

Final Report  
Evaluation of Contaminants in Fish from Lake Washington  
King County, Washington

DOH 333-061 September 2004



## **Table of Contents**

Acknowledgements	2
Foreword	3
Glossary	4
Executive Summary	6
Introduction	8
Results	11
Discussion	14
References	26
Appendix A	28
Appendix B	33
Appendix C	45
Appendix D	67
Appendix E	73

## **Acknowledgements**

We would like to thank the King County Department of Natural Resources and Parks (King County DNRP) for funding this research and providing the data for this study. Jen McIntyre and Dave Beauchamp, University of Washington School of Aquatic and Fishery Sciences, designed the study, collected and analyzed fish tissue, and assisted with data and report review. Jonathan Frodge, Randy Shuman, and Deb Lester (King County DNRP) provided valuable comments and information for the report. We wish to thank Eric Warner and Glen St. Amant (Muckleshoot Indian Tribe), Sandie O’Neill (Washington State Department of Fish and Wildlife), Dale Norton and Maggie Dutch (Washington State Department of Ecology), and Lon Kissinger (US Environmental Protection Agency) for their comments and review. We also want to thank Liz Carr, Rob Duff, and Gary Palcisko, Washington State Department of Health, for their input and review. Finally, we would like to thank Public Health Seattle and King County for their partnership in issuing fish consumption guidance based on this report.

## **Foreword**

The Washington State Department of Health (DOH) prepared this technical support document as a basis for evaluating the necessity of public advisories on fish consumption. This document is not intended to provide advice to the public. It represents a scientific analysis of fish tissue sampling data that serves as a necessary precursor to any decisions made regarding the need for a fish consumption advisory.

The toxicologists who prepared this report were Joan Hardy, Ph.D. and Dave McBride, MS.

For additional information or questions contact us at:

Washington State Department of Health  
Office of Environmental Health, Safety, and Toxicology  
P.O. Box 47825  
Olympia, WA 98504-7846  
1-877-485-7316  
Website: [www.doh.wa.gov/etoxcontact](http://www.doh.wa.gov/etoxcontact)

For people with disabilities, this document is available on request in other formats, to submit a request, please call 1-800-525-0127 (TDD/TTY call 711).

## Glossary

Acute	Occurring over a short time (compare with chronic).
Agency for Toxic Substances and Disease Registry (ATSDR)	The principal federal public health agency involved with hazardous waste issues, responsible for preventing or reducing the harmful effects of exposure to hazardous substances on human health and quality of life. ATSDR is part of the U.S. Department of Health and Human Services.
Cancer Slope Factor	A number assigned to a cancer causing chemical that is used to estimate its ability to cause cancer in humans.
Carcinogen	Any substance that causes cancer.
Chronic	Occurring over a long time (more than 1 year) (compare with acute).
Comparison value	Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.
Contaminant	A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.
Dose (for chemicals that are not radioactive)	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An “exposure dose” is how much of a substance is encountered in the environment. An “absorbed dose” is the amount of a substance that actually gets into the body through the eyes, skin, stomach, intestines, or lungs.
Environmental Protection Agency (EPA)	The federal agency that develops and enforces environmental laws to protect the environment and the public's health.
Epidemiology	The study of the occurrence and causes of health effects in human populations. An epidemiological study often compares two groups of people who are alike except for one factor, such as exposure to a chemical or the presence of a health effect. The investigators try to determine if any factor (i.e., age, sex, occupation, economic status) is associated with the health effect.
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term (acute exposure), of intermediate duration, or long-term (chronic exposure).

Hazardous substance	Any material that poses a threat to public health and/or the environment. Typical hazardous substances are materials that are toxic, corrosive, ignitable, explosive, or chemically reactive.
Ingestion	The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way (see route of exposure).
Ingestion rate	The amount of an environmental medium that could be ingested, typically on a daily basis. Units for IR are usually liter/day for water, and mg/day for soil.
Inorganic	Compounds composed of mineral materials, including elemental salts and metals such as iron, aluminum, mercury, and zinc.
Lowest Observed Adverse Effect Level (LOAEL)	The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.
Media	Soil, water, air, plants, animals, or any other part of the environment that can contain contaminants.
Minimal Risk Level (MRL)	An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), non-cancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects (see reference dose).
No Observed Adverse Effect Level (NOAEL)	The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.
Oral Reference Dose (RfD)	An amount of chemical ingested into the body (i.e., dose) below which health effects are not expected. RfDs are published by EPA.
Organic	Compounds composed of carbon, including materials such as solvents, oils, and pesticides that are not easily dissolved in water.
Parts per billion (ppb)/Parts per million (ppm)	Units commonly used to express low concentrations of contaminants. For example, 1 ounce of trichloroethylene (TCE) in 1 million ounces of water is 1 ppm. 1 ounce of TCE in 1 billion ounces of water is 1 ppb. If one drop of TCE is mixed in a railroad tank car (13,200 gallons), the water will contain about 1 ppb of TCE.
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing (inhalation), eating or drinking (ingestion), or contact with the skin (dermal contact).

## Executive Summary

The Washington State Department of Health (DOH) works to protect and improve the health of people in Washington State. Part of this mission is to reduce or eliminate exposures to health hazards in the environment, including contaminants found in fish. Recently, King County Department of Natural Resources and Parks (King County DNRP) provided DOH with fish tissue data collected by the University of Washington, School of Aquatic and Fishery Sciences (UW) as part of an ecological risk assessment to evaluate bioaccumulation of contaminants through the Lake Washington food web. Whole body fish tissue from cutthroat trout (*Oncorhynchus clarki*), northern pikeminnow (*Ptychocheilus oregonensis*), yellow perch (*Perca flavescens*), smallmouth bass (*Micropterus dolomieu*), and adult sockeye salmon (*Oncorhynchus nerka*) was collected and analyzed for contaminants that bioaccumulate, including chlordane, p,p'-dichlorodiphenyltrichloroethane (DDT), mercury, and polychlorinated biphenyls (PCBs).

Data from Lake Washington analyzed in this report are limited due to the small sample size for most size classes of fish (n =10) and the use of whole body fish tissue rather than edible muscle tissue (fillets). Regardless of these data limitations, high PCB concentrations in some species warranted a health assessment for consumers of Lake Washington fish. Findings include:

- Highest mean concentrations of PCBs and mercury in Lake Washington fish were observed in large northern pikeminnow (>300mm) (1071.4 ppb total PCBs, 387.1 ppb mercury).
- Large yellow perch (>271 mm) also had elevated mean levels of total PCBs and mercury (191.1 ppb and 183 ppb, respectively).
- Large cutthroat trout (> 300 mm) had the second highest mean concentrations of total PCBs (377.4 ppb) and relatively high concentrations of mercury (175.6 ppb).
- Only three smallmouth bass were sampled. Mean concentrations of mercury and total PCBs were 244.3 ppb and 371.2 ppb, respectively.
- Sockeye salmon had the lowest mean levels of all chemicals of concern.

Based on estimates of consumption for Lake Washington anglers and concentrations of contaminants in fish, DOH determined that the average angler may be exposed to contaminants of concern above recommended levels. In order to protect consumers of Lake Washington fish, DOH provides the following recommendations. These recommendations are emphasized for women of childbearing age and young children because of potential adverse impacts on the developing child.

- Eat a variety of fish as part of a balanced diet. Health benefits of eating fish are:
  - Fish is an excellent low-fat food, a great source of protein, vitamins, and minerals.
  - The oils in fish are important for unborn and breastfed babies.
  - Eating a variety of fish helps to reduce your chances of stroke or heart attack.
- Northern pikeminnow should not be consumed.

- Yellow perch greater than 270 mm (10½ inches) may be consumed as an eight-ounce meal once per month. Yellow perch smaller than 270 mm (10½ inches) may be consumed as an eight-ounce meal four times per month.
- Consumers of large cutthroat trout greater than 300 mm (12 inches) from Lake Washington should eat no more than one eight-ounce meal per month. For small cutthroat trout greater than 300 mm (12 inches), no more than 3 eight-ounce meals per month are recommended.
- No meal restrictions on sockeye salmon from Lake Washington. Consumers are encouraged to choose sockeye when consuming local fish.
- Prior to the issuance of this interim advisory, a statewide fish consumption advisory for large and smallmouth bass due to mercury was in place throughout water bodies in Washington State, including Lake Washington. Women of childbearing age and children six years of age or younger should eat no more than two meals per month of any bass caught in Washington state freshwaters.

The recommendations given above are based on a 60 kg (132 lbs) adult eating eight-ounce meals. In general, children should eat proportionally smaller meal sizes. Calculations for multiple chemical exposures do not change the above advice.

Since the above recommendations are based on a small sample size, DOH recommends additional sampling of northern pikeminnow, yellow perch, and cutthroat trout, to confirm initial findings of high contaminant concentrations in fish tissue. We recommend that future sampling include fillet samples for better estimation of human exposure. Other species of fish consumed by anglers such as rainbow trout, crappie, and bluegill should also be sampled.

## Introduction

The Washington State Department of Health (DOH) works to protect and improve the health of people in Washington State. Part of this mission is to reduce or eliminate exposures to health hazards in the environment. DOH's Office of Environmental Health Assessments (OEHA) conducts environmental health assessments, develops strategies, and provides education and outreach to communities in order to minimize health impacts from exposure to environmental contaminants. One focus of OEHA is on contaminants found in fish.

Recently, the King County Department of Natural Resources and Parks (King County DNRP) provided DOH with fish tissue data collected by the University of Washington, School of Aquatic and Fishery Sciences (UW) (J. McIntyre, personal communication, 2004). These data were analyzed as part of an ecological risk assessment to evaluate bioaccumulation of contaminants through the Lake Washington food web. Tissue from cutthroat trout (*Oncorhynchus clarki*), northern pikeminnow (*Ptychocheilus oregonensis*), yellow perch (*Perca flavescens*), smallmouth bass (*Micropterus dolomieu*), and adult sockeye salmon (*Oncorhynchus nerka*) was collected and analyzed for four contaminants (chlordane, p,p'-Dichlorodiphenyltrichloroethane (DDT), mercury, and polychlorinated biphenyls (PCBs)) (hexachlorocyclohexane was measured but not detected). Because this study was designed to investigate accumulation of toxicants in the food web, whole body fish were used in the analysis. When DOH conducts a human health evaluation of contaminants in fish, analysis is generally conducted on fillet tissue because the use of whole body fish tissue is likely to overestimate potential risks to humans. Nevertheless, DOH decided that data from the King County DNRP/UW study is useful in making a preliminary human health evaluation for these contaminants in Lake Washington fish.

The purpose of this report is to review and evaluate potential health risks that may result from exposure to bioaccumulative contaminants through the consumption of five Lake Washington fish species. Four chemicals are assessed for cancer and non-cancer endpoints. Consideration is given to data quality issues (i.e., use of whole body fish tissue vs. fillets and small sample size), toxicity of the chemicals, potential exposure of fish consumers, consumer body weight, comparison of contaminant levels with fish in other lakes, and the benefits of eating fish. DOH provides interim guidance for consuming Lake Washington fish and makes recommendations for future sampling.

## Background

### Lake Washington

Lake Washington is a large (87.6 km<sup>2</sup>) lake located directly east of Seattle, Washington. It is approximately 28 km long and 65 m deep (Edmondson 1991). Lake Washington is usually stratified from April through November, with average epilimnetic (the upper thermally-stratified layer of the lake) temperatures of 6° to 8° C in winter and 21° to 23° C in summer (Bartoo 1972, Brocksmith 1999; <http://dnr.metrokc.gov/wlr/waterres/lakes/monitor.htm>). Thirty native fish species and 23 introduced fish species have been documented in the Lake Washington basin (E. Warner, personal communication, 2004; Nowak 2000).

## Fish Species

Cutthroat trout, northern pikeminnow, yellow perch, smallmouth bass, and sockeye salmon were collected from Lake Washington as part of the food web study funded by King County DNRP. Cutthroat trout were collected from Lake Sammamish for comparison with Lake Washington fish. Descriptions of the five species including information on their distributions, feeding patterns, and life histories can be found in Appendix A. Other native and introduced fish species live in Lake Washington but were not collected for this study (Appendix A). For the species sampled, northern pikeminnow live the longest (up to 19 years), followed by smallmouth bass (greater than 10 years), cutthroat trout (6 – 7 years), yellow perch (less than 7 years), and sockeye salmon (S. O’Neill, personal communication, 2004). For the resident fish, northern pikeminnow feed higher in the food web than cutthroat trout, followed by smallmouth bass and yellow perch. Contaminants such as PCBs and mercury may be higher in older and larger fish because these metabolically resistant contaminants can bioaccumulate over time (i.e., exposure time is greater) and because they biomagnify as fish grow and feed at a higher trophic level.

## Chemicals of Concern

Chemicals that were analyzed and detected in whole fish tissue include chlordane, DDT, PCBs, and mercury. Hexachlorocyclohexane (lindane and other isomers) was analyzed but not detected in any samples. These chemicals were chosen since they are frequently observed in aquatic organisms due to their persistence, toxicity, and ability to bioaccumulate and/or biomagnify. A description of chemicals that were analyzed and detected in fish tissue samples from Lake Washington can be found in Appendix B.

## Methods

### Sampling

Fish were collected to obtain as large of a sample of different predator and prey fish as possible given the logistical constraints of the project. Whole fish were tested to provide data for toxic accumulation in the food web of Lake Washington as part of an ecological risk assessment being conducted by King County DNRP. Fishes were collected by a variety of methods including gillnetting, angling, mid-water trawl, electroshocking, snorkeling, minnow traps, and submerged emergent traps. Northern pikeminnow (squawfish), cutthroat trout, and yellow perch were captured opportunistically throughout the lake between October 2001 and April 2003. Four cutthroat trout (>300 mm) were sampled from Lake Sammamish. Sample sizes are given below (Table 1).

**Table 1.** Number of samples collected per fish species from Lake Washington, Seattle, Washington between October 2001 and April 2003.

Species	Size Class	Number of samples collected from Lake Washington
---------	------------	--

Species	Size Class	Number of samples collected from Lake Washington
Northern pikeminnow	<300 mm	10
	>300 mm	10
Cutthroat trout	<300 mm	10
	>300 mm	10
Yellow perch	< 200 mm	10
	201 – 271 mm	10
	> 271	9
Smallmouth bass	NA	3
Sockeye salmon	NA	10

Fishes were euthanized in tricane methanosulfonate before they were measured in length to the nearest millimeter and weighed to the nearest 0.01 gram (McIntyre, 2004). Otoliths and scales were removed for aging. Individual fish were wrapped in aluminum foil and stored in plastic bags at  $-20^{\circ}\text{C}$  until they were analyzed for contaminants.

Samples were processed at the King County Environmental Laboratory (KCEL) located in Seattle, Washington. Large fish were cut into pieces while partially frozen then homogenized with liquid nitrogen in a Hobart™ buffalo chopper. Equipment was cleaned with methanol before each sample was homogenized.

Homogenized wet samples were ground with diatomaceous earth to absorb excess water. High molecular weight (DBC) and low molecular weight (TCX) surrogates were added along with a 50:50 methylenechloride-acetone solvent. The sample was cleaned first by gel permeation chromatography (GPC) then further cleaned by Alumina. A small aliquot was set aside for analysis of pesticides (DDT, chlordane, hexachlorocyclohexane). The remainder was digested with sulphuric acid and reduced in volume. Quality assurance and control measures included method blanks (diatomaceous earth + surrogates + solvents), spike blanks (method blank + analytes), two matrix spikes (spike blank + tissue), and a laboratory duplicate (method blank + tissue). Accepted variability was 100% between duplicates and  $\pm 50\%$  for all recoveries. One small northern pikeminnow had too little volume of sample for analysis.

### **Analytical Methods**

All fish tissue was analyzed (in 6 batches) as individual whole fish. With the exception of sockeye salmon and smallmouth bass, fishes were collected and assessed by size (small and large cutthroat trout and northern pikeminnow, or small, medium and large yellow perch). Total mercury was analyzed by cold vapor atomic absorption (CVAA) using a modified EPA Method 245.6. Methylmercury was analyzed by Frontier Geosciences using a KOH-methanol digestion followed by gas chromatography and measurement by cold vapor atomic fluorescence spectroscopy (CVAFS). Organochlorine analyses followed standard protocols used by KCEL (gas chromatography with electron capture detection (GC-ECD)) and were reported as chlordane, DDT, and PCBs.

## Data Analysis

Summary statistics were calculated for compounds measured in each fish species and size class within a given species. Statistics included the mean and median values, standard deviation and error, minimum and maximum values, 95% confidence interval, sample size, and detection frequency.

All chemical concentrations were expressed in parts per billion (ppb) wet weight, unless stated otherwise. Chlordane concentrations were expressed as the sum of alpha and gamma chlordane. DDT concentrations were expressed as the sum of p,p' congeners of DDT, DDE, and DDD. PCBs were expressed as the sum of Aroclors 1254 and 1260 (Aroclors 1016, 1221, 1232, 1242, and 1248 were analyzed but not detected). Mercury was expressed as total mercury (organic and inorganic forms). Non-detected samples were assigned the value of ½ the corresponding detection limit. If samples were below a 10% detection frequency, they were not evaluated.

High detection frequencies were observed for each of the four contaminants detected. Arranging the samples into functional groups,  $\Sigma$ DDT and  $\Sigma$ PCB were detected in all predatory fish samples, and chlordanes were detected in 96% of predatory fish samples. In forage fishes,  $\Sigma$ DDT was detected in 86% of samples,  $\Sigma$ PCB in 96%, and chlordanes in 82%.

Metabolites of DDT (DDD and DDE) were detected more frequently than was DDT itself. Aroclor 1254 was detected more frequently than was Aroclor 1260. All predatory fish samples contained Aroclor 1254. Alpha-chlordane was more frequently detected than was gamma-chlordane. For all chemicals, detection frequencies were greater in predatory fish than in forage fish.

Relative magnitudes of the three organochlorine groups ( $\Sigma$ DDT,  $\Sigma$ PCB,  $\Sigma$ chlordanes) were similar among species, indicating that organochlorines behaved similarly relative to each other in terms of bioaccumulation. The most concentrated organochlorine was  $\Sigma$ PCB followed by  $\Sigma$ DDT and  $\Sigma$ chlordanes, in the average order of 13:3:1 across species.

## Results

Mean contaminant concentrations of chlordane, DDT, mercury, and PCBs were determined for five fish species by size class (Table 2, Figure 1). Highest mean concentrations of DDT, mercury, and PCBs were observed in large northern pikeminnow. Highest mean concentration of chlordane was observed in large cutthroat trout. Lowest mean concentrations for all chemicals were observed in sockeye salmon. For cutthroat trout, yellow perch, and northern pikeminnow, the highest mean concentrations for all chemicals were observed in the largest size class of each species. Mean fish length and mean contaminant concentrations with corresponding summary statistics were calculated for individual fish species by size class and are included in Appendix C, Tables C1 - C5.

**Table 2.** Summary information for fish sampled in 2001 - 2003 from Lake Washington, King County, Washington.

Species	Size Class	Mean Concentration in Whole Fish (ppb)*				Sample size
		Chlordane	DDT	Mercury	PCBs	
Smallmouth Bass	all	11.0	62.9	244.3	371.2	3
Cutthroat Trout	S (< 300mm)	15.0	47.4	42.8	79.2	10
	L (> 300mm)	44.3	168.0	175.6	377.4	10
Yellow Perch	S (< 200mm)	5.0	13.9	32.9	46.6	10
	M (201-271 mm)	9.6	48.5	86.8	66.4	10
	L (> 271 mm)	16.3	58.7	183.0	191.1	9
Northern Pikeminnow	S (< 300mm)	7.1	44.5	53.1	140.0	10
	L (> 300mm)	40.1	257.7	387.1	1071.4	10
Sockeye Salmon	all	ND	5.4	37.0	7.8	10

\* ppb = parts per billion (wet weight)

S = small, M = medium, L = large

Chlordane concentration is the sum of alpha and gamma chlordanes

DDT concentration is the sum of DDT, DDE, and DDD congeners

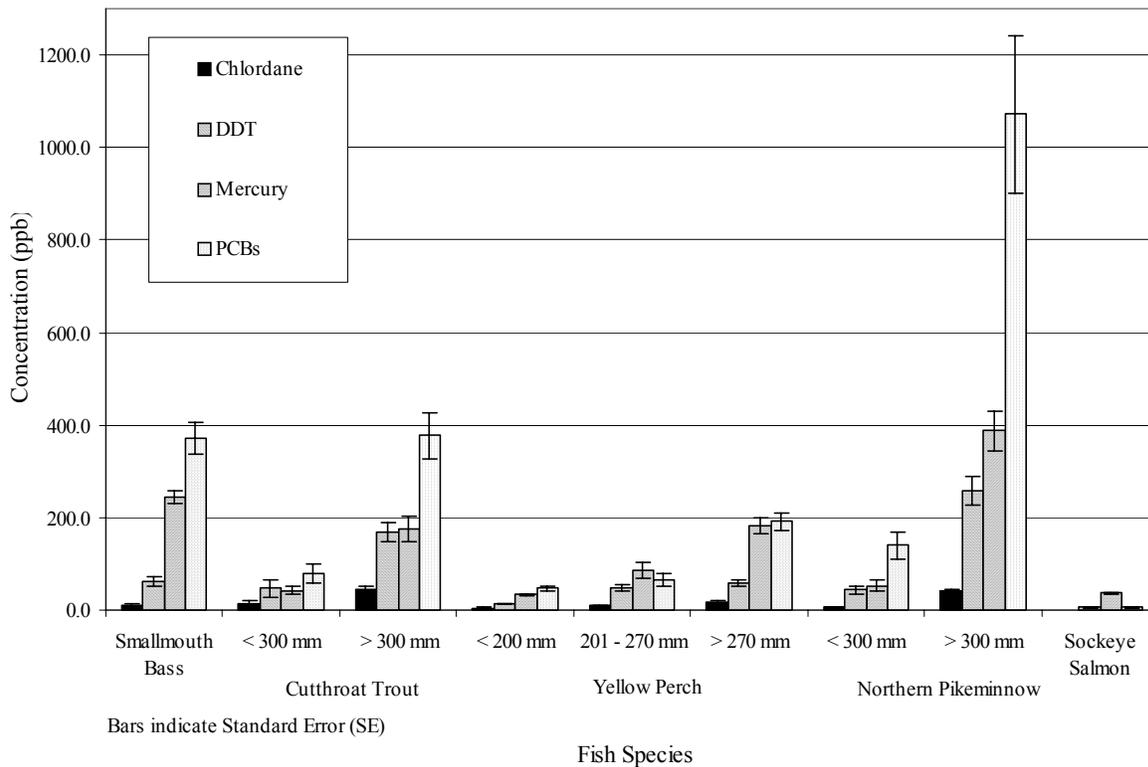
Mercury concentration is for total mercury (organic and inorganic)

PCB concentration is the sum of Aroclors 1254 and 1260

All nondetects were reported as 1/2 detection level (except where nondetected below 10%)

ND = nondetected value (below 10% detection frequency)

**Figure 1.** Mean contaminant concentrations in whole fish tissue samples from Lake Washington, King County (2001-2003)



While fish age is generally a better predictor than length in estimating contaminant levels in fish, fish age is not easily available or known to anglers. Therefore, fish length was used to establish size categories for assessment purposes. Contaminant concentrations for each chemical were

plotted in ppb (wet weight) versus fish length in millimeters (mm) for each species except smallmouth bass, which were excluded due to their small sample size (see Appendix C). For cutthroat trout, contaminants increased with greater fish length ( $p < 0.05$ ). For yellow perch, mercury and chlordane also increased with increased fish length ( $p < 0.05$ ). All contaminants increased with greater fish length for northern pikeminnow ( $p < 0.05$ ). Fish length was not correlated with contaminant levels in sockeye salmon.

### **Chlordane**

All species except sockeye had detectable levels of chlordane in whole body samples (Table 2, Figure 1, and Appendix C). For those species with detectable levels, alpha chlordane was the predominant form. Higher concentrations of chlordane were associated with increased fish size. Larger cutthroat trout and northern pikeminnow had the highest concentrations of total chlordane, with levels of 44.3 and 40.1 ppb, respectively.

### **Dichlorodiphenyltrichloroethane (DDT)**

Total DDT was detected in all fish species. Detection frequencies were high, nearly 100% for all species except for sockeye, which had a 60% detection frequency. A trend of increased total DDT concentration was observed with increased fish length (Table 2, Figure 1, and Appendix C). For all fish species, p,p'-DDE concentrations were greatest, followed by p,p'-DDD, and then p,p'-DDT. The larger size class of northern pikeminnow and cutthroat trout had the highest concentrations of total DDT at 257.7 and 168.0 ppb, respectively. Sockeye salmon had the lowest average concentration of total DDT at 5.4 ppb.

