King County
Combined Sewer Overflow
Water Quality Assessment for the
Duwamish River and Elliott Bay

Appendix B: Methods and Results
B2: Human Health Risk Assessment

Prepared by the

Duwamish River and Elliott Bay

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LIST OF ACRONYMS

ATSDR	Agency for Toxic Substances and Disease Registry
CDI	Chronic daily intake
COPC	Constituent of potential concern
CSO	Combined sewer overflow
EEC	Estimated exposure concentrations
EED	Expected environmental dose
HEAST	Health Effects Summary Tables
HI	Hazard index
HQ	Hazard quotient
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
LADI	Lifetime average daily intake
LOAEL	Lowest observed adverse effect level
mpn	Most probable number
N/AV	Not available
NOAA	National Oceanic and Atmospheric Administration
NOAEL	No observed adverse effect level
OSWER	Office of Solid Waste and Emergency Response
PAH	Polycyclic aromatic hydrocarbon
PCB	Polychlorinated biphenyl
pfu	Plaque forming unit
RDI	Recommended daily intake
RfD	Reference dose
RTECS	Registry of Toxic Effects of Chemical Substances
SEAMS	Superfund Exposure Assessment Manual
TBT	Tributyltin
TDI	Tolerable daily intake
TERA	Toxicology Excellence for Risk Assessment
TRV	Toxicity reference value
UCL	Upper confidence limit
UF	Uncertainty factor
U.S. EPA	U.S. Environmental Protection Agency
USFWS	U.S. Fish and Wildlife Service
WDFW	Washington Department of Fish and Wildlife
WDOH	Washington State Division of Health
WERF	Water Environment Research Foundation
WHO	World Health Organization
WQA	Water Quality Assessment

1. INTRODUCTION

This appendix of the Combined Sewer Overflow Water Quality Assessment for the Duwamish River and Elliott Bay presents the methods and results of the human health risk assessment. People working and recreating on the Duwamish River and Elliott Bay can be exposed to different chemicals and pathogens through a variety of exposure pathways, which are summarized in the conceptual site model presented in Appendix A - *Problem Formulation, Analysis Plan, and Field Sampling Work Plan*.

As described in the problem formulation, a wide variety of chemicals and pathogens are present in the water and sediment of the Duwamish River and Elliott Bay. This project was designed to assess potential risks to aquatic life, wildlife, and human health from exposures to these chemicals and pathogens in the river and bay. It was also designed to assess the improvements that would be observed with removal of King County's 12 combined sewer overflows (CSOs) that discharge into the river and bay. The methods and results of the wildlife risk and aquatic life risk assessments are presented in Appendices B3 and B4, respectively.

The human health risk assessment consisted of three general components.

1. A site-specific summer survey of people collecting seafood from the shores of the river and bay was conducted. This survey was designed to allow for an accurate estimation of the number of people collecting and consuming seafood from the river and bay. This information was used to estimate exposures to chemicals that accumulate in seafood from the water and sediment.

2. A human health chemical risk assessment was conducted. This assessment studied the potential for health risks resulting from exposures to chemicals in the water, sediment, and seafood in the river and bay. Health risks were estimated for people that engage in recreational activities resulting in direct exposures to the water and sediment (such as swimming or SCUBA diving, among others), and for people that consume seafood collected from the river and bay.

3. A human health pathogen risk assessment was conducted. This assessment studied the potential for illness and infection resulting from exposure to pathogens in the water and shellfish of the river and bay.

The methods and results of each of these components of the human health risk assessment are presented in the following sections.

2. METHODS AND RESULTS OF THE DUWAMISH RIVER AND ELLIOTT BAY FISHING SURVEY

The Duwamish River/Elliott Bay Water Quality Assessment (WQA) was conducted to assess the significance of potential ecological and human health risks from chemicals, physical stresses, and pathogens in the river and bay, and the relative contribution of CSOs to these potential risks. As part of this assessment, we conducted a fishing survey along the shores of the Duwamish River and Elliott Bay on 30 days during the summer of 1997. The survey was designed to determine the amount and types of seafood collected and consumed from the river and bay, as well as site-specific low, medium, and high seafood consumption rates for use in the human health risk assessment. Questions regarding each person's seafood collection, consumption frequencies and habits were asked, and seafood already collected were identified, weighed and measured. A total of 1,183 people were approached, many on more than one day, resulting in 1,947 survey attempts with an overall success rate of about 80 percent. The results indicate many people frequently collect and consume a wide variety of seafood from the river and bay. Consumption rates derived from these data were compared to those previously published for Puget Sound and other regions of the United States.

The Duwamish River and Elliott Bay are highly developed urban waterbodies within the city of Seattle that still sustain large recreational fisheries (NOAA 1987, WDOH 1985). Many steps have already been taken to remediate chemically contaminated sediments and reduce the release of toxic chemicals in the area. However, chemical contamination in seafood collected from the Duwamish River and Elliott Bay continues to be observed. The presence of potentially toxic chemicals in seafood from the Duwamish River and Elliott Bay raises concern about the level of risk to recreational anglers posed by eating chemically contaminated seafood collected in this area. These individual risk levels can be estimated using standard risk assessment techniques.

To estimate chemical exposures from seafood consumption both the chemical concentrations in seafood and the amount and type of seafood consumed must be estimated (U.S. EPA 1989a). A comprehensive sampling and analysis program was implemented by King County to obtain chemical concentration data in the tissues of salmon, rockfish, English sole, mussels, shiner perch, Dungeness crab, prawns, and squid. A seafood collection and consumption survey was also conducted to estimate the types and amount of different seafood collected and consumed from the river and bay.

Several studies have been conducted that examined seafood collection and consumption in Puget Sound (NOAA 1987, WDOH 1985, Toy et al. 1996, Pierce et al. 1981). These studies suggest that many people continue to collect seafood from Puget Sound, both from the shore and from boats. Two of these studies included surveys of fishers in Elliott Bay (NOAA 1987, WDOH 1985).

The survey conducted by King County was designed to supplement the information collected from Elliott Bay during the mid-1980s. King County surveyed individuals collecting seafood from the shores of the river and the bay. Boaters were not interviewed

because limited boat fishing was allowed within the bay during the survey, especially for salmon. The survey was designed to provide data from which we could calculate seafood collection and consumption rates. These data will be used in the risk assessment to assess whether there are risks to people from consuming seafood from the Duwamish River and Elliott Bay, and the fraction of the risks attributable to CSO discharges.

2.1 Methods

Seafood collection and consumption was estimated for people that collected seafood from the shores of the Duwamish River and Elliott Bay. Both resident and nonresident anglers were surveyed, although it is believed that non-resident anglers will collect and consume seafood from the area less frequently than resident anglers. No effort was made to identify whether the angler possessed the proper license, or was otherwise illegally collecting seafood.

Surveyors were trained on filling out the forms and approaching potential respondents. Surveyors wore no badges, caps, or other items that identified them as county employees. Surveyors worked in teams of two, and approached every individual they observed collecting seafood within the study area. The survey form was translated into three languages (Vietnamese, Korean, and Filipino) to allow for persons uncomfortable with English to participate in the survey.

Locations where seafood collection could potentially occur were identified during a pre-survey site reconnaissance. These access sites were used as survey locations. During the survey, each of these identified access sites were visited at least twice (AM and PM) each survey day.

Surveys were conducted on 30 days during a 10-week period beginning Sunday, June 22, 1997, and ending Saturday, August 30, 1997. Surveys were conducted every Saturday and Sunday (10 days each), and on 10 weekdays. Weekend days were emphasized because the reconnaissance and results of other surveys (e.g., NOAA 1987) indicated that a substantially larger number of people collected seafood on weekends.

Each survey day was divided into two shifts. On weekends the first shift began at 5 AM and lasted until 1 PM and the second shift began at 12 PM and lasted until 8 PM. On weekdays the first shift went from 5 to 11 AM, and the second shift lasted from 4 to 10 PM. Each shift visited every access point at least once. In an attempt to obtain more complete results, access points with the heaviest activity (i.e., Seacrest Park, Elliott Bay Pier, and Harbor Island) were often visited more than once during a shift.

2.2 Survey Design

The design of the survey focused on asking anglers the types of seafood they collected and consumed from the study area, and how frequently they did so. The survey consisted of a three-page questionnaire filled in by the surveyor. When allowed, surveyors also identified, measured and weighed any organisms already collected.

Each respondent was asked whether they had previously participated in the survey, and whether they were willing to participate in the survey that day. Even when an angler declined to participate, some information was often gathered. Each angler was also asked to report their age, sex and ethnicity.

To provide data on consumption rates, each angler was asked how frequently they collected and consumed seafood from the survey location each month of the year. Recall questions on the type and quantity of seafood collected and consumed during the past week were also asked. For any organisms collected the day of the survey, their plans for use were investigated. If the angler anticipated consuming the organism, the number of people with which they would share it was asked, as was their anticipated preparation method, and whether anybody sharing in its consumption would be under 10 years old.

2.3 Fishing Survey Results

A total of 1,947 interviews were attempted during the survey. Many people were approached more than once. Fewer than 1,183 different individuals were approached, with the rest of the interviews being repeat contacts[1]. About 81 percent of the different individuals agreed to be interviewed on the first time they were contacted, while 19 percent of the 764 repeat contacts agreed to be interviewed. This resulted in an overall success rate of about 56 percent. However, the surveyors were often able to gather information for many questions even when the person declined to be interviewed. There were also instances when some questions were not answered, even after the person agreed to be interviewed. Interview status responses are summarized in Table 2-1.

Repeat interviews were more successful when the interviewer was female (34 percent success) versus male (5 percent success). No obvious differences in success rates were observed between male and female interviewers on initial contacts. With only 92 of the 1,947 survey responses indicating a communication problem, the use of English-speaking interviewers did not appear to limit our ability to adequately conduct the survey. The majority of the interview attempts took place between either 5 to 10 AM (700 attempts) or 4 to 8 PM (645 attempts).

2.3.1 Survey Locations

A total of 24 survey locations were identified during the initial reconnaissance. Of these, three locations shown on Figure 2-1 (Seacrest Park in West Seattle, Elliott Bay Pier at the northwest end of Myrtle Edwards Park, and Harbor Island) accounted for 92 percent of

[1] Different individuals were assumed to be equal to the sum of the following interview status categories: agree, decline, language barrier, agree with language barrier, and decline with language barrier.

the interview attempts (1,792 out of 1,947 surveys). Fewer than 35 people were interviewed at each of the remaining sites (Table 2-2).

Table 2-1. Interview Status of 1,947 Fishing Surveys from the Elliott Bay and Duwamish River

Interview Status	Number of Respondents	Percent of Respondents (%)
Agree	925	47.5
Decline	179	9.2
Language Barrier	18	0.9
Repeat Contact	4	0.2
Agree, Repeat Contact	126	6.5
Decline, Repeat Contact	618	31.7
Decline, Language Barrier	27	1.4
Agree, Language Barrier	34	1.8
Decline, Language Barrier, Repeat Contact	7	0.4
Agree, Language Barrier, Repeat Contact	6	0.3
No Response for Question	3	0.2
Total	1,947	100

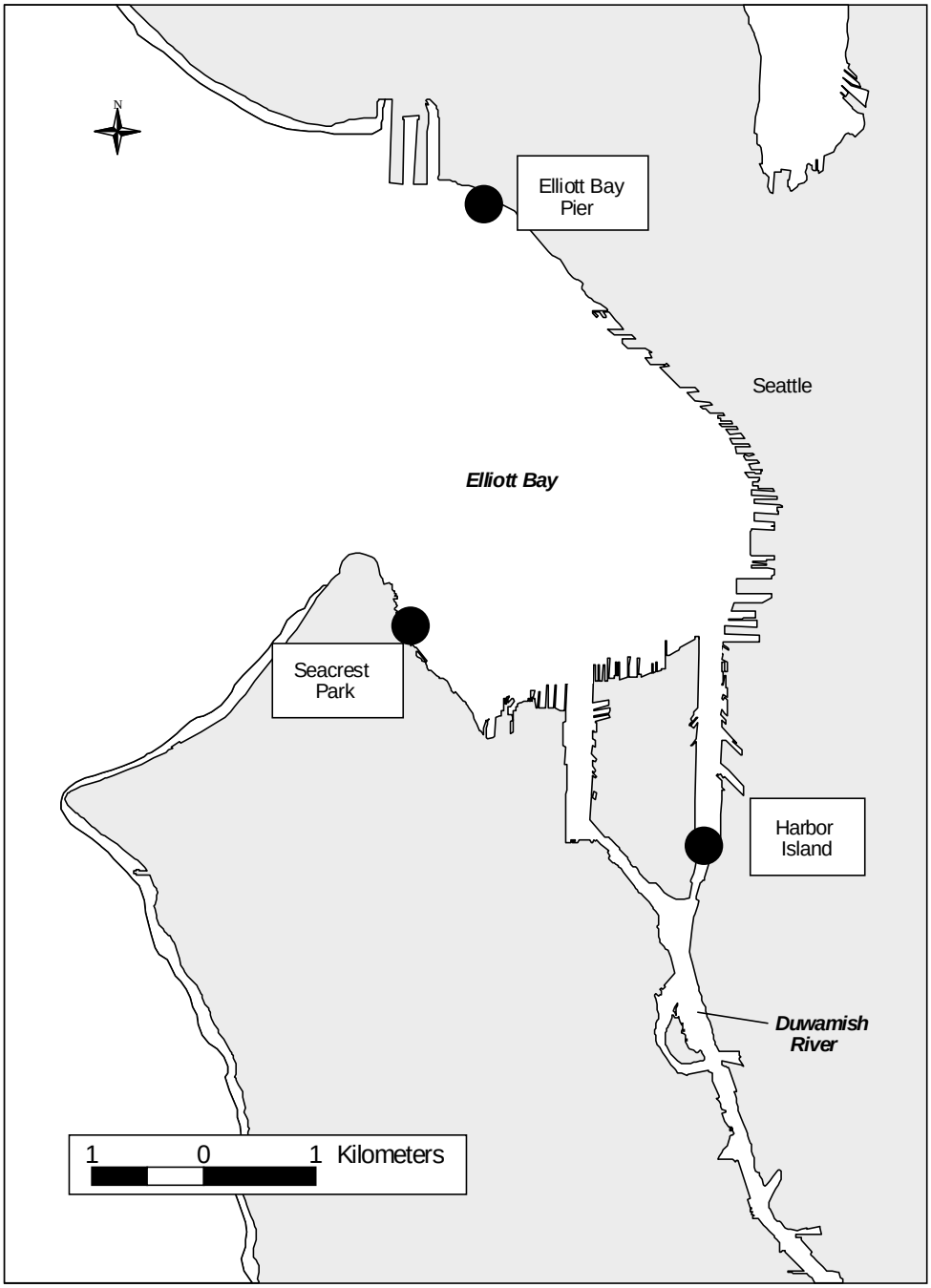

Figure 2-1 The Three Most Popular Seafood Collection Sites Along the Shores of the Duwamish River and Elliott Bay in Seattle, Washington

Table 2-2. Number of Surveys Conducted at Each Survey Location

Location	Number	Percent (%)
Boeing Employee Credit Union	12	0.62
Boeing Parking Lot	2	0.10
Diagonal Ave.	2	0.10
Don Armeni	34	1.75
Duwamish Waterway Park	1	0.05
Elliott Bay (boat)	3	0.15
Elliott Bay Pier	460	23.63
Fisher Mills	2	0.10
Harbor Island	192	9.86
Jack Perry	2	0.10
Myrtle Edwards Park	8	0.41
Seacrest	1,140	58.55
Seattle Waterfront	0	0
Shipwreck	29	1.49
Smith Cove	3	0.15
South 115th	1	0.05
Terminal 105	10	0.51
The Footbridge	9	0.46
The Rapids	16	0.82
Washington St. Public Landing	17	0.87
No Response for question	4	0.21
Total	1,947	100.00

2.3.2 Age and Sex

The majority of the people surveyed were male (85 percent) and either 15 to 30 years old (35 percent) or 30 to 50 years old (43 percent). Smaller percentages of people surveyed were less than 15 years old (7.7 percent) or over 50 years old (11 percent).

2.3.3 Ethnic Background

The majority of the respondents were Caucasian (41 percent), followed by African American (11 percent), Filipino (7.8 percent), Japanese (6 percent), Vietnamese (5.8 percent, and Chinese (4 percent). A wide variety of other ethnicities were also reported, as shown in Table 2-3.

