 \times 10^{-3}$	44
CRITFC's member tribe	average (63.2)	70	$1 \times 10^{-3}$	20
CRITFC's member tribe	high (389)	70	$9 \times 10^{-3}$	121

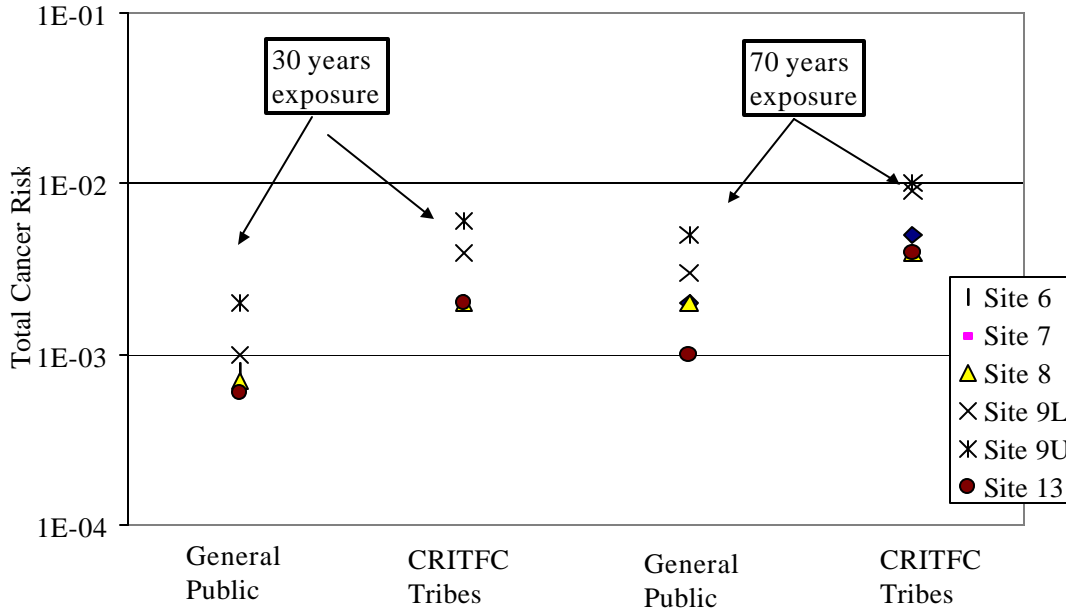


Figure 6-18. Comparison of estimated total cancer risks for consumption of white sturgeon across study sites for adults in the general public and CRITFC’s member tribes at high consumption rates. Note that cancer risks for consumption of white sturgeon are the same for study sites 7 and 13.

Figure 6-19 shows the linear relationship between fish ingestion rate and estimated upper bound basin-wide cancer risk for adults for basin-wide average concentration of chemicals in white sturgeon fillet samples from the Columbia River Basin assuming both 30 and 70 years exposure duration. It also shows that cancer risks for a 70 year exposure were about 2 fold (i.e., 70 years/30 years = 2.3) higher than those for a 30 year exposure (see Appendix N for similar figures by study site and species).

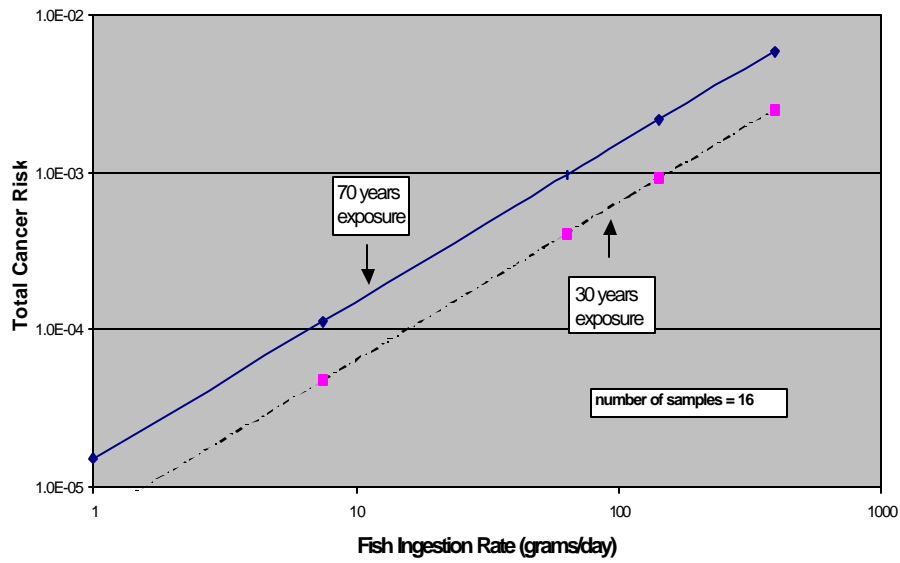


Figure 6-19. Total cancer risks versus fish consumption rate for adults. White sturgeon, basin-wide data (fillet with skin).

In the previous discussion on non-cancer results, it was shown that a small number of chemicals were responsible for most of the non-cancer health hazards from consuming fish. Tables 6-12 (fillet without skin) and Table 6-13 (whole body) show the chemicals with cancer risks at or greater than  $1 \times 10^{-5}$  for sturgeon for CRITFC's member tribal adults, average fish consumption and 70 years exposure duration. For cancer risks, a limited (but larger) number of chemicals were responsible for the majority of the cancer risk. These chemicals are:

- PCBs, including both Aroclors and dioxin-like PCB congeners,
- chlorinated dioxins and furans, with 2,3,7,8,-TCDF having the highest risk among the congeners,
- the pesticides aldrin, chlordane (total), DDD, DDE, and hexachlorobenzene, with DDE having the highest risk, and
- one metal, arsenic.

Not all chemicals were detected at every study site. For example, in the table with fillet without skin results (Table 6-12), Aroclors and PCB congeners 105, 118 and 156 were detected in all of the study site samples while other PCB congeners were detected at only one or two study sites.

**Table 6-12. Chemicals with estimated cancer risks at or greater than  $1 \times 10^{-5}$  for white sturgeon, fillet without skin. CRITFC's member tribal adult, average fish consumption, 70 years exposure.**

	Study Site*					
	CR - 6	CR-7	CR -8	SR -13	CR -9L	CR -9U
<b>PCBs</b>						
Total Aroclors**	$2 \times 10^{-4}$	$1 \times 10^{-4}$	$1 \times 10^{-4}$	$1 \times 10^{-4}$	$3 \times 10^{-4}$	$7 \times 10^{-4}$
PCB 105	$3 \times 10^{-5}$	$2 \times 10^{-5}$	$2 \times 10^{-5}$	$3 \times 10^{-5}$	$4 \times 10^{-5}$	$1 \times 10^{-4}$
PCB 114	$1 \times 10^{-5}$	<	<	$1 \times 10^{-5}$	$2 \times 10^{-5}$	$5 \times 10^{-5}$
PCB 118	$3 \times 10^{-5}$	$2 \times 10^{-5}$	$2 \times 10^{-5}$	$4 \times 10^{-5}$	$5 \times 10^{-5}$	$2 \times 10^{-4}$
PCB 126	<	$2 \times 10^{-5}$	<	<	<	<
PCB 156	$4 \times 10^{-5}$	$3 \times 10^{-5}$	$3 \times 10^{-5}$	$5 \times 10^{-5}$	$9 \times 10^{-5}$	$2 \times 10^{-4}$
PCB 157	<	<	<	<	$2 \times 10^{-5}$	$5 \times 10^{-5}$
<b>Dioxin/furans</b>						
1,2,3,7,8-PeCDD	$1 \times 10^{-5}$	$2 \times 10^{-5}$	$2 \times 10^{-5}$	$1 \times 10^{-5}$	<	<
2,3,4,7,8-PeCDF	<	$1 \times 10^{-5}$	$2 \times 10^{-5}$	<	$2 \times 10^{-5}$	$2 \times 10^{-5}$
2,3,7,8-TCDD	$4 \times 10^{-5}$	$5 \times 10^{-5}$	$6 \times 10^{-5}$	$5 \times 10^{-5}$	$1 \times 10^{-4}$	$3 \times 10^{-5}$
2,3,7,8-TCDF	$2 \times 10^{-4}$	$2 \times 10^{-4}$	$2 \times 10^{-4}$	$6 \times 10^{-5}$	$5 \times 10^{-4}$	$3 \times 10^{-4}$
<b>Pesticides</b>						
Aldrin	<	<	<	<	$2 \times 10^{-5}$	$1 \times 10^{-5}$
Chlordane (total)	<	<	<	<	$1 \times 10^{-5}$	$2 \times 10^{-5}$
DDD	$1 \times 10^{-5}$	$1 \times 10^{-5}$	$1 \times 10^{-5}$	$1 \times 10^{-5}$	$4 \times 10^{-5}$	$8 \times 10^{-5}$
DDE	$1 \times 10^{-4}$	$1 \times 10^{-4}$	$1 \times 10^{-4}$	$1 \times 10^{-4}$	$2 \times 10^{-4}$	$4 \times 10^{-4}$
Hexachlorobenzene	<	<	<	<	$2 \times 10^{-5}$	<
<b>Metals</b>						
Arsenic	$4 \times 10^{-5}$	$5 \times 10^{-5}$	$5 \times 10^{-5}$	$3 \times 10^{-5}$	$5 \times 10^{-5}$	$4 \times 10^{-5}$
<b>Total Cancer Risk for All Chemicals</b>	$8 \times 10^{-4}$	$6 \times 10^{-4}$	$7 \times 10^{-4}$	$6 \times 10^{-4}$	$1 \times 10^{-3}$	$2 \times 10^{-3}$

"<" means that estimated cancer risk was less than  $1 \times 10^{-5}$  \*Study site descriptions are in Table 1.1. CR = Columbia River; SR = Snake River

\*\* Based on "adjusted" Aroclor concentration (see Section 5.3.2)

**Table 6-13. Chemicals with estimated cancer risks at or greater than  $1 \times 10^{-5}$  for white sturgeon, whole body. CRITFC's member tribal adult, average fish consumption, 70 years exposure.**

	Study Site*		
	CR - 8	CR -9L	CR -9U
<b>PCBs</b>			
Total Aroclors**	$3 \times 10^{-4}$	$2 \times 10^{-4}$	$3 \times 10^{-4}$
PCB 105	$6 \times 10^{-5}$	$4 \times 10^{-5}$	$5 \times 10^{-5}$
PCB 114	$2 \times 10^{-5}$	$2 \times 10^{-5}$	$2 \times 10^{-5}$
PCB 118	$7 \times 10^{-5}$	$5 \times 10^{-5}$	$5 \times 10^{-5}$
PCB 156	$1 \times 10^{-4}$	$9 \times 10^{-5}$	$9 \times 10^{-5}$
PCB 157	$2 \times 10^{-5}$	$2 \times 10^{-5}$	$2 \times 10^{-5}$
<b>Dioxin/furans</b>			
2,3,4,7,8-PeCDF	$2 \times 10^{-5}$	$3 \times 10^{-5}$	$2 \times 10^{-5}$
2,3,7,8-TCDD	$9 \times 10^{-5}$	$1 \times 10^{-4}$	$9 \times 10^{-5}$
2,3,7,8-TCDF	$3 \times 10^{-4}$	$3 \times 10^{-4}$	$4 \times 10^{-4}$
<b>Pesticides</b>			
Aldrin	<	$2 \times 10^{-5}$	$2 \times 10^{-5}$
Chlordane (total)	<	$1 \times 10^{-5}$	<
DDD	$2 \times 10^{-5}$	$3 \times 10^{-5}$	$5 \times 10^{-5}$
DDE	$2 \times 10^{-4}$	$2 \times 10^{-4}$	$2 \times 10^{-4}$
Hexachlorobenzene	<	$2 \times 10^{-5}$	$1 \times 10^{-5}$
<b>Metals</b>			
Arsenic	$7 \times 10^{-5}$	$4 \times 10^{-5}$	$4 \times 10^{-5}$
<b>Total Cancer Risk for All Chemicals</b>	$1 \times 10^{-3}$	$1 \times 10^{-3}$	$1 \times 10^{-3}$

"<" means that estimated cancer risk was less than  $1 \times 10^{-5}$ . CR = Columbia River

\*\*Based on "adjusted" Aroclor concentration (see Section 5.3.2)

The total cancer risk estimates and the summary of chemicals with risks at or greater than  $1 \times 10^{-5}$  for other resident fish species are provided in Appendix O by species: Tables 1.3 and 1.4 (bridgelip sucker), 2.3 and 2.4 (largescale sucker), 3.3 and 3.4 (mountain whitefish), 4.3 and 4.4 (white sturgeon), 5.3 and 5.4 (walleye), and 6.3 and 6.4 (rainbow trout). Table 6-14 shows a summary of the total cancer risk estimates for the resident fish species for one adult population - CRITFC's member tribal adults with an average fish consumption and 70 years exposure. Results of the fillet with skin samples are shown, except for sturgeon (only fillet without skin sampled) and bridgelip sucker (only whole body sampled).

**Table 6-14. Summary of estimated total cancer risks by study site and basin-wide, resident fish species. CRITFC's tribal member adult, average fish consumption, 70 years exposure**

Species	N	Sample type	Study site name	Study Site	Study site cancer risk	Range in study site cancer risks	Basin cancer risk
Bridgelip sucker	3	WB	Yakima	48	$5 \times 10^{-4}$	$5 \times 10^{-4}$	$5 \times 10^{-4*}$
Largescale sucker	19	FS	Columbia	9U	$6 \times 10^{-4}$	1 to $6 \times 10^{-4}$	$4 \times 10^{-4}$
			Deschutes	98	$1 \times 10^{-4}$		
			Umatilla	30	$2 \times 10^{-4}$		
			Snake	13	$2 \times 10^{-4}$		
			Yakima	48	$4 \times 10^{-4}$		
			Yakima	49	$3 \times 10^{-4}$		
Mountain whitefish	12	FS	Columbia	9U	$4 \times 10^{-3}$	1 $\times 10^{-4}$ to $4 \times 10^{-3}$	1 $\times 10^{-3}$
			Deschutes	98	$3 \times 10^{-4}$		
			Umatilla	101	$1 \times 10^{-4}$		
			Yakima	48	$1 \times 10^{-3}$		
White sturgeon	16	FW	Columbia	6	$8 \times 10^{-4}$	6 $\times 10^{-4}$ to 2 $\times 10^{-3}$	1 $\times 10^{-3}$
			Columbia	7	$6 \times 10^{-4}$		
			Columbia	8	$7 \times 10^{-4}$		
			Columbia	9L	$1 \times 10^{-3}$		
			Columbia	9U	$2 \times 10^{-3}$		
Snake	13	$6 \times 10^{-4}$					
Walleye	3	FS	Umatilla	30	$2 \times 10^{-4}$	$2 \times 10^{-4}$	$2 \times 10^{-4*}$
Rainbow trout	7	FS	Deschutes	98	$2 \times 10^{-4}$	$2 \times 10^{-4}$	$2 \times 10^{-4}$
			Yakima	49	$2 \times 10^{-4}$		

N= number of samples; WB = whole body; FS = fillet with skin; FW = fillet without skin

\* Basin-wide cancer risk based on one study site

White sturgeon and mountain whitefish had the highest estimated basin-wide cancer risks at  $1 \times 10^{-3}$  (Table 6-14). All of the white sturgeon study site cancer risks were at or greater than  $6 \times 10^{-4}$  with a high of  $2 \times 10^{-3}$ . The highest cancer risks for sturgeon were from consuming fish from the Columbia River at study sites 9L ( $1 \times 10^{-3}$ ) and 9U ( $2 \times 10^{-3}$ ). The four mountain whitefish study sites span more than an order of magnitude in cancer risk -  $1 \times 10^{-4}$  for the Umatilla (study site 101),  $3 \times 10^{-4}$  for the Deschutes (study site 98),  $1 \times 10^{-3}$  for the Yakima (study site 48), and  $4 \times 10^{-3}$  for the Columbia River (study site 9U). Cancer risks were highest for the Yakima (study site 48) and Columbia River (study site 9U) for whitefish and for the Columbia River at study sites 9U and 9L for sturgeon.

Bridgelip sucker (one study site at  $5 \times 10^{-4}$ ) and largescale sucker (six study sites ranging from 1 to  $6 \times 10^{-4}$ ) had the next highest basin-wide cancer risks,  $5 \times 10^{-4}$  and  $4 \times 10^{-4}$ , respectively. Walleye (one study site at  $2 \times 10^{-4}$ ) and rainbow trout (two study sites at  $2 \times 10^{-4}$ ) had the lowest basin-wide cancer risks.



Figure 6-20 summarizes the total basin-wide cancer risks for resident fish species for adults using high and average fish consumption rates for the general public and for CRITFC’s member tribal populations assuming 70 years exposure duration. Note that the Y axis is on a logarithmic scale and that each bar begins at 0 on the Y axis. For example, the cancer risk for mountain whitefish for the general public adult, high fish consumption for 70 years, is  $3 \times 10^{-3}$ ; for CRITFC member tribal adults, high fish consumption for 70 years, the cancer risk estimates is  $8 \times 10^{-3}$ . As with Table 6-14, this figure shows that consumption of mountain whitefish and white sturgeon result in the highest cancer risks, followed by sucker, rainbow trout, and walleye. It also shows that for all species, the total cancer risks were highest for CRITFC’s member tribal adults at the high fish ingestion rates (389 g/day) followed by the general public adult, high ingestion rate (142.4 g/day); CRITFC’s member tribal adult, average ingestion rate (63.2 g/day); and general public adult, average ingestion rate (7.5 g/day).

For a more detailed comparison of cancer risks across resident fish species for each study site, see Appendix N. In this appendix, cancer risks are shown over a range of ingestion rates for all species caught at a study site.

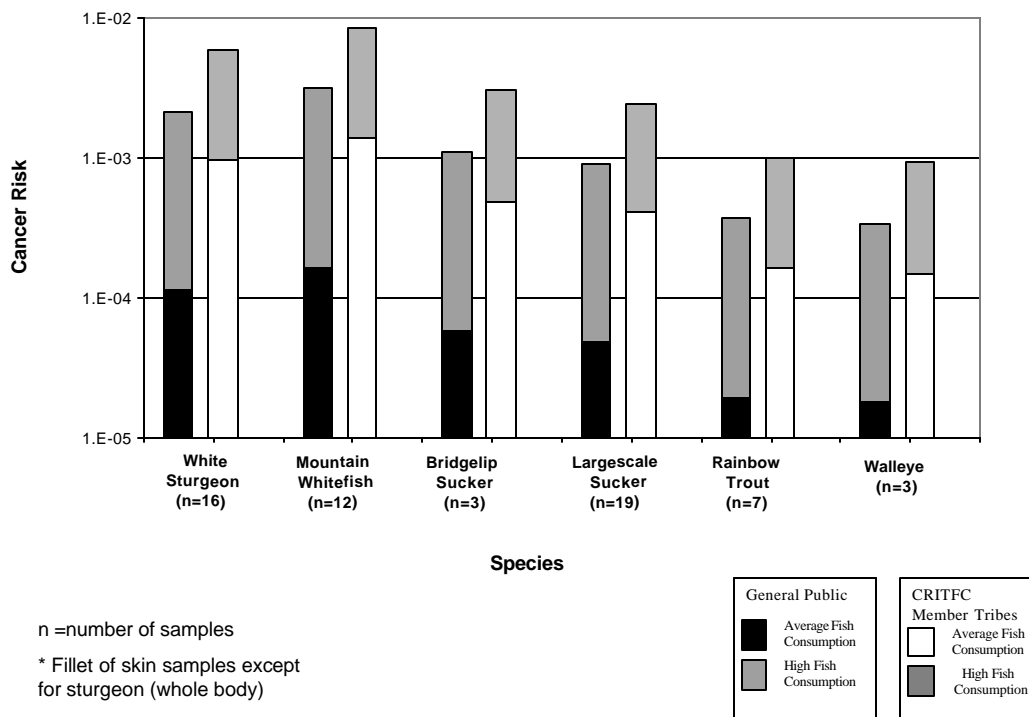


Figure 6-20. Adult cancer risks for resident fish species\*. Columbia River Basin data (70 years exposure).

The chemicals with cancer risks equal to or greater than  $1 \times 10^{-5}$  for resident fish species are shown in Appendix O for CRITFC's member tribal adults for the average fish consumption rate and 70 years exposure (Tables 1.4 (bridgelip sucker), 2.4.1 and 2.4.2 (largescale sucker), 3.4.1 and 3.4.2 (mountain whitefish), 4.4.1 and 4.4.2 (white sturgeon), 5.4.1 and 5.4.2 (walleye), and 6.4.1 and 6.4.2 (rainbow trout)).

In general, four chemical classes (PCBs, chlorinated dioxins and furans, pesticides and metals) were responsible for the cancer risks at or greater than  $1 \times 10^{-5}$  for all of the resident fish species. The exception to this was two study site samples for largescale sucker: the Snake River (study site 13, fillet with skin) had 2 semivolatiles at or greater than a  $1 \times 10^{-5}$  cancer risk, dibenz(a,h)anthracene and benzo(a)pyrene, and the Yakima River (study site 49, whole body) had one, 1,2-diphenylhydrazine.

For the metals, only one of the contaminants detected, inorganic arsenic, had an oral cancer slope factor. Thus, inorganic arsenic was the only detected metal for which cancer risks were estimated.

For the three other classes of chemicals contributing the most to the cancer risk (PCBs, dioxins/furans, and pesticides), the chemicals within each class that were at or greater than  $1 \times 10^{-5}$  vary among species and sometimes among different sample types of the same species. For example, the pesticide, hexachlorobenzene, was found at a level greater than  $1 \times 10^{-5}$  risk in only three white sturgeon samples: at Columbia River study site 9L for fillet without skin and at Columbia River study sites 9L and 9U for whole body samples. Aldrin was found at a cancer risk greater than  $1 \times 10^{-5}$  in only 2 species: at the Columbia River, study sites 9L and 9U, for both types of sturgeon samples (fillet without skin and whole body); and at Columbia River study site 9U for whitefish samples (whole body and fillet with skin).

All study sites and species had total Aroclors at or greater than a risk of  $1 \times 10^{-5}$  except for the Snake River (study site 13) for largescale sucker (fillet with skin). Up to seven different PCB congeners (105, 114, 118, 126, 156, 157 and 169) were found at or greater than a risk of  $1 \times 10^{-5}$  with the number per study site varying from zero to seven at different study sites. Up to four dioxins/furans (2,3,7,8-TCDF, 2,3,4,7,8-PCDF, 2,3,7,8-TCDD and 1,2,3,7,8-PCDD) were at or greater than a cancer risk of  $1 \times 10^{-5}$  with the number varying from two to four per study site.

Table 6-15 and Figures 6-21 through 6-26 show the percent contribution to total cancer risk from each chemical and class of chemical using the basin-wide cancer risk data for resident fish (fillet with skin for all species except sturgeon (fillet without skin) and bridgelip sucker (whole body)).

**Table 6-15. Percent contribution of contaminant groups to estimated cancer risks for resident fish species. Based on Columbia River Basin-wide averages.**

	White Sturgeon	Largescale Sucker	Mountain Whitefish	Walleye	Rainbow Trout	Bridgelip Sucker
<i>Tissue Type</i>	<i>FW</i>	<i>FS</i>	<i>FS</i>	<i>FS</i>	<i>FS</i>	<i>WB</i>
<i>Number of Samples</i>	<i>16</i>	<i>19</i>	<i>12</i>	<i>3</i>	<i>7</i>	<i>3</i>
<b>Total Metals</b>	<b>4</b>	<b>2</b>	<b>1</b>	<b>33</b>	<b>ND</b>	<b>8</b>
Arsenic	4	2	1	33	ND	8
<b>Total PCBs/Aroclors</b>	<b>39</b>	<b>46</b>	<b>83</b>	<b>31</b>	<b>68</b>	<b>46</b>
PCB 105	3	2	6	3	4	2
PCB 114	1	1	2	1	2	1
PCB 118	4	6	15	6	9	3
PCB 126	2	9	18	ND	29	14
PCB 156	6	6	12	6	8	4
PCB 157	1	1	2	ND	2	ND
PCB 169	ND	2	<1	ND	ND	1
Other PCBs	<1	<1	1	<1	<1	<1
Total Aroclors*	21	19	26	15	15	22
<b>Total Semi-Vocatives</b>	<b>ND</b>	<b>28</b>	<b>ND</b>	<b>ND</b>	<b>ND</b>	<b>1</b>
1,2-Diphenylhydrazine	ND	ND	ND	ND	ND	1
Benzo(a)pyrene	ND	8	ND	ND	ND	ND
Dibenz[a,h]anthracene	ND	17	ND	ND	ND	ND
Indeno(1,2,3-cd)pyrene	ND	2	ND	ND	ND	ND
Other Semi-Vocatives	ND	2	ND	ND	ND	ND
<b>Total Pesticides</b>	<b>23</b>	<b>21</b>	<b>10</b>	<b>11</b>	<b>5</b>	<b>32</b>
Aldrin	2	ND	2	ND	ND	ND
DDD	2	1	1	1	<1	3
DDE	15	16	8	10	4	25
DDT	<1	2	<1	<1	1	3
Heptachlor Epoxide	1	ND	ND	ND	ND	ND
Hexachlorobenzene	1	ND	<1	ND	ND	ND
Other Pesticides	2	2	<1	ND	<1	<1
<b>Total Dioxins/Furans</b>	<b>36</b>	<b>5</b>	<b>8</b>	<b>26</b>	<b>29</b>	<b>13</b>
2,3,4,6,7,8-HxCDF	<1	<1	<1	1	2	<1
2,3,4,7,8-PeCDF	1	<1	1	1	2	2
2,3,7,8-TCDD	7	1	1	7	6	2
2,3,7,8-TCDF	26	1	5	6	2	3
OCDD	<1	<1	<1	<1	<1	<1
OCDF	<1	<1	<1	ND	<1	<1
1,2,3,7,8-PeCDD	1	2	2	7	13	5
1,2,3,4,7,8-HxCDD	<1	<1	<1	1	1	<1
other dioxins	1	1	<1	2	4	1

ND=Not detected; \*Based on adjusted Aroclor concentration (See Section 5.3.2)

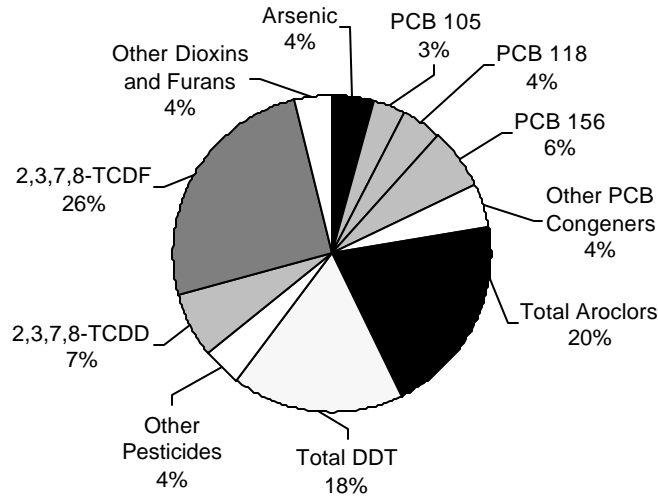


Figure 6-21. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of white sturgeon fillet without skin. Number of samples = 16.

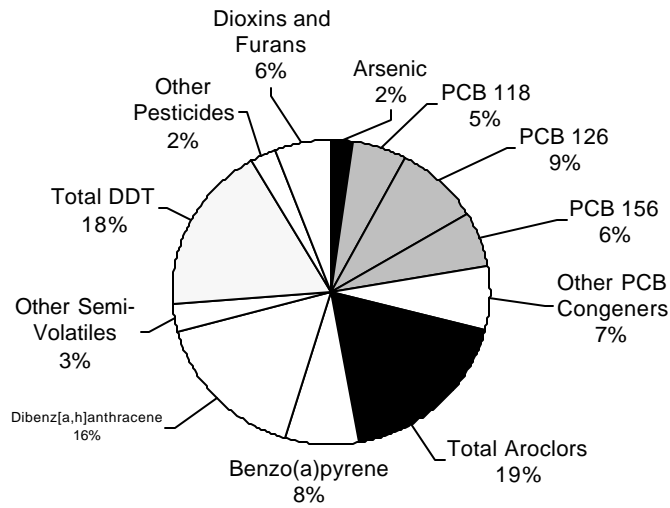


Figure 6-22. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of largescale sucker fillet with skin. Number of samples = 19.

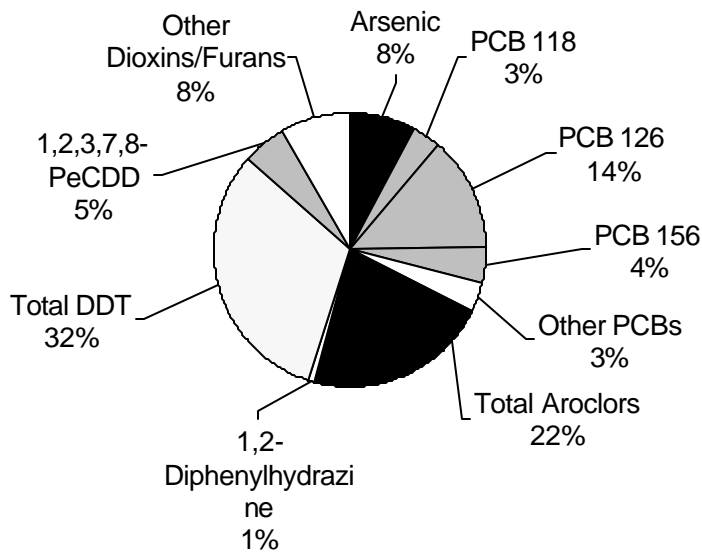


Figure 6-23. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of whole body bridgelip sucker. Number of samples = 3.

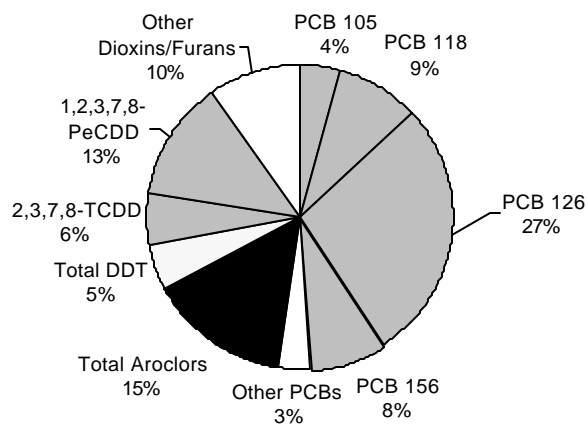


Figure 6-24. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of rainbow trout fillet with skin. Number of samples = 7.

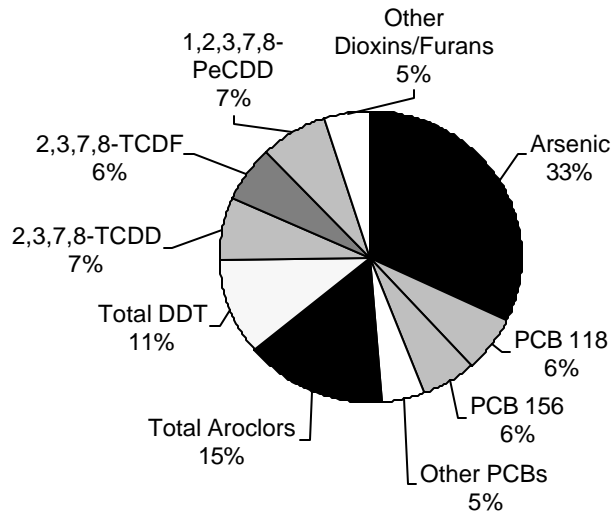


Figure 6-25. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of walleye fillet with skin. Number of samples =3.

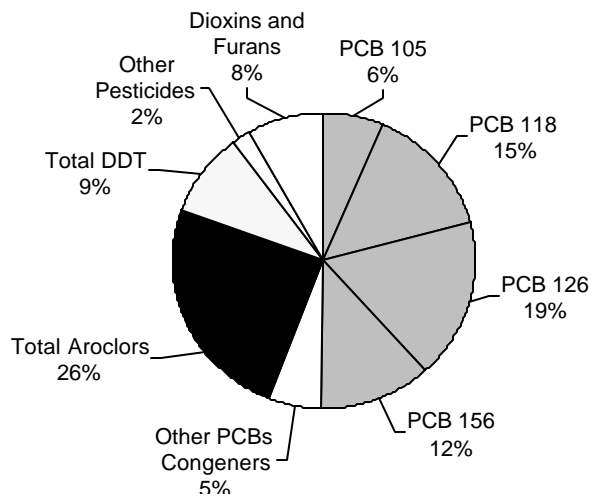


Figure 6-26. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of mountain whitefish fillet with skin. Number of samples = 12.

For all of the resident fish species except walleye, the majority of the cancer risk was from dioxins and furans, a small number of pesticides and PCBs. (Table 6-15 and Figures 6-21 through 6-26). Inorganic arsenic contributes to about 33% of the cancer risk for walleye.

- Chlorinated dioxins and furans contribute from 5% of the total cancer risk for largescale sucker to 36% for sturgeon. For sturgeon, 2,3,7,8-TCDF was by far the largest contributor of the dioxins/furans. For some of the other species, other congeners (e.g., 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD) were contributors to the dioxin/furan cancer risk.
- Pesticides contribute from about 5% to 32% of the total cancer risk, with DDE contributing more than any other pesticide.
- PCBs (both total Aroclors and dioxin-like congeners) contribute from 31% to 83% of the total cancer risk. The contribution from Aroclors (primarily 1254 and 1260) to the cancer risk for this class of chemicals was approximately 15% for rainbow trout, 26% for mountain whitefish, 19% for largescale sucker, 22% for bridgelip sucker, 15% for walleye, and 21% for sturgeon. The contribution to PCB cancer risk from the dioxin-like PCB congeners ranges from a low of 17% for walleye to a high of 56% for mountain whitefish.
- The contribution from inorganic arsenic to total cancer risk was from 0% (not detected in rainbow trout fillets) to 33% for the resident fish species. For most species, the value was less than 8%. The exception was walleye at 33%.

### 6.2.2.2 Cancer Risk Evaluation for Anadromous Fish

The total cancer risk estimates for the anadromous fish species are provided in Appendix P by species: Tables 1.3 (coho salmon), 2.3 (fall chinook salmon), 3.3 (spring chinook salmon), 4.3 (steelhead), 5.3 (eulachon), and 6.3 (Pacific lamprey).

Table 6-16 summarizes the estimates of the total cancer risks for anadromous fish species by study site and by basin for CRITFC's member tribal adults, average consumption rate (63.2 g/day), and 70 years exposure. Fillet with skin data are shown except for eulachon, which had only whole body samples collected. Figure 6-27 shows the relative differences in cancer risks for anadromous fish species using average and high fish consumption rates for the general public and CRITFC's member tribal adult assuming 70 years exposure. Note that the Y axis is on a logarithmic scale and that all of the bars begin at 0 on the Y axis. For example, the cancer risk for Pacific lamprey for the general public adult, high fish consumption for 70 years, is slightly greater than  $1 \times 10^{-3}$ ; for CRITFC member tribal adults, high fish consumption for 70 years, the cancer risk estimates is  $4 \times 10^{-3}$ . Columbia River Basin data are shown for all species (for coho salmon, eulachon and Pacific lamprey, only one study site was sampled).

**Table 6-16. Summary of estimated total cancer risks by study site and basin-wide, anadromous fish species CRITFC's tribal member adult, average fish consumption, 70 years exposure**

Species	N	Sample type	Study site name	Study site #	Study site cancer risk	Range in study site cancer risks	Basin cancer risk					
Coho salmon	3	FS	Umatilla	30	$2 \times 10^{-4}$	$2 \times 10^{-4}$	$2 \times 10^{-4*}$					
Fall chinook salmon	15	FS	Columbia	8	$2 \times 10^{-4}$	1 to $2 \times 10^{-4}$	$2 \times 10^{-4}$					
			Columbia	14	$2 \times 10^{-4}$							
			Klickitat	56	$2 \times 10^{-4}$							
			Umatilla	30	$1 \times 10^{-4}$							
			Yakima	48	$2 \times 10^{-4}$							
Spring chinook salmon	24	FS	Willamette	21	$2 \times 10^{-4}$	2 to $3 \times 10^{-4}$	$2 \times 10^{-4}$					
			Wind River	63	$2 \times 10^{-4}$							
			Little White Salmon	62	$2 \times 10^{-4}$							
			Klickitat	56	$2 \times 10^{-4}$							
			Looking Glass Creek	94	$2 \times 10^{-4}$							
			Umatilla	30	$3 \times 10^{-4}$							
			Yakima	48	$2 \times 10^{-4}$							
			Icicle Creek	51	$2 \times 10^{-4}$							
			Steelhead	21	FS			Columbia	8	$1 \times 10^{-4}$	1 to $3 \times 10^{-4}$	$2 \times 10^{-4}$
								Hood River	25	$3 \times 10^{-4}$		
Klickitat	56	$2 \times 10^{-4}$										
Snake River	93	$2 \times 10^{-4}$										
Clearwater	96	$3 \times 10^{-4}$										
Eulachon	3	WB	Columbia	3	$2 \times 10^{-4}$	$2 \times 10^{-4}$	$2 \times 10^{-4*}$					
			Pacific lamprey	3	$6 \times 10^{-4}$			$6 \times 10^{-4}$	$6 \times 10^{-4*}$			

N= Number of Samples WB = whole body; FS = fillet with skin

\* Basin-wide cancer risks based on one study site



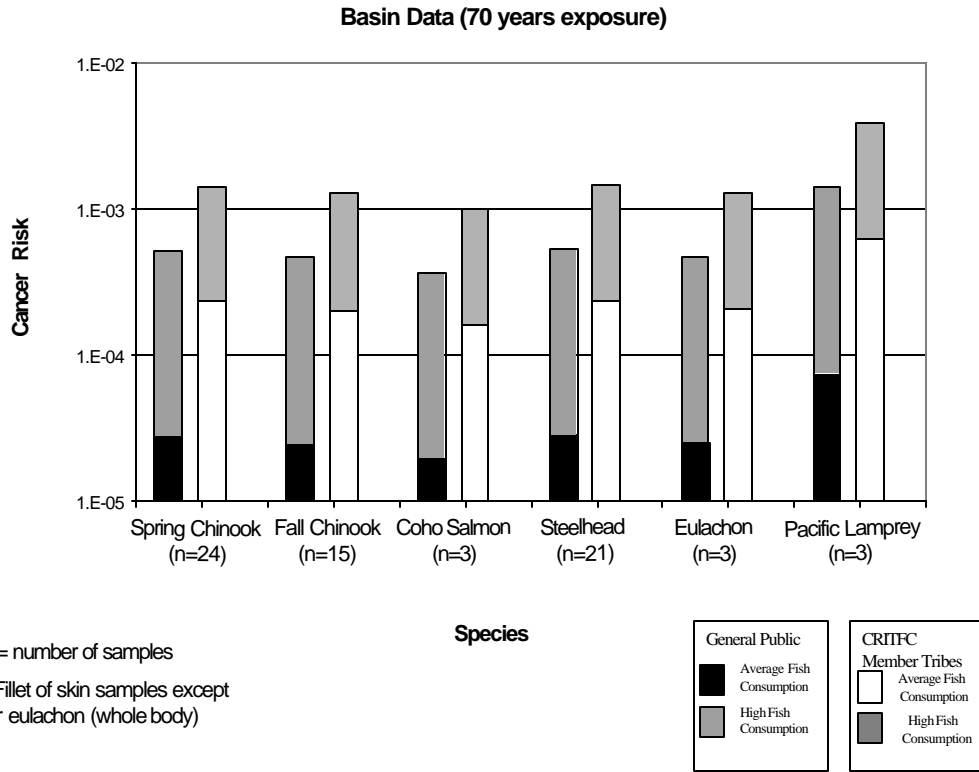


Figure 6-27. Adult cancer risks for anadromous fish species\*. Columbia River Basin-wide average data (70 years exposure).