### **Mercury**

All fish species collected from Lake Washington had detectable levels of mercury in whole body samples. With the exception of sockeye salmon, an increase in mercury concentration was seen with increased fish length (Table 2, Figure 1, and Appendix C). Highest concentrations of total mercury were observed in smallmouth bass and large northern pikeminnow at 244.3 and 387.1 ppb, respectively. Small yellow perch, sockeye salmon, and small cutthroat trout all had total mercury concentrations below 50 ppb.

### **Polychlorinated biphenyls (PCBs)**

Total PCBs were based on Aroclors 1254 and 1260; other Aroclors were analyzed but not detected. Detection frequencies for total PCBs were greater than 90% for all species except for sockeye salmon (detection frequency of approximately 25%). With the exception of sockeye, total PCB concentrations tracked well with increased fish length (Table 2, Figure 1, Appendix C). A possible explanation for the lower association in sockeye may be due to their more uniform fish size relative to other species. As with the other contaminants, concentrations of total PCBs were lowest in sockeye salmon (7.9 ppb). Large northern pikeminnow exceeded 1000 ppb total PCBs. The next highest concentration of total PCBs was observed in large cutthroat trout at 377.4 ppb.

## **Discussion**

The following is a discussion of the possible human health risks associated with eating fish from Lake Washington. Estimates of current exposure are made based on assumptions about how much fish people eat from the lake. These estimates indicate that some Lake Washington fish eaters may be exposed to contaminants above a level of concern; therefore, allowable consumption rates are calculated below.

Also considered in the discussion are data limitations, including sample size and the use of whole body tissue (instead of fillets). Finally, other factors important to the process of providing advice to fish consumers regarding exposure to contaminants are discussed, such as multiple chemical exposure, background levels of contaminants, and benefits of eating fish.

### **Estimating Exposure**

To determine whether a contaminant in fish is a health risk, we must estimate the dose to which a person may be exposed. Dose is defined as the amount of a substance to which a person is exposed over some time period, usually expressed as milligrams per kilogram per day. A basic premise of toxicology is that the “dose makes the poison,” meaning that health risks usually increase with dose. In addition to our dose estimate, we also need information regarding what dose is toxic. The most sensitive endpoints of PCB toxicity include the immune system for the general population and developmental effects for the fetus and young children. For mercury, impacts on the developing fetus are of primary concern, leading to guidance for consumption of mercury-contaminated fish that is focused on women of childbearing age. This toxicity information is used to set allowable daily intakes, also known as oral reference doses (RfDs). RfDs are doses below which adverse health effects are not expected. Specific toxicity information on PCBs, mercury, DDT, and chlordane is presented in Appendix B.

The amount of fish consumed is a key parameter when estimating exposure to contaminants in fish. One way to establish this parameter is to conduct a consumption survey, asking how much fish is being eaten, how often, and which species is being consumed. Information on consumption is used to determine if consumers are exposed to a chemical above the RfD and focus risk communication on those populations.

King County DNRP recently released results of a human use survey for shoreline areas in Lake Union, Lake Washington, and Lake Sammamish (King County DNRP 2004). The study included information on areas of highest recreational use for Lake Washington (Gene Coulon Park, Magnuson Park, Seward Park, and Kennydale Park) and areas with the highest average number of anglers per visit (Clarke Beach, Stan Sayers Park, Gene Coulon Beach Park, and Mount Baker Park). One of three goals of the survey was to provide exposure information for use in human health risk assessment for Lake Washington, Lake Sammamish, and Lake Union. The survey focused on identifying fishing frequency and consumption patterns of anglers who used these lakes.

Anglers from Lake Washington fished from either the shoreline or from a boat at relatively equal rates. Approximately 98% of all anglers sought finfish rather than other aquatic organisms.

Most anglers fished only at the site where they were interviewed. The race of anglers in Lake Washington was predominately Caucasian (62%), Asian (14%), or African American (10%).

The overall mean fish consumption rate for anglers in Lake Washington was 10.8 grams/day (95<sup>th</sup> percentile = 30.2 grams/day) and for children of Lake Washington anglers was 9.5 grams/day (95<sup>th</sup> percentile reported as 86.2grams /day). Major fish species preferred by anglers in the three lakes included perch, trout, bass, and salmon. Anglers from Lake Washington, Lake Union, and Lake Sammamish consumed their catch an average of 1.3 times per month. Consumption patterns from this study suggest that the surveyed population does not rely on self-caught fish as a large portion of their diet (King County DNRP 2004).

### Comparison with Oral Reference Dose

To determine if consumers of Lake Roosevelt fish are exposed above the RfDs for each contaminant of concern, a dose was calculated for each contaminant using the overall mean fish consumption rate of 10.8 grams/day for Lake Washington anglers (converted to 11.6 ounces/month or 1.5 eight-ounce fish meals per month). This consumption rate was then used to estimate a dose for each contaminant, which was divided by its respective RfD to yield a ratio known as a hazard quotient. A ratio less than 1.0 would indicate no expected adverse health effects. If a dose exceeds its RfD, this indicates only the potential for adverse health effects. The magnitude of this potential can be inferred from the degree to which this value is exceeded. If the estimated exposure dose is only slightly above the RfD, then that dose will fall well below the toxic effect level. The higher the estimated dose is above the RfD, the closer it will be to the toxic effect level.

**Oral Reference Dose (RfD)**  
Oral reference doses (RfDs) are levels of exposure to chemicals below which non-cancer effects are not expected. EPA sets RfDs based on chronic exposure only. An RfD is derived by dividing a toxic effect level determined in animals or humans by “safety factors” to account for uncertainty and provide added health protection.

People who eat this amount or more of large cutthroat trout (> 300 mm), large yellow perch (>271 mm), and small (<300 mm) and large (>300 mm) northern pikeminnow would exceed the RfDs for both mercury and PCBs (Appendix D, Tables D1 – D5). Adults who eat small cutthroat trout, small and medium yellow perch, and sockeye salmon at the overall mean level of consumption do not exceed this protective level of exposure. Estimated exposures based on the 95<sup>th</sup> percentile consumption rate (King County DNRP 2004) were also determined and compared with the protective level of exposure. Adults who consume fish at the 95<sup>th</sup> percentile rate would exceed the RfD for all fish except sockeye salmon (Appendix D, Tables D6 – D10).

For all respondents, the ratio of mean estimated exposure to allowable daily dose (RfD) for PCBs ranged from 0.1 (sockeye salmon) to 9.6 (northern pikeminnow) (Table 3). For children, the ratio of mean estimated exposure to allowable daily dose (RfD) for PCBs ranged from 0.1 (sockeye salmon) to 8.5 (northern pikeminnow). Ratios calculated using the 95<sup>th</sup> percentile consumption rate for all consumers ranged from 0.2 (sockeye salmon) to 27 (northern pikeminnow). For children, ratios using the 95<sup>th</sup> percentile consumption rate ranged from 0.6 (sockeye salmon) to 77 (northern pikeminnow).

**Table 3.** Ratio of estimated total polychlorinated biphenyl (PCB) dose to EPA’s oral reference dose (RfD).

Fish Species	Total PCB Concentration (ppb)	Consumption Rate Ratio			
		All Respondents		Children (ages 0-18 yrs)	
		Mean (10.8 gm/day)	95th percentile (30.2 gm/day)	Mean (9.5 gm/day)	95th percentile (86.7 gm/day)
Smallmouth Bass	371.2	3.3	9.3	2.9	26.7
Large Cutthroat Trout	377.4	3.4	9.5	3	27.1
Large Yellow Perch	191.1	1.7	4.8	1.5	13.7
Large Northern Pikeminnow	1071.4	9.6	27	8.5	77
Sockeye Salmon	7.8	0.1	0.2	0.1	0.6

Estimated dose = (consumption rate x body weight)/fish tissue PCB concentration.

Body weight of 60 kg used to calculate dose.

Sockeye salmon was the only species where the ratio was less than one if consumed at average or 95<sup>th</sup> percentile rates. Bass, large cutthroat trout, large yellow perch, and large northern pikeminnow exceeded a ratio of 1.0 using both consumption rates. Thus, anglers who consume fish from Lake Washington at or above average rates as estimated by King County DNRP are exposed to contaminants at levels that may have a negative effect on human health.

Lake Washington is within the Muckleshoot Indian Tribe’s Usual and Accustomed fishing area. Tribal members most likely consume fish at a higher rate than the mean shown in the King County study. Native Americans from the Tulalip, Squaxin Island, and Suquamish Indian Nations have been shown to consume marine fish species from Puget Sound at much higher rates than the national average (6.5 grams/day) (Toy et al. 1996, Suquamish 2000). Surveys of Asian Pacific Islanders from King County also showed fish consumption rates higher than the national average (EPA 2000). Thus, the exposure estimates based on results from the recent King County survey may underestimate potential exposure to these populations.

### Determination of Allowable Consumption Rates

DOH used an approach similar to EPA’s risk-based method to characterize and evaluate risks from exposure to chemicals (EPA 2000). The DOH approach calculates an allowable monthly consumption rate based on the RfD, the body weight of an individual, and the known contaminant concentration in fish. Current weight-of-evidence suggests that non-cancer endpoints are more sensitive and, therefore, sufficiently protective for possible cancer effects.

By using the known concentration of a contaminant in a fish species, it is possible to calculate an allowable amount that can be consumed for that species without exceeding the RfD for that contaminant. In this approach, the RfD is used to calculate the quantity of fish a person of a given weight can safely consume given varying contaminant concentrations found in fish tissue. The equation used to calculate a safe consumption rate is shown below (EPA 2000). Note that the equation is solved for “grams of fish per day” which can then be converted into meals per month.

$$\frac{\text{Grams of Fish}}{\text{Day}} = (RfD \times \text{Body Weight} \times \text{Unit Conversion Factor}) \div (\text{Concentration in Fish})$$

Where:

Parameter	Value	Units	Source
Reference Dose (RfD)	Chemical specific	mg/kg-day	EPA IRIS
Unit Conversion Factor	1000	g/kg	
Body Weight	60 (adult female)	kg	EPA Exposure Factors Handbook
Concentration in fish	Mean contaminant concentration. Specific to water body and fish species.	mg/kg	Current Study

Based on this equation, there are two variables that affect the amount of fish a person can consume and stay below the RfD. These variables include the concentration of a contaminant in fish and an individual's body weight. Both the RfD and the unit conversion factors are constant; thus, reducing the consumption rate will reduce exposure. The consumption rate is expressed as grams of fish per day, which can be converted to allowable meals per month. The RfD is expressed on a microgram per kilogram per day basis. For general advice, an assumed body weight of 70 kg is commonly used in EPA risk assessments (EPA 1997, EPA 1999). For this assessment, DOH used an assumed body weight of an average woman of 60 kg (approximately 132 lbs). This weight was chosen to ensure that women of childbearing age are appropriately considered and protected when determining a consumption rate so as to be protective of neurological and developmental endpoints in the developing fetus.

### **Allowable Consumption Rates for Lake Washington Fish**

Allowable consumption rates were calculated for various size classes of northern pikeminnow, yellow perch, cutthroat trout, and sockeye salmon (Appendix C, Tables C6 – C9). Highlighted in each table is the most restrictive consumption rate for a given species. Based on whole fish analysis for each fish species, total PCB concentrations resulted in the lowest allowable consumption rates. Thus, if fish consumers follow consumption recommendations as determined by PCB concentrations, they would be protected from possible adverse health effects due to other contaminants.

Recommended meal consumption rates based on PCB concentrations ranged from 0.2 eight-ounce fish meals per month for northern pikeminnow to over twenty meals per month for sockeye salmon. For species with different size classes, more restrictive meal limits are observed in the larger fish class. Calculated meal limits based on contaminant concentrations measured in cutthroat trout, yellow perch, northern pikeminnow, and sockeye salmon collected from Lake Washington are given below.

**Northern Pikeminnow.** Northern pikeminnow recorded the highest concentrations for three of the four detected contaminants (DDT, mercury, and PCBs) (Appendix C, Tables C6a and C6b). Calculated meal limits are the most restrictive for any species due to the high chemical levels observed. The highest mean concentration of mercury was observed in large northern

pikeminnow resulting in a calculated meal limit of 2.1 meals per month. Concentrations of PCBs in both small and large northern pikeminnow resulted in allowable meal limits of 1.2 and 0.2 meals per month, respectively.

**Yellow Perch.** Three size classes of yellow perch were analyzed for contaminants. Since concentrations for all contaminants increased as yellow perch size increased, calculated meal limits decreased with increased fish size (Appendix C, Tables C7a, C7b, and C7c). The most restrictive meal limits were based on PCB concentrations (3.5 meals per month for small yellow perch, 2.4 for medium yellow perch, and 0.8 meals per month for large yellow perch). Mercury levels in small, medium, and large yellow perch resulted in meal limits of 24.4, 9.3 and 4.4 meals per month, respectively.

**Cutthroat Trout.** Calculated consumption rates decreased with increasing size of cutthroat trout (Appendix C, Tables C8a and C8b). In both size classes of cutthroat, PCBs levels gave the most restrictive meal limits at 2.0 meals per month for small cutthroat trout and 0.4 meals per month for large cutthroat trout.

**Sockeye Salmon.** Sockeye salmon had the lowest levels for all contaminants tested in this study. All calculated meal limits were above EPA's unrestricted level of 16 meals per month for a 60 kg person (Appendix C, Table C9). Consumption rates were not calculated for chlordane because of the low detection frequency (no samples were above the detection limit).

Estimated consumption rates calculated above were based on an average woman's body weight (60 kg or 132 lbs) (EPA 1997). The amount of a contaminant that a person can safely consume varies with body weight. For example, the greater a person's weight, the greater amount of fish the consumer may safely ingest. Conversely, the lower a person's weight, the fewer fish he/she may safely consume. An illustration of how meal limit calculations vary using differing body weights is given for chlordane, DDT, mercury, and total PCBs in Appendix C (Figs. C16 – C23). These graphs provide allowable meals per month for each contaminant with the understanding that DOH used meal limits based on PCB exposure as these were the most restrictive and, therefore, the most protective.

## **Data Uncertainty**

**Sample Size.** The sample size for each fish species in this assessment is small for a lake as large as Lake Washington and limited relative to assessments conducted by DOH in other state waterbodies. For example, 21 – 176 fish per species were collected for a health assessment in Lake Roosevelt (Munn et al 1995, DOH 2001a) and 13 - 95 fish per species were collected in Lake Whatcom, near Bellingham (DOH 2001b). In particular, the number of smallmouth bass sampled ( $n = 3$ ) is inadequate to evaluate possible health concerns associated with consumption of this species. The sample size was also small for other species; numbers for most size classes of fish included 10 individuals. Determination of appropriate sample size is dependent on several factors, including variability of contaminant data. Sufficient sample size should increase with increasing variability of chemical concentrations in fish. Lake Washington is a fairly large lake (87.6 km<sup>2</sup> and 35 km long), which might affect variability within a species. Given the lack

of previous contaminant data from Lake Washington, the degree of variability to adequately determine an appropriate sample size was unknown prior to this study.

In a related study, the UW obtained a much larger sample size of aquatic organisms for diet and stable isotope investigations. Results showed a great deal of homogeneity in food habits for a given size of fish for each species, lending additional support for conclusions based on low sample sizes for contaminants. Further, Lake Washington piscivores, particularly cutthroat trout and northern pikeminnow, are very mobile, which would integrate potential regional differences in contaminants. Nevertheless, DOH recommends further sampling of cutthroat trout, northern pikeminnow, and yellow perch based on the small number of samples and the large size of the lake.

**Whole Fish Analysis.** All fish tissue samples in the Lake Washington data set were analyzed using whole body measurements. Fish tissue data for this project were originally collected by the UW as part of a collaborative project to investigate bioaccumulation of contaminants through the Lake Washington food web, not for the purpose of evaluating human health. Typically, edible muscle tissue (fillet) is used to estimate potential human health risks from consuming fish. While some individuals or groups use whole fish in various cooking methods, fillet contaminant data provides more useful information on potential contaminant exposure levels to the general population.

Using whole fish tissue data would likely overestimate potential risks to the consumer since most lipophilic contaminants (those that concentrate in fat) concentrate in areas such as head, liver, and fat near the skin. Whole body-to-fillet ratios are generally less than one for lipophilic compounds such as chlordane, DDT, and PCBs. The reverse is seen for mercury, which concentrates in muscle tissue rather than fat. Using whole body samples would likely overestimate the amount of PCBs, chlordane, and DDT for fillets (the part of the fish usually consumed) while underestimating the amount of mercury. Whole body-to-fillet contaminant concentration ratios are often used to correct for this potential bias.

Although the current study did not measure whole body-to-fillet ratios, several studies have compared whole body-to-fillet data (Appendix C, Table C10). Whole body-to-fillet ratios ranged from a low of approximately 0.1 in walleye to 1.0 in brown trout. The average ratio for salmonid species is 0.68. Information was more limited for whole body-to-fillet ratios for bass, cyprinid, and percid species and, in general, averaged about 0.5. Ratios were not available in the literature for fish species collected from Lake Washington.

For comparative purposes, DOH estimated whole body-to-fillet average tissue concentrations based on ratios found in the literature for related fish species (Table 4). Estimated concentrations were then used to calculate meal limits for northern pikeminnow, yellow perch, and cutthroat trout (by size class). Smallmouth bass consumption guidelines fall under the statewide bass advisory mentioned earlier, and sockeye salmon from Lake Washington do not require an advisory. As expected, meal limits for all species based on estimated PCB concentrations in fillets (range: 0.3 – 6.9 meals per month) were higher than those based on whole body samples (range: 0.2 – 3.5 meals per month). However, advice based on adjusted meal limits would not change appreciably. DOH recommends future sampling and analyses of fish fillets from Lake

Washington species to determine more precise measurements of contaminant levels in tissue generally ingested by fish consumers.

**Table 4.** Comparison of meal limits based on measured total PCBs in whole body fish tissue from Lake Washington, Seattle, Washington, with estimated concentrations in fillets.

Fish Species/Size Class	Allowable 8 oz. meals per month based on PCB concentrations in:	
	Whole Fish	Fillet*
Cutthroat Trout < 300 mm**	2.0	3.0
Cutthroat Trout < 300 mm**	0.4	0.6
Yellow Perch < 200 mm***	3.5	6.9
Yellow Perch 200 - 271 mm***	2.4	4.9
Yellow Perch > 271 mm***	0.8	1.7
Northern Pikeminnow < 300 mm ***	1.2	2.3
Northern Pikeminnow < 300 mm ***	0.2	0.3

\* Estimated allowable meals per month of fillet base on the following whole:fillet ratios

\*\* Fillet concentrations estimated using a whole body:fillet ratio of 0.68

\*\*\* Fillet concentrations estimated using a whole body:fillet ratio of 0.50

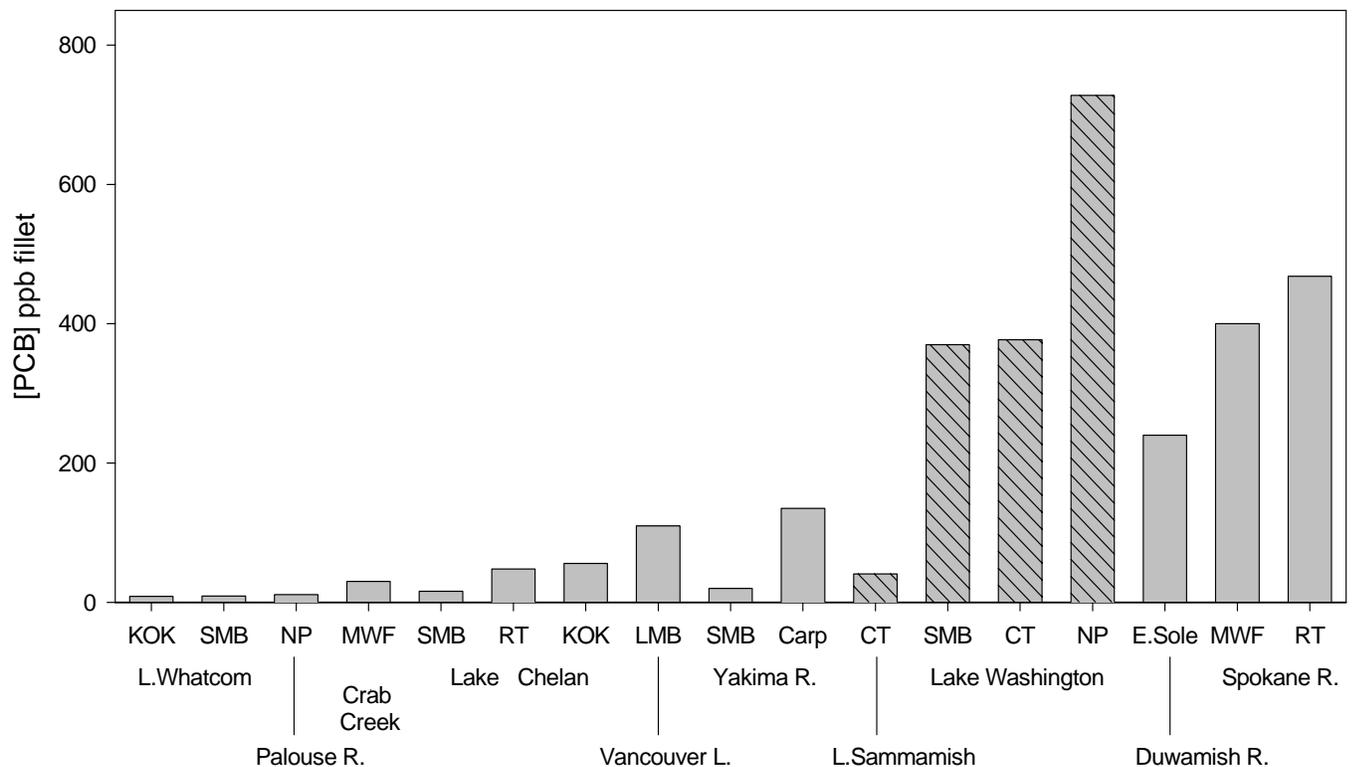
## Other Health Assessment Considerations

**Background Comparison.** Another consideration in this study is the magnitude of contaminant concentrations relative to known or estimated background concentrations. PCB concentrations are elevated in Lake Washington cutthroat trout and northern pikeminnow compared to fish from other Washington lakes and rivers. The current study also collected four cutthroat trout from Lake Sammamish > 300 mm, similar in size to large cutthroat trout collected from Lake Washington. The mean total PCB concentration of these fish was 60 ppb compared to 377.4 ppb for large cutthroat trout from Lake Washington. The large difference in concentrations in the same size fish from a neighboring waterbody suggests a localized rather than regional problem for total PCBs. A further explanation is the longer food chain in Lake Washington (compared with the food chain in Lake Sammamish) that could increase bioaccumulation in the lake through increased predation on predatory fishes (J. McIntyre, personal communication, 2004).

Studies are available with which to compare total PCB (Aroclor) levels in Lake Washington fish with fish from other fresh water bodies in Washington. Such comparisons are often complicated by differences in study design, such as analytical methods, detection limits, fish species, size, and tissue type (i.e., whole body versus fillet sample preparations). Despite these confounding factors, it is useful to consider PCB levels observed in various fish throughout the state.

Concentrations of  $\Sigma$ PCB in Lake Washington fish were many times higher than levels found in fish from many Washington State lakes and rivers, including Lake Sammamish (McIntyre 2004) (Figure 2). Levels of  $\Sigma$ PCB in Lake Washington fishes were comparable to those in fishes near PCB Superfund sites along the Lower Duwamish waterway and the Spokane River in Washington State. Levels of  $\Sigma$ PCB from fillets of fish previously sampled in Lake Union, a highly industrial site downstream of Lake Washington, were also assessed for comparison: yellow perch (~20  $\mu\text{g}/\text{kg}$ ), smallmouth bass (~50  $\mu\text{g}/\text{kg}$ ), and northern pikeminnow (~500  $\mu\text{g}/\text{kg}$ ) (McIntyre 2004).

**Figure 2.** PCB concentrations in large fishes from Washington State systems (Lake Whatcom, Palouse River, Crab Creek, Lake Chelan, Vancouver Lake, Yakima River, Lake Sammamish, Lake Washington, Lower Duwamish River, and Spokane River).



Hatched bars highlight data from the current study. L. Washington PCB values were multiplied by 0.68 to estimate fillet concentrations. KOK=kokanee, SMB=smallmouth bass, NP=northern pikeminnow, MWF=mountain whitefish, RT=rainbow trout, CT=cutthroat trout. Excluded from the comparison were sites undergoing ongoing assessment by the Washington State Department of Ecology for high levels of PCB and DDT under the Toxics Cleanup Program, including Walla Walla R., Chehalis R., and Okanogan R. PCBs at these sites ranged from 45 – 300 ppb.

Sources: (Davis et al. 1998; Serdar et al. 1999; Johnson 2001; EPA 2003b)

Figure provided by McIntyre, 2004.