Table 2-3. Ethnicity of Survey Respondents

Ethnicity	Number of people	Percent of people
Caucasian	488	41.3%
African American	128	10.8%
Filipino	92	7.8%
Japanese	71	6.0%
Vietnamese	69	5.8%
Chinese	47	4.0%
Latino(self described as: Spanish, Mexican, Chicano, Puerto Rican, Hispanic, Portuguese)	47	4.0%
Cambodian	33	2.8%
Native American (self-described as: Eskimo Native, Alaskan Native American)	33	2.8%
Laotian	21	1.8%
Korean	20	1.7%
Hawaiian	12	1.0%
Pacific Islander (self-described as: Samoan, Guam, Polynesian, New Guinean)	10	0.8%
Middle Eastern (self described as: Middle Eastern, Cypriot, Turkish, Lebanese)	5	0.4%
Thai	5	0.4%
Eastern European (self-described as: Serbian, Yugoslavian, Hungarian)	4	0.3%
Other	27	2.3%
Blank	71	6.0%
TOTAL	1,183	100%

2.3.4 Time Spent Collecting Seafood

The lengths of time that the people had been collecting seafood when the surveys began were indicated on 1,093 of the 1,947 survey forms and are summarized in Table 2-4. The majority of the people surveyed (53 percent) had collected seafood for less than one hour. 21 percent had been collecting for 1 to 2 hours, and 21 percent had been collecting between 2 and 5 hours. Less than 5 percent had been collecting for greater then five hours when the survey was conducted. Three people responded that they had been continuously collecting seafood for between 15 and 30 hours.

Table 2-4. Time Spent Collecting Seafood by the Beginning of the Interview

Time Collecting	Number of People	Percent of People[a]
15 or fewer minutes	182	17
15 to 30 minutes	165	15
30 minutes to 1 hour	237	22
1 to 2 hours	225	21
2 to 5 hours	233	21
5 to 10 hours	41	4
10 to 15 hours	7	0.6
15 to 30 hours	3	0.3

[a] Percent of people out of 1,093 responses

2.3.5 Seafood Collection Frequency

The majority of the people interviewed collect seafood only in the summertime, although approximately 10 percent of the people responding collect seafood every month of the year (Figure 2-2). These results were combined with the frequency that they collect each month to estimate the number of days they collect organisms from the survey location each year (Table 2-5). Approximately 53 percent of the 948 different people responding collect seafood less than 12 times per year, about 29 percent collect between 12 and 52 times per year, and 18 percent collect more than 52 times per year.

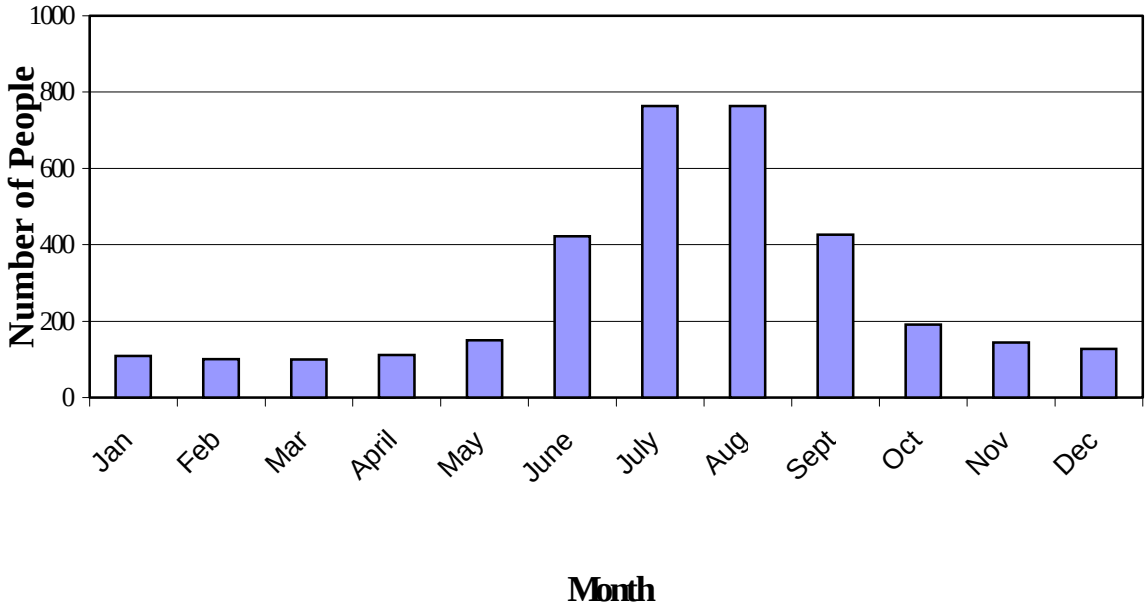

Figure 2-2. Number of People Collecting Seafood Each Month of the Year at the Survey Location

Table 2-5. Frequency with Which 1,183 People Collect and Consume Seafood from the Survey Location

Frequency (days/year)	Collect Seafood		Consume Seafood	
	Number of People[a]	Percent of People[a]	Number of People[a]	Percent of People[a]
0	0	0	466	39.5
0.1-0.9	10	0.8	1	0.08
1 - 1.9	230	19.4	78	6.6
2 - 5.9	174	14.7	114	9.6
6 - 11.9	87	7.4	65	5.5
12 – 23.9	118	9.97	69	5.8
24 – 51.9	157	13.3	72	6.1
52 – 179.9	140	11.8	41	3.5
180 – 365	32	2.7	12	1.0
No Response	235	19.9	265	22.4

[a] Out of 1,183 people surveyed

2.3.6 Seafood Consumption

About 97 percent of the 942 people responding indicated that they eat seafood. However, only 78 percent of the respondents stated that they eat seafood that they collect themselves, and only 452 people indicated that they eat seafood from the survey location.

We assumed that people that consume seafood would do so each month that they collect seafood. Based on this assumption, we combined the data on the months that people collect seafood with data on the number of meals they consume each month to estimate the number of days they consume organisms from the survey locations. Fifty-seven percent of the 452 people that said they eat seafood collected from the survey locations consume seafood less than 12 times a year. However, a large range was observed, with twelve people stating that they eat seafood from the Duwamish River or Elliott Bay at least every other day, including seven that consume seafood every day.

2.3.7 Seafood Collected

When asked whether they had recently collected seafood that had not been recorded on any survey, 209 people said that they had collected one or more type of seafood from the survey location. Of these 107 people reported that they had collected salmon, 27 collected crabs and 21 collected flounder. Dogfish, herring, lingcod, shrimp, perch, squid, rockfish, sole, sculpin, octopus, sturgeon and candlefish were each caught by fewer than 20 people.

1,218 people responded as to whether they had collected seafood on the day they were interviewed. Of these, about 14 percent had successfully collected any seafood. However, the actual success rate is likely to be higher, because the people interviewed generally continued to collect seafood after the interviews. Initial review of the data indicates a greater success rate for longer collection times.

The number of people that collected each species, the number of each species collected and the total and average weights of each seafood type collected are presented in Table 2-6. The species collected by the most people were crabs, followed by salmon and perch. Although collected by fewer people, shrimp and perch, along with crab, had the highest numbers collected. Salmon contributed the greatest portion, by weight, of seafood collected (64 percent of the total), followed by crabs (16 percent) and perch (11 percent). Seacrest was the most productive site, with more (numbers and pounds) of crab, shrimp, perch and salmon collected here than at any other location.

2.3.8 Planned Use of Collected Seafood

One hundred and thirty four people indicated during their interviews what they intended to do with the seafood that they had collected (Table 2-7). Most (74 of 134) of these people stated that they planned to eat their catch and share it with others. The remaining people stated that they would eat the seafood alone, use it as bait, release it, give it away, or responded "other." When asked about the number of people that would share the meal, 87 people stated that they would share the seafood with a total of 365 people.

Table 2-6. Number of People That Had Collected Each Species of Seafood, and the Number and Weight Collected[a]

Species	Number of People that Collected Each Species	Number Collected	Total Weight (pounds)	Average Weight per Organism (pounds)	Average Seafood Weight per Person Who Collects (pounds)
Halibut	1	4	3	0.75	3
Clams	1	25	6	0.24	6
Crabs	42	148	>90.2	0.61	2.1
Flounder	12	15	8.4	0.56	0.7
Gunnel	1	1	0.25	0.25	0.25
Herring	8	55	5.1	0.09	0.64
Ling cod	1	1	ND	ND	ND
Shrimp	11	282	>6.25	0.02	0.57
Moon snail	1	1	ND	ND	ND
Perch	19	238	>61.3	0.26	3.23
Squid	2	7	5.1	0.73	2.55
Rockfish	9	9	8.75	0.97	0.97
Sole	11	22	5.6	0.25	0.51
Salmon	33	34	>364.25	10.7	11.04
Sculpin/bullhead	9	10	2.6	0.26	0.29
Candlefish	1	30	ND	ND	ND

[a] Out of 1,218 people responding

ND = No data available

Table 2-7. Summary of Plans for Use of Seafood Collected from the Duwamish River and Elliott Bay by 134 People

Planned Use	Number of People[a]
Use as Bait	21
Eat Myself	28
Eat with Others	74
Give Away	10
Release	5
Other	6

[a] Number of people adds to greater than 134 because several people stated more than one planned use for their collected seafood.

Twenty-seven respondents also stated that they would share the seafood with children under the age of ten.

When asked what parts of the fish would be eaten, 43 out of 69 people responding (62 percent) stated that they would eat the meat only, 20 said that they would eat the meat and skin, and six said they would eat the whole fish. When asked what parts of the shellfish would be eaten, all (43 out of 43) respondents said they would eat the meat only.

Baking or frying fish was preferred 4:1 to grilling fish (Table 2-8). Other fish preparation methods (e.g., boiled) were even less preferred. Crabs, shrimp, and clams were usually boiled or steamed.

Table 2-8. Summary of the Number of People (Out of 105 Responses) Using Various Methods to Prepare Different Types of Seafood from the Duwamish River and Elliott Bay[a]

Preparation Method	Fish[b] (# of people)	Shellfish[c] (# of people)	Squid[d] (# of people)	Unknown (# of people)
Raw	0	0	1	0
Boiled	5	19	1	1
Steamed	7	13	1	1
Baked/Fried	41	5	1	8
Grilled	11	2	1	2
Soup/Broth	2	4	1	1
Other	2	1	0	2

[a] Number of people adds to greater than 105 because several people put more than one response.

[b] Fish include all species caught.

[c] Shellfish include clams, crabs and shrimp.

[d] Only one person with squid responded. This person listed six different preparation methods.

2.4 Discussion

Based on the questions on consumption frequency, 50 percent of the 452 people who eat seafood from a specific survey location eat less than eight meals a year. The national

mean intake of seafood per meal is estimated to be about four to four and one-half ounces (117 to 129 grams), while the 95th percentile[2] intake ranges from about 10 to 11.5 ounces per meal (284 to 326 grams per meal) (U.S. EPA 1996). Using these estimated meal sizes, 50 percent of the people consume an average of less than 36 ounces per year (1 kg per year) to a 95th percentile of about 86 ounces per year (2.4 kg per year) of seafood from the survey locations each year. These consumption rates are similar to the estimated average consumption rates for recreational marine anglers of 25.7 to 91.7 ounces per year (0.73 to 2.6 kg per year).

Using the same average and 95th percentile seafood meal sizes, the seven people who consume seafood from the survey locations every day consume an average of about 102.6 pounds per year (46.6 kg per year) and a worst-case of about 245 pounds per year (111 kg per year). These consumption rates are substantially larger than the worst-case consumption rates for recreational marine anglers of 21 pounds per year (9.5 kg per year) (U.S. EPA 1996), and are similar to those estimated for subsistence populations. This indicates that there is a small population of people that collect seafood from the shores of the Duwamish River and Elliott Bay that may be considered "subsistence" anglers.

The 452 people that consume study area seafood eat a grand total of 11,354 seafood meals from the Duwamish River or Elliott Bay per year. Of these, the seven people that eat one seafood meal per day (1.5 percent of the respondents) account for 20 percent of the total number of meals of Duwamish River/Elliott Bay seafood each year. Similarly, 42 percent of all such meals are consumed by 27 percent of the respondents (125 out of 452 respondents).

The size of the population that consumes seafood collected from the shores of the Duwamish River and Elliott Bay is actually larger than the observed population (Price et al. 1994, U.S. EPA 1996). We have not estimated the total population of people that collect and/or consume seafood from the shores of the Duwamish River and Elliott Bay because it is likely that the average exposures for the total population will be below the average exposure for the observed population and for risk assessment purposes, use of conservative exposure estimates is warranted.

The type of seafood collected is expected to vary throughout the year. For example, returning salmon may only be caught from the shores of the river and bay during the summer and fall. Squid is sought after during the winter, when they come close to shore to feed and spawn. Blackmouth (juvenile salmon) are mostly caught during the winter. These changes in seafood availability likely influence the numbers of people that collect organisms each month and the chemical concentrations to which people are exposed. An informal inquiry into the squid fishery indicates that many people that collect squid during the winter do not collect seafood during the summer. This implies that Figure 2-2

[2] The 95th percentile intake rate represents an intake rate that is greater than that sustained by 95% of the people. This value is an approximation of the maximum consumption rate.

(the number of people that collect seafood per month) underestimates angler pressure in winter months.

3. METHODS AND RESULTS OF THE HUMAN HEALTH CHEMICAL RISK ASSESSMENT

People recreating and working on the Duwamish River and Elliott Bay may be exposed to chemicals while engaging in a variety of activities. The human health chemical risk assessment for the Duwamish River and Elliott Bay is an attempt to quantify, when possible, the health risks from chemical exposures associated with these activities. Human health risks are estimated for baseline and without CSO conditions, risks at reference sites elsewhere in Puget Sound are also calculated.

The human health chemical risk assessment generally followed recommended U.S. EPA (1989a) methodologies. When available, regional-specific and/or site-specific data were used in the risk assessment. The human health chemical risk assessment consisted of the following components:

- In the human health chemical effects characterization, the chemical dose below which non-carcinogenic effects are not expected, and the carcinogenic potential of each chemical were obtained from toxicity databases.
- In the human health chemical exposure characterization, the concentrations and/or doses (i.e., intakes) of constituents of potential concern (COPCs) to which people may be exposed were calculated. Chemical exposure was evaluated from incidental ingestion and dermal contact exposures to both sediments and surface water, as well as from the consumption of seafood collected from the river and bay.
- In the human health chemical risk characterization, the results of the effects and exposure characterizations were combined to obtain numerical estimates of risk. The potential for non-carcinogenic effects and for carcinogenic effects were calculated separately.
- In the exposure, effects and risk uncertainty characterization sections, the uncertainties associated with the human health chemical risk assessment are discussed, along with their potential for influencing or affecting the results of the assessment.

Each of the components of the human health chemical risk assessment are discussed in the following sections.

3.1 Human Health Toxicological Effects Characterization

This section summarizes the toxicological threshold values used to estimate risks to human health from chemical exposures. This section presents an overview of different toxicity endpoints, toxicity values for non-carcinogenic health effects, carcinogenic potency "slope factors" for carcinogenic health effects, and evaluation of chemicals without chemical toxicity values.

3.1.1 Human Health Toxicity Endpoints

Human health effects are addressed separately for carcinogenic effects and all other types of adverse health effects, generically referred to as non-carcinogenic effects. The main reason for this division is that non-cancer [3] effects usually occur only after a threshold dose has been exceeded, while it is assumed that any exposure to a carcinogen will result in an increased risk of developing cancer. Issue paper number 8, "*Human Health Toxicology*" (Appendix C) presents an overview of the types of endpoints and some of the assumptions made when evaluating different effects.

Many non-cancer effects are possible from exposure to a chemical, dependent on the dose.[4] From all of the possible effect data, the values used are derived from the lowest doses at which any effect was observed. Thus, the values used to assess the potential for non-cancer health effects in this risk assessment are exposure levels at which no adverse effects are expected. In contrast to non-carcinogenic effects, the potential for carcinogenic effects is assumed to exist at any concentration[5]. Consequently, the carcinogenic toxicity values are not threshold doses, rather they are estimates of carcinogenic potency of a chemical.

The toxicity values for non-cancer-causing chemicals represent the amount that can be ingested safely on a daily basis for a long period without adverse effects. The potential for adverse effects from short-term (acute) exposure was not directly evaluated because acute effects only occur at dosages much higher than those causing long-term (chronic) effects do. Thus, chemicals for which no or only a small potential for chronic effects are predicted is also not expected to cause acute effects. Thus, the human health risk assessment relies on the assumption that we are not likely to under-estimate health effects for individuals or groups exposed for shorter periods of time.