For coho salmon, fall chinook salmon, spring chinook salmon, steelhead and eulachon, the study site cancer risks were all within a range of  $1 \times 10^{-4}$  to  $3 \times 10^{-4}$  and the basin-wide risks were at approximately  $2 \times 10^{-4}$ . The estimated cancer risk from consumption of Pacific lamprey was  $6 \times 10^{-4}$  (Table 6-16).

For all species, the total cancer risks were highest for CRITFC's member tribal adults at the high fish ingestion rates (389 g/day) followed by the general public, high ingestion rate (142.4 g/day); CRITFC's member tribal adult, average ingestion rate (63.2 g/day); and general public, average ingestion rate (7.5 g/day) (Figure 6-27).

For a more detailed comparison of cancer risks across anadromous fish species for each study site, see Appendix N. In this appendix, estimated cancer risks are shown for all species caught at a study site for a range of ingestion rates.

The chemicals with risks at or greater than  $1 \times 10^{-5}$  for each species for CRITFC's member tribal adults with average fish consumption and 70 years exposure are summarized in Appendix P by species. A review of this appendix shows that:

- For steelhead, spring chinook salmon, and fall chinook salmon, the same three chemical classes (PCBs, dioxins/furans, and one inorganic, arsenic) were responsible for the majority of the risks at or greater than  $1 \times 10^{-5}$ . Fillet with skin and whole body samples of coho had no risks greater than  $10^{-5}$  for dioxins and furans while whole body samples had a  $1 \times 10^{-5}$  risk for DDE. For spring and fall chinook salmon and steelhead, which had dioxins and furans risks at or greater than  $1 \times 10^{-5}$ , three congeners were greater than this risk level - 1,2,3,7,8-PCDD; 2,3,4,7,8-PCDF; and 2,3,7,8-TCDF. For steelhead and all three salmon, Aroclors and PCB congeners 126 and 118 were found at all study sites at or greater than  $1 \times 10^{-5}$ , as was inorganic arsenic.
- Eulachon was sampled at only one site (Columbia River, study site 3). Risks from consumption of the whole body composite sample were at or greater than  $1 \times 10^{-5}$  for two chemicals, arsenic and 1,2,3,7,8-PCDD.
- Pacific lamprey collected at two sites -Willamette Falls (21) and Fifteen Mile Creek (24) - had risks at or greater than  $1 \times 10^{-5}$  for four classes of chemicals: PCBs (Aroclors as well as PCBs 105,114,118,126, and 156); chlorinated dioxins/furans (1,2,3,7,8-PCDD and 2,3,7,8-TCDF); metals (inorganic arsenic); and pesticides (total chlordane, DDT, DDE and hexachlorobenzene).

Tables 6-17 and Figures 6-28 through 6-33 show the percent contribution to total cancer risk for each chemical and/or chemical class using basin-wide cancer risk data (based on fillet of skin data for all species except eulachon which was whole body).

A review of Table 6-17 and Figures 6-28 through 6-33 shows that:

- Arsenic contributes from 33 to 54% of the total cancer risk for salmon and steelhead; 58% for eulachon; and only about 7% for lamprey.
- PCBs (Aroclors and dioxin-like congeners) contribute from 32 to 50% of the total cancer risk for the salmon and steelhead, 77% for lamprey, and only 4% for eulachon. For the salmon, steelhead, and lamprey, Aroclors contribute from 12 to 28% of the total cancer risk. Aroclors were not detected in eulachon. Nine different PCB congeners were detected with PCB 126 contributing the most to total cancer risk (from 6 to 35%) for all species except eulachon. PCB 126 was not detected in eulachon.
- The percent contribution from all pesticides was from about 1 to 9% of the risk.
- The contribution to total cancer risk for chlorinated dioxins and furans was from 9 to 14% for all species except eulachon. For eulachon, the percent contribution to total cancer risk is about 36%.

- Salmon and steelhead look very similar in that arsenic and PCBs were the major contributors to cancer risk followed by dioxin/furans and then pesticides. For Pacific lamprey, PCBs were the major risk contributor at 77% with the rest of the risk split between arsenic, dioxin/furans and pesticides. Most of the risk for eulachon is from arsenic, then dioxins/furans with less than 4% from PCBs and pesticides combined.

**Table 6-17. Percent contribution of contaminant groups to cancer risk for anadromous fish species. Based on Columbia River Basin-wide averages.**

	Spring Chinook Salmon	Coho Salmon	Fall Chinook Salmon	Steelhead	Pacific Lamprey	Eulachon
<i>Tissue Type</i>	<i>FS</i>	<i>FS</i>	<i>FS</i>	<i>FS</i>	<i>FS</i>	<i>WB</i>
<i>Number of samples</i>	24	15	3	21	3	3
<b>Total Metals</b>	<b>50</b>	<b>45</b>	<b>54</b>	<b>33</b>	<b>7</b>	<b>58</b>
Arsenic	50	45	54	33	7	58
<b>Total PCB/Aroclors</b>	<b>32</b>	<b>43</b>	<b>32</b>	<b>50</b>	<b>77</b>	<b>4</b>
PCB 105	1	3	2	1	3	1
PCB 114	1	1	1	1	2	<1
PCB 118	3	ND	4	3	8	2
PCB 123	<1	<1	<1	<1	<1	<1
PCB 126	14	6	10	24	35	ND
PCB 156	1	5	1	2	3	1
PCB 157	<1	ND	<1	<1	1	<1
PCB 169	ND	ND	ND	<1	ND	ND
Other PCBs	<1	<1	<1	<1	<1	<1
Total Aroclors**	12	28	15	19	25	ND
<b>Total Pesticides</b>	<b>4</b>	<b>1</b>	<b>4</b>	<b>4</b>	<b>9</b>	<b>2</b>
Aldrin	ND	ND	ND	ND	ND	ND
Chlordane total	1	<1	1	1	2	ND
DDD	<1	<1	<1	<1	<1	ND
DDE	2	<1	2	2	3	2
DDT	1	<1	<1	<1	2	ND
Heptachlor Epoxide	ND	ND	ND	ND	ND	ND
Hexachlorobenzene	1	ND	1	1	2	ND
<b>Total Dioxins/Furans</b>	<b>14</b>	<b>11</b>	<b>9</b>	<b>14</b>	<b>9</b>	<b>36</b>
2,3,4,6,7,8-HxCDF	<1	ND	ND	<1	<1	1
2,3,4,7,8-PeCDF	4	2	1	6	1	4
2,3,7,8-TCDD	1	1	1	1	1	5
2,3,7,8-TCDF	4	4	5	2	3	5
OCDD	<1	<1	<1	<1	<1	<1
OCDF	<1	<1	<1	<1	ND	<1
1,2,3,7,8-PeCDD	4	3	2	4	2	16
1,2,3,4,7,8-HxCDD	<1	ND	ND	<1	<1	1
Other dioxins	1	1	<1	1	1	5

\* Number in parenthesis is number of samples in basin data \*\* Based on adjusted Aroclor concentration (see Section 5.3.2)  
 ND = not detected

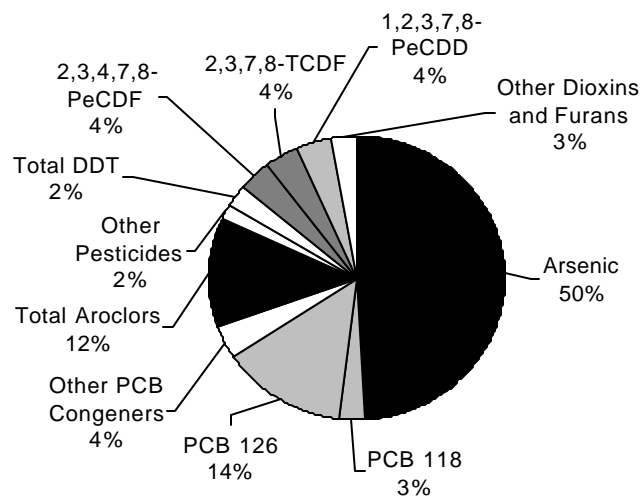


Figure 6-28. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of spring chinook fillet with skin. Number of samples = 8.

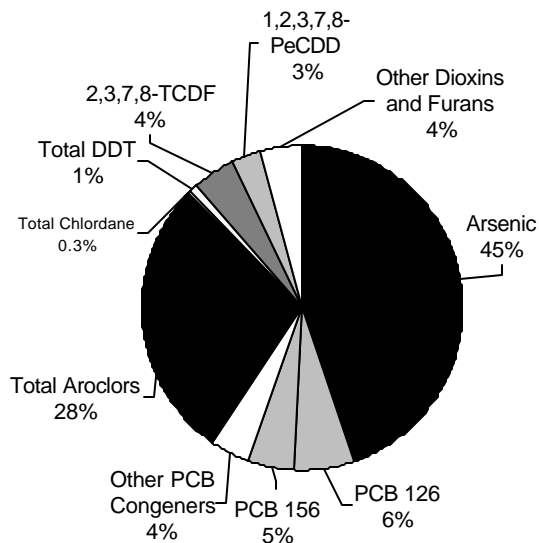


Figure 6-29. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of coho salmon fillet with skin. Number of samples = 3.

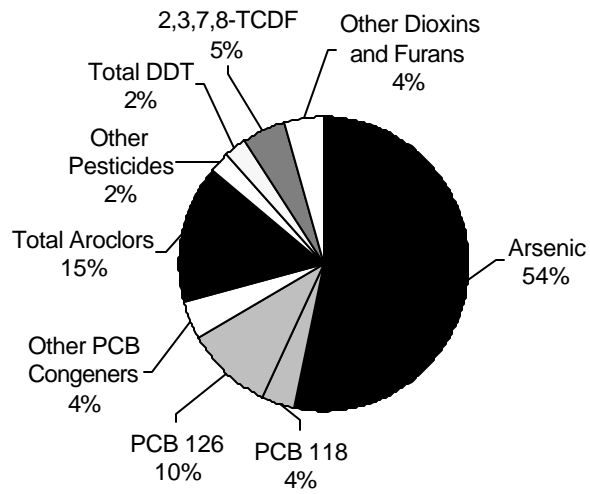


Figure 6-30. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of fall chinook salmon fillet with skin. Number of samples = 15.

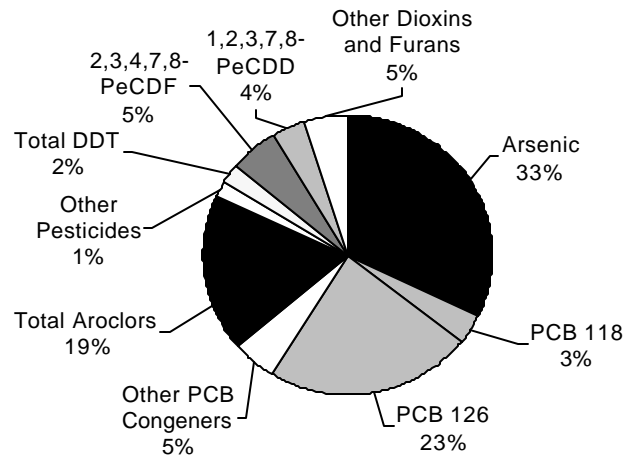


Figure 6-31. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of steelhead fillet with skin. Number of samples = 21.

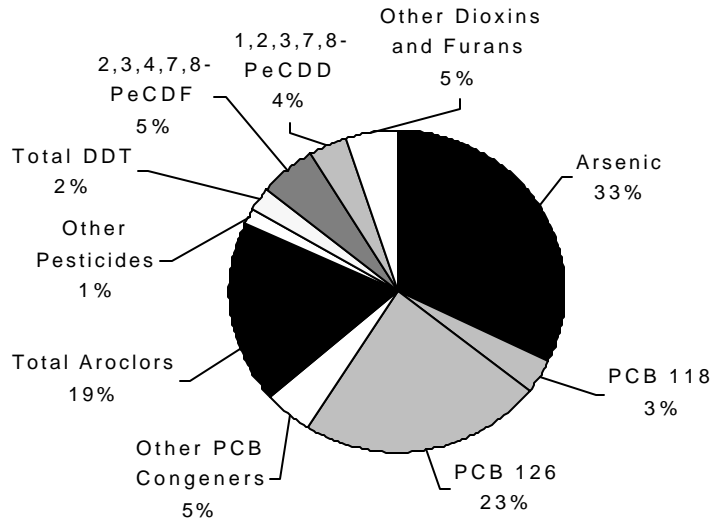


Figure 6-32. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of Pacific lamprey fillet with skin. Number of samples =3.

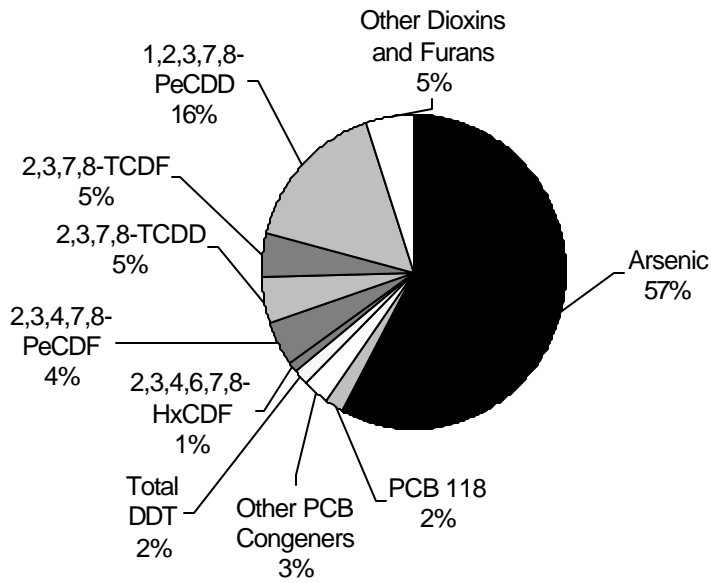


Figure 6-33. Percent contribution of basin-wide average chemical concentrations to cancer risk from consumption of whole body eulachon. Number of samples = 3.

### 6.2.2.3 Comparisons of Cancer Risks Between Anadromous Fish and Resident Fish Species

Table 6-18 shows a summary of the estimated total upper bound cancer risks for the basin and across study sites for all species at the high fish consumption rate for CRITFC's member tribal adults, 70 years exposure. It should be noted that the cancer risk estimates in Table 6-18 were calculated using high fish ingestion rates for CRITFC's member tribal adults, 70 years of exposure, while the results previously discussed for resident fish species in Table 6-14 and for anadromous fish species in Table 6-16 were calculated using average fish ingestion rates for CRITFC's member tribal adults, 70 years exposure. Conclusions from the comparisons in Table 6-18 are limited by the fact that different species were caught at different study sites and that sample numbers and types for each species varied.

Table 6-18 and the study site specific data in the tables in Appendices O and P show that for CRITFC's member tribal adults consuming fish at the high ingestion rate for 70 years:

- The basin-wide risks for *rainbow trout* and five of the anadromous fish (*coho*, *spring*, and *fall chinook salmon*, *steelhead*, and *eulachon*) were all estimated to be  $1 \times 10^{-3}$ . The range in the study site risks for the four species that had multiple study sites sampled was generally small: less than 2 fold for rainbow trout, fall chinook, and spring chinook. Steelhead had a slightly larger range ( $7 \times 10^{-4}$  to  $2 \times 10^{-3}$ ) due primarily to an estimated cancer risk of  $7 \times 10^{-4}$  at the Columbia River (study site 8); the estimated cancer risks for the other 5 study sites were at 1 or  $2 \times 10^{-3}$ .
- The basin-wide risk for *walleye* was  $9 \times 10^{-4}$ . The cancer risk for this one sample was within the range of study site risks for the species discussed in the previous bullet (rainbow trout, eulachon, the three salmon, and steelhead).
- The estimated basin-wide risks for high ingestion by adults in CRITFC's member tribes were greater than  $1 \times 10^{-3}$  among the remaining five species, with mountain whitefish and white sturgeon having the highest estimated basin-wide risks: *largescale sucker* ( $2 \times 10^{-3}$ ); *bridgelip sucker* ( $3 \times 10^{-3}$ ); *lamprey* ( $4 \times 10^{-3}$ ); *sturgeon* ( $6 \times 10^{-3}$ ), and; *whitefish* ( $8 \times 10^{-3}$ ). Three of these species had more than one study site used in the calculation of the basin-wide cancer risks, largescale sucker, sturgeon and whitefish. The range in cancer risks among the study sites sampled for sturgeon was about three-fold; for largescale sucker, about five-fold, and; for whitefish, about twenty-eight fold. The large difference in risk among study sites for whitefish was due to the low estimate of cancer risk of  $7 \times 10^{-4}$  for samples from the Umatilla (study site 101) and the high estimate of cancer risk of  $2 \times 10^{-2}$  at the Hanford Reach of the Columbia River (study site 9U). For sturgeon, no study site risk was less than  $4 \times 10^{-3}$ ; the study site with the highest estimated cancer risk was the Columbia River at study site 9U.

**Table 6-18. Summary of estimated total cancer risks by study site and basin-wide, all species. CRITFC's tribal member adult, high fish consumption, 70 years exposure**

Species	N	Sample type	Range in study site cancer risks	Basin cancer risk
<b>Resident species</b>				
bridgelip sucker	3	WB	$3 \times 10^{-3}$	$3 \times 10^{-3*}$
largescale sucker	19	FS	$8 \times 10^{-4}$ to $4 \times 10^{-3}$	$2 \times 10^{-3}$
mountain whitefish	12	FS	$7 \times 10^{-4}$ to $2 \times 10^{-2}$	$8 \times 10^{-3}$
white sturgeon	16	FW	$4 \times 10^{-3}$ to $1 \times 10^{-2}$	$6 \times 10^{-3}$
walleye	3	FS	$9 \times 10^{-4}$	$9 \times 10^{-4*}$
rainbow trout	7	FS	$1 \times 10^{-3}$ , $1 \times 10^{-3}$	$1 \times 10^{-3}$
<b>Anadromous species</b>				
coho salmon	3	FS	$1 \times 10^{-3}$	$1 \times 10^{-3*}$
fall chinook salmon	15	FS	$9 \times 10^{-4}$ to $1 \times 10^{-3}$	$1 \times 10^{-3}$
spring chinook salmon	24	FS	1 to $2 \times 10^{-3}$	$1 \times 10^{-3}$
steelhead	21	FS	$7 \times 10^{-4}$ to $2 \times 10^{-3}$	$1 \times 10^{-3}$
eulachon	3	WB	$1 \times 10^{-3}$	$1 \times 10^{-3*}$
Pacific lamprey	3	FS	$4 \times 10^{-3}$	$4 \times 10^{-3*}$

WB = whole body; FS = fillet with skin; FW = fillet without skin; N = number of samples

\* Basin-wide cancer risks based on one study site

Figure 6-34 is a summary of the cancer risks estimated to result from consumption of the resident fish and anadromous fish at all four ingestion rates for adults: general public adult, average and high fish consumption; CRITFC's member tribal adult, average and high fish consumption, assuming 70 years exposure. (Note that the Y axis is on a logarithmic scale). Basin-wide fillet with skin data were used for this figure, except for those species that had only whole body samples (bridgelip sucker and eulachon) or fillet without skin samples (sturgeon). The basin-wide cancer risks vary by species, with mountain whitefish having the highest estimated cancer risks and white sturgeon having the second highest among the species sampled. Lamprey, bridgelip sucker and largescale sucker were the next highest followed by the remaining seven species - the three salmon, steelhead, eulachon, rainbow trout, and walleye.



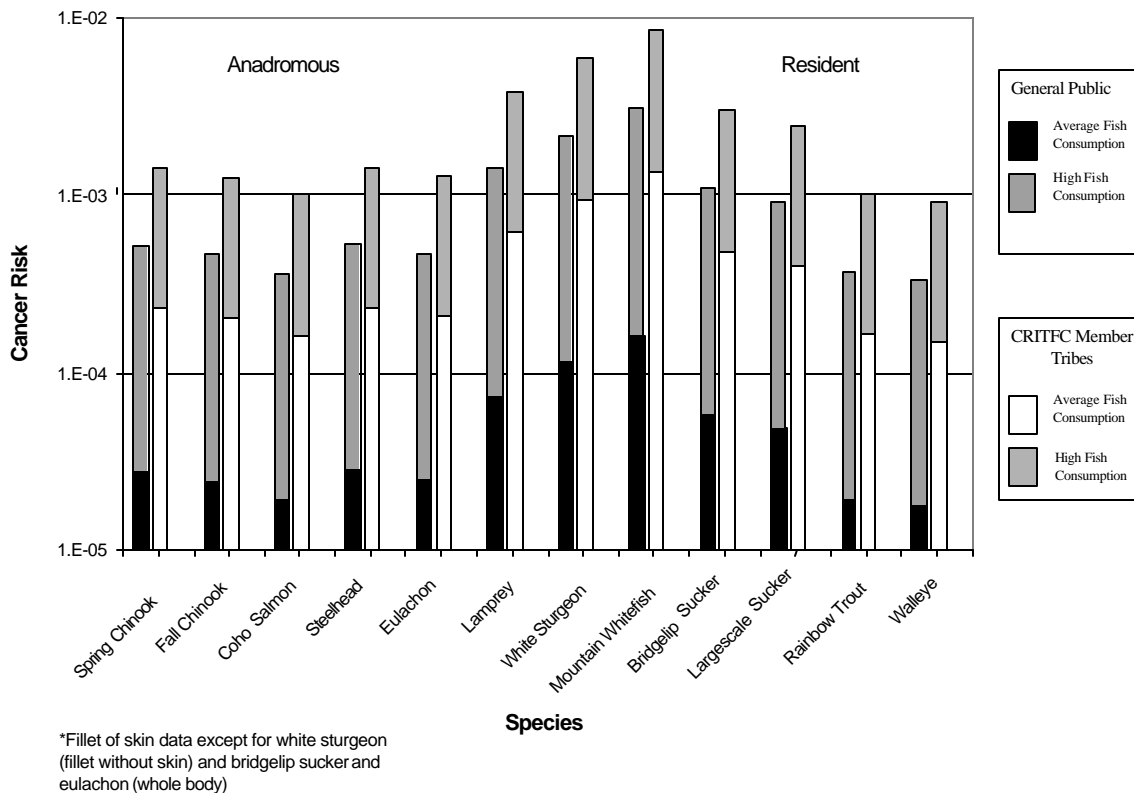


Figure 6-34. Adult estimated total cancer risks across all fish species sampled. Columbia River Basin-wide average data (70 years exposure).

For a more detailed comparison of cancer risks for anadromous fish and resident fish species for each study site, see Appendix N. In this appendix, estimated cancer risks are shown for all species caught at a sampling site using a range of fish ingestion rates.

The percent contribution of the chemicals and chemical classes to total cancer risk were shown in Tables 6-15 (resident fish species) and 6-17 (anadromous fish species) and in Figures 6-21 to 6-26 (resident fish species) and Figures 6-28 thru 6-33 (anadromous fish species). Fillet with skin data were used for these tables and figures except for sturgeon, for which fillet without skin data were used, and eulachon and bridgelip sucker, for which whole body data were used. A comparison of these tables and figures show that:

- Arsenic - For anadromous fish species, arsenic was a major contributor to cancer risk for all of the salmon and steelhead (33 to 54% for steelhead, fall and spring chinook, and

coho salmon), and eulachon (58%), but contributes only 7% to the total cancer risk for lamprey. For resident fish, such a large contribution from arsenic was seen only for walleye (33%) and less so for bridgelip sucker (8%). As discussed in Section 4, it was assumed that 10% of the total arsenic measured in fish was inorganic. The impact of this assumption on the characterization of risk is discussed more in Section 6.2.6.

- PCBs - dioxin-like PCB congeners and Aroclors contribute from 32% to 82% of the total cancer risk for the resident fish; and from 32% to 77% for five of the anadromous fish, the exception being eulachon. For eulachon, dioxin-like PCB congeners/Aroclors contribute only 4% to the total cancer risk. For those 11 fish where dioxin-like PCB congeners/Aroclors were major contributors to risk, Aroclors 1254/1260 and, in general, dioxin-like PCBs 118, 126, and 156, contribute the most to the total dioxin-like PCB congener/Aroclor risk.
- Semi-volatiles - Semi-volatiles, including, PAHs, contribute little to the total risk. The exception was largescale sucker, where the contribution to the basin-wide average was 17% for dibenz(a,h,)anthracene and 8% for benzo(a)pyrene. This was misleading, however, because these two contaminants were found only at one of the six study sites where largescale sucker fillet were sampled, the Snake River at study site 13.
- Pesticides - For resident fish species, pesticides contribute from about 5% (for rainbow trout) to 32% (for bridgelip sucker) of the total cancer risk. For anadromous fish species, the percent contribution from pesticides was lower, from 1% (for coho salmon) to 9% (for lamprey). DDE was by far the major component of the pesticide cancer risk for resident fish species.
- Chlorinated Dioxins/Furans - Chlorinated dioxins/furans contribute from 5% (for largescale sucker) to 36% (for sturgeon) of the total cancer risk for resident fish species. Dioxins/furans contribute 36% to the eulachon cancer risk, but only 9% for lamprey and chinook salmon, 11% for coho, and 14% for steelhead and spring chinook. For resident fish species, 2,3,7,8-TCDF, 1,2,3,7,8-PCDD, and 2,3,7,8-TCDD were the major contributors to the dioxin/furan cancer risk. For the anadromous fish species, 2,3,7,8-TCDF, 1,2,3,7,8-PCDD, and 2,3,4,7,8-PCDF were the major contributors.

### 6.2.3 Summary of Non-Cancer Hazards and Cancer Risks for All Species

Tables 6-19 through 6-22 are a summary of the range in endpoint specific hazard indices and cancer risks across study sites for each species at the four fish ingestion rates used for adults. Hazard indices are shown only for those endpoints that most frequently exceeded a hazard index of 1. These endpoints are for reproduction/development and the central nervous system, immunotoxicity, and liver resulting primarily from exposures greater than the reference dose for methyl mercury, Aroclors, and DDT, DDE and DDD. Cancer risks are those estimated assuming a 70 year exposure duration.

- Hazard indices and cancer risks were lowest for the general public adult at the average ingestion rate and highest for CRITFC's member tribal adults at the high ingestion rate. For the general public with an average fish ingestion (7.5 g/day or about a meal per month), hazard indices were less than 1 and cancer risks are less than  $1 \times 10^{-4}$  except for a few of the more highly contaminated samples of mountain whitefish and white sturgeon (Table 6-19).
- For CRITFC's member tribal adults at the highest fish ingestion rates (389 g/day or about 48 meals per month), hazard indices were greater than 1 for several species at some study sites. Hazard indices (less than or equal to 8 at most study sites) and cancer risks (ranging from  $7 \times 10^{-4}$  to  $2 \times 10^{-3}$ ) were lowest for salmon, steelhead, eulachon and rainbow trout and highest (hazard indices greater than 100 and cancer risks up to  $2 \times 10^{-2}$  at some study sites) for mountain whitefish and white sturgeon (Table 6-22).
- As discussed previously in Section 6.2.1, for the general public, the hazard indices for children at the average fish ingestion rate were about 0.9 those for adults at the average ingestion rate; the hazard indices for children at the high ingestion rate were about 1.3 times those for adults at the high ingestion rate. For CRITFC's member tribes, the hazard indices for children at the average and high ingestion rates were both about 1.9 times those for CRITFC's member tribal adults at the average and high ingestion rates, respectively.

**Table 6-19. Summary of Hazard Indices and Cancer Risks Across Study sites. General Public Adult, average fish consumption (7.5 grams/day or 1 meal per month).**

Species*	N*	Non-cancer endpoints which most frequently exceed a hazard index of one for all species			Cancer Risks (70 years exposure)
		Reproductive/ Developmental And Central Nervous System	Immunotoxicity	Liver	
<b>Resident species</b>					
bridgelip sucker	3	<1	<1	<1	$6 \times 10^{-5}$
largescale sucker	19	<1	<1	<1	2 to $7 \times 10^{-5}$
mountain whitefish	12	<1	<1 to 3	<1	$1 \times 10^{-5}$ to $5 \times 10^{-4}$
white sturgeon	16	<1	<1 to 2	<1	$7 \times 10^{-5}$ to $3 \times 10^{-4}$
walleye	3	<1	<1	<1	$2 \times 10^{-5}$
rainbow trout	7	<1	<1	<1	$2 \times 10^{-5}$ , $2 \times 10^{-5}$
<b>Anadromous species</b>					
coho salmon	3	<1	<1	<1	$2 \times 10^{-5}$
fall chinook	15	<1	<1	<1	2 - $3 \times 10^{-5}$
spring chinook	24	<1	<1	<1	2 - $3 \times 10^{-5}$
steelhead	21	<1	<1	<1	1 to $3 \times 10^{-5}$
eulachon	3	<1	<1	<1	$2 \times 10^{-5}$
Pacific lamprey	3	<1	<1	<1	$7 \times 10^{-5}$

\* N = number of samples. All samples are fillet with skin except sturgeon (fillet without skin) and bridgelip sucker and eulachon (whole body)

**Table 6-20. Summary of Hazard Indices and Cancer Risks Across Study sites. General Public Adult, high fish consumption (142.4 g/day or 19 meals per month).**

Species*	N*	Non-cancer endpoints which most frequently exceed a hazard index of one for all species			Cancer Risks (70 years exposure)
		Reproductive/ Developmental and Central Nervous system	Immunotoxicity	Liver	
<b>Resident species</b>					
bridgelip sucker	3	<1	6	2	1 X 10 <sup>-3</sup>
largescale sucker	19	2 to 7	1 to 8	<1 to 3	3 X 10 <sup>-4</sup> to 1 X 10 <sup>-3</sup>
mountain whitefish	12	<1 to 3	1 to 50	<1 to 4	2 X 10 <sup>-4</sup> to 9 X 10 <sup>-3</sup>
white sturgeon	16	1 to 7	6 to 40	2 to 8	1 to 5 X 10 <sup>-3</sup>
walleye	3	4	1	1	3 X 10 <sup>-4</sup>
rainbow trout	7	1 to 2	1 to 2	<1	4 X 10 <sup>-4</sup> , 4 X 10 <sup>-4</sup>
<b>Anadromous species</b>					
coho salmon	3	2	3	<1	4 X 10 <sup>-4</sup>
fall chinook	15	1 to 2	<1 to 3	<1	3 to 5 X 10 <sup>-4</sup>
spring chinook	24	<1 to 6	1 to 2	<1	4 to 6 X 10 <sup>-4</sup>
steelhead	21	1 to 3	1 to 2	<1	3 to 6 X 10 <sup>-4</sup>
eulachon	3	<1	<1	<1	5 X 10 <sup>-4</sup>
Pacific lamprey	3	<1	9	<1	1 X 10 <sup>-3</sup>

\* N = number of samples; All samples are fillet with skin except sturgeon (fillet without skin) and bridgelip sucker and eulachon (whole body)

**Table 6-21. Summary of Hazard Indices and Cancer Risks Across Study sites. CRITFC's Member Adult, average fish consumption ( 63.2 grams/day or 8 meals per month).**

Species	N	Non-cancer endpoints which most frequently exceed a hazard index of one for all species			Cancer Risks (70 years exposure)
		Reproductive/ Developmental and Central Nervous System	Immunotoxicity	Liver	
<b>Resident species</b>					
bridgelip sucker	3	<1	3	1	5 X 10 <sup>-4</sup>
largescale sucker	19	<1 to 3	<1 to 3	<1 to 1	1 to 6 X 10 <sup>-4</sup>
mountain whitefish	12	<1 to 1	<1 to 22	<1 to 2	1 X 10 <sup>-4</sup> to 4 X 10 <sup>-3</sup>
white sturgeon	16	<1 to 3	3 to 18	<1 to 3	6 X 10 <sup>-4</sup> to 2 X 10 <sup>-3</sup>
walleye	3	2	<1	<1	2 X 10 <sup>-4</sup>
rainbow trout	7	<1	<1	<1	2 X 10 <sup>-4</sup> , 2 X 10 <sup>-4</sup>
<b>Anadromous species</b>					
coho salmon	3	1	1	<1	2 X 10 <sup>-4</sup>
fall chinook	15	<1 to 1	1	<1	1 to 2 X 10 <sup>-4</sup>
spring chinook	24	<1 to 3	<1	<1	2 to 3 X 10 <sup>-4</sup>
steelhead	21	<1 to 1	<1 to 1	<1	1 to 3 X 10 <sup>-4</sup>
eulachon	3	<1	<1	<1	2 X 10 <sup>-4</sup>
Pacific lamprey	3	<1	4	<1	6 X 10 <sup>-4</sup>

N = number of samples. All samples are fillet with skin except sturgeon (fillet without skin).

Bridgelip sucker and eulachon are whole body fish tissue samples.

**Table 6-22. Summary of Hazard Indices and Cancer Risks Across Study sites. CRITFC's Member Adult, high fish consumption (389 grams/day or 48 meal per month)**

Species*	N*	Non-cancer endpoints which most frequently exceed a hazard index of one for all species			Cancer Risks (70 years exposure)
		Reproductive/ Developmental and Central Nervous System	Immunotoxicity	Liver	
<b>Resident species</b>					
bridgelip sucker	3	2	17	6	3 X 10 <sup>-3</sup>
largescale sucker	19	5 to 20	<1 to 21	<1 to 7	8 X 10 <sup>-4</sup> to 4 X 10 <sup>-3</sup>
mountain whitefish	12	<1 to 7	4 to 140	<1 to 11	7 X 10 <sup>-4</sup> to 2 X 10 <sup>-2</sup>
white sturgeon	16	3 to 20	16 to 108	6 to 21	4 X 10 <sup>-3</sup> to 1 X 10 <sup>-2</sup>
walleye	3	10	4	4	9 X 10 <sup>-4</sup>
rainbow trout	7	4 to 5	3 to 4	<1	1 X 10 <sup>-3</sup> , 1 X 10 <sup>-3</sup>
<b>Anadromous species</b>					
coho salmon	3	7	7	<1	1 X 10 <sup>-3</sup>
fall chinook	15	3 to 6	<1 to 8	<1	9 X 10 <sup>-4</sup> to 1 X 10 <sup>-3</sup>
spring chinook	24	<1 to 17	3 to 6	<1	1 to 2 X 10 <sup>-3</sup>
steelhead	21	4 to 8	3 to 6	<1	7 X 10 <sup>-4</sup> to 2 X 10 <sup>-3</sup>
eulachon	3	<1	<1	<1	1 X 10 <sup>-3</sup>
Pacific lamprey	3	<1	24	2	4 X 10 <sup>-3</sup>

N = number of samples. All samples are fillet with skin except sturgeon (fillet without skin).  
Bridgelip sucker and eulachon are whole body fish tissue samples.

#### 6.2.4 Impacts of Sample Type on Risk Characterization

For this study, both whole fish and fillet with skin samples were analyzed for all species except sturgeon, bridgelip sucker, and eulachon. Sturgeon were analyzed as whole fish and fillet without skin (since it is unlikely that sturgeon skin is eaten). For bridgelip sucker and eulachon only whole body samples were collected.

The risk characterization results for all species and sample types are included in the appendices. However, some of the risk characterization results previously discussed in Sections 6.2.1 and 6.2.2 focused on fillet with skin samples (except for those species for which fillet with skin were not collected). To determine the impact that tissue type might have on the risk characterization, the ratio of the estimated hazard indices and cancer risks for whole body to fillet samples were calculated (Table 6-23). These results were calculated for those species that had both fillet and whole body samples analyzed at a given site. For non-cancer effects, whole body to fillet ratios were calculated for the total hazard index as well as for the endpoints of immunotoxicity and reproduction. Table 6-23 also shows the number of whole body to fillet ratios that were greater than 1 compared to the total number of whole body to fillet ratios calculated for that species.

As can be seen from Table 6-23, there does not appear to be a consistent pattern in whole body to fillet ratios for the total hazard indices, the immunotoxicity hazard indices, or cancer risks at a given site for a species. The whole body to fillet ratios ranged from a low of 0.4 to a high of 6.6. Most of the ratios were less than 3. These results are consistent with the results in Section 2 of this report. In Section 2, it was shown that while whole body fish tissue samples tend to be somewhat higher in lipids and lipid soluble contaminants than fillet with skin samples for some species, these differences between whole body and fillet fish samples were not consistent across

species. For reproductive effects, the ratios of the hazard indices for reproductive effects in whole body to fillet samples appear to be less than 1 more frequently than those for the other hazard indices or cancer risks. This may be because the hazard index for reproductive effects is based largely upon the contaminant mercury which is not lipophilic and binds strongly to protein (e.g., muscle tissue). However, any conclusions on the results of whole body to fillet samples are limited by the small sample sizes (usually 3) at each site and by the fact that whole body samples were always from a composite of fish different than those used for the whole body analysis (i.e., fillet and whole body samples are not from the same fish).