**Multiple Chemical Exposure.** Different chemicals may interact in the body and increase or decrease the potential for adverse health effects. Since there are many chemicals in the environment, it is not possible to measure all interactions. However, the toxicity of contaminants with similar health endpoints should be considered additive unless information is available to suggest otherwise (ATSDR 2001a, ATSDR 2001b). In the case of contaminants measured in Lake Washington fish, mercury, PCBs and DDT impact childhood development following *in utero* exposure. Therefore, exposure doses were calculated assuming that the toxicity of PCBs, mercury, DDT and chlordane are additive by summing individual hazard quotients yielding a *hazard index*. This approach, however, did not significantly change estimated doses, or meal limits, calculated for PCB exposure alone demonstrating that efforts to reduce PCB exposure below its respective RfD will be protective with respect to each detected contaminant of concern.

Similarly, most cancer risk is attributed to PCBs. Using the mean consumption rate for Lake Washington anglers (10.8 g/day), estimated upper bound cancer risks for multiple chemicals ranged from 1.4 in one million (sockeye salmon) to 1.8 in ten thousand (large northern pikeminnow). These risks are within or close to the acceptable range of EPA's Superfund Program, which considers risks to be acceptable if within  $1 \times 10^{-4}$  to  $1 \times 10^{-6}$ . Our calculated cancer risks are upper bound estimates while actual risks are likely to be much less, possibly zero. Appendix D provides hazard indices and total cancer risk estimated for the combined exposures of each contaminant of concern.

**Sources of Contaminants.** The source of contaminants in Lake Washington fish is largely unknown. However, DDT was sprayed for mosquito control in the past, PCBs were widely used in industry prior to the 1970's, and mercury continues to be deposited aerially. King County DNRP recently collected sediment samples from 29 locations in Lake Washington. Although concentrations of most analytes were relatively low, there were some areas that exhibited sediment toxicity and an impaired benthic community. In addition, freshwater sediment guidelines were exceeded for PCBs, DDTs, mercury, and chlordane at some locations (D. Lester, personal communication, 2004). While 62% of the sites sampled (n = 29) had non-detected levels of PCBs (detection limits around 60 ppb dry weight; based on sum of Aroclors 1248, 1254, and 1260), the remaining sites where PCBs were detected had an average PCB concentration of > 200 ppb (dry weight) (range: 60 – 577 ppb dw). Sites with highest sediment concentrations were associated with combined sewage overflows (CSOs), storm drains, urban runoff, and past industrial spill locations adjacent to urban areas around the lake.

Sediment concentrations reported by King County DNRP agree well with those for areas of the Spokane River that had PCB concentrations in fish similar to those in Lake Washington (S. O'Neill, personal communication, 2004). But average total PCB sediment concentrations in Puget Sound urban bays like Elliott Bay and Sinclair Inlet (372 ppb dry weight (n = 496) and 148 ppb dry weight (n = 267), respectively) and in a known contaminated area, the Lower Duwamish (439 ppb dry weight, n = 1,079) are higher than in Lake Washington (M. Dutch, personal communication, 2004) (Puget Sound summaries were calculated on detected and undetected values). Thus, sediment levels in 40% of sampled sites in Lake Washington are elevated, but not to the highest level observed in state waterbodies. Nevertheless, these levels have resulted in demonstrable bioaccumulation in fish from the lake.

**Estimates of cancer risk.** The toxicological endpoint, or point of impact, is an important factor in determining potential effects from exposure to a chemical. Health effects that are transient, reversible, or have low severity are treated differently in a health assessment than those that produce long lasting, severe, or irreversible effects, such as those caused by PCBs and mercury. PCBs have been associated with impaired neurological development and with adverse effects on the immune system.

Another consideration in health assessments is the relative importance of cancer versus non-cancer endpoints. Current weight-of-evidence based on scientific studies that evaluated possible adverse human health effects from exposure to chlordane, DDT, mercury, and/or PCBs supports the use of non-cancer endpoints over the use of cancer endpoints in calculation of meal limit recommendations. The use of non-cancer endpoints does not disregard possible cancer

endpoints but rather places greater weight to observed and measured adverse effects rather than high dose extrapolation to probabilistic cancer endpoints. The most compelling argument against the use of cancer endpoints is in the lack of significant evidence of cancer in occupational exposure settings where exposure to contaminants is likely to be greatest both in terms of magnitude and duration. While evidence of cancer in occupational settings may not be significant, this may be due to confounding factors rather than true lack of significance. A summary of cancer classifications for chlordane, DDT, mercury, and PCBs is in Appendix E.

Recommendations based on a chronic dose, the RfD, also protect against possible cancer endpoints at risks typically used in a regulatory framework. To check this assertion, we used calculated meal limits from this study's results based on the RfD to determine potential cancer risks for each fish species (by size category). Estimated cancer risks ranged from  $1.6 - 8.2 \times 10^{-5}$ . These cancer risk estimates fall within EPA's risk range for Superfund of  $1 \times 10^{-4}$  to  $1 \times 10^{-6}$ . In short, protecting against known non-cancer will protect consumers from cancer endpoints as well.

**Benefits of fish consumption.** Recent studies have attempted to quantify risks of eating contaminated fish with benefits associated with their ingestion (Rembold 2004, Tuomisto et al. 2004, Lund et al. 2004, Sakamoto 2004). Further work is expected on this subject as more reports on fish contaminant levels and human health become available. At present, we know that fish is 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. Health benefits of eating fish have been associated with low levels of saturated versus unsaturated fats. Saturated fats are linked with increased cholesterol levels and risk of heart disease while unsaturated fats (e.g., omega-3 polyunsaturated fatty acid) are an essential nutrient.

Fish also provide a good source of vitamins and minerals. Health benefits of eating fish are well documented and linked to the reduction of cardiovascular disease, osteoporosis, and partial reduction of certain types of cancer. These major chronic diseases afflict much of the U.S. population. Replacing fish in the diet with other sources of protein such as red meat brings other considerations such as the link between saturated fat intake and cardiovascular disease. Advisories can be protective yet acknowledge benefits of eating fish, by recommending decreased consumption of fish known to have high concentrations of contaminants in favor of fish that are lower in contaminants. The American Heart Association and the U.S. Food and Drug Administration recommend two servings (12 oz.) of fish per week as part of a healthy diet.

Health benefits of eating fish deserve particular consideration when dealing with groups that consume fish for subsistence. Removal of fish from the diet of subsistence consumers may have serious health, social and economic consequences. Such populations are encouraged to consume a variety of fish species, to fish from locations with low contamination, and to follow recommended preparation and cooking methods. Further, consuming fillets rather than whole fish may reduce potential risks by another 50% (Appendix C, Table C10).

## **Conclusions and Recommendations**

Data from Lake Washington analyzed in this report are limited. Specifically, the sample size is small and the use of whole body fish tissue may overestimate exposure to contaminants. Regardless of data limitations, PCB concentrations in northern pikeminnow are some of the highest recorded in Washington, and concentrations in large yellow perch and large cutthroat trout are elevated relative to many areas throughout the state and a cause for concern.

Based on estimates of consumption for Lake Washington anglers and the concentrations of contaminants in fish discussed above, DOH estimates that the average angler is exposed to contaminants of concern above recommended levels. In order to protect consumers of Lake Washington fish, DOH provides the following recommendations.

- Eat a variety of fish as part of a balanced diet. Health benefits of eating fish are:
  - Fish is an excellent low-fat food, a great source of protein, vitamins, and minerals.
  - The oils in fish are important for unborn and breastfed babies.
  - Eating a variety of fish helps to reduce your chances of stroke or heart attack.
- Northern pikeminnow should not be consumed.
- Yellow perch greater than 270 mm (10½ inches) may be consumed as an eight-ounce meal once per month. Yellow perch smaller than 270 mm (10½ inches) may be consumed as an eight-ounce meal four times per month.
- Consumers of large cutthroat trout (>300 mm) (12 inches) from Lake Washington should eat no more than one eight-ounce meal per month. For small cutthroat trout (< 300 mm) (12 inches), no more than 3 eight-ounce meals per month are recommended.
- No meal restrictions on sockeye salmon from Lake Washington. Consumers are encouraged to choose sockeye when consuming local fish.
- Prior to the issuance of this interim advisory, a statewide fish consumption advisory for large and smallmouth bass due to mercury was in place throughout water bodies in Washington State, including Lake Washington. Women of childbearing age and children six years of age or younger should eat no more than two meals per month of any bass caught in Washington state freshwaters.

The recommendations given above are based on a 60 kg (132 lbs) adult eating an eight-ounce meal. In general, children should eat proportionally smaller meal sizes. Calculations for multiple chemical exposures do not change the above advice.

Since the above recommendations are based on a small sample size, DOH recommends additional sampling of northern pikeminnow, yellow perch, and cutthroat trout to confirm initial findings of high contaminant concentrations in fish tissue. We recommend sampling whole body fish and fillet samples with skin off for comparison with initial data. Other species of fish consumed by anglers such as rainbow trout, crappie, and bluegill should also be sampled.

## References

ATSDR 2001a. Guidance Manual for the Assessment of Joint Toxic Action of Chemical Mixtures. Draft. U.S. Department of Health and Human Services, Public Health Service. Agency for Toxic Substances and Disease Registry. February 2001.

ATSDR 2001b. Interaction Profile for Persistent Chemicals Found in Fish. Draft. U.S. Department of Health and Human Services, Public Health Service. Agency for Toxic Substances and Disease Registry. February 2001.

Bartoo, N. 1972. The vertical and horizontal distributions of northern squawfish, peamouth, yellow perch, and adult sockeye salmon in Lake Washington. M.S. Thesis, University of Washington, Seattle, WA.

Brocksmith, R. 1999. Abundance, feeding ecology and behavior of a native piscivore northern pikeminnow (*Ptychocheilus oregonensis*) in Lake Washington. MS Thesis. University of Washington, Seattle, WA. 104 pgs.

DOH 2001a. Exposure analysis of five consuming populations for overexposure to methylmercury. January 2001. Washington State Department of Health, Office of Environmental Health Assessments. Olympia, WA.

DOH 2001b. Lake Whatcom Fish Advisory. May 2001. Washington State Department of Health, Office of Environmental Health Assessments. Olympia, WA.

Dutch M, personal communication, 2004. Washington State Department of Ecology.

Ecology 2003. Mercury in Edible Fish Tissue and Sediments from Selected Lakes and Rivers of Washington State. Washington State Department of Ecology, Office of Environmental Assessment Program. June 2003. Publication No. 03-03-026.

EPA 1997. Exposure Factors Handbook. Volume 1 – General Factors. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment. August 1997. EPA/600/P-95/002Fa.

EPA 1999. Exposure Factors Handbook (EFH). U.S. Environmental Protection Agency, Office of Research and Development. February 1999. EPA/600/C-99/001.

EPA 2000. Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories. Volume 2 – Risk Assessment and Fish Consumption Limits, Third Edition. U.S. Environmental Protection Agency, Office of Water. November 2000. EPA 823-B-00-008.

King County DNRP 2004. Results of a Human Use Survey for Shoreline Areas of Lake Union, Lake Washington, and Lake Sammamish. Sammamish-Washington Analysis and Modeling Program (SWAMP). King County Department of Natural Resources and Parks, Water Treatment Division. April 2004.

- Lester D. 2004. Personal communication. King County Department of Natural Resources.
- Lund E, Engeset D, Alsaker E, Skeie G, Hjartaker A, Lundebye AK, and Niebor E. Cancer risk and salmon intake. *Science*. July 23, 2004;305(5683):476-7.
- McIntyre J. 2004. Bioaccumulation of mercury and organochlorines in Lake Washington. MS Thesis. University of Washington. Seattle, WA. 204 pgs.
- McIntyre J. 2004. Personal communication. University of Washington.
- Munn M, Cox S, and Dean C. 1995. Concentrations of mercury and other trace elements in walleye, smallmouth bass, and rainbow trout in Franklin D. Roosevelt Lake and the upper Columbia River, Washington, 1994. U.S. Geological Survey, Tacoma, WA. 95-195.
- Nowak, G. 2000. Movement patterns and feeding ecology of cutthroat trout (*Oncorhynchus clarki clarki*) in Lake Washington. MS thesis. University of Washington. Seattle, WA 75 pgs.
- O'Neill S. 2004. Personal communication. Washington State Department of Fish and Wildlife.
- Rembold 2004. Health benefits of eating salmon. *Science*. July 23, 2004;305(5683):475.
- Sakamoto M, Kubota M, Liu XJ, Murata K, Nakai K, and Satoh H. 2004. Maternal and Fetal Mercury and n-3 Polyunsaturated Fatty Acids as a Risk and Benefit of Fish Consumption to Fetus. *Environ. Sci. Technol.* 2004. 38:3860-3863.
- Suquamish 2000. Fish Consumption Survey of the Suquamish Indian Tribe of the Port Madison Indian Reservation, Puget Sound Region. The Suquamish Tribe, Port Madison Indian Reservation, Fisheries Department, Suquamish, WA. August 2000.
- Tabor R. and Chan J. 1997. Predation on sockeye salmon fry by piscivorous fishes in southern Lake Washington, 1996. US Fish & Wildlife Service. Western Washington Fishery Resource Office. Olympia, WA.
- Toy KA, Polissar NL, Liao S, and Gawne-Mittelstaedt GD. 1996. A Fish Consumption Survey of the Tulalip and Squaxin Island Tribes of the Puget Sound Region. Tulalip Tribes, Natural Resources Department, Marysville, WA. October 1996.
- Tuomisto JT, Tuomisto J, Tainio M, Niittynen M, Verkasalo P, Vartiainen T, Kiviranta H, and Pekkanen J. Risk-benefit analysis of eating farmed salmon. *Science*. July 23, 2004;305(5683):476-7.
- Warner E. 2004. Personal communication. Muckleshoot Indian Tribes.

# APPENDIX A

## Description of Lake Washington Fish Species

### **Cutthroat Trout (*Oncorhynchus clarki*)**

The cutthroat population in Lake Washington is thought to be wild, self-sustaining and mostly non-anadromous (anadromous means migrating up rivers from the ocean to spawn in fresh water) (Nowak 2000). A recent study showed that cutthroat trout generally live below the thermocline when the lake is stratified (the thermocline is the region of rapid decrease in temperature in a lake separating the upper warmer layer from the cooler lower layer) (Nowak 2000). Large cutthroat trout spend most of their time offshore to minimize contact with the warmer near-surface water, while in winter and spring they move near to shore, perhaps to prey on juvenile sockeye and Chinook salmon that migrate from streams. Cutthroat in Lake Washington do not inhabit localized areas within the lake but tend to move to various locations, most likely following prey, searching for food, or moving to more favorable conditions. Cutthroat trout consume a large number of Chinook fry and fingerlings, and predation by cutthroat trout appears to be responsible for substantial losses of juvenile sockeye salmon in Lake Washington. Average fork length of cutthroat in the littoral zone is 195.0 mm and in the limnetic zone is 366.4 mm (Nowak 2000).

### **Northern Pikeminnow (*Ptychocheilus oregonensis*)**

The northern pikeminnow is long-lived (up to age 19 in Montana) and reaches a large size (up to 25 inches) (Wydoski and Whitney 1979). Between 1996-1997, the mean length of northern pikeminnow in Lake Washington was 382.8 mm (15.07 inches). Sexual maturity is attained in about 6 years at a length of about 12 inches. The fish is edible, but the flesh is very bony. The most recent estimate of population numbers in Lake Washington ranged from 148,000 to 183,000 fish (Brocksmit 1999).

Northern pikeminnow prefer warm water. Fish appear to congregate near the mouth of the Cedar River in relatively high densities and near the mouth of the Lake Washington Ship Canal (Brocksmit 1999). The diet of northern pikeminnow varies with season, with salmon pre-smolts, smolts, and longfin smelt comprising a large part of the diet (smolts are young salmon at the life stage when they migrate from freshwater to the sea). In Lake Washington, the northern pikeminnow plays an important role as a sockeye salmon predator (Brocksmit 1999).

### **Yellow Perch (*Perca flavescens*)**

Historically, yellow perch was an important percentage of overall fish harvest from lakes in King County. This fish was first released into Lake Washington in the early 1900s (E. Warner, personal communication, 2004). The flesh of perch is firm, white, and mild in flavor (Wydoski and Whitney 1979). Young perch feed in the pelagic zone on zooplankton, and as they grow, they shift to shallower areas and feed on immature insects and mysid shrimp. Larger perch feed on forage fish when they are available. Yellow perch are often found in water 15 to 25 feet deep, making them easy to catch from shore. In Lake Washington, yellow perch range from approximately 3.8 inches at one year of age to 13.1 inches at 7 years of age. Males become mature between 1 and 2 years of age while females mature between 2 and 3 years of age.

### **Smallmouth Bass (*Micropterus dolomieu*)**

Smallmouth bass were first introduced to the western U.S. in 1874 (Wydoski and Whitney 1979). Smallmouth bass usually have a defined home range and do not travel over long distances. They prefer water temperatures of 70° – 80° F. Smallmouth bass fry eat crustaceans such as copepods and cladocerans and, when still small (between one and two inches), change to a diet of insects and begin to eat fishes. Smallmouth bass usually mature when 3 or 4 years old and, as adults, feed on insects, crayfish, and fishes.

### **Sockeye Salmon (*Oncorhynchus nerka*)**

Sockeye salmon are the second most abundant salmon on the Pacific coast and account for about 25 percent of the commercial salmon catch (Wydoski and Whitney 1979). Opportunities to fish recreationally for sockeye salmon in Lake Washington are limited because in most years the numbers returning to the lake are too low to permit a fishery (generally once every three to four years).

Sockeye differ from other species of salmon because they require a lake environment for part of their life cycle. Lake Washington sockeye return to spawn after 1 to 3 years of ocean life. About 90 percent of the fish that return have spent 1 year in fresh water and 2 - 3 years in the ocean. Although adult sockeye salmon may reach a length of 33 inches and a weight of 15.5 pounds, most adult fish weigh 3.5 to 8 pounds. Adult sockeye arrive in Lake Washington starting in early June, peaking in early July, and declining rapidly in early August. Fish remain in the lake for several months before migrating up streams and rivers to spawn. In the Cedar River, the first spawners may arrive by mid-August with the main spawning period lasting from September to early December, peaking in mid-October. The Cedar River is the largest sockeye spawning area in the basin, but over a third of the sockeye can spawn elsewhere (e.g., Bear/Cottage Creek system, Issaquah, and Lake Washington). Spawning occurs on Lake Washington beaches between early November and mid-January, peaking in mid-November. Most young fry migrate from the Cedar River into Lake Washington between January and June (peak migration from mid-February through April) and stay in the lake for 12 to 15 months until the majority become smolts and migrate to sea (D. Beauchamp, personal communication, 2004). Many smolts leave a year “early” as young-of-the-year smolts and a small percentage leaves the lake a year late as two-year-old smolts. Most sockeye smolts are from 110 – 150 mm (4.3 – 5.9 inches) long, with a maximum length of 190 mm (7.5 inches) (E. Warner, personal communication, 2004).

During their fresh-water life, juvenile sockeye salmon feed largely on zooplankton, especially crustaceans. While at sea, sockeye feed mainly on planktonic foods such as crustaceans, especially euphausiid (mysid) shrimp.

### **Other Species**

Piscivorous fish found in Lake Washington but not collected in this study include rainbow trout/steelhead (*Oncorhynchus mykiss*), coho salmon (*Oncorhynchus kisutch*), Chinook salmon (*Oncorhynchus tshawytscha*), Pacific lamprey (*Lampetra tridentata*), river lamprey (*Lampetra ayresi*), largemouth bass (*Micropterus salmoides*), brown bullhead (*Ameiurus nebulosus*), prickly

sculpin (*Cottus asper*), coast range sculpin (*Cottus aleuticus*), black crappie (*Pomoxis nigromaculatus*), warmouth (*Chaenobryttus gulosus*), bull trout (*Salvelinus confluentus*), and Dolly Varden (*Salvelinus malma*) (Tabor and Chan 1997). Some of the above species may have a much higher proportion of fish in their diet than others. The following are much less piscivorous or non-piscivorous fish found in the lake that were not included in the study: Western brook lamprey (*Lampetra richardsoni*), mountain whitefish (*Prosopium williamsoni*), peamouth (*Mylocheilus caurinus*), largescale sucker (*Catostomus macrocheilus*), longfin smelt (*Spirinchus thaleichthys*), three-spine stickleback (*Gasterosteus aculeatus*), pumpkinseed (*Lepomis gibbosus*), bluegill (*Lepomis macrocheilus*), weather loach (*Misgurnus angillicaudatus*), common carp (*Cyprinus carpio*), and tench (*Tinca tinca*) (E. Warner, personal communication, 2004).

## References

- Beauchamp, D. 2004. Personal communication. University of Washington, Seattle.
- Brocksmith, R. 1999. Abundance, feeding ecology, and behavior of a native piscivore northern pikeminnow (*Ptychocheilus oregonensis*) in Lake Washington. MS Thesis. University of Washington, Seattle, WA. 104 pgs.
- Nowak, G. 2000. Movement patterns and feeding ecology of cutthroat trout (*Oncorhynchus clarki clarki*) in Lake Washington. MS thesis. University of Washington. Seattle, WA. 75 pgs.
- Tabor R. and Chan J. 1997. Predation on sockeye salmon fry by piscivorous fishes in southern Lake Washington, 1996. US Fish & Wildlife Service. Western Washington Fishery Resource Office. Olympia, WA.
- Warner, E. 2004. Personal communication. Muckleshoot Indian Tribes. Auburn, Washington.
- Wydoski R. and Whitney R. 1974. Inland fishes of Washington. University of Washington Press. Seattle and London. 220 pgs.

# APPENDIX B

## Contaminants of Concern

## Introduction

The following is a summary of information on the background, exposure, and toxicity of four detected contaminants ( $\Sigma$ chlordane,  $\Sigma$ DDT, mercury, and total PCBs) in five fish species (northern pikeminnow (*Ptychocheilus oregonensis*), yellow perch (*Perca flavescens*), cutthroat trout (*Oncorhynchus clarki*), smallmouth bass (*Micropterus dolomieu*), and sockeye salmon (*Oncorhynchus nerka*)) collected from Lake Washington. This section represents a synopsis of information from ATSDR documents, EPA IRIS, and journal articles.

## Chlordane

### Background

Chlordane is a man-made chemical that was used as a pesticide in the United States from 1948 to 1988. Chlordane is not a single chemical but is a mixture of many related chemicals, of which about 10 are major components. Some major components are trans-chlordane, cis-chlordane,  $\beta$ -chlordane, heptachlor, and trans-nonachlor. For the first thirty years of its production, chlordane was used as a pesticide on agricultural crops, lawns, and gardens and as a fumigating agent. Mounting evidence of potential human exposure, persistence in the environment, detriments to wildlife, and knowledge of toxicity caused the U.S. Environmental Protection Agency (EPA) to phase out chlordane's use. For the last five years of its production, chlordane was approved for use only to control termites in and around homes. All approved uses within the U.S. for chlordane ended in 1988, although manufacture continues for export where it may be used in other countries (ATSDR 1994).

Chlordane enters the environment when used as a pesticide on crops, on lawns and gardens, and to control termites in houses. In soil, it attaches strongly to particles in the upper layers of soil and is unlikely to enter into groundwater. It is not known whether chlordane breaks down in most soils. However, if breakdown occurs, it is very slow and the chemical is known to remain in some soils for over 20 years. Most chlordane is lost from soil by evaporation. In water, some chlordane attaches strongly to sediment and particles in the water column and some is lost by evaporation. It is not known whether much breakdown of chlordane occurs in water or in sediment. Although chlordane breaks down in the atmosphere by reacting with light and with some chemicals in the atmosphere, it is sufficiently long-lived that it may travel long distances and be deposited on land or in water far from its source. Chlordane and its breakdown products accumulate in some form in the fat of fish, birds, mammals, and almost all humans.

### Exposure

Today, people receive the highest (but not the most common) exposure to chlordane from living in homes that were treated with chlordane for termites. Although houses built since 1988 have not been treated with chlordane for termite control, over 50 million persons have lived in chlordane-treated homes. Chlordane may be present in the air of these homes for many years after treatment. Indoor air in the living spaces of treated homes has been found to contain average levels of between 0.00003 and 0.002 milligram (mg) of chlordane in a cubic meter of air

(mg/m<sup>3</sup>). Levels as high as 0.06 mg/m<sup>3</sup> have been measured in the living areas of treated homes, and even higher levels have been found in basements and crawl spaces (ATSDR 1994).

The most common (but not the highest) source of chlordane exposure is from ingesting chlordane-contaminated food. Due to its high affinity from lipids, chlordane is almost never detected in drinking water. A survey conducted by the Food and Drug Administration (FDA) determined daily intake of chlordane from food to be 0.0013 microgram per kilogram of body weight (µg/kg) for infants and 0.0005 - 0.0015 µg/kg for teenagers and adults (a microgram is one thousandth of a milligram). The average adult would, therefore, consume about 0.11 µg of chlordane per day.

The amount of chlordane that enters the body depends on the amount in air, food, or water, and the length of time a person is exposed. Most chlordane that enters the body leaves in a few days, mostly in feces, and a much smaller amount leaves in urine. Chlordane and its breakdown products (metabolites) may be stored in body fat, where they cause no adverse effects unless released from body fat. It may take months or years before chlordane and its metabolites stored in fat are able to leave the body.