3.1.2 Non-carcinogenic Health Effects

The U.S. EPA has developed specific methodologies for establishing numerical toxicity values for chemicals causing non-cancerous effects in animals and humans (U.S. EPA 1986a,b; 1987; 1991a; and 1995). Internationally, the World Health Organization (WHO) has also developed procedures (WHO 1996a) and these procedures largely parallel those of the U.S. EPA.

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[3] See Issue paper no. 8 "*Human Health Toxicology*" (Appendix C) for a discussion of non-cancer health effects.

[4] The term dose has a specific meaning depending on the context on which it is used. In this case the term dose is used to mean the "administered dose" or the amount of chemical taken into the body by the exposure pathway evaluated. The calculated chemical intake is used as an estimate of this dose.

[5] See Issue paper no. 8 "*Human Health Toxicology*" (Appendix C) for a discussion of when this assumption might be inappropriate.

Toxicity values for ingestion exposures developed by the U.S. EPA for non-cancer health effects are referred to as reference doses (RfDs). RfDs are designed to be protective of potentially sensitive subpopulations (e.g. children) and of individuals within the general human population. Therefore, exposure to chemicals at or below the threshold (i.e., RfD) level is expected to be protective for any exposed individual. The RfD is defined as "an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime" (U.S. EPA 1998).

The RfDs are available for the ingestion route of exposure only, with no toxicity values currently available for assessing toxicity from the dermal exposure route. Guidance calls for extrapolation of a toxicity value for this route of exposure from the ingestion RfD (U.S. EPA 1989a, 1992a). In the WQA, ingestion RfDs were used directly for assessing dermal exposures without extrapolation because of uncertainties in the extrapolation methods. Extrapolation of ingestion RfDs to dermal exposure toxicity values is described in Issue Paper Number 8, "*Human Health Toxicology"* (Appendix C).

For the WQA, non-carcinogenic toxicity values developed by the U.S. EPA are used (Table 3-1). The U.S. EPA maintains two readily available sources of RfDs, the primary source is the Integrated Risk Information System Database (IRIS), which is available online at the U.S. EPA web site. The second major source of RfDs is the U.S. EPA Health Effects Summary Tables (HEAST). HEAST is updated every several years and the most recent version was published in 1997. The majority of the U.S. EPA RfDs used in the assessment were taken from IRIS, the RfD for p–cresol (4-methylphenol) was taken from HEAST. For lead and copper U.S. EPA ingestion RfDs were not available and other toxicity values were used (i.e., the WHO Tolerable Daily Intakes). For methylmercury the RfD recommended by Toxicology Excellence for Risk Assessment (TERA 1998) was used.

Many of the issues associated with the development of RfDs are presented in Issue Paper No. 8, "*Human Health Toxicology*" (Appendix C). In summary, non-cancer toxicity values used in the risk assessment were based on either (1) the lowest doses observed in laboratory toxicity studies of laboratory animals, or (2) the lowest dose associated with an

adverse effect in human epidemiological[6] studies. The specific endpoints evaluated varied for each chemical substance, and are dose-dependent because different toxic effects occur at different dosages. For example, metals may affect both liver and kidney, but at significantly different doses.

[6] An epidemiological study examines the causes and characteristics of diseases in human populations in contrast to the laboratory toxicity tests of small mammals (e.g., rats, mice) routinely used to index toxicity to humans.

Table 3-1. Non-Carcinogenic Toxicity Information for COPCs

Analyte	RfD	LOAEL (mg/kg/d)	Uncertainty Factors	Modifying Factors	Benchmark Dose (mg/kg/d)	NOAEL (mg/kg/d)	Test Animal Species	Effects	References
Metals/Metalloids									
Arsenic	0.0003	0.014	3	1	N/AV	0.0008	Human epidemiology studies	Skin changes (non-carcinogenic)	U.S. EPA (1998)[a]
Cadmium (water)	0.0005	N/AV	10	1	N/AV	0.005	Human epidemiology studies	Kidney toxicity	U.S. EPA (1998)
Cadmium (food)	0.001	N/AV	10	1	N/AV	0.01	Human epidemiology studies	Kidney toxicity	U.S. EPA (1998)
Copper	0.5	N/AV	10	1	N/AV	5	Dogs	Liver toxicity	WHO (1996a)[a]
Lead[b]	0.0035	N/AV	N/AV	N/AV	N/AV	N/AV	Human epidemiology studies	Neurological impairment	WHO (1996a)
Mercury (inorganic)	0.0003	0.226	1,000	1	N/AV	None	Rats	Neurological and kidney effects	U.S. EPA (1998)
Methyl mercury	0.0004	N/AV	N/AV	N/AV	0.0009 to 0.003	N/AV	Human epidemiology studies	Neurological impairment	TERA (1998)
Nickel	0.02	50	300	1	N/AV	5	Rats	Systemic organ weight changes	U.S. EPA (1998)
Zinc	0.3	1	3	1	N/AV	None	Human epidemiology studies	Changes in copper status[c]	U.S. EPA (1998)

Table 3-1. Non-carcinogenic Toxicity Information for COPCs (continued)

Analyte	RfD	LOAEL (mg/kg/d)	Uncertainty Factors	Modifying Factors	Benchmark Dose (mg/kg/d)	NOAEL (mg/kg/d)	Test Animal Species	Effects	References
Organometallics									
Tributyltin	0.0003	N/AV	100	1	0.03	N/AV	Rats	Immunotoxicity	U.S. EPA (1998)
Polychlorinated Biphenyls									
Aroclor 1016	0.00007	0.028	100	1	N/AV	0.007	Monkeys, animal and human epidemiology studies	Generalized clinical toxicity [d]	U.S. EPA (1998, 1996)
Aroclor 1254	0.00002	0.005	300	1	N/AV	None	Monkeys, animal and human epidemiology studies	Generalized clinical toxicity [d]	U.S. EPA (1998, 1996)
Semivolatile Organics									
4-Methylphenol	0.005	N/AV	1,000	1	N/AV	5	Rabbits	Nervous system depression/ Respiratory distress	HEAST (1997)
Bis(2-ethylhexyl) phthalate	0.02	19	1,000	1	N/AV	None	Guinea pigs	Liver Toxicity	U.S. EPA (1998)

Table 3-1. Non-carcinogenic Toxicity Information for COPCs (continued)

Analyte	RfD	LOAEL (mg/kg/d)	Uncertainty Factors	Modifying Factors	Benchmark Dose (mg/kg/d)	NOAEL (mg/kg/d)	Test Animal Species	Effects	References
PAHs									
Fluoranthene	0.04	250	3,000	1	N/AV	125	Mice	Kidney toxicity	U.S. EPA 1998
Pyrene	0.03	125	3,000	1	N/AV	75	Mice	Kidney toxicity	U.S. EPA 1998

Uncertainty Factors and Modifying Factors are adjustments to the reference dose (RfD) to account for uncertainties in toxicological data (see Issue paper no. 8 "*Human Health Toxicology*" [Appendix C] for further detail.)

Benchmark Dose is an estimate at a lower statistical confidence limit corresponding to a specified level of risk.

[a] Values referenced as U.S. EPA or HEAST are RfDs and WHO values are tolerable daily intakes (TDIs).

[b] WHO has established a provisional tolerable weekly intake based on metabolic studies in infants and children, showing that an intake of 5 micrograms of lead per kilogram of body weight resulted in lead retention in tissues, while 3-4 micrograms per kilogram of body weight did not result in any increase in lead body burden.

[c] Copper and zinc biochemistry are related, excess intake of zinc may affect the amount of copper-containing enzymes in the body.

[d] Clinical signs of toxicity included eye exudate, inflammation of the eyelid, changes in finger and toe nails, and decreased antibody response to immune challenge.

LOAEL – Lowest Observed Adverse Effect Level.

NOAEL – No Observed Adverse Effect Level.

HEAST – Health Effects Summary Tables.

N/AV –Toxicity values for non-carcinogenic effects were not available for these chemicals.

TERA – Toxicology Excellence for Risk Assessment

The general approach used by the U.S. EPA for developing RfD values for any non-cancer-causing chemical is to identify the upper end of the tolerance range (i.e., the highest dose) that is not associated with health effects. In general, the highest dose tested that is without adverse effects are the key piece of data taken from the study and used to establish the RfD. This dose is called the No-Observed-Adverse-Effect Level (NOAEL).

A series of adjustments, termed uncertainty factors, may then be made to the NOAEL dose to account for uncertainties, to derive the RfD. These adjustments are made to ensure the protectiveness of the toxicity value. Inherent in the use of these adjustments for uncertainty is the assumption that humans are at least as sensitive (and possibly more sensitive) as the animal species tested for each chemical.

Depending on the strength of the underlying toxicological database for a chemical, one or more uncertainty factors can be used to adjust the NOAEL identified. These uncertainty factors (UF) are applied in factors of 10 as shown below:

- UF of 10 to account for variability in the general population, including sensitive subpopulations (elderly, children),
- UF of 10 to address extrapolation of animal data to humans,
- UF of 10 when a NOAEL for critical effect is derived from a subchronic (intermediate exposure) rather than a chronic (long-term) exposure, and
- UF of 10 when a dose associated with an effect (rather than a NOAEL, which is associated with no effect) must be used.

In addition, another factor up to a value of 10, termed a modifying factor (MF), may be applied in addition to the UFs identified above. The modifying factor is used to address any additional uncertainties in the critical study and in the entire body of toxicological data available for the chemical. The default value for the MF is a value of 1.

Given these uncertainties, it is possible for RfD values to include uncertainty factors of up to a value of 10,000. Where data from animals is used, typically the minimum UF applied is 100; this assumes that humans are at least 100 times more sensitive than the test animals. A summary of the test data used to derive the RfDs, along with the uncertainty and modifying factors applied, the type of effect, and the test species is presented in Table 3-1.

For some chemicals, similar effects are reported due to effects on the same target organs. For example, lead and methyl mercury both have adverse effects on the central nervous system (WHO 1996b, U.S. EPA 1998). In some cases, similar effects may be due to similar mechanisms of toxic effects. For chemicals where toxic effects are due to similar

mechanisms of action the possibility of additive[7] effects exists as a result of the cumulative exposures.

Some chemicals are considered to be essential human nutrients, or are toxic only at high doses. Chemicals which are considered essential nutrients, and that have been evaluated in this risk assessment are copper and zinc (WHO 1996b). The WHO estimates daily requirements for copper and zinc are 1 to 5 mg/day and 15 to 22 mg/day, respectively (WHO 1996b). The U.S. Recommended Daily Intakes (RDIs) for copper and zinc are 2 mg/day for copper and 5 to 15 mg/day for zinc (U.S. EPA 1998; 21 CFR Vol. 2 Part 100-169). If intakes predicted for these chemicals fall within the nutritional concentration range, they have only beneficial effects.

3.1.3 Carcinogenic Health Effects

The toxicity value used in assessing carcinogenic effects is termed the "slope factor" and refers to the slope of the "dose-response" curve which estimates the carcinogenic potency of a chemical at the low-doses expected to occur from environmental exposure. The endpoint evaluated for chemical carcinogens is expressed as the probability of developing cancer from these exposures. The toxicity values for cancer effects are presented below in Table 3-2.

The term "slope factor" refers to the slope of a linear mathematical model fitted to cancer potency data, usually derived from studies in animals[8] . It is defined as a plausible upper-bound estimate of the probability of a response per unit of chemical intake over a lifetime. Because the toxicity values for carcinogenic chemicals are expressed as a "probability of response" rather than a dose as is the case for non-cancer-causing chemicals, the resulting risk estimates are also expressed as a probability of contracting cancer.

In addition to an estimate of carcinogenic potency, U.S. EPA has derived estimates of the probability that a specific chemical is a human carcinogen. This is necessary because carcinogenic potency estimates are derived from animal data and is referred to as the

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[7] Additive effects may result when two chemicals with a similar mechanism of toxic action exert effects on the same organ or tissue thereby resulting in a greater toxicity than would occur by each individual chemical.

[8] The slope factor is derived from the linear portion of the dose-response curve predicted by the selected model. Linearity of dose-response curves is postulated to occur at only the lowest environmental exposures for most chemicals. Thus, for a given chemical, the model extrapolates the dose-response curve from the study doses where tumors were observed to lower doses where exposures in the environment would be expected to occur. This concept is a key assumption in the development of the slope factor because at higher exposures than predicted by the model, the dose-response curve is not linear and the slope factor will result in inaccurate risk estimates.

carcinogenic weight-of-evidence. The weight-of-evidence rating is derived from the available body of toxicological data and includes animal test data and other supporting

Table 3-2. Carcinogenic Toxicity Information for COPCs

Analyte	Slope Factor	Test Animal Species	WOE[a]	Effects	References
Metals/Metalloids					
Arsenic	1.5	Human epidemiology studies	A	Skin and organ cancers	U.S. EPA (1998)
Polychlorinated Biphenyls					
Aroclor 1016	2/0.4[b]	Monkeys, animal and human epidemiology studies	N/AV	A variety of cancer types including liver, gastrointestinal and skin	U.S. EPA (1996, 1998)
Aroclor 1221	2/0.4	Animal and human epidemiology studies	N/AV	A variety of cancer types including liver, gastrointestinal and skin	U.S. EPA (1996)
Aroclor 1232	2/0.4	Animal and human epidemiology studies	N/AV	A variety of cancer types including liver, gastrointestinal and skin	U.S. EPA (1996)
Aroclor 1242	2/0.4	Animal and human epidemiology studies	N/AV	A variety of cancer types including liver, gastrointestinal and skin	U.S. EPA (1996)
Aroclor 1248	2/0.4	Animal and human epidemiology studies	N/AV	A variety of cancer types including liver, gastrointestinal and skin	U.S. EPA (1996)
Aroclor 1254	2/0.4	Monkeys, animal and human epidemiology studies	N/AV	A variety of cancer types	U.S. EPA (1996, 1998)
Aroclor 1260	2/0.4	Animal and human epidemiology studies	N/AV	A variety of cancer types including liver, gastrointestinal and skin	U.S. EPA (1996)
Total PCBs	2/0.4	Animal and human epidemiology studies	N/AV	A variety of cancer types including liver, gastrointestinal and skin	U.S. EPA (1996)
Semivolatile Organics					
1,4-Dichlorobenzene	0.024	Rats	C	Liver Tumors	HEAST (1997)

Table 3-2. Carcinogenic Toxicity Information for COPCs (continued)

Analyte	Slope Factor	Test Animal Species	WOE[a]	Effects	References
Bis(2-ethylhexyl)phthalate	0.014	Mice	B2	Liver carcinoma and adenoma	U.S. EPA (1998)
PAHs					
Benzo(a)anthracene	0.73	Mice	B2	Skin cancer	U.S. EPA (1993)
Benzo(a)pyrene	7.3	Mice	B2	Skin cancer	U.S. EPA (1993)
Benzo(b)fluoranthene	0.73	Mice	B2	Skin cancer	U.S. EPA (1993)
Benzo(g,h,i)perylene	N/AV	N/AV	D	N/AV	
Benzo(k)fluoranthene	0.073	Mice	B2	Skin cancer	U.S. EPA (1993)
Chrysene	0.0073	Mice	B2	Skin cancer	U.S. EPA (1993)
Dibenzo(a,h)anthracene	7.3	Mice	B2	Skin cancer	U.S. EPA (1993)
Phenanthrene	N/AV	N/AV	D	N/AV	U.S. EPA (1993)
Indeno(1,2,3-cd) pyrene	0.73	Mice	B2	Skin cancer	U.S. EPA (1993)

[a] U.S. EPA carcinogenic weight-of-evidence rating.

[b] Estimates of PCB carcinogenic potency vary dependent on a variety of factors. For food and sediment pathways an estimate of 2.0 was used; for water pathways, 0.4.

HEAST – Health Effects Summary Tables.

N/AV –Toxicity values for non-carcinogenic effects were not available for these chemicals.

information such as mutagenicity[9] tests. The weight of evidence ratings for the WQA chemicals are presented in Table 3-2. The weight of evidence scale for carcinogenicity is as follows:

A	Confirmed Human Carcinogen
B1 or B2	Probable Human Carcinogen
C	Possible Human Carcinogen
D	Not Classifiable as to Carcinogenicity
E	Evidence of Non-carcinogenicity in Humans

Health risks are difficult to measure in human or animal studies at the very low exposure levels typical of environmental risk assessments. Therefore, the development of a slope factor using current U.S. EPA protocols depends on the use of mathematical models to extrapolate from the high doses typical of laboratory animal studies (or from epidemiological studies with humans) to the lower exposure levels expected in the environment.