**Table 6-23. Comparison of site specific non-cancer hazard indices (for CRITFC's member tribal children) and cancer risks (for CRITFC's member tribal adults) from consuming whole body versus fillet for different fish species.**

Species	Hazard Indices (1)						Cancer Risk (2)	
	Immunotoxicity		Reproductive Effects		Total Hazard Index		Cancer Risk (2)	
	Range in ratios of hazard indices for whole body/fillet across sites		Range in ratios of hazard indices for whole body/fillet across sites		Range of ratios of total hazard indices for whole body/fillet across sites		Range of ratios of cancer risks for whole body/fillet	
	F	F	F	F	F	F	F	F
coho	1.1	(1/1)	0.8	(0/1)	1.1	(1/1)	1	(0/1)
fall chinook	0.9 - 6.6	(3/5)	0.7-1.1	(1/5)	1.0 - 1.6	(3/5)	1 - 2	(2/5)
spring chinook	0.9 - 1.6	(4/8)	0.3 - 1.1	(1/3)	0.6 - 1.6	(4/8)	1 - 2	(3/8)
steelhead	1.1 - 1.4	(6/6)	0.6 - 1.6	(1/6)	0.9 - 1.5	(4/6)	0.5 - 2.0	(2/6)
eulachon	na	na	na	na	na	na	na	na
Pacific lamprey	1	(0/1)	na	na	1.2	(1/1)	1	(0/1)
bridgelip sucker	na	na	na	na	na	na	na	na
largescale sucker	0.6 - 3.3	(3/5)	0.2 - 1.3	(1/6)	0.5 - 2.2	(3/6)	0.7 - 2.5	(3/6)
mountain whitefish	0.4 - 2.1	(2/4)	0.7 - 0.9	(0/3)	0.8 - 1.6	(2/4)	0.5 - 1.4	(1/4)
white sturgeon	0.4- 2.9	(1/3)	0.3 - 3.3	(2/3)	0.4 - 2.7	(1/3)	0.8 - 2.3	(1/3)
walleye	1.8	(1/1)	1	(0/1)	1	(0/1)	1	(1/1)
rainbow trout	1.2 - 1.2	(2/2)	0.7- 1.7	(½)	1.1 - 1.5	(2/2)	1.0 - 1.0	(0/2)

F=Frequency of number of whole body to fillet ratios greater than 1 divided by the total number of whole body to fillet ratios for that species.

na = Not applicable; ratios could not be calculated because chemicals (Aroclors, mercury) were less than detection limits or because fillet data were not available (i.e., for bridgelip sucker and eulachon)

(1) Hazard indices used are those calculated for CRITFC's tribal member children, high fish consumption rate

(2) Cancer risk are those calculated for CRITFC's tribal member adults, 70 years exposure, high fish consumption

### 6.2.5 Risk Characterization Using a Multiple-species Diet

As discussed in Section 4.10, a hypothetical diet consisting of multiple fish species was developed 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 percentage of the hypothetical diet assumed for each fish species and the resulting species specific ingestion rates (assuming a total fish ingestion rate of 63.2 g/day, the average for CRITFC's tribal members adults) were shown previously in Table 4-4.

Table 6-24 shows the resulting cancer risks and total non-cancer hazard indices calculated using this hypothetical diet and the average fish consumption rate (63.2 grams/day) for CRITFC's member tribal adult fish consumers. Cancer risk estimates for individual species were highest for lamprey fillets ( $1.0 \times 10^{-4}$ ) and lowest for walleye fillets ( $4.2 \times 10^{-6}$ ). The total excess cancer risk for consuming the fish used in this example was  $4.0 \times 10^{-4}$ . Total hazard indices for individual species were highest for lamprey and mountain whitefish fillets (0.7) and lowest for eulachon and largescale sucker fillets (0.1). The total hazard index for consuming the fish used in this example was 3.2.

**Table 6-24. Estimate cancer risks and non-cancer health effects for a hypothetical multiple-species diet based upon CRITFC's member average adult fish consumption (CRITFC, 1994)**

Species	Percentage of Hypothetical	Consumption Rate (g/day)	Cancer Risk <sup>a</sup>	Non-cancer Effects <sup>a</sup>
Salmon <sup>b,c,d</sup>	27.7	17.5	$5.8 \times 10^{-5}$	0.6
Rainbow Trout <sup>d</sup>	21.0	13.3	$3.5 \times 10^{-5}$	0.3
Mountain Whitefish <sup>d</sup>	6.8	4.3	$9.3 \times 10^{-5}$	0.7
Eulachon <sup>e</sup>	15.6	9.9	$3.3 \times 10^{-5}$	0.1
Pacific lamprey <sup>d</sup>	16.3	10.3	$1.0 \times 10^{-4}$	0.7
Walleye <sup>d</sup>	2.8	1.8	$4.2 \times 10^{-6}$	0.1
White Sturgeon <sup>f</sup>	7.4	4.7	$7.1 \times 10^{-5}$	0.6
Largescale Sucker <sup>d</sup>	2.3	1.5	$9.3 \times 10^{-6}$	0.1
Totals	100.0	63.2	$4.0 \times 10^{-4}$	3.2

<sup>a</sup>Risk estimates assume fish consumption by a 70 kg CRITFC's tribal member adult at the specified rate 365 days per year for 70 years

<sup>b</sup>Cancer risk estimates for salmon are the average of estimates for spring chinook ( $6.4 \times 10^{-5}$ ), fall chinook ( $5.7 \times 10^{-5}$ ), coho ( $4.5 \times 10^{-5}$ ), and steelhead ( $6.4 \times 10^{-5}$ ).

<sup>c</sup>Noncancer hazard indices for salmon are the average of estimates for spring chinook (0.6), fall chinook (0.5), coho (0.7), and steelhead (0.7).

<sup>d</sup>Risk estimates are based on analysis of uncooked composite samples of fillets with skin.

<sup>e</sup>Risk estimates are based on analysis of uncooked composite samples of whole body fish.

<sup>f</sup>Risk estimates are based on analysis of uncooked composite samples of fillets without skin.

Figure 6-35 shows the total non-cancer hazard indices and Figure 6-36 shows the total cancer risks (70 years exposure) across all species with the results for the multiple-species diet shown for comparison. The results for both general public adult (average and high fish consumption) and CRITFC's member tribal adults (average and high fish consumption) using basin-wide data are included. For all four populations, the hypothetical diet of multiple species based on CRITFC's fish consumption survey was used. The non-cancer hazards and cancer risks for the multiple-species diet were lower than those for the most contaminated species (e.g., sturgeon and whitefish) and higher than those estimated for some of the least contaminated species (e.g., salmon, steelhead, rainbow trout, and eulachon).

These results demonstrate that the non-cancer hazards and cancer risks previously discussed in Sections 6.2.1 and 6.2.2 for individual species may not adequately reflect the cancer risks and non-cancer hazards for CRITFC's member tribes or other individuals from the general public whose diets are composed of a mixture of fish types from the Columbia River Basin.

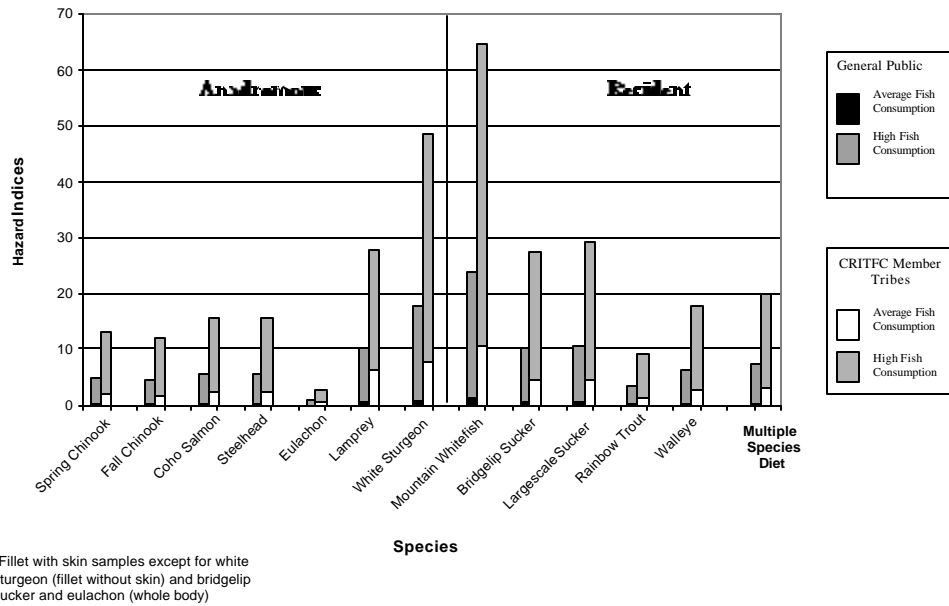


Figure 6-35. Adult total hazard indices for all fish species, with multiple-species diet results. Basin-wide average data.

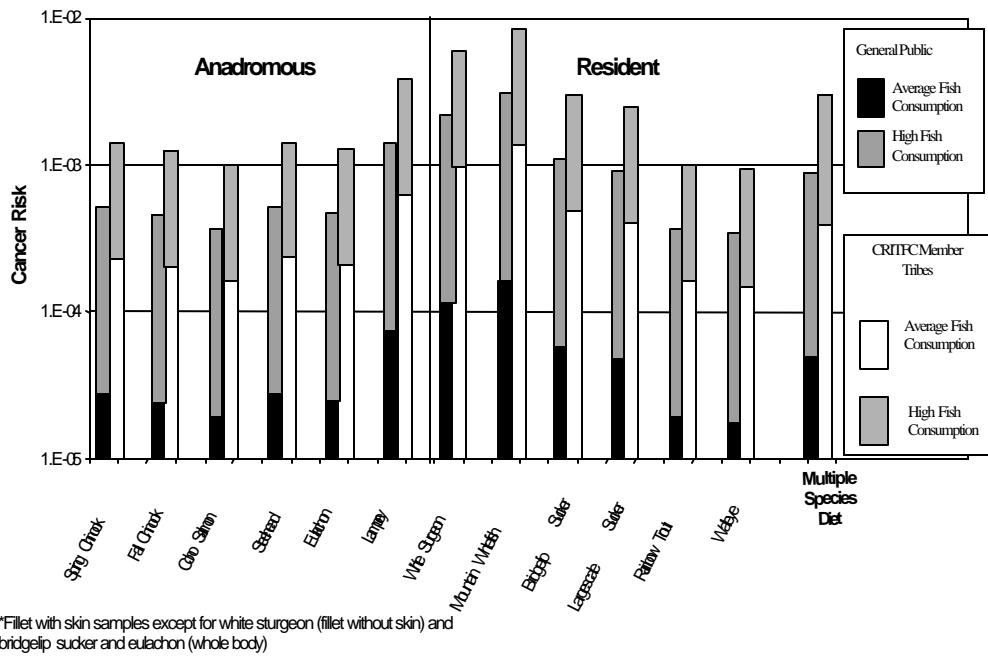


Figure 6-36. Adult cancer risks for all species, with multiple-species diet results. Columbia River Basin-wide average chemical concentration data. 70 years exposure.



## 6.2.6 Risk Characterization Using Different Assumptions for Percent of Inorganic Arsenic

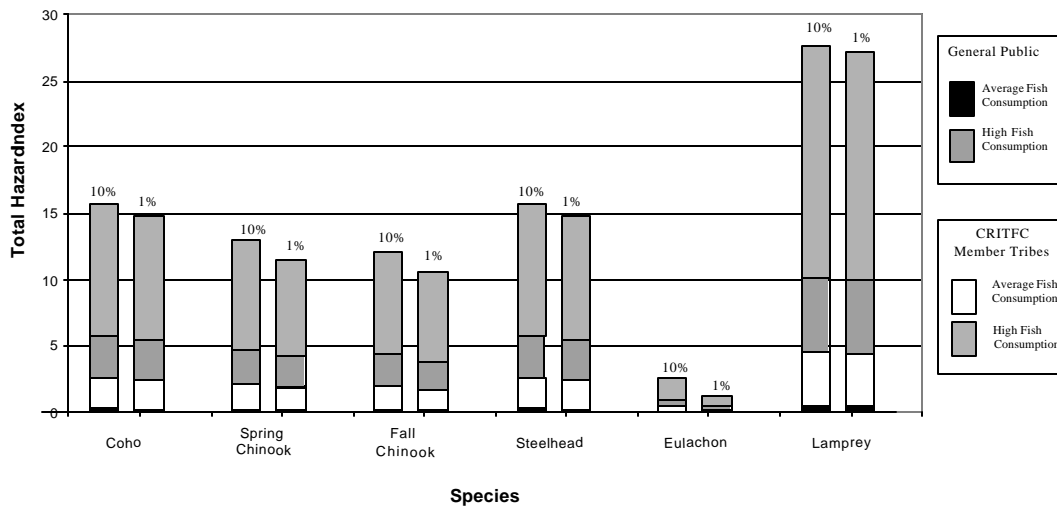
As discussed in Section 5.3.3, total arsenic was measured in fish tissue samples in this study. Because a reference dose and cancer slope factor are available for only inorganic arsenic, an assumption about the percent of inorganic arsenic in fish had to be made to estimate the non-cancer hazards and cancer risks from consuming fish. The non-cancer hazards and cancer risks discussed in Section 6.2.1 and 6.2.2, respectively, assumed that for all fish species (resident fish and anadromous fish) caught in this study, 10% of the total arsenic was inorganic arsenic. The studies used to derive this value of 10% and the rationale for its selection were discussed in Section 5.3.3. The data in Section 5.3.3 also suggests that an alternative assumption for anadromous fish species could be considered - the assumption that 1% of the total arsenic was inorganic. Therefore, the non-cancer hazards and cancer risk were recalculated for anadromous fish species using basin-wide data assuming that 1% of the total arsenic was inorganic. The assumption of 1% inorganic arsenic for anadromous fish species in effect results in a contaminant level for arsenic that one tenth of that assuming that 10% was inorganic arsenic.

Table 6-25 shows the impact of the two different assumption (10% and 1% inorganic) on the estimated total hazard indices for anadromous fish species using basin-wide data. These results are shown for general public and CRITFC's member tribal adults at both the average and high fish consumption rates. As can be seen from this table and from Figure 6-37, assuming that 1% of total arsenic was inorganic rather than 10%, the total hazard indices were reduced by 2% for lamprey, 6% for coho and steelhead, and 11% for spring and fall chinook. However, for eulachon, the assumption of 1% inorganic arsenic reduces the total basin-wide hazard index for this fish species by 56%. The effect of this assumption on risks due to ingestion of eulachon was consistent with the data in Table 6-7 which showed the percent contribution of different contaminants on the basin-wide total hazard indices for anadromous fish species. Arsenic contributed from about 2% to 13% to the total hazard index for salmon, steelhead, and lamprey but about 60% to that for eulachon. Thus, assuming that inorganic arsenic represents 1% rather than 10% of total arsenic had the largest impact on the total non-cancer hazards for eulachon (a 56% reduction in the total hazard index) and less of an impact on the other anadromous fish species.

**Table 6-25. Total hazard indices (HIs) for adults assuming that total arsenic is 1% versus 10% inorganic arsenic. Exposure concentrations used to estimate risks are Columbia River Basin-wide averages of fish tissue samples**

Species	N	Tissue Type	Percent Inorganic Arsenic as Total Arsenic	Percent Decrease In Total HI Assuming 1% Inorganic Arsenic	Total HI			
					Average Fish Consumer		High Fish Consumer	
					general public	CRITFC member tribe	general public	CRITFC member tribe
coho salmon	3	FS	10		0.3	2.5	5.7	15.7
			1	6	0.3	2.4	5.4	14.8
spring chinook	24	FS	10		0.3	2.1	4.8	13.0
			1	11	0.2	1.9	4.2	11.6
fall chinook	15	FS	10		0.2	2.0	4.4	12.0
			1	11	0.2	1.7	3.9	10.7
steelhead	21	FS	10		0.3	2.6	5.7	15.7
			1	6	0.3	2.4	5.4	14.8
eulachon	3	WB	10		0.1	0.4	1.0	2.7
			1	56	0.0	0.2	0.4	1.2
Pacific lamprey	3	FS	10		0.5	4.5	10.1	27.7
			1	2	0.5	4.4	9.9	27.1

N= Number of samples; FS = fillet with skin; WB = whole body  
 Total HI is determined by summing all hazard quotients regardless of health endpoint.



1% - One percent of total arsenic is inorganic arsenic  
 10% - Ten percent of total arsenic is inorganic arsenic  
 \*Fillet with skin samples except for eulachon (whole body)

Figure 6-37. Impact of percent inorganic arsenic on total hazard index. Basin-wide data for anadromous fish species\*.

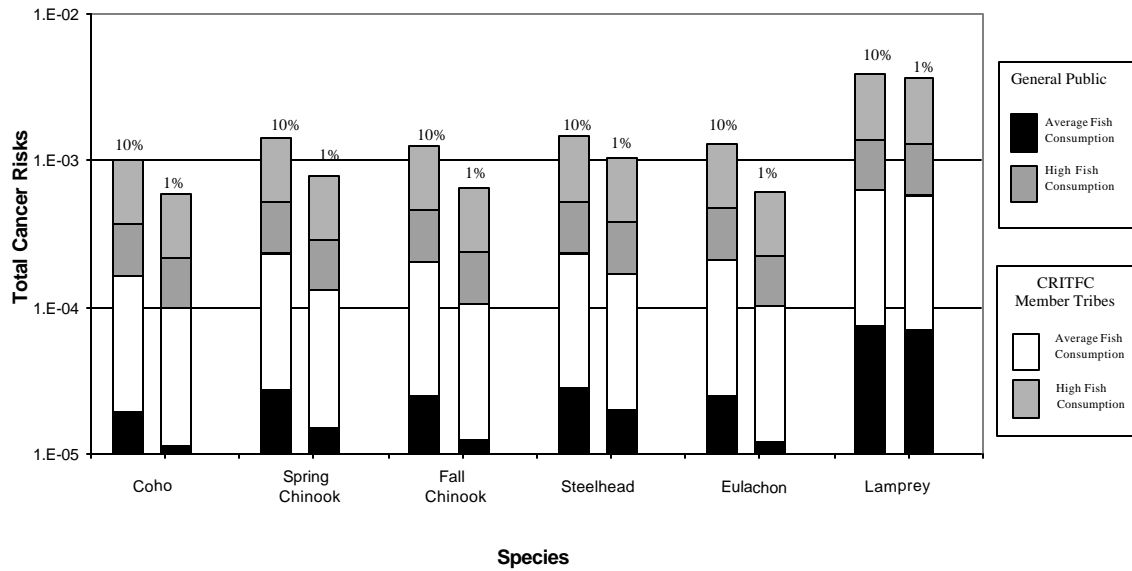
Tables 6-26 and Figure 6-38 show the impact of the two different assumptions (10% and 1% inorganic arsenic as total arsenic) on the estimated total cancer risks for anadromous fish species using basin-wide data. These results are shown for general public and CRITFC's member tribal adults at both the average and high fish consumption rates and 70 years of exposure. Assuming that 1% of total arsenic was inorganic versus 10%, the cancer risks were reduced about 6% for lamprey, 29% for steelhead, and between 40% to 52% for coho, spring chinook, fall chinook and eulachon. These results are consistent with those previously discussed for Table 6-17 (percent contribution of different contaminants on the basin-wide total cancer risk for anadromous fish species) which showed that arsenic was a major contributor to the total cancer risks for all anadromous fish species except Pacific lamprey.

**Table 6-26. Estimated total cancer risks for adults assuming that total arsenic was 1% versus 10% inorganic arsenic 70 years exposure. Exposure concentrations used to estimate risks are Columbia River Basin-wide averages of fish tissue samples.**

Species	N	Tissue Type	Percent Inorganic Arsenic as Total Arsenic	Percent Decrease In Total Cancer Risk Assuming 1% Inorganic Arsenic	Total Cancer Risk			
					Average Fish Consumer		High Fish Consumer	
					general public	CRITFC member tribe	general public	CRITFC member tribe
coho salmon	3	FS	10		1.9E-05	1.6E-04	3.7E-04	1.0E-03
			1	40.4	1.1E-05	9.7E-05	2.2E-04	6.0E-04
spring chinook	24	FS	10		2.8E-05	2.3E-04	5.2E-04	1.4E-03
			1	44.6	1.5E-05	1.3E-04	2.9E-04	7.9E-04
fall chinook	15	FS	10		2.4E-05	2.0E-04	4.6E-04	1.3E-03
			1	48.4	1.2E-05	1.1E-04	2.4E-04	6.5E-04
steelhead	21	FS	10		2.8E-05	2.3E-04	5.3E-04	1.4E-03
			1	29.3	2.0E-05	1.7E-04	3.7E-04	1.0E-03
eulachon	3	WB	10		2.5E-05	2.1E-04	4.7E-04	1.3E-03
			1	52.0	1.2E-05	1.0E-04	2.3E-04	6.2E-04
Pacific lamprey	3	FS	10		7.4E-05	6.2E-04	1.4E-03	3.8E-03
			1	6.1	6.9E-05	5.8E-04	1.3E-03	3.6E-03

N = Number of samples; FS = fillet with skin; WB = whole body

This comparison of the results from using the two different assumptions (1% versus 10%) for inorganic arsenic in fish shows that the reduction on the total non-cancer hazards was less than 12% for all anadromous fish species, except eulachon which had about a 50% reduction. However, the impact was greater on the estimates of cancer risk. With the exception of lamprey for which cancer risks were reduced by only 6%, the reductions in cancer risks for steelhead was about 29% and for the other anadromous fish species ranged from about 40 to 50%.



1% - One percent of total arsenic is inorganic arsenic  
 10% - Ten percent of total arsenic is inorganic arsenic  
 \*Fillet with skin samples except for eulachon (whole body)

Figure 6-38. Impact of percent inorganic arsenic on cancer risks. Basin-wide data for anadromous fish species.

## 7.0 Lead Risk Assessment

Lead health risks are presented separately because lead health risk methods are unique owing to the ubiquitous nature of lead exposures and the reliance on blood lead concentrations to describe lead exposure and toxicity. Lead risks are characterized by predicting blood lead levels with models and guidance developed by EPA available from the following web site:

<http://www.epa.gov/superfund/programs/lead/prods.htm> - software. In this assessment, lead exposure from fish consumption is added to all other likely sources of lead exposure to predict a blood lead level. Both the Integrated Exposure Uptake Biokinetic Model (IEUBK) for children and the EPA Adult Lead Model for the fetus predict blood lead levels from a given set of input parameters. There is no other model for lead exposures except the Adult Lead Model, so it is used for children and fetuses.

In contrast to risk assessments for cancer or non-cancer risks, lead risk assessments typically use central tendency exposure values to predict a central tendency (geometric mean) blood lead level. The predicted geometric mean blood lead level is then used in conjunction with a modeled log-normal distribution to estimate the probability of exceeding a target blood lead level of 10 µg/dl. Blood lead levels are a measure of internal dose that has been related to many adverse health effects (NRC, 1993). The emphasis on blood lead integrates exposure, toxicity and risk, which are more distinct in other types of risk assessment. For other chemicals, risk is described in terms of an external dose (e.g. mg/kg-day).

The IEUBK Model was used to predict blood lead levels in children up to 72 months of age (USEPA, 1994a,b). The EPA Adult Lead Model was used to predict blood lead levels in fetuses (USEPA, 1996b). This section on lead risk assessment is organized into separate discussions of the two lead models. Each of the two lead models was run using both central tendency and high end rates of fish ingestion. Central tendency rates of fish ingestion were used to predict both geometric mean blood lead levels and the probability of exceeding a blood lead level of 10 µg/dl in both children and fetuses. For the high end fish ingestion rates, only the most likely blood level could be predicted; it is not appropriate to predict the probability of exceeding 10 µg/dl associated with high end fish consumption.

### 7.1 Lead Concentrations in Fish

Study sites, collection methods, analytical methods, and quality assurance plans are discussed in Section 1; concentrations of lead in fish are discussed in Section 2. Whole fish had substantially higher lead levels because lead tends to concentrate in the bones and gills (Ay et al., 1999). Note that the maximum in the concentration scale for whole fish is 500 µg/kg and 100 µg/kg for fillets (Table 2-14). The highest individual sample was 1200 µg/kg in a fall chinook salmon taken from Station 14 on the Columbia River. For fish tissue samples with undetected lead concentrations, a value of half the detection limit was used (5 µg/kg) in all risk estimates.

## 7.2 Overview of Lead Risk Assessment Approach

Risk assessment methods for lead differ from other types of risk assessment because they integrate all potential sources of exposure to predict a blood lead level. Lead in the blood reflects all sources of lead exposure, regardless of its origin. Lead risk assessments reflect the widespread distribution of lead in the environment. Common sources of lead in the environment include residual contamination from past uses of lead in gasoline, paint, agricultural chemicals, and industrial sources including lead mining and smelting (NRC, 1993). People are exposed to lead through ingestion of soil and dust, inhalation of lead from the air, and consuming food with background concentrations of lead. Lead can enter drinking water through contamination of surface and groundwater as well as leaching from lead pipes and solder in plumbing systems. All of these sources and exposure pathways are included in the models used to assess lead risks. The IEUBK model is used to simulate lead exposures from air, water, diet, soil, and house dust. The Adult Lead Model accounts for the same sources of lead exposure by using a baseline blood lead level derived from the National Health and Nutrition Examination Survey (USEPA, 1996b).

Risk assessment methodologies for substances other than lead utilize a combination of central tendency and high end exposure values to estimate an aggregate reasonable maximum exposure scenario. A point value for risk derived using a reasonable maximum exposure scenario is accepted as being protective of public health. Public health protection using lead risk assessment methodology derives from a limit on the acceptable predicted blood lead values. An acceptable risk for lead exposure typically equates to a predicted probability of no more than 5% greater than the 10  $\mu\text{g}/\text{dl}$  level (USEPA, 1998b)

Risk, expressed as predicted blood lead levels, was calculated in two ways for children and fetuses. The first, and more typical, method used median fish ingestion rates to predict: 1) a geometric mean blood lead level and 2) the corresponding risk of exceeding a blood lead level of 10  $\mu\text{g}/\text{dl}$ . The probability of exceeding 10  $\mu\text{g}/\text{dl}$  was calculated with a log-normal risk model based on the model's output (the geometric mean blood lead level) and an assumed geometric standard deviation. In the second method, high-end fish ingestion rates were used to predict blood lead levels for children or mothers who consume large amounts of fish. Because the resultant high-end fish ingestion prediction does not represent a geometric mean blood lead level, the geometric standard deviation could not be applied to predict the probability of exceeding 10  $\mu\text{g}/\text{dl}$ . Predicted blood lead levels resulting from high-end fish consumption scenarios represent the most likely blood lead levels associated with high-end consumption rates.

The adverse health effects of lead have been related to blood lead concentrations in units of micrograms of lead per deciliter of whole blood ( $\mu\text{g}/\text{dl}$ ). As a result, blood lead levels have evolved as measures of exposure, risk, and toxicity. Since 1991, the national level of concern for young children and fetuses has been 10  $\mu\text{g}/\text{dl}$  (CDC, 1991). An analogous level has not been defined for other groups, but children and the developing fetus are accepted as being especially vulnerable to lead because lead interferes with the development of the central nervous system (NRC, 1993). Lead risks were evaluated by comparing predicted blood lead levels to the 10  $\mu\text{g}/\text{dl}$  standard and by determining the expected percentage to exceed the 10  $\mu\text{g}/\text{dl}$  criterion.

Adverse health effects observed at a blood lead level of 10 µg/dl are sub-clinical, meaning that, these effects cannot be diagnosed in an individual. The adverse health effects include cognitive deficits in IQ and learning, based on numerous scientific studies involving comparisons of large groups of children to control for confounding factors and account for the natural variability in cognitive function (NRC, 1993; USDHHS, 1999; CDC, 1991). The studies have incorporated both cross-sectional and longitudinal designs. The importance of primary prevention of lead exposure has been highlighted by recent studies suggesting adverse health effects at blood lead levels less than 10 µg/dl and the failure of chelation treatment to prevent cognitive impairments in treated children (Lanphear et al., 2000; Rogan et al., 2001; Rosen and Mushak, 2001).

Children are the population of greatest concern for lead exposure. Blood lead levels tend to peak in children as they become more mobile and begin to explore their surroundings. Blood lead levels normally peak at approximately 30 months of age when children are especially vulnerable to neuro-behavioral deficits (Rodier, 1995;Goldstein, 1990). The adverse effects of low-level lead poisoning can result from relatively short-term exposures on the order of months, as opposed to periods of years or longer for other chemicals. The fetus is vulnerable to the same developmental and neuro-behavioral effects as children. Although lead is harmful to fetuses, children are a greater concern because they generally have higher exposures than fetuses. Fetal exposures are lower because exposures to mothers are typically lower than exposures to children. These and other health effects are described in further detail in Appendix C (Toxicity Profiles).

### **7.3 Method for Predicting Risks to Children**

In contrast to risk assessment methodologies for predicting cancer or non-cancer risks, the lead models rely on central tendency exposure values to predict a central tendency (geometric mean) blood lead level. The predicted geometric mean blood lead level is then used in conjunction with an assumed geometric standard deviation to estimate the probability of exceeding a target blood lead level of 10 µg/dl established by the Centers for Disease Control (CDC, 1991). In this way, central tendency exposure estimates are used to estimate upper percentile blood lead levels. An example graph of an IEUBK Model run depicting the geometric mean and percent greater than 10 µg/dl is shown in Figure 7-1. In the IEUBK model, a geometric mean blood lead level of 4.6 µg/dl corresponds to a 5% chance of exceeding 10 µg/dl using the default geometric standard deviation of 1.6 (USEPA, 1994b). Although lead risk assessment methods differ from that employed for other chemicals, the goal of protecting highly exposed individuals remains the same.

The geometric standard deviation accounts for the variation in blood lead observed in children exposed to similar environmental concentrations of lead. The variation in observed blood lead levels is attributed to differences in the children (behavior and metabolism); not the environment. Because the geometric standard deviation accounts for behaviors that determine exposure levels to lead, applying the geometric standard deviation to high contact rate behaviors, including fish ingestion, would over-estimate the variability and over-predict the probability of exceeding 10 µg/dl.

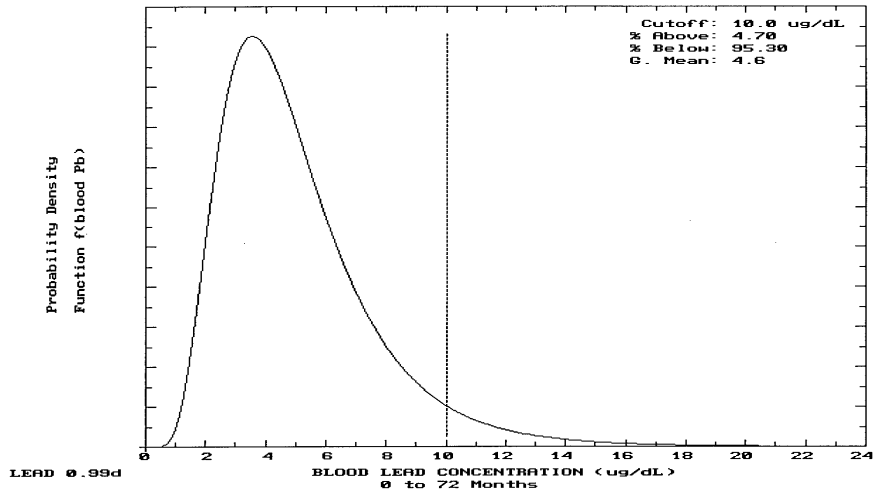


Figure 7-1. Sample IEUBK Model for Lead Output Graph.

Running the IEUBK Model with high-end fish consumption rates predicts the most likely blood lead levels for people eating large amounts of fish, although, the result does not correspond to the geometric mean of a population consuming different amounts of fish. Blood lead predictions for highly exposed individuals facilitate comparison of lead risks to risks from other chemicals, but results from high-end exposure inputs preclude application of the geometric standard deviation to calculate risks of exceeding a 10  $\mu\text{g}/\text{dL}$  blood lead level. Risks to highly exposed individuals are typically characterized by the 95<sup>th</sup> percentile of the blood lead distribution centered around the predicted geometric mean blood lead rather than using the high-end fish ingestion values.

The IEUBK Model was run with all exposure parameters set to default levels with the addition of dietary lead intake attributable to lead in fish tissue for the full range of lead concentrations observed. Default exposure parameters are based on national average levels of lead in air, water food, soil, and dirt (Table 7-1) and described in detail in EPA guidance (USEPA, 1994b).



<b>Input Parameter</b>	<b>Value</b>
Soil lead concentration	200,000 µg/kg
House dust lead concentration (proportion of soil in dust = 0.7)	140,000 µg/kg
Combined soil and dust ingestion rate by age:	
0-11 months	85 mg/day
12-23 months	135 mg/day
24-35 months	135 mg/day
36-47 months	135 mg/day
48-59 months	100 mg/day
60-71 months	90 mg/day
Lead concentration in Air	0.10 : g/cubic meter
Lead concentration in drinking water	4 : g/liter

The default concentrations of lead in soil and house dust are representative of average, national conditions. The default concentrations for lead in soil and house dust are 200,000 µg/kg and 140,000 µg/kg respectively (USEPA, 1994b). These values are appropriate for urban areas and are likely to exceed the expected concentrations in rural areas surrounding the Columbia River because lead levels increase with urbanization. A recent survey of 50 homes from small, rural towns in Northern Idaho found soil lead concentrations less than 100,000 µg/kg (Spalinger et al., 2000). These concentrations would not account for severe lead paint contamination. Lack of data on specific soil and house dust concentrations remains a large source of uncertainty in this evaluation because soil and dust in the home account for a large proportion of lead exposure in young children (Manton et al., 2000) (Lanphear et al., 1998).

The IEUBK model has the capability to simulate exposures to locally grown vegetables, game, and fish. The IEUBK default values for soil, house dust, air, diet, and water were used in conjunction with an age-specific median fish ingestion rate of 16.2 g/day based on the fish consumption survey of CRITFC's member tribes (CRITFC, 1994). Fish ingestion was specified as the percentage of meat (Table 7-2) consisting of locally caught fish and the lead concentrations in the fish. There are other ways to simulate fish ingestion in the IEUBK Model (e.g. by specifying dietary lead intakes as µg/day), but it was preferred to specify fish ingestion as a percentage of meat to preserve the caloric and protein intake assumptions of the model. This approach substitutes fish for other protein sources rather than adding fish to the default diet. This approach conforms with IEUBK body weight and biokinetic assumptions and is described in EPA guidance (USEPA, 1994b).

<b>Age Range (months)</b>	<b>Meat Consumption grams/day</b>
12-24	87
25-36	96
37-48	102
49-60	107
61-72	112
Average	101

The CRITFC study examined Columbia River fish consumption in young children as surveyed by their parents. This study was selected as the most relevant study to assess the Columbia River lead hazard for all children because it is specific to the place, CRITFC’s member tribes, and the age range specified by the IEUBK (CRITFC, 1994). The tribal ingestion rates are likely to overestimate fish consumption for non-tribal members. Because the CRITFC study presents consumption rates for children up to 72 months of age, the IEUBK Model was run for the same age range.

To facilitate comparisons between risks from lead and other chemicals presented in Section 6, the ingestion rates used for other chemicals are summarized in Table 7-3. Fish ingestion rates used to estimate risks from chemicals other than lead are based on mean and 99<sup>th</sup> percentiles of both the CRITFC survey and national data for the general public described in Section 4 of this report.

The distribution of child fish consumption rates from the CRITFC study is statistically skewed because it included individuals with very high fish consumption rates relative to others. For skewed data, the arithmetic mean is not an appropriate measure of central tendency because it is highly influenced by the individuals with large fish consumption rates. The median (50<sup>th</sup> percentile) is a preferred central tendency measure of skewed data because it is less sensitive to extreme values. The fish consumption data for CRITFC’s member tribes (CRITFC, 1994) were re-analyzed to omit children who did not consume fish from the data set (Kissinger and Beck, 2000). The re-analysis calculated a median consumption rate occurred between 13 and 16.2 g/day, the 39<sup>th</sup> and 65<sup>th</sup> percentiles, respectively (see Table 7-4). Rather than interpolate a median value of 14.4 g/day between the 39<sup>th</sup> and 65<sup>th</sup> percentiles, the higher value was selected as a protective central tendency consumption rate.

**Table 7-3. Fish Ingestion Rates (grams/day) Used to Assess Risk for Lead and other Chemicals**

Target Population				Non-lead		Non-lead	
Assessment	Lead		Native American		General Public		
Population	Native American						
Exposure Level	Central	High End	Central	High End	Central	High	
	Mother and Fetus		Adult		Adult		
Ingestion Rate	39.2	389	63.2	389	7.5	142.4	
Basis	50 <sup>th</sup> CRITFC	99 <sup>th</sup> CRITFC	Mean CRITFC	99 <sup>th</sup> CRITFC	Mean EPA	99 <sup>th</sup>	
Age Range	Children < 72 Months		Children < 72 Months		Children < 15 years		
Ingestion Rate	16	101	24.8	162	2.83	77.95	
Basis	50 <sup>th</sup> CRITFC	IEUBK MAX*	Mean CRITFC	99 <sup>th</sup> CRITFC	Mean	99 <sup>th</sup>	

\* A fish ingestion rate of 101 g/day assumes that locally caught fish comprise 100% of all dietary protein sources and represents an upper constraint of the IEUBK Lead Model for Children

**Table 7-4. Percentages of Child Fish Consumption Rates for Consumers of Fish From (Kissinger and Beck, 2000) analysis of (CRITFC, 1994)**

Grams/day	Cumulative Percent	Grams/day	Cumulative Percent	Grams/day	Cumulative Percent
0.4	1%	8.1	33%	32.4	84%
0.8	1%	9.7	35%	48.6	89%
1.6	5%	12.2	38%	64.8	93%
2.4	5%	13.0	39%	72.9	95%
3.2	9%	16.2	65%	81.0	97%
4.1	14%	19.4	66%	97.2	98%
4.9	16%	20.3	67%	162.0	100%
6.5	18%	24.3	70%		

### 7.4 Risk Characterization for Children

Predicted blood lead levels spanning the full range of observed fish tissue concentrations are shown in Figure 7-2. Predicted geometric mean blood lead levels are plotted on the left axis with a solid line. The corresponding probabilities of exceeding 10 µg/dl are shown as percentages on the right axis with a dashed line. Each of the 11 pairs of points represents a separate IEUBK Model run at successively increasing concentrations of lead in fish. These results indicate that for fish containing lead up to 500 µg/kg, the probability of achieving a blood lead level greater than 10 µg/dl is no more than 5% and the predicted geometric mean blood lead level is 4.6 µg/dl. For comparison, only the average concentration of whole body eulachon had a lead concentration of 500 µg/kg. The next highest whole fish species is fall chinook, with an average lead concentration of 220 µg/kg. Average lead concentrations in all other whole fish and fillet samples occur well below 500 µg/kg and concentrations in fillets averaged 200 µg/kg (Table 2-14).

