AN ABSTRACT OF THE THESIS OF

Doolalai Sethajintanin for the degree of Master of Science in Toxicology presented on June 21, 2002.
Title: Bioaccumulation Profiles of Chemical Contaminants in Fish from the Lower Willamette River, Portland Harbor Superfund Site and Human Health Risk Assessment.

Abstract approved: Redacted for privacy

Kim A. Anderson

Concentrations of twenty-five PCBs, fifteen organochlorine pesticides and mercury were determined in recreational fish from the Willamette River, Oregon during the summer of 2000. Thirty-six fish samples of three fish species including black crappie, smallmouth bass and common carp were analyzed. The data reported here provides new information and recent residue data in fish from the main stem of lower Willamette River. Concentrations of total PCBs and total DDT (sum of p,p'-homologs) in fish varied from 14 to 528 and from 18 to 510 ng/g wet weight, respectively. Fish samples from Portland Harbor superfund site were most contaminated because this river segment is the primary depositional area of the Willamette River system. Among three fish species analyzed, smallmouth bass contained the highest contaminant levels. Distribution profiles of analyzed PCBs were similar in three fish species. Hexachlorobiphenyl congener 153 was the most abundant and pentachlorobiphenyls congeners 118, 101 and heptachlorobiphenyls congeners 180, 187 were second most abundant. Among DDT derivatives, p,p'-DDE was the most abundant species. Other organochlorine pesticides were not detected or present below detection limit. Mercury levels in tested fish were in a range of 0.013 to 0.52 mg/g. Hazard quotient indices ($\sum HQ>1$) indicated

consumption of contaminated fish by recreational fishers and subsistence fishers harvesting fish from the lower portion of the river might cause chronic adverse health effects posed by the presence of these chemicals. Total cancer risk at all sites of this study exceeded acceptable lifetime cancer risk level (10^{-5}). The greatest contributors to hazard quotients for non-carcinogenic risk and carcinogenic risk were total PCBs and dioxin-like PCBs, respectively. The 10^{-5} upper limit of lifetime cancer risk as the health protection standard, suggested no fish consumption in the unit of meals/year for smallmouth bass and black crappie from the lower Willamette River were acceptable because the presence of PCBs were at the concentrations that can pose a long term toxic threat to local fish consumers.

Bioaccumulation Profiles of Chemical Contaminants in Fish from the Lower Willamette River, Portland Harbor Superfund Site and Human Health Risk Assessment

By
Doolalai Sethajintanin

A THESIS

submitted to

Oregon State University

in partial fulfillment of
the requirements for the
degree of

Master of Science

Presented June 21, 2002
Commencement June 2003

Master of Science thesis of Doolalai Sethajintanin presented on June 21, 2002.

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ACKNOWLEDGEMENTS

I would like to express my sincere gratitude to Dr. Kim A. Anderson, my advisor, for her understanding, patience and encouragement. Her valuable advice is appreciated. I acknowledge Dr. Lawrence R. Curtis for his instruction for getting started. I would like to thank Dr. Brian W. Smith for his contribution to risk analysis. I would like to thank Dr. T. Mark Zabriskie as my graduate representative. I also thank The Oregonian for partial funding of this study.

I am grateful to Eugene R. Johnson and Bobby R. Loper for their laboratory instruction and contribution to this work. I thank Roderick Inman for training me in fundamental laboratory practices. Special thanks go to the staff and colleagues at the Food Safety and Environmental Stewardship Program who made my days at the lab memorable. I thank all of my friends at Oregon State University for their support, especially Arusa Chaovanalikit, Angkana Tantituvanont, Magalie Rosemond and Oraphin Krissanakriangkrai.

I am deeply indebted to the Royal Thai Government Scholarship for financial support. I thank all of my teachers for contributing to my knowledge.

Last, I thank the most important people in my life, mom and dad, for always being there and loving support although physically thousands of miles away. Without their loves and inspiration this accomplishment could not be possible.

CONTRIBUTION OF AUTHORS

This study was conducted with the contributions of the following co-authors: Dr. Kim A. Anderson has provided good advice and expertise in method development. She contributed guidance and helpful discussion in preparation of all manuscripts; Dr. Brian W. Smith helped in assessment of human health risk of organochlorines and mercury exposures; Eugene R. Johnson and Bobby R. Loper provided laboratory assistance and participated in the chemical analysis work.

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CHAPTER 1

INTRODUCTION

Polychlorinated biphenyls (PCBs), organochlorine pesticides, and mercury are some of the most serious global environmental contaminants of concern. Because of their lipophilicity, ubiquity, persistence, and toxicity, these contaminants are potentially harmful to humans and other biota. After these contamintants are released into the environment, they tend to undergo bioaccumulation and biomagnification along the food chain. The concentration in organisms increases due in part to bioaccumulation as the level of trophic position and the length of the food chain increases (Kucklick and Baker, 1998; Rasmussen *et al*, 1990; Chiu *et al*, 2000; Allen-Gil *et al*, 1997). For PCBs and some organochlorine pesticides such as dieldrin and p,p'-DDE, their concentrations in the tissue of the predators at the top level of the food chain can be 10^4 to 10^5-fold higher than in organisms at the lowest trophic levels (Walker, 2001). These toxic pollutants affect organisms in the food chain in different ways, and they ultimately may affect on humans as well.

Human exposure to these chemicals is predominantly through the diet (Fisher, 1999). Fish and seafood products are the largest potential source for human exposure to PCBs and methylmercury (Tollefson, 1989; Dickman and Leung, 1998; Fisher, 1999; National Research Council, 2000). Although fish and seafood

are not significant sources of exposure for organochlorine pesticides compared to dairy products and animal meat, the ubiquity and high levels of some organochlorine pesticides in fish and shellfish such as total DDTs (sum of DDT and its derivatives, DDD, DDE) and dieldrin were reported (Newsome and Andrews, 1993; Datta *et al.,* 1998; Brown *et al.,* 1998; Schmitt *et al.,* 1999). There is evidence of correlation between fish consumption history and body burdens of organochlorine compounds and mercury in local fish consumers from contaminated water bodies (Hovinga *et al.,* 1993; Fitzgerald *et al.,* 1996, 1999; Mergler *et al.,* 1998; Hanrahan *et al.,* 1999). Determining fish tissue contaminant concentrations, therefore, is important to protect residents from the health risks of consuming contaminated non-commercially caught fish, especially from the areas in which a water is known to have high degree of contamination such as a superfund site. Recreational and subsistence fishers are prone to health risk problems if they regularly eat fish containing hazardous chemicals. Recreational and subsistence fishers are a significant target population because they consume large quantities of fish from the same water bodies repeatedly over many years.

Additionally, a fish tissue contaminant study also serves as another indicator for assessing sediment and water quality. Contaminant concentrations in fish tissue reflect contaminant concentrations in the aquatic environment. Brown et al (1998) reported a highly significant correlation between concentrations of PCBs, chlordanes, DDT and its metabolites, hexachlrobenzene and dieldrin in sediment and levels of these compounds in fish livers of all target bottom-dwelling fish. Distributions and patterns of chemical contaminants in fish liver and whole fish tissue were virtually identical and their concentrations were related to surrounding water and sediment concentrations (Bright *et al.,* 1995). Measuring concentration of contaminants in fish tissue provides evidence of contamination over a wider area and over a longer time event than those obtained from measuring contaminants in sediment and water (Brown *et al.,* 1998). Fish take up lipophilic organic contaminants through their contaminated food as well as from direct contact with

sediment and water. Low water solubility and high lipophilicity of the refractory organic contaminants as well as slow metabolism and low excretion of these compounds in fish contribute to potential bioconcentration and bioaccumulation in fish tissue and biomagnification in the food chain. Bottom-feeding fish can represent surrounding sediment quality while predator fish can be a good indicator of persistent pollutant biomagnification. Fish also represent a real bioavailable endpoint, especially relevant for health risk investigation

POLYCHLORINATED BIPHENYLS (PCBs)

PCBs were commercially produced as complex mixtures of congeners, generated by the chlorination of biphenyl. Because of their insulating properties, chemical stability, miscibility with organic compounds and relative inflammability, they were widely used for a variety of industrial applications, especially in capacitors, transformers and other electrical equipments. They were also used in plasticizers, surface coatings, inks, adhesives, flame-retardants, pesticide extenders, paints, and carbonless duplicating paper. Properties that lead to these uses contribute to their environmental persistence (Safe, 1994). Although commercial production of PCBs in the United States was banned in 1979, depositions from past uses, past disposal practices, illegal disposal and accidental release are still environmental sources (Erickson, 2001). In addition, PCBs can be unintentionally produced as by-products of particular chemical process such as combustion of polyvinyl chloride (Katami, 2002).

Many studies have reported PCB-toxicity in both in vitro and in vivo studies (Safe, 1994). The toxicity of individual PCBs is related to the molecular structure. The most notable congeners are those that contain non-ortho or mono-ortho chlorine substituent which have a similar structure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (dioxin). These dioxin-like PCBs act upon aryl

hydrocarbon (Ah) receptors and show similar toxicity to dioxin (i.e., immunotoxicity, disruption of multiples endocrine pathways, developmental toxicity, reproductive toxicity, carcinogenicity, tumor promotion, hepatotoxicity, porphyria, and thymic atrophy) (Safe, 1998). However, non-planar ortho substituted PCBs also elicit toxic responses including neurobehavioral, neurotoxic, carcinogenic and endocrinial changes by acting through multiple unrelated mechanisms (Geisey and Kannan, 1998). Some hydroxylated metabolites interfered with thyroid function and estrogenic activity (James, 2001). Some methylsulfone metabolites induced xenobiotic-metabolizing enzymes, affected thyroid function and promoted tumors (James, 2001). Yusho (Japan, 1968) and Yu-Cheng (Taiwan, 1978) were human poisoning episodes from consumption of rice bran oil contaminated with PCBs where chloracne, fatigue, nausea and liver disorders were reported in the exposed population (Guo and Hsu, 2001).

ORGANOCHLORINE PESTICIDES

Organochlorine pesticides analyzed in this study are listed in Table 1.1. They all are listed as target analytes for assessing chemical contamination in fish by the U.S. Environmental Protection Agency (U.S.EPA, 2000c). They were once widely used to control agricultural and forest pest before production and manufacturing ended in the U.S. because of concerns about their persistence and potential to cause adverse effects to human and wildlife. Potential sources of release are agricultural and urban runoff and deposition from past use.

Table 1.1 Chemical compounds determined in fish tissue

Metal	Organochlorine Pesticides	Polychlorinated Biphenyls
Mercury	DDT and related compounds	37; 3,4,4'-TrCB
	p,p'-DDT	44; 2,2',3,5'-TeCB
	p,p'-DDD	49; 2,2',4,5'-TeCB
	p,p'-DDE	52; 2,2',5,5'-TeCB
		60; 2,3,4,4'-TeCB
	Cyclodiene insecticides	74; 2,4,4',5-TeCB
	dieldrin	77; 3,3',4,4'-TeCB
	aldrin	87; 2,2',3,4,5'-PeCB
	endrin,	99; 2,2',4,4',5-PeCB
	heptachlor	101; 2,2',4,5,5'-PeCB
	heptachlor epoxide	105; 2,3,3',4,4'-PeCB
	α-chlordane	114; 2,3,4,4',5-PeCB
	γ-chlordane	118; 2,3',4,4',5-PeCB
		126; 3,3',4,4',5-PeCB
	Hexachlorocyclohexanes (HCH)	128; 2,2',3,3',4,4'-HxCB
	or	138; 2,2',3,4,4',5'-HxCB
	benzene hexachloride (BHC)	153; 2,2',4,4',5,5'-HxCB
	α-BHC.	156; 2,3,3',3,4,4',5-HxCB
	β-BHC	166; 2,3,4,4',5,6-HxCB
	γ-BHC (lindane)	169; 3,3',4,4',5,5'-HxCB
	δ-BHC	170; 2,2',3,3',4,4',5-HpCB
		180; 2,2',3,4,4',5,5'-HpCB
		183; 2,2',3,,4,4',5',6-HpCB
		187; 2,2',3,4',5,5',6-HpCB
		189; 2,3,3',4,4',5,5'-HpCB

TrCB = trichlorobiphenyls
TeCB = tetrachlorobiphenyls
PeCB = pentachlorobiphenyls
HxCB = hexachlorobiphenyls
HpCB = heptachlorobiphenyls

Eating food contaminated with large amounts of DDT can be acutely toxic to the nervous system from the action upon the Na^+ channels (Walker, 2001). Apart from the nervous system, liver is the organ significantly affected by DDT and its derivatives. DDT-induced liver lesion was reported (WHO, 1979) and U.S. EPA refers to liver toxicity as the critical effect in chronic health hazard assessments for noncarcinogenic effect (U.S.EPA, 2002). U.S. EPA classifies total DDTs as probable human carcinogen based on observation of increased incidence of liver tumors including carcinomas in mice and rats (U.S.EPA, 2002).

Cyclodiene insecticides and hexachlorocyclo-hexanes are antogonists of the neurotransmitter GABA (gamma-aminobutyric acid) found in the nervous system (Walker, 2001). Acute toxicity of occupational exposure to aldrin and dieldrin was reported (i.e., hyperirritability, nausea, muscle twitching and convulsion). Liver abnormalities are the typical chronic toxic effect and oral animal studies have shown liver carcinogenic effect (U.S.EPA, 2002).

MERCURY

Mercury occurs naturally and is distributed throughout the environment by both natural processes and human activities (National Research Council, 2000). Sources of mercury releases to surface waters include natural release from rocks and soils, mining and smelting and industrial activities such as pulp and paper mills, leather tanning, electroplating and chemical manufacturing (U.S.EPA, 2001). Dietary intake is one of the most important sources of non-occupational exposure. The most extreme catastrophes are Minamata Bay (1953-1960) and Niigata (1965) in Japan where a severe neurological disorder was reported in the population as the consequent of consumption of seafood contaminated by mercury from plant effluents (Tollefson, 1989).

Once in the environment, mercury can exist in a number of inorganic and organic forms but methyl mercury is the most common bioavailable form (National Research Council, 2000). Methyl mercury quickly enters the aquatic food chain and can build up in certain fish to levels that are many times greater than levels in the surrounding water. The major human health effects from exposure to methyl mercury are neurotoxicity to adult and neurological dysfunctions and developmental abnormalities in children born to mothers exposed to methyl mercury during pregnancy (National Research Council, 2000). Neuropathological observations showed focal necrosis of neurons in the cortex of cerebrum and cerebellum (Goyer, 1996). Mitochondria changes, induction of lipid peroxidation, microtuble disruption and protein synthesis disruption have been proposed as possible mechanisms, however, there is no definitive evidence of the primary mechanism for methyl mercury toxicity (National Research Council, 2000). In addition to the extreme disaster of Minamata and Niigata episodes, many studies have been conducted to monitor the evidence of neurotoxicity associated with consuming fish from mercury-contaminated areas. Mergler et al (1998) reported deficiency in some nervous system function tests in fish consumers from the upper St. Lawrence River. High blood levels of mercury were associated with consumption rate, however, observed neurotoxic effects as the consequent of mercury burden in fish cannot be warranted because fish from the St. Lawrence River were known to contain multiple neurotoxic substances.

OBJECTIVE

There are local sources of organochlorine compounds, heavy metal and other chemical pollutants into the Willamette River, Oregon. The Willamette River receives direct inputs of treated and untreated municipal and industrial effluents. Industrial operations have been identified as potential sources of contamination to

Portland Harbor, primary depositional area of the Willamette River system (U.S.EPA, 2000b; U.S.EPA and Oregon DEQ, 2000). Besides, non-point source inputs from agriculture, silviculture, residential, urban, and industrial land uses are also significant inputs (Oregon DEQ, 1999). The Oregon Department of Environmental Quality reported decreased water quality at the lower Willamette River. The U.S.EPA listed Portland Harbor a superfund site in December 2000.

However, the most recent published data for PCBs and organochlorine pesticides residues in fish from this area is over decade old. Therefore, the analysis of PCBs, organochlorine pesticides and mercury in fish tissue is proposed to update water quality status as well as to protect local fish consumers from health risk if contaminant levels were unsafe.

The objectives of this pilot study are:

- To evaluate spatial distribution of organochlorines and mercury in fish from the lower 20-miles of the Willamette River including the superfund site at Portland Harbor
- To evaluate temporal persistence of these environmental contaminants in order to understand fate and degradation of persistent chemicals
- To investigate the influence of fish species-difference on the bioaccumulation of persistent chemicals and also to evaluate chemical contaminant levels in fish in different trophic levels.
- To obtain characteristics of PCB composition in fish available for transfer to other organisms at upper trophic levels. The PCB compositions in fish may help to predict the influences of fish consumption on human profiles.
- To assess human health risk from consumption of fish from the lower 20-miles of the Willamette River

CHAPTER 2

BIOACCUMULATION PROFILES OF CHEMICAL CONTAMINANTS IN FISH FROM THE WILLAMETTE RIVER PORTLAND HARBOR SUPERFUND SITE, OREGON

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Manuscript under submission to

The Journal of Environmental Toxicology and Chemistry

ABSTRACT

Twenty-five PCBs, fifteen organochlorine pesticides and mercury were determined in three recreational fish species from the Willamette River in Oregon, including the Portland Harbor superfund site during the spring and summer of 2000. The most recent published data for PCBs and organochlorine pesticides residues in fish from the Willamette River, Oregon is over a decade old. The data presented here provides both new information and updated residue data in fish from the main stem of the lower Willamette River. A series of sample extraction, cleanup, and fractionation was conducted and GC/ECD equipped with dual column/dual detector was employed for identification and confirmation of organochlorine contaminants. Concentrations of total PCBs and total DDT (sum of p,p'-DDT, p,p'-DDE and p,p'-DDD) in fish varied from 14 to 528 and from 18 to 510 ng/g-wet weight, respectively. Hexachlorobiphenyl congener 153 was the most abundant of the PCBs detected and p,p'-DDE was the most abundant of DDT and its derivatives. Average total PCBs concentrations exceeded US EPA's Screening Values. Contaminant concnetrations were highest in fish from the superfund site and lower further upriver. Smallmouth bass were the most contaminated of three fish species examined. Mercury levels in tested fish were in a range of 0.013 to 0.52 mg/g. Other organochlorine agrochemicals were not detected or were below detection limits.

INTRODUCTION

Many streams throughout the Willamette Basin have been monitored for chemical contaminants, and failed to meet water quality criteria due to the occurrence of pesticides, heavy metals, dioxins/furans, and other pollutant in the water, sediments, and fish [1-5]. However, most studies have not focused on the

Portland Harbor area, a recently declared superfund site. The most recent published studies are a decade old [2, 6, 7]. Schmitt et al [6, 7] included the Willamette River at Oregon City (~ River Mile, RM, 26) as one station in the National Contaminant Biomonitoring Program (NCBP) project; residues of organochlorine chemicals in U.S. freshwater fish during 1976-1984; however, this station is 20 miles upriver of the Portland Harbor and the superfund site. Also Schmitt's data is nearly two decades old. Curtis et al [2] also studied the occurrence of PCBs and organochlorine pesticides in fish from six sites along the main stem of the Willamette River in 1990. However their study was focused on the upper river (River Mile 72-195) and fish sampled at Portland were limited (three whole squawfish and three carp muscle samples). To our knowledge, our study is the first published report on organochlorine contaminants in fish collected from the lower Willamette River since 1990.

The Willamette River in western Oregon is one of only 14 American Heritage Rivers [8]. The river is the 13th largest river in the United States in terms of stream flow and yields more runoff per square mile than any of other larger river in the U.S [8]. It flows north from Eugene for approximately 187 miles through Portland, Oregon's metropolitan area, before joining the Columbia River just 10 feet above sea level. The Columbia River flows another 100 miles west to the Pacific Ocean. The Willamette Valley is renowned as one of the most highly productive agricultural regions in the Pacific Northwest [3, 4]. The Willamette basin is home to 70% of Oregonians [3]. The Willamette basin land use is classified as 70% forest, 22% agriculture and 5% urban [3, 4]. In addition, the Willamette River provides a significant migratory corridor, nursery habitat and adult forage for runs of salmon, and nearly 50 species of fish have been identified in the river[4]. Recreational or sport fishing is extremely popular, and resident species are fished throughout the year. Numerous animal species utilize the Willamette River during various seasons [1-5].

The U.S. Environmental Protection Agency (U.S.EPA) [9, 10] declared a six-mile stretch of the lower Willamette within Portland Harbor, between the southern tip of Sauvie Island and Swan Island (RM 3.5 to RM 9.5), as a superfund site on December 1, 2000, see Figure 1. Chemical contaminants within the Portland Harbor sediments included polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), pesticides and heavy metals [9, 10]. Much of the Portland Harbor is industrialized and marine traffic is considered intensive. Possible sources of contamination include historical or current industrial operations. The Oregon Department of Environmental Quality reported in 1999 that the river's overall health was marginal with water quality decreasing steadily from the headwater above Eugene to the mouth at the Columbia River [5].

Although use and production of organochlorine pesticides and polychlorinated biphenyls (PCBs) were banned in most countries, their residues are still ubiquitous in the environment and continue to raise public concern for human and ecosystem health. Historical deposits are the typical source of exposure and aquatic sediment is considered as the ultimate sink. Due to the lipophilic nature of many of these contaminants and their resistance to degradation and metabolism, some of these contaminants bioaccumulate and biomagnify in the food chain and can persist in the environment for many years. PCBs and organochlorine pesticides are among the types of contaminant at Portland, which are known to bioaccumulate and/or biomagnify [9, 10].

There have been several geographic regions of concern in the US considered highly contaminated by organochlorines as a consequence of contamination from historical activities. Portions of Hudson River, New Bedford Harbor, and the Great Lakes have been reported as areas of high organochlorine contamination, as well as, the Chesapeake Bay where fish are known to have high concentrations of PCBs and organochlorine pesticides [6, 11-16]. Schmitt et al [7] determined concentrations of organochlorine chemical residues and heavy mental contaminants in freshwater fish collected from a nationwide network of stations

and concluded a downward trend of mean contaminant concentrations at many stations. However, DDT, PCBs and mercury concentrations in fish from some stations were still high enough to constitute a threat to piscivorous wildlife. Brown et al [17] reported concentrations of chlorinated hydrocarbon pesticides and PCBs in sediment and in indigenous marine fish species were highest at urbanized areas from selected sites on the Pacific Coast and the highest mean concentrations of DDT were probably related to production plants. McCain et al [18] investigated organochlorine contamination associated with fish diseases in San Diego Bay. Beyond contamination in U.S. regions, organochlorine and heavy metal contaminant residues in aquatic system are of global concern. Many studies have reported the ubiquity of PCBs, organochlorine pesticides and mercury in other regions around the world and some were detected at harmful levels to either human or wildlife health [19-23].