U.S. EPA slope factors do not consider possible threshold mechanisms for non-genotoxic[10] carcinogenic chemicals, though this is likely to change[11]. Some of the carcinogenic chemicals evaluated in the WQA, such as arsenic, are thought to be non-genotoxic. The result of not considering the possibility of a threshold to the Duwamish River and Elliott Bay WQA is a potential to over-estimate the cancer risk to people from exposure to cancer-causing chemicals in WQA site media (fish, sediment, water). King County has, however, elected to be conservative in their evaluation of potential health risks in the WQA, and thus, U.S. EPA slope factors are used in evaluating human cancer risks.

Similar to additive effects for non-carcinogenic effects, the potential exists that exposure to two or more carcinogens may result in a greater risk of developing an environmentally induced cancer than would occur by exposure to a single carcinogen if these carcinogens act on a single organ or tissue. In the WQA, the effects of multiple carcinogen exposures are assumed to be additive, and total risks from exposure to multiple carcinogens is assessed by summing the chemical-specific risks. The mechanism of carcinogenicity varies for the various chemicals considered in the WQA (i.e., arsenic and PCBs). Therefore, it is unlikely that the carcinogenic effects from combined exposures are

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[9] Mutagenicity (i.e., the ability of a chemical to cause genetic mutations) is thought to be related to the carcinogenic process.

[10] Genotoxic carcinogens are those chemicals that are directly toxic to cell DNA (e.g., mutagenic) and may cause cancer by this mechanism.

[11] In 1996 the U.S. EPA, recognizing the need to update the science underlying the current cancer assessment process, proposed revisions to their 1986 Cancer Assessment Guidelines (U.S. EPA 1996). See Issue paper no. 8 *"Human Health Toxicology"* (Appendix C).

strictly additive. Rather, they are likely to be independent. Thus, the additivity assumption is a conservative approach in assessing total carcinogenic risks.

3.1.4 Evaluation of Chemicals without Available Toxicity Values

At least one chronic toxicity value (RfD or slope factor) was available for every COPC except benzo(g,h,i)perylene and phenanthrene. Chronic toxicity values were unavailable from U.S. EPA or WHO for these two PAHs. Consequently, neither of these compounds could be quantitatively evaluated for chronic effects. No information was identified in the review of toxicological data for this report to suggest that either benzo(g,h,i)perylene or phenanthrene are carcinogenic in humans. The International Agency for Research on Cancer (IARC 1992) has evaluated these two PAHs and concluded that they are not classifiable with respect to carcinogenic potential. Similarly, the U.S. EPA has categorized these two PAHs as group D (U.S. EPA 1998). This designation indicates that there is insufficient information to suggest that these compounds are carcinogenic. Both benzo(g,h,i)perylene and phenanthrene have been shown to be ineffective at initiating genetic damage[12] and have not induced cancers in animal tests (ATSDR 1994). Therefore, the potential carcinogenic risks from exposure to these PAHs are uncertain, and the limited information available does not indicate that they are likely to be carcinogenic.

3.1.5 Uncertainties in Toxicity Assessment

Uncertainty in using chronic toxicity values occurs because data for the majority of chemicals evaluated are derived through extrapolation of animal data to humans, from high experimental doses to low environmental doses, from sub-chronic (short-term laboratory) to chronic (long-term environmental exposure), and from average to more sensitive members of the population (U.S. EPA 1989a).

Chronic toxicity values have additional uncertainties specific to chronic exposures. For non-carcinogenic RfDs, these uncertainties are generally accounted for by the use of "safety factors" that can be as high as 10,000. As described in Section 3.1, safety factors are typically applied to the experimentally derived toxicity values to provide a "margin-of-safety." Safety factors are incorporated into a non-carcinogenic RfD, with the intent to protect the entire potentially exposed population, including sensitive people. However, the use of these safety factors may greatly under-estimate the "safe dose" for a chemical to which they are applied.

For carcinogenic slope factors, the general sources of uncertainty described above apply (i.e., high dose to low dose, subchronic to chronic, and animal to human). Additionally,

[12] Genetic changes (i.e., mutagenesis) are thought be causally related to the development of cancer for PAHs.

for carcinogenic chemicals the slope factors make no assumption regarding the existence of a "safe dose" (i.e., toxic threshold) any dose is assumed to have some probability of cancer induction. This assumption in itself is very conservative, due to the possible existence of a threshold dose for many non-genotoxic carcinogens. The current state of knowledge regarding arsenic carcinogenicity suggests that it may have a threshold (Wilson 1996, Barrett 1993, U.S. EPA 1998). Therefore the carcinogenic potential of arsenic may be over predicted.

Additional uncertainty is introduced in the toxicity assessment because toxicity values for some PAHs are lacking. However, the toxicity of PAHs without toxicity values (benzo(g,h,i)perylene, phenanthrene) are not expected to be significantly different from those with toxicity values. Additionally, PAHs were infrequently detected in seafood tissues and are not expected to represent significant contributions to total risk.

3.2 Human Health Chemical Exposure Characterization

This section presents the methods and assumptions used to estimate human exposures to chemicals. Chemical toxicity information is presented above in Section 3.1. The methods for estimating health risks from chemicals are presented below in Section 3.3. The results of the human health chemical risk assessment are presented below in Section 3.4. Potential human exposures and health risks from pathogens (i.e., bacteria, viruses, and parasites) are discussed separately in Section 4.

It was determined in Appendix A *Problem Formulation, Analysis Plan, and Field Sampling Work Plan* that people may be exposed to chemicals in water and sediment through a variety of direct exposure activities, such as swimming and SCUBA diving, among others. It was also determined that people may be exposed to chemicals through indirect activities, such as the consumption of seafood harvested from the river or the bay. A fishing survey was conducted during the summer 1997 to allow for better estimates of the type and amount of seafood collected and consumed, and the number of people that engage in these activities (Section 2).

As described in Appendix A and discussed below, the exposure assessment stage of the human health risk assessment consists of identifying potentially exposed populations and exposure pathways, estimating exposure concentrations, and calculating the range of chemical exposures.

3.2.1 Identification of Potentially Exposed Populations

People may be exposed to chemicals in Duwamish River and Elliott Bay waters and sediments through a variety of activities. As described in Appendix A - *Problem Formulation, Analysis Plan, and Field Sampling Work Plan,* populations potentially exposed to chemicals in the Duwamish River and Elliott Bay include those persons who use the river and bay for recreational activities (e.g., swimming), and those who may catch and consume fish, shellfish, and other seafoods from the river and bay. Each of these is further described below.

Recreational Users. The Duwamish River and Elliott Bay are heavily used urban waterbodies. There are many access points along the shoreline of the river and bay, and many people may use these access points for recreational activities. A more complete discussion of uses is presented in Issue Paper No. 3 "*Human Site Use*" in Appendix C. People may engage in a wide variety of recreational activities that will result in exposure to chemicals in water and sediment, such as:

- Swimming at Duwamish Park or Duwamish Head
- Scuba diving at Seacrest Park
- Wading at Duwamish Park, Duwamish Head, or other access points
- Sailing in Elliott Bay
- Wind surfing in Elliott Bay
- Boating in the Duwamish River and Elliott Bay
- Kayaking in the Duwamish River and Elliott Bay
- Parasailing in Elliott Bay
- Water skiing in Elliott Bay
- Jet skiing in Elliott Bay

The study area is accessible from private and public properties along much of the shoreline. During the summer months, and to a lesser extent the winter months, the public, including adults and children may recreate in the study area. Because these individuals may potentially come into contact with the surface water and bottom sediments, they have been identified as potentially exposed populations.

People Collecting and Consuming Marine Organisms. Many people collect fish, shellfish, and other organisms throughout the Duwamish River and Elliott Bay for food and recreation. Specifically, people:

- Line fish from the shores of the Duwamish River and Elliott Bay, and from boats in Elliott Bay
- Net fish in the Duwamish River
- Gather shellfish and other organisms (e.g., mussels, crabs, sea cucumbers, and seaweed) at access points in the Duwamish River and Elliott Bay
- Consume fish, shellfish or other organisms gathered (by themselves or others) from the river or bay.

King County conducted a survey of people line fishing and collecting shellfish and other organisms from the shores of the river and bay during summer 1997 (Section 2 above).

The survey confirmed that people do line fish and harvest shellfish and other organisms from the shores of the river and bay. About 40 percent of the people interviewed stated that they consumed seafood from the study area. Most (50 percent) of the observed people that consume seafood from the study area eat no more than eight meals per year. However, seven individuals (about 1.5 percent of the observed people that consume seafood from the study area) were identified that eat seafood from the study area every day of the year. In addition, people that collect marine organisms from the Duwamish River and Elliott Bay may be directly exposed to chemicals in sediment and water during their collection activities. Accordingly, people that collect and consume fish, shellfish, and other organisms have been identified as potentially exposed populations.

3.2.2 Identification of Potential Exposure Pathways

In risk assessments, the exposure pathway is defined as the course a chemical takes from its source to a given receptor (U.S. EPA 1989a). Each exposure pathway includes a source (where chemicals originate), an exposure point (point of contact) and an exposure route (route of entry into the body).

As discussed in the previous section and in the issue paper discussing human site use, the study area is used for a variety of activities that could involve direct or indirect exposure to chemicals in surface water, sediments, and the tissues of fish, shellfish and other organisms. Exposure pathways evaluated in the human health risk assessment are summarized in Table 3-3. As shown, incidental ingestion of water and sediment, dermal contact with water and sediment, and ingestion of seafood harvested from the Duwamish River and Elliott Bay were quantitatively assessed.

Table 3-3. Exposure Pathways Evaluated in Human Health Risk Assessment [a]

Exposure Pathways	Human Site Uses Evaluated				
	Swimming	SCUBA Diving	Windsurfing	Net Fishing	Seafood Consumption
Incidental Ingestion of Water	X	X	X	X	
Skin Contact with Water	X	X	X	X	
Incidental Ingestion of Sediments	X			X	
Skin Contact with Sediments	X			X	
Ingestion of Fish					X
Ingestion of Shellfish and other organisms					X

[a] Other exposure pathways are assumed to result in lower exposures than those estimated.

Several of the identified exposure activities were not explicitly quantified because their exposures resulting from these activities are expected to be similar or less than comparable exposure pathways that were selected for evaluation. Direct exposures to sediment and water via sailing, boating, kayaking, parasailing, water skiing and jet skiing were not evaluated because exposures during these activities are expected to be similar or less than exposures occurring while wind surfing. Similarly, direct exposures to sediment and water while wading were not assessed because exposures while swimming are expected to be larger and provide a more conservative estimate of exposure. Finally, direct exposures to sediment and water while line fishing and gathering shellfish and other organisms were not assessed because these exposures are expected to be smaller than those experienced while net fishing.

Cumulative exposures through multiple exposure pathways were not quantified in the exposure assessment. Such cumulative exposures were not quantified because of the uncertainties associated with such exposures. For example, it is possible an individual may be exposed while swimming (via incidental ingestion and dermal contact with water and sediment) and while SCUBA diving. Other individuals may be exposed while net fishing and from consuming seafood from the river and bay. These types of cumulative exposures were not quantitatively assessed for three reasons. First, for possible combinations of the direct exposure pathways, no data were available to indicate the frequency that people would engage in multiple activities. Second, a range of exposure frequencies was assumed (discussed below) to account for worst-case exposure scenarios. Third, by calculating exposure, and subsequent risks, by activity, the reader is then able to estimate their personal level of exposure (and subsequent risk) based on their personal uses of the river and bay.

Both adults and children may be exposed through one or more of the evaluated activities. It was expected that children would swim in the river and bay. Results from King County's fishing survey (Section 2 above) indicated that children both collect and consume seafood from the Duwamish River and Elliott Bay. Exposures by both children and adults were assessed separately for swimming in the Duwamish River and Elliott Bay and consuming seafood from both areas. Children were not evaluated for SCUBA diving, wind surfing or net fishing, as it was assumed that children rarely engage in these or comparable recreational activities.

3.2.3 Exposure Concentrations

Chemical concentrations to which people may be exposed were estimated from the results of the water and sediment quality model and the fish and invertebrate tissue data. Exposure concentrations were estimated for baseline conditions (with CSO discharges), and without CSO discharges and at reference sites in Puget Sound. Exposure concentrations of COPCs in water, sediment, and biota are described in the sections that follow.

Water and Sediment Chemical Exposure Concentrations. Chemical concentrations in water and sediment were estimated using the results of the water and sediment quality model. The water and sediment quality model was calibrated to the results of the sampling and analysis program, which included the collection of about 2,000 samples and about 13,000 chemical analyses. The model used is a dynamic three-dimensional hydrodynamic and chemical fate and transport model developed for the Duwamish River. The model divides the river (north of the Interstate 405 Bridge) and Elliott Bay into 512 cells, which are then divided into 10 layers resulting in 5,120 cell-layers. Sediments are also modeled for each of the 512 cells. Chemical inputs from the Green River upstream of the study area, the Puget Sound boundary, CSOs, sediments, and other sources are accounted for within the model. The model was used to calculate water and sediment concentrations for baseline conditions (i.e., including CSO discharges) and without CSO discharges. Annual average chemical concentrations were used at the locations where exposures are expected to occur. The locations of the exposures are presented in Table 3-4.

Table 3-4. Locations of Direct Human Exposures to Water and Sediment

Activity	Location	Water Cells	Sediment Cells
Swimming	Duwamish River at Duwamish Park	Surface cells only	Yes
	Elliott Bay at Duwamish Head	Surface cells only	Yes
SCUBA Diving	Elliott Bay at Seacrest Park	All depths	No
Wind Surfing	Elliott Bay – entire bay	Surface cells only	No
Net Fishing	Duwamish River – turning basin to mouth	All depths	Yes

Chemical exposure concentrations in water and sediment were estimated as the annual average concentrations for each exposure location. When a location included more than one cell (e.g., all cells within Duwamish River), the exposure concentrations were calculated as the average of the annual average concentration of each cell.

Chemical concentrations in Duwamish River and Elliott Bay water and sediment (wet weight) used in the risk assessment are presented by exposure pathway in Tables 3-5, 3-6, and 3-7, for baseline conditions, without CSO conditions, and reference sites, respectively.

Baseline Chemical Concentrations in Fish and Shellfish. Chemical concentrations in fish and shellfish were estimated using the fish and invertebrate tissue data, and supplemented with concentration data available from the Washington State Department of Fish and Wildlife (WDFW). Each tissue sample consisted of a composite of 3 to 20 individuals, depending on the species (see the *Field Sampling Work Plan* in Appendix

A.) Species for which data were available, and the locations from which they were obtained, are presented in Table 3-8.