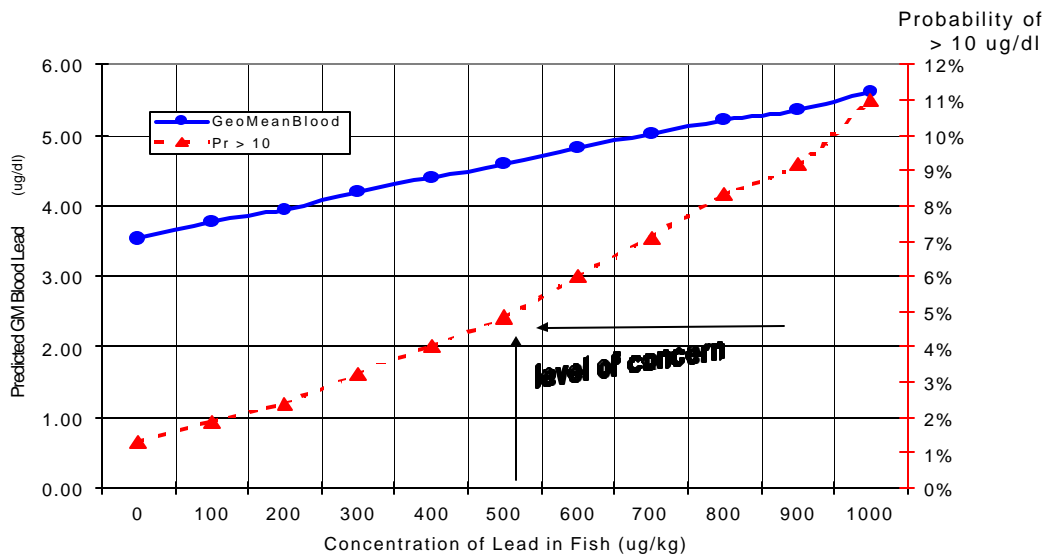


Figure 7-2. Predicted blood lead levels for children who consume of fish collected from the Columbia River Basin assuming fish is 16% of dietary meat.

To explore the effect of an extremely high fish consumption rate in children, the IEUBK Model was run assuming that fish replaced 100% meat in the diet (101 g/day) (Figure 7-3). The IEUBK Model was run repeatedly to determine the fish tissue concentration associated with a predicted blood lead level of 10 µg/dl. A lead concentration of 500 µg/kg in fish tissue corresponded to a predicted blood lead concentration of 10 µg/dl. This is the same concentration associated with a 5% risk of exceeding 10 µg/dl under the 16.2 g/day fish consumption scenario described in the previous paragraph.

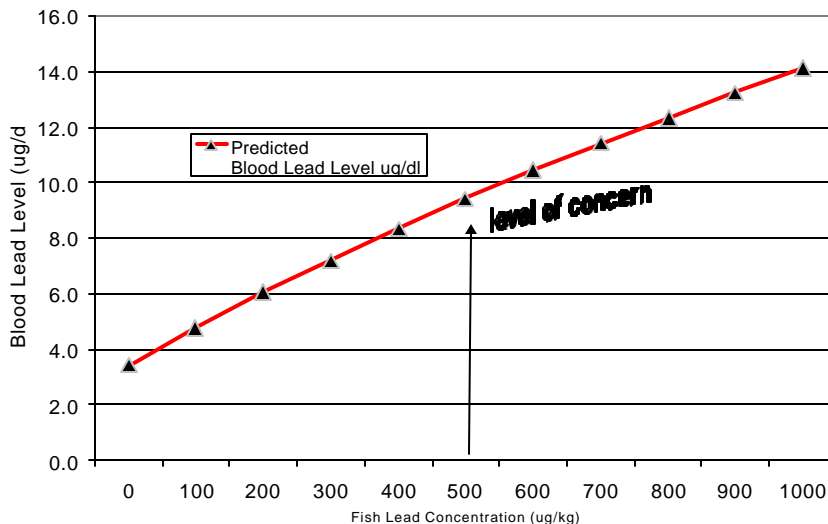


Figure 7-3. Predicted blood lead levels for children (0-72 months) who consume 101 g/day of fish collected from the Columbia River Basin, 1996-1998.

## 7.5 Uncertainties in risk estimates for Children

Lead risk assessment methods are unique because they use cumulative exposures to predict blood lead levels in contrast to methods used for other chemicals which generally limit evaluation of exposures to discreet sources. Because lead risks are cumulative, uncertainties are compounded by the many sources of exposure in addition to uncertainties arising from fish consumption. In children, lead exposure occurs primarily from lead in soil and house dust rather than from typical dietary sources (Manton et al., 2000). Sources of lead exposure common to children and fetuses include industrial or agricultural sources, occupational exposures, and environmental lead originating from gasoline or leaded paint. Occupational exposures can track contaminants from the workplace into the home, potentially spreading exposure among children and adults in a household (Fenske et al., 2000). A major source of uncertainty in this risk assessment may be attributable to sources of lead other than Columbia River fish. The magnitude of lead exposure from fish consumption varies with selection of fish parts eaten (e.g. whole versus fillet), species of fish, and the study site of the fish relative to sources of lead contamination.

The IEUBK model is normally used to simulate blood lead levels for children up to 84 months of age. However, because the fish consumption data from the CRITFC study were reported for children up to 72 months of age, IEUBK evaluation was limited to 72 months. A 72-month

model run predicts higher blood lead concentrations than an 84-month model run because blood lead levels peak during the first 36 months. In the absence of data to estimate specific, concurrent residential exposures, the default concentrations of lead in soil and house dust represent a large source of uncertainty in the IEUBK evaluation because these sources are expected to account for most of the lead exposure to young children. However, the default soil and dust concentrations are unlikely to underestimate average levels of lead in the homes.

## 7.6 Method for Predicting Risks to Fetuses

The Adult Lead Model begins with a baseline blood lead level for adult women and then predicts an incremental increase in blood lead levels associated with an increase in exposure that is not included in the baseline blood lead levels (USEPA, 1996b and USEPA, 1999a). In the Adult Lead Model, fetal blood lead levels are set equal to 90% of the mother's blood lead level. If the baseline blood lead reflects the modeled incremental exposure, then the exposure is counted twice and the modeled blood lead level would be too high. In this study, the Adult Lead Model was used to evaluate fish ingestion as the source of incremental exposure greater than the baseline blood lead level.

The assumptions used in this approach include:

- 1) Lead exposures from all sources except consuming fish from the Columbia River are captured in the baseline blood lead level, based on high end estimates from national blood lead surveys, and
- 2) incremental ingestion of fish is not included in the baseline blood lead level.

Selection of a high baseline blood lead level minimized the possibility of underestimating risk. The lead ingested from fish is converted to a blood lead level by using a constant ratio of an increase in blood lead concentration associated with a mass of absorbed lead. This ratio is the Biokinetic Slope Factor (BKSF). The baseline blood lead level, the blood level in the absence of lead exposure via Columbia River fish ingestion, is critical to this calculation. A complete listing of all the Adult Lead Model input values is included in Table 7.5.

The equations used in the Adult Lead Model are (USEPA 1999b):

*Equation 7-1*

*Adult Blood Lead Level = Baseline Blood Lead Level + Increase in Blood Lead*

*Equation 7-2*

*Increase in Blood Lead =*

*[(BKSF) \* Fish Ingestion Rate \* Fish Concentration \* Absorbed Fraction for Fish]*

*Equation 7-3*

*Fetal Blood Lead = Adult Blood \* 0.9*

*Equation 7-4*

Probability that Fetal Blood Lead is greater or equal to 10 µg/dl using the z-value where:  

$$z = \ln(10) - \ln(\text{Fetal Blood Lead}) / \ln(\text{Geometric Standard Deviation})$$

Analysis of the lead hazard associated with adult consumption of Columbia River fish was conducted using the formula:

$$\text{Equation 7-5 } PbB_{adult, central} = PbB_{adult,0} + BKSF * (PBF * IR_F * AF_F * EF_F) / AT$$

**Table 7-5. Input Parameters Used for the EPA Adult Lead Model**

Variable	Description	Value Used
PbB <sub>adult,0</sub>	Adult blood lead concentration in the absence of other lead exposure.	Central 1.7 µg/dl High End 2.2 µg/dl
BKSF	Biokinetic slope factor relating the (quasi-steady state) increase in blood lead concent	
PbF	Fish lead concentration	full range of values: 0-1000 µg/kg
IR <sub>F</sub>	Intake rate of fish in g/day median of CRITFC Adult Consumption	39.2 g/day
AF <sub>F</sub>	Absolute gastrointestinal absorption factor for ingested lead fish (dimensionless)	0.10
EF <sub>F</sub>	Exposure frequency for ingestion of fish (days of exposure during the averaging period); may be taken as days per year in continuing long term exposures.	365 days per year
AT	Averaging time, the total period during which exposure may occur	365 days per year

Because study site-specific baseline blood lead levels and geometric standard deviations are not available for consumers of Columbia River fish, the Adult Lead Model was run using both central tendency and high-end estimates of the baseline blood lead level and the geometric standard deviation described in (USEPA, 1996b). The larger baseline blood lead level increased the predicted blood lead levels. An increase in the Geometric Standard Deviation increased the probability of exceeding 10 µg/dl. All input parameters are listed in Table 7.6.

**Table 7-6. Adult Lead Model Baseline Blood Lead and Geometric Standard Deviations**

Input Parameter	Baseline Blood Lead Level	Geometric Standard Deviation
Central Values	1.7 µg/dl	1.8
High End Values	2.2 µg/dl	2.1

Fish ingestion rates for adult consumers of Columbia River fish are based on the median ingestion rate of 39.2 g/day interpolated from Table 10 of the 1994 CRITFC consumption survey (CRITFC, 1994). Consumption rates were reported as 38.9 g/day and 40.5 g/day for the 49<sup>th</sup> and 53<sup>rd</sup> percentiles respectively (CRITFC, 1994). For comparison, EPA provides a mean estimate of national per capita fish consumption of 7.5 g/day (USEPA, 2000b). The Model was also run using the 99<sup>th</sup> percentile ingestion rate from the CRITFC survey (389 g/day) to facilitate comparison with the risks from chemicals other than lead (Table 7.1).

## 7.7 Risk Characterization for Fetuses

The Adult Lead Model was used to evaluate potential lead risks to the fetus following maternal consumption of Columbia River fish. Predicted fetal geometric mean blood lead levels and associated probabilities of exceeding the 10  $\mu\text{g}/\text{dl}$  for a range of lead levels in fish are summarized in Figures 7-4 and 7-5. Figure 7-4 shows results using the maximum recommended exposure parameters for the baseline blood lead level of 2.2  $\mu\text{g}/\text{dl}$  and geometric standard deviation of 2.1 (USEPA, 1996b). Figure 7-5 is identical to Figure 7-4, but uses central tendency estimates of baseline blood lead level of 1.7  $\mu\text{g}/\text{dl}$  and geometric standard deviation of 1.8. Although, the predicted risks of exceeding 10  $\mu\text{g}/\text{dl}$  are substantially higher in Figure 7-4, the fish concentration associated with a 5% risk of exceeding 10  $\mu\text{g}/\text{dl}$  is 700  $\mu\text{g}/\text{kg}$ . Average fish concentrations in whole fish and fillets were 0.12 and 0.02 respectively. The highest lead concentrations were found in whole-body samples of eulachon with an average fish tissue concentration of 500  $\mu\text{g}/\text{kg}$  lead. For the fetus of an adult consuming 39.2 grams of whole fish per day (129  $\mu\text{g}/\text{kg}$ ), the Adult Lead Model predicts that fetal blood lead levels will exceed 10  $\mu\text{g}/\text{dl}$  less than 2% of the time using the high end values for baseline blood lead level and geometric standard deviation. Using high end values for baseline blood lead level and geometric standard deviation with the 389 g/day ingestion rate results in a predicted fetal blood lead level at a fish concentration of 600  $\mu\text{g}/\text{kg}$ .

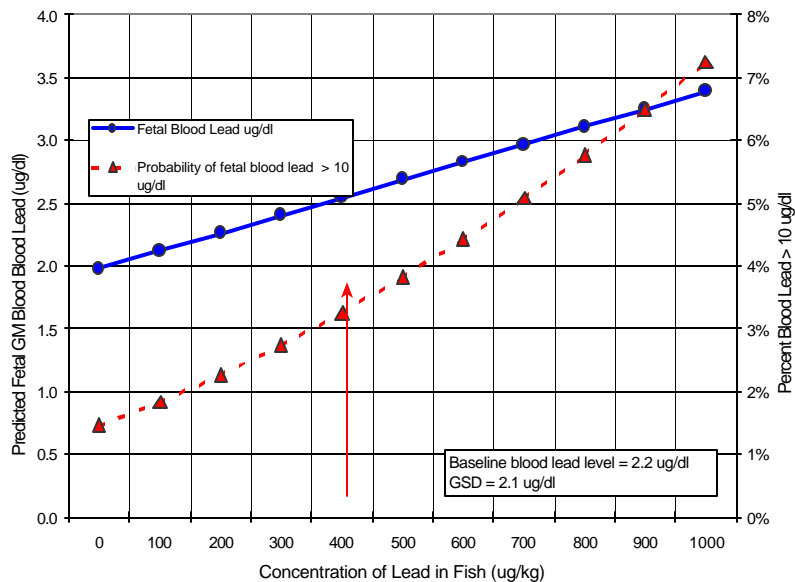


Figure 7-4. Predicted fetal blood lead levels with maternal fish ingestion rate of 39.2 g/day with baseline blood lead level at 2.2  $\mu\text{g}/\text{dl}$  and GSD = 2.1  $\mu\text{g}/\text{dl}$ .

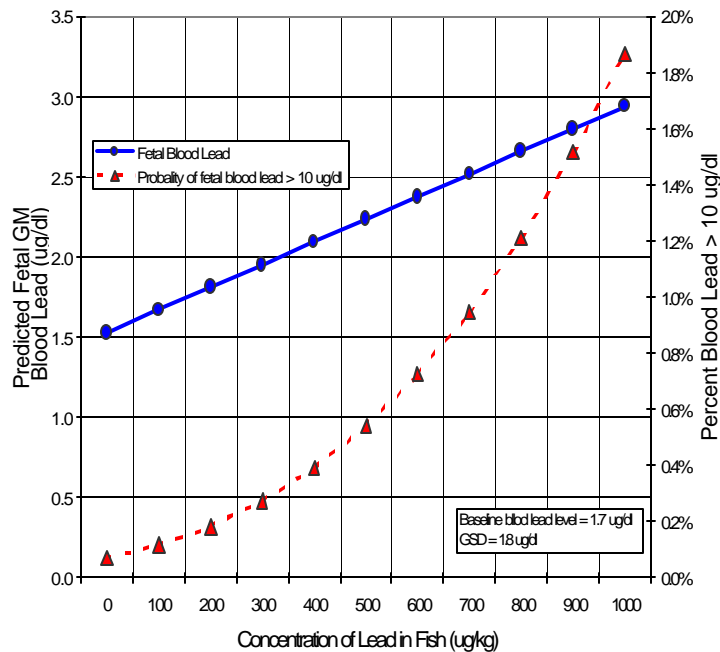


Figure 7-5. Predicted fetal blood lead level with maternal fish ingestion rate of 39.2 g/day with baseline blood lead level at 1.7  $\mu\text{g}/\text{dl}$  and GSD = 1.8  $\mu\text{g}/\text{dl}$ .

## 7.8 Uncertainty Analysis for Risk to Fetuses

Fetal risk estimates share common sources of uncertainties with the estimates for child risks including the assumed fish lead concentrations and fish consumption rates. Uncertainties unique to the Adult Lead Model include the assumed baseline blood lead level and geometric standard deviation parameters from the National Health and Nutrition Examination Survey (USEPA, 1996b). The results are based on the highest recommend values for the baseline blood lead levels and the geometric standard deviation. They are unlikely to underestimate risk.

## 7.9 Conclusions

Despite uncertainties in this assessment, lead levels in fish analyzed from the Columbia River occur at levels unlikely to cause a blood level greater than 10  $\mu\text{g}/\text{dl}$ . Risks to children from fish consumption are unlikely to exceed 5% at lead concentrations less than 500  $\mu\text{g}/\text{kg}$  (Figure 7-2, 7-3). Similarly, fetal risks are unlikely to exceed 5% at concentrations less than 700  $\mu\text{g}/\text{kg}$  (Figure 7-4, 7-5). These levels of concern occur at lead concentrations near the maximum values of the samples. This conclusion is supported by several analyses using health protective exposure assumptions that are unlikely to underestimate risks from fish consumption. The exposure assumptions are based on default and high end exposure parameters recommended by EPA lead risk assessment guidance used in conjunction with fish ingestion rates from the CRITFC fish consumption survey (CRITFC, 1994).



## 8.0 Radionuclide Assessment

### 8.1 Radionuclide Data Reporting and Use

A unique characteristic of some radionuclide analytical data is the occurrence of numerically negative results. Radionuclide analyses usually require the subtraction of an instrument background measurement from a gross sample measurement. Both results are positive, and when sample activity is low (close to background), random variations in measurements can cause the resulting net activity to be less than zero. Although negative activities have no physical significance, they do have statistical significance, as for example in the evaluation of trends or the comparison of groups of samples. Good practice for laboratory reporting of radionuclide analysis results therefore dictates reporting results as generated: whether positive, negative, or zero, together with associated uncertainties.

This is consistent with EPA guidance (USEPA, 1980a), which states: “When making measurements near background levels, one can expect to frequently obtain values that are less than the estimated lower limit of detection or minimum detectable concentration. If these values are not recorded and used in making average estimates, then these estimates are always going to be greater than the “true” representation in the environment. Therefore it is recommended that every measurement result should be recorded and reported directly as found.”

The general principles for evaluation of radionuclide data for this project were:

- a. It is generally best to use reported values plus the associated uncertainties.
- b. Reported values are better estimates of actual concentrations than are minimum detectable concentrations.
- c. J-qualified (estimated) data should not be used for quantitative purposes where unqualified data is available to substitute.
- d. All reported data (including U-qualified (nondetect) data, should be used in averages.
- e. Quantitative analyses should only be performed for those radionuclides which have at least one positive unqualified result reported.
- f. For gamma data, the EPA’s National Air and Radiation Exposure Laboratory (NAREL) reported minimum detectable concentration values for certain radionuclides of interest even in cases where the radionuclide was not detected and no value was reported. If these minimum detectable concentrations are used for quantitative analyses, the results should clearly note the use of minimum detectable concentration-based input. If minimum detectable concentrations are to be used for quantitative purposes, the minimum detectable concentrations may need additional decay corrections where holding times exceeded 10 half lives. This should not be an issue since no radionuclide with a half-life

less than 10% of holding time was detected in any of the gamma analyses and therefore these short-lived radionuclides would not be used for analytical purposes.

## **8.2 General Information on Radiation Risk**

Radiation is a known human carcinogen. As such, the models used to estimate risk from radiation exposure assume that at low levels of exposure, the probability of incurring cancer increases linearly with dose, and without a threshold.

All of the epidemiological studies used in the development of radiation risk models involve high radiation doses delivered over relatively short periods of time. Evidence indicates that the response per unit dose at low doses and dose rates from low-linear energy transfer radiation (primarily gamma rays) may be overestimated if extrapolations are made from high doses acutely delivered. The degree of overestimation is often expressed in terms of a dose and dose rate effectiveness factor that is used to adjust risks observed from high doses and dose rates for the purpose of estimating risks from exposures at environmental levels. EPA models for radiation risk include a dose and dose rate effectiveness factor of 2 applicable to most low-linear energy transfer radiation exposure. For high-linear energy transfer radiation (e.g. alpha particles), the differences in relative biological effect are accounted for in weighting factors applied in the calculation of dose and risk.

In addition to cancer risk, radiation can also represent a risk for hereditary effects. Radiation-induced genetic effects have not been observed in human populations, however, and cancers generally occur more frequently than genetic effects. The radiation-related risk of severe hereditary effects in offspring is estimated to be smaller than that for cancer. The risk of severe mental retardation from radiation exposure to the fetus is estimated to be greater per unit dose than the risk of cancer in the general population, but the period of susceptibility is very much shorter. Based on these considerations, EPA generally considers the risk of cancer to be limiting and uses it as the sole basis for assessing radiation-related human health risks.

The risk coefficients used in this risk assessment are derived using age-specific models and are age-averaged. This means that the risk coefficients are appropriate for use in estimating exposure over a lifetime, since they are derived by taking into account the different sensitivities to radiation as a function of age. The risk coefficients in this assessment may be used to assess the risk due to chronic lifetime exposure of an average individual to a constant environmental concentration. The risk estimates in this report are intended to be prospective assessments of estimated cancer risks from long-term exposure to radionuclides in the environment. The use of the risk coefficients listed for retrospective analyses of radiation exposures to populations should be limited to estimation of total or average risks in large populations. The risk coefficients are not intended for application to specific individuals or to specific subgroups.

Estimates of lifetime risk of cancer to exposed individuals resulting from radiological and chemical risk assessments may be summed to determine the overall potential human health hazard. It is standard practice, however, to tabulate the two sets of risk estimates separately. This

is due to important differences in the two kinds of risk estimates. For many chemical carcinogens, laboratory experiments and animal data are the basis for estimates of risk. In the case of radionuclides, however, the data come primarily from epidemiological studies of exposure to humans. Another important difference is that the risk coefficients used for chemical carcinogens generally represent an upper bound or 95<sup>th</sup> percent upper confidence level of risk, while radionuclide risk coefficients are based on best estimate values.

### 8.3 Risk Calculations

Data qualifiers assigned during the data verification and validation process were used in making decisions about numerical values for input into risk calculations. Reported values were used with the following exceptions: zero was used where negative values were reported and one half of the reported minimum detectable concentration was used where the result was reported as minimum detectable concentration.

The naturally-occurring radionuclide potassium-40 (K-40) is a special case in the risk calculations. Potassium is an essential nutrient which contains the naturally radioactive isotope potassium-40, which has a half-life of more than one billion years. K-40 constitutes 0.01% of natural potassium which as a result has a specific activity of approximately 800 pCi/g of potassium. Variations in diet have little effect on the radiation dose received, since the amount of potassium in the body is under close hemostatic control. Although K-40 is the predominant source of radiation exposure from food, calculation of dose or risk for specific food pathways is not meaningful since the biological control of potassium content in the body (and hence the radiation dose due to potassium) means that the dose is independent of intake. Therefore, K-40 concentrations were not included in the calculations of cumulative risk from radionuclides in samples. K-40 concentrations and risks are discussed separately for comparison.

Quantitative analyses were performed only for those radionuclides which had at least one positive unqualified result reported. Those radionuclides and their associated risk coefficients are:

<u>Radionuclide</u>	<u>Risk Coefficient (risk/Bq)</u>
Uranium -234 (U-234)	2.58 x 10 <sup>-9</sup>
Uranium-235+D (U-235+D)	2.63 x 10 <sup>-9</sup>
Uranium-238+D (U-238+D)	3.36 x 10 <sup>-9</sup>
Strontium-90+D (Sr-90+D)	2.58 x 10 <sup>-9</sup>
Plutonium-239 (Pu-239)	4.70 x 10 <sup>-9</sup>
Bismuth-212 (Bi-212)	included in Th-228+D coefficient
Bismuth-214 (Bi-212)	included in Ra-226+D coefficient
Cesium-137+D (CS-127+D)	1.01 x 10 <sup>-9</sup>
Potassium-40 (K-40)	9.26 x 10 <sup>-10</sup>
Lead-212(Pb-212)	included in Th-228+D coefficient
Lead-214(Pb-214)	included in Ra-226+D coefficient
Raon-224(Ra-224)	included in Th-228+D coefficient
Thorium-228+D (Th-228+D)	1.14 x 10 <sup>-8</sup>
Radon-226+D (Ra-226+D)	1.39 x 10 <sup>-8</sup>
Tellurim-208 (Tl-208)	included in Th-228+D coefficient

Risks

for individual radionuclides were calculated using morbidity coefficients for dietary intake from EPA guidance (USEPA 1999c). Many of the radionuclides detected are members of important naturally-occurring decay chains (e.g. Ra-226 series, Th-228 series). For these radionuclides, risks were calculated based on risk from the entire decay series in secular equilibrium. Risk coefficients representing the entire decay series (identified with “+D” designation) were derived by summing the risk coefficients for all decay chain members. For some decay series members (e.g. Po-218) no data is available in EPA guidance and these radionuclides were not included in the calculation of risk coefficients (USEPA, 1999d). Based on data for these radionuclides reported in HEAST the risks from radionuclides which are not included in EPA guidance are insignificant in comparison to the risks from the other members of the decay series for which EPA guidance provides data (USEPA, 1994c; USEPA, 1999d).

The general approach used in selecting data for input into decay series calculations was to:

- 1) use measured data wherever possible,
- 2) prioritize measured data in accordance with assigned data qualifiers, and
- 3) to use minimum detectable concentration values ( minimum detectable concentrations) for input only when other sources of data were not available.

In selecting the value to use for the concentration of the radionuclide at the head of the chain, decay products were used as surrogates. This is consistent with the physical principles of radioactive decay and secular equilibrium. Where more than one decay product was available to act as surrogate, positive values were selected over nondetect. The largest positive value was used where two or more otherwise equally suitable results were available.

In cases where Tl-208 was used as a surrogate for the Th-228 decay series, the branching ratio of the Bi-212 decay (36% decaying to Tl-208) was taken into account. If no decay chain member data is available, one-half of the minimum detectable concentration value for Ra-226 was used for input into the calculation for the Ra-226+D subchain. Similarly, one-half the minimum detectable concentration for Ra-228 was used as input into the Th-228+D subchain calculation where necessary. In the case of Cs-137, if no gamma peak was reported, one-half of the Cs-137 minimum detectable concentration was used as input for this radionuclide.

If there was a choice between uranium data from uranium alpha analyses and from gamma analyses (e.g. U-235), the uranium alpha analysis data was used. Alpha analysis for uranium is a more sensitive technique than gamma analysis. In particular, U-235 analysis by gamma spectroscopy involves additional analytical uncertainty resulting from Ra-226 interference with the spectral line used to quantify U-235. If only the gamma data was available, it was used with appropriate consideration of data qualifiers.

Analytical results used for risk calculations included three samples which had a total of six “J” qualified (estimated) results among them. Five of these estimated values represented uranium isotopes which are expected to be present, and for which the estimated values represent the best available data for input into the risk calculation. In one case the estimated value used represented a result for Pu-239. These estimated values were included in the calculations for completeness,

and their inclusion did not significantly alter the magnitude of the risks calculated.

## **8.4 Composite Study site Results**

Plutonium, strontium and uranium analyses were not performed on all samples sent for radionuclide analysis. For some of the composite groups of samples (composites 53 (study site Columbia River 9U), 24 (study site Columbia River 7), and 25 (study site Columbia River 8), only gamma analyses were performed. Risks were calculated based on the gamma component of these samples only. Risks were calculated based on a nominal consumption rate of 1 gram per day and also for consumption rates of 7.5 g/day (average public consumption), 142.4 g/day (99<sup>th</sup> percentile public consumption), 63.2 g/day (average CRITFC's member tribe consumption) and 389 g/day (99<sup>th</sup> percentile CRITFC's member tribe consumption). These consumption rates are the same as used for the nonradionuclide risk analysis. Risks were calculated for a 70 year lifetime. Composites of particular interest include Composite 54 (study site -K-Basin ponds) and 30 (study site Snake River 13). Table 8-1 presents a summary of the calculated risks for each consumption rate.

### **8.4.1 Potassium-40 Results**

As expected, the results for K-40 analyses are very consistent throughout the samples and represent one of the most prominent sources of radioactivity in all samples analyzed. The concentrations in samples ranged between 1.7 pCi/g and 3.7 pCi/g with an average value of 2.8 pCi/g. If this value were used to calculate risk in the same manner as the other radionuclides detected, the resulting calculated average risk would be  $1 \times 10^{-3}$ . As noted previously, however, although K-40 is the predominant source of radiation exposure from food, calculation of dose or risk for specific food pathways is not meaningful since the biological control of potassium content in the body (and hence the radiation dose due to potassium) means that the dose is independent of intake. Therefore, K-40 concentrations were not included in the calculations of cumulative risk from radionuclides in samples. K-40 concentrations and risks are presented separately for the purposes of comparison.

## **8.5 Background**

As anticipated, many of the radionuclides present in naturally-occurring background were also present in the samples analyzed. The sampling and analysis for radionuclides was not designed to provide the statistical power necessary to quantitatively define background. The mobile nature of the species sampled together with normal regional and local variations in concentrations of naturally-occurring radionuclides in the environment make such an effort impractical in the context of this project. However, an effort was made to obtain data that would provide a qualitative perspective on background concentrations in fish. To this end, samples were taken from the Snake River (composite group number 30; study site Snake River 13) to represent fish that would not be affected by the operations of nuclear facilities in the Tri-Cities area. Examination of the analytical results for the Snake River samples shows that in none of the samples was there any Pu-239 or Sr-90 detected. Cs-137 was detected, as could be expected from

the worldwide distribution of this radionuclide as a result of the atmospheric testing of nuclear weapons during the 1950's and early 1960's. In addition, naturally occurring radionuclides in the uranium and thorium decay series were also detected.

**Table 8-1. Composite risks for consumption of fish contaminated with radionuclides from the Columbia River Basin for the general public and CRITFC's member Tribes .**

Composite number (study sites)	Species	Unit (1 g/d)	Fish Consumption Rates			
			Average Public (7.5 g/d)	High Public (142.4 g/d)	Average CRITFC's member tribe (63.2 g/d)	High CRITFC's member tribe (389 g/d)
52 (9E,9F)	Largescale sucker	$6 \times 10^{-7}$	$5 \times 10^{-6}$	$9 \times 10^{-5}$	$4 \times 10^{-5}$	$2 \times 10^{-4}$
53 (9F,9H)	Largescale sucker	$9 \times 10^{-7}$ *	$7 \times 10^{-6}$ *	$1 \times 10^{-4}$ *	$6 \times 10^{-5}$ *	$4 \times 10^{-4}$ *
54 (9K)	White sturgeon	$6 \times 10^{-7}$	$5 \times 10^{-6}$	$9 \times 10^{-5}$	$4 \times 10^{-5}$	$2 \times 10^{-4}$
24 (7A)	White sturgeon	$1 \times 10^{-6}$ *	$8 \times 10^{-6}$ *	$1 \times 10^{-4}$ *	$6 \times 10^{-5}$ *	$4 \times 10^{-4}$ *
25 (8F)	White sturgeon	$8 \times 10^{-7}$ *	$6 \times 10^{-6}$ *	$1 \times 10^{-4}$ *	$5 \times 10^{-5}$ *	$3 \times 10^{-4}$ *
29 (8E,8B)	White sturgeon	$6 \times 10^{-7}$	$5 \times 10^{-6}$	$9 \times 10^{-5}$	$4 \times 10^{-5}$	$2 \times 10^{-4}$
84 (8F)	Channel catfish	$8 \times 10^{-7}$	$6 \times 10^{-6}$	$1 \times 10^{-4}$	$5 \times 10^{-5}$	$3 \times 10^{-4}$
85 (8F,8I)	Largescale sucker	$9 \times 10^{-7}$	$7 \times 10^{-6}$	$1 \times 10^{-4}$	$6 \times 10^{-5}$	$3 \times 10^{-4}$
86 (8C)	Channel catfish	$6 \times 10^{-7}$	$5 \times 10^{-6}$	$9 \times 10^{-5}$	$4 \times 10^{-5}$	$3 \times 10^{-4}$
30 (13E,13F)	White sturgeon	$8 \times 10^{-7}$	$6 \times 10^{-6}$	$1 \times 10^{-4}$	$5 \times 10^{-5}$	$3 \times 10^{-4}$
87 (9I)	White sturgeon	$7 \times 10^{-7}$	$5 \times 10^{-6}$	$1 \times 10^{-4}$	$4 \times 10^{-5}$	$3 \times 10^{-4}$
88 (9I)	White sturgeon	$7 \times 10^{-7}$	$5 \times 10^{-6}$	$1 \times 10^{-4}$	$4 \times 10^{-5}$	$3 \times 10^{-4}$
78 (9Q,9P)	Mountain whitefish	$8 \times 10^{-7}$	$6 \times 10^{-6}$	$1 \times 10^{-4}$	$5 \times 10^{-5}$	$3 \times 10^{-4}$
79 (9O,9N)	Mountain whitefish	$6 \times 10^{-7}$	$5 \times 10^{-6}$	$9 \times 10^{-5}$	$4 \times 10^{-5}$	$2 \times 10^{-4}$
82 (9D,9B,9A)	White sturgeon	$8 \times 10^{-7}$	$6 \times 10^{-6}$	$1 \times 10^{-4}$	$5 \times 10^{-5}$	$3 \times 10^{-4}$
83 (9A)	White sturgeon	$5 \times 10^{-7}$	$4 \times 10^{-6}$	$7 \times 10^{-5}$	$3 \times 10^{-5}$	$2 \times 10^{-4}$

\*Composites 53, 24, and 25 did not have uranium, strontium or plutonium analyses performed, and the composite risks do not include contributions from those radionuclides .

## 8.6 Uncertainties

The uncertainty associated with cancer risk estimates for ingestion of fish contaminated with radionuclides includes contributions from the analytical uncertainties of the reported results, and risk coefficients. The analytical uncertainties associated with the laboratory results are reported at the two standard deviation level. For radionuclide analyses, uncertainties related to counting statistics depend on the number of counts obtained, which varies with the analytical technique used as well as the concentrations of radionuclide in the sample. As a percentage of the reported result, their magnitude typically varies from a few percent in the case of gamma results which are significantly greater than detection limits (e.g. K-40 results), to 20-40% for uranium results, to more than 100% in cases of reported results which are classified as non-detect.

Some analytical results are qualified as estimated values due to interferences from other radionuclides in the analysis. Additional uncertainty results from the use of some radionuclides as surrogates for other radionuclides in decay series, the assumption of secular equilibrium, and the use of minimum detectable concentration data in calculating risk. These uncertainties likely result in overestimates of risk.

The uncertainties associated with the risk coefficients are likely to be larger than those due to analytical uncertainties. EPA guidance does not provide specific quantitative uncertainty estimates of the cancer risk coefficients (USEPA 1999d). National Council on Radiation Protection and Measurements. (NCRP) Report 126 (NCRP, 1997), examined the question of uncertainties in risk coefficients for the relatively simple case of external radiation exposure to low linear energy transfer (primarily gamma) radiation. The conclusion was that the 90% confidence interval encompassed a range approximately a factor of 2.5 to 3 higher and lower than the value of the risk estimate. Since estimates of risk from ingestion of food necessarily involve the added complexity of modeling of physiological processes to determine dose and risk, the uncertainties in this context are likely to be even greater.

The National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation (BEIR), in their report, addressed the issue of uncertainty in risk estimates for low doses from low linear energy transfer radiation (NAS, 1990). BEIR V considered the assumptions inherent in modeling such risks and concluded that at low doses and dose rates it must be acknowledged that the lower limit of the range of uncertainty in the risk estimates extends to zero.

## 8.7 Discussion

Considering the number of samples, the mobility of the fish, and the range of results obtained, it does not appear to be possible to attribute results to specific sources. Most of the radionuclides detected are known to be present naturally in the environment. Cs-137 is also widespread in the environment and was detected in many samples without apparent pattern. There were three samples in the vicinity of the Hanford Reach (Columbia River study site 9U) which showed positive detection results for Sr-90.



Sr-90, like Cs-137, is a widespread radionuclide resulting from atomic testing in the atmosphere. It is also associated with Hanford operations and is known from other environmental studies to be present in Columbia River sediments near Hanford.

The estimated risks are similar across all composite groups (Table 8-1). This is consistent with the observation that the majority of the estimated risk is generally due to radionuclides which are members of naturally occurring decay chains.

## **8.8 Conclusions**

The risks calculated for fish consumption (Table 8-1) are small relative to the estimated risks associated with radiation from naturally-occurring background sources, to which everyone is exposed. In the US, the average annual effective dose equivalent is approximately 300 millirem including exposure to radon. The lifetime risk associated with this background dose can be estimated to be approximately  $1 \times 10^{-2}$ , or 1%.

## **9.0 Comparisons of Fish Tissue Chemical Concentrations**

### **9.1 Comparison by Chemical Concentration**

In this section the fish tissue residues from our study are compared to other food types and studies of contaminants in fish reported in literature. This section also includes a comparison of fish tissue concentration data for smallmouth bass and channel catfish in addition to the 13 fish species which were the main focus of this report.

#### **9.1.1 Chlordane**

Chlordane was used as a pesticide from the 1940's until the late 1980's. Until 1983 it was used on corn and citrus fruits, lawns and gardens. It was banned in 1988.

Like most of the other cyclodiene pesticides (heptachlor, heptachlor epoxide, aldrin, dieldrin, endrin, and endosulfans I and II) chlordane degrades very slowly. Various of its metabolites can stay in the soil for over 20 years and can bioaccumulate in tissues of higher organisms.

Exposure to chlordane occurs largely from eating contaminated foods, such as root crops, meats, fish, and shellfish, or from touching contaminated soil. In the early 1980's chlordane was detected in 4 of 324 food composites: 3 potato composites ranging from trace to 2 µg/kg, and 1 garden fruit composite at a trace level (Gartrell et al., 1986). In the 1980 U.S. Food and Drug Administration (USFDA) market basket survey of infant and toddler diet samples, chlordane was detected at 5 µg/kg in one of 143 toddler food composites (Gartrell et al., 1985).

Chlordane concentrations of 118 to 290 µg/kg were measured in various estuarine fish in coastal states surveyed (Butler and Schutzmann, 1978). In a more recent survey, Munn and Gruber (1997) reported fish concentrations of 140 - 610 µg/kg of the sum of chlordane in composite samples of whole body fish from the Central Columbia Plateau.