Chlordane and its breakdown products can be measured in human blood, urine, feces, and breast milk and measurements have shown that most Americans have low levels of chlordane metabolites in their body fat. The breakdown products can stay in body fat for very long periods, so finding them in body fat or breast milk does not tell how much or how long ago exposure to chlordane occurred.

## **Toxicity**

Most health effects in humans linked to chlordane exposure are on the nervous system, the digestive system, and the liver. These effects have been observed mostly in people who swallowed chlordane mixtures. No harmful effects on health have been confirmed in studies of workers who produced chlordane. One study found minor changes in liver function in workers in Japan who used chlordane as a pesticide. There are indications that chlordane may cause anemia and other changes in blood cells, but the evidence is not very strong.

The EPA guidelines for drinking water suggest that no more than 60 ppb chlordane should be present for longer than 10 days in drinking water that children consume. Drinking water should contain no more than 0.5 ppb for children or 2 ppb for adults if they drink the water for longer periods. FDA has established that levels of chlordane and its breakdown products in most fruits and vegetables should not be greater than 300 ppb, while animal fat and fish should not exceed 100 ppb.

EPA's Fish Advisory Guidance Manual (2000) states that, "Multiple neurological effects have been reported in humans exposed both acutely and chronically to chlordane." Adults (n = 109 women and n = 97 men) exposed to uncertain levels of chlordane from air and ingestion showed significant (p < 0.05) differences in neurophysiological and neuropsychological function tests. Profiles of mood states such as tension, depression, anger, vigor, fatigue and confusion were affected significantly (p < 0.0005) compared to "a referent population." The RfD listed in IRIS

for chlordane is  $5.0 \times 10^{-4}$  mg/kg-d based on a NOAEL of 0.15 mg/kg-d for hepatic necrosis in a 2-yr feeding study in mice (EPA IRIS 1999). FDA action level for chlordane in fish tissue is 300 ppb.

Chlordane is classified as B2 (probable human carcinogen) using the Guidelines for Carcinogen Risk Assessment (EPA 1986). The oral cancer slope factor is 0.35 ug/kg/day. Under the 1996 Proposed Guidelines, it would be characterized as a likely carcinogen by all routes of exposure. These characterizations are based on the following summaries of the evidence available: (1) human epidemiology studies showing non-Hodgkin's lymphoma in farmers exposed to chlordane and case reports of aplastic anemia (chlordane data associated with home use are inadequate to demonstrate carcinogenicity); (2) animal studies in which benign and malignant liver tumors were induced in both sexes of four strains of mice and liver toxicity but no tumors in rats of two strains; and (3) structural similarity to other rodent liver carcinogens.

## **Dichlorodiphenyltrichloroethane (DDT)**

### **Background**

DDT is a pesticide that was once used to control insects on agricultural crops. It was also used to control insects that carry diseases like malaria and typhus, but it is now used in only a few countries to control malaria (ATSDR 2002). Technical grade DDT is a mixture of three forms, p,p'-DDT, o,p'-DDT, and o,o'-DDT. All of these are white, crystalline, tasteless, and almost odorless solids. DDT may also contain p,p'-Dichlorodiphenyldichloroethylene (DDE) and p,p'-Dichlorodiphenyldichloroethane (DDD) as contaminants. DDD was used to a lesser extent than DDT to kill pests, and one form of DDD was used medically to treat cancer of the adrenal gland. DDE and DDD are breakdown products of DDT.

DDT does not occur naturally in the environment. The use of DDT was no longer permitted in the United States after 1972 except in the case of a public health emergency, but it is still used in some areas of the world for controlling malaria. Most DDT in the environment is a result of past use, but current use in other countries is still introducing DDT into the environment. DDE is only found in the environment as a result of contamination or breakdown of DDT. DDD also enters the environment during the breakdown of DDT.

DDT enters the atmosphere when it evaporates from contaminated water and soil and is then deposited on land or surface water. This cycle may be repeated many times, with the result that DDT, DDE, and DDD are carried long distances in the atmosphere, including Arctic and Antarctic regions.

DDT, DDE, and DDD persist in the soil for a very long time (decades), depending on many factors such as temperature, type of soil, and whether the soil is wet. In surface water, DDT binds to particles, settles, and is deposited in the sediment. It can accumulate to high levels in fish and marine mammals, with the highest levels found in adipose tissue. DDT in soil can also be absorbed by some plants and by animals or people who eat those crops.

## **Exposure**

Since the ban on DDT in the US and other parts of the world, environmental concentrations of DDT and metabolites have decreased. Average adult intakes of DDT have fallen over the years, as levels in food items have decreased. However, there are still measurable quantities of DDT, DDE, and DDD in many food groups. Mean concentrations of DDT in fish as measured by FDA between 1991 and 1999 range from 0.2-9.2 ppb (ATSDR 2002). People who eat fish caught in the Great Lakes consume larger amounts of DDT in their diets than average; however, as levels of DDT in the environment decline this exposure route is also expected to decline. At this time, low levels of DDT, DDE, and DDD are expected to be present in the diet for several more decades (ATSDR 2002).

DDT and its metabolites accumulate in adipose tissue. Indigenous peoples of the arctic are considered at risk to DDT exposure since their diets are particularly high in fatty tissues from marine mammals. Another route of potential exposure of DDT to children is through breast-feeding.

## **Toxicity**

Most information on health effects in humans comes from studies of workers in plants that manufacture DDT or applicators who spray DDT over an extended period (ATSDR 2002). DDT impairs nerve impulse conduction. Observed effects vary from mild altered sensations to tremors and convulsions. DDT is also capable of inducing alterations on reproduction and development in animals, an effect attributed to the alteration of hormones. The o,p'-DDT isomer has the strongest estrogen-like properties. The p,p'-DDE isomer has anti-androgenic properties and can alter development of reproductive organs in rats (ATSDR 2002). An RfD of  $5.4 \times 10^{-4}$  mg/kg/d was established based on liver effects in rats.

Animal studies have shown that DDT, DDE, and DDD can cause cancer in the liver. There is no conclusive evidence to link DDT to cancer in humans, although possible genotoxic effects have been reported. EPA assigned DDT, DDE, and DDD a weight-of-evidence classification of B2, probable human carcinogens (EPA IRIS 2001). An oral slope factor of  $0.34 \text{ (mg/kg-day)}^{-1}$  was derived for DDT. IARC assigned a weight-of-evidence classification of B2 to DDT, possibly carcinogenic to humans (IARC 2002).

## **Mercury**

### **Background**

Mercury is widespread in the environment as a result of natural and anthropogenic releases. Everyone is exposed to small amounts of mercury (Clarkson 1993, and Clarkson 1997, in Goldman and Shannon, 2001). Most mercury in the atmosphere is elemental mercury vapor and inorganic mercury, and mercury in water, soil, plants, and animals is in organic or inorganic forms. Organic mercury is primarily in the form of methylmercury.

Mercury is released into surface waters from natural weathering of rocks and soils from volcanic activity. Mercury is also released from human action such as industrial activities, fossil fuel burning, and disposal of consumer products. Global cycling of mercury via air deposition occurs when mercury evaporates from soils and surface waters to the atmosphere. From the atmosphere, mercury is redistributed on land and surface water then absorbed by soil or sediments. Once inorganic mercury is released into the environment, bacteria convert it into organic mercury, the primary form that accumulates in fish and shellfish (ATSDR 1999).

## **Exposure**

In the aquatic food chain, methylmercury biomagnifies as it is passed from lower to higher trophic levels through consumption of prey organisms. Fish at the top of the food chain can biomagnify methylmercury approximately 1 to 10 million times greater than concentrations in the surrounding waters. Nearly all of the mercury found in fish is in the methylmercury form. Predatory ocean fish that live for a long time may have increased methylmercury content because of exposure to natural and industrial sources of mercury (Goldman and Shannon 2001). Methylmercury content of fish varies by species and size of the fish as well as harvest location. The top ten commercial fish species represent about 85% of the seafood market and contain a mean mercury level of approximately 0.1 µg/g.

Some states have issued advisories about consumption of fish containing mercury. DOH issued a statewide fish consumption advisory for women of childbearing age and young children based on elevated levels of mercury in various commercially bought fish as well as freshwater bass caught for recreation (DOH 2003). <http://www.doh.wa.gov/fish/FishAdvMercury.htm>

## **Toxicity**

Most organic mercury compounds are readily absorbed by ingestion and appear in the lipid fraction of blood and brain tissue. Organic mercury readily crosses the blood-brain barrier and also crosses the placenta. Fetal blood mercury levels are equal to or higher than maternal levels (Goldman and Shannon 2001). Methylmercury also appears in human milk. Organic mercury compounds are most toxic in the central nervous system and may also affect the kidneys and immune system (Clarkson 1993, and Clarkson 1997, in Goldman and Shannon, 2001).

Methylmercury is toxic to the cerebral and cerebellar cortex in the developing brain and is a known teratogen. In Minamata Bay, Japan, mothers who were exposed to high amounts of mercury but were asymptomatic gave birth to severely affected infants. The infants often appeared normal at birth but developed psychomotor retardation, blindness, deafness, and seizures over time. Since the fetus is susceptible to neurotoxic effects of methylmercury, several studies have focused on subclinical effects among children whose mothers were exposed to high levels of methylmercury. A study in Iraqi children exposed to high levels of methylmercury in contaminated seeds demonstrated motor retardation in children whose mothers had hair mercury levels ranging from 10-20 ppm. Two prospective epidemiologic studies were conducted in the Seychelles and the Faroe Islands. Results from the Faroe Islands suggest that exposure in utero to mercury at lower levels is associated with subtle adverse effects on the developing brain (maximum level in hair was 39.1 ppm and in blood was 351 ppb). Memory, attention, and

language tests were inversely associated with higher methylmercury exposures in children up to 7 years of age (Grandjean et al. 1997, Goldman and Shannon 2001). In the Seychelles study, adverse effects on development or IQ have not been found up to 66 months of age. The Faroe Islands and Seychelles studies are continuing, in order to provide a long-term developmental evaluation of exposed children. Further support for the developmental effects seen in Faroese children is demonstrated in a study of New Zealand children exposed *in utero* to methylmercury consumed in fish by their mothers.

In 1998, the National Academy of Sciences (NAS) was directed by the United States Congress to evaluate methylmercury toxicity and provide recommendations on exposure limits (NRC 2000). The study established a reference dose for mercury of 0.1 µg/kg-day. The EPA has recently re-confirmed 0.1 µg/kg-day as its Reference Dose (RfD) (EPA IRIS, 2003). This RfD is based on health effects data specific to the protection of the developing fetus. As the developing fetus represents the population of greatest concern, the RfD is considered protective of all other populations that are less exposed and/or less sensitive. The current action level of FDA for mercury in fish tissue is 1 ppm (1000 ppb).

## **Polychlorinated Biphenyls (PCBs)**

### **Background**

PCBs are persistent environmental contaminants that are ubiquitous in the global environment due to intensive industrial use. PCBs were used as commercial mixtures (Aroclors) that contain up to 209 different chlorinated biphenyl congeners, which are structurally similar compounds that vary in toxicity. Each congener has a biphenyl ring structure but differs in the number and arrangement of chlorine atoms substituted around the biphenyl ring. PCBs are lipid soluble and are stable; their stability depends on the number of chlorine atoms and the position of the chlorine atoms on the biphenyl molecule. Their lipophilic character and resistance to metabolism enhances concentration in the food web and exposure to humans and wildlife.

PCBs were produced commercially in the U.S. from the 1930's to 1977 and sold primarily as mixtures under the trade name Aroclor. The name Aroclor 1254, for example, means that the molecule contains 12 carbon atoms (the first 2 digits) and approximately 54% chlorine by weight (second 2 digits) (ATSDR 2000). Each mixture (1016,1242,1254, and 1260) contained many different PCB congeners. In 1971, the sole US producer of PCBs (Monsanto Chemical Company) voluntarily stopped open-ended uses of PCBs and in 1977 ceased their production. Because PCBs do not burn easily and are good insulators, they were commonly used as lubricants and coolants in capacitors, transformers, and other electrical equipment. Old capacitors and transformers that contain PCBs are still in operation. Over the years, PCBs have been spilled, illegally disposed, and leaked into the environment from transformers and other electrical equipment. PCBs in the environment have decreased since the 1970's but are still detectable in our air, water, soil, food, and in our own bodies.

The breakdown of PCBs in water and soil occurs over many years. The lower chlorinated PCBs are more easily broken down in the environment, while adsorption of PCBs generally increases as chlorination of the compound increases. The highly chlorinated Aroclors (1248, 1254, and

1260) resist both chemical and biological degradation in the environment. Microbial degradation of highly chlorinated Aroclors to lower chlorinated biphenyls has been reported under anaerobic conditions, as has the mineralization of biphenyl and lower chlorinated biphenyls by aerobic microorganisms. Although they are slow processes, volatilization and biodegradation are the major pathways of removal of PCBs from water and soil (ATSDR 2000). In water, photolysis appears to be the only viable chemical degradation process. The chemical composition of the original Aroclor mixtures released to the environment changes over time since the individual congeners degrade and partition at different rates (ATSDR 2000).

Many PCB congeners persist in ambient air, water, marine sediments, and soil at low levels throughout the world. The half-life of PCBs (the time it takes for one-half of the PCBs to breakdown) in the air is 10 days or more, depending on the type of PCB. PCBs in the air can be carried long distances and may be deposited onto land or water. Once in water, most PCBs tend to stick to organic particles and sediments.

In Lake Washington and other waterbodies, PCBs in sediments are taken up in the bodies of aquatic organisms, which are in turn consumed by creatures higher in the food web. Fish, birds, and mammals tend to accumulate certain congeners over time in their fatty tissue. Concentrations of PCBs can reach levels thousands of times higher than the levels in water. Bioconcentration is the uptake of a chemical from water alone, while bioaccumulation is the result of combined uptake via food, sediment, and water. These processes can lead to high levels in the fat of predatory animals (ATSDR 2000). Also, PCBs can biomagnify in fresh and saltwater ecosystems. Humans may be exposed to PCBs when they eat fish, use fish oils in cooking, or consume meat, milk or cheese.

## **Exposure**

The general population is exposed to PCBs through inhalation and ingestion of contaminated water and food. The dominant source of PCBs to humans is through consumption of seafood, meat, and poultry. Of particular concern is exposure through consumption of fish. Some groups may consume greater amounts of fish than others; for example, Native Americans, Asian immigrant populations and sport anglers are three groups with high rates of seafood ingestion in the Puget Sound area (Landolt et al. 1985, Landolt et al. 1987, Toy et al. 1996, EPA 1999, Suquamish 2000). Several studies have found PCBs in local seafood (Landolt et al. 1987, PSAMP 1997, O'Neill et al. 1998, West and O'Neill 1998, PSAMP 2000, O'Neill and West 2001, West et al. 2001).

## **Toxicity**

Toxic responses to PCBs include dermal toxicity, immunotoxicity, carcinogenicity, and adverse effects on reproduction, development, and endocrine functions. Some epidemiological studies indicate that consumption of fish containing PCBs may cause slight but measurable impairments in physical growth and learning behavior in children. Some PCB congeners have a structure and biological activity that is similar to dioxin. EPA has determined that PCBs are probable human carcinogens and assigned them the cancer weight-of evidence classification B2 based on animal studies. Human studies are being updated; current available evidence is inadequate, but

suggestive regarding cancer to humans. The upper-bound cancer slope factor for PCBs is 2.0 per (mg/kg)/day.

Part of the uncertainty in assessing PCB effects from consuming fish is that PCB congeners selectively bioaccumulate in fish in different patterns than found in commercial mixtures of PCBs (Schwartz et al 1987). The congener mix that a fetus would encounter during pregnancy and via nursing may be quite different than congener patterns initially released into the environment. Since PCB congeners differ in their potency and in the specific ways they interact with biological systems, health criteria based on data from Aroclor mixtures fed to animals (e.g., the EPA RfD) may not account for the effects of biodegradation that result in differing initial and final congener patterns. While some information on pattern changes is available in studies in the Great Lakes (Kostyniak et al., 1999 and Humphrey et al., 2000), data from Lake Washington did not include enough information to account for this occurrence (i.e., no congener data). This issue is one that is being investigated at a national and international level.

DOH recently conducted a thorough review of recent scientific literature in an attempt to set a state standard for exposure to PCBs through consumption of fish and shellfish. DOH concluded that ATSDR's minimal risk level (MRL) of 0.02 µg/kg-day for chronic-duration oral exposure to PCBs would be protective of the most sensitive population (fetus) for the most sensitive endpoints reviewed (immune and developmental). The chronic oral MRL is based on a lowest-observed adverse effect level (LOAEL) of 0.005 mg/kg-day for immunological effects seen in adult monkeys exposed to Aroclor 1254 (ATSDR 2000). EPA verified an oral reference dose (RfD) of 0.02 µg/kg-day for Aroclor 1254 (EPA IRIS 2000), based on dermal/ocular and immunological effects in monkeys. For comparison, FDA set residue levels in fish and edible shellfish as 2 mg/kg.

## References

ATSDR 1994. Toxicological Profile for Chlordane. U.S. Department of Health and Human Services, Public Health Service. Agency for Toxic Substances and Disease Registry. May 1994.

ATSDR 1999. Toxicological Profile for Mercury. U.S. Department of Health and Human Services, Public Health Service. Agency for Toxic Substances and Disease Registry. March 1999.

ATSDR 2000. Toxicological Profile for Polychlorinated Biphenyls (PCBs). U.S. Department of Health and Human Services, Public Health Service. Agency for Toxic Substances and Disease Registry. November 2000.

ATSDR 2002. Toxicological Profile for DDT, DDE and DDD. U.S. Department of Health and Human Services, Public Health Service. Agency for Toxic Substances and Disease Registry. September 2002.

Clarkson TW. 1993. Mercury: major issues in environmental health. *Environ Health Perspect.* 1993 Apr;100:31-8.

Clarkson TW. 1997. The Toxicology of Mercury. *Crit Rev Clin Lab Sci.* 1997 Aug;34(4):369-403.

DOH 2003. Statewide Bass Advisory. September 2003.  
[http://www.doh.wa.gov/ehp/oeas/publications%20pdf/statewide\\_%20bass\\_%20advisory-2003.pdf](http://www.doh.wa.gov/ehp/oeas/publications%20pdf/statewide_%20bass_%20advisory-2003.pdf).

EPA IRIS – Integrated Risk Information System  
<http://www.epa.gov/iris/index.html>.

EPA 1986. *Guidelines for Carcinogen Risk Assessment*. U.S. Environmental Protection Agency, Office of Research and Development. 1986. 51 Federal Register 33992 (September 24, 1986).

EPA 1996. *Proposed Guidelines for Carcinogen Risk Assessment*. U.S. Environmental Protection Agency, Office of Research and Development. April 1996. EPA/600/P-92/003C.

EPA 1999. Asian and Pacific Islander Seafood Consumption Study in King County, WA. U.S. Environmental Protection Agency, Office of Environmental Assessment. May 1999. EPA 910/R-99-003.

Goldman LR and Shannon MW. 2001. Technical Report: Mercury in the Environment: Implications for Pediatricians. *Pediatrics* 108:197-205, 2001.

Grandjean P, Weihe P, White R, Debes F, Araki S, Yokoyama K, Murata K, Sorensen N, Dahl R, and Jorgensen P. 1997. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicol. Teratol.* 19(6):417-428.

Humphrey H, Gardiner J, Pandya J, Sweeney A, Gasior D, McCaffrey R, and Schantz S. 2000. PCB congener profile in the serum of humans consuming Great Lakes fish. *Environ. Health Perspect.* 2000 Feb.;108(2):167-72.

IARC 2002. <http://193.51.164.11/htdocs/monographs/vol53/04-ddt.htm>

Kostyniak P, Stinson C, Greizerstein H, Vena J, Buck G, and Mendola P. 1999. Relation of Lake Ontario fish consumption, lifetime lactation, and parity to breast milk polychlorobiphenyl and pesticide concentrations. *Environ. Res.* 1999 Feb;80(2):S166-S174.

Landolt M, Hafer F, Nevissi A, van Belle G, Van Ness K, and Rockwell C. 1985. Potential toxicant exposure among consumers of recreationally caught fish from urban embayments of Puget Sound. NOAA Technical Memorandum NOS OMA 23. Rockville, MD.

Landolt M, Kalman D, Nevissi A, van Belle G, Van Ness K, and Hafer F. 1987. Potential toxicant exposure among consumers of recreationally caught fish from urban embayments of Puget Sound: Final Report. NOAA Technical Memorandum NOS OMA 33. Rockville, MD.

NRC 2000. Toxicological Effects of Methylmercury. Committee on the Toxicological Effects of Methylmercury, Board on Environmental Studies and Toxicology, Commission on Life Sciences. National Academy of Science National Research Council. National Academy Press. 2000.

O'Neill S, West J, and Hoeman J. 1998. Spatial trends in the concentration of polychlorinated biphenyls (PCBs) in Chinook (*Oncorhynchus tshawytscha*) and coho salmon (*O. kisutch*) in Puget Sound and factors affecting PCB accumulation: results from the Puget Sound Ambient Monitoring Program. In R.Strickland (ed.). Proceedings of the 1998 Puget Sound Research Conference. Puget Sound Water Quality Action Team, Olympia, WA.

O'Neill S, and West J. 2001. Exposure of Pacific Herring (*Clupea pallasii*) to persistent organic pollutants in the Puget Sound and the Georgia Basin. Puget Sound Research '01 Proceedings. Puget Sound Water Quality Action Team, Seattle, WA.

PSAMP 1997. Draft. Summary report of contaminants in Puget Sound fishes and factors affecting contaminant uptake and accumulation. Puget Sound Ambient Monitoring Program, Fish Monitoring Component, Marine Resources Division, Washington Dept. of Fish and Wildlife, Olympia, WA.

PSAMP 2000. Toxic Contaminants in Fish. In 2000 Puget Sound Update: Seventh Report of the Puget Sound Ambient Monitoring Program. Puget Sound Water Quality Action Team. Olympia, WA.

Schwartz P, Jacobson W, Fein G, Jacobson J, and Price H. 1983. Lake Michigan fish consumption as a source of polychlorinated biphenyls in human cord serum, maternal serum, and milk. *Am. J. Public Health.* 1983 Mar;73(3):293-6.

Suquamish 2000. Fish Consumption Survey of the Suquamish Indian Tribe of the Port Madison Indian Reservation, Puget Sound Region. The Suquamish Tribe, Port Madison Indian Reservation, Fisheries Department, Suquamish, WA. August 2000.

Toy KA, Polissar NL, Liao S, and Gawne-Mittelstaedt GD. 1996. A Fish Consumption Survey of the Tulalip and Squaxin Island Tribes of the Puget Sound Region. Tulalip Tribes, Natural Resources Department, Marysville, WA. October 1996.

West J, and O'Neill S. 1998. Persistent Pollutants and Factors Affecting their Accumulation in Rockfishes (*Sebastes* spp.) from Puget Sound, Washington. In E. R. Strickland, editor. Puget Sound Research '98 Proceedings. Puget Sound Water Quality Action Team, Seattle, WA.

West J, O'Neill S, Lippert G, and Quinnell S. 2001. Toxic contaminants in marine and anadromous fishes from Puget Sound, Washington: Results of the Puget Sound Ambient Monitoring Program fish component, 1989-1999. Technical Report FTP01-14, Washington Dept. of Fish and Wildlife, Olympia, WA.