Table 3-5. Baseline Chemical Concentrations in Water and Sediment Used in Human Health Risk Assessment

COPC	Swimming				Net Fishing		SCUBA	Wind Surfing
	Duwamish Park		Duwamish Head		Duwamish River		Seacrest	Elliott Bay
	Water (mg/L)	Sediment (mg/kg wet weight)	Water (mg/L)	Sediment (mg/kg wet weight)	Water (mg/L)	Sediment (mg/kg wet weight)	Water (mg/L)	Water (mg/L)
Metals/Metalloids								
Arsenic	7.40E-04	3.61E+00	1.17E-03	6.31E+00	1.12E-03	9.09E+00	1.24E-03	1.15E-03
Cadmium	4.09E-05	1.34E-01	6.30E-05	7.82E-01	6.36E-05	1.14E+00	6.50E-05	6.23E-05
Copper	2.08E-03	1.50E+01	7.90E-04	4.10E+01	1.62E-03	5.73E+01	5.51E-04	8.02E-04
Lead	7.45E-04	5.45E+00	2.20E-04	2.81E+01	6.12E-04	4.88E+01	1.05E-04	2.03E-04
Mercury	6.06E-06	6.53E-03	1.53E-06	1.77E-01	3.39E-06	1.84E-01	8.31E-07	1.52E-06
Nickel	1.08E-03	6.25E+00	6.73E-04	1.48E+01	9.27E-04	1.44E+01	5.80E-04	6.57E-04
Zinc	4.66E-03	2.44E+01	1.48E-03	6.95E+01	3.19E-03	8.60E+01	8.06E-04	1.41E-03
Organometallics								
Tributyltin	1.43E-06	4.03E-03	3.06E-07	2.67E-01	1.08E-06	4.75E-01	1.11E-07	2.98E-07
Polychlorinated Biphenyls								
Total PCBs	1.44E-05	6.12E-02	1.16E-05	1.21E-01	1.74E-05	2.08E-01	6.08E-06	1.51E-05
Semivolatile Organics								
1,4-Dichlorobenzene	5.67E-06	5.41E-04	2.07E-06	4.83E-02	4.55E-06	1.60E-01	8.92E-07	2.45E-06
4-Methylphenol	5.99E-05	2.92E-03	1.38E-05	6.06E-02	3.66E-05	1.47E-01	5.42E-06	1.60E-05
Benzo(a)anthracene	1.32E-06	8.84E-03	7.12E-08	2.10E-01	5.28E-07	3.57E-01	2.82E-08	7.20E-08
Benzo(a)pyrene	1.51E-07	1.01E-03	8.16E-09	2.40E-02	6.06E-08	4.09E-02	3.23E-09	8.25E-09
Benzo(b)fluoranthene	2.91E-06	3.48E-02	4.72E-07	1.78E-01	2.33E-06	3.06E-01	1.70E-07	4.48E-07
Benzo(g,h,i)perylene	1.11E-06	1.32E-02	1.80E-07	6.76E-02	8.84E-07	1.16E-01	6.44E-08	1.70E-07
Benzo(k)fluoranthene	9.89E-07	1.47E-02	1.71E-07	2.14E-01	9.27E-07	2.30E-01	6.20E-08	1.59E-07
Bis(2-ethylhexyl) phthalate	1.20E-04	2.62E-01	7.82E-05	9.82E-02	9.44E-05	5.77E-01	6.86E-05	7.88E-05
Chrysene	1.38E-06	9.21E-03	7.42E-08	2.18E-01	5.51E-07	3.72E-01	2.94E-08	7.50E-08
Dibenzo(a,h)anthracene	1.16E-07	1.39E-03	1.89E-08	7.11E-03	9.31E-08	1.22E-02	6.78E-09	1.79E-08
Fluoranthene	1.11E-05	3.25E-02	1.43E-06	3.29E-01	5.43E-06	5.33E-01	5.18E-07	1.43E-06
Phenanthrene	2.55E-05	8.40E-03	2.61E-06	2.00E-01	8.39E-06	2.62E-01	9.52E-07	2.39E-06
Pyrene	2.26E-05	2.23E-01	5.56E-07	3.59E-01	3.43E-06	5.07E-01	4.91E-07	5.29E-07
Indeno(1,2,3-cd)Pyrene	4.37E-07	5.23E-03	7.09E-08	2.67E-02	3.49E-07	4.58E-02	2.54E-08	6.71E-08

Table 3-6. Chemical Concentrations in Water and Sediment Under Without CSO Conditions Used in the Human Health Risk Assessment

	Swimming				Net Fishing		SCUBA	Wind Surfing
	Duwamish Park		Duwamish Head		Duwamish River		Seacrest	Elliott Bay
COPC	Water (mg/L)	Sediment (mg/kg)	Water (mg/L)	Sediment (mg/kg)	Water (mg/L)	Sediment (mg/kg)	Water (mg/L)	Water (mg/L)
Metals/Metalloids								
Arsenic	7.31E-04	4.77E+00	1.17E-03	6.31E+00	1.13E-03	9.25E+00	1.26E-03	1.16E-03
Cadmium	4.14E-05	1.77E-01	6.36E-05	7.82E-01	6.47E-05	1.15E+00	1.38E-07	6.28E-05
Copper	2.02E-03	1.91E+01	7.67E-04	4.10E+01	1.58E-03	5.79E+01	5.44E-04	7.79E-04
Lead	7.10E-04	7.17E+00	2.10E-04	2.81E+01	5.94E-04	4.94E+01	1.04E-04	1.93E-04
Mercury	6.10E-06	5.86E-03	1.57E-06	1.77E-01	3.43E-06	1.84E-01	8.38E-07	1.57E-06
Nickel	1.03E-03	8.26E+00	6.68E-04	1.48E+01	9.14E-04	1.45E+01	5.86E-04	6.56E-04
Zinc	4.51E-03	3.17E+01	1.43E-03	6.95E+01	3.12E-03	8.71E+01	8.03E-04	1.35E-03
Organometallics								
Tributyltin	1.47E-06	3.62E-03	3.33E-07	2.67E-01	1.11E-06	4.74E-01	1.17E-07	3.33E-07
Polychlorinated Biphenyls								
Total PCBs	1.44E-05	1.61E-02	1.15E-05	1.40E-01	1.72E-05	7.77E-01	6.02E-06	1.50E-05
Semivolatile Organics								
1,4-Dichlorobenzene	5.73E-06	4.83E-04	2.04E-06	4.83E-02	4.43E-06	1.60E-01	8.73E-07	2.40E-06
4-Methylphenol	6.14E-05	2.58E-03	1.36E-05	6.06E-02	3.52E-05	1.45E-01	5.24E-06	1.54E-05
Benzo(a)anthracene	1.43E-06	7.26E-03	7.11E-08	2.09E-01	5.51E-07	3.57E-01	2.82E-08	6.82E-08
Benzo(a)pyrene	1.64E-07	8.32E-04	8.15E-09	2.40E-02	6.31E-08	4.09E-02	3.24E-09	7.81E-09
Benzo(b)fluoranthene	3.07E-06	2.98E-02	4.91E-07	1.78E-01	2.40E-06	3.05E-01	1.73E-07	4.74E-07
Benzo(g,h,i)perylene	1.17E-06	1.13E-02	1.87E-07	6.75E-02	9.13E-07	1.16E-01	6.56E-08	1.80E-07
Benzo(k)fluoranthene	1.06E-06	1.24E-02	1.80E-07	2.14E-01	9.66E-07	2.30E-01	6.37E-08	1.71E-07
Bis(2-ethylhexyl) phthalate	1.68E-04	1.85E-01	6.78E-04	4.28E-02	4.79E-04	1.53E+00	6.37E-04	7.45E-04
Chrysene	1.49E-06	7.56E-03	7.41E-08	2.18E-01	5.74E-07	3.72E-01	2.94E-08	7.10E-08
Dibenzo(a,h)anthracene	1.23E-07	1.19E-03	1.97E-08	7.10E-03	9.61E-08	1.22E-02	6.91E-09	1.89E-08
Fluoranthene	1.16E-05	2.72E-02	1.46E-06	3.29E-01	5.52E-06	5.32E-01	5.21E-07	1.47E-06
Phenanthrene	2.63E-05	7.46E-03	2.61E-06	1.99E-01	8.50E-06	2.60E-01	9.60E-07	2.41E-06
Pyrene	2.31E-05	8.73E-03	5.35E-07	3.59E-01	3.45E-06	5.23E-01	4.90E-07	5.24E-07
Indeno(1,2,3-cd)Pyrene	4.61E-07	4.47E-03	7.37E-08	2.66E-02	3.60E-07	4.58E-02	2.59E-08	7.10E-08

Table 3-7. Reference Site (Puget Sound) Chemical Concentrations in Sediment Used in the Human Health Risk Assessment

COPC	Sediment (mg/kg)
Metals/Metalloids	
Arsenic	6.45E+00
Cadmium	3.92E-01
Copper	2.24E+01
Lead	6.31E+00
Mercury	N/AV
Nickel	5.22E+01
Zinc	4.74E+01
Organometallics	
Tributyltin	N/AV
Polychlorinated Biphenyls	
Aroclor 1016	N/AV
Aroclor 1221	N/AV
Aroclor 1232	N/AV
Aroclor 1242	N/AV
Aroclor 1248	N/AV
Aroclor 1254	N/AV
Aroclor 1260	N/AV
Total PCBs	N/AV
Semivolatile Organics	
1,4-Dichlorobenzene	N/AV
4-Methylphenol	N/AV
Benzo(a)anthracene	7.92E-03
Benzo(a)pyrene	7.79E-03
Benzo(b)fluoranthene	3.38E-02
Benzo(g,h,i)perylene	6.11E-03
Benzo(k)fluoranthene	1.18E-02
Bis(2-ethylhexyl)phthalate	
Chrysene	8.67E-03
Dibenzo(a,h)anthracene	7.67E-03
Fluoranthene	1.67E-02
Phenanthrene	2.10E-02
Pyrene	1.44E-02
Indeno(1,2,3-cd)Pyrene	7.89E-03

Table 3-8. Tissue Data with Associated Collection Locations Used in the Human Health Risk Assessment

Species	Tissue Available	Preparation	Location	Reference Site
Chinook and Coho Salmon	Fillet	Uncooked	Duwamish River and Elliott Bay	Apple Cove Point, Budd Inlet (chinook only), Central Sound, Colvos Passage (coho only), Deschutes River, Nisqually River, Nooksack River, Sinclair Inlet, Skagit River, South Sound
Rockfish	Fillet	Uncooked	Elliott Bay	Hood Canal
English Sole	Fillet and whole body	Uncooked and cooked	Elliott Bay	Port Susan
Shiner Perch	Whole body	Uncooked	Duwamish River	Port Susan
Crab	Meat and hepatopancreas	Uncooked and cooked	Duwamish River and Elliott Bay	Port Susan
Mussels	Meat	Uncooked	Duwamish River and Elliott Bay	Totten Inlet
Squid	Meat and whole body	Uncooked	Elliott Bay	None
Prawns	Meat	Uncooked	Elliott Bay	Port Susan

In general, the tissue most frequently consumed (e.g., fillet) was included in the assessment, with the exception of shiner perch, for which only whole body data were available. In addition, whole body English sole[13] chemical concentrations, and crab hepatopancreas were analyzed. Chemical concentrations were analyzed in all tissues uncooked and in cooked English sole fillets, whole body English sole, crab meat, and crab hepatopancreas. Tissue data are presented on the WQA homepage at: http://splash/metrokc.gov/wlr/waterres/wqa/wqapage.htm.

[13] Whole body English sole concentrations were calculated as the weighted average of the fillet (assumed to be 30 percent of weight) and remainder (assumed to be 70 percent of weight) concentrations. Remainder concentrations were estimated as a composite from 20 fish of equal amounts of (1) skin, fins, and tail, (2) backbone, (3) head, jaws, and gills, and (4) kidney and gonads. Liver, blood and other fluids were samples separately and were not included in the composite sampling of the remainder.

Exposure concentrations in each tissue type were estimated as the 95th percentile upper confidence limit (UCL)[14] of the mean (U.S. EPA 1989a).

$$95UCL = C_{tissue} + (t_{tissue} \times SE_{tissue}) \qquad \text{Equation 3-1}$$

Where:

95UCL	=	95th upper confidence limit of the mean tissue concentration
T_{tissue}	=	t value, based on sample
SE_{tissue}	=	Standard error in tissue concentration

When only one sample was available, the observed concentration was used as the exposure concentrations for each COPC. In cases, where the COPC was undetected, it was assumed for the purposes of the risk assessment, that it was present at one-half the detection limit concentration. However, exposures and risks were not calculated unless a chemical was detected in tissues in at least one sample.

A thorough review of the quality assurance/quality control (QA/QC) analytical results was conducted to aid in the interpretation of the tissue data. For the majority of the COPCs in the majority of the samples, the data were found to be of sufficient quality to be used without qualification. For some chemicals in some tissue types, the QA/QC results indicated that the analytical results may under-estimate the actual concentrations. When this situation was observed, the analytical results were multiplied by a "adjustment factor". The adjustment factors were numerical values estimated from the QA/QC results to account for the worst possible under-estimation of the actual tissue concentrations. Adjustment factors and other QA/QC are described on the WQA homepage.

Baseline condition chemical concentrations in Duwamish River and Elliott Bay fish and shellfish used in the risk assessment are summarized in Table 3-9 and Table 3-10, respectively. Reference site chemical concentrations in fish and shellfish used in the risk assessment are also summarized in Table 3-11.

[14] The upper confidence limit of the mean captures the variability in the mean, by using an estimate of the upper bound of possible values of the true average tissue concentrations. Since the 95th UCL of the mean was used, the exposure concentrations may over-estimate the actual mean, but there is only one chance in twenty that the actual mean is greater the estimated exposure concentration.

Table 3-9. Baseline Condition Chemical Concentrations (mg/kg) in Duwamish River Seafood Used in the Human Health Risk Assessment

COPC	Raw English Sole	Count	# Detects	English Sole Carcass	Whole Body English Sole	Count	# Detects	Perch Whole Body	Count	# Detects	Salmon	Count	# Detects
Metals/Metalloids													
Arsenic	1.6E+0	3	3	6.0E-1	9.0E-1	3	3	1.6E-1	3	3	9.7E-2	36	36
Cadmium	4.0E-3	3	0	4.0E-3	4.0E-3	3	0	2.3E-2	3	3	N/AV	N/AV	N/AV
Copper	3.0E-1	3	3	4.3E-1	4.0E-1	3	3	2.5E+0	3	3	7.2E-1	36	36
Lead	1.0E-2	3	0	2.8E-1	2.0E-1	3	3	1.9E-1	3	3	1.6E-2	35	1
Mercury	8.6E-2	3	3	8.0E-2	8.2E-2	3	3	9.3E-2	3	3	8.2E-2	36	36
Nickel	1.0E-2	3	0	2.9E-1	2.1E-1	3	3	2.1E-1	3	3	N/AV	N/AV	N/AV
Zinc	8.4E+0	3	3	3.0E+1	2.4E+1	3	3	1.9E+1	3	3	N/AV	N/AV	N/AV
Organometallics													
Tributyltin	6.3E-3	3	3	2.1E-2	1.7E-2	3	3	2.1E-1	3	3	N/AV	N/AV	N/AV
Polychlorinated Biphenyls													
Aroclor 1016	2.7E-3	3	0	4.9E-2	3.5E-2	3	0	4.0E-3	3	0	N/AV	N/AV	N/AV
Aroclor 1221	2.7E-3	3	0	4.9E-2	3.5E-2	3	0	4.0E-3	3	0	N/AV	N/AV	N/AV
Aroclor 1232	2.7E-3	3	0	4.9E-2	3.5E-2	3	0	4.0E-3	3	0	N/AV	N/AV	N/AV
Aroclor 1242	2.7E-3	3	0	4.9E-2	3.5E-2	3	0	4.0E-3	3	0	N/AV	N/AV	N/AV
Aroclor 1248	2.7E-3	3	0	1.5E-1	1.0E-1	3	3	4.0E-3	3	0	1.4E-3	36	0
Aroclor 1254	2.8E-1	3	3	1.6E+0	1.2E+0	3	3	4.3E-1	3	3	3.3E-2	36	36
Aroclor 1260	1.8E-1	3	3	1.1E+0	8.4E-1	3	3	2.9E-1	3	3	2.4E-2	36	30
Total PCBs	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	5.9E-2	36	36
Semivolatile Organics													
1,4-Dichlorobenzene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	1.2E-2	3	0	N/AV	N/AV	N/AV
4-Methylphenol	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.0E-2	3	0	N/AV	N/AV	N/AV
Benzo(a)anthracene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	1.2E-2	3	0	N/AV	N/AV	N/AV
Benzo(a)pyrene	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.0E-2	3	0	N/AV	N/AV	N/AV
Benzo(b)fluoranthene	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	3.2E-2	3	0	N/AV	N/AV	N/AV
Benzo(g,h,i)perylene	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.0E-2	3	0	N/AV	N/AV	N/AV
Benzo(k)fluoranthene	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	3.2E-2	3	0	N/AV	N/AV	N/AV
Bis(2-ethylhexyl)phthalate	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	1.2E-2	3	0	7.4E-1	36	8
Chrysene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	1.2E-2	3	0	N/AV	N/AV	N/AV
Dibenzo(a,h)anthracene	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	3.2E-2	3	0	N/AV	N/AV	N/AV
Fluoranthene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	1.2E-2	3	0	N/AV	N/AV	N/AV
Phenanthrene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	1.2E-2	3	0	N/AV	N/AV	N/AV
Pyrene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	1.2E-2	3	0	N/AV	N/AV	N/AV
Indeno(1,2,3-cd)Pyrene	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.0E-2	3	0	N/AV	N/AV	N/AV

Table 3-9. Baseline Condition Chemical Concentrations (mg/kg) in Duwamish River Seafood Used in the Human Health Risk Assessment (continued)