One important issue for PCB analyses in the environment is congener composition. PCB congener composition in environmental samples significantly differs from the original commercial mixtures [24, 25]. The composition of PCB mixtures after they are released into the environment changes over time through partitioning, chemical transformation, biotransformation and preferential bioaccumulation [26]. The adverse effect of PCBs on the environment and living organisms are due to specific individual congeners, and their additive and/or non-additive interactions with themselves and other pollutants [27]. Furthermore, risk assessment of PCB in food products and environmental samples considers the potential adverse impact and concentration of specific individual congeners [26]. Characterization of PCB in environmental matrices in terms of the original commercial mixtures, Aroclor®, can therefore be both imprecise and inappropriate [24, 27]. The congener-specific approach of the analysis and risk assessment of PCBs was proposed and has been conducted in several laboratories and regulatory agencies [24, 26-28]. PCBs analysis in our study is based on the congener-specific approach.

Dietary intake is the major source of exposure for humans to organochlorine pesticides, PCBs and methylmercury, especially from fish caught in contaminated water. Fish have low metabolic potential to degrade organochlorine contaminants [29, 30], similarly methylmercury has a very long half-life in fish [31]. Consequently, fish tend to accumulate relatively high concentrations of persistent organochlorines and methylmercury. Fish may bioaccumulate some types of contaminants in their tissues from the surrounding water or sediment unless those contaminants are readily metabolized [4, 17]. Therefore, measuring contaminant concentrations in fish tissues can provide important evidence of occurrence and level of contaminants in upper trophic levels of an aquatic system. Also residue data in fish tissues reflect contamination over a longer period of time and over a wider area than in water and sediments from a single location. Furthermore, study of contaminant residues in resident fish is more relevant since contaminants detected are bioavailable. Bioavailable contaminants have the potential to transfer through and up the food chain and have the ability to cause deleterious effects to human and wildlife health.

The main purpose of our study was to evaluate the occurrence and distribution of PCBs, organochlorine pesticides and mercury on fish samples from the Willamette River at and around the Portland Harbor superfund site. The temporal persistence of the residue data was then evaluated by comparing with the most recent fish data from this region. Since fish residue data in this area are very limited and the most recent published studies are rather old (1990), this data is an important contribution to the environmental site assessment. In addition, the influence of fish species difference on the bioaccumulation of persistent chemicals was also investigated. These residue data will update the community awareness of hazard contamination in the Willamette River fish, particularly from Portland Harbor superfund site if contaminant levels are potentially harmful to local fish consumers.

MATERIALS AND METHODS

Study area

The sampling sites were chosen as a pilot study to investigate current chemical contaminant concentrations in the Willamette River at the Portland Harbor superfund site, upriver and at a reference area (Figure 2.1). Four sites were designated, each 1-3 miles long, and were selected throughout the lower 20 mile-portion of the Willamette River, from river mile 3 (RM3) at the head of Multnomah Channel to RM 25 at Milwaukee. These four sites were generally classified as industrial and urban land use [1]. Site 1 and site 2 represented the superfund site, which were designated as the lower superfund site and the upper superfund site, respectively. Each was a 3-mile segment, containing all 6 miles of the Portland Harbor superfund site, see Figure 2.1. Site 3 and site 4 were within the Portland metropolitan area but are not within the industrial Portland Harbor area or the superfund site. Site 5 and site 6 were designated as the reference sites and classified as agricultural/forestry land use [1], see Figure 2.1.

Sampling procedure

Field sampling was conducted during April – September 2000. Based on the recommendation from the state of Oregon and U.S. Environmental Protection Agency (US.EPA) [1, 32] for selecting target species in tissue contamination monitoring study, one bottom-feeding fish species and two predator fish species were collected. Common carp (*Cyprinus carpio*) represents the bottom-feeding species and smallmouth bass (*Micropterus dolomeieui*) and black crappie (*Pomoxis nigromaculatus*) represent the predator species.

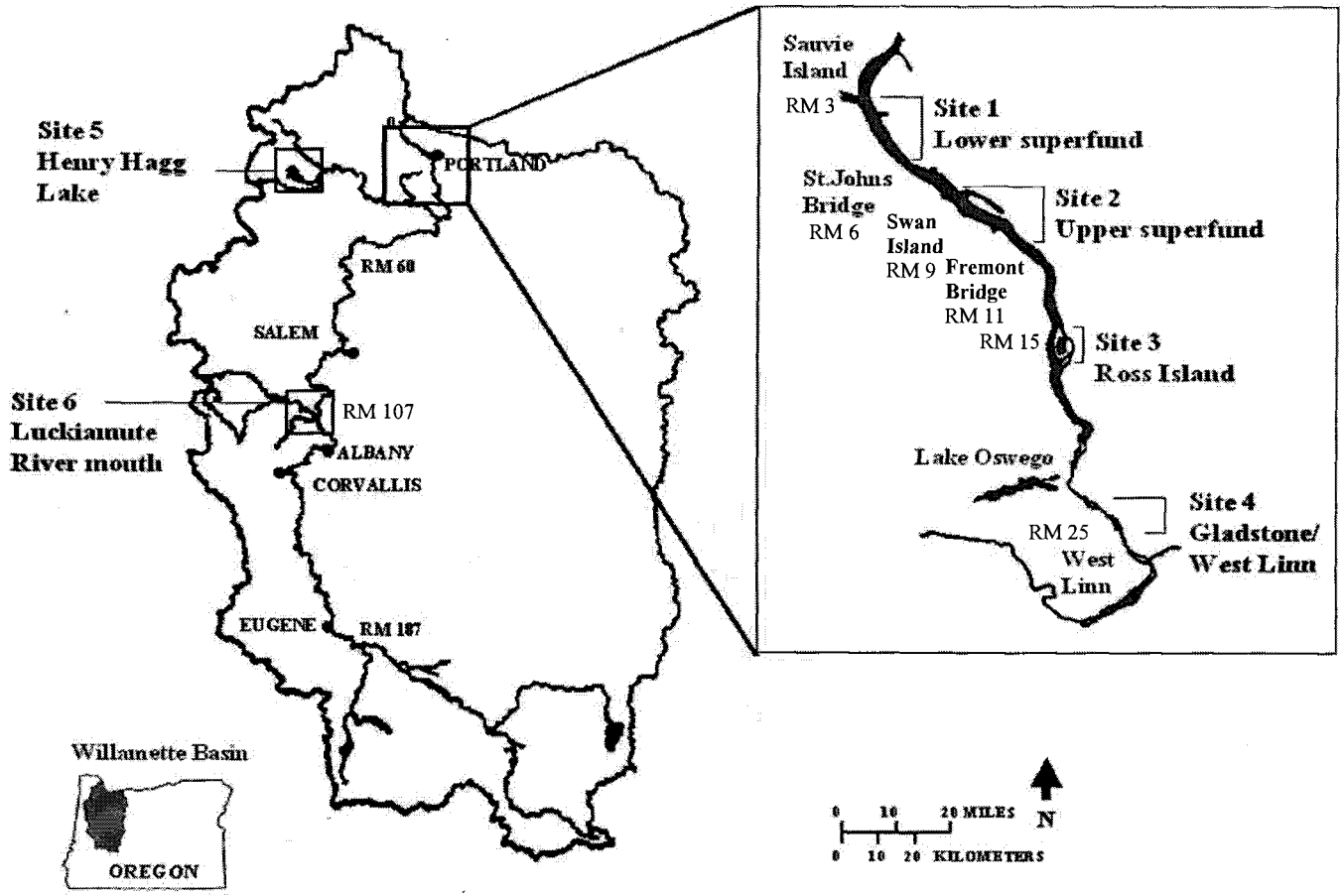


Figure 2.1 Willamette River Basin, Oregon, showing location of sampling site in the study during summer 2000. Site 5 and 6 were designated as reference site

A total of 36 fish representing 3 species were collected along the study area, see Figure 2.1. Our starting objective was to have triplicate of each fish species at each site. Unfortunately we were unable to collect common carp at all sites and this resulted in having triplicate carp at only the lower superfund site and the upper superfund site. Fish were caught by hook and line, and killed by a quick blow to the head. Each individual fish was double wrapped in aluminum foil placed in zip lock bag and immediately packed in coolers with ice packs for transport to the laboratory. All fish were delivered to the laboratory within 24 hours of collection and kept at 4 °C until processing.

Sample preparation

Each fish was prepared within 24 hours of delivery. Sex, weight, and length were recorded. Individual whole fish was cut into small pieces and ground using a high-speed blender, Robot Coup® Bixer RSI BX6. Liquid nitrogen was used to facilitate grinding and homogenization of fish tissue. Fish samples were transferred to sterile glass jars and maintained frozen at –20 °C until analysis.

Sample extraction, cleanup, and fractionation was based on the method of Lazar et al. [33] and modified slightly as necessary for adaptation to our facility's technical requirements. Briefly, 50 µl of 400 ng/ml tetrachloro m-xylene (TCMX) in hexanes was fortified on 5 g of homogenized sample as the internal surrogate standard. Other fortification samples were spiked with 100 µl of an organochlorines mixture (γ-BHC, heptachlor, aldrin each at 100 ng/ml, dieldrin, endrin, p,p'-DDT each at 40 ng/ml). Samples were then ground in a mortar with 25 g Na_2SO_4 anhydrous. Sample mixtures were applied to chromatographic columns (2 cm x 40 cm) plugged with glass wool and filled with Na_2SO_4 anhydrous and 30 ml of 1:1, dichloromethane: hexanes. Another 10 g of Na_2SO_4 anhydrous was mixed into the mortar to remove sample residues and added to the column. The column was kept

for 1 hour and then eluted with 310 ml of 1:1, dichloromethane: hexanes. The extract was concentrated to ca. 5 ml using rotary evaporators. The solvent was subsequently evaporated to 0.5 ml under a mild stream of N_2 (Pierce Racti – Therm^{TM}, heating module).

The extract was subsequently cleaned up using gel permeation chromatography (GPC) column (2.2 cm x 50 cm) packed with a slurry of 50 g Biobead S-X3 (200- 400 mesh, Bio-Rad, solvent dichloromenthane: cyclohexanse, 1:1) connected with a micro fraction collector. The eluent, dichloromethane: cyclohexane, 1:1 had a flow rate of 5 ml/min. Collection of GPC fractions was based on analysis of a series of standard PCB and organochlorine pesticide standards and reference fish tissue analyzed previously. The first fraction (0-144 ml), containing mainly lipids, was saved for lipid content determination; the second fraction (144-240 ml) was collected for organochlorine analysis. The solvent was then evaporated to 1 ml. The solvent extract was eluted through a Florisil® column (6 g, 60-100 mesh, EM Science, activated overnight at 130 °C) for further cleanup and fractionation purposes. Confirmation and validation of the fraction content were performed using a series of known standards and with fortified reference fish samples. Three fractions were collected by consecutive elution with 60 ml hexanes (Fraction A, containing PCBs, mono-orthosubstituted PCBs, α-BHC, heptachlor, aldrin, α -chlordane, p,p'-DDE, p,p'-DDT), 60 ml 15% dichloromethane: hexanes (v:v) (Fraction B, containing non-orthosubstituted PCBs,β-BHC, γ-BHC, δ-BHC, heptachlor epoxide, γ -chlordane, p,p'-DDD), and 110 ml 50% dichloromethane: hexanes (v:v) (Fraction C, containing dieldrin, endrin, methoxychlor).

Each of three fractions was separately concentrated to 0.5 ml and analyzed by gas chromatography with dual capillary columns (DB-xlb and DB-17ms, J&W Scientific Inc.) and dual ECD detectors (Ni ^{63}) with an injection volume of 2 μL. DB-xlb and DB-17ms (each was 30 m x 0.23 mm ID x 0.25 μm film thickness) were used for quantification and confirmation. The GC-ECD was a Varian Star ® Model 3600 operated with the 8200 autosampler with the splitless mode. Helium

and nitrogen were the carrier gas and makeup gas, respectively. Both columns were temperature programmed as follows; initial column temperature was 100 °C with a 1- min hold, then increased from 100 to 130 °C at 10 °C/min, and from 130 to 285 °C at 3 °C/min. The final temperature was held for 4 min. Injector and detector were set at 250 °C and 350 °C, respectively. Chromatographic data were integrated and calculated using Varian Star 4.0-® software. Quantification was accomplished using external calibration. The method detection limits of 2 ng/g for fish samples were typical for PCBs and organochlorine pesticides in our study. Analytes were reported when detected on both columns and only samples containing residues exceeding the blanks were considered positive.

Sixteen fish samples were randomly selected and total mercury content was determined by using an inductive coupled plasma mass spectrometer (ICP-MS).

Quality control

All QC sample types were included in each batch of analysis. QC samples included reagent blanks, fortified samples and sample duplicates, each QC type represented 10 % of the total number of samples analyzed in any given batch. They were prepared and analyzed in the same fashion as the fish samples. Recoveries of internal standard surrogates ranged from 74 –117 %. Average recoveries of fortification fish samples ranged from 72 to 107% (Table 2.1). Relative percentage difference (RPD) for duplicates for each analyte were ≤ 30 %. Standard curves were typically composed of ≥5 standard concentrations. Standard calibration regressions (R^2) ranged from 0.91 to 1.0 for 25 PCBs and 15 organochlorine pesticides. In addition, 10% of the fish samples were sent to the Great Lake Environmental Research Institution for third party quality control and their results were consistent with our data (RPD ≤ 20 %).

Table 2.1 Percent recovery of fortified fish sample (n = 3)

Chemical	Percent Recovery (%)
γ-BHC	72 ± 9.2
Heptachlor	84 ± 5.6
Aldrin	83 ± 1.0
Dieldrin	75 ± 9.2
Endrin	76 ± 10
p,p'-DDT	107 ± 24

The values are arithmetic mean ± standard deviation of three fish samples for each chemical

Statistical analysis

To provide summary statistics comparable to other studies, the data presented are reported as means ± 1 standard deviation. For the human health analyses, if samples were detected on both columns but below the detection limit, they were given a value of one-half of the detection limit (1 ng/g) and samples that were not detected on either column, they were assigned a value of zero [34]. All statistical analyses were conducted using the SAS system for window V.8. One-way analysis of variance (ANOVA) and Tukey's Studentized Range (HSD) were used to examine difference in means of lipid-effect, site-effect and species-effect on analyte concentrations. Linear regression was used to determine whether the relationship between contaminant and lipid concentration existed. Tests were considered significant if $p \leq 0.05$.

RESULTS AND DISCUSSIONS

An example of a GC/ECD chromatogram of a typical fish sample is displayed in Figure 2.2; biological and physical data for fish samples recorded during sample preparation and sample analysis are summarized in Table 2.2. Because concentrations of lipophilic contaminants such as organochlorine pesticides, polychlorinated biphenyl (PCBs) are often correlated to tissue lipid content [35], the relationship between analytes and lipid content was examined. Since the existence of a relationship between contaminant concentration and lipid content among species and among sites was not correlated, the adjustment of tissue contaminant concentration for lipid covariation was not applied in our study. The lipid normalization approach is appropriate when a statistically significant relationship between contaminant concentration and lipid concentration exist [35]. Hebert and Keenleyside [35] concluded if there was no relationship between lipid and contaminant concentration, lipid-normalized data might be inconsistent with interpretation of the wet-weight data. This approach may create more unexplained variability and mask other processes influencing contaminant concentration. Huckins et al [36] also reported application of lipid normalization approach to total PCBs concentration increased variability among fish samples instead of decreasing it. Similarly, our data indicate that lipid normalization is not applicable; therefore, all data are reported on a wet weight basis.

As shown later, concentration of analytes within the same species from some sampling sites presented high variability, as shown by large standard deviation in some cases. However, this is typical in field studies [37], and therefore data points that might be considered as outliers were not omitted from our data sets.

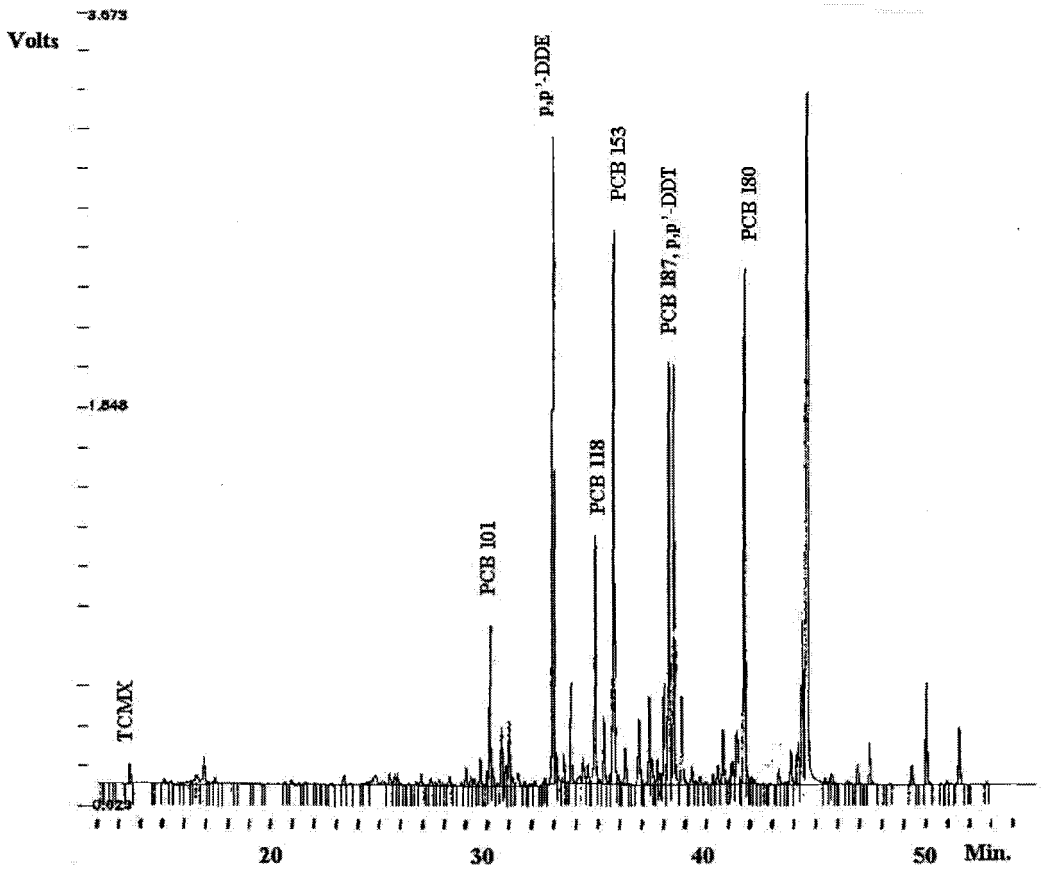


Figure 2.2 GC/ECD chromatogram on db-17ms column of one smallmouth bass (*Micropterus dolomeieui*), this chromatogram is of fraction A from the Florisil column separation.

Table 2.2 Summary of biological and physiological data of fish sample

	Lower superfund site	Upper superfund site	Ross Island	Gladstone/ West Linn	Reference site
Black crappie					
n	3	3	3	3	2
Sex (M/F)	1/2	1/2	2/1	3/0	1/1
Weight (g)	102 ± 6.93	111 ± 15.1	148 ± 20.6	114 ± 16.3	79.8 ± 12.5
Length (cm)	18.0 ± 0.55	20.4 ± 6.11	20.1 ± 1.68	18.7 ± 1.50	16.3 ± 0.28
Percent moisture	70.7 ± 0.29	72.6 ± 0.38	71.9 ± 0.84	72.1 ± 0.87	73.2 ± 0.59
Percent lipid	3.15 ± 0.40	2.54 ± 1.85	2.82 ± 0.36	2.19 ± 0.51	3.11 ± 1.63
Smallmouth Bass					
n	3	3	3	3	4
Sex (M/F)	1/2	2/1	1/2	0/3	1/2[a]
Weight (g)	870 ± 68.5	154 ± 64.7	326±177	277 ± 90.1	395 ± 222
Length (cm)	37.1 ± 1.87	24.9 ± 9.10	27.2 ± 3.32	25.1 ± 2.54	29.5 ± 5.82
Percent moisture	71.3 ± 1.76	73.4 ± 2.11	71.9 ± 1.44	71.1 ± 1.26	72.0 ± 0.99
Percent lipid	4.93 ± 0.33	3.33 ± 1.8	4.17 ± 1.34	5.03 ± 0.58	3.09 ± 1.08
Carp					
n	3	3			
Sex (M/F)	1 / 2	1 / 2			
Weight (g)	876±103	902±766			
Length (cm)	31.6 ± 4.99	33.1 ± 10.1			
Percent moisture	69.2 ± 0.75	72.5 ± 4.74			
Percent lipid	6.31 ± 1.88	5.26 ± 4.62			

Values are arithmetic mean ± 1 standard deviation
n = number, M = male, F = female
[a] Not available

Polychlorinated biphenyls

There are 209 possible PCB congeners; however, only a select group was analyzed as part of this Willamette River superfund site fish pilot study. Inclusion of PCB congeners in the analysis is based on toxicity, frequency of occurrence and abundance in environmental samples, relative abundance in animal tissues and analytical capability [26, 28]. The PCB congeners analyzed in our study were PCB 37, 44, 49, 52, 60, 74, 77, 87, 99, 101, 105, 114, 118, 126, 128, 138, 153, 156, 166, 169, 170, 180, 183, 187 and 189. According to US EPA guidelines [32], concentrations of individual PCB were summed and reported as total PCBs concentration.

Total PCB concentrations in fish increased as the distance to the superfund site decreased (Figure 2.3) and the highest concentrations were detected in fish from within the superfund site. This finding corresponded to river land use in which higher concentrations were found at industrial areas rather than at agricultural/forestry area. Industries and marine traffic are intensive in Portland Harbor and historic industrial activities at this area i.e., electrical power generation and other related activities [38], are considered to be likely potential source of PCBs. At the lower superfund site (see Figure 2.3), there was moderate evidence that the means of total PCBs concentration differed among three fish species ($p= 0.01$, ANOVA F-test) where mean concentration in smallmouth bass was higher than those in black crappie and common carp ($p < 0.05$, Tukey's Studentized Range, HSD). However, species effect was not observed at any other sites. Site effect was also investigated in each individual species. There was moderate evidence of difference in means of PCBs concentrations in smallmouth bass among study sites ($p = 0.02$, ANOVA F-test). Total PCBs in smallmouth bass at the lower superfund site was significantly higher than total PCBs in smallmouth bass at the reference sites or agricultural/forestry area ($p< 0.05$ Tukey's Studentized Range, HSD). On the other hand, the differences in means of total PCBs concentration in

common carp and black crappie among sites were not significant (common carp: p=0.07, t-test; black crappie: p=0.94, ANOVA F-test).