# APPENDIX C

## Lake Washington Fish Contaminant Data and Analysis

**Table C1.** Summary statistics for length and contaminant concentrations in smallmouth bass.

<i>Smallmouth Bass</i>	<i>Length (mm)</i>	<i>Concentration in Whole Fish (ppb)*</i>			
		<i>Chlordane</i>	<i>DDT</i>	<i>Mercury</i>	<i>PCBs</i>
Mean	418.9	11.0	62.9	244.3	371.2
Standard Error	24.1	2.6	10.7	12.6	35.7
Median	405.8	10.9	69.7	251.0	385.0
Standard Deviation	41.8	4.5	18.5	21.8	61.9
Minimum	385.2	6.5	41.9	220.0	303.6
Maximum	465.7	15.5	77.1	262.0	425.0
Confidence Level (95.0%)	103.9	11.2	46.1	54.1	153.7
Sample Size	3	3	3	3	3
Detection Frequency	-	50%	100%	100%	100%

\* ppb = parts per billion (wet weight)

Chlordane conc. is the sum of alpha and gamma chlordanes

DDT conc. is the sum of DDT, DDE, and DDD congeners

Mercury conc. is for total mercury (organic and inorganic)

PCB conc. is the sum of Aroclors 1254 and 1260

All nondetects were reported as 1/2 detection level

**Table C2a.** Summary statistics for length and contaminant concentrations in cutthroat trout <300 mm.

<i>Cutthroat Trout</i> < 300 mm	<i>Length</i> (mm)	<i>Concentration in Whole Fish (ppb)*</i>			
		<i>Chlordane</i>	<i>DDT</i>	<i>Mercury</i>	<i>PCBs</i>
Mean	247.8	15.0	47.4	42.8	79.2
Standard Error	12.2	6.3	19.3	7.1	21.2
Median	239.7	9.1	22.9	37.0	52.7
Standard Deviation	38.5	20.0	60.9	22.6	67.2
Minimum	188.5	1.0	10.2	21.7	21.4
Maximum	294.6	68.3	210.6	98.4	239.5
Confidence Level (95.0%)	27.5	14.3	43.6	16.1	48.1
Sample Size	10	10	10	10	10
Detection Frequency	-	70%	100%	100%	95%

**Table C2b.** Summary statistics for length and contaminant concentrations in cutthroat trout >300 mm.

<i>Cutthroat Trout</i> > 300 mm	<i>Length</i> (mm)	<i>Concentration in Whole Fish (ppb)*</i>			
		<i>Chlordane</i>	<i>DDT</i>	<i>Mercury</i>	<i>PCBs</i>
Mean	429.1	44.3	168.0	175.6	377.4
Standard Error	14.0	7.9	21.5	26.3	49.4
Median	431.7	37.5	181.6	159.5	359.0
Standard Deviation	44.3	25.1	68.0	83.1	156.1
Minimum	350.0	2.3	14.6	35.0	38.8
Maximum	500.0	73.7	238.0	299.0	563.3
Confidence Level (95.0%)	31.7	17.9	48.7	59.5	111.6
Sample Size	10	10	10	10	10
Detection Frequency	-	70%	100%	100%	100%

\* ppb = parts per billion (wet weight)

Chlordane conc. is the sum of alpha and gamma chlordanes

DDT conc. is the sum of DDT, DDE, and DDD congeners

Mercury conc. is for total mercury (organic and inorganic)

PCB conc. is the sum of Aroclors 1254 and 1260

All nondetects were reported as 1/2 detection level

**Table C3a.** Summary statistics for length and contaminant concentrations in yellow perch <200 mm.

<b>Yellow Perch &lt; 200 mm</b>	<b>Length (mm)</b>	<b>Concentration in Whole Fish (ppb)*</b>			
		<b>Chlordane</b>	<b>DDT</b>	<b>Mercury</b>	<b>PCBs</b>
Mean	136.0	5.0	13.9	32.9	46.6
Standard Error	4.0	0.6	0.9	1.9	5.2
Median	134.5	4.4	13.6	33.3	37.9
Standard Deviation	12.7	1.8	2.9	5.9	16.3
Minimum	116.0	2.6	10.7	22.5	29.2
Maximum	158.0	8.3	18.9	41.3	74.9
Confidence Level (95.0%)	9.1	1.3	2.1	4.2	11.7
Sample Size	10	10	10	10	10
Detection Frequency	-	50%	93%	100%	90%

**Table C3b.** Summary statistics for length and contaminant concentrations in yellow perch 201 - 271 mm.

<b>Yellow Perch 201 - 271 mm</b>	<b>Length (mm)</b>	<b>Concentration in Whole Fish (ppb)*</b>			
		<b>Chlordane</b>	<b>DDT</b>	<b>Mercury</b>	<b>PCBs</b>
Mean	243.5	9.6	48.5	86.8	66.4
Standard Error	4.2	1.3	6.6	16.9	14.1
Median	238.0	9.2	48.4	65.6	50.7
Standard Deviation	13.3	4.2	20.7	53.4	44.7
Minimum	231.0	4.5	21.8	40.3	24.0
Maximum	271.0	18.9	87.5	207.0	166.9
Confidence Level (95.0%)	9.5	3.0	14.8	38.2	32.0
Sample Size	10	10	10	10	10
Detection Frequency	-	100%	100%	100%	95%

**Table C3c.** Summary statistics for length and contaminant concentrations in yellow perch >271 mm.

<b>Yellow Perch &gt; 271 mm</b>	<b>Length (mm)</b>	<b>Concentration in Whole Fish (ppb)*</b>			
		<b>Chlordane</b>	<b>DDT</b>	<b>Mercury</b>	<b>PCBs</b>
Mean	303.7	16.3	58.7	183.0	191.1
Standard Error	6.7	2.8	6.8	17.4	18.3
Median	302.0	13.4	64.9	180.0	184.5
Standard Deviation	20.2	8.4	20.4	52.3	55.0
Minimum	273.0	8.0	26.0	121.0	119.2
Maximum	332.8	34.8	84.3	293.0	286.6
Confidence Level (95.0%)	15.6	6.5	15.6	40.2	42.3
Sample Size	9	9	9	9	9
Detection Frequency	-	50%	100%	100%	100%

\* ppb = parts per billion (wet weight)

Chlordane conc. is the sum of alpha and gamma chlordanes

DDT conc. is the sum of DDT, DDE, and DDD congeners

Mercury conc. is for total mercury (organic and inorganic)

PCB conc. is the sum of Aroclors 1254 and 1260

All nondetects were reported as 1/2 detection level

**Table C4a.** Summary statistics for length and contaminant concentrations in northern pikeminnow <300 mm.

<i>Northern Pikeminnow</i> < 300 mm	<i>Length</i> (mm)	<i>Concentration in Whole Fish (ppb)*</i>			
		<i>Chlordane</i>	<i>DDT</i>	<i>Mercury</i>	<i>PCBs</i>
Mean	234.3	7.1	44.5	53.1	140.0
Standard Error	13.9	1.2	8.6	11.1	29.2
Median	234.5	7.0	38.2	35.1	118.0
Standard Deviation	43.9	3.6	25.7	35.2	87.7
Minimum	145.6	2.3	14.6	18.6	57.9
Maximum	297.3	13.8	90.3	106.0	325.0
Confidence Level (95.0%)	31.4	2.8	19.8	25.2	67.4
Sample Size	10	9	9	10	9
Detection Frequency	-	50%	96%	100%	100%

**Table C4b.** Summary statistics for length and contaminant concentrations in northern pikeminnow >300 mm.

<i>Northern Pikeminnow</i> > 300 mm	<i>Length</i> (mm)	<i>Concentration in Whole Fish (ppb)*</i>			
		<i>Chlordane</i>	<i>DDT</i>	<i>Mercury</i>	<i>PCBs</i>
Mean	458.7	40.1	257.7	387.1	1071.4
Standard Error	16.8	5.3	29.9	42.4	171.0
Median	444.3	37.3	274.7	334.0	998.0
Standard Deviation	53.0	16.7	94.5	134.0	540.8
Minimum	401.9	15.1	72.7	212.0	210.1
Maximum	568.0	68.3	436.4	598.0	2289.0
Confidence Level (95.0%)	37.9	11.9	67.6	95.8	386.8
Sample Size	10	10	10	10	10
Detection Frequency	-	50%	100%	100%	100%

\* ppb = parts per billion (wet weight)

Chlordane conc. is the sum of alpha and gamma chlordanes

DDT conc. is the sum of DDT, DDE, and DDD congeners

Mercury conc. is for total mercury (organic and inorganic)

PCB conc. is the sum of Aroclors 1254 and 1260

All nondetects were reported as 1/2 detection level

**Table C5.** Summary statistics for length and contaminant concentrations in sockeye salmon.

<b>Sockeye Salmon</b>	<b>Length (mm)</b>	<b>Concentration in Whole Fish (ppb)*</b>			
		<b>Chlordane</b>	<b>DDT</b>	<b>Mercury</b>	<b>PCBs</b>
Mean	606.2	1.0	5.4	37.0	6.3
Standard Error	12.5	0.0	0.7	1.4	1.4
Median	603.5	1.0	4.6	36.5	5.2
Standard Deviation	39.7	0.0	2.3	4.4	4.4
Minimum	553.0	1.0	3.2	30.2	2.5
Maximum	660.0	1.0	10.9	44.8	14.3
Confidence Level (95.0%)	28.4	0.0	1.7	3.2	3.1
Sample Size	10	10	10	10	10
Detection Frequency	-	0%	60%	100%	25%

\* ppb = parts per billion (wet weight)

Chlordane conc. is the sum of alpha and gamma chlordanes

DDT conc. is the sum of DDT, DDE, and DDD congeners

Mercury conc. is for total mercury (organic and inorganic)

PCB conc. is the sum of Aroclors 1254 and 1260

All nondetects were reported as 1/2 detection level

**Table C6a.** Calculated consumption rates of northern pikeminnow <300 mm for a 132 lb (60 kg) woman based on contaminant concentration.

Northern Pikeminnow < 300 mm				
Contaminant	RfD (ug/kg day)	Concentration in Whole Fish ppb (ug/kg)*	Consumption rate (ounces of fish per month)**	Consumption rate (number of 8 oz. meals per month)***
Chlordane	0.5	7.1	4536.9	567.1
DDT	0.5	44.5	723.9	90.5
Mercury	0.1	53.1	121.3	15.2
PCBs	0.02	140.0	9.2	1.2

**Table C6b.** Calculated consumption rates of northern pikeminnow >300 mm for a 132 lb (60 kg) woman based on contaminant concentration.

Northern Pikeminnow > 300 mm				
Contaminant	RfD (ug/kg day)	Concentration in Whole Fish ppb (ug/kg)*	Consumption rate (ounces of fish per month)**	Consumption rate (number of 8 oz. meals per month)***
Chlordane	0.5	40.1	803.3	100.4
DDT	0.5	257.7	125.0	15.6
Mercury	0.1	387.1	16.6	2.1
PCBs	0.02	1071.4	1.2	0.2

\* ppb = parts per billion (wet weight)

\*\* Consumption rate = (RfD x body weight)/(conc. in fish) x (30.4 days/month)

\*\*\* Number of 8 oz. meals per month that would put a 60 kg person at the RfD

RfD - EPA's chemical specific reference dose

Chlordane conc. is the sum of alpha and gamma chlordanes

DDT conc. is the sum of DDT, DDE, and DDD congeners

Mercury conc. is for total mercury (organic and inorganic)

PCB conc. is the sum of Aroclors 1254 and 1260

Highlighted value indicates most restrictive meal limit for each size class

**Table C7a.** Calculated consumption rates of yellow perch < 200 mm for a 132 lb (60 kg) woman based on contaminant concentration.

Yellow Perch < 200 mm				
Contaminant	RfD (ug/kg day)	Concentration in Whole Fish ppb (ug/kg)*	Consumption rate (ounces of fish per month)**	Consumption rate (number of 8 oz. meals per month)***
Chlordane	0.5	5	6442.3	805.3
DDT	0.5	13.9	2317.4	289.7
Mercury	0.1	32.9	195.2	24.4
PCBs	0.02	46.6	27.6	3.5

**Table C7b.** Calculated consumption rates of yellow perch 201 - 270 mm for a 132 lb (60 kg) woman based on contaminant concentration.

Yellow Perch 201 - 271 mm				
Contaminant	RfD (ug/kg day)	Concentration in Whole Fish ppb (ug/kg)*	Consumption rate (ounces of fish per month)**	Consumption rate (number of 8 oz. meals per month)***
Chlordane	0.5	9.6	3355.4	419.4
DDT	0.5	48.5	664.2	83.0
Mercury	0.1	86.8	74.2	9.3
PCBs	0.02	66.4	19.4	2.4

**Table C7c.** Calculated consumption rates of yellow perch > 271 mm for a 132 lb (60 kg) woman based on contaminant concentration.

Yellow Perch > 271 mm				
Contaminant	RfD (ug/kg day)	Concentration in Whole Fish ppb (ug/kg)*	Consumption rate (ounces of fish per month)**	Consumption rate (number of 8 oz. meals per month)***
Chlordane	0.5	16.3	1976.2	247.0
DDT	0.5	58.7	548.8	68.6
Mercury	0.1	183.0	35.2	4.4
PCBs	0.02	191.1	6.7	0.8

\* ppb = parts per billion (wet weight)

\*\* Consumption rate = (RfD x body weight)/(conc. in fish) x (30.4 days/month)

\*\*\* Number of 8 oz. meals per month that would put a 60 kg person at the RfD

RfD - EPA's chemical specific reference dose

Chlordane conc. is the sum of alpha and gamma chlordanes

DDT conc. is the sum of DDT, DDE, and DDD congeners

Mercury conc. is for total mercury (organic and inorganic)

PCB conc. is the sum of Aroclors 1254 and 1260

Highlighted value indicates most restrictive meal limit for each size class

**Table C8a.** Calculated consumption rates of cutthroat trout <300 mm for a 132 lb (60 kg) woman based on contaminant concentration.

<b>Cutthroat Trout &lt; 300 mm</b>				
<b>Contaminant</b>	<b>RfD (ug/kg day)</b>	<b>Concentration in Whole Fish ppb (ug/kg)*</b>	<b>Consumption rate (ounces of fish per month)**</b>	<b>Consumption rate (number of 8 oz. meals per month)***</b>
Chlordane	0.5	15.0	2147.4	268.4
DDT	0.5	47.4	679.6	84.9
Mercury	0.1	42.8	150.5	18.8
PCBs	0.02	79.2	16.3	2.0

**Table C8b.** Calculated consumption rates of cutthroat trout >300 mm for a 132 lb (60 kg) woman based on contaminant concentration.

<b>Cutthroat Trout &gt; 300 mm</b>				
<b>Contaminant</b>	<b>RfD (ug/kg day)</b>	<b>Concentration in Whole Fish ppb (ug/kg)*</b>	<b>Consumption rate (ounces of fish per month)**</b>	<b>Consumption rate (number of 8 oz. meals per month)***</b>
Chlordane	0.5	44.3	727.1	90.9
DDT	0.5	168.0	191.7	24.0
Mercury	0.1	175.6	36.7	4.6
PCBs	0.02	377.4	3.4	0.4

\* ppb = parts per billion (wet weight)

\*\* Consumption rate = (RfD x body weight)/(conc. in fish) x (30.4 days/month)

\*\*\* Number of 8 oz. meals per month that would put a 60 kg person at the RfD

RfD - EPA's chemical specific reference dose

Chlordane conc. is the sum of alpha and gamma chlordanes

DDT conc. is the sum of DDT, DDE, and DDD congeners

Mercury conc. is for total mercury (organic and inorganic)

PCB conc. is the sum of Aroclors 1254 and 1260

Highlighted value indicates most restrictive meal limit for each size class

**Table C9.** Calculated consumption rates of sockeye salmon for a 132 lb (60 kg) woman based on contaminant concentration.

Sockeye Salmon				
Contaminant	RfD (ug/kg day)	Concentration in Whole Fish ppb (ug/kg)*	Consumption rate (ounces of fish per month)**	Consumption rate (number of 8 oz. meals per month)***
DDT	0.5	5.4	5965.1	745.6
Mercury	0.1	37.0	174.1	21.8
PCBs	0.02	7.9	163.1	20.4

\* ppb = parts per billion (wet weight)

\*\* Consumption rate = (RfD x body weight)/(conc. in fish) x (30.4 days/month)

\*\*\* Number of 8 oz. meals per month that would put a 60 kg person at the RfD

RfD - EPA's chemical specific reference dose

DDT conc. is the sum of DDT, DDE, and DDD congeners

Mercury conc. is for total mercury (organic and inorganic)

PCB conc. is the sum of Aroclors 1254 and 1260

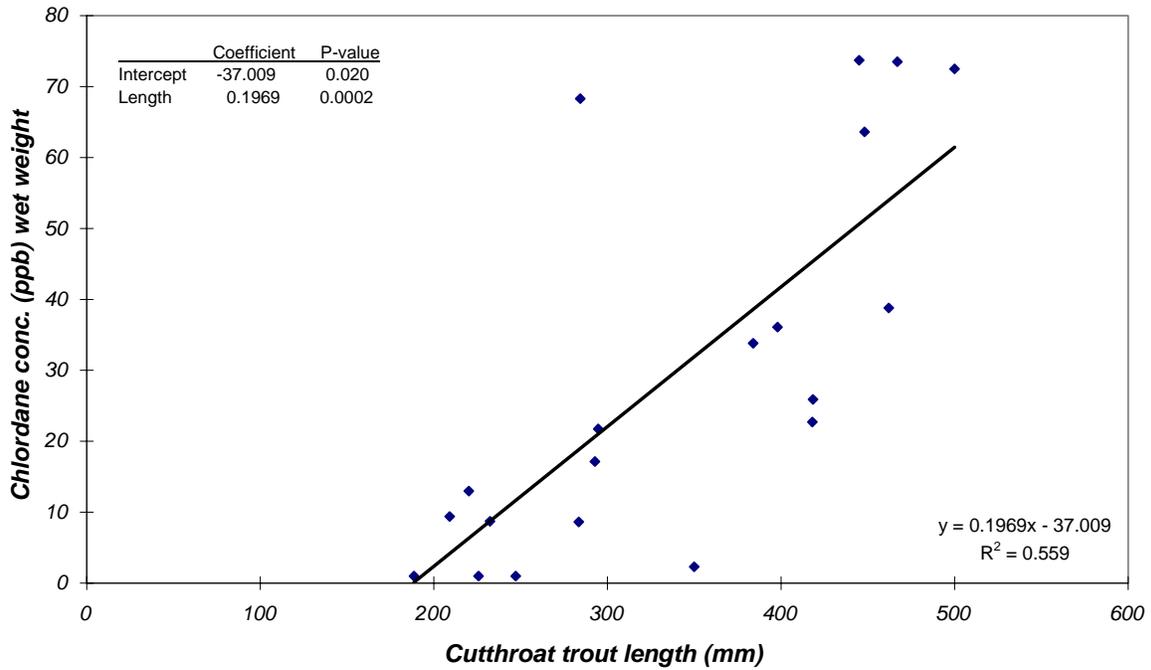
Highlighted value indicates most restrictive meal limit for each size class

**Table C10.** Fillet-to-whole body PCB ratios for various fish species.

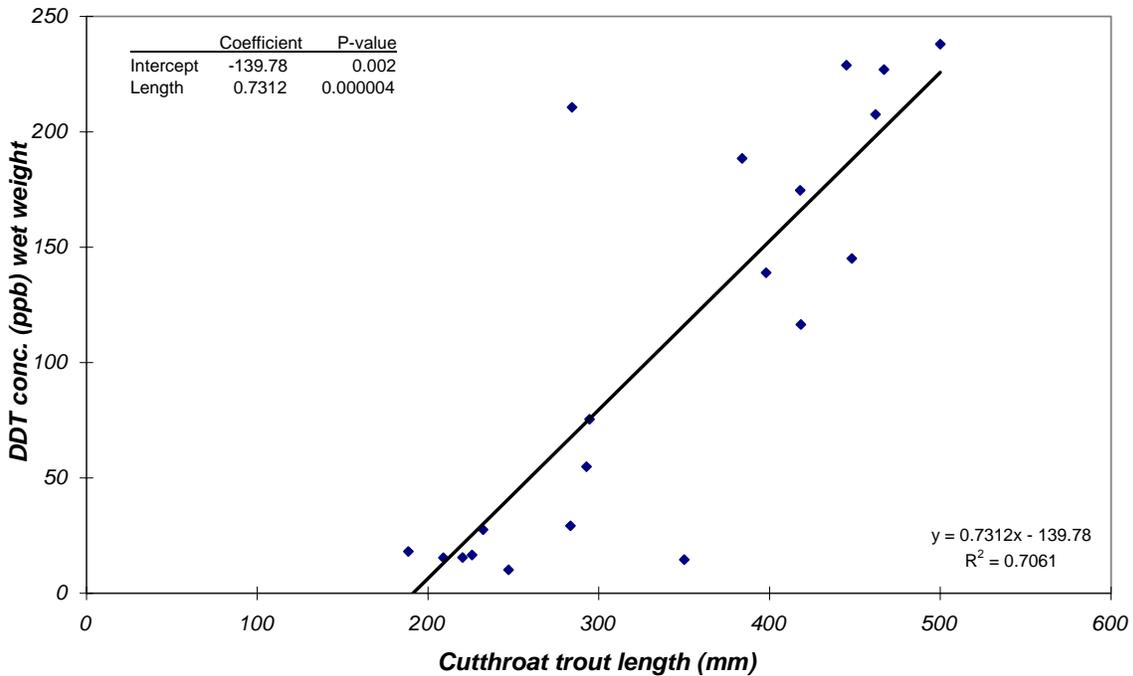
Fillet-to-Whole Body Ratios for PCBs			
Species	Group	Fillet:Whole Body Ratio	Reference
Black Bass	Bass	0.43	Bevelheimer et al. 1997
White Bass	Bass	0.44	Thompson et al. 2002
Carp	Cyprinid	0.53	Thompson et al. 2002
White sucker	Cyprinid	0.48	Thompson et al. 2002
Walleye	Percid	0.17	Thompson et al. 2002
Walleye	Percid	0.1	Parkerton 1993
Walleye	Percid	0.09	Connolly et al. 1992
Brown trout	Salmoid	1.0	Connolly et al. 1992
Brown trout	Salmoid	0.88	Connolly et al. 1992
Brown trout	Salmoid	0.57	Connolly et al. 1992
Coho	Salmoid	0.59	Amrhein et al. 1999
Coho	Salmoid	0.89	Connolly et al. 1992
Rainbow trout	Salmoid	0.68	Amrhein et al. 1999
Rainbow trout	Salmoid	0.34	Niimi and Oliver 1983
Rainbow trout	Salmoid	0.43	Carline et al. 2001

Table courtesy of Jan McIntyre

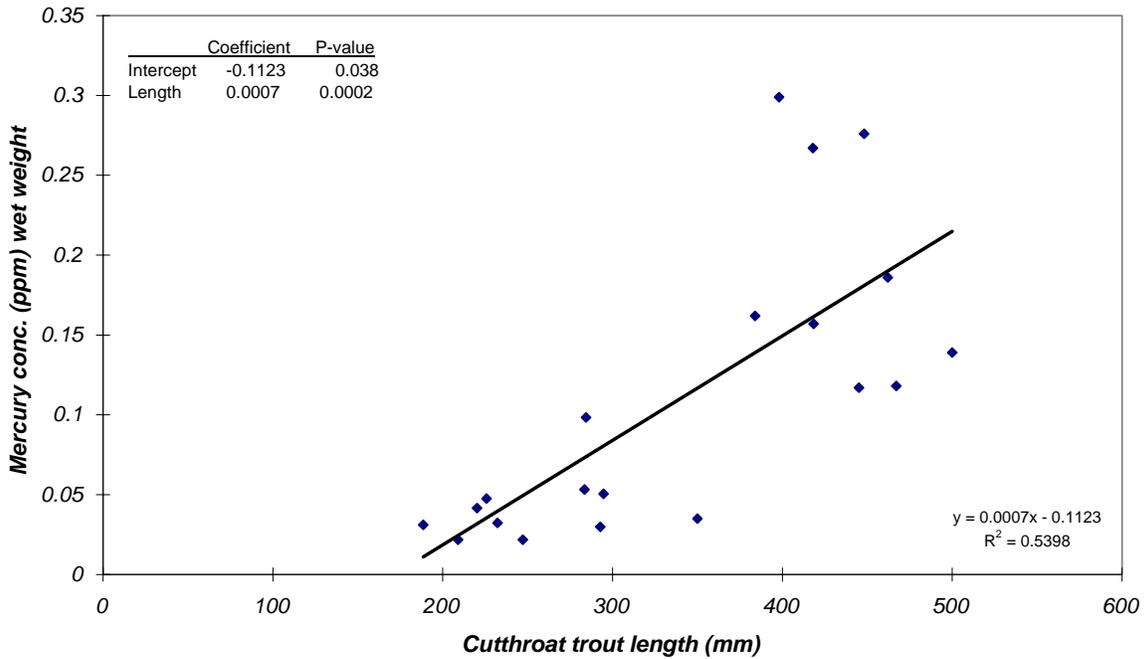
Data presented for discussion only, and where not used in determining consumption rates.