COPC	Crab Hepato-pancreas (Raw)	Count	# Detects	Dungeness Crab	Count	# Detects	Mussels	Count	# Detects	Cooked Sole	Count	# Detects	Cooked Crab	Count	# Detects
Metals/Metalloids															
Arsenic	7.0E-1	1	1	2.6E+0	2	2	9.4E-2	29	29	2.3E+0	3	3	9.1E-1	2	2
Cadmium	1.1E-1	1	1	4.9E-2	2	2	5.4E-1	29	29	4.0E-3	3	0	8.0E-2	2	2
Copper	4.3E+1	1	1	2.2E+1	2	2	1.4E+0	29	29	3.5E-1	3	3	2.6E+1	2	2
Lead	1.8E-1	1	1	2.5E-1	2	2	5.2E-1	29	29	1.0E-2	3	0	5.3E-2	2	2
Mercury	6.7E-2	1	1	1.7E-1	2	2	1.3E-2	29	29	1.4E-1	3	3	2.3E-1	2	2
Nickel	2.4E-1	1	1	3.0E-1	2	2	6.1E-1	29	29	3.3E-2	3	3	1.0E-1	2	2
Zinc	2.6E+1	1	1	7.0E+1	2	2	4.7E+1	29	29	8.8E+0	3	3	5.9E+1	2	2
Organometallics															
Tributyltin	5.9E-2	1	1	1.8E-1	2	2	7.3E-2	29	29	1.7E-2	3	3	8.9E-2	2	2
Polychlorinated Biphenyls															
Aroclor 1016	2.0E-2	1	0	2.7E-3	2	0	6.5E-3	29	0	6.5E-3	3	0	2.7E-3	2	0
Aroclor 1221	2.0E-2	1	0	2.7E-3	2	0	6.5E-3	29	0	6.5E-3	3	0	2.7E-3	2	0
Aroclor 1232	2.0E-2	1	0	2.7E-3	2	0	6.5E-3	29	0	6.5E-3	3	0	2.7E-3	2	0
Aroclor 1242	2.0E-2	1	0	2.7E-3	2	0	6.5E-3	29	0	6.5E-3	3	0	2.7E-3	2	0
Aroclor 1248	1.2E-1	1	1	2.6E-2	2	1	6.5E-3	29	0	3.2E-2	3	3	2.7E-3	2	0
Aroclor 1254	1.1E+0	1	1	2.0E-1	2	2	3.5E-2	29	21	5.3E-1	3	3	1.2E-1	2	2
Aroclor 1260	4.5E-1	1	1	5.1E-2	2	2	6.5E-3	29	0	3.1E-1	3	3	6.6E-2	2	2
Total PCBs	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Semivolatile Organics															
1,4-Dichlorobenzene	1.2E-2	1	0	8.0E-3	2	0	8.0E-3	29	0	1.3E-2	3	0	8.0E-3	2	0
4-Methylphenol	2.0E-2	1	0	1.4E-2	2	0	2.9E-2	29	0	2.2E-2	3	0	1.4E-2	2	0
Benzo(a)anthracene	1.2E-2	1	0	8.0E-3	2	0	1.9E-2	29	14	1.3E-2	3	0	8.0E-3	2	0
Benzo(a)pyrene	2.0E-2	1	0	1.4E-2	2	0	1.4E-2	29	0	2.2E-2	3	0	1.4E-2	2	0
Benzo(b)fluoranthene	3.2E-2	1	0	2.2E-2	2	0	2.4E-2	29	1	3.5E-2	3	0	2.2E-2	2	0
Benzo(g,h,i)perylene	2.0E-2	1	0	1.4E-2	2	0	1.4E-2	29	0	2.2E-2	3	0	1.4E-2	2	0
Benzo(k)fluoranthene	3.2E-2	1	0	2.2E-2	2	0	2.2E-2	29	0	3.5E-2	3	0	2.2E-2	2	0
Bis(2-ethylhexyl)phthalate	1.2E-2	1	0	8.0E-3	2	0	4.2E-2	29	2	1.3E-2	3	0	8.0E-3	2	0
Chrysene	1.2E-2	1	0	8.0E-3	2	0	3.2E-2	29	16	1.3E-2	3	0	8.0E-3	2	0
Dibenzo(a,h)anthracene	3.2E-2	1	0	2.2E-2	2	0	2.2E-2	29	0	3.5E-2	3	0	2.2E-2	2	0
Fluoranthene	3.2E-2	1	0	8.0E-3	2	0	5.1E-2	29	28	1.3E-2	3	0	8.0E-3	2	0
Phenanthrene	1.2E-2	1	0	8.0E-3	2	0	1.5E-2	29	3	1.3E-2	3	0	8.0E-3	2	0
Pyrene	1.2E-2	1	0	8.0E-3	2	0	3.6E-2	29	17	1.3E-2	3	0	8.0E-3	2	0
Indeno(1,2,3-cd)Pyrene	2.0E-2	1	0	1.4E-2	2	0	1.4E-2	29	0	2.2E-2	3	0	1.4E-2	2	0

N/AV = Not Available

Table 3-10. Baseline Condition Chemical Concentrations (mg/kg) in Seafood from Elliott Bay Used in the Human Health Risk Assessment

COPC	English Sole	Count	# Detects	Eng. Sole Carcass	Whole Body Eng. Sole	Count	# Detects	Raw Rock Fish	Count	# Detects	Perch – Whole body	Count	# Detects	Cooked Sole	Count	# Detects
Metals/Metalloids																
Arsenic	8.9E-1	3	3	6.6E-1	7.3E-1	3	3	1.3E-1	3	3	1.2E-1	3	3	1.2E+0	3	3
Cadmium	4.0E-3	3	0	4.0E-3	4.0E-3	3	0	4.0E-3	3	0	3.3E-2	3	3	4.0E-3	3	0
Copper	2.6E-1	3	3	4.6E-1	4.0E-1	3	3	2.2E-1	3	3	7.8E-1	3	3	2.6E-1	3	3
Lead	1.0E-2	3	0	1.8E-1	1.3E-1	3	3	1.0E-2	3	0	1.7E-1	3	3	1.0E-2	3	0
Mercury	1.1E-1	3	3	7.0E-2	8.1E-2	3	3	7.1E-1	3	3	3.1E-2	3	3	1.5E-1	3	3
Nickel	1.0E-2	3	0	2.0E-1	1.5E-1	3	3	1.0E-2	3	0	2.6E-1	3	3	3.5E-2	3	3
Zinc	7.7E+0	3	3	3.3E+1	2.5E+1	3	3	2.9E+0	3	3	1.8E+1	3	3	7.0E+0	3	3
Organometallics																
Tributyltin	2.4E-3	3	3	1.9E-2	1.4E-2	3	3	5.4E-2	3	3	2.0E-1	3	3	4.1E-3	3	3
Polychlorinated Biphenyls																
Aroclor 1016	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	4.4E-3	3	0
Aroclor 1221	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	4.4E-3	3	0
Aroclor 1232	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	4.4E-3	3	0
Aroclor 1242	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	4.4E-3	3	0
Aroclor 1248	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	4.4E-3	3	0
Aroclor 1254	1.5E-2	3	1	1.8E-1	1.3E-1	3	3	6.8E-2	3	1	1.9E-1	3	3	7.1E-2	3	3
Aroclor 1260	1.3E-2	3	1	2.7E-1	2.0E-1	3	3	1.3E-1	3	3	1.1E-1	3	3	6.5E-2	3	3
Total PCBs	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Semivolatile Organics																
1,4-Dichlorobenzene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	8.0E-3	3	0
4-Methylphenol	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	1.4E-2	3	0	2.0E-2	3	0	1.4E-2	3	0
Benzo(a)anthracene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	8.0E-3	3	0
Benzo(a)pyrene	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	1.4E-2	3	0	4.9E-2	3	1	1.4E-2	3	0
Benzo(b)fluoranthene	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.2E-2	3	0	3.2E-2	3	0	2.2E-2	3	0
Benzo(g,h,i)perylene	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	1.4E-2	3	0	2.0E-2	3	0	1.4E-2	3	0
Benzo(k)fluoranthene	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.2E-2	3	0	3.2E-2	3	0	2.2E-2	3	0
Bis(2-ethylhexyl)phthalate	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	8.0E-3	3	0
Chrysene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	8.0E-3	3	0
Dibenzo(a,h)anthracene	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.2E-2	3	0	3.2E-2	3	0	2.2E-2	3	0
Fluoranthene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.1E-1	3	2	8.0E-3	3	0
Phenanthrene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.0E-1	3	2	8.0E-3	3	0

Pyrene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.3E-1	3	2	8.0E-3	3	0
Indeno(1,2,3-cd)Pyrene	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	1.4E-2	3	0	2.0E-2	3	0	1.4E-2	3	0

Table 3-10. Baseline Condition Chemical Concentrations (mg/kg) in Seafood from Elliott Bay Used in the Human Health Risk Assessment (continued)

COPC	Crab Hepato. (Raw)	Count	# Detects	Dungeness Crab	Count	# Detects	Mussels	Count	# Detects	Prawns	Count	# Detects	Whole Squid	Count	# Detects	Cooked Crab	Count	# Detects	Cooked Crab Hepato	Count	# Detects
Metals/Metalloids																					
Arsenic	8.0E-1	1	1	2.1E+0	4	4	1.2E-1	3	3	2.3E+0	1	1	5.6E-1	3	3	1.8E+0	3	3	5.4E-1	2	2
Cadmium	1.7E+0	1	1	5.2E-1	4	4	7.1E-1	3	3	1.3E-2	1	1	1.8E-1	3	3	2.2E-1	3	3	3.5E+0	2	2
Copper	2.9E+1	1	1	2.0E+1	4	4	1.3E+0	3	3	5.8E+0	1	1	5.3E+1	3	3	2.6E+1	3	3	4.5E+1	2	2
Lead	1.3E-1	1	1	3.5E-1	4	4	9.0E-1	3	3	1.0E-2	1	0	1.0E-2	3	0	8.0E-2	3	3	3.0E-1	2	2
Mercury	5.4E-2	1	1	1.3E-1	4	4	1.5E-2	3	3	5.2E-2	1	1	5.2E-2	3	3	4.4E-1	3	3	5.0E-2	2	2
Nickel	4.3E-1	1	1	1.3E-1	4	4	9.2E-1	3	3	2.6E-2	1	1	1.0E-2	3	0	1.6E-1	3	3	3.4E-1	2	2
Zinc	4.9E+1	1	1	6.7E+1	4	4	5.2E+1	3	3	1.1E+1	1	1	1.7E+1	3	3	8.5E+1	3	3	6.0E+1	2	2
Organometallics																					
Tributyltin	6.1E-2	1	1	8.9E-2	4	4	5.9E-2	3	3	6.9E-2	1	1	3.8E-2	3	3	1.2E-1	3	3	8.4E-2	2	2
Polychlorinated Biphenyls																					
Aroclor 1016	2.0E-2	1	0	3.8E-3	4	0	6.5E-3	3	0	4.0E-3	1	0	4.0E-3	3	0	1.0E-2	3	0	2.0E-2	2	0
Aroclor 1221	2.0E-2	1	0	3.8E-3	4	0	6.5E-3	3	0	4.0E-3	1	0	4.0E-3	3	0	1.0E-2	3	0	2.0E-2	2	0
Aroclor 1232	2.0E-2	1	0	3.8E-3	4	0	6.5E-3	3	0	4.0E-3	1	0	4.0E-3	3	0	1.0E-2	3	0	2.0E-2	2	0
Aroclor 1242	2.0E-2	1	0	3.8E-3	4	0	6.5E-3	3	0	4.0E-3	1	0	4.0E-3	3	0	1.0E-2	3	0	2.0E-2	2	0
Aroclor 1248	2.0E-2	1	0	3.8E-3	4	0	6.5E-3	3	0	4.0E-3	1	0	4.0E-3	3	0	1.0E-2	3	0	2.0E-2	2	0
Aroclor 1254	9.6E-1	1	1	1.5E-1	4	3	6.5E-3	3	0	4.0E-3	1	0	3.1E-2	3	3	5.1E-2	3	3	6.9E-1	2	2
Aroclor 1260	1.0E+0	1	1	1.1E-1	4	3	6.5E-3	3	0	1.4E-2	1	1	4.0E-3	3	0	3.8E-2	3	3	6.8E-1	2	2
Total PCBs	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Semivolatile Organics																					
1,4-Dichlorobenzene	1.2E-2	1	0	1.1E-2	4	0	8.0E-3	3	0	1.2E-2	1	0	1.2E-2	3	0	3.0E-2	3	0	1.2E-2	2	0
4-Methylphenol	2.0E-2	1	0	1.9E-2	4	0	2.9E-2	3	0	2.0E-2	1	0	2.0E-2	3	0	5.0E-2	3	0	2.0E-2	2	0
Benzo(a)anthracene	1.2E-2	1	0	3.7E-2	4	1	3.2E-2	3	1	1.2E-2	1	0	1.2E-2	3	0	3.0E-2	3	0	1.2E-2	2	0
Benzo(a)pyrene	2.0E-2	1	0	3.8E-2	4	1	1.4E-2	3	0	2.0E-2	1	0	2.0E-2	3	0	5.0E-2	3	0	2.0E-2	2	0
Benzo(b)fluoranthene	3.2E-2	1	0	3.0E-2	4	0	2.2E-2	3	0	3.2E-2	1	0	3.2E-2	3	0	7.8E-2	3	0	3.2E-2	2	0
Benzo(g,h,i)perylene	2.0E-2	1	0	1.9E-2	4	0	1.4E-2	3	0	2.0E-2	1	0	2.0E-2	3	0	5.0E-2	3	0	2.0E-2	2	0
Benzo(k)fluoranthene	3.2E-2	1	0	3.0E-2	4	0	2.2E-2	3	0	3.2E-2	1	0	3.2E-2	3	0	7.8E-2	3	0	3.2E-2	2	0
Bis(2-ethylhexyl)phthalate	1.2E-2	1	0	1.1E-2	4	0	1.3E-2	3	0	5.3E-2	1	1	1.2E-1	3	2	3.0E-2	3	0	1.2E-2	2	0
Chrysene	1.2E-2	1	0	3.5E-2	4	1	3.1E-2	3	1	1.2E-2	1	0	1.2E-2	3	0	3.0E-2	3	0	1.2E-2	2	0
Dibenzo(a,h)anthracene	3.2E-2	1	0	3.0E-2	4	0	2.2E-2	3	0	3.2E-2	1	0	3.2E-2	3	0	7.8E-2	3	0	3.2E-2	2	0
Fluoranthene	1.2E-2	1	0	9.7E-2	4	2	5.7E-2	3	3	1.2E-2	1	0	1.2E-2	3	0	3.2E-2	3	1	1.2E-2	2	0
Phenanthrene	1.2E-2	1	0	2.0E-1	4	2	3.0E-2	3	3	1.2E-2	1	0	1.2E-2	3	0	1.6E-1	3	1	2.3E-1	2	1
Pyrene	1.2E-2	1	0	8.0E-2	4	2	4.6E-2	3	3	1.2E-2	1	0	1.2E-2	3	0	3.0E-2	3	0	1.2E-2	2	0

Indeno(1,2,3-cd)Pyrene	2.0E-2	1	0	1.9E-2	4	0	1.4E-2	3	0	2.0E-2	1	0	2.0E-2	3	0	5.0E-2	3	0	2.0E-2	2	0

N/AV = Not Available

Table 3-11. Reference Site Chemical Concentrations (mg/kg) in Seafood Used in the Human Health Risk Assessment