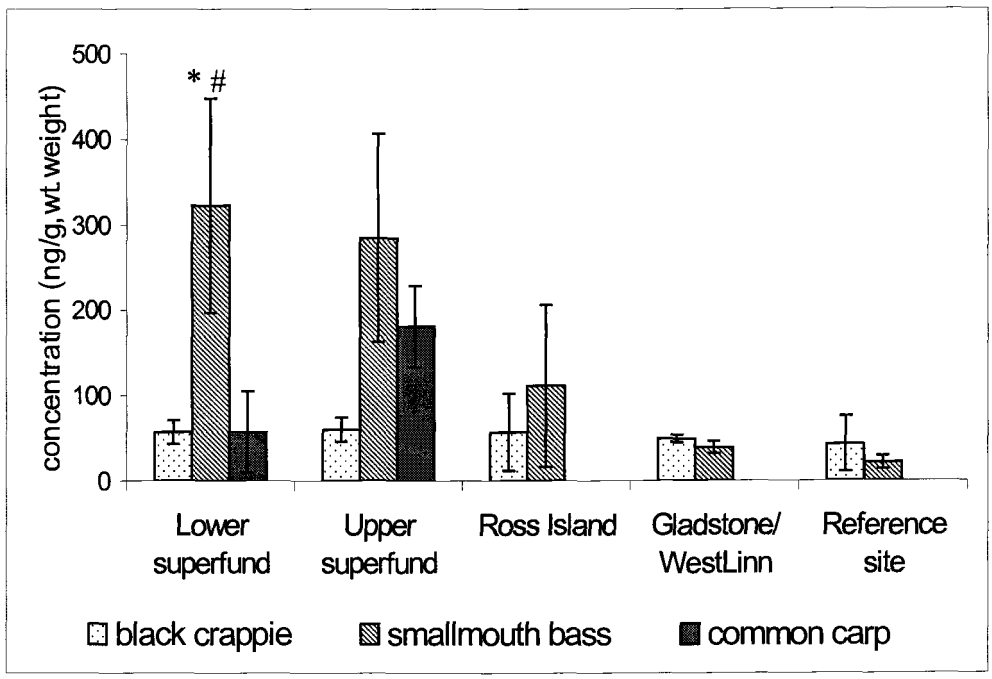


Figure 2.3 Total PCBs concentration (ng/g, wet weight) in whole fish (n=36). The results are mean $\pm$ 1 SD for three fish ($n_{\text{black crappie}} = n_{\text{smallmouth bass}} = n_{\text{common carp}} = 3$) at each site, except at the reference site ($n_{\text{black crappie}} = 2$ and $n_{\text{smallmouth bass}} = 4$).
* Species-effect significant, # Site-effect significant, see text. Significant is considered when $p < 0.05$. U.S. EPA's screening values for carcinogenic effect and non-carcinogenic effect of total PCBs in fish are 20 ng/g and 80 ng/g for recreational fishers and 2.45 ng/g and 9.83 ng/g for subsistence fishers, respectively

Total PCBs concentrations in individual fish were in the range of 14 to 528 ng/g, wet weight. Comparing fish from all sites, fish at the superfund site had the highest level of PCBs contaminant. Of the fish examined smallmouth bass overall

had the highest contamination, with the highest PCBs levels detected of 528 ng/g and 459 ng/g at the upper and lower superfund site, respectively. Average total PCBs in each fish species are illustrated in Figure 2.3.

Although data for purpose of historical comparison are limited, total PCBs in fish at Portland Harbor has not changed significantly since the last published study in 1990 [2]. Target species and PCB analysis approach conducted in the previous studies [2, 6] and our study is somewhat different. Curtis et al [2] reported 127 and 1400 ng/g of total PCBs as Aroclor® 1260 in whole squawfish and carp muscle collected in Portland (RM 7) during July and October of 1990. They also did congener-specific analysis for three coplanar PCBs in a single carp muscle sample from RM 7 and found 37 ng/g of PCB 77, 6 ng/g of PCB 105 and 21 ng/g of PCB 169. In our study, we detected 93 and 181 ng/g, wet weight of total PCBs (sum of 25 congeners) in whole common carp collected at the lower and the upper superfund site, respectively. Of all common carp samples, the highest concentration of PCB 105 was 3.35 ng/g and PCB 77 and PCB 169 were not detected in any common carp from Portland Harbor superfund site. If we assume squawfish and smallmouth bass are equivalent in terms of trophic position in the aquatic food chain; both are piscivorous predator fish species [4], means of total PCBs concentrations in predator fish at Portland Harbor have not changed significantly. We detected 323 and 285 ng/g, wet weight in whole smallmouth bass collected at the lower and the upper superfund site, respectively. Curtis et al [2] found Aroclor® 1260 in whole squawfish, comparable trophic position fish species to smallmouth bass, at 127 ng/g. This suggests that bioaccumulation and biomagnification of PCBs in piscivorous fish species and the aquatic food web in Portland area have not changed significantly. This comparison scenario is not an ideal comparison for temporal trend analysis since total PCBs in fish analyzed in our study and the Curtis et al study [2] utilized different target fish species and analysis approach (Aroclor ® vs. congener specific approach). However, the data presented here can serve as the reference for any future work of contaminant

residue analysis in fish. Schmitt et al [6] studied total PCBs as Aroclor® mixtures (Aroclor®1248, 1254, and 1260) in whole northern squawfish and whole peamouth from the Willamette River at Oregon City (~RM 26). They detected 200 and 100 ng/g, wet weight of Aroclor® 1254 and 1260 in whole squawfish, and 100 ng/g, wet weight of Aroclor® 1254 and 1260 in whole peamouth. Comparative fish species were compared; peamouth and black crappie are insectivores and squawfish and smallmouth bass are piscivores [4]. We detected 49 and 39 ng/g, wet weight in whole black crappie and whole smallmouth bass collected from Gladstone/West Linn site (RM 23-25). Acknowledging the difference in analysis approach between the studies, there however generally appears to be little difference in current PCB levels in Willamette fish compared to Schmitt et al work [6] of nearly two decades ago. In summary, the trend of PCBs bioaccumulation in bottom-feeding fish and predator fish at Portland area and in predator fish at RM 23 –26 have not changed significantly during two decades although the production and use of PCBs has been prohibited for nearly thirty years.

Figure 2.3 shows relatively low PCB concentrations in black crappie as compared with other fish species in our study. Average PCB concentrations detected in black crappie at all sites, however, are still higher than Oregon State and U.S. EPA's warning levels, even at the reference site initially believed to be less PCB contaminated. Oregon State health screening level is 3.3 ng/g, which was exceeded by all fish samples in our pilot study. U.S. EPA's screening values for carcinogenic effect (Risk Level, RL = 10^{-5}) and non-carcinogenic effect of total PCBs in fish are 20 ng/g and 80 ng/g for recreational fishers and 2.45 ng/g and 9.83 ng/g for subsistence fishers, respectively [32]. Therefore, the high level of total PCBs detected in our study is of concern for human health due to consumption of contaminated fish.

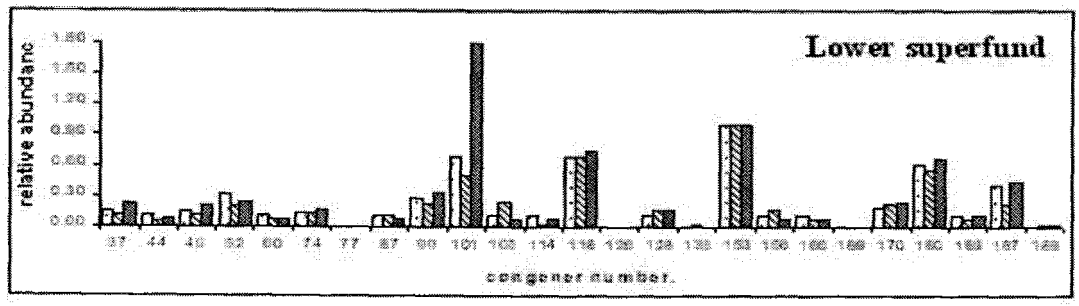
All three fish species in this pilot study are commonly consumed in the area where they occur by recreational and subsistence fishers and bioaccumulate high concentrations of environmental contaminants in their tissues [32, 39]. Lipophilic

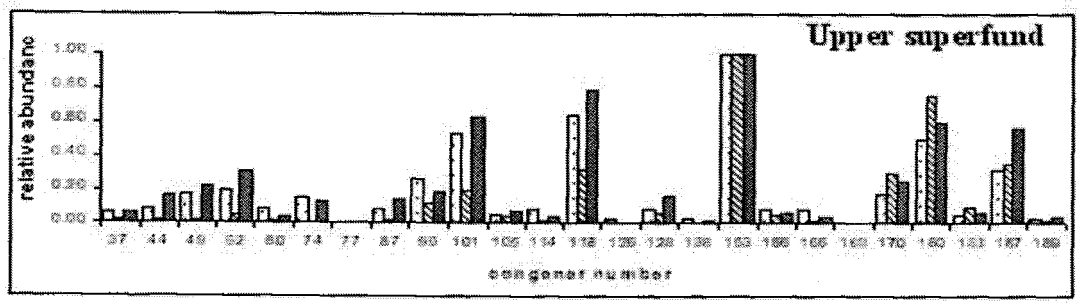
contaminants biomagnify as they pass from prey to predator. Carp, a bottom-feeding fish species, accumulate organochlorines from direct physical contact with contaminated sediment and by consuming benthic invertebrates and epibenthic organisms [32]. Contaminant concentration in sediment usually positively correlates with the level of these compounds or their derivatives in bottom-feeding fish [17]. High PCB concentrations in carp tissues imply the sediments at the superfund site are highly polluted. Whereas smallmouth bass and black crappie, which are predator species, are good indicators of contaminant biomagnification through trophic level of the food chain [32]. We observed different patterns of PCB accumulation in these two predator fish. Total PCBs level in smallmouth bass was much higher than the level found in black crappie. Smallmouth bass is piscivorous; crayfish and fish are primary components of their diets, whereas black crappie is insectivorous [4, 40]. Probably, food source can explain much of the difference between PCBs level in smallmouth bass and black crappie. Insects may not bioaccumulate significant amounts of organochlorine contaminants, compared to crayfish and small fish that associate with the sediment and have longer life cycles. Generally, position in the food chain correlates to the magnitude of bioaccumulative contaminant residues in fish tissues. Compared with organisms at the bottom of the food chain, organisms at the higher end tend to accumulate more highly lipophilic chemicals such as PCBs and organochlorine pesticides. Therefore, food source, trophic level and rate of uptake and loss in individuals may play a role in this difference [39].

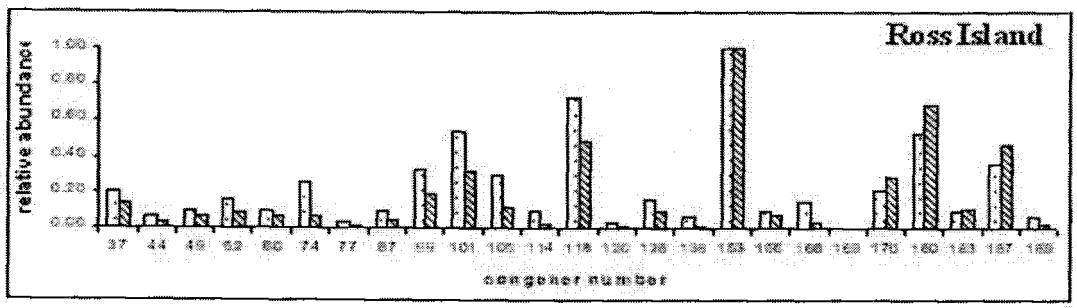
Individual congener distribution was investigated, PCB 153 (2,2', 4,4'5, 5'-HxCB) was found to be the most abundant and PCB 180 (2,2', 3,4,4', 5,5'-HpCB) and PCB 118 (2,3', 4,4'5-PeCB) were second most abundant as well as PCB101 (2,4,5,2', 5'-PeCB) and PCB187 (2,3,5,6,2', 4', 5'-HpCB), as shown in Figure 2.4. The PCB pattern can vary within a study area, trophic levels, or even in animals of the same species due to different kinds of PCBs that they are exposed to [41]. Our findings correspond to the study by McFarland and Clarke [28] in which PCB

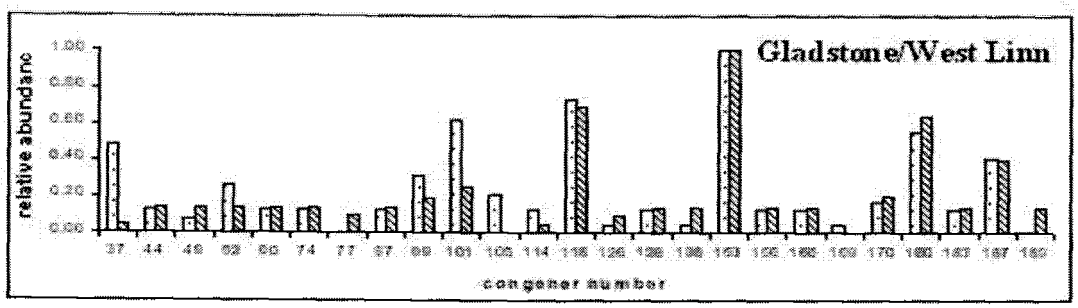
congers were identified and classified by their toxicity and frequency of occurrence in the environment, as well as, many other environmental PCBs studies [21, 25, 37, 42, 43]. PCB residue profiles are influenced by many factors such as a profile of exposure sources, species difference, and other effects under field conditions [25]. All fish samples in this study showed relatively similar profiles in which highly chlorinated PCBs tended to accumulate more than less chlorinated PCBs. This congener pattern suggests relatively low metabolism of high chlorine content PCB congeners in fish. Less chlorinated PCBs have more available positions (unsubstituted ring) for metabolism than highly chlorinated PCBs [44]. PCB 138, one of the predominant PCB congener often found in animal tissues [12, 24, 45] was not very abundant in fish from our study. PCB 153 is among the most frequently reported as an abundant PCB congener in environmental samples and animal tissues [25, 45]. This hexachlorinated congener is often used as a reference congener and the bioaccumulation ratios of other PCBs are expressed relative to PCB 153 [46]. PCB 153 was typically the most abundant or second most abundant congener in our fish at all sites, see Figure 4. PCB153 is one of the major components of commercial PCB mixtures (e.g. 4.26% in Aroclor 1254 and 10.20% in Aroclor 1260) [30]. Its toxicity appears to have the greatest potency among the di-ortho coplanar congeners but less potent than the non-ortho coplanar and mono-ortho coplanar congeners [28].

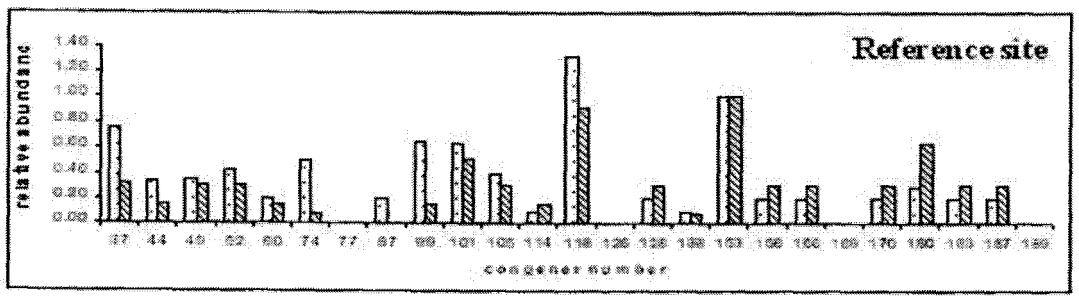
Figure 2.4 Polychlorinated biphenyl congener profile in three fish species (n= 36). Data are averages of three fish sample ($n_{\text{black crappie}} = n_{\text{smallmouth bass}} = n_{\text{common carp}} = 3$) at each site, except at the reference site ($n_{\text{black crappie}} = 2$ and $n_{\text{smallmouth bass}} = 4$). Concentrations are normalized to PCB 153 as shown as relative abundant.











Black crappie Smallmouth bass Common carp

Interestingly, the PCB profile is modestly different at the reference site. Specifically, there are proportionally more lower chlorinated PCBs in fish at the reference site. Trichlorobiphenyls (PCB 37) and tetrachlorobiphenyls (PCB 49, 52, 60, and 74) at the reference site represent the relative ratios of 0.20 to 0.75 which are higher than the ratios at the other sites, especially PCB 37. Exposure to different original Aroclor® mixtures can contribute to profile differences in fish. Aroclor® 1016 and Aroclor® 1242, for example, contain about 50% of trichlorobiphenyls and 25 % of tetrachlorobiphenyls while Aroclor® 1260 have no trichloro- and tetrachloro biphenyls [26]. Therefore, it is possible that historical input of PCBs at the reference site is different from the other sites. In addition, age of the original source can also contribute to this difference. Environmental degradation processes such as photolysis and microbial degradation can change individual PCB congeners to the lower chlorine content congeners. PCBs at the older source tend to be lower chorine substituted congeners than PCBs recently released. Changing in environmental PCB composition occurs over time. Therefore, source and age of a release are several factors that can contribute to the differences in PCB profiles in environmental samples at different locations.

There are 12 dioxin-like PCB congeners currently defined [47]. We analyzed eight of these congeners. Detection of dioxin-like PCBs suggested the potential for dioxin toxicity from the dioxin-like congeners should be considered. Dioxin-like PCBs elicit biochemical and toxic response resembling those caused by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) [47-49]. The toxic responses by TCDD are mediated through the aryl hydrocarbon (Ah) receptor signal transduction. The Toxic Equivalency Factors (TEFs) approach converts measured concentrations of individual congeners to TCDD-Toxic Equivalent Concentration (TEQ) by using TEF values set by World Health Organization (WHO-TEFs) for individual dioxin-like PCBs [47]; $TEQ = \Sigma\ [PCBi \times TEF_i]_n$ where i = individual congener, and n = the numbers of congeners. TEQs for each fish species are given in Table 2.4.

Table 2.3 TEQ concentrations (pg/g, wet weight) in whole fish (n = 36). Data are averages of three fish sample ($n_{\text{black crappie}} = n_{\text{smallmouth bass}} = n_{\text{common carp}} = 3$) at each site, except at the reference site ($n_{\text{black crappie}} = 2$ and $n_{\text{smallmouth bass}} = 4$)

Congeners	TEF [a]	Lower Superfund site			Upper Superfund site			Ross Island		Gladstone/West Linn		Reference Site	
		SB	BC	CC	SB	BC	CC	SB	BC	SB	BC	SB	BC
77	0.0001	ND	ND	ND	0.03	ND	ND	0.03	0.03	0.07	ND	ND	ND
126	0.1	ND	ND	ND	33.0	33.0	ND	33.0	33.0	67.0	33.0	ND	ND
169	0.01	ND	ND	ND	3.30	ND	ND	ND	ND	ND	3.30	ND	ND
105	0.0001	1.57	0.10	0.10	0.27	0.07	0.23	0.30	0.29	ND	0.17	0.10	0.20
114	0.0005	0.79	0.50	0.50	0.50	0.50	0.50	0.34	0.50	0.17	0.50	0.25	0.25
118	0.0001	4.47	0.71	0.99	2.63	0.82	2.59	1.20	0.71	0.50	0.59	0.30	0.67
156	0.0005	5.26	0.50	0.50	1.94	0.50	1.03	0.97	0.50	0.50	0.50	0.50	0.50
189	0.0001	0.10	ND	0.03	0.10	0.03	0.10	0.07	0.07	0.10	ND	ND	ND
PCBs-TEQs		12.2	1.81	2.12	41.8	34.9	4.44	35.9	35.1	68.3	38.1	1.15	1.62

[a] Toxic Equivalency Factor (TEF) based on Van den Berg et al. 1998 [47]
SB = smallmouth bass
BC = black crappie
CC = common carp
ND = non detected

The relative contributions of coplanar PCBs and mono-ortho PCBs to TEQ concentration varied depending on site and fish species. Since no coplanar PCBs were detected in any fish at the Lower superfund site, only mono-ortho PCBs contributed to TEQ concentration at this superfund site. At the site where coplanar PCBs were detected, coplanar PCBs were the major contributors to TEQs. This can be explained by the greater value of the respective TEFs of coplanar PCBs than the respective TEFs for mono-ortho PCBs. When the levels of coplanar PCBs and mono-ortho PCBs are similar, coplanar PCBs will dominate the TEQ concentration due to the larger TEFs value, in particular, TEFs of PCB 126 and PCB 169. TEQ concentrations in all individual fish species at all sites of this study exceeded the recommended Screening Values (SVs) for dioxin (RL = 10^{-5}) [32], which are 2.56 x 10^{-7} ppm and 3.15 x 10^{-8} ppm for recreational fishers and subsistence fishers, respectively.

Organochlorine pesticides

Concentrations of total DDTs (sum of p,p'-DDT, p,p'-DDD, p,p'-DDE) [32] in individual fish ranged from of 17 to 510 ng/g, wet weight, arithmetic mean concentrations of each species at each site are illustrated in Figure 2.5. Smallmouth bass at the lower superfund site had the highest level of total DDT compared with all the other sites and other species at the same site. There was moderate evidence of differences in the means of total DDT concentration among fish species at the lower superfund site ($p=0.04$, ANOVA F-test). Average total DDT concentration in whole smallmouth bass was significantly higher than in black crappie but was not different from the DDT level in common carp (Tukey's Studentized range, HSD). Average total DDT concentration in smallmouth bass sampled at the lower superfund site was significantly different from smallmouth bass at other sampling sites ($p=0.007$, ANOVA F-test and $p < 0.05$, Tukey's Studentized Range, HSD). The highest concentration was detected in smallmouth bass from the lower superfund site (510 ng/g,wet weight). Interestingly, it was not the same fish that had the highest level of total PCBs. Total DDT concentrations in other fish species were not different among other sampling sites (black crappie: $p =0.8$, ANOVA F-test; common carp: $p=0.39$, t-test). Only average total DDT in smallmouth bass at the lower superfund site exceeded the U.S. EPA's screening values [32] for DDT-induced carcinogenic effect (117 ng/g) but not for DDT-induced noncarcinogenic effect (2,000 ng/g). All other fish at all other sites were below U.S. EPA's screening values.