The average concentrations of total chlordane found in anadromous fish tissue from our study ranged from <4 µg/kg in eulachon and coho salmon to 43 µg/kg in Pacific lamprey (Table 2-3). Egg samples from spring chinook sample had the highest average concentration (66 µg/kg) in our study (Table 2-3). The average concentrations of total chlordane in the resident fish species in our study ranged from < 2.4 µg/kg in rainbow trout and bridgelip sucker to 29 µg/kg in white sturgeon (Table 2-3).

#### **9.1.2 Total DDT**

The legal use of DDT in agriculture has been banned in the United States since 1972. DDT and its derivatives are persistent, bioaccumulative compounds which are ubiquitous in the organisms, sediments, and soils.

Exposure to DDT and its structural analogs (DDE, DDD) occurs primarily from eating contaminated foods, such as root and leafy vegetables, meat, fish, and poultry. From 1967 to 1972 the concentrations of total DDT in meat, fish and poultry decreased from 3,200 µg/kg to 900 µg/kg (IARC, 1978). From 1970 to 1973, DDE residues decreased only 27%, compared to a decrease of 86% and 89% for DDT and DDD, respectively (USEPA, 1980).

Based on data from the US Fish and Wildlife Service National Pesticides Monitoring Program (Schmitt et al., 1981), the DDT concentrations in fish ranged from 100 to 11,000 µg/kg.

DDT was detected in meats (0.3 µg/kg) and raw berries (2.0 µg/kg) consumed by indigenous residents of the Canadian Arctic (Berti et al., 1998).

The maximum concentration of DDE in the fish from several USGS surveys was in a whole body composite sample of carp (3,300 µg/kg) from the Brownlee Reservoir on the Snake River, Idaho (Table 9-1). The maximum concentration of DDE in our study was in the whole body composite sample of white sturgeon (1400 µg/kg) from the Hanford Reach of the Columbia River (study site 9U). The maximum concentrations of DDE in bridgelip sucker, rainbow trout, and largescale sucker levels in our study were higher than levels found by Munn and Gruber (1997) in the Central Columbia Plateau (Table 9-1). The largescale sucker levels in our study were similar to the largescale sucker levels reported by Clark and Maret (1998) for the Snake River Basin.

**Table 9-1. Comparison of range concentrations of sum of DDE (o,p' & p,p') in whole body composite fish samples Columbia River Basin.**

<b>Fish</b>	<b>µg/kg</b>	<b>Location</b>	<b>Reference</b>
carp	3300	Brownlee Reservoir, Snake River, Idaho	Clark and Maret ,1998
bridgelip sucker	87	Palouse River, Central Columbia Plateau	Munn and Gruber, 1997
bridgelip sucker	120-340	Northern Desert, Central Columbia	Munn and Gruber ,1997
<i>bridgelip sucker</i>	<i>347 - 612</i>	<i>Columbia River Basin</i>	<i>Our study, 1996-1998</i>
rainbow trout	9.5-32	Northern Desert, Central Columbia	Munn and Gruber, 1997
<i>rainbow trout</i>	<i>5-89</i>	<i>Columbia River Basin</i>	<i>Our study, 1996-1998</i>
largescale sucker	33-1300	Snake River Basin	Clark and Maret ,1998
largescale sucker	120-400	Palouse River, Central Columbia Plateau	Munn and Gruber, 1997
<i>largescale sucker</i>	<i>29-1312</i>	<i>Columbia River Basin</i>	<i>Our study, 1996-1998</i>

### 9.1.3 PCBs

PCBs, are stable, man-made chemicals that only degrade at very high temperatures. They do not conduct electricity and most of the various types of PCBs and PCB mixtures take the form of liquids. For these reasons, PCBs have been used extensively in much of the world as electrical insulating fluids, especially in capacitors and transformers which deliver high voltage in critical devices and situations where fire prevention is of great concern. PCBs have also been used extensively as hydraulic fluids, as well as in the manufacture of carbonless copy paper, etc. Environmental contamination with PCBs has resulted from industrial and domestic discharges, landfills, and atmospheric transport of incompletely incinerated PCBs.

Under environmental conditions, PCBs are extremely stable and slow to chemically degrade

(Eisler, 1986b). PCBs enter the environment as mixtures containing a variety of individual components (congeners) and impurities that vary in toxicity. The chlorinated nature of the various PCB molecules also makes them more fat soluble, and thus capable of bioaccumulating in aquatic food webs. The lipid solubility of the PCBs increases with increased chlorine substitution. This lipophilicity also tends to increase resistance to biodegradation.

Because of the relatively great environmental persistence and lipophilicity of this group of pollutants, low-level PCB contamination is now a global phenomenon, with PCB residues occurring almost universally in human milk, other human tissues, food, etc. For the general population, likely routes of ongoing chronic exposure to PCBs are primarily from food (Table 9-2).

**Table 9-2. PCB residues in raw agricultural commodities, 1970-76.**  
(Source: Duggan et al, 1971)

<b>Food Type</b>	<b>Number of samples</b>	<b>Percent Detected</b>	<b>Average (µg/kg)</b>
fish	2,901	46	892
eggs	2,302	9.6	72
milk	4,638	4.1	67
cheese	784	0.9	11
red meat	15,200	0.4	8
poultry	11,340	0.6	6

The estimated PCB content of a typical teenage boy's diet was about 15 µg/day in 1971, decreasing by 1975, to about 8.1 µg/day (IARC, 1978). The levels of PCBs have declined in ready-to-eat foods from 1978 to 1982 (Table 9-3). However, the human body burden remains high. The body burden of PCBs in human fat ranged between 500 and 1,500 µg/kg in 1987 (USEPA, 1987).

**Table 9-3. The declining trends in PCBs in ready-to-eat foods collected in markets of a number of US cities (Source: Duggan et al., 1971).**

<b>Year</b>	<b>Number of samples</b>	<b>Percent Detected</b>	<b>Average (µg/kg)</b>
1978	360	9	trace - 50
1979	360	4	<1 - 2
1980	360	2	2
1981- 82	324	2	1

In the 1980 -1981 USFWS survey of PCBs in fish from 107 locations the geometric was 530 µg/kg (Schmitt et al., 1985). This was lower than mean PCB levels from previous monitoring efforts, in which geometric means for PCBs were 880 µg/kg (1976-1977) and 850 µg/kg from (1978- 1979) (Schmitt et al., 1985).

In a 1976-1980 EPA survey of PCB residues in finfish from the Chesapeake Bay watershed, the concentrations ranged from non detects to 4,640 µg/kg (Tale 9-5). There was no trend over time as was observed in the USFWS Pesticide Monitoring Program.

**Table 9-4. The 1976-80 ranges for PCB residues from 547 finfish from the Chesapeake Bay and its tributaries ( Source: USEPA, 1987a).**

<u>Year</u>	<u>µg/kg</u>
1976	ND - 980
1977	30 - 510
1978	60 - 4,640
1979	10 - 1,600
1980	3 - 1,450

In later studies concentrations of total PCBs in a variety of fish tissue types ranged from 10 µg/kg in white sucker fillets in Saginaw Bay, Lake Huron, Michigan to 14,500 µg/kg in fish from the Spokane River, Washington (Table 9-5). Measurements of Aroclor 1254 and 1260 in white croaker muscle in California ranged from 1 µg/kg to 713 µg/kg (Table 9-6).

**Table 9-5. Total PCB concentrations in fish tissue from studies reported in the literature from 1978-1994.**

<u>Species &amp; Tissue type</u>	<u>µg/kg</u>	<u>Location/date of study</u>	<u>Reference</u>
fish livers	132 - 772	near the outfall for the Los Angeles County wastewater treatment plant 1980-81,	Gossett et al., 1983.
750 fish samples	70 - 14,500	11 major lakes and rivers in Alberta, Canada	Chovelon et al., 1984
25 white suckers fillets	10-180	Saginaw Bay, Lake Huron, 1979-1980	Kononen, 1989
freshwater fish (whole body) mean = 36 maximum =930		Spokane River, WA, 1999	Johnson, 2001

**Table 9-6. Concentrations Aroclor 1254 & 1260 in white croaker muscle tissue from California water bodies in the spring of 1994. (Source: Fairey et al., 1997)**

<u>µg/kg</u>	<u>Location</u>
137 - 613	13 locations throughout San Francisco Bay
1	Southern California Dana Point,
757	Malibu

The concentration of Aroclor 1254 ranged from 480 µg/kg to 9,930 µg/kg in lake trout from lakes in Michigan (Table 9-7). The concentration of Aroclor 1254 in resident fresh water species from our study ranged from 10 µg/kg in rainbow trout to 930 µg/kg in mountain whitefish.

**Table 9.7. Concentrations of Aroclor 1254 in lake trout from lakes in Michigan during 1978-82 (Devault et al., 1986).**

<u>µg/kg</u>	<u>Location</u>
5630 - 9930	Lakes Michigan
2100 - 3660	Lake Huron
480-1890	Lake Superior

The concentration of Aroclors in chinook salmon eggs from Lake Michigan were much higher

than the levels found in our study (Table 9-8).

**Table 9-8. Aroclor concentrations in chinook salmon eggs reported for Lake Michigan, Michigan, compared to our study of Aroclors in the chinook salmon eggs.**

$\mu\text{g}/\text{kg}$	N	salmon	Location/date of study
<b>Aroclor 1254</b>			
5,400		chinook	Lake Michigan, 1982 (Jaffet et al., 1985)
<i>12</i>	<i>1</i>	<i>fall chinook</i>	<i>Columbia River Basin, 1996-1998</i>
<i>15 - 20</i>	<i>6</i>	<i>spring chinook</i>	<i>Columbia River Basin, 1996-1998</i>
<b>Aroclor 1260</b>			
1,100		chinook	Lake Michigan, 1982 (Jaffet et al., 1985)
<i>&lt;19</i>	<i>1</i>	<i>fall chinook</i>	<i>Columbia River Basin, 1996-1998</i>
<i>&lt;18</i>		<i>spring chinook</i>	<i>Columbia River Basin, 1996-1998</i>

< = detection limit

Concentrations of PCBs measured in fish from our study were compared to other fish surveys in Lake Roosevelt on the upper Columbia River in Washington (Table 9-9). The maximum concentration of Aroclors 1254 and 1260 in walleye and rainbow trout were lower in our study of the Columbia River Basin than the EPA (USEPA, 1998c) and USGS (Munn, 2000) surveys of Lake Roosevelt, Washington. Concentrations of the Aroclors in white sturgeon were higher in our study than the EPA study of Lake Roosevelt, Washington (Table 9-9).

**Table 9-9. Concentrations of Aroclors 1254 and 1260 in composite samples of fish fillets from Lake Roosevelt, Washington compared concentrations measured in our study of the Columbia River Basin.**

Fish Species	$\mu\text{g}/\text{kg}$	N	Location	Reference
<b>Aroclor 1254</b>				
small walleye	30 - 10	9	Lake Roosevelt, 1994	USEPA, 1998c
large walleye	35 - 89	2	Lake Roosevelt, 1994	USEPA, 1998c
<i>walleye</i>	<i>12 - 14</i>	<i>7</i>	<i>Columbia River Basin, 1996-1998</i>	<i>our study</i>
white sturgeon*	15 - 77	2	Lake Roosevelt, 1994	USEPA, 1998c
<i>white sturgeon*</i>	<i>10 - 190</i>	<i>16</i>	<i>Columbia River Basin, 1996-1998</i>	<i>our study</i>
rainbow trout	13 - 45	10	Lake Roosevelt, 1994	USEPA, 1998c
rainbow trout	3 - 49	16	Lake Roosevelt, 1998	Munn, 2000
<i>rainbow trout</i>	<i>10 - 20</i>	<i>7</i>	<i>Columbia River Basin, 1996-1998</i>	<i>our study</i>
smallmouth bass	ND - 8	9	Lake Roosevelt, 1994	USEPA, 1998c
<i>smallmouth bass</i>	<i>38 - 83</i>	<i>3</i>	<i>Columbia River Basin, 1996-1998</i>	<i>our study</i>
kokanee	28 - 40	4	Lake Roosevelt, 1994	USEPA, 1998c
lake whitefish	31 - 51	3	Lake Roosevelt, 1994	USEPA, 1998c
<b>Aroclor 1260</b>				
small walleye	4 - 13	9	Lake Roosevelt, 1994	USEPA, 1998c
large walleye	23 - 32	2	Lake Roosevelt, 1994	USEPA, 1998c
<i>walleye</i>	<i>&lt;19</i>	<i>7</i>	<i>Columbia River Basin, 1996-1998</i>	<i>our study</i>
white sturgeon*	13 - 102	2	Lake Roosevelt, 1994	USEPA, 1998c
<i>white sturgeon*</i>	<i>13 - 200</i>	<i>16</i>	<i>Columbia River Basin, 1996-1998</i>	<i>our study</i>
rainbow trout	5 - 72	10	Lake Roosevelt, 1994	USEPA, 1998c
<i>rainbow trout</i>	<i>&lt;18</i>	<i>7</i>	<i>Columbia River Basin, 1996-1998</i>	<i>our study</i>
smallmouth bass	3 - 6	9	Lake Roosevelt, 1994	USEPA, 1998c
<i>smallmouth bass</i>	<i>68 - 220</i>	<i>3</i>	<i>Columbia River Basin, 1996-1998</i>	<i>our study</i>
kokanee	10 - 14	4	Lake Roosevelt, 1994	USEPA, 1998c
lake whitefish	16 - 29	3	Lake Roosevelt, 1994	USEPA, 1998c

N - number of samples < = detection limit \*White sturgeon were individual fillets without skin

#### 9.1.4 Chlorinated Dioxins and Furans

Because of their chlorination and specific chemical structures, most chlorinated dioxins and furans are highly fat soluble, and difficult for the body to quickly degrade and excrete. They are similar to some of the other persistent chlorinated residues like DDT and PCBs. Also like PCBs and DDTs, chlorinated dioxins and furans can bioaccumulate in fish. The amount of furans in fish can sometimes be tens of thousands times higher than the levels in the surrounding water.

The chlorinated dibenzodioxins and chlorinated dibenzofurans are not produced intentionally by industrial processes. Rather, most chlorinated dioxins and furans are generated in very small amounts as unwanted impurities during the manufacture of several chlorinated chemicals and consumer products, including certain wood treatment chemicals, some metals, and paper products. When the waste water, sludge, or solids from these processes are released into waterways or soil in dump sites, the sites may become contaminated with chlorinated dioxins and furans. These unwanted contaminants also enter the environment from burning municipal and industrial waste in incinerators, as well as from gasoline exhaust, and the burning of coal, wood, or oil for home heating and production of electricity. Other production chemicals which can generate unwanted trace amounts of 2,3,7,8-TCDD have included the forestry herbicide 2,4,5-trichlorophenoxy propionic acid (Silvex), and the industrial chemical 2,4,5-trichlorophenol. Unwanted trace amounts of some of the higher-chlorinated dioxins, especially the hexa and octa isomers, have also been associated with the production of the widely used wood preservative, pentachlorophenol.

Many of the various chemicals and processes which significantly produce chlorinated dioxins and furans in the environment are either being slowly phased out or are strictly controlled. It is currently believed that chlorinated dioxin and furan emissions associated with incineration and combustion activities are the predominant environmental source of these contaminants (USEPA, 2000e). Chlorinated dioxins and furans also arise from natural processes in the environment such as forest fires and volcanos.

TCDF is often found in fish tissue because of its affinity for lipids and because of its formation as a by-product in the industrial processes, especially pulp and paper mills (USEPA, 2000e). The concentration of 2,3,7,8-TCDF was measured in a variety of fish species from Lake Roosevelt, Washington by the USEPA in 1994 (Table 9-10). The concentrations of 2,3,7,8-TCDF in walleye ranged from 0.0001 to 0.0063  $\mu\text{g}/\text{kg}$  (Table 9-10). The maximum concentration from our study was lower than the maximum reported for Lake Roosevelt, Washington. The white sturgeon 2,3,7,8-TCDF maximum concentration in our study was higher than the maximum from the 1994 Lake Roosevelt study (Table 9-10). The rainbow trout 2,3,7,8-TCDF concentrations were similar in both studies.

**Table 9-10. Concentrations of 2,3,7,8-TCDF in composite samples of fish filets collected from Lake Roosevelt, Washington in 1994 compared with our 1996-1998 survey of the Columbia River Basin.**

<b>Fish</b>	<b>µg/kg</b>	<b>N</b>	<b>Collection date</b>	<b>Reference</b>
small walleye	0.0001 - 0.0016	9	Lake Roosevelt, 1994	USEPA, 1998c
large walleye	0.0007 - 0.0063	2	Lake Roosevelt, 1994	USEPAc 1998c
<i>walleye</i>	<b>0.0006 - 0.00085</b>	<b>3</b>	<b><i>Columbia River Basin, 1996-98</i></b>	<b><i>our study</i></b>
white sturgeon	0.016 - 0.025	2	Lake Roosevelt, 1994	USEPA, 1998c
<i>white sturgeon</i>	<b>0.0025 - 0.054</b>	<b>16</b>	<b><i>Columbia River Basin, 1996-98</i></b>	<b><i>our study</i></b>
small rainbow trout	0.000098 - 0.0015	6	Lake Roosevelt, 1994	USEPA, 1998c
large rainbow trout	0.0015 - 0.00188	10	Lake Roosevelt, 1994	USEPA, 1998c
<i>rainbow trout</i>	<b>0.0001 - 0.0003</b>	<b>7</b>	<b><i>Columbia River Basin, 1996-98</i></b>	<b><i>our study</i></b>
kokanee	0.0028 - 0.0031	4	Lake Roosevelt, 1994	USEPA, 1998c
smallmouth bass	0.00001 - 0.0041	9	Lake Roosevelt, 1994	USEPA, 1998c
lake whitefish	0.0038 - 0.01610	3	Lake Roosevelt, 1994	USEPA, 1998c

N= number of samples

In the USEPA National Dioxin Survey (USEPA, 2000d) background levels of toxicity equivalence concentrations for chlorinated dioxins, furans, and dioxin-like PCB congeners were  $0.00116 \pm 0.00121$  µg/kg in fish and  $0.00046 \pm 0.00099$  µg/kg in beef. In our study the average toxicity equivalence concentrations ranged from a low of 0.0004 µg/kg in fall chinook salmon to the highest average concentration of 0.0063 µg/kg in mountain whitefish.

### 9.1.5 Metals

The metals measured in our study are naturally occurring substances. Some of these metals are essential at trace levels for survival of vertebrates. These chemicals may combine with other chemicals to form compounds, (e.g. methylmercury, dimethylarsenic, arsenocholine, arsenosugars) which alters their bioavailability and toxicity. Most can become toxic if sufficiently high levels are encountered in the environment. Many of the metals which are taken up by fish tend to increase in concentration as the organisms age and increase in body size (Wiener and Spry, 1996, reported in Clark and Maret, 1998).

Information about barium, beryllium, cobalt, and manganese and are not included in this section. Background information on these chemicals is included in the Toxicity Profiles (Appendix C)

### 9.1.6 Aluminum

Aluminum is the most common and widely distributed metal in the earth's crust. Concentrations as high as 150,000 - 600,000 mg/kg have been reported in soil. The average ingestion of aluminum by humans has been estimated at 30 - 50 mg/day (Bjorksten, 1982). This estimate may be low, in light of a 1997 United Kingdom (UK) total diet study involving 20 different food groups from 20 representative towns, for the general UK population, where the highest mean concentrations of aluminum were found in the bread (6,600 µg/kg) and fish (6,100 µg/kg) (Ysart et al., 2000). Aluminum is present in the natural diet, in amounts varying from very low in animal products to relatively high in plants.



In our study the basin-wide average aluminum concentrations ranged from non-detect in coho salmon (whole body and fillet) to 69,000 µg/kg in whole body largescale sucker. The maximum concentration was 190,000 µg/kg in the largescale sucker composite sample from the main-stem Columbia River (study site 8).

### 9.1.7 Arsenic

Arsenic is found widely in nature, and occurs most abundantly in sulfide ores. Arsenic levels in the earth's crust average about 5,000 µg/kg. Arsenic is found in trace amounts in aquatic environments. As was described in Section 5, arsenic exists in both organic and inorganic forms. The most common combined form of arsenic is the inorganic compound, arsenopyrite (FeAsS). The organic arsenic compounds are less toxic than the inorganic arsenic compounds.

Arsenic does not readily bioconcentrate in aquatic organisms. It is typically water soluble and does not combine with proteins. Since, aquatic invertebrates accumulate arsenic more readily than fish biomagnification is unlikely (Spehar et al., 1980). Planktivorous fish are more likely to concentrate arsenic than omnivorous or piscivorous fishes (Hunter et al., 1981). Eisler (1988a) found no evidence that biomagnification occurs in aquatic food chains. In 1995, Robinson et al., found no evidence of arsenic uptake or accumulation from water in both rainbow and brown trout. The rainbow trout in our study had the lowest arsenic concentrations (<25 µg/kg fillet; 120 µg/kg whole body) of the fish species sampled.

In a 1997 UK study, dietary exposures to arsenic were estimated to be about 65 µg /day (Ysart et al., 2000). The “fish” food group had the highest mean arsenic concentration (400 µg/kg; Ysart et al., 2000).

Arsenic levels recorded for fish tissues seem to be quite variable. Fish taken from the Great lakes contained 5.6 - 80 µg/kg arsenic; primarily in the lipid fraction of the fish tissue (Lunde, 1970). In a study of African tilapia fish, muscle tissue contained arsenic levels ranging from 110 µg/kg (Ikdu and Marget Lakes) to one specimen with 10,500 µg/kg (Abu Quir Bay) ( El Nabawi et al., 1987). Ashraf and Jaffar (1988) measured arsenic levels of 2,880 µg/kg and 2510 µg/kg in two tuna species from the Arabian Sea. The authors noted that increased arsenic content was proportional to increased weight in the tuna species.

The average arsenic levels in resident, fresh water fish species in our study ranged from not detect in rainbow trout fillet to 490 µg/kg in whole body walleye (Table 2-14). The average concentrations in anadromous species from our study ranged from 310 µg/kg in Pacific lamprey fillet to 890 µg/kg in whole body eulachon. There was no correlation between lipid and arsenic in fish in our study, as was observed in the Great Lakes study (Lunde, 1970) or body weight and arsenic as observed by Asraf and Jaffar (1988).

### 9.1.8 Cadmium

Cadmium naturally occurs in the aquatic environment, but is of no known biological use and is considered one of the most toxic metals. While cadmium is released through natural processes, anthropogenic cadmium emissions have greatly increased its presence in the environment. In aquatic systems, cadmium quickly partitions to sediment, but is readily remobilized through a variety of chemical and biological processes (Currie et al., 1997). Cadmium does not bioconcentrate significantly in fish species, but does tend to accumulate more readily in invertebrates. Omnivorous and insectivorous predators tend to accumulate cadmium in their tissues more than piscivorous predators (Scheuhammer, 1991). Saiki et al., (1995) found no evidence of biomagnification of cadmium in steelhead on the Upper Sacramento River. Eisler (1985a) also maintains that evidence for cadmium biomagnification suggests that only the lower trophic levels exhibit biomagnification. Cadmium tends to form stable complexes with metallothionein (a sulfhydryl-rich protein). The resulting cadmium complexes have long half-lives and a tendency to accumulate with age in exposed organisms. As such, long lived species tend to be at a higher risk from chronic low-level dietary cadmium exposure.

People who are smokers are exposed to significant levels of inhaled cadmium. The major exposure route for the non-smoking human population is via food. In a 1997 UK study, the mean population dietary exposures to cadmium was estimated to be about 12 µg/kg/day for the general UK population (Ysart et al., 2000). Cadmium concentrations were highest in the viscera and trimmings of animals (77 µg/kg), and nuts (59 µg/kg), while the bread and potato food groups made up the greatest contributions (both 25%) to dietary exposure of the general population.

Certain cruciferous vegetable crops are known to be able to sequester elevated cadmium levels if grown in sufficiently contaminated soils. Queiroloa et al. (2000) reported ranges of 0.2 to 40 µg/kg for cadmium, with highest levels being found in potato skin in a study of vegetables (broad beans, corn, potato, alfalfa and onion) from farming villages in Northern Chile.

The WHO (1992) indicates that marine organisms generally contain higher cadmium residues than their freshwater and land-dwelling counterparts. In our study the highest cadmium levels were in whole body samples of largescale sucker (250 µg/kg ) followed by spring chinook salmon (170 µg/kg) and Pacific lamprey (150 µg/kg).

Average cadmium concentrations ranged from non detect in fillet samples of walleye, coho salmon, and fall chinook salmon to 120 µg/kg in whole body spring chinook salmon. The maximum concentration (250 µg/kg) was in the largescale sucker composite sample from the Hanford Reach of the Columbia River (study site 9U).

### 9.1.9 Chromium

Chromium is widely distributed in the earth's crust, with an average concentration of about 125,000 µg/kg. It is found in small amounts in all soils and plants. Most of the chromium present in food is in the trivalent form [Cr(III)], which is an essential nutrient. The hexavalent

form is more toxic, but is not normally found in food. In freshwater environments, hydrolysis and precipitation are the most important processes in determining the environmental fate of chromium, while absorption and bioaccumulation are considered minor. Chromium (VI) is highly soluble in water and thus very mobile in aquatic systems (Ecological Analysts, 1981).

The mean daily dietary intake of chromium from air, water, and food, is estimated to be about 0.2 - 0.4 µg, 2.0 µg, and 60 µg, respectively (ATSDR, 2000). The predicted intakes from air chromium are probably exceeded considerably in the case of smokers, and those who are occupationally exposed.

In a 1997 UK study, meat products contained the highest mean chromium concentration (230 µg/kg), but beverages made the greatest dietary contribution (19%) to the population exposure to chromium (Ysart et al., 2000). The US Food and Nutrition Board has recommended a safe and adequate dietary intake of chromium of 0.05 - 0.20 µg/day (Seller and Sigel, 1988).

Chromium was found in fish sampled from 167 lakes in the northeast United States at levels ranging from 30-1,460 µg/kg with a mean of 190 µg/kg (Yeardley et al., 1998). Seaweeds have been shown to sequester total chromium by a bioaccumulation factor of about 100 times greater than ambient levels in seawater (Boothe and Knauer, 1972). Snails showed an accumulation factor of  $1 \times 10^6$  for total chromium (Levine, 1961).

In our study, basin-wide average chromium concentrations ranged from <100 µg/kg in eulachon to 360 µg/kg in the whole body white sturgeon (Table 2-14). The maximum concentration (1000 µg/kg) was measured in the whole body white sturgeon sample from the main-stem Columbia River (study site 8)

### **9.1.10 Copper**

Because of its ubiquitous occurrence in the environment, and its essentiality for life, copper is found naturally at trace levels in aquatic and terrestrial organisms. Copper is not strongly bioconcentrated in vertebrates, but is more strongly bioconcentrated in invertebrates. In salmonids the accumulation of copper in muscle, kidney, and spleen tissues occurred at copper concentrations ranging from 0.52-3 µg/L in both seawater and freshwater (freshwater hardness=46-47 mg/L)(Camusso and Balestrini, 1995; Peterson et al., 1991; Saiki et al., 1995). The concentrations of copper in fish tissues reflect the amount of bioavailable copper in the environment. Baudo (1983, Wren et al. (1983), and Mance (1987) have all concluded that copper, along with zinc and cadmium do not biomagnify in the aquatic environment.

Intake of copper from food tends to be about one order of magnitude greater than intake from drinking water (USEPA, 1987). Exceptions to this are in relatively rare situations involving consumption of “soft” drinking water sources supplied by copper pipes; which can result in daily individual drinking water intakes of copper in excess of 2 mg/day. In a 1997 UK diet study, copper was highest in viscera and trimmings (50,000 µg/kg) and nuts (8,500 µg/kg), with mean concentrations in the other food groups ranging from 50 to 2,100 µg/kg (Ysart et al., 2000).

In our study, the copper concentrations ranged from 250 µg/kg in white sturgeon fillet sample to 4500 µg/kg in whole body Pacific lamprey. The maximum concentration (14,000 µg/kg) was in the whole body fall chinook salmon composite sample from the main-stem Columbia River (study site 14).

#### **9.1.11 Lead**

Lead is a naturally occurring, ubiquitous compound that can be found in rocks, soils, water, plants, animals, and air. Lead is the fifth most prevalent commercial metal in the US. Lead is found naturally in all plants, with normal concentrations in leaves and twigs of woody plants of about 2,500 µg/kg, pasture grass 1,000 µg/kg, and cereals from 100 -1,000 µg/kg (IARC, 1980).

Absorption of lead by aquatic animals is affected by the age, gender and diet of the organism, as well as the particle size, chemical species of lead, and presence of other compounds in the water (Eisler, 1988b; Hamir et al., 1982). Although inorganic lead is poorly accumulated in fish, it has been shown to bioconcentrate in aquatic species. Invertebrates tend to have higher lead bioconcentration factors than vertebrates. A bioconcentration factor of 42 was observed in brook trout embryos (Eisler, 1988b). Bioconcentration factors decrease as waterborne lead concentrations increase, thus suggesting accelerated depuration or saturation of uptake mechanisms (Hodson et al., 1984). Exposures of rainbow trout to 3.5-51 µg/L tetramethyl lead from 7 - 14 days resulted in rapid accumulation of lead. However, once the fish were removed to clean water, lead decreased rapidly from organs, followed by a slower release from other body components, until baseline levels were reached. An increase in dietary calcium of 0-8400 µg/kg reduced the uptake of waterborne lead in coho salmon, possibly due to interactions with gill membrane permeability (Hodson et al., 1984). In vertebrates, lead concentrations tend to increase with age and localize in hard tissues such as bone or teeth.

The primary exposure route for lead is food (Table 9-11). Foods which are likely to have elevated lead levels are dried foods, liver, canned food, and vegetables which have a high area-to-mass ratio. Historic use of soldered food cans greatly increased the lead content of prepared and processed foods. Sherlock (1987) reported that while ravioli from welded (no lead) cans contained 30 µg/kg lead, ravioli from a 98% lead soldered can was found to contain a mean content of 150 µg/kg lead.

**Table 9-11. Lead concentrations in food purchased in five Canadian cities between 1986 - 1988 (Source: Dabeka and McKenzie, 1995).**

category	% contribution to dietary intake	mean $\mu\text{g}/\text{kg}$	maximum $\mu\text{g}/\text{kg}$
fruits and fruit juice	13.9	44.4	372.7
miscellaneous	6.1	41.7	178.9
vegetables	16.8	24.4	331.7
meat and poultry	7.6	20.2	523.4
<i>fish</i>	0.7	19.3	72.8
sugar and candies	1.5	18.3	111.6
soups	4.5	15.5	48.7
bakery goods and cereals	20.6	13.7	66.4
beverages	20.9	9.9	88.8
fats and oils	0.3	9.6	19.7
milk and milk products	7.1	7.7	44.7
canned and raw cherries			203
canned citrus fruit			126
canned beans			158
canned luncheon meats			163

The basin-wide average lead concentrations in fish from our study of the Columbia River Basin ranged from non detect in fillets of Pacific lamprey, walleye, and rainbow trout to 500  $\mu\text{g}/\text{kg}$  in whole body eulachon (Table 2-14). The maximum concentration (1200  $\mu\text{g}/\text{kg}$ ) in our study was in the whole body fall chinook salmon from the main-stem Columbia River (study site 14).

### 9.1.12 Mercury

While mercury does occur naturally in small amounts in aquatic environments, the cycling of mercury prolongs the influence of man-made mercury compounds (Hudson et al., 1995). Mercury is cycled through the environment through an atmospheric-oceanic exchange. This cycling is facilitated by the volatility of the metallic form of mercury. Natural bacterial transformation of mercury results in stable, lipid soluble, alkylated compounds such as methyl mercury (Beijer and Jernelov, 1979). In sediments, mercury is usually found in its inorganic forms, but aquatic environments are a major source of methyl mercury (USEPA, 1985). In background freshwater systems, mercury occurs naturally at concentrations of 0.02-0.1  $\mu\text{g}/\text{L}$  (Moore and Ramamoorthy, 1984).

Mercury has been shown to bioconcentrate in a variety of aquatic organisms. Aquatic predators face the greatest danger of bioconcentrating mercury, and thus their tissue concentrations best reflect the amount of mercury available to aquatic organisms in the environment. Fish have been shown to concentrate mercury as methyl mercury even when they are exposed to inorganic mercury. Fish, such as rainbow trout, have been found to accumulate mercury in the form of methyl mercury at aquatic concentrations as low as 1.38  $\text{ng}/\text{L}$  (Ponce and Bloom, 1991).

Some evidence supports the biomagnification of mercury in aquatic food chains. When comparing benthic feeding fish, fish that feed on plankton, invertebrates, and vertebrates, the

greatest mercury concentrations were found in piscivorous fishes. Thus, the authors of this study concluded that mercury content in fish increased with higher trophic levels (Wren and MacCrimmon, 1986).

Freshwater ecosystems historically associated with heavy gold mining activity have often been impacted by elevated mercury levels in fish. This is in large part due to the use of liquid elemental mercury, or quicksilver, as a means of separating out gold during the mining process, especially during historic times.

Dietary sources greatly exceed other media like air and water as a source of human mercury exposure and uptake. In a 1997 UK diet study, fish contained the highest mean concentration (43 µg/kg), and made the greatest contribution (33%) to the population dietary exposure estimate (Ysart et al., 2000). The World Health Organization, EPA, and others indicate that risk to humans from mercury contamination via ocean fish is mainly through the consumption of predator species like swordfish, king mackerel, and shark (WHO, 1976).

In a monitoring study of fish in British Columbia, Canada, mercury concentrations in muscle tissue of various fish ranged from 40 µg/kg in rainbow trout to 2,860 µg/kg in lake trout (Table 9-12). In our study, rainbow trout the average mercury concentrations ranged from 73 µg/kg in whole body samples to 77 µg/kg in the fillet samples (Table 2-14).

<b>Fish Species (study location)</b>	<b>µg/kg</b>
Rainbow trout (Tezzeron Lake)	40
herring	70
dolly varden or char (Carpenter Lake)	410-1,940
dogfish or shark (English Bay)	1,080
lake trout (Pinchi Lake)	2,860

A 1984 EPA national survey of fish tissue found mercury ranging from 50 µg/kg in salmon to 610 µg/kg in pike (Table 9-13). In our study average mercury concentrations in fillet samples of salmon was 84 µg/kg in fall chinook, 100 µg/kg in spring chinook, and 120 µg/kg in coho. (Table 2-14).

**Table 9-13. EPA 1984 survey of total mercury concentrations in edible fish tissue, shrimp, and prepared foods. (Source USEPA, 1984b)**

Fish Species	µg/kg	Invertebrates	µg/kg	Prepared food	µg/kg
salmon	50	shrimp	460	fish sticks	210
whiting	50			canned tuna	240
sardines	60				
flounder	100				
snapper	450				
bass	210				
catfish	150				
trout	420				
pike	610				

In a more recent EPA national survey of mercury in fish tissue, median mercury levels ranged from 1 µg/kg in largemouth bass, channel catfish, bluegill sunfish, and common carp to 8,940 µg/kg in largemouth bass (Table 9-14). The concentrations of mercury fillets of fish tissue in our study were 380 - 470 µg/kg in smallmouth bass, 160 - 200 µg/kg in walleye, and 240 - 280 µg/kg in channel catfish (Table 9-27). All of these fish species had lower concentrations in our study than in the EPA 1990-1995 survey (USEPA, 1999e).

**Table 9-14. Mercury concentrations from an EPA 1990 - 1995 national survey of fish fillets (Source : USEPA, 1999e).**

Species	µg/kg
largemouth bass	1 - 8,940
Smallmouth bass	8 - 3,340
walleye	8 - 3,000
northern pike	100 - 4,400
channel catfish	1 - 2,570
bluegill sunfish	1 - 1,680
common carp	1 - 1,800
white sucker	2 - 1,710
yellow perch	10 - 2,140

In 1999, May et al. (2000) collected 141 samples of fish from reservoir and stream areas in the Bear and South Yuba River watersheds in the Sierra Nevada of Northern California (Table 9-15). Fish concentrations in the California survey ranged from 20 µg/kg to 1,500 µg/kg (Table 9-15). Rainbow trout mercury concentrations in fillets ranged from 45 - 150 µg/kg (Table 9-27). Channel catfish mercury concentrations ranged from 240 - 280 µg/kg (Table 9-27).

**Table 9-15. USGS survey of mercury concentrations in fish tissue from reservoirs and streams in Northern California. (Source: May et al, 2000). Fish were fillets without skin**

<b>Reservoir</b>	<b>µg/kg</b>
largemouth bass	20 - 1,500
Reservoir sunfish	< 100 - 410
channel catfish	160 - 750
<b>Streams</b>	<b>µg/kg</b>
Brown trout	20 - 430
rainbow trout	60 - 380

Several recent surveys in Washington measured concentrations of mercury in resident fish species (Table 9-16). The walleye samples from our study were within the range of the samples from Munn and Short (1997) and Munn (2000). Smallmouth bass from our study were within the range of the studies by Munn et al. (1995) and Sedar et al. (2001) although the maximum concentrations in our smallmouth bass were lower than the levels found in Lake Roosevelt, Washington (Munn et al., 1995) and Lake Whatcom (Serdar et al., 2001). Serdar et al., (2001) reported a mean concentration of (70 µg/kg) in most fish species in Washington State. The authors found higher concentrations of mercury in 6 of 8 fillets with the skin off. In our study all the fillets, except white sturgeon, were analyzed with skin. There was also no consistent pattern between fillets with skin or whole body. Rainbow trout concentrations from our study were also within the range observed in rainbow trout from Lake Roosevelt, Washington, although the maximum was lower than the maximum observed in Lake Roosevelt (Munn et al, 1995).