COPC	English Sole (Port Susan)	Count	# Detects	Eng. Sole Carcass	Whole Body Eng. Sole	Count	# Detects	Raw Rock Fish	Count	# Detects	Perch – Whole body	Count	# Detects	Salmon	Count	# Detects	Cooked Sole	Count	# Detects
Metals/Metalloids																			
Arsenic	1.3E+0	3	3	7.3E-1	8.9E-1	3	3	2.7E-1	3	3	1.1E-1	3	3	8.3E-01	168	158	1.8E+0	3	3
Cadmium	4.0E-3	3	0	4.0E-3	4.0E-3	3	0	4.0E-3	3	0	3.5E-2	3	3	N/AV	N/AV	N/AV	4.0E-3	3	0
Copper	1.7E-1	3	3	4.5E-1	3.7E-1	3	3	2.0E-1	3	3	8.4E-1	3	3	5.8E-01	168	168	2.9E-1	3	3
Lead	1.0E-2	3	0	6.3E-2	4.7E-2	3	3	1.0E-2	3	0	6.8E-2	3	3	1.5E-02	168	10	1.0E-2	3	0
Mercury	7.8E-2	3	3	7.7E-2	7.7E-2	3	3	8.8E-1	3	3	1.3E-1	3	3	7.4E-02	178	178	1.3E-1	3	3
Nickel	1.0E-2	3	0	2.7E-1	1.9E-1	3	3	1.0E-2	3	0	2.0E-1	3	3	N/AV	N/AV	N/AV	3.7E-2	3	3
Zinc	6.7E+0	3	3	2.3E+1	1.8E+1	3	3	2.6E+0	3	3	2.1E+1	3	3	N/AV	N/AV	N/AV	6.4E+0	3	3
Organometallics																			
Tributyltin	4.0E-4	3	3	1.6E-3	1.2E-3	3	3	1.2E-2	3	3	5.7E-2	3	3	N/AV	N/AV	N/AV	8.5E-4	3	3
Polychlorinated Biphenyls																			
Aroclor 1016	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	N/AV	N/AV	N/AV	4.4E-3	3	0
Aroclor 1221	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	N/AV	N/AV	N/AV	4.4E-3	3	0
Aroclor 1232	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	N/AV	N/AV	N/AV	4.4E-3	3	0
Aroclor 1242	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	N/AV	N/AV	N/AV	4.4E-3	3	0
Aroclor 1248	2.7E-3	3	0	8.5E-3	6.7E-3	3	0	2.7E-3	3	0	4.0E-3	3	0	1.4E-03	344	1	4.4E-3	3	0
Aroclor 1254	2.7E-3	3	0	2.5E-2	1.8E-2	3	2	2.7E-3	3	0	8.2E-2	3	3	2.8E-02	344	330	2.0E-2	3	2
Aroclor 1260	2.7E-3	3	0	2.5E-2	1.8E-2	3	0	2.7E-3	3	0	3.0E-2	3	3	1.5E-02	344	316	4.4E-3	3	0
Total PCBs	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	4.4E-02	344	334	N/AV	N/AV	N/AV
Semivolatile Organics																			
1,4-Dichlorobenzene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	N/AV	N/AV	N/AV	8.0E-3	3	0
4-Methylphenol	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	1.4E-2	3	0	2.0E-2	3	0	N/AV	N/AV	N/AV	1.4E-2	3	0
Benzo(a)anthracene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	N/AV	N/AV	N/AV	8.0E-3	3	0
Benzo(a)pyrene	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	1.4E-2	3	0	2.0E-2	3	0	N/AV	N/AV	N/AV	1.4E-2	3	0
Benzo(b)fluoranthene	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.2E-2	3	0	3.2E-2	3	0	N/AV	N/AV	N/AV	2.2E-2	3	0
Benzo(g,h,I)perylene	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	1.4E-2	3	0	2.0E-2	3	0	N/AV	N/AV	N/AV	1.4E-2	3	0
Benzo(k)fluoranthene	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.2E-2	3	0	3.2E-2	3	0	N/AV	N/AV	N/AV	2.2E-2	3	0
Bis(2-ethylhexyl)phthalate	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	2.0E-01	168	16	8.0E-3	3	0
Chrysene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	N/AV	N/AV	N/AV	8.0E-3	3	0
Dibenzo(a,h)anthracene	2.2E-2	3	0	N/AV	N/AV	N/AV	N/AV	2.2E-2	3	0	3.2E-2	3	0	N/AV	N/AV	N/AV	2.2E-2	3	0
Fluoranthene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	N/AV	N/AV	N/AV	8.0E-3	3	0
Phenanthrene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	N/AV	N/AV	N/AV	8.0E-3	3	0

Pyrene	8.0E-3	3	0	N/AV	N/AV	N/AV	N/AV	8.0E-3	3	0	1.2E-2	3	0	N/AV	N/AV	N/AV	8.0E-3	3	0
Indeno(1,2,3-cd)Pyrene	1.4E-2	3	0	N/AV	N/AV	N/AV	N/AV	1.4E-2	3	0	2.0E-2	3	0	N/AV	N/AV	N/AV	1.4E-2	3	0

Table 3-11. Reference Site Chemical Concentrations (mg/kg) in Seafood Used in the Human Health Risk Assessment (continued)

COPC	Crab Hepato-pancreas (Raw)	Count	# Detects	Dungeness Crab	Count	# Detects	Mussels (totten Inlet-baseline-predeployment deployment)	Count	# Detects	Prawns	Count	# Detects	Cooked Crab	Count	# Detects	Cooked Crab Hepatopan creas	Count	# Detects
Metals/Metalloids																		
Arsenic	2.4E-1	1	1	4.2E-1	3	3	8.8E-2	3	3	2.6E+0	2	2	7.8E-1	3	3	1.2E+0	1	1
Cadmium	1.0E+0	1	1	1.5E-1	3	3	4.2E-1	3	3	1.0E-1	2	2	3.4E-1	3	3	1.0E+0	1	1
Copper	5.4E+1	1	1	1.4E+1	3	3	9.4E-1	3	3	7.8E+0	2	2	2.1E+1	3	3	5.3E+1	1	1
Lead	2.5E-2	1	1	4.6E-2	3	2	4.5E-2	3	3	1.3E-2	2	0	1.0E-2	3	0	3.6E-2	1	1
Mercury	5.9E-2	1	1	7.7E-2	3	3	5.6E-3	3	2	2.9E-1	2	2	2.0E-1	3	3	8.9E-2	1	1
Nickel	1.0E-1	1	1	4.2E-1	3	3	7.2E-1	3	3	8.9E-2	2	1	1.5E-1	3	3	2.6E-1	1	1
Zinc	1.6E+1	1	1	5.5E+1	3	3	9.2E+0	3	3	1.2E+1	2	2	6.0E+1	3	3	3.7E+1	1	1
Organometallics																		
Tributyltin	1.3E-3	1	1	3.3E-3	3	3	3.8E-3	3	3	1.7E-2	2	2	4.1E-3	3	3	4.7E-3	1	1
Polychlorinated Biphenyls																		
Aroclor 1016	4.0E-3	1	0	2.7E-3	3	0	6.5E-3	3	0	2.7E-3	2	0	2.7E-3	3	0	4.0E-3	1	0
Aroclor 1221	4.0E-3	1	0	2.7E-3	3	0	6.5E-3	3	0	2.7E-3	2	0	2.7E-3	3	0	4.0E-3	1	0
Aroclor 1232	4.0E-3	1	0	2.7E-3	3	0	6.5E-3	3	0	2.7E-3	2	0	2.7E-3	3	0	4.0E-3	1	0
Aroclor 1242	4.0E-3	1	0	2.7E-3	3	0	6.5E-3	3	0	2.7E-3	2	0	2.7E-3	3	0	4.0E-3	1	0
Aroclor 1248	4.0E-3	1	0	2.7E-3	3	0	6.5E-3	3	0	2.7E-3	2	0	2.7E-3	3	0	4.0E-3	1	0
Aroclor 1254	9.8E-2	1	1	2.7E-3	3	0	6.5E-3	3	0	2.7E-3	2	0	1.7E-2	3	3	1.7E-1	1	1
Aroclor 1260	7.0E-2	1	1	2.7E-3	3	0	6.5E-3	3	0	2.7E-3	2	0	2.7E-3	3	0	1.1E-1	1	1
Total PCBs	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Semivolatile Organics																		
1,4-Dichlorobenzene	1.2E-2	1	0	8.0E-3	3	0	8.0E-3	3	0	8.0E-3	2	0	8.0E-3	3	0	8.0E-3	1	0
4-Methylphenol	2.0E-2	1	0	1.4E-2	3	0	2.9E-2	3	0	1.4E-2	2	0	1.4E-2	3	0	4.0E-2	1	0
Benzo(a)anthracene	1.2E-2	1	0	8.0E-3	3	0	8.0E-3	3	0	8.0E-3	2	0	8.0E-3	3	0	2.4E-2	1	0
Benzo(a)pyrene	2.0E-2	1	0	8.0E-3	3	0	1.4E-2	3	0	1.4E-2	2	0	2.2E-2	3	0	4.0E-2	1	0
Benzo(b)fluoranthene	3.2E-2	1	0	2.2E-2	3	0	2.2E-2	3	0	2.2E-2	2	0	2.2E-2	3	0	6.5E-2	1	0
Benzo(g,h,i)perylene	2.0E-2	1	0	1.4E-2	3	0	1.4E-2	3	0	1.4E-2	2	0	1.4E-2	3	0	4.0E-2	1	0
Benzo(k)fluoranthene	3.2E-2	1	0	2.2E-2	3	0	2.2E-2	3	0	2.2E-2	2	0	2.2E-2	3	0	6.5E-2	1	0
Bis(2-ethylhexyl)phthalate	1.2E-2	1	0	8.0E-3	3	0	1.3E-2	3	0	5.2E-2	2	1	8.0E-3	3	0	2.4E-2	1	0
Chrysene	1.2E-2	1	0	8.0E-3	3	0	1.0E-2	3	0	8.0E-3	2	0	8.0E-3	3	0	2.4E-2	1	0
Dibenzo(a,h)anthracene	3.2E-2	1	0	2.2E-2	3	0	2.2E-2	3	0	2.2E-2	2	0	2.2E-2	3	0	6.5E-2	1	0
Fluoranthene	1.2E-2	1	0	8.0E-3	3	0	1.1E-2	3	0	8.0E-3	2	0	8.0E-3	3	0	2.4E-2	1	0
Phenanthrene	1.2E-2	1	0	8.0E-3	3	0	1.2E-2	3	0	8.0E-3	2	0	8.0E-3	3	0	2.4E-2	1	0

Pyrene	1.2E-2	1	0	8.0E-3	3	0	1.2E-2	3	0	8.0E-3	2	0	8.0E-3	3	0	2.4E-2	1	0
Indeno(1,2,3-cd)Pyrene	2.0E-2	1	0	1.4E-2	3	0	1.4E-2	3	0	1.4E-2	2	0	1.4E-2	3	0	4.0E-2	1	0

N/AV = Not available

Estimating Chemical Concentrations in Fish and Shellfish in the Without CSO Scenario. Fish and shellfish tissue concentrations were adjusted for the without CSO scenario using existing tissue concentration data and the results of the water and sediment quality model. To conduct this analysis, it was assumed that chemical concentrations in the fish and shellfish were in equilibrium with surface water and sediment COPCs.

Using the annual average chemical concentrations in water (for shiner perch, rockfish, mussels, and crabs) and sediment (for English sole) predicted by the water and sediment quality model, a concentration ratio of baseline conditions (with CSO discharges) to without CSO conditions was calculated. Tissue concentrations were assumed to be linearly related to chemical concentrations in surface water and sediment. Therefore, any changes in water and sediment concentrations should result in a corresponding change in tissue concentrations. Predicted chemical concentrations for the without CSO scenario were calculated using the annual average water concentrations over the entire study area. The 95th UCL baseline tissue concentrations were also used to estimate the future concentrations. Concentrations of each chemical in each tissue type except salmon,[15] after elimination of CSO discharges, were calculated by adjusting the average and variance in the tissue concentration based on changes in water concentrations. The adjusted 95th UCL was then calculated. First, the water concentrations in the total study area were calculated as a weighted mean as shown in Equation 3-2:

$$C_{study\ area-water} = \frac{(2{,}140 \times C_{Elliot\ Bay-water}) + (1{,}290 \times C_{Duwamish-water})}{3{,}490} \qquad \text{(Equation 3-2)}$$

Where:

$C_{study\ area-water}$	=	Weighted average study area water concentration, either baseline or without CSOs
2,140	=	Number of cells in Elliott Bay
$C_{Elliott\ Bay-water}$	=	Annual average water concentration in Elliott Bay patch, either baseline or without CSOs
1,290	=	Number of cells in Duwamish River
$C_{Duwamish-water}$	=	Annual average water concentration in Duwamish River patch, either baseline or without CSOs
3,490	=	Number of cells in Elliott Bay and Duwamish River

Once study area water (and sediment for English sole) concentrations were calculated, adjusted mean tissue concentrations were calculated according to Equation 3-3:

[15] As adult salmon most likely acquire any chemical tissue burdens outside of the study area, no adjustments were made to salmon tissues concentrations in the without CSO scenario.

$$C_{tissue-wthout\,CSO} = C_{tissue-baseline} \text{ x } \frac{C_{study\,area-without\,CSO}}{C_{study\,area-baseline}} \quad \text{(Equation 3-3)}$$

Where:

$C_{tissue\ without\ CSO}$	=	Mean concentration in tissue, without CSOs
$C_{tissue\ baseline}$	=	Mean concentration in tissue, baseline
$C_{study\ area\ without\ CSO}$	=	Weighted average study area water (or sediment for English Sole) concentration, without CSOs
$C_{study\ area\ baseline}$	=	Weighted average study area water (or sediment for English Sole) concentration, baseline

The adjusted standard errors were calculated according to Equations 3-4 and 3-5:

$$VAR_{tissue-without\,CSO} = VAR_{tissue-baseline} \text{ X } \left(\frac{C_{study\,area-without\,CSO}}{C_{study\,area-baseline}}\right)^2 \quad \text{(Equation 3-4)}$$

Where:

$VAR_{tissue\ without\ CSO}$	=	Variance in tissue concentrations, without CSOs
$VAR_{tissue\ baseline}$	=	Variance in tissue concentrations, baseline
$C_{study\ area\ without\ CSO}$	=	Weighted average study area water concentration, without CSOs
$C_{study\ area\ baseline}$	=	Weighted average study area water concentration, baseline

$$SE_{tissue-without\ CSO} = \sqrt{\frac{VAR_{tissue-without\ CSO}}{C_{study\,area\,-\,baseline}}} \quad \text{(Equation 3-5)}$$

Where:

$SE_{tissue\ -\ without\ CSO}$	=	Standard error in tissue concentrations, without CSOs
$VAR_{tissue\ -\ without\ CSO}$	=	Variance in tissue concentrations, without CSOs
n	=	Number of tissue samples

Finally, the adjusted 95th UCL on the mean was calculated using Equation 3-6 as follows:

$$95UCL = C_{\,tissue\,-\,without\,CSO} + (t \text{ x } SE_{tissue\,-\,without\,CSO}) \quad \text{(Equation 3-6)}$$

Where:

95UCL	=	95th UCL on the mean, without CSOs
$C_{tissue\ -\ without\ CSO}$	=	Mean concentration in tissue, without CSOs
$SE_{tissue\ -\ without\ CSO}$	=	Standard error in tissue concentrations, without CSOs

T = t value, based on sample size

Without CSO condition chemical concentrations in tissues for the Duwamish River, and Elliott Bay presented in Table 3-12 and Table 3-13, respectively.

Quantification of Exposure. Average daily chemical exposures were quantified for both adults and children aged 1 to 6, 7 to 12, and 13 to 18. Additionally, a range of exposure assumptions was used to evaluate all possible human exposures. This range included low, medium, and high estimates for each parameter, along with an estimate of the "average" chemical exposure each time the activity occurs (e.g., exposure per meal or recreational event).

Equations Used to Estimate Exposures from Ingestion of Seafood, Sediment, and Water. Chronic chemical exposures to people from seafood ingestion, and incidental ingestion of water and sediment, were calculated using Equations 3-7 and 3-8 (U.S. EPA 1989a). Equation 3-7 was used to estimate the average intake during the period of exposure, which was then used in the risk characterization to estimate non-carcinogenic risks. Equation 3-8 was used to estimate the intake averaged over the course of a lifetime, which was used in the risk characterization to estimate carcinogenic risks. Exposures were calculated for all four age groups: adults and children aged 1 to 6, 7 to 12, and 13 to 18, and for all exposure levels, low, medium, and high.

$$\text{Ingestion CDI} = \frac{\text{EEC} \times \text{IR} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}} \qquad \text{(Equation 3-7)}$$

$$\text{Ingestion LADI} = \frac{\text{EEC} \times \text{IR} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{L}} \qquad \text{(Equation 3-8)}$$

Where:

CDI	=	Chronic daily intake of chemical, mg/kg-day
LADI	=	Lifetime average daily intake of chemical, mg/kg-day
EEC	=	Expected exposure concentration in seafood (mg/kg), sediment (mg/kg) or water (mg/L)
IR	=	Ingestion rate of seafood (g/day), sediment (g/day), or water (mL/day)
EF	=	Exposure frequency to seafood, sediment, or water, day/yr
ED	=	Exposure duration, years
CF	=	Conversion factor, 1×10^{-3} kg/g (seafood and sediment), 1×10^{-3} L/ml (water)
BW	=	Body weight, kg
AT	=	Averaging time, years x 365 day/yr
L	=	Lifespan, years x 365 day/yr

Table 3-12. Without CSO Condition Chemical Concentrations in Duwamish River Seafood Used in the Human Health Risk Assessment

can be found in excel in human health chemical conc.xls [Duwamish River] needs 2 pages.

Table 3-12 Without CSO Condition Chemical Concentrations in Duwamish
(Continued)

Table 3-13 Without CSO Condition Chemical Concentrations in Seafood from Elliott Bay Used in the Human Health Risk Assessment

can be found in excel in human health chemical conc.xls [Elliott Bay] needs 3 pages.