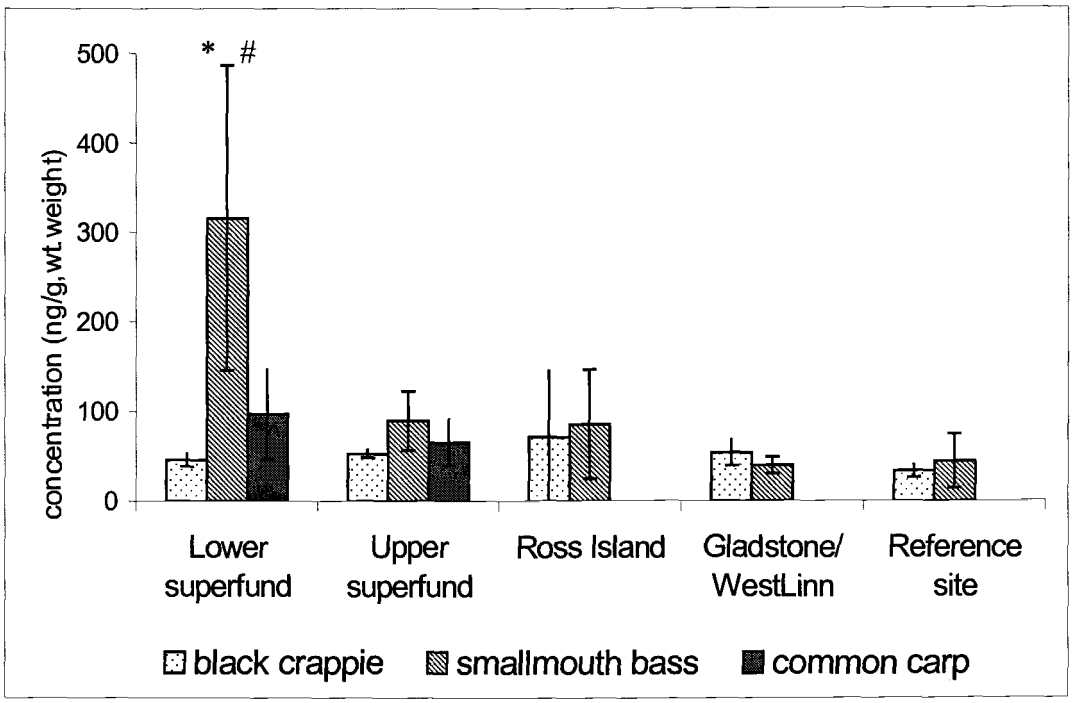


Figure 2.5 Total DDT (sum of p,p'-DDT, p,p'-DDD, p,p'-DDE) concentration (ng/g, wet weight) in whole fish (n=36). The results are mean $\pm$ 1 SD for three fish ($n_{\text{black crappie}} = n_{\text{smallmouth bass}} = n_{\text{common carp}} = 3$) at each site, except at the reference site ($n_{\text{black crappie}} = 2$ and $n_{\text{smallmouth bass}} = 4$).* Species-effect significant, # Site-effect significant, see text. Significant is considered when $p < 0.05$.
U.S. EPA's screening values for recreational fishers are 2000 ng/g (non-carcinogenic effects) and 117 ng/g (carcinogenic effect) and the values for subsistence fishers are 245 ng/g (non-carcinogenic effects) and 14 ng/g (carcinogenic effect).

Anderson et al [1] reported the occurrence of DDT and other organochlorine pesticides in the Willamette Basin depended on local land use and pesticide use history. Manufacturing of DDT during 1947-1954 [38] is one of the historic industrial activities at the Portland harbor superfund site. In addition to site-specific industrial activity, accumulation from upstream sources and other runoff can be another possible potential source of contamination. Average total DDT level in smallmouth bass at the lower superfund site is significantly higher than in

smallmouth bass at any other sites, as shown in Figure 5. When the highest total DDT concentration from one smallmouth bass was omitted from statistical analysis, average total DDT level at the lower superfund site still was significantly higher than any other sites ($p = 0.003$, ANOVA F-test and Tukey's Studentized range, HSD). Average total DDT residues in this species at other sites are much lower and all are below the U.S. EPA's safety level. The DDT manufacturing facility was directly in the middle of the Portland Harbor (~RM 7) and probably represents the large increase of DDT in fish downstream. The lower superfund site (RM 3-6.1) is located adjacent to a historic DDT manufacturing/shipping plant (~RM 7) and DDT residues from disposal and/or spills when the plant operated may be contributing to high DDT contamination in this area. DDT adheres tenaciously to soil particles [50] and the sediments in water eventually move downstream. The other possible sources of contamination such as non-point urban runoff and/or upstream agricultural runoff are sources of contamination at other sites in this study; however their DDT contribution is relatively small in comparison. All sites above lower superfund are below U.S. EPA safety level.

The major component of technical DDT is p,p'-DDT, it usually accounts for 70% or more of the total, whereas o,p'-DDT is a less toxic and less persistent isomer, and generally accounts for only 20%, p,p'-DDD, also an insecticide in its own right, typically accounts for 3% [44]. Although p,p'-DDT is chemically and biochemically stable, once released into the environment, DDT undergoes environmental processes and biotransformations generating DDD and DDE. The details of specific metabolism and environmental process of DDT in the environment is still under some controversy [44, 50]. The most abundant and widespread residues of DDT in the environment are p,p'-DDT, p,p'-DDD and p,p'-DDE. The latter is a highly persistent metabolite in the environment, as well as, in organisms [50]. DDT and its metabolites undergo strong biomagification along trophic transfer. Schmitt et al [7] addressed metabolism of DDT in fish, which is generally accomplished through dechlorination to DDE but generally not to DDD.

Therefore the presence of p,p'-DDD in fish tissue can be from a metabolite of DDT in environment and/or direct input of p,p'-DDD. We found the proportion of p,p'-DDE in fish was predominant and the patterns of percent contribution of each residue to total DDT were similar among species and sites; p,p'-DDT, p,p'-DDD and p,p'-DDE on average account for 16 %, 12% and 73%, respectively.

The average p,p'-DDE levels in bottom-feeding fish and piscivorous fish at Portland have increased from the previous study in 1990 [2]. Curtis et al [2] detected DDE residues in common carp fillet (~30 ng/g) and whole squawfish (~20 ng/g) sampled from the Willamette River at Portland (RM 7) in July 1990. The average DDE levels in whole common carp were 65 and 40 ng/g, wet weight at the lower superfund site (RM 3-6.1) and the upper superfund site (RM 8-11), respectively. Smallmouth bass is comparable to squawfish in terms of trophic level in aquatic food web because both are piscivorous fish species [4]. We detected 228 and 56 ng/g of p,p'-DDE in whole smallmouth bass at the lower superfund site (RM 3-6.1) and at the upper superfund site (RM 8-11). The increase in p,p'-DDE residue in fish tissue is not surprising. A reported DDT half-life in the environment is between 2-15 years [50] and the major route of environmental degradation and biotransformation is dechlorination to the highly persistent metabolite, p,p'-DDE [44]. p,p'-DDE is far more highly persistent than p,p'-DDT , while p,p'-DDD has half-life of greater than 10 years [44]. This therefore appears to be the main reason why p,p'-DDE residues are still detected at significant levels in environmental samples while the levels of p,p'-DDT have been decreasing after banned in the 1970s. Schmitt et al [6] reported total DDT in whole northern squawfish (~160 ng/g) and in whole peamouth (~50 ng/g) collected at Oregon City (~RM 26), in 1984. The averages of total DDT in our comparable fish species collected at Gladstone/West Linn site (RM 23-25) were 40 ng/g in whole smallmouth bass and 54 ng/g in whole black crappie. The unchanged trend of total DDT level at this site supports the evidence of high persistence of DDT and its derivatives in the environment. Our findings and other studies [6, 37] indicate DDT and its

metabolites are still ubiquitous in environmental samples and their levels in fish tissue have changed little although the use of DDT and its derivatives have been banned for 30 years.

All fish contained detectable dieldrin (2 ng/g or higher) with the highest levels in fish from the superfund site (4.6 ng/g in whole common carp from lower superfund site). Aldrin, endrin and methoxychlor were not detected in any fish. Other organochlorine pesticides tested (α-BHC, β-BHC, γ-BHC, δ-BHC, heptachlor, heptachlor epoxide, γ -chlordane, α –chlordane) were not detected or present at low levels in some fish (below detection limit). No correlation was found between organochlorine pesticides distribution and fish species or sampling sites.

Mercury

All fish tested contained mercury (as total mercury) in a range of 0.01 to 0.52 μg/g with the highest level occurring in one black crappie collected at the mouth of Luckiamute River. Despite the fact that much of the mercury in fish tissue is present as methyl mercury, which is known to be neurotoxic to human [51], due to the high cost of methyl mercury analysis, U.S. EPA [32] recommends that total mercury be analyzed and the conservative assumption be made that all mercury measured is methyl mercury. No pattern of mercury distribution was found to correlate with fish species or sampling sites. Surface water can be polluted with mercury from run-off water contaminated by either natural or anthropogenic sources, or from air deposition [51]. Comparison of our mercury fish data to historical mercury fish residues [4], supports the declining residue trend. Recommended screening value for adverse health effect of mercury in fish by U.S.EPA is 0.4 μg/g [32], and only one fish exceeded that warning level in our study (0.52 μg/g) in whole black crappie.

CONCLUSION

This study suggested the average background levels of organochlorine compounds in fish from the Portland Harbor Superfund site and upriver are of public concern to water quality and human health. Average PCB levels in fish in the Willamette River have not declined over the last decade. In addition, the fish PCBs residues still exceed the state and U.S. EPA's safety screening levels. PCBs residues in fish increased as the distance to the superfund site decreased and the highest concentrations were detected in fish from the superfund site. This finding substantiated correlation between total PCBs levels and amount of industrial land use. Portland Harbor is an intensively industrialized and urbanized area in which historical industrial activities may be the potential sources for PCBs contamination. PCBs bioaccumulation profiles in fish were dominated by high chlorine content congeners in which hexachlorobiphenyl congener 153 was the most abundant, and heptachlorobiphenyl congener 180 and pentachlorobiphenyl congener 118 were second most abundant. This finding confirmed relatively low metabolism of high chlorine content congeners in fish.

The highest average total DDT concentration (sum of p, p'-homologs) was detected in smallmouth bass collected at the lower superfund site. No mean total DDT in any fish species at any site exceeded U.S. EPA's Screening values, except at the lower superfund site where the screening value for DDT-induced carcinogenic effect was exceeded in whole smallmouth bass. The p,p'-DDE, stable lipophilic and highly persistent metabolite, was most abundant in fish tissue (~73 %). Residues from a former DDT manufacturing facility appear to be the dominant source of DDT at the lower superfund site. A lower background level of DDT is present throughout the study area due to general non-point urban runoff and/or general agricultural runoff at the watershed level. However, other agricultural organochlorine pesticides tested in this study were not detected or detected below

detection limit (typical 2 ng/g fish sample). Only one tested fish contained mercury exceeding US.EPA screening value for mercury in fish.

According to US.EPA [32], exceedance of screening values is an indication that a monitoring study of contaminant levels should continue and assessment of human health risk due to consumption of fish in this area and risk management should be taken to protect the local consumer health and ecosystem. For local fish consumers, reduce consumption of fish from this area, avoiding whole body preparation and cooking fish can reduce the intake and associated adverse health effects of these chemicals [52]. The present study may serve as a bases for future study of occurrence and abundance of environmental contaminants at the Willamette River, particularly at the Portland harbor area. Substantial analysis of other environmental samples such as sediments, water, and other aquatic biota are needed to better understand and update the distributions of chemical contaminant fate and distribution in this region.

ACKNOWLEDGEMENTS

We thank Beth Siddens and Rod Inman for their assistance in the completion of this work and Dr. Carl Schreck and Dr. Lawrence Curtis, Oregon State University, for helpful discussions, Bill Monroe of the Oregonian, and the Oregon Bass and Panfish Club for collecting the fish, and The Oregonian for partial funding of this project. The Royal Thai Government Scholarship to Doolalai Sethajintanin is acknowledged.

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CHAPTER 3

ESTIMATION OF MERCURY AND ORGANOCHLORINES EXPOSURES AND POTENTIAL HEALTH RISK BY CONSUMPTION OF FISH FROM THE LOWER WILLAMETTE RIVER, OREGON

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Manuscript in preparation to submit to

The Journal of Environmental Health Perspectives

ABSTRACT

Polychlorinated biphenyls (PCBs), organochlorine pesticides and mercury have been detected in three recreation fish species from the lower Willamette River, Oregon. Total PCBs levels (sum of 25 individual congeners) were detected at higher levels than the health protection standard levels. Risks from these persistent chemical compounds present in fish were evaluated for the public health risk of eating fish from this segment of the river. Carcinogenic risks from PCBs and organochlorine pesticides and non-carcinogenic risks from organochlorine compounds and mercury were assessed. Hazard quotient indices ($\sum HQ>1$) indicated consumption of contaminated fish by recreational fishers and subsistence fishers at the lower portion of the river might cause chronic adverse health effects. Total cancer risk at all sites of this study exceeded acceptable lifetime cancer risk level (10^{-5}). The greatest contributors to hazard quotients for non-carcinogenic risk and carcinogenic risk were total PCBs and dioxin-like PCBs, respectively. The 10^{-5} upper limit of lifetime cancer risk as the health protection standard, suggested no fish consumption in the unit of meals/year for smallmouth bass and black crappie from the lower Willamette River is acceptable because of the presence of PCBs at the concentrations that can pose a long term toxic threat to local fish consumers.

INTRODUCTION

The ultimate goal of human health risk assessment is to protect human health from current and potential threats posed by uncontrolled hazardous substances released into the environment, which may contaminate food or other environmental compartments [1]. Human health risk is the prediction of an adverse

health effect and its consequential severity caused by current and future exposures to a chemical or a hazard. Since toxicological experiments often cannot be conducted directly in humans, extrapolation from the animal experimental model is most often used [2].

The risk assessment process has four main components, which are hazard identification, toxicity assessment or dose-response assessment, exposure assessment, and risk characterization. Hazard identification and toxicity assessment are the science-based characterization while exposure assessment and risk characterization utilize the scientific database to predict risk. Since interpretation of contaminant levels in environmental samples (i.e., water, soils, air, and fish tissue) are complicated by environmental and biological processes, the risk to society may be real or perceived [2]. The perceived risk may have just as much influence on important regulatory decisions that affect both remedial response processes and public health regulations [1, 3].

The Portland Harbor located on the lower Willamette River, Oregon, has been recently declared a superfund site [4, 5]. Portland is the largest metropolitan area in Oregon; industrial facilities and marine traffic are considered intensive in the Portland Harbor area. Chemical contaminants within the Portland Harbor sediments contain polycyclic aromatic hydrocarbon (PAHs), polychlorinated biphenyls (PCBs), chlorinated pesticides and heavy metals [4, 5]. Possible sources of contamination include historical or current industrial operations, surface runoff, direct discharge, stormwater outfalls, spill releases from ships or barges and upstream sources [6]. Several studies have reported contaminant residues in the water, sediments, and/or fish residing in the Willamette River and its tributaries and some contaminants exceeded the safety levels established by either the State of Oregon or the U.S. Environmental Protection Agency (U.S. EPA) [7-11].

The Willamette River has important fish habitat, which provides a favored sport fishing area, nearly fifty fish species are present within the Willamette basin [11]. The main stem near Portland provides recreational fishing for resident fish

such as; black crappie, white crappie, smallmouth bass, and anadromous salmonid including chinook, steelhead, and coho salmon. Fish may be contaminated with chemical contaminants by direct intake from the physical environment, as well as, by intake from food [12]. Lipophilic contaminants such as PCBs and chlorinated pesticides are accumulated in the food chain and increase in concentration as they pass from prey to predator. Therefore, recreational and subsistence fishers are susceptible to exposure to hazardous lipophilic chemical residues in fish from contaminated areas.

Recently, our laboratory has evaluated and updated chemical contaminant residues in three recreational fish species collected during summer 2000 from the Portland Harbor superfund site and up the Willamette River [13]. The study area covered the lower 20-mile portion of the Willamette River, from river mile 3 (RM3) at the head of Multnomah Channel to RM 25 at Milwaukee. This section of the Willamette River was divided into four sections, described as lower superfund site, upper superfund site, Ross Island and Gladstone/West Linn (Figure 3.1). These four sites are considered as dominantly industrial and urban land use [9]. Two of our additional sites considered as dominantly agricultural land and forest uses were included in the study for purposes of comparison and were designated as the reference sites. One of reference sites was Henry Hagg Lake near Forest Grove, which is 25 miles west of Portland. The second reference site was the Willamette River at the mouth of Luckiamute River, south of Salem (RM 107 –RM 108), Figure 3.1. Three popular recreational fish species collected were common carp (*Cyprinus carpio*), smallmouth bass (*Micropterus dolomeieui*) and black crappie (*Pomoxis nigromaculatus*). The contaminants determined consisted of 25 individual PCB congeners, 15 organochlorine pesticides (including p,p'-DDT , p,p'-DDD, p,p'-DDE, α-BHC, β-BHC, γ-BHC, δ-BHC , α -chlordane, γ -chlordane , heptachlor, heptachlor epoxide, aldrin , dieldrin, endrin, and methoxychlor), and mercury. Our results indicated chemical contaminant levels are of concern for local fish consumers. Average total PCB concentrations and total DDT (sum of p,p'-

homologs) exceeded U.S.EPA Screening Values (SVs). Exceedance of the SVs is an indication that assessment of human health risk is required for these sites [14].

When contaminants such as PCBs in fish tissues exceed the U.S. EPA screening values, the potential risk of consuming fish to cause chronic deleterious health effects posed by these chemicals is possible. High concentrations of PCBs, organochlorine pesticides and mercury contamination in fish can be transferred to humans through the food chain. Therefore human health risk calculations from our recent fish data will be valuable for assessing risk for recreational fishers and subsistence fishers in this area. One of the main purposes of this pilot study is to evaluate human health risk due to consumption of specific contaminated fish species from the lower Willamette River including the Portland Harbor superfund site. The estimates of hazard quotients and lifetime cancer risks from consuming fish contaminated by these chemicals are calculated. Chemical contaminant data are based on our recent fish data [13] while the risk assessment approach and risk parameters are based on U.S.EPA guidelines [1].

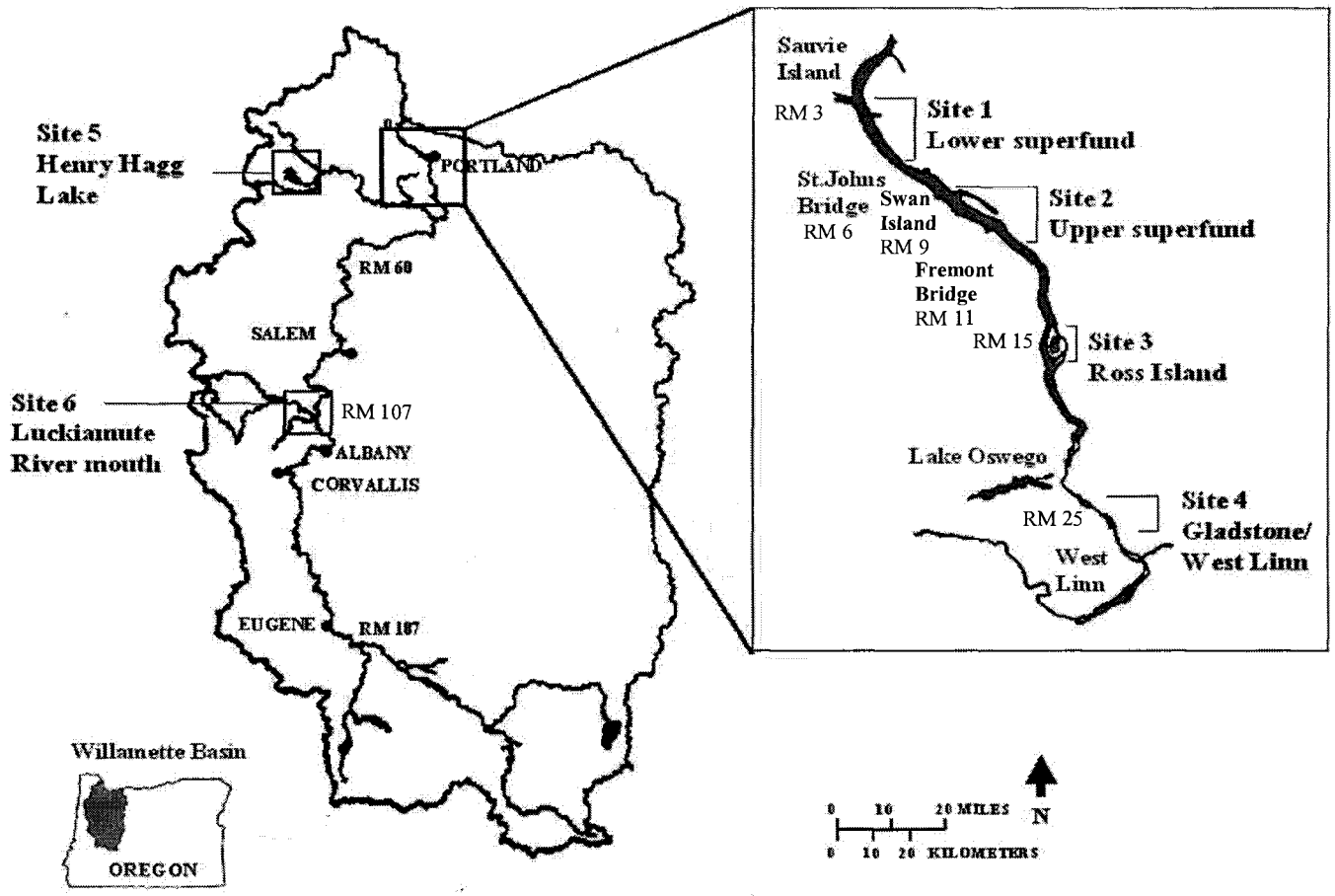


Figure 3.1 Willamette River Basin, Oregon, showing location of sampling site in the study during summer 2000. Site 5 and 6 were designated as reference site

RISK ASSESSMENT APPROACH

Risk assessment is the method for evaluating risk to humans exposed to toxicants. It involves the evaluation of scientific information on the hazardous properties of environmental agents that may pose adverse health effects on exposed humans, and the estimation of the probability that exposed populations will be harmed due to their occurrences and the characteristics of the resulting risk [3]. This process generally consists of hazard identification, toxicity assessment, exposure assessment, and risk characterization. After the risk assessment is completed, risk management in which risk assessment is integrated with other issues such as political, social, economic, and engineering consideration are used to make a decision about the need and method for risk reduction at a remedial site [1, 3].

HAZARD IDENTIFICATION

Hazard identification encompasses identification and quantification of potential chemicals that are suspected to pose health hazards and characterization of their toxicological effects. Inclusion of chemical contaminants evaluated in our study was based on historical contaminant residue data in this region [6-11] along with U.S. EPA guidance for recommended target analytes in a fish contaminant study [14]. PCBs, organochlorine pesticides and mercury in the Willamette River due to historical anthropogenic contamination remain a public and regulatory concern. These chemicals are persistent in the aquatic environment and have high potential for bioaccumulation and biomagnification in fish tissue and in the aquatic food chain. Methods of sample collection and chemical analysis used in this risk assessment are described in detail in chapter 2.