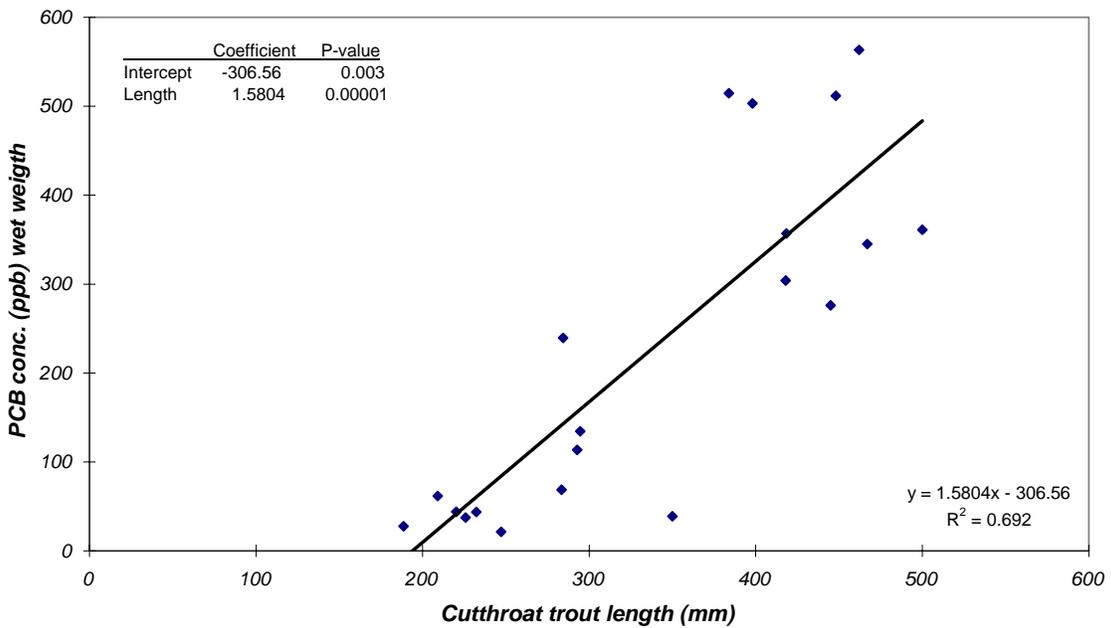
**Figure C1.** Correlation between cutthroat trout length (mm) and associated chlordane concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represents the sum of alpha and gamma chlordanes. N = 20.



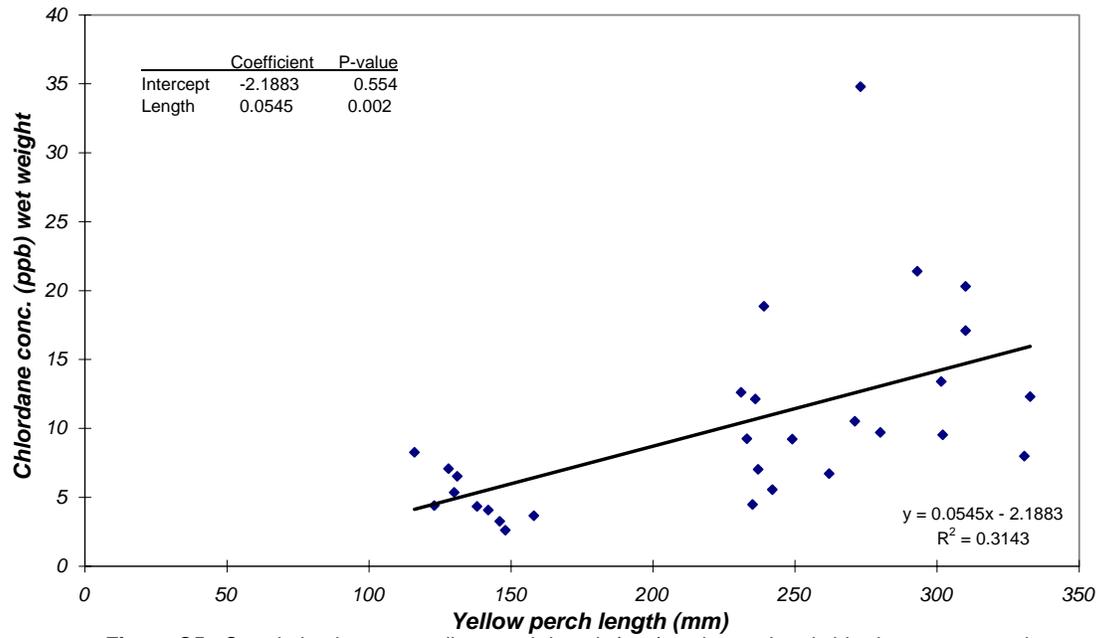
**Figure C2.** Correlation between cutthroat trout length (mm) and associated DDT concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represents the sum of DDT, DDE, and DDE. N=20.



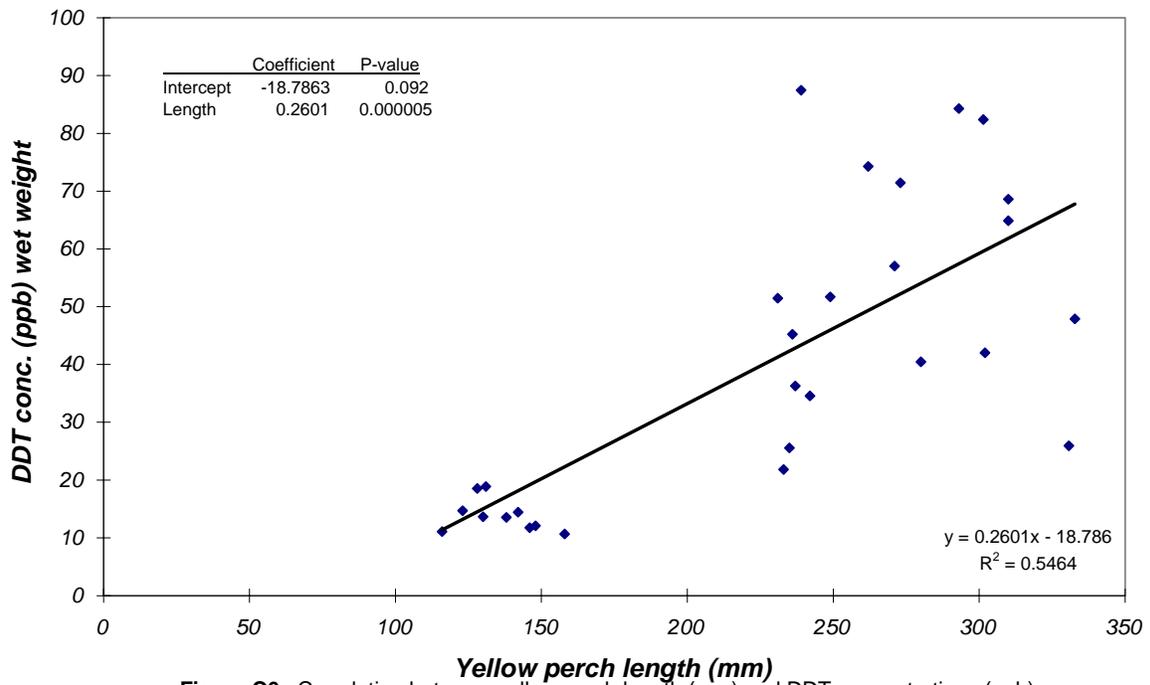
**Figure C3.** Correlation between cutthroat trout length (mm) and associated mercury concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represents the sum of organic and inorganic mercury. N=20.



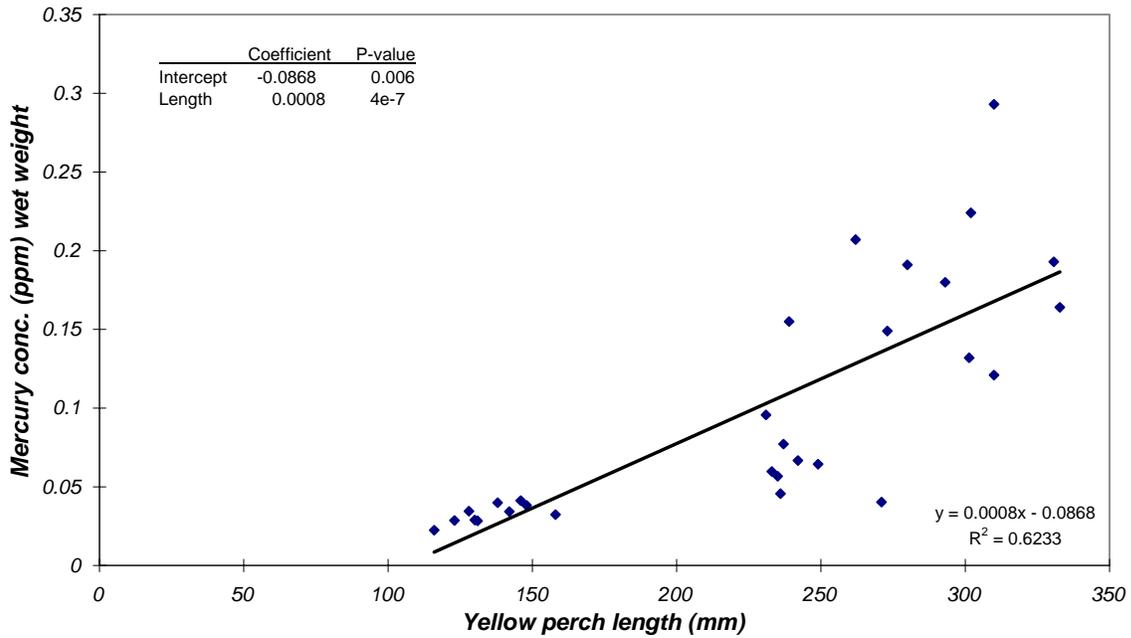
**Figure C4.** Correlation between cutthroat trout length (mm) and associated PCB concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represent sum of PCB arclochs 1254 and 1260. N=20.



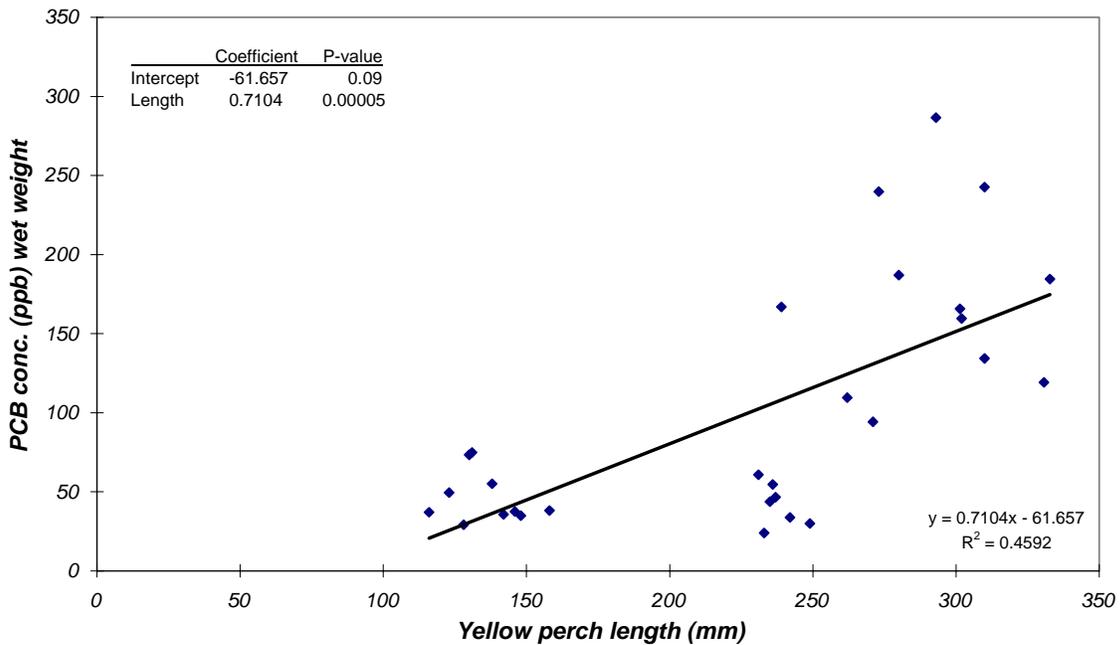
**Figure C5.** Correlation between yellow perch length (mm) and associated chlordane concentration (ppb) measured in whole body tissue samples on a wet weight basis. Data represents the sum of alpha and gamma chlordanes. N=29.



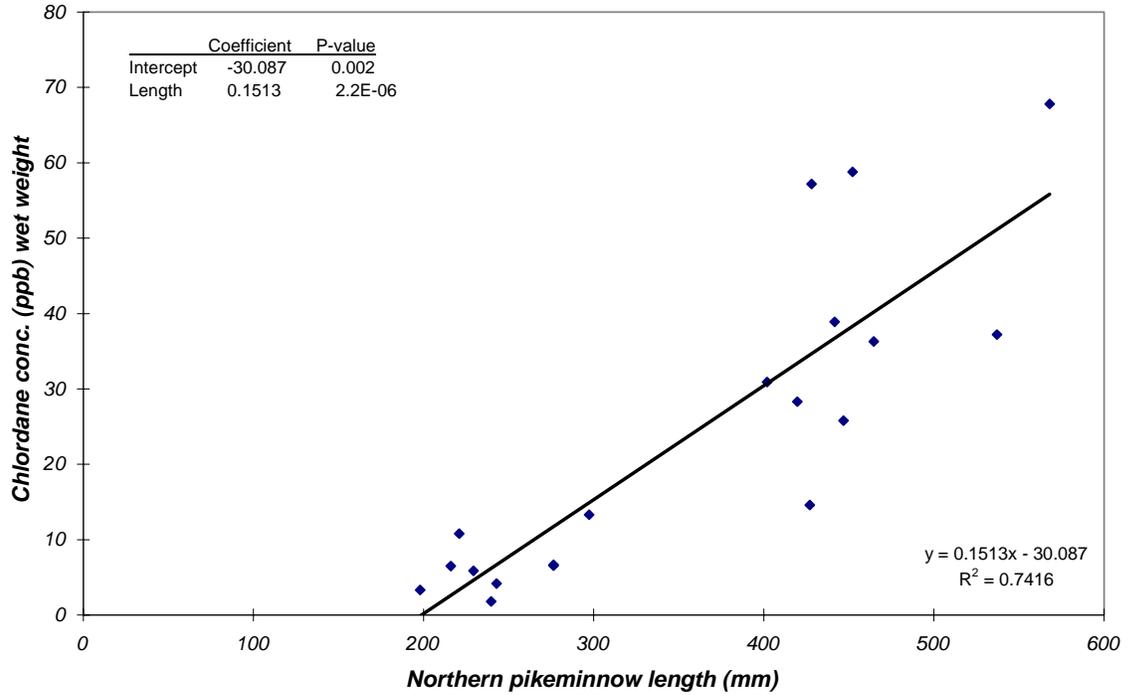
**Figure C6.** Correlation between yellow perch length (mm) and DDT concentrations (ppb) measured in whole body tissues samples on wet weight basis. Data represent the sum of DDT, DDE, and DDD. N=29.



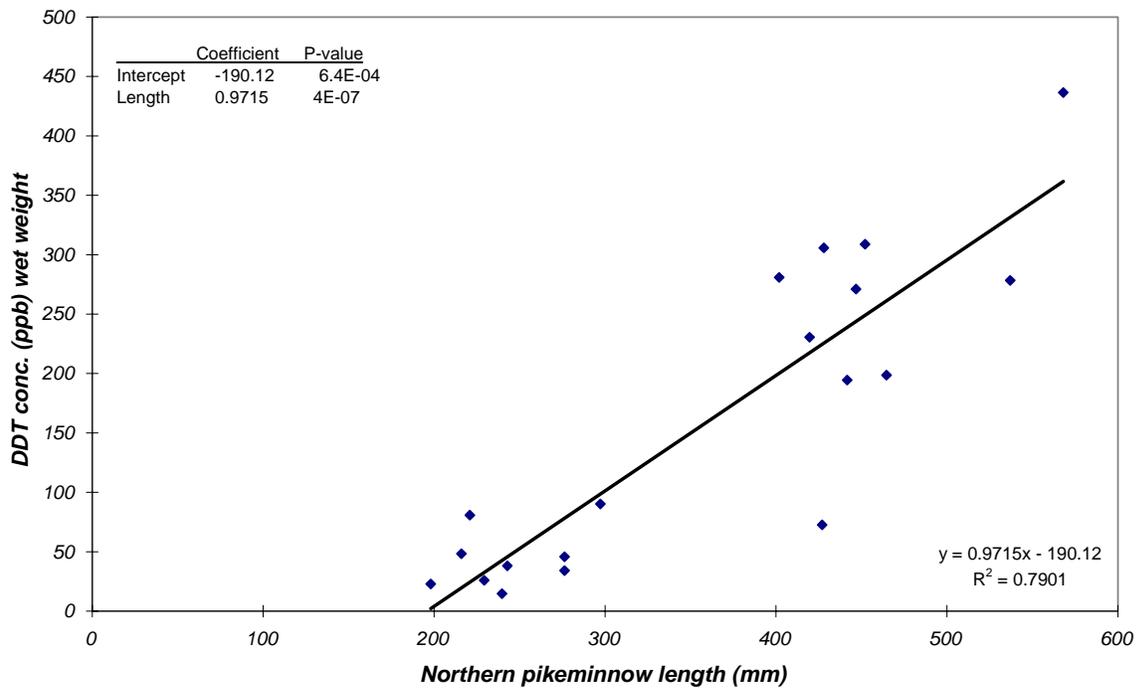
**Figure C7.** Correlation between yellow perch length (mm) and associated mercury concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represent the sum of organic and inorganic mercury. N=29.



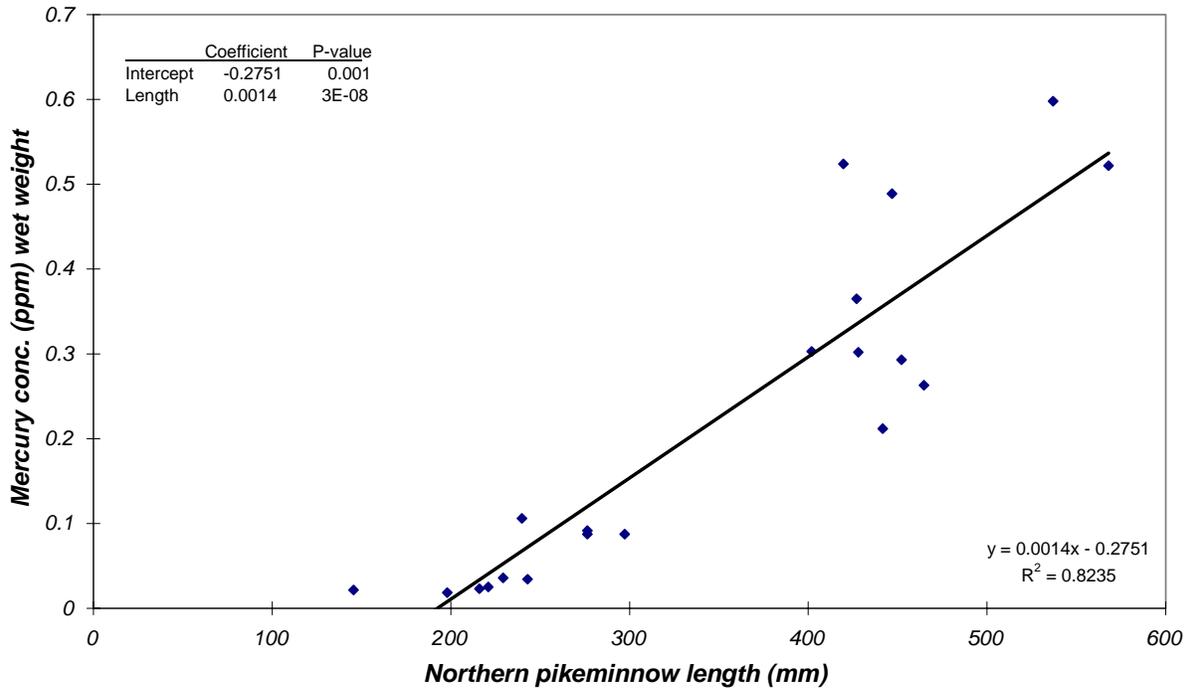
**Figure C8.** Correlation between yellow perch length (mm) and associated PCB concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represent the sum of PCB aroclors 1254 and 1260. N=29.



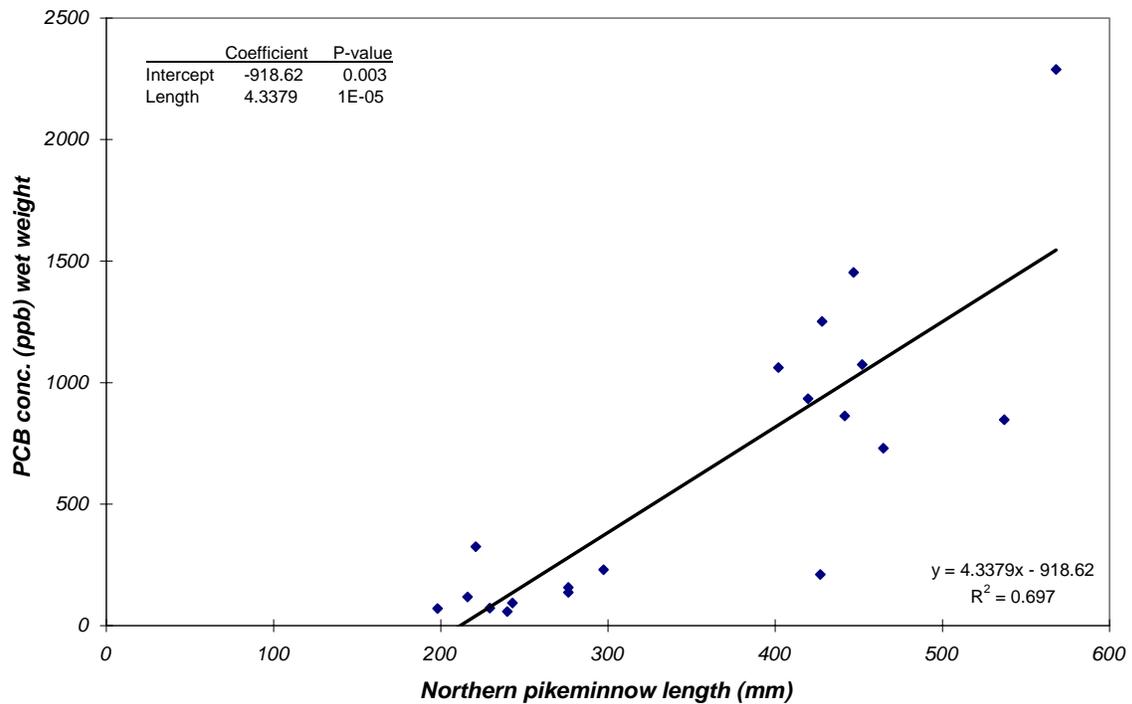
**Figure C9.** Correlation between northern pikeminnow length (mm) and chlordane concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represent the sum of alpha and gamma chlordanes. N=20.



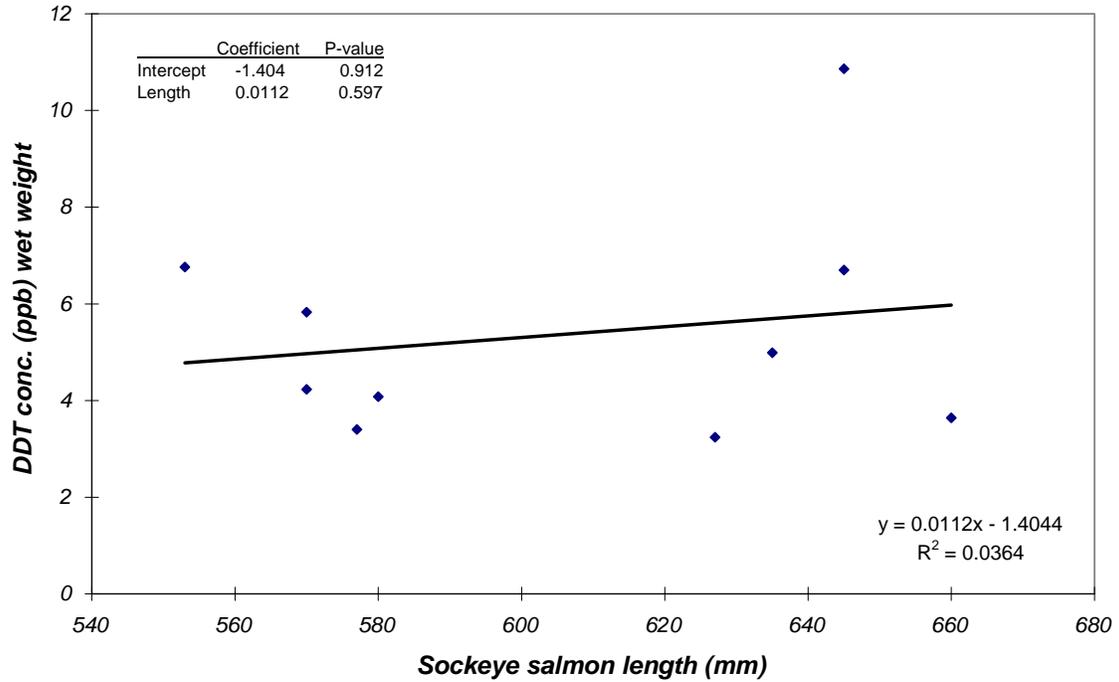
**Figure C10.** Correlation between northern pikeminnow length and associated DDT concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represent the sum of DDT, DDE, and DDE. N=20.