**Table 9-16. Mercury concentrations in fish fillets collected in Lake Whatcom and Lake Roosevelt, Washington compared to our study of the Columbia River Basin .**

<b>Fish species</b>	<b>Tissue Type</b>	<b>µg/kg</b>	<b>N</b>	<b>Location</b>	
walleye	composite	110 - 440	34	Lake Roosevelt, 1994	Munn and Short 1997
walleye	individual	110 - 150	8	Lake Roosevelt, 1998	Munn 2000
<b>walleye</b>	<b>composite</b>	<b>160 - 200</b>	<b>3</b>	<b>Columbia River Basin, 1996-1998</b>	<b>our study</b>
smallmouth bass	composite	160 - 620	5	Lake Roosevelt, 1994	Munn et al., 1995
smallmouth bass	individual	100 - 1840	96	Lake Whatcom, 2000	Serdar et al., 2001
<b>smallmouth bass</b>	<b>composite</b>	<b>380 - 470</b>	<b>3</b>	<b>Columbia River Basin, 1996-1998</b>	<b>our study</b>
rainbow trout	individual	110 - 240	6	Lake Roosevelt, 1994	Munn et al., 1995
<b>rainbow trout</b>	<b>composite</b>	<b>45 - 150</b>	<b>7</b>	<b>Columbia River Basin, 1996-1998</b>	<b>our study</b>
perch	individual	120 - 290	30	Lake Whatcom, 2000	Serdar et al., 2001
kokanee	individual	100 - 130	30	Lake Whatcom, 2000	Serdar et al., 2001
pumpkinseed	individual	70 -120	30	Lake Whatcom, 2000	Serdar et al., 2001
cutthroat trout	individual	60 - 80	30	Lake Whatcom, 2000	Serdar et al., 2001
brown bullhead	individual	70 - 440	30	Lake Whatcom, 2000	Serdar et al., 2001

N= Number of samples

### 9.1.13 Nickel

Nickel occurs naturally in rocks and soils and can leach into aquatic environments. However, weathering of nickel-containing substrates results in only small amounts of nickel entering into aquatic systems. Manmade sources of nickel include mining, combustion of coal, petroleum and tobacco, manufacture of cement and asbestos, food processing, textile and fur fabrication,



laundries, and car washes (USEPA, 1983). The National Academy of Sciences reports that fish contain nickel at a maximum of 1,700 µg/kg (NAS, 1975).

Nickel concentrations the maximum nickel concentration was 17,000 µg/kg in a whole body steelhead sample from the Klickitat River (study site 56). This sample was an anomaly since the other samples from this site were 170 and 520 µg/kg. The average concentrations in fillet samples ranged from 15 µg/kg in Pacific lamprey to 260 µg/kg in walleye; whole body ranged from 50 µg/kg in eulachon to 1200 µg/kg in Coho salmon.

#### **9.1.14 Selenium**

While selenium is ubiquitous in the earth's crust, only trace levels normally occur in aquatic environments. Selenium enters aquatic habitats from a number of anthropogenic and natural sources. Elevated levels in aquatic systems are found in regions where soil is selenium-rich or where soils are extensively irrigated (Dobbs et al., 1996). As an essential micronutrient, selenium is used by animals for normal cell functions. However, the difference between useful amounts of selenium and toxic amounts is small. Selenium at low levels in the diet is an essential element for humans. At elevated dose levels, it exhibits toxicity (selenosis). Organic and reduced forms of selenium (e.g. seleno-methionine and selenite) are generally more toxic and will bioaccumulate (Besser et al., 1993; Kiffney and Knight, 1990). Bioconcentration of selenium may be modified by water temperature, age of receptor organism, organ and tissue specificity, and mode of administration (Eisler, 1985a). Fish bioconcentrate selenium in their tissues with particularly high concentrations observed in ovaries when compared to muscle tissues (Lemly, 1985; Hamilton et al., 1990) and milt (Hamilton and Waddall, 1994). Selenium that is bioconcentrated appears to occur in its most harmful concentrations in predator species such as chinook salmon (Hamilton et al., 1990). Bioconcentration factors (BCFs) in rainbow trout range from 2-20 after exposure to 220-410 µg/L selenium. The magnitude of the BCFs appeared to be inversely related to exposure concentrations (Adams and Johnson, 1977). Biomagnification of selenium has also been well documented. The magnitude of the biomagnification ranges from 2-6 times between producers and lower consumers (Lemly and Smith, 1987). Piscivorous fish accumulate the highest levels of selenium and are generally one of the first organisms affected by selenium exposure, followed by planktivores and omnivores (Lemly, 1985).

Selenium has been frequently detected in a great variety of commonly consumed foods. In a 1997 UK diet study the mean selenium concentrations in the viscera and trimmings was estimated to be 490 µg/kg and 250 µg/kg in nuts (Ysart et al., 2000). Meat products (15%), fish (13%), and bread (13%) groups make the greatest contributions to diet (Ysart et al., 2000).

In the US infant diet the average concentration of selenium was highest in grains and cereals followed by fish (Table 9-17).

**Table 9-17. Selenium concentrations in US infant diet. (Source: Gartrell et al., 1985 and 1986).**

Food Group	1979 µg/kg	1981-1982 µg/kg
other dairy products	2	15
potatoes	2	2
beverages	2	
whole milk	4	9
vegetables	4	7
sugars and adjuncts	11	
oils and fats	12	5
meat, fish and poultry	107	112
grain and cereals	156	192

Selenium is well known to accumulate in living tissues. Selenium has been found in marine fish meal at levels of about 2,000 µg/kg, which is about 50,000 times greater than the selenium levels in seawater (Wilbur, 1980). Table 9-18 is a list of selenium concentrations in a variety of fish tissue types.

**Table 9-18. Concentrations of selenium in fish reported in the literature.**

Fish type	µg/kg	Location and date	Reference
<b>Mean</b>			
Razorback sucker eggs	3,700 - 10,600	Utah (1992)	Hamilton and Waddell, 1994
largemouth bass and bluegills gonads	2,630 - 4,640	power plant cooling reservoirs (1994)	Baumann and Gillespie, 1986
rainbow trout, edible portion	270	Toronto Harbor, Canada 1980	Davies, 1990
northern pike, edible portion	250	Toronto Harbor, Canada 1980	Davies, 1990
<b>Geometric mean</b>			
freshwater fish	560	112 selected US monitoring stations during from 1976-1979	Lowe et al., 1985
	460		
	470		
brown trout liver	6,290	South Platte River Basin in 1992 -93	Heiny and Tate, 1997
carp liver	8,130	South Platte River Basin in 1992 -93	Heiny and Tate, 1997
white sucker liver	17,900	South Platte River Basin in 1992 -93	Heiny and Tate, 1997
lake trout	500 to 860	Lake Huron from 1980 - 85	Great Lakes Water Quality Board, 1989
walleye and splake /backcross lake trout	650 to 790	Lake Huron 1980 - 85	Great Lakes Water Quality Board, 1989
walleye and splake /backcross lake trout	700 to 790	Lake Huron 1979 and 1985,	Great Lakes Water Quality Board, 1989
<b>Maximum</b>			
carp	3,650	Colorado River 1978 -79,	Lowe et al., 1985

The average concentrations of selenium in our study ranged from 220 µg/kg in a rainbow trout fillet to 1,100 µg/kg in the white sturgeon fillet (Table 2-14). The maximum concentration (2700 µg/kg) was in a white sturgeon fillet sample from the Hanford Reach of the Columbia River (study site 9U).

### 9.1.15 Vanadium

Vanadium is found in vegetables from about 0.5 to 2 µg/kg, with an average of about 1 µg/kg (Beyerrum, 1991). Veal and pork have been found to contain about 0.1 µg/kg. According to ATSDR (1992), foods containing the highest levels of vanadium include ground parsley, 1,800 µg/kg; freeze-dried spinach, 533 - 840 µg/kg; wild mushrooms, 50 - 2,000 µg/kg; and oysters, 455 µg/kg. Intermediate levels are found in certain cereals, like maize (0.7 µg/kg), and Macedonian rice 30 µg/kg). Also vanadium has been found in beef at 7.3 µg/kg, and in chicken at about 38 µg/kg. Seller and Sigel (1988) indicate that beverages, fats, oils, and fresh fruits and vegetables contained the least vanadium, ranging from less than 1 to about 5 µg/kg. Grains, seafoods, meats, and dairy products were generally from about 5 to 30 µg/kg. Prepared food ranged from 11 to 93 µg/kg, and dill seed and black pepper contained 431 and 987 µg/kg vanadium, respectively. ATSDR (ATSDR, 1992) indicates that in general, seafoods have been found to contain somewhat higher levels of vanadium than do tissues from terrestrial animals.

Mackeral has been found to contain about 3.5 µg/kg of vanadium, with 28 µg/kg in freeze-dried tuna (ATSDR, 1992). Konasewich et al. (1978) found vanadium in whole-fish samples of burbot and bloater chub taken from Lake Huron at concentrations of 75 µg/kg and 260 µg/kg, respectively. The same authors also found vanadium in whole samples of lake trout from Lake Superior, at 85 µg/kg. Nakamoto and Hassler (1992) found vanadium in the carcasses of male and female bluegill taken from the Merced River and the Salt Slough, California, at mean concentrations of 2,200 and 1,700 µg/kg, respectively.

In our study the average vanadium concentrations ranged from 5 µg/kg in fillet samples of spring chinook salmon and walleye to 310 µg/kg in whole body largescale sucker. The maximum concentration (770 µg/kg) was in a whole body rainbow trout composite sample from the Umatilla River (study site 101).

### 9.1.16 Zinc

Zinc occurs naturally in the earth's crust at an average concentrations of about 70,000 µg/kg. It is introduced into aquatic systems via leaching from igneous rocks. Zinc is found in all living organisms and is an essential element for growth, development and reproduction. However aquatic animals tend to accumulate excess zinc which can result in growth retardation, hyperchromic anemia, and defective bone mineralization. Because zinc combines with biomolecules in target species and most of these species accumulate more than they need for normal metabolism, data showing bioconcentration factors for target receptors may be misleading. Bioconcentration factors (BCF's) reported by EPA ranged from 51 in Atlantic salmon (*Salmo salar*) to 1,130 for the mayfly (*Ephemera grandis*) (USEPA, 1987c). Little to no evidence exists indicating the successive biomagnification of zinc in tissues of fish and avian receptors (USEPA, 1987c).

In the ATSDR survey of food groups the levels for zinc ranged from 29,200 µg/kg in fish/meat/poultry to 2,300 µg/kg in leafy vegetables (Table 9-19).

**Table 9-19. Concentrations of zinc in food groups. (Source: ATSDR, 1993)**

<b>Food Group</b>	<b>µg/kg</b>	<b>Food Group</b>	<b>µg/kg</b>
meat/fish/poultry	29,200	dairy products	4600
grain/cereals	8,700	legumes	8300
legumes	8,300	leafy vegetables	2300
legumes	8,300		

The average concentrations of zinc in whole body fish tissue from our study ranged from 3800 µg/kg in the white sturgeon fillet to 30,000 µg/kg in the whole body coho salmon (Table 2-14). The maximum concentration (40,000 µg/kg) was in the whole body mountain whitefish from the Deschutes River (study site 98).

## **9.2 Comparisons By Fish Species**

This section includes general descriptions of each of the chemicals measured in this study followed by brief comparisons of these chemicals with data reported in databases or other studies. More information about each chemical is provided in Appendix C (Toxicity Profiles). In addition to chemical descriptions, this section includes a summary of the life history of the fish species. This brief discussion of the habitat preferences and feeding habits is intended to provide some understanding of how the fish may be exposed to pollutants. Appendix B (Fish Life Histories) contains detailed information on each fish species.

The chemical levels measured in fish tissue from our study in largescale and bridgelip sucker, mountain whitefish, rainbow trout, channel catfish, smallmouth bass, fall and spring chinook, and coho were compared with levels reported in 4 databases and two other similar studies in the Columbia River Basin. Only those concentrations which had more than a 10 fold difference are discussed.

Information on white sturgeon, walleye, steelhead, eulachon, and Pacific lamprey was not found in these databases or reports. However their life histories and a synopsis of the literature information described in Section 9.1 are added to this section to complete the summary for all species from this study.

The 4 databases were developed by:

- 1) the USGS, National Contaminant Biomonitoring Program (NCBP) database (Schmitt et al., 1999a),
- 2) the USGS, Biomonitoring of Environmental Status and Trends (BEST) database (Schmitt et al., 1999b)
- 3) the State of Washington, Puget Sound Ambient Monitoring Program (PSAMP) (West et al., 2001 and
- 4) EPA's 1994 survey of literature reports on chemical data from the Columbia River

## Basin (USEPA 1994d)

The NCBP database includes data on persistent organochlorine insecticides, industrial chemicals, herbicides, and potentially toxic contaminants that may threaten fish and wildlife resources (Schmitt et al., 1999a). The NCBP database, from the early 1960's through 1986, contains measured values of average whole-body composite fish samples where each composite sample was comprised of five individual fish samples.

The BEST database includes data from the smallmouth bass sampled from the Mississippi River drainage during August-December 1995 (Schmitt et al., 1999b). Fish tissue data consisted of whole body composite samples, where, ideally, each composite sample consisted of 10 individual fish samples.

The PSAMP database consists of measured chemical concentrations in fillet (without skin) composites of adult chinook and coho salmon (West et al., 2001). Composite samples include 2-5 individual fish, with five individual fish per composite being the most common.

EPA's 1994 database includes a compilation of data from 1984 to 1994 on chemical concentrations in fish tissue and sediments from the Columbia River Basin. The information in the database includes individuals and agencies contacted, data sources, abstracts for contaminant studies, and an overview of future or ongoing studies (USEPA, 1994d).

The data from two surveys of chemicals in fish from the Columbia River Basin were also compared to fish tissue residues from our study:

- 1) The Lower Columbia River Bi-State Water Quality Program (Tetra Tech, 1996) and
- 2) Willamette River Human Health Technical Study (EVS, 2000)

The Lower Columbia River Bi-State Water Quality Program (Tetra Tech, 1996) characterized potential human health risks associated with consuming fish from the lower Columbia River, below the Bonneville Dam. The Bi-State study was conducted during two periods: 1991-1993 and 1995. Data from 1991-1993 consisted of data that measured chemical contaminant concentrations in fillet tissues of five different resident target fish species (largescale sucker, carp, peamouth, white sturgeon, and crayfish). Five individual fish were composited to form single composite samples. Data from 1995 included measured chemical concentrations in fillet fish tissue from largescale sucker, smallmouth bass, chinook salmon, and coho salmon. Fish tissue data for these species consists of range and mean data from three composite samples where each sample was made up of eight fish.

The Willamette River Human Health Technical Study (EVS, 2000) included data from four fish species of which smallmouth bass and largescale sucker were used for comparisons with our study. Data were compared for both fillet with skin and whole body tissue. All samples from the

Willamette study were composite samples formed by homogenizing tissue from five to eight individual fish.

### 9.2.1 Largescale Sucker (*Catostomus macrocheilus*) and Bridgelip Sucker (*C. columbianus*)

The largescale sucker is native to the Pacific Northwest in tributaries to the Pacific Ocean from the Skeena River in British Columbia to the Sixes River in Oregon (Scott and Crossman 1973). Largescale suckers are abundant throughout the Columbia River and are the most common resident fish species collected in the Hanford Reach (Gray and Dauble 1977).

Dauble (1986) found that algal periphyton was the major food item for fry, juvenile, and adult largescale suckers in the Columbia River. The stomachs of adults may also contain crustaceans, aquatic insect larvae, snails, fish eggs, sand, and bottom debris (Dauble 1986, Scott and Crossman 1973). Stream fish appear to feed upon more algae, diatoms, and aquatic insect larvae other than Chironomidae, whereas lake fish include Amphipoda and Mollusca (Carl 1936).

The bridgelip sucker is found in the Fraser and Columbia river basins from British Columbia to southeastern Oregon, including the Harney basin, below Shoshone Falls in the Snake River, and in northern Nevada (Scott and Crossman 1973, Lee et al. 1980). Throughout its range it coexists and hybridizes with the largescale sucker (*C. macrocheilus*) (Dauble and Buschbom 1981).

The life history and behavior of the bridgelip sucker are poorly understood. According to Scott and Crossman (1973), this fish usually inhabits small, swift, cold-water rivers with gravel to rocky substrates, whereas Wydoski and Whitney (1979) report it inhabits quiet backwater areas or the edges of the main current of rivers with sand or mud bottoms. In the Yakima River, Patten et al. (1970) found this fish in warm flowing waters. In the mid Columbia River during the day, Dauble (1980) found that subadult and adult bridgelip suckers were common in the tailouts of pools, at the end of riffles, and above boulders in the main current. At night, these fish were more abundant near shore in flowing water 0.6 to 1.5 m deep.

The diet of *C. columbianus* is almost entirely periphyton during all seasons. This fish has an expanded cartilaginous lower lip on its mouth that enables it to efficiently crop algae attached to the bottom. However, like almost all other suckers, this species also feeds to some extent on aquatic insect larvae and crustaceans (Dauble 1978, Wydoski and Whitney 1979). Mammals and some birds prey on this species (Scott and Crossman 1973).

Chemical concentrations in largescale sucker fish tissue were compared for arsenic, cadmium, copper, mercury, lead, selenium, zinc, p,p'-DDE, p,p'-DDT, Aroclor 1254, and Aroclor 1260 were compared data in the NCBP databases and the Bi-State and Willamette River studies (Table 9-20a).

While the metal concentrations in largescale sucker from our study were within the range of the other studies and databases examined, the maximum concentrations of metals were higher or

lower depending on the chemical (Table 9-20a). Cadmium concentrations were 25 times higher in our study than in the Willamette River study and National NCBP database. Lead in largescale sucker from our study was 9 times higher than in largescale sucker from the NCBP National database.

The organic chemical comparisons in largescale sucker were also quite variable (Table 9-20a). With exception of the Aroclors the organic chemical concentrations in our study were all within the range of the other databases and studies. However, the maximum concentrations were different. The maximum concentration of p,pDDE in largescale sucker was 9 times higher in our study than in the Bi-State study, and 14 times higher than in the NCBP Columbia River station 98.

The maximum Aroclor 1254 concentrations in largescale sucker were higher in the Columbia River NCBP stations (from 8x to 46x) than in our study. The detection limits were too high in the National NCBP database to discern a difference in Aroclor 1254 and our study.

With the exception of cadmium, the Willamette River study results for metals and organic chemicals were similar to our study.

The concentrations of chemicals in bridgelip sucker were within the range found in largescale sucker, except the largescale sucker had higher maximum concentrations (Table 9-20a,b).

**Table 9-20a. Comparison of chemical concentrations in composites samples of whole body largescale sucker.**

Station	USGS- NCBP- Columbia River Basin				USGS- NCBP		Willamette	Bi-State		EPA	
	Columbia	Columbia	Columbia	Snake	National	single composite		mean	max	ave	range
	(46)	(47)	(98)	(41,42,96)							
Chemical	range µg/kg	range µg/kg	range µg/kg	range µg/kg	range µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	
Arsenic	<50 - 870	130 - 290	111 - 333	<50 - 260	40 - 270	120	8	385	160	74- 320	
Cadmium	<50 - 160	<50 - 600	50 - 410	<50 - 260	<5 - 9	10	37	66	55	13-250	
Copper	850 - 1340	1070 - 1283	720 - 1150	490- 4318	600 - 1010	1780	912	1230	1400	800-5600	
Lead	90 - 390	100 - 520	160 - 2570	10 - 290	20 - 120	37	171	860	170	27-1100	
Mercury	50 - 320	<10 - 160	20 - 130	10 - 230	10 - 370	121	122	264	130	<58-250	
Selenium	60 - 430	60 - 386	190 - 250	170 - 450	80 - 340	ND	132	260	310	<180-500	
p,p'-DDE	20 - 2000	20 - 1100	10-90	50 - 560	10 - 970	835	59	150	370	28-1300	
p,p'-DDT	10 - 270	10 - 430	10-70	10 - 440	10 - 190	190	10	56	33	<1-180	
Aroclor 1254	100 - 2100	5 - 3000	100 - 600	<5 - 500	<100	53	176	270	30	<14-65	
Aroclor 1260	100 - 700	<5 - 100	100 - 300	<5 - 300	<100 - 300	36	35	1300	38	<12-100	

Min= minimum; Max = maximum, Ave = average <= detection limit

NCBP = USGS National Contaminant Biomonitoring Program 1969-1986. Range of average whole body composites. Station numbers are in parentheses.

Willamette = composites without replication, EVS, 2000.

Bi-State = whole body concentrations of fish collected during 1991-1993 from the lower Columbia River, below Bonneville Dam. Mean and maximum (max) TetraTech, 1996

EPA- Our study = range of composite fish samples from sites in the Columbia River Basin. See table 1-1 and 1-2 for description of sites.



**Table 9-20b . Comparison of ranges of chemical concentration in composite samples of whole body bridgelip sucker.**

Station Chemical	USGS - NCBP- Columbia River Basin			NCBP	EPA
	Salmon (43) <i>µg/kg</i>	Snake (96) <i>µg/kg</i>	Columbia (98) <i>µg/kg</i>	National <i>µg/kg</i>	Our Study <i>µg/kg</i>
Arsenic	160 - 330	No Data	180 - 270	60	260 - 300
Cadmium	20 - 50	No Data	70 - 280	<50 - 60	22 - 32
Copper	680 - 1900	No Data	No Data	No Data	880 - 1800
Lead	100 - 220	No Data	530 - 1000	<100 - 110	37 - 78
Mercury	40 - 80	120	20 - 70	80 - 160	<40 - 53
Selenium	200 - 470	No Data	200 - 260	No Data	280
p,p''-DDE	10 - 30	340 - 440	<10 - 40	200 - 350	310 - 560
p,p''-DDT	<10 - 20	190 - 200	<10 - 40	180 - 380	37 - 52
PCB1254	<100	<100 - 500	<100	1000 - 2800	18 - 32
PCB1260	<100	<100	<100 - 4800	No Data	27 - 49

< = detection limit

NCBP = USGS National Contaminant Biomonitoring Program 1969-1986 Range of average whole body composites. Station numbers are in parentheses.

EPA- Our Study = range of composites from the Yakima River (study site 48).

### 9.2.2 Mountain Whitefish (*Prosopium williamsoni*)

The mountain whitefish is native to cold water rivers and lakes in western North America, both east and west of the Continental Divide (Scott and Crossman 1973). Seven-year old fish range in length and weight from 307 to 387 mm and from 475 to 890 g, respectively, while the ranges for 8-year old fish are 330 to 410 mm and 501 to 944 g (Scott 1960, Pettit and Wallace 1975, Thompson and Davies 1976). Mountain whitefish feed primarily on immature forms of bottom-dwelling aquatic insects such as Diptera (true flies and midges), Trichoptera (caddisflies), Ephemeroptera (mayflies), and Plecoptera (stoneflies) (Wydoski and Whitney 1979, Cirone et al. 2002).

The ranges of chemical concentrations in the whole body mountain whitefish, from the present study were compared with mountain whitefish data from the NCBP database (Table 9-21). There was no consistent pattern between the metal concentrations in our study of mountain whitefish and NCBP database (Table 9-21). The maximum arsenic and cadmium levels were similar in our study and the NCBP database. The maximum copper concentrations in mountain whitefish in our study were 6 to 9 times higher than the concentrations in the NCBP database. Lead concentrations were higher in the NCBP database. The maximum mercury levels measured in the Salmon River in NCBP database were higher than the levels measured in our study; the levels in the NCBP Snake River mountain whitefish were lower. The maximum selenium concentrations were lower in the NCBP database than in our study.

The maximum p,p' DDE concentrations in mountain whitefish in our study were 700 times higher than the concentrations in mountain whitefish from the NCBP Salmon River station. The Aroclor concentrations were not comparable because of the higher detection limits in the NCBP database.

**Table 9-21. Comparison of ranges chemical concentrations in composite samples of whole body mountain whitefish.**

Station	USGS -NCBP - Columbia River Basin			EPA
	Salmon (43)	Snake (96)	Columbia (97)	Our Study
Chemical	$\mu\text{g}/\text{kg}$	$\mu\text{g}/\text{kg}$	$\mu\text{g}/\text{kg}$	$\mu\text{g}/\text{kg}$
Arsenic	120	No data	No data	120 - 180
Cadmium	40	No data	No data	<4 - 54
Copper	840	590	No data	620 - 5000
Lead	100	103	No data	10 - 72
Mercury	290	65	190	<47 - 130
Selenium	680	472	No data	590 - 1800
p,p'-DDE	<10	590	1410	13 - 770
p,p'-DDT	20	30	350	<2 - 49
Aroclor 1254	<100	100	<100	<21 - 140
Aroclor 1260	<100	100	100	<18 - 130

<= detection limit

NCBP = USGS National Contaminant Biomonitoring Program 1969-1986. Range of average whole body composites. Station numbers are in parentheses.

EPA- Our Study = range of composite fish samples from sites in the Columbia River Basin. See table 1-1 and 1-2 for description of sites

### 9.2.3 White Sturgeon (*Acipenser transmontanus*)

White sturgeon is native to the Pacific Northwest where it has evolved life history characteristics that have allowed them to thrive for centuries in large, dynamic river systems containing diverse habitats. These characteristics include opportunistic food habits, delayed maturation, longevity, high fecundity, and mobility (Beamesderfer and Farr 1997). White sturgeon may attain lengths and weights of more than 6 m and 580 kg, respectively, during a life span of over 100 years (Scott and Crossman 1973). White sturgeon body weight ranged from 9 to 34 kg.

White sturgeon take advantage of scattered and seasonal food sources by moving between different riverine habitats. They feed on a wide range of food items including zooplankton, molluscs, amphipods, aquatic larvae, benthic invertebrates, and fish (McCabe et al. 1993). White sturgeon are more predaceous than any other North American sturgeon (Semakula and Larkin 1968) and can capture and consume large prey (Beamesderfer and Farr 1997). Seasonal migrations occur in the Lower Columbia River where sturgeon move to feed on eulachon (*Thaleichthys pacificus*), northern anchovy (*Engraulis mordax*), American shad (*Alosa sapidissima*), moribund salmonids, amphipods, and other invertebrates (DeVore et al. 1995).

Concentrations of the Aroclors and 2,3,7,8-TCDF and in white sturgeon from our study of the Columbia River Basin were higher than the EPA 1994 (USEPA, 1998c) studies of Lake Roosevelt, Washington (Tables 9-9 and 9-10).

### 9.2.4 Walleye (*Stizostedion vitreum*)

The original range of the walleye generally east of the Rocky Mountains was expanded when it was introduced to the Columbia River below Roosevelt Dam in the 1940's or 50's (Wydoski and Whitney 1979). This species shows a preference for large, semi-turbid waters, but is capable of inhabiting a large range of physical and chemical conditions (Colby et al. 1979).

Feeding usually occurs near or at the bottom, and walleye may move into shallow water to feed. Walleye fry feed on rotifers, copepods, and cladocerans. Juvenile and adult walleye are largely piscivorous, but invertebrates (e.g., mayfly nymphs and amphipods) may be a large part of their diet in the late spring and early summer. Cannibalism is common with this species (Colby et al. 1979, Eschmeyer 1950). Prey for this species in the Columbia River includes mainly cottids, cyprinids, catostomids, and percopsids; out migrating juvenile salmonids were a smaller part of their diet (Zimmerman 1999).

Adult walleye are not usually preyed upon by other fish. However, in its native range northern pike and muskellunge do prey on this fish (Colby et al. 1979). They are also probably preyed upon by fish eating birds and mammals (Sigler and Sigler 1987).

The maximum concentration of Aroclors 1254 and 1260 and 2,3,7,8-TCDF in walleye were lower in our study of the Columbia River Basin than levels found in surveys of Lake Roosevelt, Washington, (USEPA, 1998c; Munn, 2000) (Tables 9-9 and 9-10).

### **9.2.5 Channel catfish (*Ictalurus punctatus*)**

The original range of the channel catfish, east of the Rock Mountains was expanded when it was introduced to Idaho waters in 1893, but the date of its introduction to Washington waters is unknown (Wydoski and Whitney 1979, Simpson and Wallace 1982).

Young channel catfish tend to feed primarily on aquatic insects and bottom arthropods, but after attaining about 100 mm in length they are usually omnivorous or piscivorous (Carlander 1969). Adult channel catfish consume a wide variety of plant and animal material including clams, snails, crayfish, pondweed, and small terrestrial vertebrates (Eddy and Underhill 1976, Moyle 1976).

Young channel catfish are prey to a variety of fishes and piscivorous birds but the adults, due to their size and bottom occurrence, are probably free of predation (Scott and Crossman 1973, Schramm et al. 1984).

The concentrations of chemicals measured in channel catfish our study were compared to levels reported in the NCBP database (Table 9-22). The concentrations of metals were higher in the National and Columbia Basin NCBP databases with two exceptions. The maximum concentrations of arsenic and selenium concentrations in channel catfish were 10 times higher in our study than the NCBP Willamette station. The concentrations of the following metals were higher in the NCBP national database: cadmium 29x, lead 60x, mercury 14x, and selenium 4 times higher.

The concentrations of organic chemicals were higher in the NCBP National database than in our study. The maximum concentrations of the following chemicals in channel catfish from the National NCBP database were higher than the levels in channel catfish in our study: p,p'DDE 47x, p,p'DDT 166x, Aroclor 1260 672x, and Aroclor 1260 42 times higher. The concentrations

of p,p' DDT in the NCBP Columbia Basin stations were 5 - 23 times higher than in our study. The maximum concentrations of Aroclor 1254 in channel catfish was from the NCBP Columbia Basin Stations were 24 to 76 times higher than in our study.

**Table 9-22. Comparison of ranges of chemical concentrations in whole body channel catfish tissue from our study with the USGS-NCBP database.**

Station	USGS - NCBP			EPA	
	Willamette (45)	Snake (96)	National	Our Study	
Chemical	$\mu\text{g}/\text{kg}$	$\mu\text{g}/\text{kg}$	$\mu\text{g}/\text{kg}$	ave	$\mu\text{g}/\text{kg}$
Arsenic	<50	<50 - 610	10 - 630	230	110 - 430
Cadmium	<50	<50	3 - 760	17	13 - 26
copper	no data	no data	no data	510	410 - 590
Lead	100	<100 - 210	30 - 2000	21	12 - 33
Mercury	290	80 - 900	<10 - 4500	210	140 - 320
Selenium	60	70 - 180	<50 - 2500	500	410 - 630
p,p'-DDE	570	<10 - 1050	10 - 42300	570	280 - 900
p,p'-DDT	<10 - 1050	<10 - 220	<5 - 7500	21	0.8 - 45
Aroclor 1254	4400	<10 - 1400	<50 - 39000	38	25 - 58
Aroclor 1260	No Data	<100 - 500	<50 - 5900	77	32 - 140

\*Samples are fillet with skin;

Ave= average

NCBP = USGS National Contaminant Biomonitoring Program 1969-1986. Range of average whole body composites. Station numbers are in parentheses.

EPA-Our Study = whole body composite samples from the Columbia River (study site 8) and the Yakima River (study site 8)

### 9.2.6 Smallmouth Bass (*Micropterus dolomieu*)

The range of the smallmouth bass, originally restricted to freshwaters of eastern-central North American, was expanded by plantings in the Pacific Northwest in the late 1800s and early 1900s. In Washington, smallmouth bass are most numerous in the Columbia and Snake rivers (Wydoski and Whitney 1979, Simpson and Wallace 1982).

Smallmouth bass fry initially eat copepods and cladocerans and at lengths of 2 to 5 cm change to a diet of insects and small fish (Hubbs and Bailey, 1938). Tabor et al. (1993) found that salmonids made up from 4 to 59% (by weight) and from 19 to 30% (by volume) of the diet of smallmouth bass in the Columbia River Basin. The authors concluded that predation rates on salmonids were high during the spring and early summer when subyearling salmon were abundant and of suitable forage size and shared habitat with the smallmouth bass.

Smallmouth bass in the Columbia River grow at a rate equal to or better than that of bass from other locations in the United States. In a 1952 study, the weights and total lengths of the Columbia River fish at age four were 510 g and 32 cm; age six, 794 g and 38 cm; age eight, 1,304 g and 43 cm; and at age ten, 1,814 g and 47 cm, respectively (Henderson and Foster 1957, Wydoski and Whitney 1979). The body weight of smallmouth bass in our study ranged from 1300 to 1400 g.

Smallmouth bass from our study were compared to data reported in the BEST and NCBP databases (Table 9-23). The concentrations of all chemicals in smallmouth bass from the NCBP National database were higher than in our study. In particular, Aroclor 1254 was higher (68x) in

the NCBP National database. The Aroclor concentrations in Columbia River Basin NCBP stations had higher detection limits than in our study.

**Table 9-23. Comparison of ranges of chemical concentrations in whole body smallmouth bass.**

Chemical	USGS- NCBP					USGS	EPA
	Yakima (44)	Snake (42)	Salmon (43)	Willamette(45)	National	BEST	Our Study
Chemical	$\mu\text{g/kg}$	$\mu\text{g/kg}$	$\mu\text{g/kg}$	$\mu\text{g/kg}$	$\mu\text{g/kg}$	$\mu\text{g/kg}$	$\mu\text{g/kg}$
Arsenic	No data	50 - 60	<30 - 50	250	40 - 670	<178 - 263	160 - 170
Cadmium	No data	10 - 50	6 - 60	50	2 - 50	<36 - 43	5 - 19
Copper	No data	380	1182	No data	257 - 1950	445 - 591	500 - 560
Lead	No data	<100	100 - 170	120	10 - 320	8 - 100	10 - 140
Mercury	140 - 270	150 - 280	210 - 360	130	60 - 1200	80 - 280	220 - 360
Selenium	No data	440	606 - 830	No data	80 - 1260	203 - 491	480 - 710
p,p'-DDE	940 - 1660	80 - 2540	280 - 690	60	10 - 950	10 - 65	970 - 1700
p,p'-DDT	200 - 420	80 - 170	80 - 170	20	<5 - 590	10 - 84	44 - 80
Aroclor 1254	100 - 600	<100	<50 - 400	<400	<50 - 6400	No data	46 - 94
Aroclor 1260	200	<100 - 800	<50 - 100	<200	<50 - 1300	No data	80 - 190

NCBP = USGS National Contaminant Biomonitoring Program 1969-1986. Range of average whole body composites. Station numbers are in parentheses.

BEST = USGS Biomonitoring of Environmental Status and Trends Program - 1995 Fish Samples from the Mississippi Delta.

EPA- Our Study = whole body composite samples from the Yakima River (study site 48)

### 9.2.7 Rainbow and Steelhead (*Oncorhynchus mykiss*)

*Oncorhynchus mykiss* are native to the Pacific Northwest and appear in two forms: the resident rainbow trout and the anadromous steelhead, both of which occur in the Columbia River Basin. It also has the greatest diversity of life history patterns of any Pacific salmonid species (Wydoski and Whitney 1979, Pauley et al. 1986). This diversity includes degrees of anadromy, differences in reproductive biology, and plasticity of life history between generations (Peven 1990, Busby et al. 1996).

The diet of rainbow trout and juvenile steelhead changes seasonally, depending on food availability. They may feed on aquatic insects, amphipods, leaches, snails, and fish eggs. The steelhead's diet in the ocean includes crustaceans, squid, herring, and other fish (Withler, 1966; Wydoski and Whitney, 1979). Adult non-migratory rainbow trout average 0.9 to 1.8 kg in weight and usually have a life span of 5 to 6 years (Simpson and Wallace, 1982; Sigler and Sigler, 1987). Steelhead can achieve 9 years of age, weights of 16 kg, and lengths to 122 cm (Scott and Crossman, 1973; Wydoski, and Whitney, 1979). The average body weight of rainbow trout in our study ranged from 47 - 571g. The steelhead average body weight ranged from 1633 to 6440g.

The chemical residues in rainbow trout measured in our study were compared to the NCBP databases (Table 9-24). The maximum concentration of p,p' DDE in rainbow trout was 300 times higher in the NCBP Columbia River Basin station (Snake River) than in our study.

Steelhead concentrations of metals in fish tissue were within the range of rainbow trout (Table 9-24). The maximum concentrations of arsenic and lead were higher (4x and 2x respectively) in the steelhead, while p,p'DDE was lower in the steelhead than the rainbow trout.

**Table 9-24. Comparison of ranges of chemical concentrations in composite samples of whole body rainbow trout.**

Station Chemical	USGS - NCBP		EPA ( Our Study)	
	Snake (41) µg/kg	National µg/kg	rainbow trout µg/kg	steelhead
Arsenic	<50 - 145	<50 - 260	<50 - 560	290 - 1200
Cadmium	5 - 50	10 - 70	<4 - 58	29 - 88
Copper	680 - 3130	1130 - 4620	900 - 5000	1900 - 6800
Lead	9 - 100	10 - 650	<10 - 88	<10 - 360
Mercury	30 - 130	10 - 270	<33 - 380	<50 - 420
Selenium	220 - 540	170 - 3000	230 - 790	460 - 940
p,p'-DDE	80 - 25400	10 - 140	3 - 84	5 - 33
p,p'-DDT	5 - 70	5 - 40	<2 - 12	<1 - 6
Aroclor 1254	100 - 600	<50 - 300	<10 - 20	9 - 29
Aroclor 1260	<50	<50 - 100	<6 - 22	<6 - 21

NCBP = USGS National Contaminant Biomonitoring Program 1969-1986. Range of average whole body composites. Station numbers are in parentheses.

EPA- Our study = range of composite fish samples from sites in the Columbia River Basin. See table 1-1 and 1-2 for description of sites.

### 9.2.8 Chinook Salmon (*Oncorhynchus tshawytscha*)

Chinook salmon are the largest of the Pacific salmon and have a variable life history. Timing of migration and spawning, and the duration of freshwater, estuarine, and ocean residencies varies for this species (Meehan and Bjornn 1991). 'Stream-type' and 'ocean-type' chinook are the two main races. Stream-type chinook are also referred to as spring or summer chinook salmon, and ocean-type as fall chinook salmon. Most (78%) of the chinook salmon in the Columbia River are ocean-type and they spawn from mid-September to late December. Ocean-type juveniles migrate to the estuary at 3 to 6 months of age when they are 70 to 90 mm in length (Meehan and Bjornn 1991). In the estuary, these juveniles prefer low banks and subtidal refuge areas and their diet consists of insect and crab larvae and small fish (Healey 1991). Stream-type juveniles overwinter in freshwater before out migrating as yearlings from April to June. Some will spend two winters in freshwater. Deep pools with rock crevices provide over wintering habitat. In freshwater, juvenile diet is primarily insects, both aquatic larvae and terrestrial adults. During outmigration, yearling smolts spend a brief period in the estuary where they occupy the outer part of the estuary, thus, their habitat does not overlap with the smaller ocean type chinook (Healey 1991).