Table 3.1 summarizes the fish residue data. All fish tested contained mercury as total mercury in a range of 0.01-0.52 μg/g. α-BHC, β-BHC, γ-BHC, δ-BHC, heptachlor, heptachlor epoxide, γ-chlordane, α-chlordane were not detected or were present at low levels in only some fish. Total PCBs are the sum of all 25 PCB congeners analyzed in our study. Inclusion of PCB congeners in the analysis is based on toxicity, frequency of occurrence and abundance in environmental samples, relative abundance in animal tissues and analytical capability [15, 16]. Toxicity of some PCB congeners is associated with induction of mixed oxidases [15]. Some PCB congeners elicit toxic effects similar to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD; dioxin) [15, 17]. Biochemical and toxic response of these dioxin-like congeners are mediated through binding to the cytosolic aryl hydrocarbon receptor (Ah receptor) in target cells [18]. Besides binding to the Ah receptor, dioxin-like PCBs show a structural relationship to dibenzo-p-dioxins and dibenzofurans, exhibit dioxin-mediated biochemical and toxic responses and are persistent and bioaccumulate through the food chain [17]. These toxic responses include dermal toxicity, immunotoxicity, adverse effects on reproduction, development and endocrine functions, decreased vitamin A levels, altered lipid metabolism, carcinogenicity and tumor promotion activity [18]. However, only a small portion of the constituents of PCB mixtures, 12 congeners from a total of 209 possible congeners, exhibits dioxin-like activity [17]. Other congeners elicit their toxicity including neuro-behavioral, neurotoxic, carcinogenic and endocrine changes via multiple unrelated mechanisms of action but not the Ah receptor mediated-mechanism [19]. Both dioxin-like and non dioxin-like PCBs contribute to overall PCB toxicity. Risk assessment of only dioxin-like PCBs or non dioxin-like PCBs could result in an underestimate of adverse health effects of environmental PCB mixtures. Therefore, we analyzed both dioxin-like congeners (coplanar PCBs; PCB 77, 126, 169, and mono-ortho PCBs; PCB 105, 114, 118, 156, 189) and non dioxin-like congeners (PCB 37, 44, 49, 52, 62, 74, 87, 99, 101, 128, 138, 153, 166, 170, 180,183, 187,189) [16, 17, 19].

Table 3.1 Concentrations (ng/g, wet weight) of chemical contaminants determined in whole fish (n=36)

Chemical	Lower superfund site			Upper superfund site			Ross Island		Gladstone/West Linn		Reference site	
	SM	BC	CC	SM	BC	CC	SM	BC	SM	BC	SM	BC
Total PCBs	323 (126)	56.9 (13.6)	92.9 (47.5)	285 (212)	60.2 (14.3)	181 (48.1)	111 (95.0)	56.5 (45.1)	38.7 (6.96)	48.7 (4.38)	22.5 (7.45)	42.9 (32.1)
[a]Non dioxin-like PCBs	249 (97.6)	46.8 (12.1)	79.7 (44.1)	249 (191)	48.7 (12.0)	149 (40.2)	91.6 (76.0)	43.3 (33.7)	30.0 (5.79)	38.4 (5.89)	17.0 (5.37)	32.7 (25.0)
[a]Dioxin-like PCBs	73.4 (29.4)	10.1 (1.66)	13.5 (5.15)	35.9 (21.1)	11.9 (2.7)	32.9 (11.0)	19.0 (19.3)	13.3 (11.4)	8.71 (1.89)	10.5 (1.79)	5.48 (2.08)	10.5 (7.48)
[a,b]PCBs-TEQs	12.2	1.81	2.12	41.8	34.9	4.44	35.9	35.1	68.3	38.1	1.15	1.62
Total DDT	316 (171)	45.7 (7.14)	97.3 (50.1)	89.5 (32.8)	52.5 (4.4)	65.6 (26.1)	86.1 (60.7)	72.4 (73.4)	40.2 (9.27)	53.6 (14.3)	45.2 (30.6)	33.9 (7.0)
Dieldrin	2.37 (1.27)	1.21 (0.37)	2.93 (1.81)	1.00 (0)	1.57 (0.98)	2.00 (1.73)	2.41 (1.31)	1.43 (0.75)	1.47 (0.81)	1.00 (0)	1.00 (0)	1.00 (0)

SB = smallmouth bass, BC = black crappie, CC = common carp
Values in parenthesis are 1 standard deviation
[a]See text for details
[b]Toxic equivalent concentrations (TEQs) based on toxic equivalent factors (TEFs) by Van den Berg et al.,1998 [17], PCBs-TEQs in pg/g

TOXICITY ASSESSMENT OR DOSE-RESPONSE ASSESSMENT

The toxicity assessment evaluates the toxicity information and characterizes the relationship between the dose of contaminant intake and the incidence of adverse health effects in the exposed population [1, 20]. In order to evaluate the inherent toxicity of hazardous compounds at the superfund site, identification of toxic endpoints and selection of toxicity values to assess the significance of exposure receptor to such compounds are necessary. Toxicity information and toxicity values used in this study were primarily obtained from U.S.EPA databases (the Integrated Risk Information System, IRIS and health effects assessment summary tables, HEAST)[21, 22] and other literatures [12, 15, 18, 19, 23, 24].

Tables 3.2 and 3.3 present toxicity values and a summary of critical toxic effects of chemicals identified in this study. Toxic potency of dioxin-like PCBs is compared relative to the toxic potency of the most toxic congener, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) [17, 19, 25]. To assess toxicity of dioxin-like PCBs, an interim approach of TCDD-toxic equivalency factor (TEF) has been utilized to yield TCDD-toxic equivalent concentrations or TEQs [17, 19, 25]. U.S. EPA has classified 2,3,7,8-TCDD as a probable human carcinogen with a slope factor of 1.56×10^{5} $(mg/kg\text{-}d)^{-1}$[26]. However, EPA's office of Research and Development has been re-evaluating the potency of dioxins and dibenzofurans, the information for these compounds is therefore subject to change depending on the result of this re-evaluation [14].

Table 3.2 Non-carcinogenic toxicity values for target analytes and a summary of their critical effects

Chemical	Oral RfD[a] (mg/kg-d)	Confidence	UF/MF[b]	Critical toxic effect
alpha-BHC	NA[c]	NA	NA	NA
beta-BHC	NA	NA	NA	NA
gamma-BHC (lindane)	3×10^{-4}	medium	10/-	Liver, kidney toxicity
delta-BHC	NA	NA	NA	NA
Heptachlor	5×10^{-4}	low	100/-	liver weight increases
Heptachlor Epoxide	1.3×10^{-5}	low	1000/-	liver weight increases
Aldrin	3×10^{-5}	medium	1000/-	liver toxicity
Total Chlordane	5×10^{-4}	medium	300/1	hepatic necrosis
Dieldrin	5×10^{-5}	medium	100/-	liver lesion
Endrin	3×10^{-4}	medium	100/-	mild histological lesion in liver occasional convulsion
total DDTs (sum of p,p'-DDT, p,p'-DDE and p,p'-DDD)	5×10^{-4}	medium	10/-	liver lesion
Methoxychlor	5×10^{-3}	low	100/-	excessive loss of litter
PCBs	2×10^{-5}	medium	300/-	immunological effect, developmental effect hepatotoxicity
Mercury (methyl mercury)	1×10^{-4}	high	10/1	developmental, neuropsychological impairment

Source: U.S.EPA IRIS and HEAST database [21,22]
[a]Oral reference dose, see text
[b]UF = uncertainty factor, MF = modifying factor
[c]NA = not available

Table 3.3 Oral carcinogenic toxicity values of target analytes

chemical	Oral SF[a] $(mg/kg\text{-}d)^{-1}$	Tumor type/location	EPA carcinogenicity Classification[b]
alpha-BHC	6.3	hepatic nodule and hepatocellular carcinomas	B2
beta-BHC	1.8	hepatic nodule and hepatocellular carcinomas	C
gamma-BHC(lindane)	1.3	liver tumor	B2/C
delta-BHC	NA	NA[c]	D
Heptachlor	4.5	hepatocellular carcinomas	B2
Heptachlor Epoxide	9.1	hepatocellular carcinomas	B2
Aldrin	17	liver carcinoma	B2
Total Chlordane	0.35	hepatocellular carcinomas	B2
Dieldrin	16	liver carcinoma	B2
Endrin	NA	NA	D
total DDTs (sum of p,p'-DDT, p,p'-DDE and p,p'-DDD)	0.34	liver benign and malignant	B2
Methoxychlor	NA	NA	D
PCBs - dioxin-like - non dioxin-like	 1.56×10^5 2	hepatocellular carcinomas hepatocellular carcinomas	 B2 B2
Mercury (methylmercury)	NA	NA	C

Source: U.S.EPA IRIS and HEAST database [21,22]
[a]Oral slope factor, see text
[b]A= human carcinogen, B1= probable human carcinogen (limited human data are available), B2= probable human carcinogen (sufficient evidence in animals and inadequate or no evidence in humans), C= possible human carcinogen, D= not classified as to human carcinogenicity, E= evidence of non-carcinogenicity [1]
[c]NA = not applicable

Reference dose (RfD) for oral exposure is the toxicity value used to evaluate non-carcinogenic effects resulting from exposure to harmful substances at a superfund site. U.S.EPA [1] has defined the RfD as an estimate (with uncertainty spanning an order of magnitude) of a daily exposure level for the human population, including sensitive subgroups, during a lifetime of exposure, with this level an appreciable risk is unlikely to cause deleterious effects. Since toxicological experiments cannot be conducted directly in humans, RfDs are extrapolated from animal experimental models. Uncertainty factors and a modifying factor are used to account for any inherit variables associated within the data extrapolations for estimating the RfD [1]. Uncertainty factors are used to account for interspecies variability between human and laboratory animals, variation in the general population to protect sensitive subgroups, variability from extrapolating from a sub-chronic study to chronic exposure, and uncertainty associated with extrapolating from lowest observed adverse effect level (LOAEL) to no observed adverse effect level (NOAEL). In addition, a modifying factor is based upon an evaluation of additional uncertainties of the data used to create RfD for the chemical not explicitly addressed by the uncertainty factors mentioned above. Generally, uncertainty factor and modifying factor are a tenfold factor [27]. The sum of all uncertainty/modifying factors can range widely depending on toxicity database. Therefore uncertainty factors in some cases can reach 10,000.

Cancer slope factor (SF) and its associated potential for carcinogenicity are the toxicity data used to assess potential human carcinogenic risk. U.S. EPA assigned a weight of evidence classification system for carcinogenicity based on supporting evidence to determine the likelihood that the substance is a human carcinogen. The EPA classification system for weight of evidence is available in EPA's Risk Assessment Guidance for Superfund [1]. Slope factor is used to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of potential toxic chemical over a lifetime. Unlike, non-carcinogenic effect, cancer risk is a non-threshold value. It is based on

the assumption that there is no safe dose with a risk of zero (except at zero dose) because even a small number of molecular events can evoke changes in a single cell resulting in uncontrolled cellular proliferation and then leading to a clinical state of disease [1, 3].

EXPOSURE ASSESSMENT

There are several exposure pathways that residents can be exposed to toxic agents at a superfund site (i.e., inhalation, direct contact, ingestion). Consumption of contaminated fish is the potential pathway for exposure to PCBs, organochlorine pesticides and mercury residues from fish from the Portland Harbor superfund site and upriver sampling sites. Because commercial fishing is minimal in this area whereas recreational fishing is very popular; therefore, recreational fishers and the subsistence fishers are the direct target population. Recreational fishers and subsistence fishers are potentially the exposed population because they are more likely to eat large amounts of locally caught fish. To evaluate risk associated with consumption of contaminated fish, fish species at each sampling site were assessed independently.

Quantification of exposure depends on chemical contaminant concentration detected in fish tissues, which is estimated by using the arithmetic means from the current fish contaminant data. The exposure estimates are the maximum exposures that are reasonably expected to occur at a site for a specific pathway [1]. Chemical intake or exposure is normalized for time and body weight and defined as "Chronic Daily Intake", which is expressed in a unit of mg chemical per kg body weight per day (mg/kg-day) [1]. The equation for chemical intake is

$$CDI\ (mg/kg\text{-}day) = \frac{C \times CF \times IR \times EF \times ED}{BW \times AT} \qquad \text{equation (1)}$$

Where: CDI = Chronic daily intake of a specific chemical (mg/kg-day)

C = Chemical concentration in fish (mg/kg)

CF = Conversion factor (kg/g)

IR = Ingestion rate (g/day)

EF = Exposure frequency (days/year)

ED=Exposure duration (years)

BW = body weight (kg)

AT = Averaging time (period over which exposure is averaged, days)

The exposure parameters used to calculate exposure concentrations are specific in particular scenarios. However, this information is often unavailable or inadequate at the superfund site or varies from time to time depending on the nature of exposure sources and/or lifestyle of the target populations. When exposure parameters are not available or not adequate, the choices of specific values and assumptions used in CDI calculation are generally based on U.S. EPA guidance for risk assessment at a superfund site [1, 14, 28]. Exposure parameter values and their sources and rationale are presented in Table 3.4.

Table 3.4 Exposure parameters used in chronic daily intake calculation

Parameter	Value	Source/Rationale
Concentration (C)	arithmetic mean (mg/kg)	Species and chemical specific
Conversion factor (CF)	0.001 kg/g	
Ingestion rate (IR)	variable 17.5 g/day 142.4 g/day	 Default value for the general adult population and recreational fishers[a] Default value for subsistence fishers[a]
Exposure Frequency (EF)	350 days/year 365 days/year	Recreational Scenario[b] Assume 365 days/year exposure[b]
Exposure Duration (ED)	30 years 75 years	National upper-bound time (90th percentile) at one residences[c] Average lifetime expectancy[a]
Body Weight (BW)	70 kg	Mean adult body weight[d]
Average time (AT)	Noncarcinogen: ED x 365 days/year Carcinogen: 75 years x 365 days/year	By definition[c]

Source: various sources [a, b, c, d,] see reference [14], [32], [1], [25], respectively

Consumption rates critically affect reliability and accuracy of risk assessment calculations. Choosing inaccurate consumption rates for the target population may underestimate or overestimate exposure resulting in no protection or overprotection fish consumer health. Several surveys reporting fish consumption patterns relied on fish species, fishing calendar, cultural factors (socio-

demographics), economic determinants, demand for certain protein and nutrients available extensively in fish such as omega-3 fatty acids eicosapentanoic acid (EPA) and decosahexanoic acid (DHA) in fatty fish species [29-32].

The Oregon Department of Environmental Quality (Oregon DEQ) is evaluating site-specific fish consumption information to identify the consumption rates for local fish consumers at Portland Harbor area. The available consumption rate information ranged widely with large variance and they were not specific for the Willamette River (Mike Poulson, the Oregon Department of Environmental Quality, Portland, Oregon (Personal communication, June 6, 2002). However, the most recent rates at the middle Willamette River were 17.5 g/day and 142 g/day for the 90^{th} and 95^{th} percentile of the consumers, respectively whereas the estimates at the Columbia Slough area, north of Portland, ranged from 14 to 105 g/day. A likely consumption rate that The Oregon DEQ is favoring at this stage of evaluation is 78 g/day (90^{th} percentile). The U.S. EPA recommended ingestion rate for the general population and recreational fishers is 17.5 g/day and ingestion rate for subsistence fishers is 142.4 g/day [14]. The estimate of 17.5 g/day is the 90^{th} percentile and the estimate of 142.4 is the 99^{th} percentile of national per capita rate for fish consumption that includes all individuals who may eat fish and those who do not eat fish. Because local fish consumption rates for recreational fishers and subsistence fishers at Portland Harbor are not available; therefore the default values by U.S. EPA are appropriate. These values can capture both the low and high end of the consumption rates for the surrounding areas of the lower Willamette River.

Exposure concentrations for carcinogenic and non-carcinogenic effects were calculated separately by using different exposure parameters due to the difference in their critical toxicity values. Exposure duration and exposure frequency are based on national statistics of the upper bound of years spent by an individual at one residence, and an upper value of 30 years is considered a reasonable maximum residential exposure to a toxicant at a superfund site [1, 28]. However, for cancer risk assessment that is described in terms of lifetime

probability, lifetime exposure (75 years by convention) is considered a reasonably approximation [28]. There is uncertainty associated with estimates of exposure regarding to the exposure parameter (i.e., consumption rate, consumption frequency, exposure duration, type and portion of fish consumed and fish preparation method) used in assessing human health risk.

Individual PCB congeners have different physicochemical properties that lead to different toxicity and different distributions in the environment. Dioxin-like PCBs and non dioxin-like PCBs have different toxic mechanisms as mentioned early, however, both contribute to the overall carcinogenic effect of environmental PCB mixtures. Calculations of PCBs exposure concentration for cancer risk without accounting for dioxin-like congeners could result in an underestimate of the potential carcinogenic effect of environmental mixture [19]. Total PCBs cancer risk is assessed by combining risks of dioxin-like and non dioxin-like congeners [16, 33] and the toxic equivalency factor (TEF) approach is applied for dioxin-like congeners [16-19, 25]. The TEF approach enables the expression of overall toxic potential of the mixture of individual dioxin-like congeners as one integrated parameter, TCDD-toxicity equivalent concentration (TEQ), in which the toxic potency of dioxin-like PCBs is related to the toxicity of 2,3,7,8-TCDD. Individual dioxin-like PCB concentrations are converted to the TEQ by multiplying their concentrations with their respective TEF values established by World Health Organization [17], equation (2). The TEQs are then summed within a sample to generate the total concentration of TEQ contributed by dioxin-like congeners. PCB-TEQs are shown in Table 3.1.

$$TEQ = \Sigma [PCB_i \times TEF_i]_n \qquad \text{Equation (2)}$$

Where i = individual congener, n= the numbers of congeners

TEQ then replaces C in equation (1) for the dioxin-like PCBs exposure assessment calculation and will be treated as dioxin in cancer risk characterization evaluation. Whereas, non dioxin-like PCBs concentrations are summed within a fish sample and the product is then calculated for non-dioxin congeners exposure concentration. For non-carcinogenic risk of PCBs, total PCBs concentration is applied to equation (1) regardless of dioxin-like congeners or non dioxin-like congeners [16].

HUMAN HEALTH RISK CHARACTERIZATION

Risk characterization is the integration of exposure assessment and toxicity assessment to yield qualitative and quantitative estimates of carcinogenic risk and systemic hazards. Quantitative risk assessment approach is different for carcinogenic and noncarcinogenic effects. Noncarcinogenic effects are assumed to manifest after exposure to the threshold dose while carcinogenic effects are not considered to have a threshold dose to express a response [34]. Carcinogenic risk is estimated as the increase in probability of an individual developing cancer over a lifetime of exposure [1].

Carcinogenic risk

Carcinogenic risk estimates are assessed by multiplying the oral slope factor for the carcinogen by the chronic daily intake, assuming linearity in the low-dose portion of the multistage model dose-response curve [1].

$$Risk = CDI \times SF \qquad \text{equation (3)}$$

Where: Risk = a unitless probability of estimated chemical specific individual excess lifetime cancer risk

CDI = Chemical specific chronic daily intake (mg/kg-day)

SF = Chemical specific carcinogenic slope factor $(mg/kg\text{-}day)^{-1}$

An excess individual lifetime cancer risk level (RL) is an assigned level of maximum acceptable individual lifetime risk, i.e., RL = 10^{-5} for a level of risk not to exceed one excess case of cancer per 100,000 individuals exposed over a lifetime [14]. A risk range of 10^{-4} to 10^{-7} is typically acceptable and the states have the flexibility to select an appropriate RL value based on site-and population-specific factors [14]. Generally, cancer risks below 10^{-6} are usually considered to be of minimal concern, but risks greater than 10^{-4} are unacceptable and may call for remediation or control to restrict access to the site [34]. Consistence with U.S.EPA fish advisories [14], the risk level of 10^{-5} is used in this study to evaluate possible cancer risk for adult local fish consumers at the lower Willamette River.

For simultaneous exposures to multiple chemicals such as at the superfund site, estimating risk by considering one chemical at a time might underestimate the risks associated with simultaneous exposure to multiple chemicals. To account for simultaneous exposure to multiple chemicals, U.S.EPA assumes dose additivity and independence of action by the compound involved [1]. The overall carcinogenic risk, therefore, is the summation of each individual chemical's risk regardless of species, carcinogenic classification or tumor type, or mechanism.

Tables 3.5, 3.6, 3.7 and Figure3.2 show cancer risk values for individually detected chemicals and overall cancer risk values for each species for two different scenarios of fish consumption. Figure 3.2 displays the trend of overall cancer risks in each species at each study site for the general population and recreational fishers

at 30-year exposure duration. For the "average" scenario (general population and recreational fishers, consumption rate = 17.5 g/d and 30-year exposure duration), all estimates for total risk in each species at all sites exceeded the health protection standard (cancer risk level = 10^{-5}) (Table 3.7). Under this exposure assumption, total risks ranged from 2.5 x 10^{-5} in smallmouth bass at the reference site to 1.0 x 10^{-5} in smallmouth bass at Gladstone/West Linn site. For a lifetime exposure assumption (75-year exposure duration) for the same population group, the total risk ranged from 6.6 x 10^{-5} in smallmouth bass at the reference site to 2.2 x 10^{-2} in smallmouth bass at Gladstone/West Linn. Considering individual fish species, the relative total cancer risk listed in decreasing order are smallmouth bass, black crappie and common carp, see Figure 3.2. The relative total cancer risks for smallmouth bass regarding to study site listed in decreasing order are Gladstone/West Linn site, the upper superfund site and Ross Island site, the lower superfund site, and the reference site, see Figure 3.2. The total cancer risks in black crappie at the upper superfund site, Ross Island and Gladstone/West Linn were similar and higher than the total cancer risks at the lower superfund site and the reference site. In common carp, the total cancer risks were approximately equivalent at the lower and the upper superfund site. The trends of total cancer risks in other scenarios are similar, see Figure 3.2 with increases in the risk values as the consumption rate and exposure duration increased.