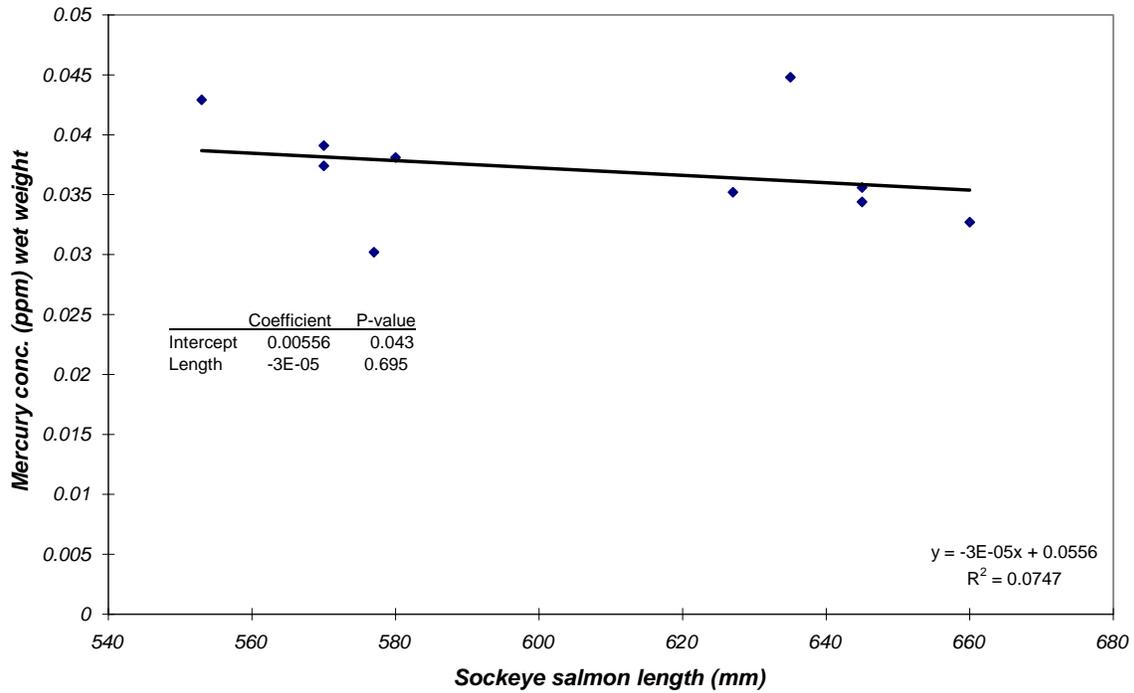
**Figure C11.** Correlation between northern pikeminnow length (mm) and associated mercury concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represents the sum of organic and inorganic mercury. N=20.



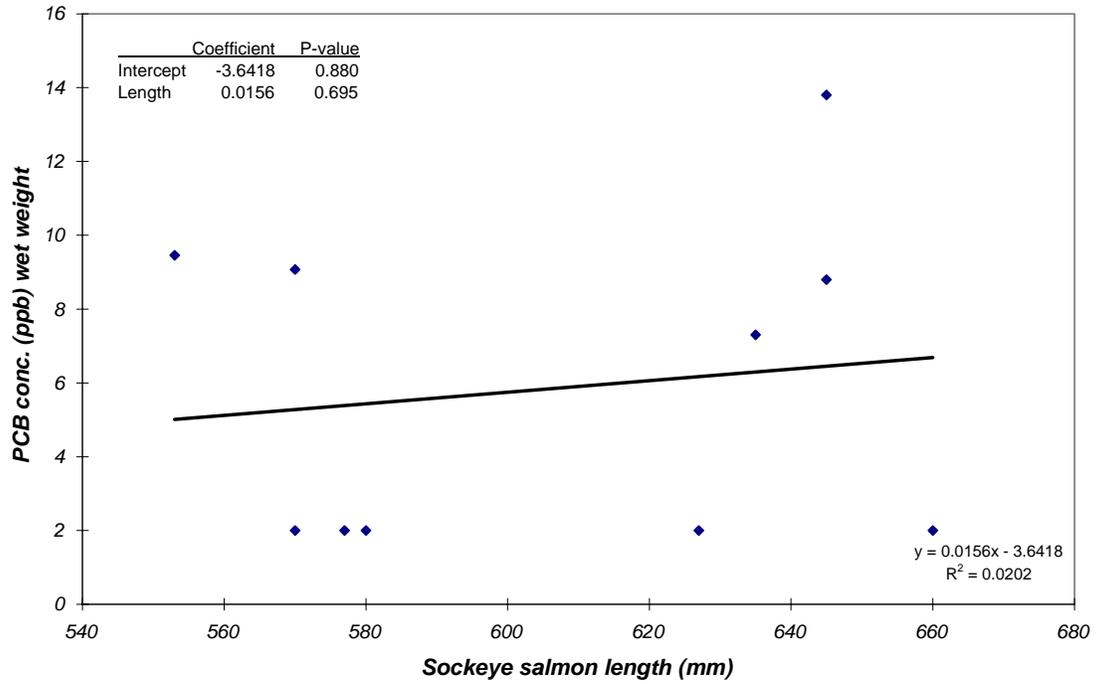
**Figure C12.** Correlation between northern pikeminnow length (mm) and PCB concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represent the sum of PCB aroclors 1254 and 1260. N=20.



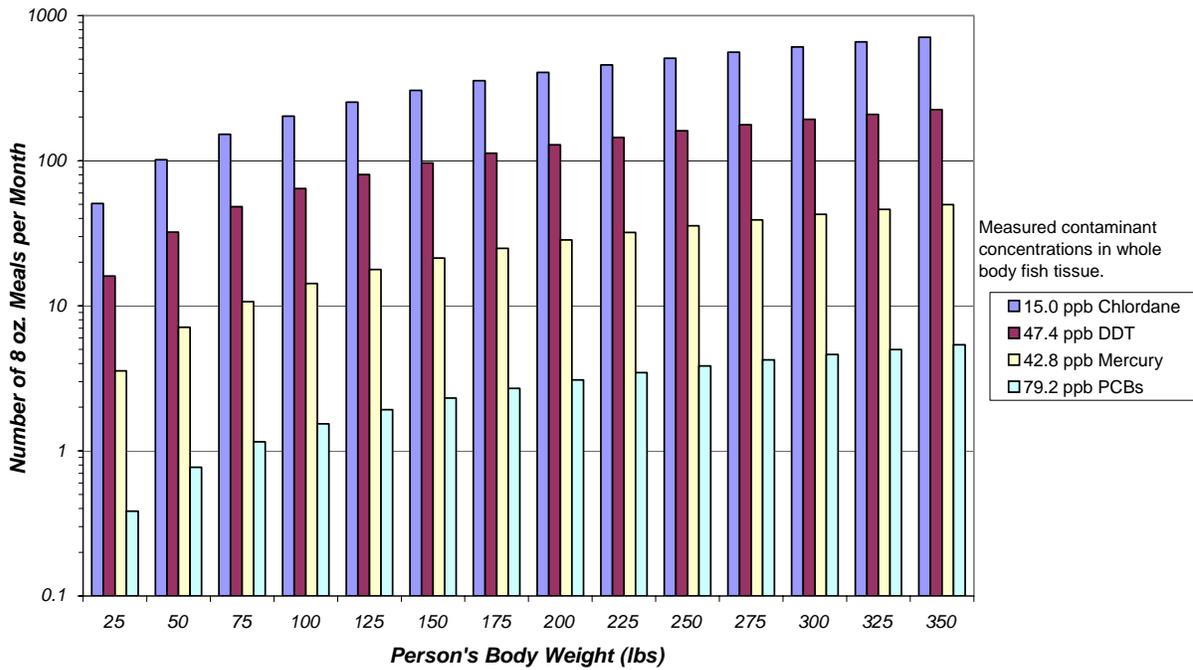
**Figure C13.** Correlation between sockeye salmon length (mm) and associated DDT concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represent the sum of DDT, DDE, and DDT. N=10.



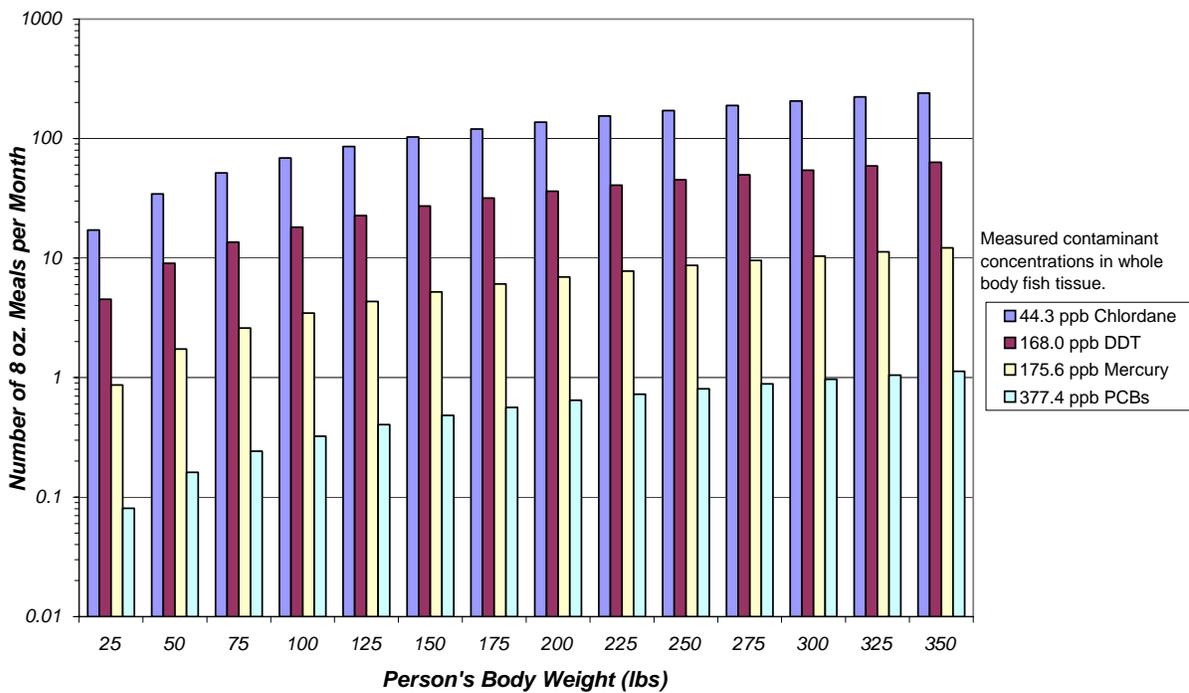
**Figure C14.** Correlation between sockeye salmon length (mm) and associated mercury concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represents the sum of organic and inorganic mercury. N=10.



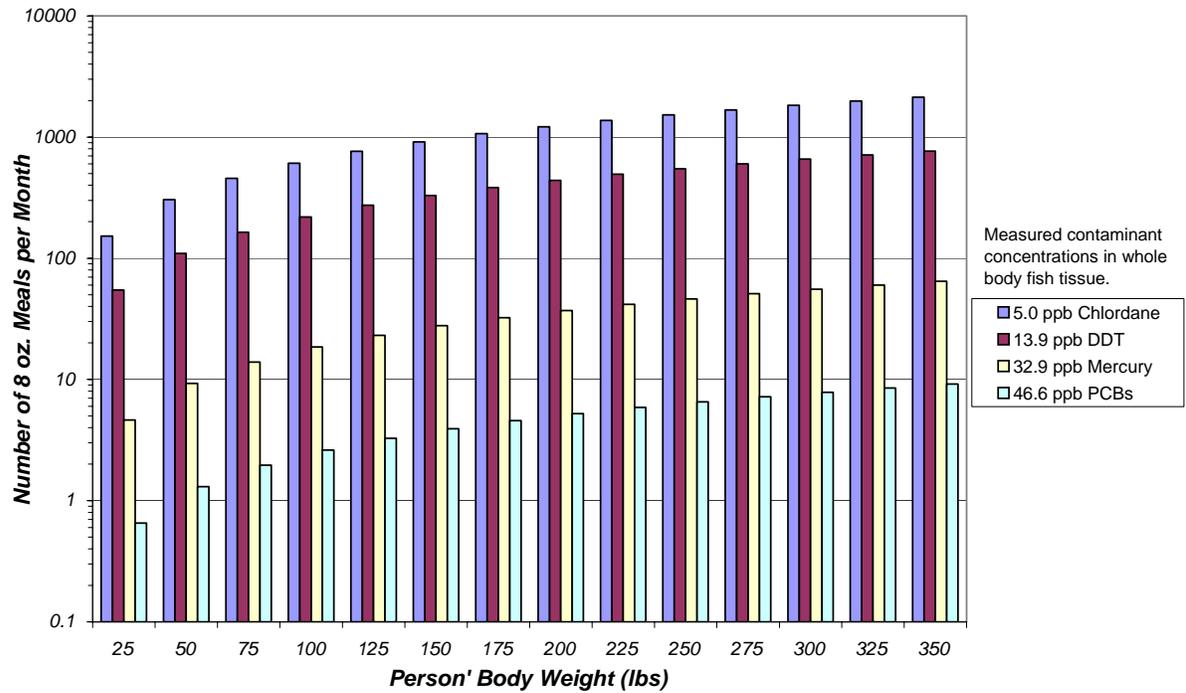
**Figure C15.** Correlation between sockeye salmon length (mm) and associated PCB concentration (ppb) measured in whole body tissue samples on wet weight basis. Data represents the sum of PCB aroclors 1254 and 1260. N=10.



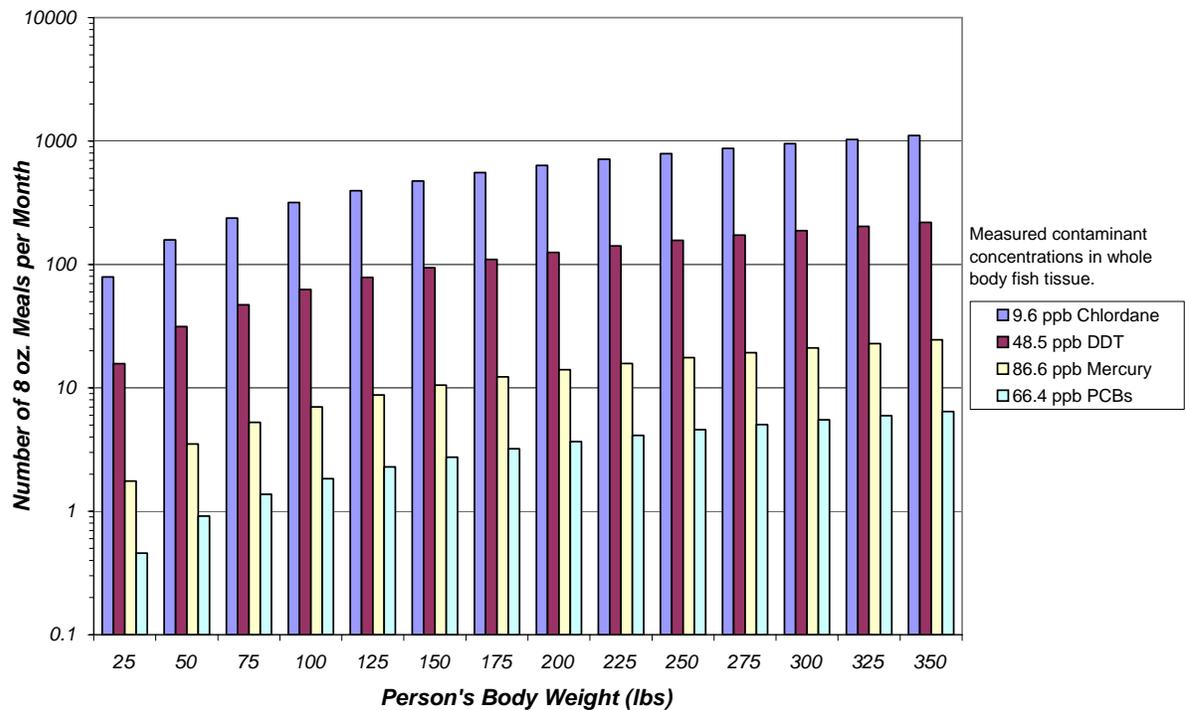
**Figure C16.** Allowable number of 8 oz. meals per month of cutthroat trout < 12 inches (300 mm) from Lake Washington for varying body weights based on contaminant concentration.



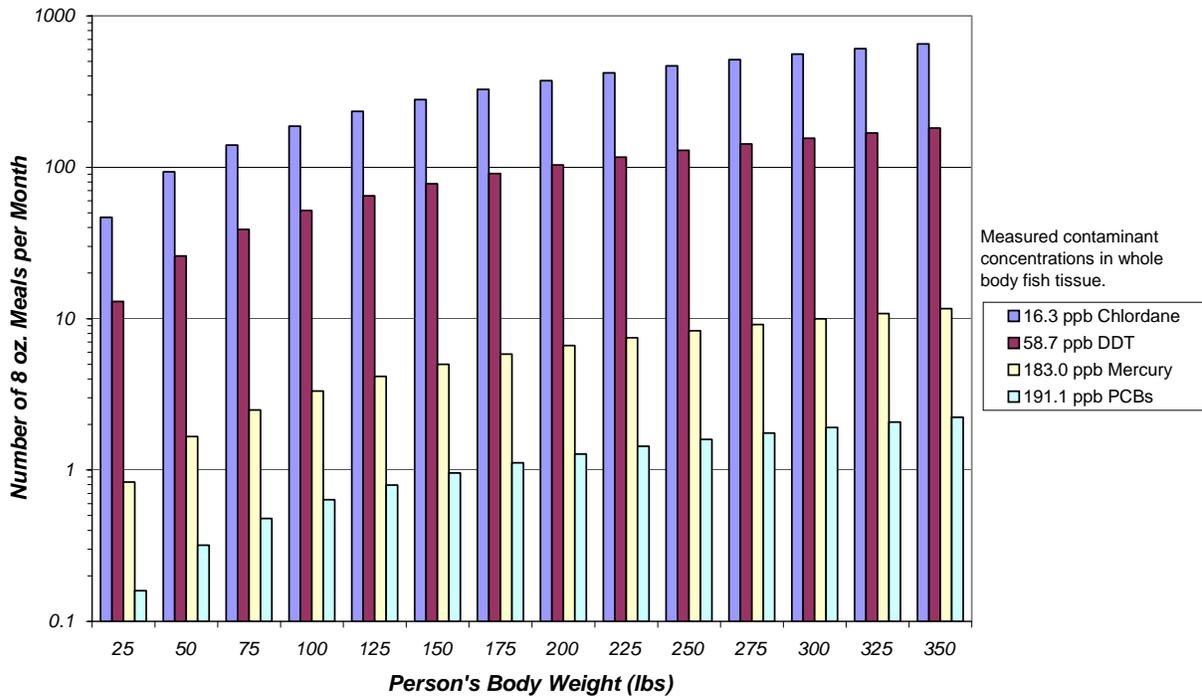
**Figure C17.** Allowable number of 8 oz. meals per month of cutthroat trout > 12 inches (300 mm) from Lake Washington for varying body weights based on contaminant concentration.



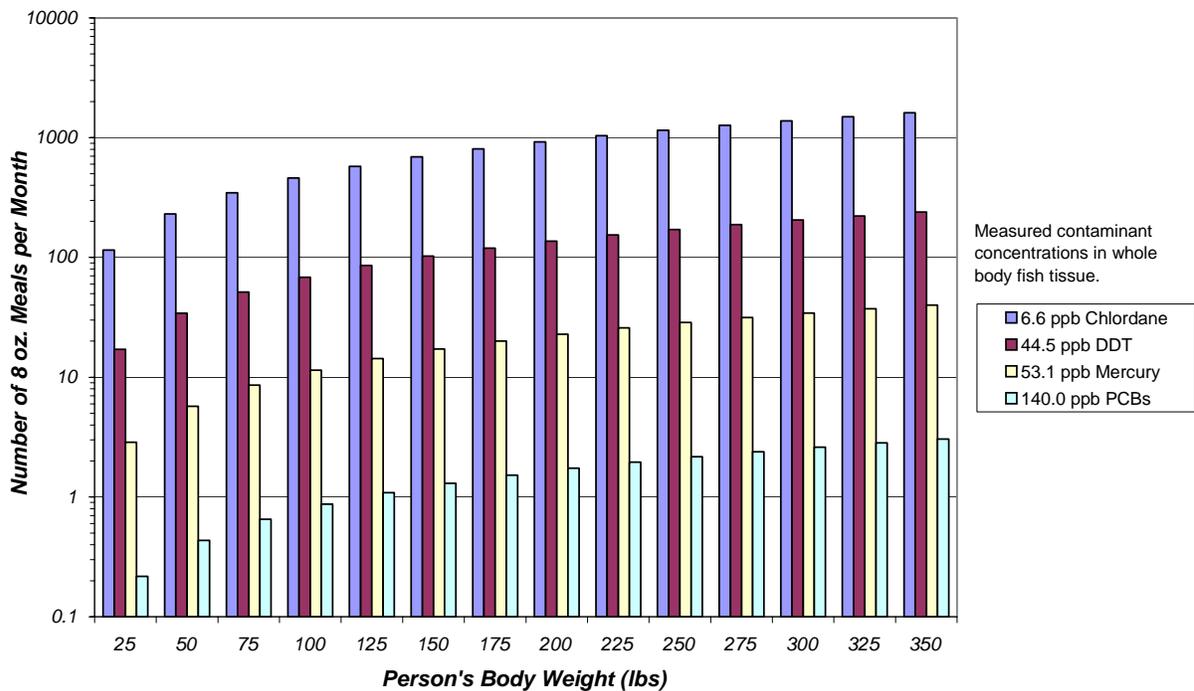
**Figure C18.** Allowable number of 8 oz. meals per month of yellow perch <8 inches (200 mm) from Lake Washington for varying body weights based on contaminant concentration.



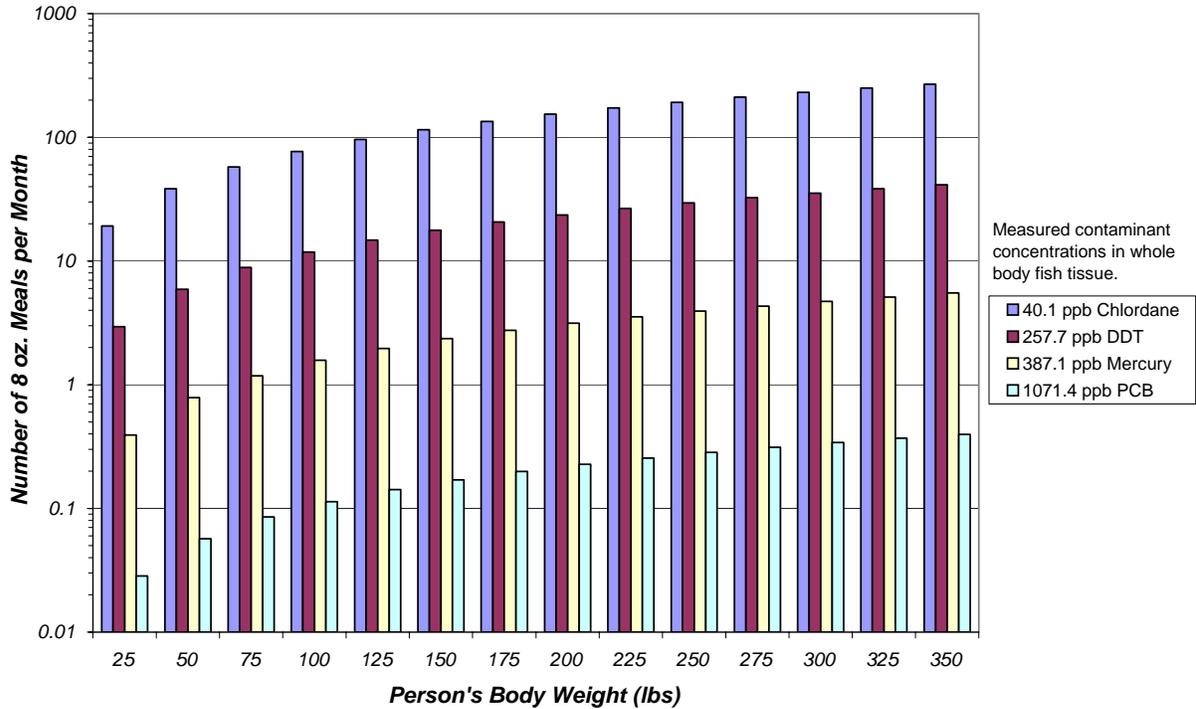
**Figure C19.** Allowable number of 8 oz. meals per month of yellow perch 8 -10.5 inches (201 - 270 mm) from Lake Washington for varying body weights based on contaminant concentration.