Chemical concentrations of metals and organic chemicals measured in fall chinook salmon from our study of the Columbia River Basin were compared to fall chinook salmon measurements in PSAMP database and the Bi-State study (Table 9-25).

The concentration of arsenic in chinook salmon was similar in our study, PSAMP, and the EPA 1994 database, while the Bi-State arsenic concentrations were lower (48x for fall chinook salmon; 52x for spring chinook salmon). The cadmium levels in chinook salmon were higher (13x fall chinook salmon; 3x spring chinook salmon) in the EPA 1994 database than our study. The maximum lead concentrations were higher in the spring chinook salmon in our study than in the Bi-State study (14x). Fall chinook and spring chinook salmon from our study had higher concentrations of Aroclor 1254 than the Bi-State study (35x and 24x, respectively).

The chemical concentrations in fall and spring chinook salmon from our study were similar to each other with the exception of cadmium, lead, and mercury which were higher in spring chinook (15x, 8x, and 5x, respectively; Table 9-25).

**Table 9-25. Comparison of chemical concentrations in chinook salmon fillet with skin.**

Station	EPA 1994		EPA					
	Database	PSAMP	Bi-State		Our Study			
	range µg/kg	range µg/kg	ave µg/kg	max µg/kg	fall chinook salmon		spring chinook salmon	
Chemical					ave µg/kg	range µg/kg	ave µg/kg	range µg/kg
Arsenic	20 - 1110	570 - 1600	13	23	810	530 - 1100	850	560 - 1200
Cadmium	20 - 50	No data	2	2.5	<2	<4	2	<4 - 15
Copper	240 - 1900	370 - 1200	860	1010	640	540 - 760	790	240 - 1000
Lead	20 - 40	no data	7	10	7	<10 - 16	14	<10 - 140
Mercury	62 - 164	58 - 160	100	130	84	<50 - 150	100	<83 - 510
Selenium	360 - 370	no data	280	340	330	280 - 380	350	290 - 430
p,p'-DDE	no data	4 - 48	8.5	11	12	4 - 26	12	6 - 18
p,p'-DDT	3	0.5 - 4	1.5	3	2.5	<2 - 8	4	3 - 8
Aroclor 1254	18 - 20	5 - 88	0.9	0.9	17	9 - 35	16	9 - 24
Aroclor 1260	16 - 30	1 - 72	10	15	9.9	<19	11	<18
2,3,7,8-TCDD	0.00014	no data	0.0002	0.0006	0.00002	<0.00001-0.00005	0.00002	<0.00001-0.00005
2,3,7,8-TCDF	0.0009	no data	0.0016	0.00027	0.00068	<0.00003-0.0014	0.0006	0.0004-0.00074

Ave = average; max = maximum <= detection limit

EPA 1994 database = EPA survey of data from the Columbia River Basin from 1983-1994. Does not differentiate between spring and fall chinook salmon

Bi-State = 1995 concentrations in fillets of fish from the lower Columbia River, below Bonneville Dam. Does not differentiate between fall and spring chinook salmon (Tetra Tech, 1996).

PSAMP = 1992-1995, data is for fillet without skin. Does not differentiate between fall and spring chinook salmon

EPA- Our study = range of composite fish samples from sites in the Columbia River Basin. See table 1-1 and 1-2 for description of sites

### 9.2.9 Coho Salmon (*Oncorhynchus kisutch*)

Coho salmon are one of the five Pacific salmon species in North America. The life span of most coho is three years, during which they attain average weights ranging from about 3,000 to 6,000g (Wydoski and Whitney 1979). The average body weight of the coho salmon in our study was 2,855g to 3,960g.

The coho salmon fish typically spend up to 21 months in freshwater followed by approximately 16 months in the ocean before returning to freshwater where they will spawn and die. These fish rarely feed on non-moving food or off the bottom in streams (Sandercock 1991). Juveniles consume insects (larvae, pupae, and adults), worms, small fish, and fish eggs. In reservoirs, coho juveniles feed primarily on zooplankton and emerging insects (Wydoski and Whitney 1979).

Samples of coho salmon from our study were compared to data from PSAMP and the Bi-State study (Table 9-26). The maximum concentrations of several chemicals were higher in coho salmon from our study than the coho salmon from the Bi-State study: arsenic (85x), lead (25x), and Aroclor 1254 (19x).

**Table 9-26. Comparison of chemical concentrations in coho salmon fillet with skin.**

Station	PSAMP	Bi-State		EPA - Our study	
	range	mean	max	ave	range
Chemical	$\mu\text{g}/\text{kg}$	$\mu\text{g}/\text{kg}$	$\mu\text{g}/\text{kg}$	$\mu\text{g}/\text{kg}$	$\mu\text{g}/\text{kg}$
Arsenic	570 - 1600	2.7	7	540	450 - 600
Cadmium	No data	3	5		<4
Copper	410 - 1010	810	850	1700	680 - 3600
Lead	No data	4	9	81	<10 - 230
Mercury	58 - 160	44	48	120	110 - 120
Selenium	No data	168	188	290	270 - 310
p,p'-DDE	1.3 - 26	3	5	33	29 - 35
p,p'-DDT	0.52 - 1.4	0.8	1	2	<2 - 4
Aroclor 1254	2 - 66	0.6	0.9	16	12 - 19
Aroclor 1260	1 - 32	3	4		<18
2,3,7,8-TCDD	No data	0.0003	0.0009	0.000017	<0.00001 - 0.00004
2,3,7,8-TCDF	No data	0.0007	0.0009	0.0005	0.0004 - 0.0005

Ave = average; max = maximum; < = detection limit

PSAMP = 1992-1995, data is for fillet without skin

Bi-State = 1995 whole body concentrations of fish from the lower Columbia River, below Bonneville Dam. (TetraTech, 1996)

EPA - Our study = range of composite fish samples from sites in the Columbia River Basin. See table 1-1 for site descriptions.

### 9.2.10 Pacific Lamprey (*Lampetra tridentata*)

The Pacific lamprey is a native anadromous fish with a widespread distribution in the Columbia River Basin (Wydoski and Whitney 1979).

The adults overwinter in freshwater, do not feed during this time, and spawn the following spring (Beamish 1980). Larvae (ammocoetes) leave the gravel approximately 2 to 3 weeks after hatching, drift down current, settle in slow back water areas, burrow in soft substrates with organic debris, and take up a filter feeding existence (Pletcher 1963, Kan 1975). The ammocoete life stage may range from 4 to 7 years, during which time they remain buried in the sediment (Beamish and Levings 1991, Close et al. 1995). Ammocoetes are reported to feed on vegetative material (Clemens and Wilby 1967), diatoms and desmids (Pletcher 1963), and detritus and algae suspended above and within the substrate (Moore and Mallatt 1980). Juvenile lampreys play an important role in the diets of many freshwater fishes, including channel catfish, northern pike minnow, and several species of cyprinids and cottids. Salmonid fry prey upon lamprey eggs, but do not feed on the ammocoetes. The larvae are also taken by several species of gulls and terns (Pletcher 1963, Close et al. 1995).

Metamorphosis occurs from July to October. Shortly thereafter, the downstream migration of young adult lampreys begins usually at night and with an abrupt increase in river flow. Pacific lampreys migrate to salt water where they take up a parasitic life, but feeding may start in freshwater (Pletcher 1963, Beamish 1980, Beamish and Levings 1991).

The ocean phase of the adult life cycle may last 3.5 years (Beamish 1980). In ocean and estuarine areas, adults are important prey for several pinniped species. After entering the Columbia River they become a prey item for white sturgeon (Wydoski and Whitney 1979, Roffe and Mate 1984, Close et al. 1995).



There were no comparable studies of Pacific lamprey in the literature.

### **9.2.11 Eulachon (*Thaleichthys pacificus*)**

The eulachon occurs only on the west coast of North America, including the Columbia River Basin (Scott and Crossman 1973). This anadromous species spawns in the main channel of the Columbia River and periodically in the Grays, Cowlitz, Kalama, Lewis, and Sandy Rivers (Smith and Saafeld 1955).

It is believed that developing larvae do not feed in freshwater, but rely on their yolk sac for nourishment until they reach the ocean (Smith and Sallfeld 1955, Scott and Crossman 1973). At sea, post-larval eulachon move into deeper water as they grow. They feed on plankton, mysids, ostracods, copepods and their eggs, and barnacle, cladoceran, and polychaete larvae (Hart 1973). Juvenile and adult fish feed primarily on euphausiid shrimp, crustaceans, and cumaceans. Adults do not feed after they return to freshwater (Barraclough 1964).

As are other smelts, *T. pacificus* is a very important food item for a wide variety of predators. Adults are fed on by many piscivorous fishes including Pacific salmon and white sturgeon, marine mammals ranging from the harbor seal to the finback whale, seabirds, waterfowls, and gulls (Scott and Crossman 1973). The larval and post larval stages contribute modestly to the diet of small salmon off the Fraser River (Hart 1973).

There were no comparable studies of eulachon in the literature.

## **9.3 Comparisons across all species**

### **9.3.1 Resident Fish**

White sturgeon, mountain whitefish, whole body walleye, largescale sucker, smallmouth bass, and channel catfish had the highest concentrations of organic chemicals of all the species tested in this study (Table 9-27a,b). Bridgelip sucker and walleye fillet samples had much lower chemical residues, similar to the salmonids and eulachon.

The largescale sucker was the fish species with the most frequent detection of PAHs (Table 2-1a). The phenols were detected in only one white sturgeon sample from the main-stem Columbia River (study site 8) (Table 2-1a).

The basin-wide average concentrations of total DDT (Table 2-4) in the salmonids (chinook, coho, rainbow trout, and steelhead ) and eulachon were much lower than, white sturgeon, mountain whitefish, largescale sucker, and smallmouth bass. The maximum concentrations p,p'DDE was found in whole body smallmouth bass followed by white sturgeon fillet, channel catfish fillet, and whole body largescale sucker (Table 9-27a).

The white sturgeon, mountain whitefish, whole body walleye, and smallmouth bass had the

highest concentrations of Aroclors. The maximum concentration of TCDF was in the white sturgeon (Table 9-27a,b). The next highest average concentration was in the mountain whitefish.

The maximum concentrations of metals (arsenic, cadmium, copper, lead, mercury, selenium) were lower in the resident species than in the anadromous species, except for largescale sucker which had the highest concentration of cadmium (Table 9-27a,b). When doing a comparison of fish tissue across all species it is important to not only consider the maximum concentrations but also some measure of the variability. In this study, the average concentration is a measure of variability. While the maximum mercury and selenium concentrations were in the spring chinook salmon, the basin-wide average concentrations of mercury were highest in the largescale sucker, walleye, and white sturgeon.

The higher concentration of organic chemicals may be attributed to size in some species or lipid content. The white sturgeon were some of the largest fish measured in the study. The samples included only single fish. It is also known to have a very long life span. Thus, it is not clear whether the high levels of organic chemicals in this fish may be due to an anomaly in the few fish that were sampled, their size, or their age.

The association of organic chemical concentrations in the tissues of resident species and percent lipid was not particularly evident in this study. There was an association with lipid in the white sturgeon samples from one study site (study site 6). The difference in chemical content between the whole body walleye and the fillet was also associated with lipid. However, there were no other clear associations of whole body and fillet with lipid and organic chemicals in fish tissue.

There was an indication of high concentrations of organic chemicals in the resident fish collected from the Hanford Reach of the Columbia River (study site 9U). However, there is no information in this study to explain the levels in fish from this study site.

### **9.3.2 Pacific lamprey and eulachon**

Of the anadromous fish species, Pacific lamprey had maximum concentration of organic chemicals (DDE and Aroclor 1254; Table 9-27b). The high concentration of organic chemicals in the Pacific lamprey may have been due to its high lipid content.

The metals content of the Pacific lamprey was not consistent across different metals. For example when compared to the other anadromous species, the arsenic concentrations were low for Pacific lamprey while concentrations of copper, lead, mercury, and selenium were within the range of the range of these other fish species.

While eulachon also had a high lipid content, they had some of the lowest levels of organic chemicals of all the species test. Aroclors and chlordane were not detected in the eulachon. Eulachon had the highest average concentration of arsenic and lead.

### 9.3.3 Salmonids

The salmonids had the lowest concentrations of organic chemicals with a few exceptions. There were no semi-volatile chemicals detected in the fall chinook salmon or coho salmon tissue samples. Pyrene was found at the highest concentrations of all the PAHs in a rainbow trout collected from the upper Yakima River (study site 49). The fillet or whole body samples of rainbow trout, eulachon, and coho salmon had no detectable concentrations of any of the chlordanes compounds.

The concentrations of metals in the chinook salmon and steelhead were higher than the other resident or anadromous fish species. Steelhead had the maximum concentration of arsenic. When doing a comparison of fish tissue across all species it is important to not only consider the maximum concentrations but also some measure of the variability. In this study, the average concentration is a measure of variability. Thus, while steelhead had the maximum concentration of arsenic, the average concentrations were higher in eulachon, and chinook salmon (Table 2-14). From this study, the salmon, steelhead, and eulachon had higher concentrations of arsenic than the resident species and Pacific lamprey. Fall chinook salmon had the maximum concentration of lead (Table 9-27b). The average concentrations of lead were highest in eulachon, fall chinook salmon, and whole body walleye (Table 2-14).

Although the egg samples from the salmon and steelhead had high percent lipid, the concentration of organic compounds was generally lower than the fish tissue of the anadromous or resident fish with a few exceptions. The highest concentrations of total chlordanes were in egg samples from the spring chinook salmon. The maximum concentrations of copper and selenium were in egg samples from the salmon and steelhead (Table 9-27b). The basin-wide average concentrations of copper were highest in the egg samples from the salmon and steelhead followed by the whole body Pacific lamprey. The basin-wide average concentrations for selenium were highest in spring chinook salmon egg samples followed by white sturgeon and mountain whitefish. The high concentration of selenium may also be associated with the high percent lipid in the egg samples.

**Table 9-27a. Range of chemical concentrations in resident fish tissue samples from our study of the Columbia River Basin, 1996-1998.**

Chemical	T	largescale	Bridgelip	rainbow	mountain	white	walleye	channel	smallmouth
		sucker	sucker	trout	whitefish	sturgeon**		catfish	bass
		µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg	µg/kg
<i>N-FS</i>		19		7	12	16	3	5	
<i>N-WB</i>		23	3	12	12	8	3	6	
Arsenic	FS	50 - 100	NS	<50	51 - 140	150 - 640	290 - 400	50 - 330	110 - 170
	WB	74 - 320	260 - 300	<50 - 560	120 - 180	<200 - 640	480 - 510	110 - 430	160 - 170
Cadmium	FS	<4 - 24*	NS	<4 - 5*	<4 - 14*	<4 - 6*	<4	ND	ND
	WB	13 - 250	22 - 32	<4 - 58	4 - 54	15 - 95	100 - 110	13 - 26	5 - 19
Copper	FS	430 - 870	NS	440 - 610	510 - 840	<210 - 410	500 - 600	310 - 360	510 - 560
	WB	800 - 5600	880 - 1800	900 - 5000	620 - 5000	260 - 1800	730 - 5700	410 - 590	500 - 560
Lead	FS	10 - 140	NS	<10	<10 - 26	<10 - 29*	<10	10 - 11*	10 - 55
	WB	27 - 1100	37 - 78	<10 - 88	10 - 72	27 - 330	<10 - 490	12 - 33	10 - 140
Mercury	FS	71 - 370	NS	45 - 150	<49 - 140	38 - 430	160 - 200	240 - 280	380 - 470
	WB	<58 - 250	40 - 53	<33 - 380	<47 - 130	73 - 250	120 - 220	140 - 320	220 - 360
Selenium	FS	130 - 400	NS	180 - 250	300 - 720	310 - 2700	380 - 400	240 - 500	450 - 530
	WB	<180 - 500	<280	230 - 790	590 - 1800	<420 - 1100	410 - 540	410 - 630	480 - 710
p,p'-DDE	FS	14 - 740	NS	4 - 54	8 - 910	100 - 1400	44 - 52	330 - 1300	480 - 1200
	WB	28 - 1300	310 - 560	3 - 84	13 - 770	400 - 1100	350 - 440	280 - 900	970 - 1700
p,p'-DDT	FS	<2 - 92*	NS	<2 - 5*	<2 - 58	2 - 31	<2 - 3	2 - 87	23 - 48
	WB	<1 - 180	37 - 52	<2 - 12*	<2 - 49	<4 - 38	7 - 12	0.8 - 45	44 - 80
Aroclor 1254	FS	10-46	NS	10 - 20	<16 - 930	10 - 190	12 - 14	29 - 69	38 - 83
	WB	<14 - 65	18 - 32	<7 - 30	<21 - 140	38 - 120	54 - 98	25 - 58	46 - 94
Aroclor 1260	FS	<11 - 75	NS	<18	<9 - 190	<13 - 200	<19	37 - 130	68 - 220
	WB	<12 - 100	27 - 49	<6 - 22*	<18 - 130	41 - 160	47 - 61	32 - 140	80 - 190
2,3,7,8-TCDD	FS	<0.00001 - 0.00007	NS	<0.0000 - 0.00015	<0.00001 - 0.00021	0.0001 - 0.0014	0.00007 - 0.00008	0.001 - 0.0014	NA
	WB	<0.00001-0.00021	0.00006-0.00008	<0.00001 - 0.0002	<0.00001 - 0.00023	0.00006 - 0.0013	0.00036 - 0.00042	0.0010 - 0.0014	NA
2,3,7,8-TCDF	FS	0.0001 - 0.0015	NS	0.00014 - 0.00028	0.00014 - 0.014	0.0025 - 0.054	0.0006 - 0.00075	0.0022 - 0.0034	NA
	WB	0.0008 - 0.0036	0.0008 - 0.001	<0.0004 - 0.00048	0.0002 - 0.012	0.008 - 0.047	0.0038 - 0.0055	0.0022 - 0.0034	NA

N=number of samples; FS- Fillet with Skin; WB = whole body;E=egg; NA = not analyzed; < detection limit; \* detection frequency was less than 50% of the samples

\*\*whitesturgeon were single fish and fillets without skin.

**Table 9-27b. Range of chemical concentrations ( µg/kg) in anadromous fish tissue samples from our study of the Columbia River Basin.**

	T	steelhead	fall chinook salmon	spring chinook	coho salmon	eulachon	Pacific lamprey
<i>N-Egg</i>		1	1	6	3		
<i>N-FS</i>		21	15	24	3		3
<i>N-WB</i>		21	15	24	3	3	9
Arsenic	E	ND	240	<410 - 510	310 - 360		
	FS	280 - 1500	530 - 1100	560 - 1200	450 - 600	NS	280 - 360
	WB	290 - 1200	610 - 1000	570 - 1100	450 - 560	860 - 930	150 - 370
Cadmium	E	34	<4	22 - 72	<4		
	FS	<4 - 9	<4	<4 - 15	<4	NS	16 - 30
	WB	29 - 88	5 - 10	6 - 170	19 - 27	9 - 10	56 - 150
Copper	E	18,000	5800	5300 - 6600	4100 - 5000		
	FS	540 - 940	540 - 760	240 - 1000	680 - 3600	NS	1100 - 1400
	WB	1900 - 6800	1000 - 14000	1100 - 2300	720 - 2400	920 - 970	3700 - 5500
Lead	E	41	<10	<10 - 50*	<10		
	FS	<10 - 23*	<11 - 16	<10 - 140	<10 - 230	NS	<10
	WB	<10 - 360	11 - 1200	<10 - 92	11 - 20	370 - 680	<10 - 69*
Mercury	E	<43	<50	<79	<100		
	FS	70 - 210	<50 - 150	<83 - 510*	110 - 120	NS	<110
	WB	<50 - 420	<50 - 200	<71 - 130*	11 - 20	<35	<91 - 210
Selenium	E	4500	2400	3700 - 5500	1100 - 1300		
	FS	<250 - 500	280 - 380	290 - 430	270 - 310	NS	410 - 450
	WB	460 - 940	<380 - 570	360 - 680	330 - 420	270 - 300	520 - 760
p,p'-DDE	E	7	7	10 - 16	31 - 33		
	FS	5 - 28	4 - 26	6 - 18	29 - 35	NS	46 - 55
	WB	5 - 33	5 - 53	11 - 22	31 - 37	10 - 11	35 - 77
p,p'-DDT	E	<2	<2	4 - 7	<2		
	FS	<1 - 5	<2 - 8	<2 - 7	<2 - 4	NS	28 - 38
	WB	<1 - 6	<2 - 7	3 - 8	<2 - 4	<4	6 - 29
Aroclor 1254	E	15	12	15 - 20	11 - 17		
	FS	8 - 21	9 - 35	9 - 24	12 - 19	NS	80 - 100
	WB	9 - 29	10 - 47	13 - 26	18 - 19	<37	60 - 150
Aroclor 1260	E	<20	<19	<18	<18		
	FS	<6 - 21*	<19	<18	<18	NS	<19
	WB	<6 - 21*	<19	<18	<18	<37	<13 - 20*
2,3,7,8-TCDD	E	<0.00003	<0.00004	<0.00001 - 0.00004	<0.00001-0.00005		
	FS	<0.00001 - 0.00008	<0.00001 - 0.00005	<0.00001-0.00005	<0.00001-0.00004		0.00001-0.00006
	WB	<0.00001-0.00006	<0.0000 - 0.00006	<0.00001 - 0.0001	<0.00001	<0.00005-0.0001	0.00002 - 0.0007
2,3,7,8-TCDF	E	<0.00022	0.00043	0.00036 - 0.00065	0.00029-0.00066		
	FS	<0.00018-0.00065	<0.00003-0.0014	0.0004-0.00074	0.00035-0.00054		0.0012-0.0017
	WB	<0.00025-0.0006	0.00043-0.0014	0.00057 - 0.0011	0.00036-0.00049	0.00058-0.00078	0.0011-0.0032

## 10.0 Uncertainty Evaluation

There are many uncertainties in completing a survey of contaminants in fish tissue and in estimating risks from consumption of these fish. This section provides a summary of the assumptions and uncertainties in evaluating the fish contaminant data and preparing the risk assessment. Some of the types of uncertainty which were encountered in this study include:

- 1) errors in sampling, fish preparation, and chemical analysis,
- 2) variability in fish tissue concentrations within fish, across species and tissue types, and among stations,
- 7) lack of comparable data-sets for comparisons, and
- 3) lack of knowledge regarding human exposure and toxicity.

## 10.1 Fish Tissue Collection

Uncertainty in toxic chemical levels is primarily associated with variability in fish tissue concentrations over space and time as well as errors in chemical analytical methods. The temporal (seasonal, annual) range of chemical concentrations in fish species was not known.

There was some measure of spatial variability in certain fish species which were collected at a number of sites (largescale sucker, white sturgeon, mountain whitefish, rainbow trout, chinook salmon, steelhead, Pacific lamprey). Coho salmon, bridgelip sucker, and eulachon were each only collected at one location, therefore there was no measure of spatial variability in these species. Pacific lamprey and walleye were only collected at two locations. Therefore, there were gaps in our information on contaminant levels in these species from other sections of the Columbia River Basin. In addition to a limited number of sampling locations, some of the sites included large stream reaches (Table 1-1). Therefore, the average concentrations from these sites represent sampling areas of several miles.

Individual fish tissue were composited to obtain a representative sample of the mean concentrations of fish tissue. However, by compositing the fish there is a loss of certainty in the variance among individual fish samples. To reduce some of the uncertainty associated with composites, an attempt was made to collect fish: 1) at the same time and 2) of the same size.

To maintain uniformity in sample size within composites the smallest individual within a composite was supposed to be no less than 75% of the total length of the largest individual. Seventy-nine percent of the composites were within this guideline. Of the composite samples not meeting the guideline, roughly one-half were within 70% of the total length of the largest individual. The compositing goals were not fully met in all samples because:

- 1) larger fish (rainbow trout and mountain whitefish) were added to some composites to gain enough fish tissue for analyses,
- 2) tribal members requested that small fall chinook salmon (jacks) be added to samples of larger adults, or
- 3) spatial and temporal variability in fish species limited the number of fish available for sampling.

To maintain uniformity across composites the relative difference between the average length of the individuals in the smallest-sized composite (i.e., the one with the smallest average body lengths) was to be within 10% of the average length of the largest-sized composite. Eighty-nine percent of the composites were within the 10% guideline. Of the 11% not meeting the guideline, 5 composites were steelhead, and one each were walleye, largescale sucker, rainbow trout, and spring chinook salmon.

In addition to collecting composites of the same size an attempt was made to collect replicate samples at each study site to provide a more accurate estimate of the variance in tissue analyses. The goal of collecting at least three replicate composite samples for each sample type from each study site was met at 92% of the study sites. Only two replicates or less were collected at 8% of the study sites. Replication was limited at study site 30 on the Umatilla River because the electro-fishing boat broke down, which prohibited additional collections of walleye and largescale sucker. There were a low number of rainbow trout available from study site 98 in the Deschutes River.

The uncertainty in the tissue concentrations is also associated with the sampling design. The fish type, tissue type, and sample location were all predetermined during the planning conference. This type of sampling is biased with unequal sample sizes and predetermined sample locations rather than a random design. This bias is to be expected when attempting to provide information for individuals or groups based on their preferences. The results of this survey should not be extrapolated to any other fish or fish from other locations.

EPA's guidance for preparing fish tissue for chemical analysis recommends scaling fish (USEPA, 2000f). However, CRITFC's member tribes do not typically scale their fish (CRITFC tribes, personal communication). The results of some of the chemical analyses in this report may be affected by the amount of certain chemicals (e.g. metals) which may be concentrated in the fish scales.

The homogeneity of ground fish tissue can vary considerably, depending upon the nature of the tissue sample and the grinding procedures. In this project we attempted to minimize variability of chemical measurements by specifying the fish grinding procedure (See Volume 5) and by monitoring the homogeneity of composite samples.

With the exception of white sturgeon, fish tissue chemical residues were measured in fillet with skin and whole body. White sturgeon were the only species which were analyzed as fillet without skin. As discussed in Section 2, whole body fish tissue samples tend to be somewhat higher in

lipids than fillet with skin samples for some fish species. This difference in lipids between whole body and fillet fish samples was not consistent across species. This was not surprising since the preparation of fillets with skin usually left a thin layer of subcutaneous fat remaining under the skin.

The fillet and whole body samples were not from the same fish. Therefore, any comparisons between them will be affected by the natural variability in fish samples as well as the tissue type.

## 10.2 Chemical Analyses

All data quality objectives established for this project were met. However, there were uncertainties in the chemical analysis due to interferences, detection limits, and method development.

A number of problems were encountered in the measurement of target compounds. For dioxins/furans, dioxin-like PCBs, non-acid labile chlorinated pesticides, and Aroclors, the primary analytical problem encountered by the laboratories was the interference of chlorinated and brominated non-target compounds in extracts of project fish samples. For dioxin-like PCBs, many sample extracts had to be diluted and re-measured because of high levels of dioxin-like PCB target compounds in some samples.

The metallic equipment used to grind fish samples was tested prior to sample analysis for possible interferences. The results indicated that lead, manganese, nickel, copper, aluminum, zinc, and PCB 105 were found in the rinsate blanks from the fish grinder. The levels of manganese, nickel, copper, aluminum, zinc, and PCB 105 were in negligible quantities and should not affect the study results. However, the lead levels (77 µg/l) in the rinsate were higher; therefore, the results reported in this study for lead may be increased over levels that would be found in tissue samples.

Modifications to digestion procedures for high levels of lipids in some project samples improved measurements of metals and mercury using EPA methods 200.8 and 251.6. The chemical analysis of chlorinated phenolics (EPA Method 1653) and neutral semi-volatiles (EPA Method 8270) had the largest number of data which were not acceptable due to high quantitation limits.

For this project, analytical methods were chosen to provide detection or quantitation limits which were as low as possible given available analytical methods and resources. The true value of chemicals which were “not detected” is actually somewhere between the reported detection limit and zero. For this study ½ the detection limit was used to estimate chemical concentrations. Appendix E lists each chemical concentration as equal to: 1) the detection limit, 2) zero, and 3) one-half the detection limit. The use of ½ the detection limit may have over or underestimated the true fish tissue concentration.

In the quality assurance review of the chemical data, certain chemical concentrations were qualified with a “J”. The “J” qualifier designates a concentration which is estimated. EPA



recommends that the J-qualified concentrations be treated in the same way as data without this qualifier with acknowledgment that there is more uncertainty associated with “estimated” data (USEPA, 1989). We chose to use these data in this assessment without conditions. Use of this data to calculate fish tissue concentrations may overestimate the true concentration since these levels may be incorrect. The data qualifiers are listed with each data point in Appendix D of Volume 1 and in Volume 4.

The percent difference in field duplicates was estimated for all chemicals analyzed. There was less than 10% difference between most of the duplicate samples. The samples with greater than 10% difference are shown in Table 10-1. The maximum difference was 157% in cobalt concentrations in fall chinook from study site 48 (Table 10-1). There was no consistent pattern of error in field duplicate by study site, chemical, or fish species.

The difference in duplicate fillets from the same fish is an indication of the variability of chemicals within fish tissue, since the fillets were from the opposite sides of the same fish. In this study, the duplicate values were averaged. By averaging the concentration of the duplicate samples fish tissue concentrations and risk estimates may be lower than the actual exposure that would occur if the higher fish tissue concentration was used.

**Table 10-1 . Percent difference in field duplicate samples from the Columbia River Basin. Fish are listed with study site ID in parentheses. The maximum percent difference is given for the chemical within a chemical group.**

Species (study sites)	Percent difference for analytes (greater than 10%)			
	Dioxins & Furans	Metals	PCBs	Pesticides
steelhead (96)	46 (OCDD)	68 (Ba)	56 (PCB 123)	67 (DDT)
spring chinook (94)	13 (HxCDF)	62 (Cd)	17 (PCB 189)	15 (DDT)
fall chinook (8)		29 (Hg)	14 (PCB 157)	11 (DDD)
fall chinook (48)	18 (TCDF)	107 (Cr); 157 (Co)	28 (PCB 126); 18 (Aroclor 1254)	
mountain whitefish (98)	29 (TCDD)	70 (Pb)	32 (PCB 167); 32 (Aroclor 1254)	35 (DDE)
white sturgeon (13)	29 (HxCDF)	54 (Hg)	15 (PCB 118); 11 (Aroclor 1260)	124 (nonaolcor)
white sturgeon (6)	57 (TCDF & HxCDF)	42 (Co)	39 (PCB 105); 109 (Aroclor 1254)	119 (DDT)
white sturgeon (9)	50 (OCDD)	144 (Co)	27 (PCB 169)	59 (oxychlordan)

### 10.2.1 Lipid analyses

All samples were measured for percent lipids according to the procedure described in EPA Method 1613B. Other percent lipid procedures such as the three extraction methods described in EPA Method 8290 would have produced different percent lipid results because of the different extraction solvents used and different extraction conditions. While the lipid values reported in our study were consistent because the analyses were all done within one laboratory using one

method, there would be considerable uncertainty in comparing the lipid levels measured in this study with other data generated by different methods or different laboratories.

### **10.3 Comparing Chemical Data Across Fish Species and with Other Studies**

The comparison of this study with other studies is confounded by the methods that were used to collect the samples, the tissue type, number of samples, and species as well as the inconsistency in chemical methods. In particular, methods for analyzing fish tissue for dioxins, furans, and PCB congeners have changed recently. Thus, chemical analysis of fish tissue data for these particular chemicals from the 1970's through the early 1990's will not necessarily give the same results as were seen in this study.

### **10.4 Risk Assessment**

Uncertainties can occur in all parts of the risk assessment--exposure assessment, toxicity assessment, and risk characterization. An uncertainty evaluation has been done as a part of this risk assessment to show how the risk characterization could be affected if alternative assumptions had been made and/or different parameters had been used to calculate the cancer risks and non-cancer hazard indices.

#### **10.4.1 Exposure Assessment**

##### **10.4.1.1 Contaminant Concentrations in Fish Tissue**

As discussed earlier in this report, the fish species collected and the sampling study sites selected were based primarily on data from CRITFC's Fish Consumption Report (CRITFC, 1994) and discussions with tribal staff. Although samples were taken from the study sites used most frequently by the tribes, many other study sites used for fishing were not sampled. In addition, as discussed in Section 4.5, there were limited data on the species collected and fishing locations used by non-tribal populations in the Columbia River Basin. Therefore, while the concentrations of chemicals in fish tissue have been used to characterize risk for the general public in this study, this characterization was uncertain due to the lack of data on fishing practices for the general public.

Another source of uncertainty for this risk assessment involves the use of the average chemical concentrations for fish collected over a short period of time to estimate human exposure over 30 and 70-year durations. If average chemical concentrations in fish tissue have changed over time, or were likely to change in the future, the risk estimates presented in this report may either underestimate or overestimate the risk to individuals. The relatively small amount of existing historical data on chemical contaminants in fish within the Columbia River Basin was insufficient to reliably evaluate trends in chemical concentrations. The seasonal range of chemical concentrations in the target species evaluated in this risk assessment is also not known.

Thus, the risk estimates presented in this report could increase or decrease depending upon how

concentrations vary over location and time.

As discussed in Section 1.7.5, to calculate average contaminant levels in fish, a value of one-half the detection limit was used in some cases for non-detected chemicals. Risk characterization based upon one-half the detection limit could be either an overestimate or an underestimate of the actual risks.

#### **10.4.1.2 Tissue Type**

For this study, both whole fish and fillets were analyzed when possible. The fillet and whole body sample types were chosen based on the fish consumption survey for CRITFC's member tribes (CRITFC, 1994). In this study, respondents were asked to identify the fish parts they consume for each species. For most of the fish species sampled as a part of this study, 50% or more of the respondents said that they consume fish skin. A smaller proportion of the tribal members consumed other fish parts (head, eggs, bones and organs). In addition to the question of people consuming fish parts, some chemicals preferentially accumulate in fat or internal organs, thus having both whole body and fillet fish tissue samples provides a more comprehensive picture of the amount of chemical accumulated throughout the fish tissue. Fillets were analyzed with skin because most tribal members consumed the skin with the muscle tissue.

Information on the portions of fish that are consumed most frequently by the general public were not available. However, respondents to the qualitative fish consumption survey of people from Wheatland Ferry to Willamette Falls Reach of the Willamette River, Oregon indicated that they consume primarily fish fillets as well as other fish parts and the whole body (EVS, 1998).

In Section 6.2.4, the ratios of the estimated hazard indices and cancer risks for whole body to filleted fish samples were calculated to determine the possible impact of tissue type on the risk characterization. These results were calculated for those species that had both fillet and whole body samples analyzed at a given site. For non-cancer effects, whole body to fillet ratios were calculated for the total hazard index as well as for the endpoints of immunotoxicity and reproduction. The number of whole body to fillet ratios that were greater than 1 compared to the total number of samples was also shown. These calculations (Table 6-23) did not show a consistent pattern in whole body to fillet ratios for the total hazard indices, the immunotoxicity hazard indices, or cancer risks at a given site for a species. The whole body to fillet ratios ranged from 0.2 to greater than 1 for a few species/sites (e.g. high of a ratio 6.6 for fall chinook, immunotoxicity hazard index). For reproductive effects, the ratios of the hazard indices for reproductive effects in whole body to fillet samples appear to be less than 1 more frequently than those for the other hazard indices or cancer risks. This may be because the hazard index for reproductive effects is based largely upon the contaminant mercury which is not lipophilic and binds strongly to protein (e.g., muscle tissue).

Any conclusions, however, on the results of whole body to fillet samples are limited by the small sample sizes (usually 3 or less) at each site and by the fact that whole body samples were always from a composite of fish different than those used for the whole body samples (i.e., fillet and

whole body samples are not from the same fish).

### 10.4.1.3 Exposure Duration

Exposure duration is defined as the time period over which an individual is exposed to one or more contaminants. For adults, two different exposure durations were used for the risk assessment: 70 years, which represents the approximate average life expectancy of all individuals born in the United States in the late 1960s; and 30 years, which represents the 90<sup>th</sup> percentile length of time that an individual stays at one residence (USEPA, 1997b).

The value of 70 years was assumed for lifetime exposure in this risk assessment because it is the value commonly assumed for the general population in most EPA risk assessments. Also, 70 years is the primary assumption used in the derivation of many of the cancer slope factors found in IRIS (USEPA, 2000c).

As was discussed in Section 4, changes in exposure duration do not impact the exposures estimated for calculating non-cancer health impacts. This is because the product of the exposure frequency (EF) times exposure duration (ED) is always equivalent to the averaging time (AT) (see Equation 4-1 in Section 4.3).

However, since the averaging time for estimating exposure for cancer risks is always a person's lifetime, changing exposure duration does impact the estimated risk. The cancer risk estimates for an individual who consumes fish over an exposure duration that differs from the exposure durations used in this report ( $ED_{new}$ ) can be determined using the following equation:

$$(Equation 10-1) \quad ECR_{new} = ECR_{70} \times ED_{new}/ED_{70}$$

where:

- $ECR_{new}$  = Excess cancer risk for the new exposure duration
- $ECR_{70}$  = Excess cancer risk estimate for a lifetime exposure duration of 70 years
- $ED_{new}$  = Individual exposure duration in years
- $ED_{70}$  = Default lifetime exposure duration of 70 years

Equation 10-1 shows that the excess cancer risk will change in direct proportion to the ratio of the new and default exposure durations. For example, if an exposure duration of 9 years was selected, which is the median length of time an individual stays at one residence, the lifetime exposure cancer risk estimates would be multiplied by a factor of 0.13 (9 years ÷ 70 years = 0.13) to obtain revised cancer risk estimates for a 9-year exposure duration. Thus, all total excess cancer risk estimates for 70 years exposure duration for the fish species and tissue types evaluated in this report would decrease by approximately an order of magnitude (i.e. ten-fold) for an exposure duration of 9 years.

### 10.4.1.4 Consumption Rate

In this risk assessment, exposures were estimated for both the general public and for members of CRITFC's member tribes. For the general public, adequate quantitative information on fish consumption rates for those areas of the Columbia River Basin sampled in this study was not available. Therefore, the ingestion rates assumed for those individuals in this risk assessment

were based on a national report of fish consumption (USEPA, 2000b). For CRITFC's member tribes, ingestion rates were taken from CRITFC's fish consumption study (CRITFC, 1994). For both the general population and the tribes, mean and a 99<sup>th</sup> percentile ingestion rates for children and adults were selected to evaluate potential risks over a range of possible ingestion rates.