Table 3.5 Cancer risk values of individually detected chemicals in each fish species at a 30-year exposure duration

site	IR[a] (g/day)	fish species	dioxin-like PCBs	non-dioxin like PCBs	α-BHC	β-BHC	γ-BHC	Hepata-chlor	heptachlor epoxide	aldrin	total chlordane	dieldrin	total DDTs
lower superfund	17.5	BC	2.7E-05	9.0E-06			1.2E-07		8.7E-07	5.4E-07	6.7E-08	1.9E-06	1.5E-06
		SB	1.8E-04	4.8E-05		5.7E-08	1.2E-07	1.4E-07	8.7E-07	5.4E-07	9.8E-08	3.6E-06	1.0E-05
		CC	3.2E-05	1.5E-05			8.4E-08	1.4E-07	8.7E-07		8.4E-08	4.5E-06	3.2E-06
	142.4	BC	2.2E-04	7.3E-05			1.0E-06		7.1E-06	4.4E-06	5.5E-07	1.5E-05	1.2E-05
		SB	1.5E-03	3.9E-04		4.6E-07	1.0E-06	1.2E-06	7.1E-06	4.4E-06	8.0E-07	3.0E-05	8.4E-05
		CC	2.6E-04	1.2E-04			6.8E-07	1.2E-06	7.1E-06		6.8E-07	3.7E-05	2.6E-05
upper superfund	17.5	BC	5.2E-04	9.3E-06	2.0E-07	5.7E-08	1.2E-07	2.9E-07	2.9E-07		6.7E-08	2.4E-06	1.7E-06
		SB	6.2E-04	4.8E-05	4.0E-07	1.2E-07	1.2E-07	4.3E-07	4.4E-07	1.1E-06	6.7E-08	1.5E-06	2.9E-06
		CC	6.6E-05	2.9E-05	2.0E-07	5.7E-08	1.2E-07	2.9E-07	8.7E-07	5.4E-07	1.8E-07	3.1E-06	2.1E-06
	142.4	BC	4.3E-03	7.6E-05	1.6E-06	4.6E-07	1.0E-06	2.4E-06	2.3E-06		5.5E-07	2.0E-05	1.4E-05
		SB	5.1E-03	3.9E-04	3.3E-06	9.4E-07	1.0E-06	3.5E-06	3.6E-06	8.9E-06	5.5E-07	1.2E-05	2.4E-05
		CC	5.4E-04	2.3E-04	1.6E-06	4.6E-07	1.0E-06	2.4E-06	7.1E-06	4.4E-06	1.5E-06	2.5E-05	1.7E-05
Ross Island	17.5	BC	5.2E-04	8.3E-06			8.4E-08	2.9E-07	2.9E-07		6.7E-08	2.2E-06	2.4E-06
		SB	5.4E-04	1.8E-05	2.0E-07	5.7E-08	1.2E-07	1.4E-07	5.8E-07		6.7E-08	3.7E-06	2.8E-06

Table 3.5 Continued

site	IR[a] (g/day)	fish species	dioxin-like PCBs	non-dioxin like PCBs	α-BHC	β-BHC	γ-BHC	Hepata-chlor	heptachlor epoxide	aldrin	total chlordane	dieldrin	total DDTs
Ross Island	142.4	BC	4.3E-03	6.8E-05			6.8E-07	2.4E-06	2.3E-06		5.5E-07	1.8E-05	1.9E-05
		SB	4.4E-03	1.4E-04	1.6E-06	4.6E-07	1.0E-06	1.2E-06	4.8E-06		5.5E-07	3.0E-05	2.3E-05
Gladstone West Linn	17.5	BC	5.7E-04	7.4E-06			1.2E-07	1.4E-07	5.8E-07		6.7E-08	1.5E-06	1.7E-06
		SB	1.0E-03	5.8E-06	6.0E-07		1.2E-07	4.3E-07			6.7E-08	2.3E-06	1.3E-06
	142.4	BC	4.6E-03	6.0E-05			1.0E-06	1.2E-06	4.8E-06		5.5E-07	1.2E-05	1.4E-05
		SB	8.3E-03	4.7E-05	4.9E-06		1.0E-06	3.5E-06			5.5E-07	1.8E-05	1.1E-05
Reference site	17.5	BC	2.4E-05	6.3E-06				4.3E-07		1.6E-06	5.0E-08	1.5E-06	1.1E-06
		SB	1.7E-05	3.3E-06	1.5E-07	8.6E-08		2.2E-07	4.4E-07	8.2E-07	5.0E-08	1.5E-06	1.5E-06
	142.4	BC	2.0E-04	5.1E-05				3.5E-06		1.3E-05	4.1E-07	1.2E-05	9.0E-06
		SB	1.4E-04	2.7E-05	1.2E-06	7.0E-07		1.8E-06	3.6E-06	6.6E-06	4.1E-07	1.2E-05	1.2E-05

Values in shading show lifetime cancer risks > 10^{-5} (chemical of potential concern)
BC= black crappie, SB = smallmouth bass, CC = common carp
[a]Ingestion rate = 17.5 g/day for general population and recreational fisher
Ingestion rate = 142.4 g/day for subsistence fishers

Table 3.6 Cancer risk values of individually detected chemicals in each fish species at 75-year exposure duration

site	IR[a] (g/day)	fish species	dioxin-like PCBs	non-dioxin like PCBs	α-BHC	β-BHC	γ-BHC	Hepata-chlor	heptachlor epoxide	aldrin	total chlordane	dieldrin	total DDTs
lower	17.5	BC	7.1E-05	2.3E-05			3.3E-07		2.3E-06	1.4E-06	1.8E-07	4.8E-06	3.9E-06
superfund		SB	4.8E-04	1.2E-04		1.5E-07	3.3E-07	3.7E-07	2.3E-06	1.4E-06	2.6E-07	9.5E-06	2.7E-05
		CC	8.3E-05	4.0E-05			2.2E-07	3.7E-07	2.3E-06		2.2E-07	1.2E-05	8.3E-06
	142.4	BC	5.7E-04	1.9E-04			2.6E-06		1.9E-05	1.1E-05	1.4E-06	3.9E-05	3.2E-05
		SB	3.9E-03	1.0E-03		1.2E-06	2.6E-06	3.0E-06	1.9E-05	1.1E-05	2.1E-06	7.7E-05	2.2E-04
		CC	6.7E-04	3.2E-04			1.8E-06	3.0E-06	1.9E-05		1.8E-06	9.5E-05	6.7E-05
upper	17.5	BC	1.4E-03	2.4E-05	5.2E-07	1.5E-07	3.3E-07	7.5E-07	7.5E-07		1.8E-07	6.3E-06	4.5E-06
superfund		SB	1.6E-03	1.2E-04	1.1E-06	3.0E-07	3.3E-07	1.1E-06	1.1E-06	2.8E-06	1.8E-07	4.0E-06	7.6E-06
		CC	1.7E-04	7.5E-05	5.2E-07	1.5E-07	3.3E-07	7.5E-07	2.3E-06	1.4E-06	4.8E-07	8.0E-06	5.6E-06
	142.4	BC	1.1E-02	1.9E-04	4.2E-06	1.2E-06	2.6E-06	6.1E-06	6.1E-06		1.4E-06	5.1E-05	3.6E-05
		SB	1.3E-02	9.7E-04	8.6E-06	2.5E-06	2.6E-06	9.2E-06	9.3E-06	2.3E-05	1.4E-06	3.3E-05	6.2E-05
		CC	1.4E-03	5.8E-04	4.2E-06	1.2E-06	2.6E-06	6.1E-06	1.9E-05	1.1E-05	3.9E-06	6.5E-05	4.5E-05
Ross	17.5	BC	1.4E-03	2.2E-05			2.2E-07	7.5E-07	7.5E-07		1.8E-07	5.7E-06	6.2E-06
Island		SB	1.4E-03	4.6E-05	5.2E-07	1.5E-07	3.3E-07	3.7E-07	1.5E-06		1.8E-07	9.6E-06	7.3E-06

Table 3.6 Continued

site	IR[a] (g/day)	fish species	dioxin-like PCBs	non-dioxin like PCBs	α-BHC	β-BHC	γ-BHC	Hepata-chlor	heptachlor epoxide	aldrin	total chlordane	dieldrin	total DDTs
Ross Island	142.4	BC	1.1E-02	1.8E-04			1.8E-06	6.1E-06	6.1E-06		1.4E-06	4.7E-05	5.0E-05
		SB	1.1E-02	3.7E-04	4.2E-06	1.2E-06	2.6E-06	3.0E-06	1.2E-05		1.4E-06	7.8E-05	6.0E-05
Gladstone West Linn	17.5	BC	1.5E-03	1.9E-05			3.3E-07	3.7E-07	1.5E-06		1.8E-07	4.0E-06	4.6E-06
		SB	2.7E-03	1.5E-05	1.6E-06		3.3E-07	1.1E-06			1.8E-07	5.9E-06	3.4E-06
	142.4	BC	1.2E-02	1.6E-04			2.6E-06	3.0E-06	1.2E-05		1.4E-06	3.3E-05	3.7E-05
		SB	2.2E-02	1.2E-04	1.3E-05		2.6E-06	9.2E-06			1.4E-06	4.8E-05	2.8E-05
Reference site	17.5	BC	6.3E-05	1.6E-05				1.1E-06		4.3E-06	1.3E-07	4.0E-06	2.9E-06
		SB	4.5E-05	8.5E-06	3.9E-07	2.3E-07		5.6E-07	1.1E-06	2.1E-06	1.3E-07	4.0E-06	3.8E-06
	142.4	BC	5.1E-04	1.3E-04				9.2E-06		3.5E-05	1.1E-06	3.3E-05	2.3E-05
		SB	3.6E-04	6.9E-05	3.2E-06	1.8E-06		4.6E-06	9.3E-06	1.7E-05	1.1E-06	3.3E-05	3.1E-05

Values in shading show lifetime cancer risks > 10^{-5} (chemical of potential concern)
BC= black crappie
SB =smallmouth bass
CC = common carp
[a]Ingestion rate = 17.5 g/day for general population and recreational fisher and 142.4 g/day for subsistence fishers

Table 3.7 The overall cancer risk values at 30-year and 75 –year exposure duration

site	Target population	IR (g/day)	fish species	Overall cancer risk	
				30-year exposure	75-year exposure
lower superfund	general population and recreational fishers	17.5	black crappie	4.1E-05	1.1E-04
			smallmouth bass	2.5E-04	6.4E-04
			commom carp	5.6E-05	1.5E-04
	subsistence fishers	142.4	black crappie	3.3E-04	8.7E-04
			smallmouth bass	2.0E-03	5.2E-03
			commom carp	4.5E-04	1.2E-03
upper superfund	general population and recreational fishers	17.5	black crappie	5.4E-04	1.4E-03
			smallmouth bass	6.8E-04	1.8E-03
			commom carp	1.0E-04	2.7E-04
	subsistence fishers	142.4	black crappie	4.4E-03	1.1E-02
			smallmouth bass	5.5E-03	1.4E-02
			commom carp	8.3E-04	2.1E-03
Ross Island	general population and recreational fishers	17.5	black crappie	5.4E-04	1.4E-03
			smallmouth bass	5.6E-04	1.5E-03
	subsistence fishers	142.4	black crappie	4.4E-03	1.1E-02
			smallmouth bass	4.6E-03	1.2E-02
Gladstone/ WestLinn	general population and recreational fishers	17.5	black crappie	5.8E-04	1.5E-03
			smallmouth bass	1.0E-03	2.7E-03
	subsistence fishers	142.4	black crappie	4.7E-03	1.2E-02
			smallmouth bass	8.4E-03	2.2E-02
Reference Site	general population and recreational fishers	17.5	black crappie	3.5E-05	9.2E-05
			smallmouth bass	2.5E-05	6.6E-05
	subsistence fishers	142.4	black crappie	2.9E-04	7.5E-04
			smallmouth bass	2.1E-04	5.3E-04

Values in shading show lifetime cancer risks > 10^{-5}

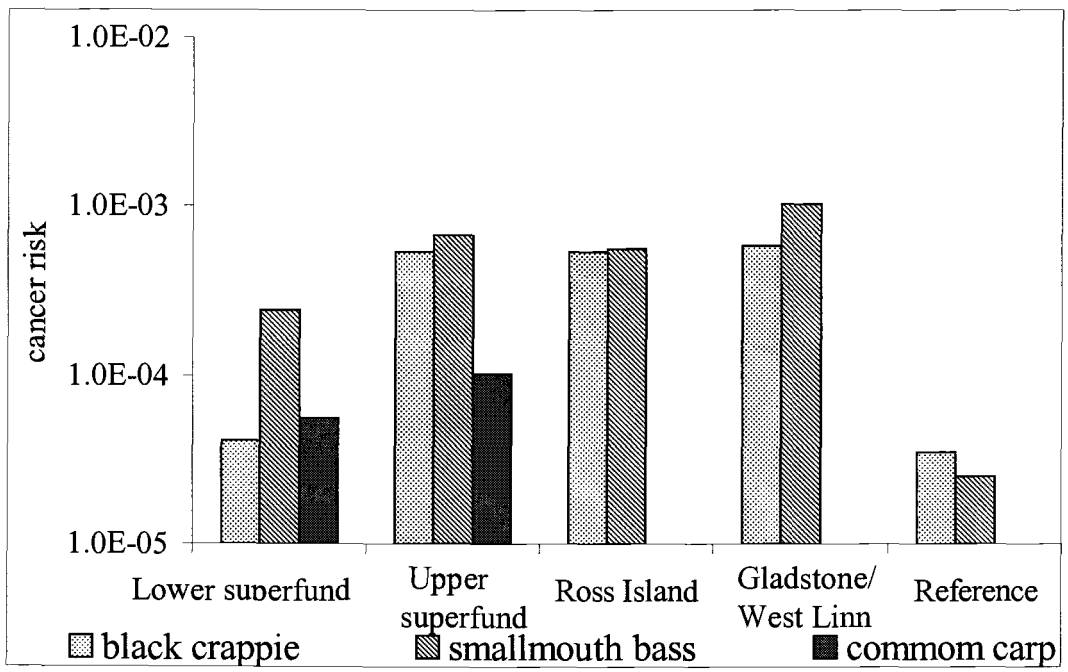


Figure 3.2 Estimated overall carcinogenic risk values caused by consuming PCBs and organochlorine pesticides contaminated fish collected from Portland Harbor superfund site and the Willamette River for general and recreational fishers (consumption rate = 17.5 g/day) at 30-year exposure duration assumption

Although average contaminant concentrations in fish at Gladstone/West Linn site were lower than the superfund site and Ross Island site, fish from this site posed relatively higher cancer risks. Among individual cancer risks of analyzed carcinogens, PCB-TEQs cancer risks contributed significantly to the relatively higher proportion of estimated total lifetime cancer risk, which subsequently directed risk estimates (Tables 3.5, 3.6 and Figure 3.3). The relative contribution of PCB-TEQs cancer risk to total cancer risk varied depending on site and fish species. PCB 126 and PCB 169 are the major contributors to PCB-TEQs with Toxic Equivalency Factor (TEFs) 0.1 and 0.01, respectively [17]. The presence of these two congeners in fish or any other environmental samples drives PCB-TEQs

values up significantly and subsequently significantly increases the potential cancer risk once they are multiplied by the 2,3,7,8-TCDD's slope factor (1.56×10^5). The greater the concentration of dioxin-like compounds, the higher the cancer risk level. The non ortho-coplanar PCBs (PCB 77, PCB 81, PCB 126 and PCB 169) are reported rarely in environmental samples but they are expected to be toxicologically most active based on the concept of co-planarity enhancing the potential toxicity [15]. These congeners, particularly PCB 126, pose a greater threat to human and wildlife than other PCBs. Harris et al [35] reported among coplanar PCBs tested, PCBs 126 was the most potent inducer of embryotoxicity and hepatic aryl hydrocarbon hydroxylase (AHH) in fish. In this study, PCB 126 was detected in most of the smallmouth bass and one black crappie at Gladstone/West Linn, and in one smallmouth bass and one black crappie at the upper superfund site and Ross Island site, but it was not detected in any fish from the lower superfund site and the reference site. The distributions of PCB 126 and other co-planar PCBs (see details in Chapter 2) were contrary to the distribution of total PCBs in fish among the sites in which total PCBs levels were reported highest at the superfund site and lower upriver. The distributions in the opposite direction of co-planar PCBs to the total PCBs were responsible for higher TCDD-toxic equivalent concentrations (TCDD-TEQs) and subsequently higher cancer risk calculation at Gladstone/West Linn and Ross Island.

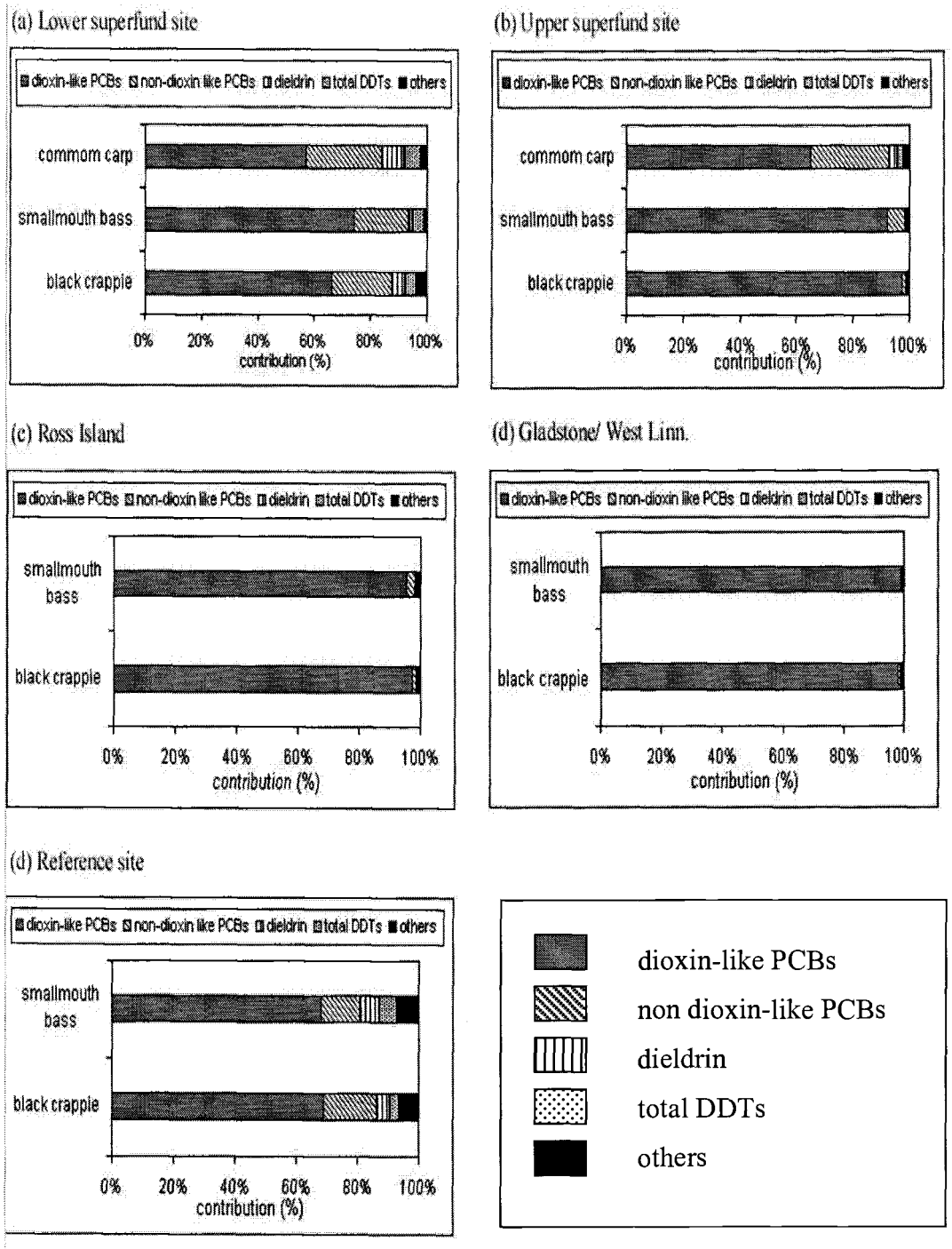


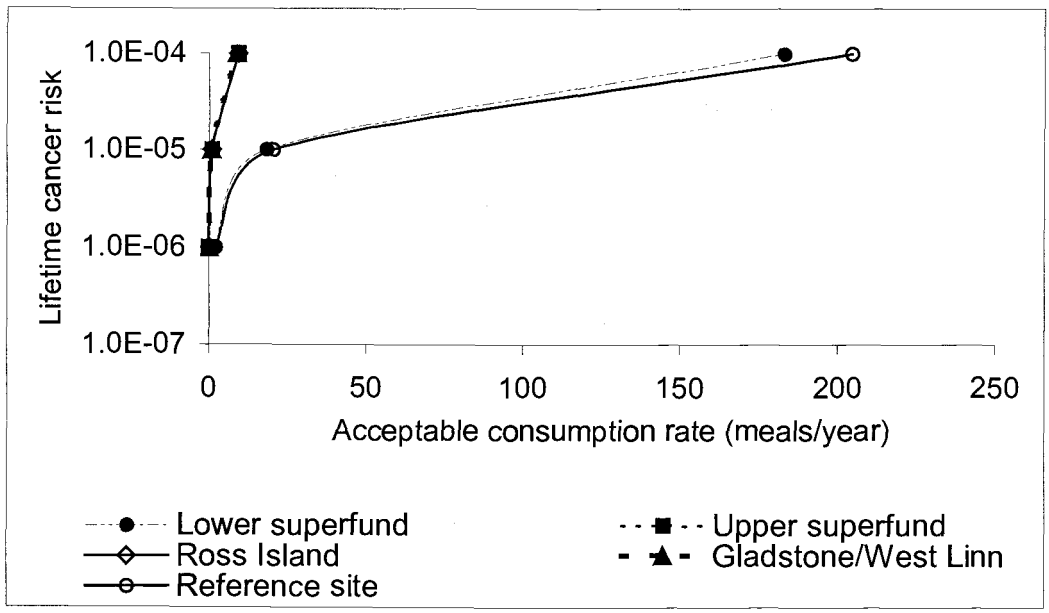
Figure 3.3 Percent contribution to total carcinogenic risk by various chemicals for three fish species collected from the Portland Harbor superfund site and the Willamette River during summer 2000

When all scenario assumptions were taken into consideration (Tables 3.5 and 3.6), PCBs (both dioxin-like and non dioxin-like PCBs) were the primary chemicals of potential concern in smallmouth bass at all study sites. Cancer risks for dioxin-like and non dioxin-like PCBs in this species exceeded the acceptable level (10^{-5}). As consumption rates and exposure duration increased, dieldrin, and total DDT (DDT and its derivatives) were added as chemicals of potential concern at most study sites. Dioxin-like PCBs were the primary chemicals of potential concern in black crappie, and non dioxin-like PCBs, DDT and its derivatives, and dieldrin were included as additional chemicals of potential concern when consumption rate or exposure duration increased. For the most extreme scenario (subsistence fisher and lifetime exposure duration) for smallmouth bass and black crappie, heptachlor epoxide and aldrin were added at the superfund site, α-BHC was added at Gladstone/West Linn, and aldrin was added as chemicals of concern at the reference site. Dioxin-like and non dioxin-like PCBs were primarily chemicals of potential concern in common carp at the superfund site. Additional chemicals of potential concern for this species included DDT and its derivatives, dieldrin, aldrin and heptachlor epoxide as consumption rate and exposure duration increased.