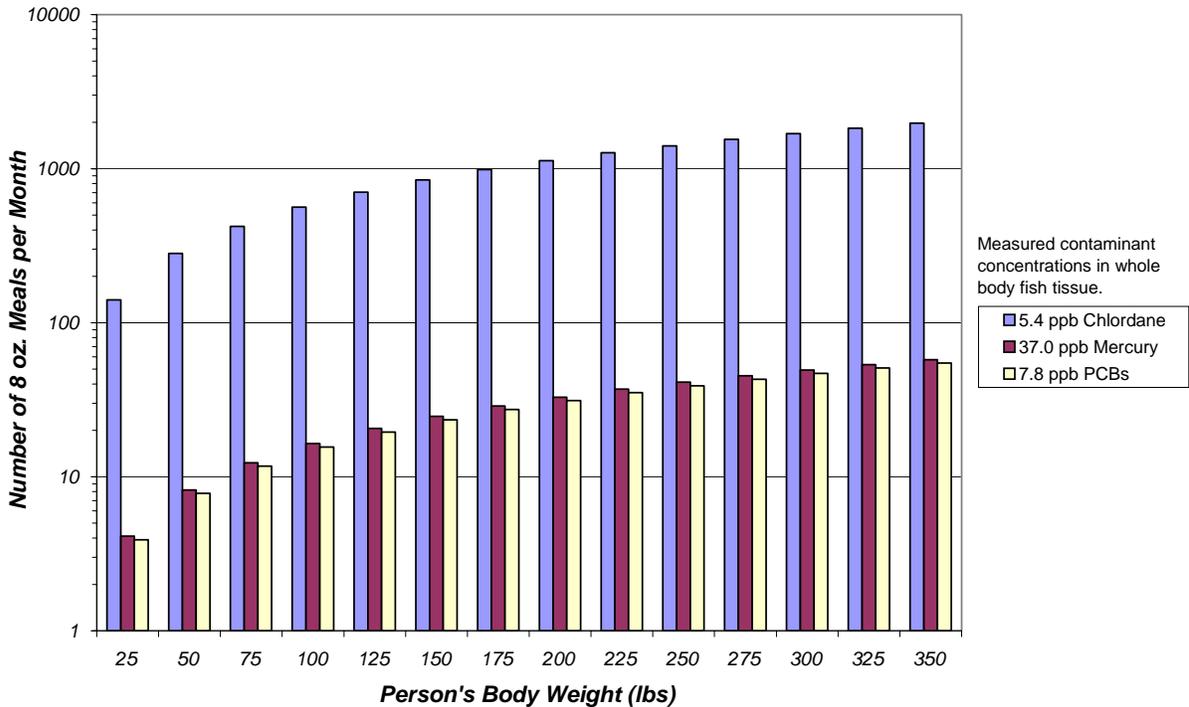
**Figure C20.** Allowable number of 8 oz. meals per month of yellow perch >10.5 inches (270 mm) from Lake Washington for varying body weights based on contaminant concentration.



**Figure C21.** Allowable number of 8 oz. meals per month of northern pikeminnow <12 inches (300 mm) from Lake Washington for varying body weights based on contaminant concentration.



**Figure C22.** Allowable number of 8 oz. meals per month of northern pikeminnow >12 inches (300 mm) from Lake Washington for varying body weights based on contaminant concentration.



**Figure C23.** Allowable number of 8 oz. meals per month of sockeye salmon from Lake Washington for varying body weights based on contaminant concentration.

## APPENDIX D

### Multiple Chemical Exposure Calculations

**Table D1.** Cancer and non-cancer calculations for multiple chemicals for large northern pikeminnow using the mean consumption rate (i.e., 1.5 eight-ounce meals per month).

Large Pikeminnow	PCB	Hg	DDT	Chlordane	
Exposure parameters	Value	Value	Value	Value	Units
BW body weight	30.4	30.4	30.4	30.4	Days/month
MS meal size	60	60	60	60	kg
C conc.	0.227	0.227	0.227	0.227	kg/meal
ED exposure duration - cancer	1071.4	387.1	257.7	40.1	ug/kg
AT averaging time - cancer	30	na	30	30	years
	70	na	70	70	years

meals/month\* 1.5

\* based on King County DNRP consumption study (ave. consumption rate)

	Noncancer Endpoints				Noncancer Hazard Index
	PCB	Hg	DDT	Chlordane	
Dose (ug/kg/day)	0.200	0.072	0.048	0.007	
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	
Hazard Quotient (HQ)	10.00	0.72	0.10	0.01	10.8

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

	Cancer Endpoints				Potential Cancer Risk
	PCB	Hg	DDT	Chlordane	
Dose cancer (ug/kg/day)	0.0857	na	0.0206	0.0032	
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	
Risk	1.7E-04	na	7.0E-06	1.1E-06	1.8E-04

**Table D2.** Cancer and non-cancer calculations for multiple chemicals for large cutthroat trout using the mean consumption rate (i.e., 1.5 eight-ounce meals per month).

Large Cutthroat trout	PCB	Hg	DDT	Chlordane	
Exposure parameters	Value	Value	Value	Value	Units
BW body weight	30.4	30.4	30.4	30.4	Days/month
MS meal size	60	60	60	60	kg
C conc.	0.227	0.227	0.227	0.227	kg/meal
ED exposure duration - cancer	377.4	175.6	168	44.3	ug/kg
AT averaging time - cancer	30	na	30	30	years
	70	na	70	70	years

meals/month\* 1.5

\* based on King County DNRP consumption study (ave. consumption rate)

	Noncancer Endpoints				Noncancer Hazard Index
	PCB	Hg	DDT	Chlordane	
Dose (ug/kg/day)	0.070	0.033	0.031	0.008	
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	
Hazard Quotient (HQ)	3.52	0.33	0.06	0.02	3.9

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

	Cancer Endpoints				Cancer Risk
	PCB	Hg	DDT	Chlordane	
Dose cancer (ug/kg/day)	0.0302	na	0.0134	0.0035	
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	
Risk	6.0E-05	na	4.6E-06	1.2E-06	6.6E-05

**Table D3.** Cancer and non-cancer calculations for multiple chemicals for large yellow perch using the mean consumption rate (i.e., 1.5 eight-ounce meals per month).

Large perch	PCB	Hg	DDT	Chlordane	
Exposure parameters	Value	Value	Value	Value	Units
BW body weight	30.4	30.4	30.4	30.4	Days/month
MS meal size	60	60	60	60	kg
C conc.	0.227	0.227	0.227	0.227	kg/meal
ED exposure duration - cancer	191.1	183	58.7	16.3	ug/kg
AT averaging time - cancer	30	na	30	30	years
	70	na	70	70	years

meals/month\* 1.5

\* based on King County DNRP consumption study (ave. consumption rate)

Noncancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose (ug/kg/day)	0.036	0.034	0.011	0.003	Noncancer Hazard Index
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	
Hazard Quotient (HQ)	1.78	0.34	0.02	0.01	

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

Cancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose cancer (ug/kg/day)	0.0153	na	0.0047	0.0013	Cancer Risk
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	
Risk	3.1E-05	na	1.6E-06	4.6E-07	

**Table D4.** Cancer and non-cancer calculations for multiple chemicals for large yellow perch using the mean consumption rate (i.e., 1.5 eight-ounce meals per month).

Smallmouth bass	PCB	Hg	DDT	Chlordane	
Exposure parameters	Value	Value	Value	Value	Units
BW body weight	30.4	30.4	30.4	30.4	Days/month
MS meal size	60	60	60	60	kg
C conc.	0.227	0.227	0.227	0.227	kg/meal
ED exposure duration - cancer	371.2	244.3	62.9	11	ug/kg
AT averaging time - cancer	30	na	30	30	years
	70	na	70	70	years

meals/month\* 1.5

\* based on King County DNRP consumption study (ave. consumption rate)

Noncancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose (ug/kg/day)	0.069	0.046	0.012	0.002	Noncancer Hazard Index
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	
Hazard Quotient (HQ)	3.46	0.46	0.02	0.004	

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

Cancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose cancer (ug/kg/day)	0.0297	na	0.0050	0.0009	Cancer Risk
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	
Risk	5.9E-05	na	1.7E-06	3.1E-07	

**Table D5.** Cancer and non-cancer calculations for multiple chemicals for large yellow perch using the mean consumption rate (i.e., 1.5 eight-ounce meals per month).

<b>Sockeye</b>	<b>PCB</b>	<b>Hg</b>	<b>DDT</b>	<b>Chlordane</b>	
<b>Exposure parameters</b>	<b>Value</b>	<b>Value</b>	<b>Value</b>	<b>Value</b>	<b>Units</b>
30.4	30.4	30.4	30.4	30.4	Days/month
BW body weight	60	60	60	60	kg
MS meal size	0.227	0.227	0.227	0.227	kg/meal
C conc.	7.8	37	5.4	1	ug/kg
ED exposure duration - cancer	30	na	30	30	years
AT averaging time - cancer	70	na	70	70	years

meals/month\* 1.5

\* based on King County DNRP consumption study (ave. consumption rate)

<b>Noncancer Endpoints</b>					
	<b>PCB</b>	<b>Hg</b>	<b>DDT</b>	<b>Chlordane</b>	
Dose (ug/kg/day)	0.001	0.007	0.001	0.0002	<b>Noncancer Hazard Index</b>
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	
Hazard Quotient (HQ)	0.07	0.07	0.002	0.0004	

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

<b>Cancer Endpoints</b>					
	<b>PCB</b>	<b>Hg</b>	<b>DDT</b>	<b>Chlordane</b>	
Dose cancer (ug/kg/day)	0.0006	na	0.0004	0.0001	<b>Cancer Risk</b>
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	
Risk	1.2E-06	na	1.5E-07	2.8E-08	

**Table D6.** Cancer and non-cancer calculations for multiple chemicals for large northern pikeminnow using the 95<sup>th</sup> percentile consumption rate (i.e., four eight-ounce meals per month).

<b>Large Pikeminnow</b>	<b>PCB</b>	<b>Hg</b>	<b>DDT</b>	<b>Chlordane</b>	
<b>Exposure parameters</b>	<b>Value</b>	<b>Value</b>	<b>Value</b>	<b>Value</b>	<b>Units</b>
3	30.4	30.4	30.4	30.4	Days/month
BW body weight	60	60	60	60	kg
MS meal size	0.227	0.227	0.227	0.227	kg/meal
C conc.	1071.4	387.1	257.7	40.1	ug/kg
ED exposure duration - cancer	30	na	30	30	years
AT averaging time - cancer	70	na	70	70	years

meals/month\* 4

\* based on King County DNRP consumption study (95 percentile consumption rate)

<b>Noncancer Endpoints</b>					
	<b>PCB</b>	<b>Hg</b>	<b>DDT</b>	<b>Chlordane</b>	
Dose (ug/kg/day)	0.533	0.193	0.128	0.020	<b>Noncancer Hazard Index</b>
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	
Hazard Quotient (HQ)	26.67	1.93	0.26	0.04	

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

<b>Cancer Endpoints</b>					
	<b>PCB</b>	<b>Hg</b>	<b>DDT</b>	<b>Chlordane</b>	
Dose cancer (ug/kg/day)	0.2286	na	0.0550	0.0086	<b>Potential Cancer Risk</b>
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	
Risk	4.6E-04	na	1.9E-05	3.0E-06	

**Table D7.** Cancer and non-cancer calculations for multiple chemicals for large cutthroat trout using the 95<sup>th</sup> percentile consumption rate (i.e., four eight-ounce meals per month).

Large Cutthroat trout	PCB	Hg	DDT	Chlordane	
Exposure parameters	Value	Value	Value	Value	Units
BW body weight	30.4	30.4	30.4	30.4	Days/month
MS meal size	60	60	60	60	kg
C conc.	0.227	0.227	0.227	0.227	kg/meal
ED exposure duration - cancer	377.4	175.6	168	44.3	ug/kg
AT averaging time - cancer	30	na	30	30	years
	70	na	70	70	years

meals/month\* 4

\* based on King County DNRP consumption study (95 percentile consumption rate)

Noncancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose (ug/kg/day)	0.188	0.087	0.084	0.022	Noncancer
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	Hazard Index
Hazard Quotient (HQ)	9.39	0.87	0.17	0.04	10.5

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

Cancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose cancer (ug/kg/day)	0.0805	na	0.0358	0.0095	Cancer
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	Risk
Risk	1.6E-04	na	1.2E-05	3.3E-06	1.8E-04

**Table D8.** Cancer and non-cancer calculations for multiple chemicals for large yellow perch using the 95<sup>th</sup> percentile consumption rate (i.e., four eight-ounce meals per month).

Large perch	PCB	Hg	DDT	Chlordane	
Exposure parameters	Value	Value	Value	Value	Units
BW body weight	30.4	30.4	30.4	30.4	Days/month
MS meal size	60	60	60	60	kg
C conc.	0.227	0.227	0.227	0.227	kg/meal
ED exposure duration - cancer	191.1	183	58.7	16.3	ug/kg
AT averaging time - cancer	30	na	30	30	years
	70	na	70	70	years

meals/month\* 4

\* based on King County DNRP consumption study (95 percentile consumption rate)

Noncancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose (ug/kg/day)	0.095	0.091	0.029	0.008	Noncancer
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	Hazard Index
Hazard Quotient (HQ)	4.76	0.91	0.06	0.02	5.7

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

Cancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose cancer (ug/kg/day)	0.0408	na	0.0125	0.0035	Cancer
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	Risk
Risk	8.2E-05	na	4.3E-06	1.2E-06	8.7E-05

**Table D9.** Cancer and non-cancer calculations for multiple chemicals for smallmouth bass using the 95<sup>th</sup> percentile consumption rate (i.e., four eight-ounce meals per month).

Smallmouth bass	PCB	Hg	DDT	Chlordane	
Exposure parameters	Value	Value	Value	Value	Units
BW body weight	30.4	30.4	30.4	30.4	Days/month
MS meal size	60	60	60	60	kg
C conc.	0.227	0.227	0.227	0.227	kg/meal
ED exposure duration - cancer	371.2	244.3	62.9	11	ug/kg
AT averaging time - cancer	30	na	30	30	years
	70	na	70	70	years

meals/month\* 4

\* based on King County DNRP consumption study (95 percentile consumption rate)

Noncancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose (ug/kg/day)	0.185	0.122	0.031	0.005	Noncancer Hazard Index
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	
Hazard Quotient (HQ)	9.24	1.22	0.06	0.011	

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

Cancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose cancer (ug/kg/day)	0.0792	na	0.0134	0.0023	Cancer Risk
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	
Risk	1.6E-04	na	4.6E-06	8.2E-07	

**Table D10.** Cancer and non-cancer calculations for multiple chemicals for sockeye salmon using the 95<sup>th</sup> percentile consumption rate (i.e., four eight-ounce meals per month).

Sockeye	PCB	Hg	DDT	Chlordane	
Exposure parameters	Value	Value	Value	Value	Units
30.4	30.4	30.4	30.4	30.4	Days/month
BW body weight	60	60	60	60	kg
MS meal size	0.227	0.227	0.227	0.227	kg/meal
C conc.	7.8	37	5.4	1	ug/kg
ED exposure duration - cancer	30	na	30	30	years
AT averaging time - cancer	70	na	70	70	years

meals/month\* 4

\* based on King County DNRP consumption study (95 percentile consumption rate)

Noncancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose (ug/kg/day)	0.004	0.018	0.003	0.0005	Noncancer Hazard Index
RfD (ug/kg/day)	0.02	0.10	0.50	0.50	
Hazard Quotient (HQ)	0.19	0.18	0.005	0.0010	

intake = (conc x Ingestion freq x exp freq x exp duration) / (bw x ave time)

Cancer Endpoints					
	PCB	Hg	DDT	Chlordane	
Dose cancer (ug/kg/day)	0.0017	na	0.0012	0.0002	Cancer Risk
Slope Factor (kg-day/ug)	2.0E-03	na	3.4E-04	3.5E-04	
Risk	3.3E-06	na	3.9E-07	7.5E-08	

APPENDIX E  
Cancer Evaluation

## Introduction

Some chemicals detected in Lake Washington fish have the ability to cause cancer. In order to quantify a fish consumer's increased cancer risk, a cancer slope factor describing the potency of a chemical's carcinogenicity must be determined through scientific study. Some cancer slope factors are derived from human population data. Others are derived from laboratory animal studies involving doses much higher than those encountered in the environment. Use of animal data requires extrapolation of the cancer potency obtained from these high dose studies down to real-world exposures. This process involves much uncertainty. Despite uncertainties associated with cancer slope factors for each contaminant, it is possible to calculate the potential cancer risk by applying the following equation (EPA 1986):

$$\text{Risk of cancer} = (\text{Chronic Daily Intake}) \times (\text{Cancer Slope Factor}).$$

In this equation, the chronic daily intake is replaced with the chemical-specific reference dose (RfD) with units of mg/kg-day. The cancer slope factor is also chemical specific with units of (mg/kg-day)<sup>-1</sup>. CSFs for each chemical are listed above. The product of the RfD and CSF results in a unitless value that represents the population risk, expressed as the probability of developing cancer over a lifetime. The resulting calculated cancer risks ranged from 2 in 10,000 for chlordane and DDT, to 4 in 100,000 for PCBs. These risks are upper bound estimates, while true risks may be as low as zero. These calculated values fall under current typical regulatory guidelines used by EPA for acceptable risk levels and range from 1 in one million to one in ten thousand.

The following summaries provide an overview of available cancer data and assessment for each contaminant of concern in Lake Washington fish.

### **Chlordane**

Chlordane is classified by EPA as B2 (probable human carcinogen) using the 1986 Guidelines for Carcinogen Risk Assessment. These characterizations are based on the following summaries of the evidence available: (1) human epidemiology studies showing non-Hodgkin's lymphoma in farmers exposed to chlordane and case reports of aplastic anemia (chlordane data associated with home use are inadequate to demonstrate carcinogenicity); (2) animal studies in which benign and malignant liver tumors were induced in both sexes of four strains of mice and occurred with an elevated, but not statistically significant, incidence in a fifth strain, as well as liver toxicity but no tumors in rats of two strains; and (3) structural similarity to other rodent liver carcinogens. EPA's IRIS has assigned a cancer slope factor (CSF) for chlordane of 0.35 (mg/kg-day)<sup>-1</sup>.

### **Dichlorodiphenyltrichloroethane (DDT)**

DDT is classified also as B2 (probable human carcinogen). The basis for this classification is from observations of tumors (generally of the liver) in seven studies in various mouse strains and three studies in rats. DDT is structurally similar to other probable carcinogens, DDE and DDD. The existing human epidemiological data are inadequate and autopsy studies relating tissue levels of DDT to cancer incidence have yielded conflicting results. Three studies reported that

tissue levels of DDT and DDE were higher in cancer victims than in those dying of other diseases (Casarett et al.1968, Dacre and Jennings 1970, Wasserman et al.1976). In other studies, no such relationship was seen (Robinson et al.1965, Hoffman et al.1967). Studies of occupationally exposed workers and volunteers have been of insufficient duration to be useful in assessment of the carcinogenicity of DDT to humans. EPA's IRIS database shows a CSF for DDT of  $0.34 \text{ (mg/kg-day)}^{-1}$ .

## **Mercury**

Mercury has been classified as C (possible human carcinogen). This is based on inadequate data in humans and limited evidence of carcinogenicity in animals. Three studies were identified that examined the relationship between methylmercury exposure and cancer in humans. No persuasive evidence of increased carcinogenicity attributable to methylmercury exposure was observed in any of the studies. In animal studies, male mice exposed to methylmercuric chloride in the diet had an increased incidence of renal adenomas, adenocarcinomas, and carcinomas. The tumors were observed at a single site and in a single species and single sex. Renal epithelial cell hyperplasia and tumors were observed only in the presence of profound nephrotoxicity and were suggested to be a consequence of reparative changes in the cells. A CSF has not been calculated for mercury in the IRIS database.

## **Polychlorinated biphenyls (PCBs)**

PCBs are also classified as a B2 (probable carcinogen) and the following studies showed possible associations between PCBs and occupational exposure. A cohort study by Bertazzi et al. (1987) analyzed cancer mortality among 2,100 workers at a capacitor manufacturing plant in Italy. Male workers showed a statistically significant increase in death from gastrointestinal tract cancer compared with national and local rates. In females, a statistically significant excess risk of death from hematologic cancer was reported. Analyses by exposure duration, latency, and year of first exposure revealed no trend; however, the numbers were small. A cohort study by Brown (1987) analyzed cancer mortality among workers at two capacitor manufacturing plants in New York and Massachusetts. The cohort included 2,588 workers employed at least three months in areas of the plants considered to have potential for heavy exposure to PCBs. Cancer rates of workers were compared with national rates. Analyses by time since first employment or length of employment revealed no trend; again, the numbers were considered small. A third study involving 3,588 workers at a capacitor manufacturing plant in Indiana by Sinks et al. (1992) analyzed cancer mortality. Workers were classified into five exposure zones based on distance from the impregnation ovens. Compared with national rates, a statistically significant excess risk of death from skin cancer was reported. A proportional hazards analysis revealed no pattern of association with exposure zone; however, the numbers are small.

Other occupational studies by NIOSH (1977), Gustavsson et al. (1986) and Shalat et al. (1989) looked for an association between occupational PCB exposure and cancer mortality. The studies examining the cancer causing effect of PCBs often have methodological limitations. However, the evidence, taken in totality, indicates a potential cancer causing effect for PCBs. EPA determined that the human data are inadequate, but suggestive of carcinogenicity (EPA IRIS 2000), and IARC (1998) concluded that the evidence for carcinogenicity to humans is limited.

Cancer studies in animals are more conclusive in demonstrating a link with PCB exposure. A 1996 study found liver tumors in female rats exposed to Aroclors 1260, 1254, 1242, and 1016, and in male rats exposed to 1260. These mixtures contain overlapping groups of congeners that, together, span the range of congeners most often found in environmental mixtures. Earlier studies found high, statistically significant incidences of liver tumors in rats ingesting Aroclor 1260 or Clophen A 60 (Kimbrough et al., 1975; Norback and Weltman, 1985; Schaeffer et al., 1984). Mechanistic studies are beginning to identify several congeners that have dioxin-like activity and may promote tumors by different modes of action. PCBs are absorbed through ingestion, inhalation, and dermal exposure, after which they are transported similarly through the circulation. This provides a reasonable basis for expecting similar internal effects from different routes of environmental exposure. The current CSF for PCBs is  $2 \text{ (mg/kg-day)}^{-1}$ .

## References

- Bertazzi, P.A., L. Riboldi, A. Pesatori, L. Radice and C. Zocchetti. 1987. Cancer mortality of capacitor manufacturing workers. *Am. J. Ind. Med.* 11(2):165-176.
- Brown DP. 1987. Mortality of workers exposed to polychlorinated biphenyls - An update. *Arch. Environ. Health.* 42(6):333-339.
- Casarett LJ, Fryer GC, Yaeger WL, and Klemmer HW. 1968. Organochlorine pesticide residues in human tissue-Hawaii. *Arch. Environ. Health.* 17:306-311.
- Dacre JC and Jennings RW. 1970. Organochlorine insecticides in normal and carcinogenic human lung tissues. *Toxicol. Appl. Pharmacol.* 17:277.
- EPA 1986. *Guidelines for Carcinogen Risk Assessment*. U.S. Environmental Protection Agency, Office of Research and Development. 1986. 51 Federal Register 33992 (September 24, 1986).
- EPA IRIS – Integrated Risk Information System  
<http://www.epa.gov/iris/index.html>.
- Gustavsson P, Hogstedt C, and Rappe C. 1986. Short-term mortality and cancer incidence in capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). *Am. J. Ind. Med.* 10:341-344.
- Hoffman WS, Adler H, Fishbein WI, and Bauer FC. 1967. Relation of pesticide concentrations in fat to pathological changes in tissues. *Arch. Environ. Health.* 15:758-765.
- IARC 1998. <http://193.51.164.11/htdocs/monographs/suppl7/polychlorinatedbiphenyls.html>
- Kimbrough RD, Squire RA, Linder RE, Strandberg JD, Montali RJ, and Burse VW. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. *J. Natl. Cancer Inst.* 55(6):1453- 1459.
- NIOSH (National Institute for Occupational Safety and Health). 1977. Criteria for a Recommended Standard: Occupational Exposure to Polychlorinated Biphenyls (PCBs). U.S. DHEW, PHS, CDC, Rockville, Md. Publ. No. 77-225.
- Norback DH and Weltman RH. 1985. Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. *Environ. Health Perspect.* 60:97-105.
- Robinson J, Richardson A, Hunter CG, Crabtree AN, and Rees HS. 1965. Organo-chlorine insecticide content of human adipose tissue in south-eastern England. *Br. J. Ind. Med.* 22:220-229.

Schaeffer E, Greim H, and Goessner W. 1984. Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. *Toxicol. Appl. Pharmacol.* 75:278-288.

Shalat SL, True LD, Fleming LE, and Pace PE. 1989. Kidney cancer in utility workers exposed to polychlorinated biphenyls (PCBs). *Br. J. Ind. Med.* 46(11):823-824.

Sinks T, Steele G, Smith AB, Watkins K, and Shults RA. 1992. Mortality among workers exposed to polychlorinated biphenyls. *Am. J. Epidemiol.* 136(4):389-398.

Wasserman M, Nogueira DP, Tomatis L. 1976. Organochlorine compounds in neoplastic and adjacent apparently normal breast tissue. *Bull. Environ. Contam. Toxicol.* 15(4):478-484.