It is not known if the ingestion rates selected for this risk assessment are representative of the actual consumption practices of individuals consuming fish from the study area. The exposures estimated in this report are likely to be higher than those expected for a recreational fisherman who infrequently fishes at any of the study sites. On the other hand, as discussed in Section 4, Harris and Harper (1997) suggest that an ingestion rate of 540 g/day is more appropriate for a tribal member who pursues a traditional lifestyle. This is higher than the 99<sup>th</sup> percentile CRITFC member tribal fish consumption rate of 389 g/day used in this report.

#### **10.4.1.5 Multiple-Species Consumption Patterns**

The hazard indices and cancer risk estimates in this report were primarily based upon the consumption of individual fish species and tissue types. However, these estimates which are based upon individual fish species may not be an adequate representation of risk for most individuals since most people likely eat a diet composed of multiple fish species. Therefore, as a part of the risk characterization, a hypothetical multiple-species diet was also evaluated using tribal fish consumption data from CRITFC's fish consumption study. For this hypothetical multiple-species diet, information from Table 17 of the CRITFC fish consumption study (CRITFC, 1994) was used. This table from the CRITFC consumption survey provides information on the percentage of adults that consumed 10 fish species evaluated in the study (CRITFC, 1994). As was shown in Table 6-24 and Figures 6-35 and 6-36 the resultant cancer risk and non-cancer hazards of the multiple species diet reflect the proportion of the different types of fish in the diet and the contaminant levels in those fish. Therefore, the estimated cancer risks and non-cancer hazards from consuming fish from the Columbia River Basin for any one individual depend upon the types and amounts of fish they eat and may be very different from those estimated in this report for individual species.

As part of this uncertainty analyses, an estimate of the total cancer risks and non-cancer hazards from a multiple species diet using data from Table 18 in the CRITFC fish consumption study in addition to that in Table 17 was calculated (CRITFC, 1994). Table 18 provides average consumption rates (grams per day) for each species for those adult respondents in the survey who consume fish. These rates were determined by combining the average consumption rate for each individual who consumed a particular species with the average serving size in ounces for that individual and then calculating the mean of all of the individual consumption rates. The differences in the consumption rates for the hypothetical multiple diet using the two CRITFC tables (Table 17 versus Table 18) are shown in Table 10-2. As can be seen from Table 10-2, the

consumption rates, cancer risks and total hazards for each individual fish species differ using the results from the two different tables in the CRITFC consumption study (CRITFC, 1994). However, the total estimated cancer risks and total non-cancer hazard indices from consuming all species are approximately the same using either table.

**Table 10.2. Comparison of estimated total cancer risks and hazard indices for a hypothetical multiple species diet using data from Table 17 and Table 18 in the CRITFC fish consumption report (Source: CRITFC, 1994).**

Fish Species	T	Results using Table 17 in the CRITFC fish consumption study <sup>(1)</sup>				Results using Table 18 in the CRITFC fish consumption study		
		Percentage of Hypothetical Diet	Consumption Rate (grams/day)	Total Cancer Risk	Non- Cancer Effects (total HI)	Consumption Rate (grams/day)	Total Cancer Risk	Non Cancer Effects (total HI)
salmon	FS	27.7%	17.5	6E-05	0.6	25.7	8E-05	0.9
trout	FS	21.0%	13.3	3E-05	0.3	9.6	2E-05	0.2
whitefish	FS	6.8%	4.3	9E-05	0.7	8.9	2E-04	1.5
smelt	WB	15.6%	9.9	3E-05	0.1	4.8	2E-05	0.0
lamprey	FS	16.3%	10.3	1E-04	0.7	4.7	5E-05	0.3
walleye	FS	2.8%	1.8	4E-06	0.1	3.8	9E-06	0.2
sturgeon	FW	7.4%	4.7	7E-05	0.6	3.3	5E-05	0.4
sucker	FS	2.3%	1.5	9E-06	0.1	2.8	2E-05	0.2
Totals		100.0%	63.2	4E-04	3.2	63.6	4E-04	3.8

(1) These results are those presented in Section 6.2.5 and Table 6-24  
 FS = fillet with skin FW = fillet without skin WB = whole body

T= tissue type  
 HI = hazard index

#### 10.4.1.6 Effects of Cooking

It was assumed for this risk assessment, that (with the exception of skinless white sturgeon fillets) the skin and fatty areas of the fish are not removed during preparation, and that there is no net reduction in contaminant concentrations during cooking. Anglers who prepare fillets by skinning and trimming away the fatty area may reduce their exposure to chemicals (such as organochlorines) that accumulate in fatty areas. It has also been shown that cooking the fish may affect exposure concentrations of such chemicals, depending on the cooking method.

EPA's guidance (USEPA, 2000a) provides a summary of the effects on organochlorine (e.g., PCBs, DDT, chlordane, dioxins/furans) contaminant levels in fish as a result of fish preparation and cooking. This summary shows that the reductions in chemical concentrations vary considerably among the different studies because of different fish species, contaminants, cooking methods, etc. In these studies most of the percent reductions in chemical concentrations ranged from about 10 to 60%. However, much higher losses were also seen as were net gains of one contaminant (PCBs). Overall, these studies support the conclusion that organochlorines can be lost during cooking. But, based on the available information, it is difficult to quantify these losses for use in a risk assessment since the actual losses from cooking depend upon the cooking method (i.e., baking, frying, broiling, etc.), the cooking duration, the temperature during cooking, preparation techniques (i.e., trimmed or untrimmed, with or without skin), the lipid content of the fish, the fish species, and the contaminant levels in the raw fish.

Also as discussed in EPA guidance (USEPA, 2000a), several studies indicate that some organo-metal compounds bind to different fish tissues than the tissue which bind organochlorines. Mercury, for example, binds strongly to protein, thereby concentrating in the muscle tissue of fish. Mercury also concentrates in liver and kidney, though at generally lower rates. Thus, preparations such as trimming and gutting, can actually result in a greater average concentration of mercury in the remaining tissues compared with the concentration in the whole fish (Gutenmann and Lisk, 1991). As discussed previously in the discussion on effects of sample type on the risk characterization (Section 6.2.4 and Table 6-23), the ratios of the hazard indices for reproductive effects in whole body to fillet samples appear to be less than 1 more frequently than the ratios for the total hazard index, hazard index for immunotoxicity, and cancer risks. This may be because the hazard index for reproductive effects is based largely upon the contaminant mercury which is not lipophilic and binds strongly to protein (e.g., muscle tissue). However, any conclusions based on the ratios of whole body to fillet samples are limited by the small sample sizes (usually 3 or less) at each site and by the fact that whole body samples were always from a composite of fish different than those used for the whole body analysis (i.e., fillet and whole body samples are not from the same fish).

The impact of cooking on mercury levels was studied by Morgan et al., 1997. They found that mercury concentrations (wet weight basis) in pan-fried, baked and boiled walleye fillet ranged from 1.1 to 1.5 times higher than in the corresponding raw portions; in lake trout the range was 1.5 to 2.0 times higher.

#### **10.4.2 Toxicity Assessment**

There are also uncertainties in the toxicity assessment. These include uncertainties (1) in the toxicity values (i.e., reference doses and cancer slope factors) used; (2) in the toxicity equivalence factors developed for dioxins/furans and dioxin-like PCBs and in the relative potency factors used for PAHs; (3) in the lack of toxicity data for some of the chemicals that were detected in fish, and; (4) in the manner in which certain chemicals (Aroclors, dioxin-like PCBs, DDT/DDE/DDD, and arsenic) were evaluated.

##### **10.4.2.1 Toxicity Values**

As discussed in Section 5.0, the majority of the toxicity factors used in estimating hazard indices and cancer risks were taken from EPA's IRIS database which is a database of human health effects that may result from exposure to various substances found in the environment. For a small number of chemicals whose toxicity factors were not available in IRIS, toxicity factors developed by NCEA were used. Although the development of the IRIS toxicity factors has been reviewed by a group of EPA health scientists using consistent chemical hazard identification and dose-response assessment methods, there are still several sources of uncertainty in these factors and their relevance to the populations for which the risk assessment is being conducted. As discussed in EPA's guidance (USEPA, 1989), some of these uncertainties may include:

- using dose-response information from effects observed at high doses to predict the

adverse effects that may occur in humans following exposure to the lower levels expected from human exposure in the environment;

- using dose-response information from short-term studies to predict the effects of long-term exposures;
- using dose-response information from animal studies to predict effects in humans; and
- using dose-response information from homogenous populations or healthy human populations to predict the effects likely to be observed in the general population consisting of individuals with a wide range of sensitivities.

In addition to the uncertainties in developing reference doses and cancer slope factors based upon the data that are available, there are also uncertainties in the fact that specific types of effects data are often not available for a given chemical. Some examples include the lack of data on a chemical's cancer and non-cancer impact on vulnerable populations (e.g., children) and a lack of information for some chemicals on non-cancer endpoints such as reproductive, developmental, and endocrine disruption. However, the lack of data on non-cancer effects is usually considered when determining what uncertainty factors and modifying factors should be used to develop a reference dose for a given chemical. The lack of data on cancer is partially addressed by using conservative assumptions (e.g., upper confidence levels, the most sensitive species) in estimating cancer slope factors. All of these assumptions are intended to provide a margin of safety to ensure that the health impacts for an individual chemical are not likely to be underestimated.

To better understand the uncertainties associated with the toxicity factors for each of the chemicals evaluated in this risk assessment, refer to the Toxicity Profiles in Appendix C. These profiles review the data upon which the reference doses and cancer slope factors were developed.

#### **10.4.2.2 Toxicity Equivalence Factors for Dioxins, Furans, and Dioxin-like PCB Congeners and Relative Potency Factors for PAHs**

Toxicity equivalence factors were used for the chlorinated dioxins and furans and the dioxin-like PCBs measured in this study to calculate toxicity equivalence concentration. These toxicity equivalence factors were calculated using all of the available data and were selected to account for uncertainties in the available data and to avoid underestimating risk (Van den Berg et al., 1998). Alternative approaches, including the assumption that all dioxin-like PCBs carry the toxicity equivalence of 2,3,7,8-TCDD, or that all chlorinated dioxins, furans, and dioxin-like PCB congeners other than 2,3,7,8-TCDD can be ignored, have been generally rejected as inadequate for risk assessment purposes by EPA and many other countries and international organizations. These toxicity equivalence factors are order-of-magnitude estimates relative to the toxicity of 2,3,7,8-TCDD. Therefore, their use creates uncertainty in the risk assessment, especially since chlorinated dioxins/furans and dioxin-like PCBs contribute significantly to the cancer risks estimated in this risk assessment.



Also, it should be noted that the cancer slope factor for 2,3,7,8-TCDD is being re-evaluated as part of a current review by EPA (USEPA, 2000e). A review of the most current draft document suggests that this cancer slope factor may increase. This change would affect both the cancer risk estimates associated with 2,3,7,8-TCDD as well as those risk estimates calculated for the other chlorinated dioxins, furans, and dioxin-like PCB congeners having toxicity equivalence factors. If the slope factor increases, cancer risks estimated for these classes of compounds would also increase.

As discussed in Section 5, EPA has developed provisional guidance on estimating risk from exposure to PAHs (USEPA, 1993). A cancer slope factor is available for only one PAH, benzo(a)pyrene. In this provisional guidance, relative potency factors have been developed for six PAHs relative to benzo(a)pyrene. These relative potency factors were used to estimate cancer risk from PAHs in this risk assessment. As with the toxicity equivalence factors 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 considered to be more uncertain because they do not meet all of the criteria for the application of toxicity equivalence factors to mixtures.

In our study, with the exception of one composite sample of largescale sucker taken at study site 13 (see discussion in Section 6.2), PAHs do not contribute significantly to the levels of contaminants in fish or to cancer risk estimates from consuming fish. Therefore, the uncertainties in the use of relative potency factors for PAHs should not greatly impact the overall risks characterized in this report.

#### **10.4.2.3 Chemicals Without Quantitative Toxicity Factors**

As shown in Table 5-1, there were 23 chemicals that were analyzed for in fish tissue that do not have a cancer slope factor or reference dose. Of the 23 chemicals without toxicity values, the following 14 chemicals were not detected in any fish species: delta-BHC, dibenzofuran, gamma-chlordene, tetrachloroguaiacol, 4-bromophenyl-phenylether, 4-chloroguaiacol, 4-chlorophenyl-phenylether, 3,4-dichloroguaiacol, 4-chloro-3-methylphenol, 4,5-dichloroguaiacol, 4,6-dichloroguaiacol, 3,4,5-trichloroguaiacol, 3,4,6-trichloroguaiacol, and 3,5,6-trichloroguaiacol. Six additional chemicals were detected in less than 3% of the samples: acenaphthylene, alpha-chlordene, benzo(ghi)perylene, phenanthrene, retene, and 1-methyl-naphthalene. Of the remaining 3 chemicals, DDMU was detected less than 10%; 2-methyl-naphthalene and pentachloroanisole were detected greater than 10% of the time.

As discussed in the Toxicity Profiles (Appendix C), the toxicity and mechanism(s) of action(s) of pentachloroanisole are similar to those of its parent chemical, pentachlorophenol. However, methylation of the chlorophenols makes them more polar, and thus likely to be somewhat less reactive in biological systems. Thus the extent of both acute and chronic toxicity of pentachloroanisole can be reasonably anticipated to be somewhat less than its chlorinated parent, PCP. DDMU is a breakdown product of the DDT. Little information is available on DDMU or 2-methyl-naphthalene.

It is impossible to predict how the lack of toxicity information on these 23 chemicals might impact the characterization of risk in this report. However, given the fact that only 2 of these chemicals (2-methyl-naphthalene and pentachloroanisole) were detected in greater than 10% of the samples, any under estimation of cancer risk and non-cancer hazards is unlikely to be great.

There are no EPA consensus reference doses available for the chlorinated dioxins and furans and the dioxin-like PCB congeners, therefore, the possible non-cancer health effects from exposure to these chemicals from fish consumption could not be estimated in this report. From the most recent draft of EPA's reassessment of the toxicity of these compounds (USEPA, 2000e), it is clear that these compounds can cause non-cancer effects at very low levels of exposure. The inability to characterize the non-cancer hazards from these compounds may result in an underestimate of the non-cancer hazards calculated in this report.

#### **10.4.2.4 Risk Characterization for PCBs**

As discussed in Section 1, two different measurements were used in this study to determine PCB concentrations in fish tissue: 1) analysis of Aroclors which are commercial mixtures of both dioxin-like and non-dioxin-like PCB congeners, and 2) analysis of individual dioxin-like PCB congeners. The Aroclor methodology included the analysis of 7 Aroclors: Aroclor 1016, Aroclor 1221, Aroclor 1232, Aroclor 1242, Aroclor 1248, Aroclor 1254, and Aroclor 1260. Only Aroclors 1242, 1254, and 1260 were detected. Eleven dioxin-like PCB congeners that exert toxicity similar to 2,3,7,8-TCDD were also measured. PCB 170 and PCB 180, though measured, were not considered in the risk assessment as dioxin-like PCB congeners because they do not currently have associated toxicity equivalence factors.

#### **Cancer Risks for PCBs**

Because Aroclors are a mixture of both dioxin-like and non-dioxin-like PCB congeners, calculating and summing the risk associated with both Aroclors and with individual dioxin-like PCB congeners would likely overestimate cancer risk by accounting for the dioxin-like PCB congener risk both individually and within the risk estimates for Aroclors. Therefore, before using the Aroclor fish concentrations to calculate cancer risk, an adjustment was made to the Aroclor concentrations by subtracting the concentration of dioxin-like PCB congeners from the total Aroclor concentrations for each sample. This resulted in what is called the "adjusted Aroclor" value.

To estimate the impact of using this method on the cancer risk, a comparison was made for estimates of cancer risk from PCBs using different methods. The excess cancer risks calculated with these methods (using basin averages) for each fish species are shown in Table 10-3. The risk from dioxin-like PCB congeners alone ranged from 0.5 (coho salmon) to 3.5 (rainbow trout) times (column B/A) the risk calculated for total unadjusted Aroclors alone. Because the mass of dioxin-like PCB congeners is so small compared to that of the Aroclors, the risk estimated for adjusted Aroclors (subtracting the concentration of dioxin-like PCB congeners from the total Aroclor concentrations) (column C) is only slightly lower than that for total unadjusted Aroclors

(Column A). Characterizing PCB risks by combining either total Aroclors plus dioxin-like PCB congeners (A + B) or adjusted Aroclors plus dioxin-like PCB congeners (B + C) is approximately the same. The PCB risks estimated from using “adjusted Aroclors plus dioxin-like PCB congeners” is from 1.5 to 4.3 times that estimated from using total unadjusted Aroclors alone (Column B+C /A).

**Table 10-3. Estimated Cancer Risks for PCBs Using Different Methods of Calculation. CRITFC’s member tribal adult, average fish consumption, 70 years exposure using average Columbia River Basin-wide chemical concentrations.**

	A	B	B/A	C	A+B	B+C	(B+C)/ (A+B)	(B+C)/A
	Total unadjusted Aroclors	Dioxin- like PCB congeners	Risk Ratio	Adjusted Aroclors only	Total Aroclors plus dioxin- like PCB congeners	Adjusted Aroclors plus dioxin-like PCB congeners	Risk Ratio	Adjusted Aroclors plus dioxin- like PCB congeners / total unadjusted Aroclors
bridgelip sucker	1.1E-04	1.2E-04	1.1	1.0E-04	2.3E-04	2.3E-04	0.98	2.1
largescale sucker	7.6E-05	1.1E-04	1.4	7.1E-05	1.8E-04	1.8E-04	0.97	2.4
mountain whitefish	3.5E-04	7.7E-04	2.2	3.0E-04	1.1E-03	1.1E-03	0.96	3.1
white sturgeon	2.0E-04	1.7E-04	0.8	1.9E-04	3.7E-04	3.6E-04	0.97	1.8
walleye	2.3E-05	2.6E-05	1.1	2.1E-05	4.9E-05	4.6E-05	0.95	2.0
rainbow trout	2.5E-05	8.7E-05	3.5	2.2E-05	1.1E-04	1.1E-04	0.97	4.3
coho	4.6E-05	2.5E-05	0.5	4.5E-05	7.0E-05	7.0E-05	0.99	1.5
fall chinook	3.1E-05	3.6E-05	1.2	3.0E-05	6.8E-05	6.6E-05	0.98	2.1
spring chinook	2.9E-05	4.8E-05	1.7	2.8E-05	7.7E-05	7.6E-05	0.98	2.6
steelhead	4.4E-05	7.5E-05	1.7	4.2E-05	1.2E-04	1.2E-04	0.99	2.7
eulachon	ND	9.5E-06	NA	ND	9.5E-06	9.5E-06	1.00	NA
Pacific lamprey	1.6E-04	3.3E-04	2.1	1.5E-04	4.8E-04	4.7E-04	0.98	3.0

ND = not detected    NA = not applicable

### Non-Cancer Effects from Aroclors

The immunological endpoint was based upon the toxicity of Aroclors. However, only one of the three Aroclors detected in the fish samples has a reference dose - Aroclor 1254. Therefore, two possible methods were available to estimate the non-cancer hazard for the immunotoxicity endpoint.

- (A) - estimate the hazard index using the concentration of Aroclor 1254 only and the reference dose for Aroclor 1254, or
- (B) - assume that the reference dose for Aroclor 1242 and 1260 are equivalent to that for Aroclor 1254; estimate the hazard index by summing all three Aroclor concentrations and use this sum with the reference dose for Aroclor 1254.

Method B was used in this risk assessment. To show the potential uncertainties with using Method B, the hazard indices calculated with both methods (using basin averages) for each fish species are shown in Table 10-4.

**Table 10-4. Comparison of Hazard Indices for the Immunological Endpoint Based on Alternative Treatments of Aroclor Data. CRITFC's member tribal adult, average fish consumption, using average Columbia River Basin-wide chemical concentrations.**

	Endpoint specific hazard index for immunotoxicity		(B/A) Ratio of the hazard index for the sum of Aroclors to the hazard index for Aroclor 1254 only
	(A) Aroclor 1254	(B) sum of Aroclors 1242, 1254, and 1260	
bridgelip sucker	1.1	2.7	2.5
largescale sucker	0.8	1.9	2.4
mountain whitefish	5.1	8.7	1.7
white sturgeon	2.6	5	1.9
walleye	0.6	0.6	1.0
rainbow trout	0.6	0.6	1.0
coho salmon	0.7	1.1	1.6
fall chinook salmon	0.8	0.8	1.0
spring chinook salmon	0.7	0.7	1.0
steelhead	0.7	1.1	1.6
eulachon	ND	ND	ND
Pacific lamprey	3.9	3.9	1.0

ND = Not Detected

Table 10-4 also shows the ratio of the hazard index calculated using (A) Aroclor 1254 concentrations only or (B) the sum of all three Aroclors. For walleye, rainbow trout, spring chinook, fall chinook, and Pacific lamprey, the method used has no impact on the hazard index calculated for the immunotoxicity endpoint. This is because for these five species, only Aroclor 1254 was detected in the fish sampled. For the other species, the hazard index based on Method B (using the sum of all Aroclor concentrations) is from 1.6 to 2.5 times higher than the hazard index based upon Aroclor 1254 alone (column B/A).

#### 10.4.2.5 Non-Cancer Effects from DDT, DDD, and DDE

DDT and its derivatives, DDD and DDE, were measured in fish tissue samples; however, only DDT has a reference dose. The reference dose for DDT is based upon its toxic effects on the liver (hepatotoxicity). For the non-cancer hazard assessment done in this report, two possible methods for the estimation of the hazard quotient and hazard index from these chemicals were possible:

- (A) - estimate the hazard quotient using the concentrations of DDT only and the reference dose for DDT, or
- (B) - assume that the reference doses for DDD and DDE are equivalent to that for DDT. Therefore, first sum the concentrations of all of the DDD, DDE and DDT species in each sample and utilize the reference dose for DDT to estimate the hazard quotient from the summed concentrations of DDD, DDE, and DDD

**Table 10-5. Comparison of Hazard Quotients and Hazard Indices for the Hepatic Health Endpoint Based on Alternative Treatments of DDT, DDD, and DDE Data. CRITFC's member tribal adult, average fish consumption, using average Columbia River Basin-wide chemical concentrations.**

Species	Hazard quotient		Hazard Index for hepatic endpoint			
	A	B	(B/A)		(D/C)	
	DDT only	Total DDT	HQ (Total DDT)/ HQ (DDT)	DDT only	sum of DDT, DDE, and DDD	HI (Total DDT)/ HI (DDT)
bridgelip sucker	0.08	0.95	11	0.13	1.00	7.5
largescale sucker	0.04	0.44	11	0.10	0.50	5.0
mountain whitefish	0.03	0.76	27	0.19	0.93	4.8
white sturgeon	0.02	1.04	52	0.36	1.38	3.9
walleye	0.00	0.10	28	0.47	0.57	1.2
rainbow trout	0.01	0.05	8	0.04	0.09	2.1
coho salmon	0.00	0.01	4	0.06	0.07	1.2
fall chinook	0.00	0.03	7	0.08	0.10	1.4
spring chinook	0.01	0.04	4	0.08	0.11	1.3
steelhead	0.00	0.03	8	0.07	0.10	1.4
eulachon	ND	0.02	NA	0.05	0.07	1.4
Pacific lamprey	0.06	0.17	3	0.22	0.33	1.5

ND = not detected; NA = not applicable  
 HS = hazard quotient  
 HI = Hazard index  
 Total DDT = sum of DDT, DDD, DDE

Method B was used to characterize non-cancer health effects in this study. Because DDT has been identified as having a hepatic (liver) toxicity endpoint, the treatment of DDT and its derivatives will affect not only the hazard quotient for the these species, but also the hazard index for the hepatic (liver) toxicity endpoint.

Table 10-5 compares the hazard quotients for DDT and its derivatives (in columns A and B) as well as the hazard indices for the hepatic endpoint (in columns C and D) using the two methods. As can be seen from Table 10-5, the hazard quotient increased from about 3 times for Pacific lamprey to 52 times for white sturgeon when all three species (DDT, DDE, DDD) are summed to calculate the hazard quotient compared to calculating the hazard quotient using DDT data alone. The impact on the hepatic endpoint is less because for some fish species other chemicals in addition to DDT and its derivatives are included in the calculation of the hazard index for hepatotoxicity. The ratio between the hepatic hazard index using DDT, DDE, and DDD to the hepatic hazard index using DDT alone ranges from between 1.2 for coho salmon to 7.5 for bridgelip sucker, with the highest ratios seen in some of the resident fish species. Thus, the endpoint specific hazard indices for hepatotoxicity that are discussed in Section 6 may be an overestimate if DDE and DDD are less toxic to the liver than DDT. This is primarily true for several of the resident species.

#### 10.4.2.6 Risk Characterization for Arsenic

As discussed in Section 5.3.3, total arsenic was measured in fish tissue samples in this study. Because a reference dose and cancer slope factor are available for only inorganic arsenic, an

assumption about the percent of inorganic arsenic in fish had to be made to estimate the non-cancer hazards and cancer risks. The non-cancer hazards and cancer risks discussed in Section 6.2.1 and 6.2.2, respectively, assumed that for all fish species (resident fish and anadromous fish) caught in this study, 10% of the total arsenic was inorganic arsenic. The data in Section 5.3.3 also suggests that an alternative assumption for anadromous fish species should be considered - the assumption that 1% of the total arsenic is inorganic. Therefore in Section 6.2.6, the non-cancer hazards and cancer risks were recalculated for anadromous fish species using basin data assuming that 1% of the total arsenic was inorganic.

This comparison of the results from using the two different assumptions (1% versus 10%) for arsenic in fish shows that the reduction of the non-cancer hazards is less than 12% for all anadromous fish species, except eulachon which had about a 50% reduction. However, the impact is greater on the estimates of cancer risk. With the exception of lamprey for which cancer risks were reduced by only 6%, the reductions in cancer risks for steelhead were about 29%. The cancer risks for the other anadromous fish species were reduced from about 40% to 50%. Thus, the assumptions used for percent inorganic arsenic have the most impact on the cancer risks estimated for salmon, steelhead and eulachon and on the non-cancer hazards for eulachon.

### **10.4.3 Risk Characterization**

#### **10.4.3.1 Cancer Risk Estimates**

As recommended by EPA's guidance on mixtures (USEPA, 2000g), the total cancer risk from a sample is calculated by summing the risk of individual carcinogenic compounds in that sample. This approach for carcinogens (response addition) assumes independence of action by the components in a mixture (i.e., that there are no synergistic or antagonistic interactions among the carcinogens in fish and that all chemicals produce the same effect, cancer). If these assumptions are incorrect, over- or under-estimation of the actual risks could result. The underlying biological basis for assuming synergism is that cancer is a multistage process where a series of events transforms a normal cell into a malignant tumor. If two carcinogens act at different stages, their combined effect can be greater than either acting alone. For example, initiation-promotion studies have demonstrated synergistic effects for some pairs of carcinogens. On the other hand, similar-acting carcinogens can compete with each other to result in antagonism. For example, the presence of one metal can decrease the absorption or effectiveness of a similar metal. Interactions can be quite complex and can depend on dose or other factors, including background exposures to other carcinogens. In general, available information seldom allows quantitative inferences to be made about potential interactions among carcinogens. In the absence of such information, the practice is to assume additivity, particularly at low doses for mixtures.

Summation of carcinogenic risks for substances with different weights-of-evidence for human carcinogenicity is also an uncertainty. The cancer risk equation for multiple substances sums all carcinogens equally, giving as much weight to class B or C as to class A carcinogens. Using the assumption of additivity gives equal weight to all slope factors without regard to their basis from human data. In this assessment, only arsenic is in the class A carcinogen group (human carcinogen based on human data) and all of the other major contributors to cancer risk (e.g., DDT

and DDE, DDD, Aroclors, dioxin-like PCB congeners and chlorinated dioxins and furans) are in the class B2 group (probable human carcinogen based on sufficient evidence in animals and inadequate or no evidence in humans). It should be noted, however, that EPA's most recent draft document on the toxicity of 2,3,7,8-TCDD and related compounds (USEPA, 2000e) characterizes the complex mixtures of dioxins to which humans are exposed as "likely human carcinogens".

The cancer slope factors used in this risk characterization are primarily from EPA's database, IRIS. Most of the IRIS cancer slope factors are considered to be plausible upper bounds to the actual lifetime excess cancer risk for a given chemical. Concern has often been raised that adding multiple carcinogens, whose slope factor are upper bound estimates, will lead to unreasonably high estimates of the actual risk. Statistical examination of this issue suggests that the error in the simple addition of component upper bounds is small compared to other uncertainties, and that as the number of mixture components increases, summing their upper bounds yields an inflated but not misleading estimate of the overall risk (Cogliano, 1997). In fact, division by a factor of two can be sufficient to convert a sum of upper bounds into a plausible upper bound for the overall risk. If one or two carcinogens predominate the risk, however, this is not of concern.

#### **10.4.3.2 Non-Cancer Health Effects**

In Section 6, non-cancer health impacts were evaluated in several ways. First, the hazard quotient was calculated. The hazard quotient, which is the ratio between an individual's estimated exposure to a chemical compared to the reference dose for that chemical, assumes that there is a level of exposure (i.e., the reference dose) below which it is unlikely for even sensitive populations to experience adverse health effects. As a rule, the greater the value of the hazard quotient, the greater the level of concern. However, it is important to emphasize that the level of concern does not increase linearly as the reference dose is approached or exceeded for each chemical because reference doses for different chemicals do not have equal accuracy or precision and are not based on the same severity of toxic effects. Therefore, the possible health impacts resulting from exposures greater than the reference dose can vary widely depending upon the chemical.

Based on EPA guidance (USEPA, 1986a; USEPA, 1989; USEPA, 2000g), the hazard quotients calculated for each chemical in a sample were then summed to give a hazard index. This approach of adding all of the hazard quotients regardless of endpoint (dose addition) has several uncertainties because it assumes that all compounds in a mixture have similar uptake and pharmacokinetics (absorption, distribution, and elimination in the body) and it results in combining chemicals with reference doses that are based upon very different critical effects, levels of confidence, uncertainty/modifying factors, and dose-response curves. Since the assumption of dose additivity is most properly applied to compounds that induce the same effect by the same mechanism of action, EPA guidance recommends that when the total hazard index for a mixture exceeds 1, the chemicals in that mixture should be segregated by effect and mechanism to derive endpoint-specific hazard indices (USEPA, 1986a).

Although deriving endpoint specific hazard indices, as was done for this risk assessment, likely reduces the uncertainty in the non-cancer hazard evaluation in this risk assessment, these

uncertainties are not eliminated. For example, calculation of endpoint specific hazard indices may still be incorrect estimates of non-cancer health impacts. Although two chemicals may affect the same organ (e.g. the liver), they may not necessarily do so by the same specific toxicological process.

However, it should be noted that in this assessment the majority of the estimated non-cancer hazards resulted from a limited number of chemicals: Aroclors, mercury, total DDTs, and arsenic. The highest endpoint specific hazard indices were for immunotoxicity (due to Aroclors), central nervous system and reproduction/developmental (due to mercury), liver (due primarily to DDT, DDE and DDD), and hyperpigmentation/cardiovascular (due to arsenic). These endpoint specific hazard indices are based in large part on a single chemical or class of chemical (e.g. total DDTs). Therefore, the many uncertainties regarding calculation of endpoint specific hazard indices using a mixture of chemicals should not play a major role in the characterization of non-cancer hazards.

#### **10.4.3.3 Cumulative Risk from Chemical and Radionuclide Exposure**

Risks were combined for all carcinogens to equal a total cancer risk. However, radionuclides were not included in this estimate because radionuclide analyses were not completed for all species in this assessment.

### **10.5 Risk Characterization for Consumption of Fish Eggs**

As discussed in Section 4.5, a small number of egg samples were collected for some of the anadromous fish species. Although the fish consumption studies discussed in this report suggest that both CRITFC's member tribes and some of the general public consume eggs, none of these studies provided information on the amount of eggs consumed. Therefore, a risk characterization of eggs was not included in Section 6. However, to provide information on the potential risks from consuming eggs, the average fish ingestion rates for adults and children (general public and CRITFC's member tribes) were used for estimating cancer risk (adults only) and non-cancer hazards (adults and children) for eggs. These estimates for eggs, which are shown in Appendix P, are very uncertain but they serve as a useful comparison to the results for fish consumption.

Three samples of eggs were collected from coho salmon (Umatilla), fall chinook (Columbia, site 8), and steelhead (Columbia, site 8) and six egg samples were collected from spring chinook (3 at the Umatilla and 3 at Looking Glass Creek).

Endpoint specific and total hazard indices for eggs were calculated using the average fish ingestion rates for each population (adult and child, general public and; adult and child, CRITFC's member tribes)(Tables 1.1 and 1.2 (coho salmon), 2.1 and 2.2 (fall chinook salmon), 3.1 and 3.2 (spring chinook salmon), 4.1 and 4.2 (steelhead)). This provides estimates of the non-cancer hazards for two ingestion rates for adults (7.5 and 63.2 g/day) and children (2.83 g/day, up to age 6; and 24.8 g/day, up to age 15). No endpoint specific hazard indices and no total hazard indices greater than 1 were found using the average fish consumption rate for the general public, adult or child. At the average consumption rate for CRITFC's member tribal adults and children,



some of the total hazard indices were greater than 1 for eggs, the highest being approximately 4 for steelhead eggs at the average fish consumption rate for CRITFC's member tribal children. Endpoint specific hazard indices greater than 1 (high of 2) for liver, immunotoxicity, and selenosis were seen for CRITFC's member tribal child, average ingestion rate for spring chinook and steelhead; an immunotoxicity endpoint specific hazard index of approximately 1 was seen for coho. Endpoint specific hazard indices greater than 1 were due to exposures greater than the reference dose for total Aroclors (immunotoxicity) and selenium (selenosis and liver).

Cancer risks for eggs were calculated using the average fish ingestion rates for both adult populations (general public adult and CRITFC's member tribal adult) for both 30 and 70 years of exposure. These results are found in the tables in Appendix P (Tables 1.3 (coho salmon), 2.3 (fall chinook salmon), 3.3 (spring chinook salmon), and 4.3 (steelhead)). As can be seen from these tables, cancer risks from consumption of eggs ranged from  $4 \times 10^{-6}$  for both fall chinook and steelhead at the lowest exposures (general public adult, average fish ingestion rate, 30 years exposure) to a high of  $8 \times 10^{-5}$  for the highest exposure calculated (average fish consumption rate, CRITFC's member tribal adult, 70 years of exposure). For these same exposures, coho salmon eggs ranged from  $7 \times 10^{-6}$  to  $1 \times 10^{-4}$  and spring chinook eggs from  $9 \times 10^{-6}$  to  $2 \times 10^{-4}$ .

## 11.0 Conclusions

The goals of this study were to determine:

- 1) if fish were contaminated with toxic chemicals,
- 2) the difference in chemical concentrations among fish species and study sites, and
- 3) the potential human health risk due to consumption of fish from the Columbia River Basin.

The results of the study showed that all species of fish had some levels of toxic chemicals in their tissues and in the eggs of chinook and coho salmon and steelhead. The concentration of organic chemicals in the egg samples was lower than expected, given the high lipid content of the egg samples. The fish tissue chemical concentrations were quite variable within fish (duplicate fillets), across tissue type (whole body and fillet), across species, and study sites. However, the chemical residues exhibited some trends in distribution. The concentrations of organic chemicals in the salmonids (chinook and coho salmon, rainbow and steelhead trout) were lower than any other species. The concentrations of organic chemicals in three fish species (white sturgeon, mountain whitefish, largescale sucker) were higher than any other species. Pacific lamprey had higher organic chemical concentrations than anadromous species but lower than resident species. The concentrations of metals were variable with maximum levels of different metals occurring in a variety of species. The distribution across stations was variable although fish collected from the Hanford Reach of the Columbia River and the Yakima River tended to have higher concentrations of organic chemicals than other study sites.

The concentrations of toxic chemicals found in fish from the Columbia River Basin may be a risk to the health of people who eat them depending on:

- A. the toxicity of the chemicals,
- 2) the concentration of chemicals in the fish,
- 3) fish ingestion rates
- 4) fish species, and tissue type

The chemicals which contributed the most to the hazard indices and cancer risks were the persistent bioaccumulative chemicals (PCB, DDE, chlorinated dioxins and furans) as well as some naturally occurring metals (arsenic, mercury). Some pollutants persist in the food chain largely due to past practices in the United States and global dispersion from outside North America. Although some of these chemicals are no longer allowed to be used in the United States, a survey of the literature indicates that these chemical residues continue to accumulate in a

variety of foods including fish. Human activities can alter the distribution of the naturally occurring metals (e.g. mining, fuel combustion) and thus increase the likelihood of exposure to toxic levels of these chemicals through inhalation or ingestion of food and water.

Many of the chemical residues in fish identified in this study were not unlike levels found in fish from other studies in comparable aquatic environments in North America. The results of this study, therefore, have implications not only for tribal members but also the general public.

While contaminants remain in fish, it is useful for people to consider ways to still derive beneficial effects of eating fish, while at the same time reducing exposure to these chemicals. Fish are a good source of protein, low in saturated fats, and contain oils which may prevent coronary heart disease. Risks can be reduced by decreasing the amount of fish consumed, by preparing and cooking fish to reduce contaminant levels, or by selecting fish species which tend to have lower concentrations of contaminants.

Reducing dietary exposure through cooking or by eating a variety of fish will decrease the consumer's exposure, but not eliminate these chemicals from the environment. Reduction of many of the man-made chemicals from the environment will take decades to centuries. Regulatory limits for new waste streams and clean up of existing sources of chemical wastes can help to reduce exposure. The exposure to naturally occurring chemicals can be reduced through better management of our natural resources. The results of this study confirm the need for regulatory agencies to continue to pursue rigorous controls on environmental pollutants and to remove those pollutants which have been dispersed into our ecosystems.

There are many uncertainties in this risk assessment which could result in alternate estimates of risk. These uncertainties include our limited knowledge of the mechanisms which cause disease, the variability of contaminants in fish, changes in fish tissue concentrations over time, ingestion rates, and the effects of food preparation. The uncertainties in our estimates may increase or decrease the risk estimates reported in this study.

The chemicals which were estimated to contribute the most to potential health effects (PCB, DDE, chlorinated dioxins and furans, arsenic, mercury) are the chemicals for which regulatory strategies need to be defined to eliminate or reduce these chemicals in our environment.

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