Because dioxin-like PCBs are the major contributor to overall cancer risk, allowable consumption rates without a potential cancer risk is calculated based on PCB-TEQs with an average of 129 g fish per 1 serving size [33]. Figures 3.4 and 3.4 illustrate the number of meals/year associated with various cancer risk levels for the 30-year and 75-year exposure durations, respectively. As shown in Figures 3.4 and 3.5, different assumptions (i.e. exposure duration) have a significant impact on the final risk estimates. This illustration provides a fish consumption limit for local fish consumers at various acceptable risk levels. For the 30-year exposure duration, a 10^{-5} dioxin-like PCBs cancer risk is estimated from the following number of meals/year for smallmouth bass: 3 (the lower superfund site) and 29 (the reference site), for black crappie: 18 (the lower superfund site) and 20 (the

reference site), and for common carp: 16 (the lower superfund site) and 7 (the upper superfund site). Neither smallmouth bass consumption nor black crappie consumption is acceptable at the upper superfund site, Ross Island site, and Gladstone/ West Linn site.

a) Black crappie



b) Smallmouth bass

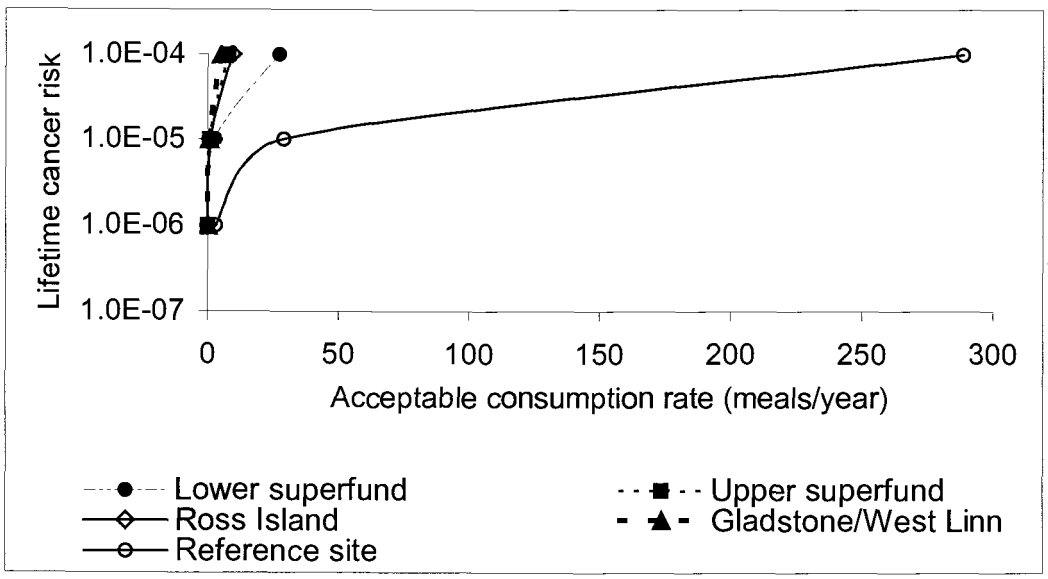


Figure 3.4 Total carcinogenic risk as a function of consumption rate for 30-year exposure duration a) in black crappie b) in smallmouth bass c) in common carp Consumption rate based on assumption of 129 g of fish per 1 serving size

c) Common carp

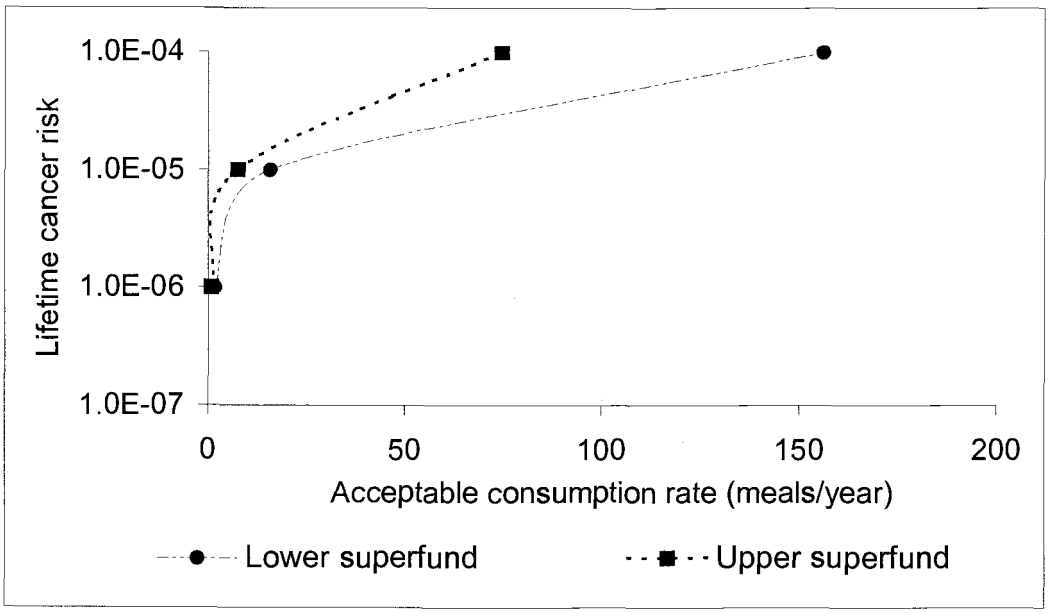
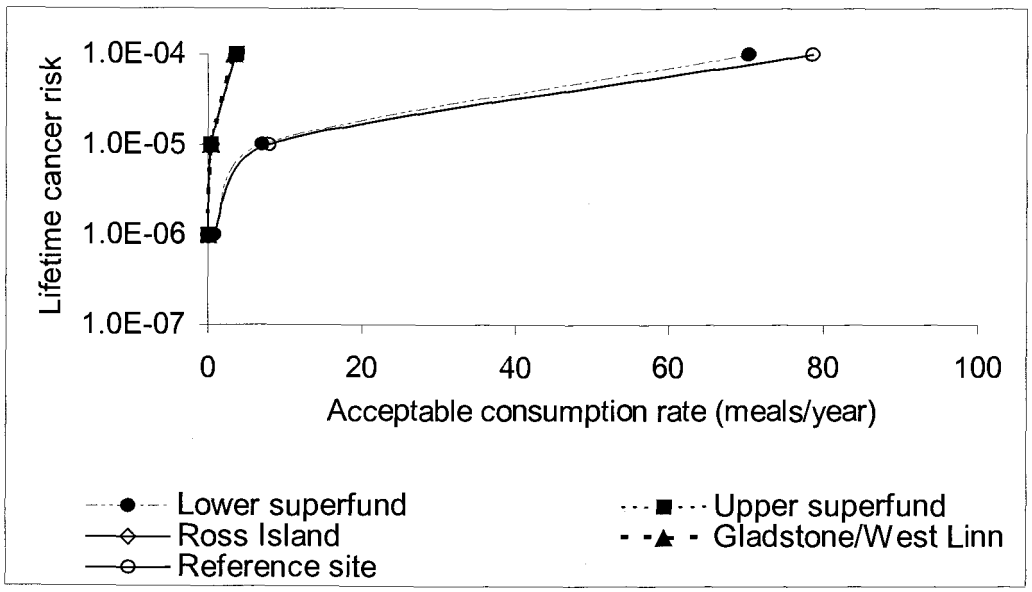


Figure 3.4 Continued

a) Black crappie



b) Smallmouth bass

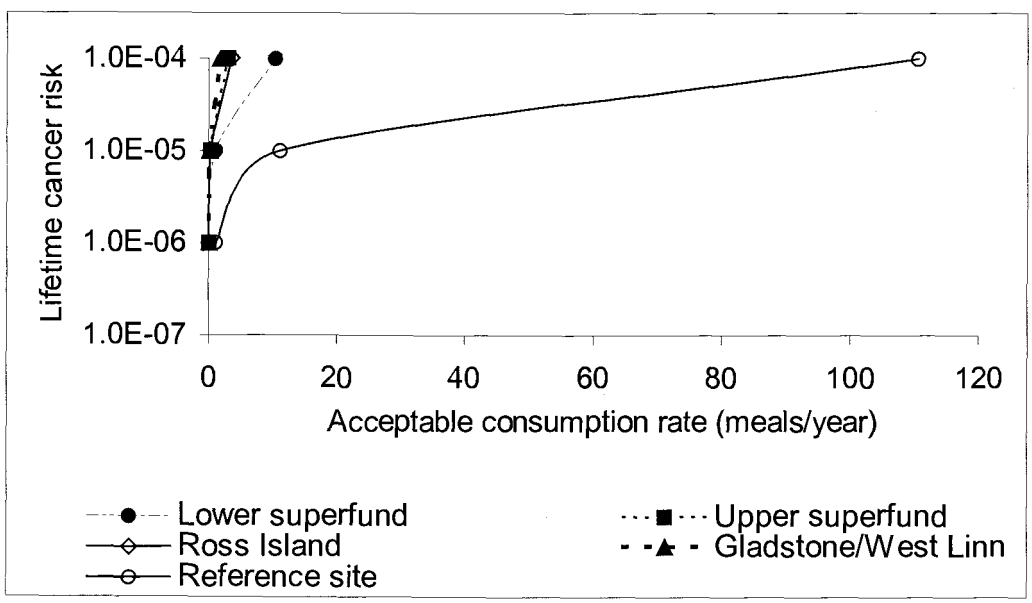


Figure 3.5 Total carcinogenic risk as a function of consumption rate for 75-year exposure duration a) in black crappie b) in smallmouth bass c) in common carp Consumption rate based on assumption of 129 g of fish per 1 serving size

c) Common carp

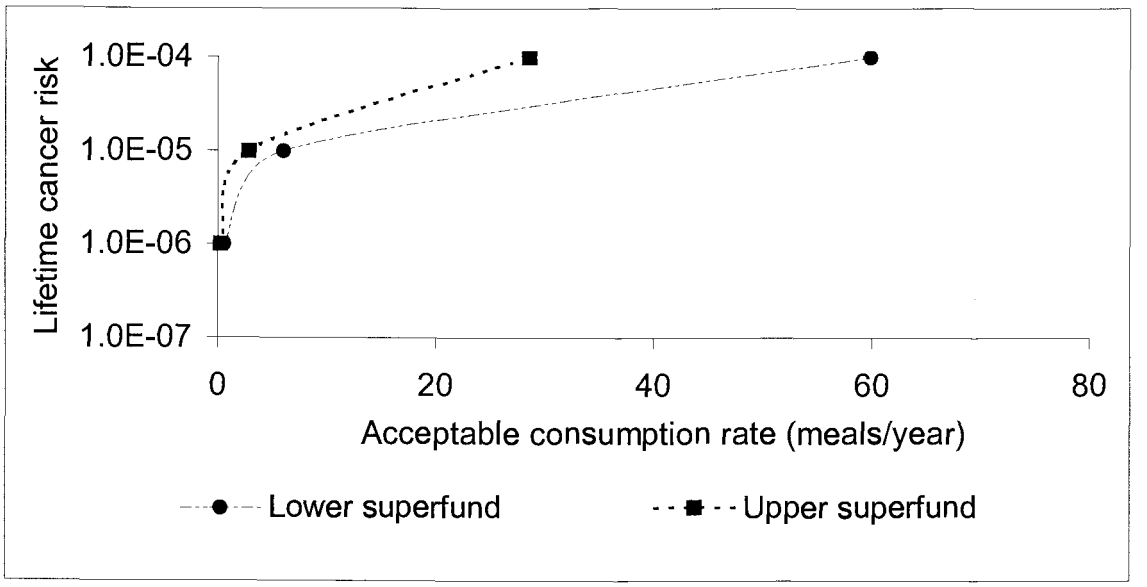


Figure 3.5 Continued

Non-carcinogenic risk

Non-carcinogenic risk is represent by the ratio of the average daily dose and the route- specific reference dose (RfD) which is referred to as "Hazard Quotient" [1]. At the level below the threshold dose (RfD), adverse health effects are unlikely even for sensitive population [1, 34]

$$HQ = \frac{CDI}{RfD} \qquad \text{equation (4)}$$

Where: HQ = Chemical specific Hazard Quotient (unitless)
CDI = Chemical specific chronic daily intake (mg/kg-day)
RfD = Chronic route-specific reference dose (mg/kg-day)

A hazard quotient less than 1 indicates that chemical exposure under the considered circumstances is unlikely to cause adverse health effect. If the hazard quotient is greater than 1, there is the potential for adverse health effects due to the chemical exposure. A hazard index is derived when simultaneous exposures to several substances are evaluated [1]. It is assumed that the magnitude of the adverse health effects will be proportional to the sum of the ratios of the sub-threshold exposures to acceptable exposures. Thus, hazard index (HI) is the summation of the hazard quotients. For multiple chemical exposures, the HI can exceed 1 even if no individual chemical exposure exceeds its RfD.

Tables 3.8 and 3.9 show the results of the estimated non-carcinogenic hazard indices and non-carcinogenic hazard quotient for detected chemicals in three fish species at all study sites. The potential for significant non-carcinogenic risk (HI > 1) for the general population and recreational fishers is indicated in each species as followed (Table 3.8); smallmouth bass at the lower superfund site (HI = 4.9), the

upper superfund site (HI = 3.8), Ross Island site (HI = 1.7) and Gladstone/West Linn (HI = 1.2); black crappie at the lower superfund site (HI = 1.1) and the reference site (HI = 1.8); common carp at the lower superfund site (HI = 1.4) and the upper superfund site (HI = 2.4). The hazard indices increase as the consumption rates increase (subsistence fishers).

Table 3.8 Estimated non-carcinogenic hazard indices for detected chemicals in each fish species

Site	Target population	IR (g/day)	Fish species	Hazard Index
lower superfund	general population and recreational fishers	17.5	black crappie smallmouth bass commom carp	1.1 4.9 1.4
	subsistence fishers	142.4	black crappie smallmouth bass commom carp	8.7 40.2 11.4
upper superfund	general population and recreational fishers	17.5	black crappie smallmouth bass commom carp	1.0 3.8 2.4
	subsistence fishers	142.4	black crappie smallmouth bass commom carp	8.4 30.7 19.5
Ross Island	general population and recreational fishers	17.5	black crappie smallmouth bass	1.0 1.7
	subsistence fishers	142.4	black crappie smallmouth bass	8.4 14.1
Gladstone/ West Linn	general population and recreational fishers	17.5	black crappie smallmouth bass	0.7 1.2
	subsistence fishers	142.4	black crappie smallmouth bass	5.3 9.9
Reference Site	general population and recreational fishers	17.5	black crappie smallmouth bass	1.8 0.9
	subsistence fishers	142.4	black crappie smallmouth bass	14.6 7.6

Values in shading show hazard quotient > 1

Table 3.9 Estimated non-carcinogenic hazard quotients for detected chemicals in each fish species

site	IR (g/day)	fish species	total PCBs	total DDT	dieldrin	endrin	heptachlor epoxide	γ-BHC	total chlodane	hepta-chlor	aldrin	methoxy -chlor	mercury
lower superfund	17.5	BC	6.8E-01	2.2E-02	5.8E-03		1.8E-02	8.0E-04	9.6E-04		2.6E-03		3.4E-01
		SB	3.9E+00	1.5E-01	1.1E-02	2.6E-04	1.8E-02	8.0E-04	1.4E-03	1.6E-04	2.6E-03	1.6E-05	8.9E-01
		CC	1.2E+00	4.7E-02	1.4E-02		1.8E-02	5.4E-04	1.2E-03	1.6E-04			1.7E-01
	142.4	BC	5.6E+00	1.8E-01	4.7E-02		1.5E-01	6.5E-03	7.8E-03		2.1E-02		2.7E+00
		SB	3.1E+01	1.2E+00	9.2E-02	2.1E-03	1.5E-01	6.5E-03	1.1E-02	1.3E-03	2.1E-02	1.3E-04	7.2E+00
		CC	9.4E+00	3.8E-01	1.1E-01		1.5E-01	4.4E-03	9.8E-03	1.3E-03			1.4E+00
upper superfund	17.5	BC	7.2E-01	2.5E-02	7.5E-03		6.1E-03	8.0E-04	9.6E-04	3.2E-04		3.2E-05	2.6E-01
		SB	3.4E+00	4.3E-02	4.8E-03	5.4E-04	9.2E-03	8.0E-04	9.6E-04	4.8E-04	5.4E-03	3.2E-05	2.9E-01
		CC	2.2E+00	3.1E-02	9.6E-03	2.6E-04	1.8E-02	8.0E-04	2.6E-03	3.2E-04	2.6E-03	1.6E-05	1.2E-01
	142.4	BC	5.9E+00	2.0E-01	6.1E-02		5.0E-02	6.5E-03	7.8E-03	2.6E-03		2.6E-04	2.1E+00
		SB	2.8E+01	3.5E-01	3.9E-02	4.4E-03	7.5E-02	6.5E-03	7.8E-03	3.9E-03	4.4E-02	2.6E-04	2.3E+00
		CC	1.8E+01	2.6E-01	7.8E-02	2.1E-03	1.5E-01	6.5E-03	2.1E-02	2.6E-03	2.1E-02	1.3E-04	9.8E-01
Ross Island	17.5	BC	7.2E-01	3.5E-02	6.9E-03		6.1E-03	5.4E-04	9.6E-04	3.2E-04			2.6E-01
		SB	1.3E+00	4.1E-02	1.2E-02		1.2E-02	8.0E-04	9.6E-04	1.6E-04			3.4E-01
	142.4	BC	5.8E+00	2.8E-01	5.6E-02		5.0E-02	4.4E-03	7.8E-03	2.6E-03			2.1E+00
		SM	1.1E+01	3.4E-01	9.4E-02		1.0E-01	6.5E-03	7.8E-03	1.3E-03			2.7E+00

Table 3.8 Continued

site	IR (g/day)	fish species	total PCBs	total DDT	dieldrin	endrin	heptachlor epoxide	γ-BHC	total chlodane	hepta-chlor	aldrin	methoxy-chlor	mercury
Gladstone WestLinn	17.5	BC	5.9E-01	2.6E-02	4.8E-03		1.2E-02	8.0E-04	9.6E-04	1.6E-04		0.0E+00	2.4E-02
		SB	4.6E-01	1.9E-02	7.0E-03			8.0E-04	9.6E-04	4.8E-04		3.2E-05	7.2E-01
	142.4	BC	4.8E+00	2.1E-01	3.9E-02		1.0E-01	6.5E-03	7.8E-03	1.3E-03		0.0E+00	2.0E-01
		SB	3.8E+00	1.6E-01	5.7E-02			6.5E-03	7.8E-03	3.9E-03		2.6E-04	5.9E+00
Reference site	17.5	BC	5.2E-01	1.6E-02	4.8E-03				7.2E-04	4.8E-04	8.0E-03		1.2E+00
		SB	2.7E-01	2.2E-02	4.8E-03	2.0E-04	9.2E-03		7.2E-04	2.4E-04	4.0E-03	2.4E-05	6.2E-01
	142.4	BC	4.2E+00	1.3E-01	3.9E-02				5.9E-03	3.9E-03	6.5E-02		1.0E+01
		SB	2.2E+00	1.8E-01	3.9E-02	1.6E-03	7.5E-02		5.9E-03	2.0E-03	3.3E-02	2.0E-04	5.1E+00

Values in shading show hazard quotient > 1 (chemical of potential concern)
BC= black crappie
SB =smallmouth bass
CC = common carp
[a]Ingestion rate = 17.5 g/day for general population and recreational fisher
Ingestion rate = 142.4 g/day for subsistence fishers

Figure 3.6 displays the relationship between hazard index and study sites for individual fish species by using the recreation fisher scenario. Overall, the hazard indices increased as the distance to the superfund site decreased and hazard indices for the subsistence fisher scenario elicited similar trends with increasing hazard index values as consumption rates increased. Considering all fish species at the superfund site (the lower and the upper superfund), the relative potential for non-carcinogenic risk, listed in decreasing order, is smallmouth bass, common carp and black crappie.

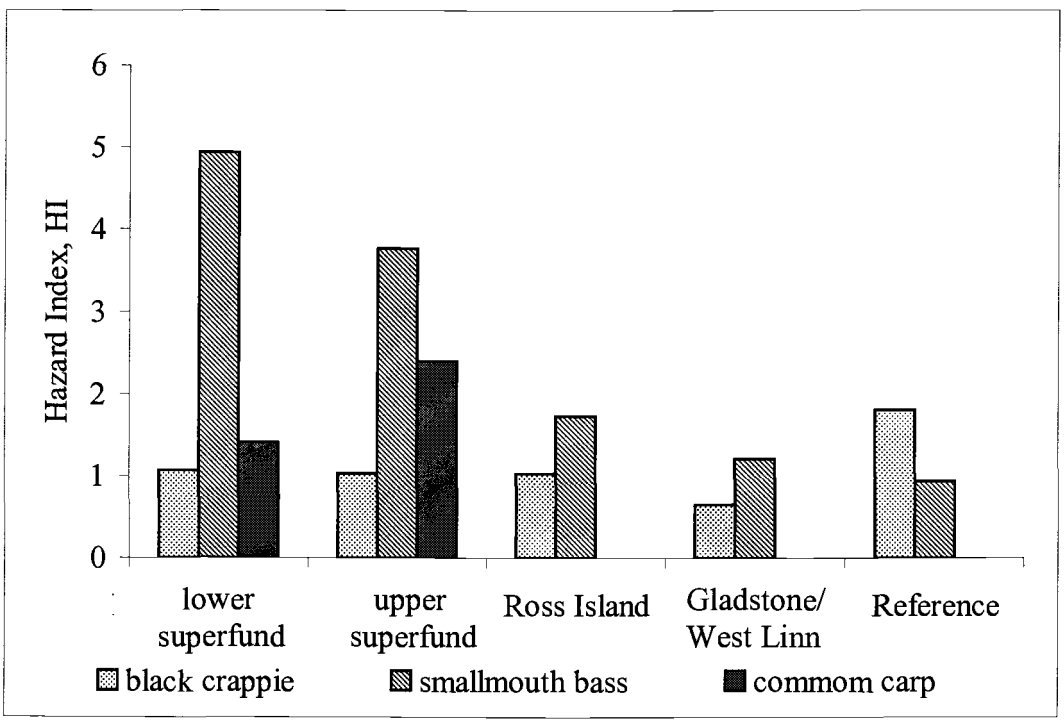


Figure 3.6 Hazard index estimates for general population and recreational fishers (consumption rate = 17.5 g/day)

Dose additive assumption (HI) is a simple appropriate approach at a screening level and most properly applied to compounds that induce the same effect by the same mechanism of action [1]. Application of hazard index approach to a number of compounds that are not expected to affect the same target organ or that do not act by the same mechanism could overestimate the potential for effects [34]. However, this possibility is generally not of concern if only one or two substances are responsible for driving the hazard index greater than 1[1]. In this study, PCBs are the significant contributor and drive hazard indices in fish at most sites, except in smallmouth bass at Gladstone/West Linn site and fish at the reference site where mercury is the major contributor to the hazard index (Figure 3.7). Considering the hazard index for neurotoxicity by mercury for recreational fishers (Table 3.9), no fish species at any site has a hazard quotient greater than 1, except for black crappie at the reference site. Therefore, neurotoxic effects do not seem to be the primary critical endpoint for non-carcinogenic effect due to consumption of fish from the lower Willamette River. Liver toxicity by PCBs and organochlorine pesticides appears to be the significant endpoint due to consumption of contaminated fish from the Portland Harbor superfund site. Hazard indices greater than 1 for hepatotoxic effect in each species are estimated as followed; smallmouth bass at the lower superfund site HI = 4.05, at the upper superfund site HI = 3.48, at Ross Island site HI = 1.39; common carp at the lower superfund site HI = 1.24, at the upper superfund site HI = 2.28.

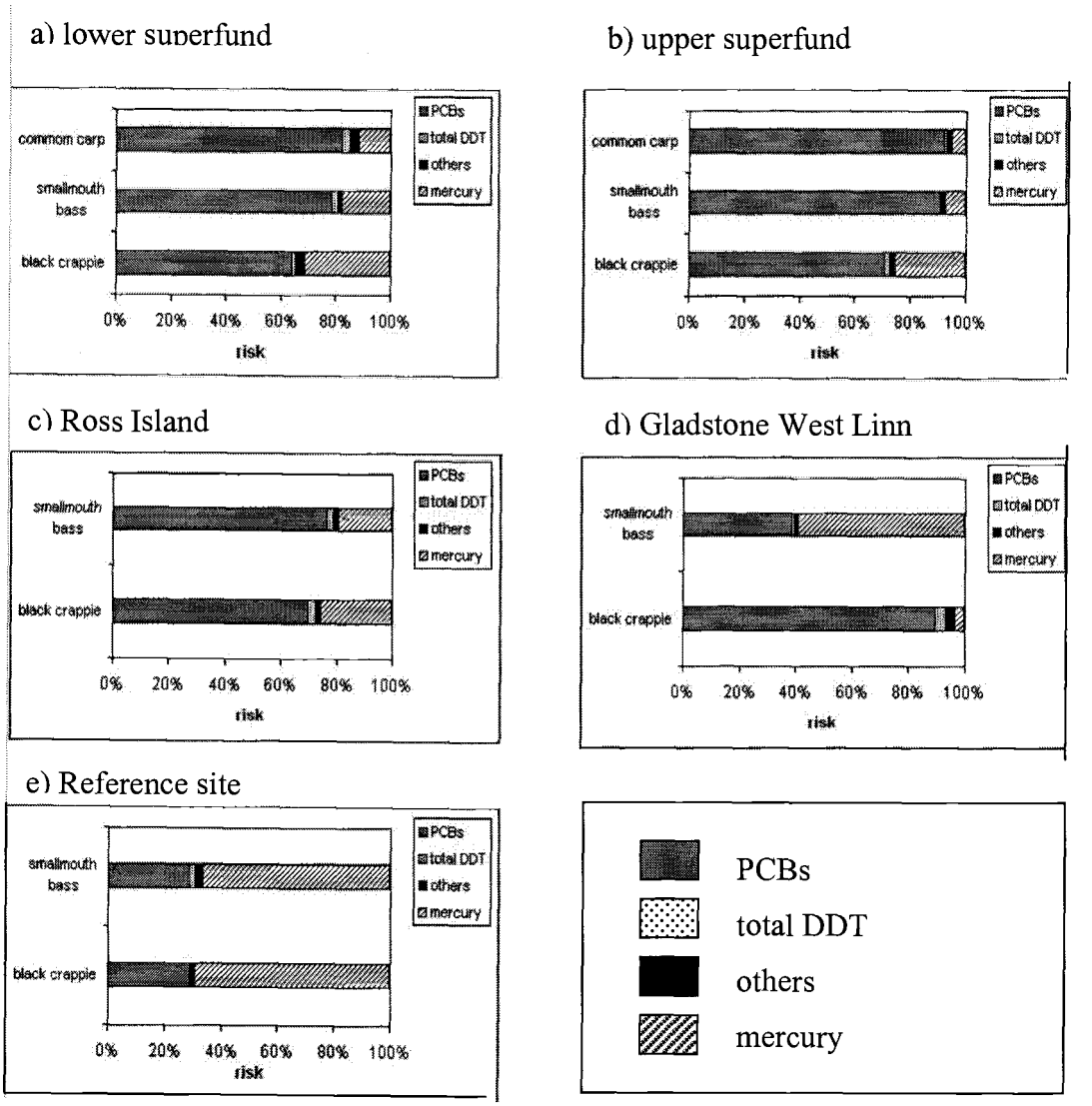


Figure 3.7 Percent contribution to hazard index by various chemicals for three fish species collected from the Portland Harbor superfund site and the Willamette River during summer 2000

As mentioned early, PCBs are responsible for the majority of the HI above 1 at the Portland Harbor superfund site. PCBs are primary chemicals of potential concern in smallmouth bass at the superfund site and Ross Island site and in common carp at the superfund site. As the consumption rate increases, DDT and its derivatives are included as additional chemicals of potential concern in smallmouth bass at the lower superfund site and mercury is added to all three fish species at the superfund site. At the reference site, mercury is the primary chemical of potential concern in black crappie and PCBs are additional concern chemicals as consumption rate increases. Although, the overall hazard indices in black crappie for recreation fisher consumer at the lower superfund site, the upper superfund site and Ross Island site exceeded 1, there are no chemical-specific hazard quotients that exceeded 1. Thus, there is no primary chemical of potential concern for black crappie at these sites.

While Figure 3.4 and 3.5 show estimates of allowable consumption rate at different acceptable risk levels, Table 3.10 shows estimates of acceptable consumption rate without non-carcinogenic effects by using different endpoints. Because PCBs are responsible for the majority of HI above 1 at most sites, estimates of acceptable consumption rate without non-carcinogenic effects are calculated based on total PCBs concentrations. Table 3.10 shows consumption rate of meals/year based on hazard quotient equal 1. Considering PCB-induced liver toxicity as endpoint, acceptable consumption rates increase as the distance from the superfund site increases. Eating fish less than these numbers, local fish consumers are assumed to be free from the potential risk of PCBs-induced liver toxicity. Mercury is another chemical of potential concern at the reference site. Local fish consumers are likely to suffer from neurotoxic effects if they eat large quantity of fish from this site repeatedly. Considering neurotoxic effects induced by mercury as the toxic end point, acceptable consumption rates are decreased to 40 meals/year for black crappie and 79 meals/year for smallmouth bass.

Table 3.10 Estimates of acceptable consumption rate (meals/year) without appreciable adverse effects induced by mercury and PCBs

Site	Fish species	Acceptable consumption rate (meals/year)[a]	
		Mercury	PCBs
lower superfund	black crappie	148	73
	smallmouth bass	56	13
	common carp	295	44
upper superfund	black crappie	188	69
	smallmouth bass	172	15
	common carp	413	23
Ross Island	black crappie	188	73
	smallmouth bass	148	37
Gladstone/ WestLinn	black crappie	2066	85
	smallmouth bass	69	107
Reference Site	black crappie	40	96
	smallmouth bass	79	184

[a]Consumption rate based on assumption of 129 g fish per 1 serving size [33]

UNCERTAINTY EVALUATION

Uncertainty can be introduced at various stages of the risk assessment process [27]. Uncertainty can arise as a consequence of the techniques used to sample and analyze chemical residues, chemical fate and transport factors, population variability, selection of exposure scenarios and assumptions, and the toxicity data used [33]. The resulting risks can either overestimate or underestimate risks and may or may not protect human health. Therefore, uncertainty analysis needs to be included in risk assessment because it is critical to the credibility of risk estimates. Risk managers will balance between risk characterization results and acceptable uncertainty and justify whether the sites need to be remediated or other action taken to protect human health.

Uncertainty can be inherent in the toxicity values (i.e., oral reference dose and slope factor). Although uncertainty factors and modifying factors are applied to the obtained values to account for the various types of uncertainty and variability inherent in estimating the toxicity values, the overestimation or underestimation of the potential risk are still possible. In addition, in the case of PCBs, the toxicity values are typically derived from laboratory studies based on commercial PCB mixtures that have congener composition different from environmental PCB mixtures due to environmental processes and biotransformation. This difference can also contribute to the uncertainty in risk assessment when PCBs are the target analytes.

Because most risk parameters are often unavailable, the exposure assumptions, (i.e. consumption rate and exposure duration) are generally based on U.S. EPA guidance. The potential for risk may be overestimated if an individual tends to live in a place less than the default exposure duration or consume fish less than the default consumption rate. However, the benefit of this assumption (reasonable maximum exposure, RME) is to protect the majority of the population and conservativeness.

Chemical concentrations in fish are one of the factors that contribute to the uncertainty. Contaminant concentrations detected in fish samples represent chemical residues in fish at a single sampling period, but these mean concentrations are assumed as the chemical concentrations for the entire 30-year exposure duration and lifetime exposure duration. In addition, if new fish samples are collected or if the monitoring study in fish is continued, the mean concentrations would likely be different. The new mean concentrations may be higher or lower than the values used in this study. Subsequently, the risk values presented here may overestimate or underestimate the risk obtained from future or multiple sampling periods. Additionally, banned chemicals such as organochlorine compounds may likely decline over time due to source control and environmental and biological degradation. With the decrease in contaminant concentrations, the potential for risk provided here may be overestimated. On the other hand, the risk estimates can also be underestimated if new inputs of contaminants are introduced to the site.

Assigning values to non-detected for chemical residues in human health food exposure assessment is also in question for risk assessment since no detection does not necessarily mean that the chemical is not present at any level but simply that the laboratory instrumentation or method cannot detect a residue at some level below the detection limit. Use of one-half of detection limit, the full detection limit, or true zero for these non-detected residues depend on different assumptions and risk assessor decisions [36]. The potential risk for carcinogenic and non-carcinogenic effect obtained from different assigned values for non-detected samples will differ. Using one-half of the detection limits can increase cancer risks estimated in this study up to seventy-fold (data not shown). Although, the basic concept of assigning one-half of the detection limit to non-detectable residues is to avoid underestimating exposure to highly exposed population but the possibility of overestimating of risk can occur and may cause misinterpretation, particularly, if the detection limit is high. Environmental data can be confidently used if the detection limits are not more than 20% of the concentrations of concern [20]. The

detection limit of 2 ng/g met the requirement of the usability of environmental data for risk assessments [20]

Processing and/or cooking effect is another factor that can affect the certainty of human health food exposure assessment. This risk assessment relied on the conservative assumption, that the whole fish instead of only fillets were examined and no cooking process was accounted for. It has been proposed that preparation and cooking methods have lowered the levels of PCBs and other organochlorine contaminants in certain fish species [37-39]. Zabik et al [38] demonstrated that removing the skin and lateral line in raw chinook salmon and carp as compared to the skin-on fillets with only the belly-flab removed reduced the level of organochlorine pesticides and total PCBs up to 50%. DDT derivatives, dieldrin, hexachlrobenzene, chlordane complex, heptachlor epoxide, and total PCBs were also significantly lower in cooked fillets than in the raw fillets by 30% to 41% [38]. Different cooking methods have different impacts on losses of PCB. Smoking and microwave baking can remove PCBs up to 65% [37, 39]. Therefore, it is likely that the potential exposure concentration of PCBs and organochlorine pesticides can be reduced if a fisherman skins off and cooks the fish. However, cooking effects on mercury residues were not in agreement [40-42]. Morgan et al [40] reported mercury concentrations in pan fried, baked and boiled portion of walleye and lake trout ranged from 1.1 to 2.0 times higher than in the corresponding raw portions, but total mercury levels were constant before and after cooking, indicating the concentration effect was caused by loss of moisture and fat. In contrast, some studies reported the effect of cooking was reduction of mercury residues in fish samples. D' Arrigo [41] indicated roasting produced a reduction up to 20% of mercury present in the fish samples and Anand [42] also reported losses of mercury in fried fish. Hence, consideration of preparing and cooking effect is important in assessing the health risk of eating contaminated fish.

Uncertainties are also associated with the additive approach. According to U.S. EPA guidance, risk from simultaneous exposures to multiple chemicals at the

superfund site is based on the additive assumption. The assumption of dose additive overlooks possible synergisms or antagonisms among chemicals and similarity in modes of action and metabolism are assumed. This assumption may cause subsequently over-or underestimated risk if there is any evidence of non-additive (antagonistic or synergistic interaction) of the chemicals of concern. Non-additive effects of PCB mixtures with themselves and with other environmental contaminants have been studied. PCB153, one of the major congeners in the environment, has been reported as an antagonist for some certain PCB congeners [43-45]. Zhao et al [43] investigated the inhibition of PCB 126-induced immunotoxicity and fetal cleft palate in mice by PCB 153. The ability of PCB 153 to inhibit PCB 126-induced embryotoxicity in chicken was also reported [44]. The other example of non-additive effect of PCBs is the study by Haag-Gronlund et al where weak antagonisms between PCB 126 and PCB 153 and between PCB 105 and PCB 153 in a rat liver tumor promotion bioassay were observed [45].

CONCLUSION

The non-carcinogenic risks and cancer risks from the presence of PCBs, organochlorine pesticides and mercury in fish from the lower stretch of the Willamette River were higher than the acceptable levels. Risks increased as consumption rate and exposure duration/frequency increased. This study indicated recreational and subsistence fishers may be threaten from eating fish from this segment of the river over the long term. However, avoiding eating fish from this area or processing and cooking fish before eating can significantly reduce the potential risks. All exposure parameters are critical in risk evaluation. Risk can be real or flawed depending on the accuracy and reliability of these parameters. Because the toxicity values are normally derived form the animal experimental model, uncertainty inherent in these values can cause risk over- or underestimated.

Fish consumption rate is another factor that affects on risk calculation. Risk will be reliable as long as consumption rate used in risk evaluation represents local fish consumer behavior. Chemical concentration is also the important factor that can affect on the reliability of risk evaluation. Because risk evaluation is based on concentrations of chemical detected in fish or in other environmental samples, assessment the useability of environmental data should be taken into account before conducting risk assessment. Monitoring study of fish and updating information of fish consumer behavior are needed to update fish advisories and to protect local fish consumer from health risks from eating contaminated fish.

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CHAPTER 4

CONCLUSIONS

The results presented in this study show the average background levels of organochlorine compounds in fish from the lower 20-mile stretch on the Willamette River are of concern to water quality and human health. Smallmouth bass are the most contaminated of three fish species analyzed in this study. The averages of PCbs and DDT derivatives in fish tissue are highest at the Portland Harbor superfund site and decline with increasing distance from the superfund site. The marked increase of organochlorine compounds reflects poor water quality at the lower stretch of the river. The marked increase of chemical contaminants also indicates that this river segment is the primary depositional area of the Willamette River system. This river stretch is significantly impacted by historical and current industrial activities and urban runoff, as well as, other non point sources such as urban and agricultural land uses. The p,p'-DDE, persistent metabolite of DDT, is the most abundant among the fifteen organochlorine pesticides tested. Deposit residues from the former DDT manufacturing plant appear to be the dominant source of total DDTs at the superfund site. A lower background level of total DDTs is present throughout the study area due to general non-point urban runoff and/or general agricultural runoff at the watershed level. Other agricultural organochlorine pesticides tested are not detected or present below detection limit (typically 2 ng/g fish sample). Only one tested fish contains mercury higher than the U.S. EPA's screening value for recreational fishers.

PCB congener profiles in fish from each site of study are similar and dominated by high chlorine content congeners. Hexachlorobiphenyl congener 153 is the most abundant congener, followed by pentachlorobiphenyl congener 118 and

heptachlorobiphenyl congener 180. Interestingly, the PCB profile at the reference site is slightly different from the other sites. The ratios of trichlorobiphenyls and tetrachlorobiphenyls to the recalcitrant congener PCB 153 are considerably higher in fish from the referent site. Source of input (original PCB mixtures) and age of release could contribute to the difference of PCB profiles at the reference site. The difference of PCB profiles in the environmental samples substantiates the appropriateness of congener-specific analysis. Congener-specific PCB analysis is more appropriate than commercial mixtures (Aroclor®)-based analysis because PCB composition in the environment differs from the commercial mixtures due to weathering, chemical transformation and preferential bioaccumulation.

This study also presents the potential health risk of chemical contaminants in fish to the local fish consumers and suggests initial advisory for local fish consumers to reduce risk. The assessments for non-carcinogenic risks and cancer risks indicate recreational fishers and subsistence fishers may be threaten from eating fish from this area. Hazard quotient indices greater than 1 indicated the adverse health effects associated with chemical contaminants in fish are likely. Total PCBs are the major contributor to overall non-carcinogenic risk with the hazard quotients above 1 at most sites, except at the reference site where mercury is the main contributor. The overall cancer risks at all sites exceed acceptable lifetime cancer risk level (10^{-5}) and dioxin-like congeners are the major contributor to the cancer risks. The excess lifetime cancer risks in fish at Gladstone/West Linn are higher than risks at the superfund site although fish at the superfund site has higher total PCB levels. The presence of non ortho-coplanar PCBs (PCB 126, 169) in fish at Gladstone/West Linn drives PCB-TEQs values up significantly and subsequently significantly increases the potential cancer risk. Therefore, the presence of non ortho-coplanar PCBs, especially PCB 126, although at the low level is critical for PCB risk assessment. The analytical improvement to decrease detection limit of PCB 126 and PCB 169 is needed to reduce uncertainty associated with their presence or absence in fish tissue. Therefore, the sensitivity of detection limit is

desired and the evaluation of environmental data before conducting risk assessment is recommended.

The present study may serve as a base for fortune study on monitoring occurrence and distribution of environmental contaminants at the Willamette River. Monitoring of fish tissue contaminant concentration need to be continue to protect local fish consumer health and to assess surrounding sediment and water quality. In addition chemical analyses in other environmental samples such as water, sediment or other aquatic biota are needed to better understand fate, behavior process and environmental impacts on PCB and organochlorine pesticides in the Willamette Basin system. However, their residues in non-biotic samples such as water and sediment may not be relevant to health risk investigation because some of them are not in bioavailable forms which are potentially uptake by aquatic organisms. Furthermore, aquatic organisms, fish for instance, are sometimes difficult to collect resulting in limitation in number of samples. Thus, the development of other sampling devices such as passive sampling device, which are mimic the membrane of organisms and selectively sample bioavailable chemical contaminants, are needed.

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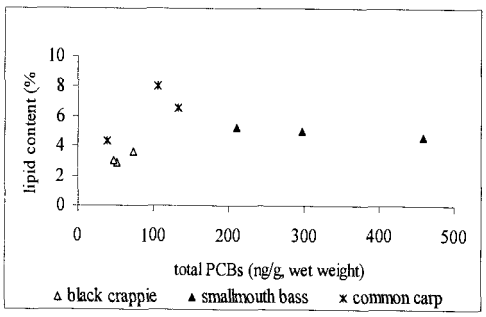
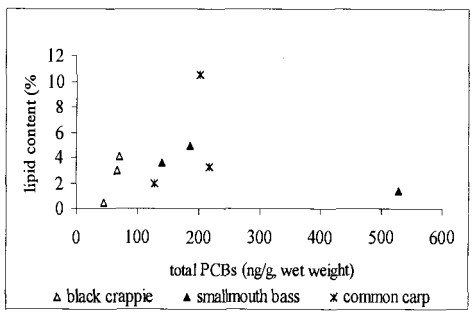
APPENDICES

Appendix A Scatter plots between lipid content and total PCB concentration

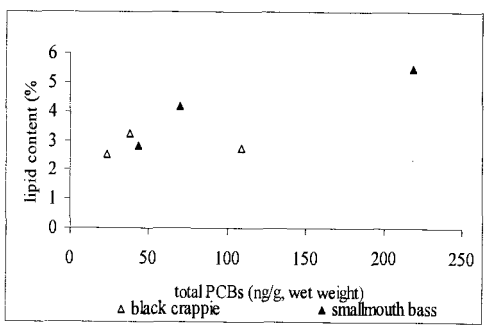
a) lower superfund site



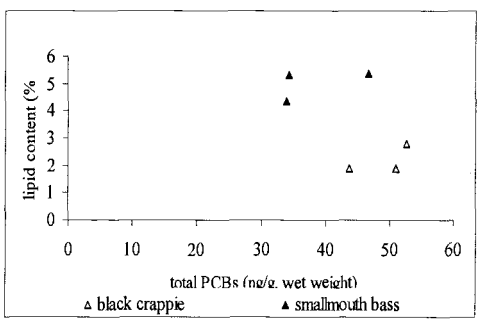
b) upper superfund site



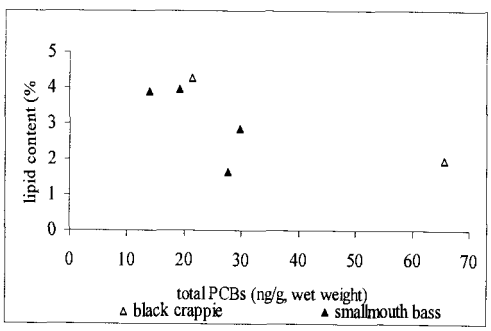
c) Ross Island



d) Gladstone/ West Linn



e) Reference site



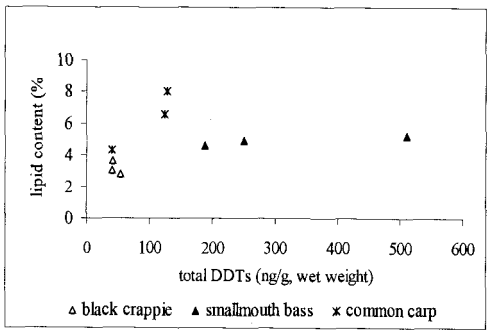
△ black crappie

▲ smallmouth bass

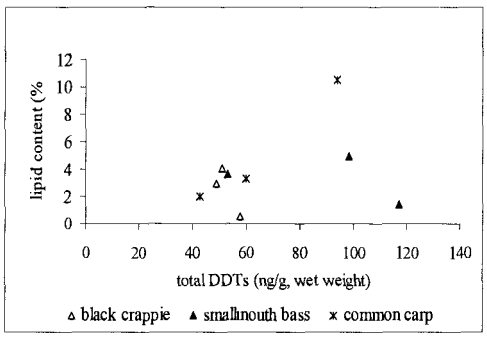
* common carp

Appendix B Scatter plots between lipid content and total DDT concentration (sum of p,p'-DDT, p,p'-DDD and p,p'-DDE)

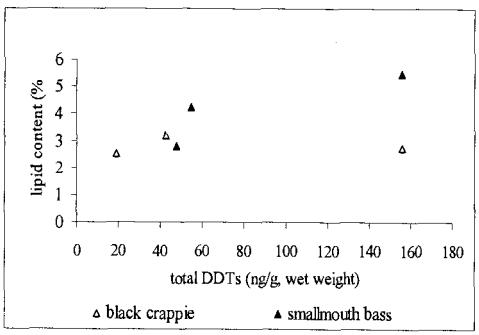
a) lower superfund site



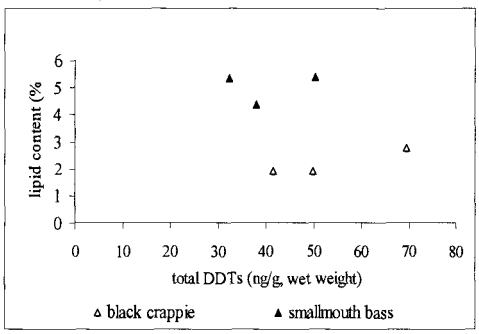
b) upper superfund site



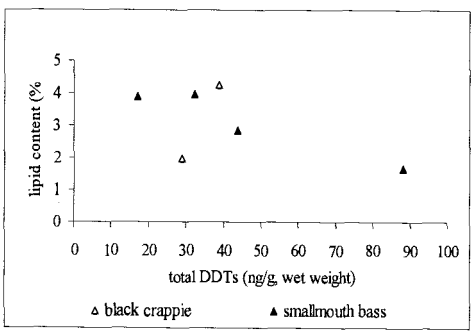
c) Ross Island



d) Gladstone/ West Linn



e) Reference site



△ black crappie

▲ smallmouth bass

* common carp