Table 3-13. Without CSO Condition Chemical Concentrations in Seafood from Elliott Bay Used in the Human Health Risk Assessment. (Continued)

Table 3-13. Without CSO Condition Chemical Concentrations in Seafood from Elliott Bay Used in the Human Health Risk Assessment. (Continued)

Both the chronic daily intake (CDI) and the lifetime average daily intake (LADI) are expressed in units of mass of chemical taken into the body on a daily basis per unit of body weight (mg/kg-day). The CDI is an estimate of the average daily exposure during the exposure period, and is used to estimate potential non-carcinogenic risks. The LADI is an estimate of the average daily exposures averaged over an entire lifespan, and is used to estimate potential carcinogenic risks. As indicated in Equations 3-7 and 3-8, the level of exposure is directly proportional to the chemical concentration in the environment, the ingestion rate (how much each day), frequency (how many days per year) and duration (how many years) of exposure. In contrast, the level of exposure is inversely proportional to the body weight of the exposed individual (i.e., the dose decreases as weight increases) and the time over which the exposure is averaged (or the lifespan of the exposed individual when calculating the LADI.

Equations Used to Estimate Exposure from Dermal Contact with Surface Water. The methodology recommended by the U.S. EPA for assessing dermal exposures was used to quantify chemicals exposures to the skin (U.S. EPA 1992b). The U.S. EPA recently issued, then rescinded, more current guidance for assessing dermal exposures to water. Because the more recent guidance was rescinded, the 1992 guidance was used in this risk assessment. The methodologies presented are theoretically derived, and are subject to future revision by U.S. EPA. The procedures are "not official agency guidance, rather they represent the judgments of the authors and are offered as a starting point for Agency programs to adopt/modify in light of programmatic consideration" (U.S. EPA 1992b). Although, the procedures used are highly uncertain, it is believed that the procedures presented represent the best available methods for estimating dermal absorption. The U.S. EPA (1992b) analysis included a detailed overview of the mechanisms for dermal absorption, techniques for measuring dermal absorption, and mathematical description for dermal absorption. Two different procedures are recommended: one for inorganic chemicals and one for organic chemicals. Each procedure is discussed below.

To estimate the dose of inorganic chemicals received via dermal absorption, a steady-state equation was used. Steady-state conditions require that the rate of chemical absorption remains constant throughout the period of contact with the chemical. This simplification does not account for the period of time that exists between first contact with the chemical and the attainment of steady state. During this time, chemicals enter the skin at a faster rate than during steady-state conditions. Thus, the initial non-steady-state condition results in a buildup of chemical in the skin. Once steady state has been reached, the chemical enters and leaves the skin at equal rates. After dermal contact has ended, chemicals are no longer absorbed by the skin.

The intake of inorganic chemicals from water by skin dermal absorption was estimated using a steady-state approach. While this is a gross oversimplification of the absorption process, the steady-state equation is thought to provide an adequate approximation of the dose received via dermal absorption of inorganic chemicals. The skin intakes were estimated using Equation 3-9 below (U.S. EPA 1992b):

$$DA = K_p \times C_{sw} \times ET \times cf \qquad \text{(Equation 3-9)}$$

Where:

DA	=	Dose absorbed per unit area per day, mg/cm^2-day
K_p	=	Permeability coefficient from water, cm/hr
C_{sw}	=	Concentration of chemical in water, mg/L
ET	=	Adult and child exposure time while swimming, hr/day
cf	=	Conversion factor, 0.001 L/cm^3

While there have been some chemical-specific studies, it is not currently possible to accurately extrapolate absorption rates from one inorganic constituent to another based on chemical properties. For chemicals with available skin absorption data, chemical-specific permeability coefficients (Kp) were used. For inorganic chemicals with no available data, default Kp of 0.001 cm/hr was chosen (U.S. EPA 1992b). Inorganic chemical Kp values used in this risk assessment are presented in Table 3-14.

Table 3-14. Chemical Specific Parameters used in Dermal Exposure Assessment

COPC	Kp (cm/hr)	T (hr)	Tss (hr)	B (unitless)
Metals/Metalloids				
Arsenic	0.001	NA/P	NA/P	NA/P
Cadmium	0.001	NA/P	NA/P	NA/P
Copper	0.001	NA/P	NA/P	NA/P
Lead	0.000004	NA/P	NA/P	NA/P
Mercury	0.001	NA/P	NA/P	NA/P
Nickel	0.0001	NA/P	NA/P	NA/P
Zinc	0.0006	NA/P	NA/P	NA/P
Organometallics				
Tributyltin	0.005962	5.144424	18.21126	0.154882
Polychlorinated Biphenyls				
Aroclor 1016	0.987643	3.2783	15.48228	109.6478
Aroclor 1221	2.199379	1.472139	6.952401	109.6478
Aroclor 1232	1.422984	2.275353	10.7457	109.6478
Aroclor 1242	0.870362	3.72005	17.56851	109.6478
Aroclor 1248	0.54752	5.913556	27.92768	109.6478
Aroclor 1254	0.369488	8.762918	41.38423	109.6478

Table 3-14. Chemical Specific Parameters used in Dermal Exposure Assessment (continued)

COPC	Kp (cm/hr)	T (hr)	Tss (hr)	B (unitless)
Aroclor 1260	0.188278	17.19684	81.21474	109.6478
Total PCBs	0.369488	8.762918	41.38423	109.6478
Semivolatile Organics				
1,4-Dichlorobenzene	0.07324	0.689523	3.702738	0.312608
4-Methylphenol	0.018	0.398702	0.956886	0.009333
Benzo(a)anthracene	0.892072	2.151041	10.18297	52.48075
Benzo(a)pyrene	1.119648	3.013344	14.22923	116.1944
Benzo(b)fluoranthene	1.691317	3.013344	14.21633	207.7304
Benzo(e)pyrene	1.119648	3.013344	14.22923	116.1944
Benzo(g,h,i)perylene	3.241778	4.221325	19.89819	834.9619
Benzo(k)fluoranthene	2.091823	3.013344	14.21211	280.2206
Bis(2-ethylhexyl)phthalate	0.022522	21.22994	103.5529	7.413102
Chrysene	0.886258	2.151041	10.1834	51.9996
Dibenzo(a,h)anthracene	1.561421	4.341589	20.47494	310.456
Fluoranthene	0.550287	1.492962	7.144943	15.88953
Phenthrene	0.224153	1.065734	5.585153	2.789687
Pyrene	0.438707	1.492962	7.188555	11.54782
Indeno(1,2,3-cd)pyrene	4.34E+00	4.221325	19.89628	1258.925

N/AP –Not applicable

For organic chemicals, a theoretical non-steady-state equation was used (U.S. EPA 1992b) which accounts for the increased rate of dermal absorption during the initial non-steady-state condition. The equation used to predict the organic chemical dose received via dermal absorption depends on the length of the exposure relative to the time required to reach a steady state. The following two equations were used to estimate the dose received per event per exposed skin area for organic chemicals. Equations 3-10 and 3-11 below were used to estimate the dose of organic chemicals received for each recreational event. The equation used depends on Exposure time (ET) relative to the estimated time required to reach steady state (Tss). Chemical-specific estimates of (Kp), (Tss), (T), and (B) were obtained from the U.S. EPA (1992b). Chemical-specific Kp, Tss, and T values are presented in Table 3-14.

If $ET < t_{ss}$, then: $DA = 2 \times Kp \times C_{sw} \times cf \times \sqrt{\frac{6 \times T \times ET}{3.14}}$ (Equation 3-10)

If $ET > t_{ss}$, then:

$$DA = Kp \times C_{sw} \times cf \times \left[\left(\frac{ET}{1+B}\right) + \left(2 \times T \times \left[\frac{1+(3 \times B)}{1+B}\right]\right)\right] \quad \text{(Equation 3-11)}$$

Where:

ET	=	Exposure time for swimming, hr/day
Tss	=	Time chemical takes to reach steady-state
DA	=	Dose absorbed per unit area per day, mg/cm^2-day
Kp	=	Permeability coefficient from water, cm/hr
Csw	=	Concentration of chemical in surface water, mg/L
T	=	Lag time chemical takes to diffuse through skin, hr
B	=	Ratio of permeability coefficients of the chemical in the stratum corneum and the viable epidermis of the skin, dimensionless
Cf	=	Conversion factor, 0.001 L/cm^3

Once an estimate of the dose absorbed per unit of surface area was estimated the intakes (CDI and LADI) were derived as follows in Equations 3-12 and 3-13. The dermal CDIs and LADIs differ from those calculated by the ingestion pathway in one important respect. Since they are estimates of the amount of chemical that is absorbed across the skin into the blood, they are estimates of *absorbed* doses. In contrast, the ingestion intakes are estimates of the *administered* doses. That is the ingestion intakes do not account for the fraction absorbed across the intestinal tract and into the body for distribution to tissues and organs. Therefore, intake estimates by these two pathways are not directly comparable and are a source of uncertainty in the dermal risk assessment.

$$CDI_{WD} = \left[\frac{ES \times EF \times ED}{BW}\right] \times \left[\frac{DA}{AT}\right] \quad \text{(Equation 3-12)}$$

$$LADI_{WD} = \left[\frac{ES \times EF \times ED}{BW}\right] \times \left[\frac{DA}{L}\right] \quad \text{(Equation 3-13)}$$

Where:

CDI_{WD}	=	Chronic daily intake from dermal contact with surface water, mg/kg-day
$LADI_{WD}$	=	Lifetime average daily intake from dermal contact with surface water, mg/kg-day
DA	=	Dose absorbed per unit area per day, mg/cm2-day
ES	=	Exposed skin area, cm2
EF	=	Exposure frequency, days/yr
ED	=	Exposure duration, yr
BW	=	Body weight, kg

AT	=	Averaging time, yr x 365 day/yr
L	=	Lifespan, yr x 365 day/yr

Equations Used to Estimate Exposures from Dermal Contact with Sediment. The absorption of chemicals from sediment depends on many factors, including the amount of soil adhering to skin, the time the sediment is in contact with the skin, and the physical characteristics of the sediment and condition and age of the skin. For example, physical characteristics of the sediment, such as particle size and organic carbon content are site-specific and affect the degree to which chemicals are available for absorption. Data on the dermal absorption of chemicals from sediment are scarce. A theoretical model that estimates the dose received based on these variables has been proposed (U.S. EPA 1992b). This model has not been validated however, and may result in inaccurate estimates of exposure. The authors do not recommend using the theoretical model, and instead suggest a simpler model based on percent absorption of an applied dose. This recommendation appears prudent based on the high degree of uncertainty associated with each of the variables involved in the more complex model. Intakes from dermal exposure to chemicals in sediment were estimated according Equations 3-14 and 3-15 below:

$$\text{Dermal CDI}_{(\text{sediment})} = \frac{\text{CS x SA x AF x ABS x EF x ED x CF}}{\text{BW x AT}} \qquad \text{(Equation 3-14)}$$

$$\text{Dermal LADI}_{(\text{sediment})} = \frac{\text{CS x SA x AF x ABS x EF x ED x CF}}{\text{BW x L}} \qquad \text{(Equation 3-15)}$$

Where:

LADI	=	Lifetime average daily intake, mg/kg-day
CDI	=	Chronic daily intake, mg/kg-day
CS	=	Concentration of chemical in sediment, mg/kg
SA	=	Skin surface area available for contact, cm^2
AF	=	Sediment-to-skin loading rate, mg/cm^2-event
ABS	=	Chemical-specific absorption factor, unitless
EF	=	Event frequency, events/yr
ED	=	Exposure duration, yr
CF	=	Conversion factor, 1E-06 kg/mg
BW	=	Body weight, kg
AT	=	Averaging time, yr x 365 d/yr
L	=	Lifespan, yr x 365 d/yr

Human Exposure Assumptions Used to Estimate Exposures.

Seafood Consumption Pathway. To the extent practicable, site-specific data obtained from the 1997 fishing survey (Section 2 above) were used to estimate exposures. Assumptions derived from the survey include seafood exposure frequencies. The remaining parameters were derived using reasonable assumptions from the published

literature and using best professional judgement. The exposure parameters used are summarized in Tables 3-15 through 3-17 below.

The frequencies with which seafood was consumed from the study area were derived using the results of the fishing survey conducted in 1997 (Section 2 above). The 50th and 75th percentile seafood consumption frequencies from the survey data were used to estimate the "low" and "medium" seafood consumption rates, respectively. The maximum reported consumption frequency of 365 days per year was used to represent high exposures. However, a very small number of individuals reported this level of seafood consumption. Therefore, the majority of exposed individuals are expected to consume seafood from the study area at the medium level (75th percentile) or lower.

Table 3-15. General Human Health Exposure Assumptions

Exposure Parameter	Exposure Level			Units	Source
	Low Value	Medium Value	High Value		
Adult Body Weight	79	70	60	Kg	U.S. EPA (1991b)
Child age 1 to 6 Body Weight	18	17	15	Kg	U.S. EPA (1991b)
Child age 7 to 12 Body Weight	37	35	29	Kg	U.S. EPA (1991b)
Child age 13 to 18 Body Weight	64	59	52	Kg	U.S. EPA (1991b)
Adult Exposure Duration	9	33	75	Yrs	U.S. EPA (1991b)
Child Exposure Duration	6	6	6	Yrs	Best Professional Judgement
Adult Averaging Time for Non-carcinogens	9	33	75	Yrs	U.S. EPA (1996)
Child Averaging Time for Non-carcinogens	6	6	6	Yrs	Best Professional Judgement
Lifespan	75	75	75	Yrs	U.S. EPA (1996)

Table 3-16. Human Health Exposure Assumptions for Seafood Consumption Pathway

Exposure Parameter	Exposure Level			Units	Source
	Low Value	Medium Value	High Value		
Adult Fish Consumption Frequency	8	24	365	Meals/yr	See Section 2.3 above
Child 1 to 6 Fish Consumption Frequency	8	24	365	Meals/yr	See Section 2.3 above
Child 7 to 12 Fish Consumption Frequency	8	24	365	Meals/yr	See Section 2.3 above
Child 13 to 18 Fish Consumption Frequency	8	24	365	Meals/yr	See Section 2.3 above
Adult Fish Ingestion Rate	93	152	305	g/meal	U.S. EPA (1991b)
Adult Shellfish Ingestion Rate	93	152	305	g/meal	U.S. EPA (1996)
Adult Crab Hepatopancreas Ingestion Rate	36	55	91	g/meal	Best Professional Judgement
Child 1 to 6 Fish Ingestion Rate	57	85	170	g/meal	U.S. EPA (1996)
Child 7 to 12 Fish Ingestion Rate	76	112	212	g/meal	U.S. EPA (1996)
Child 13 to 18 Fish Ingestion Rate	82	124	229	g/meal	U.S. EPA (1996)
Child 1 to 6 Shellfish Ingestion Rate	57	85	170	g/meal	U.S. EPA (1996)
Child 7 to 12 Shellfish Ingestion Rate	76	112	212	g/meal	U.S. EPA (1996)
Child 13 to 18 Shellfish Ingestion Rate	82	124	229	g/meal	U.S. EPA (1996)
Child 1 to 6 Crab Hepatopancreas Ingestion Rate	18	36	55	g/meal	Best Professional Judgement
Child 7 to 12 Crab Hepatopancreas Ingestion Rate	18	36	55	g/meal	Best Professional Judgement
Child 13 to 18 Crab Hepatopancreas Ingestion Rate	18	36	55	g/meal	Best Professional Judgement

Table 3-17. Human Health Exposure Assumptions for the Direct Pathways

Exposure Parameter	Exposure Level			Units	Source
	Low Value	Medium Value	High Value		
General Exposure Pathways					
Adult Incidental Water Ingestion Rate	25	50	75	ml/hr	U.S. EPA (1991b)
Adult Incidental Sediment Ingestion Rate	25	50	75	mg/event	U.S. EPA (1988)
Adult Sediment Deposition Rate to Skin	0.16	0.036	0.66	mg/sq.cm	U.S. EPA (1991b)
Child Sediment Deposition Rate	5	16	25	mg/soc.	U.S. EPA (1996)
Absorption Fraction from Sediment for Inorganics	0.001	0.005	0.01	unitless	U.S. EPA (1992)
Absorption Fraction from Sediment for Organics	0.01	0.05	0.1	unitless	U.S. EPA (1992)
Net Fisher Scenario					
Adult Net Fishing Frequency	2	24	91	event/yr	U.S. EPA (1988); Best Professional Judgement
Adult Net Fishing Event Time	0.17	1	2.6	hr/event	U.S. EPA (1988); Best Professional Judgement
Adult Skin Surface Area Exposed to Water	4,900	19,400	21,800	sq. cm	U.S. EPA (1996)
Adult Skin Surface Area Exposed to Sediment	4,900	9,300	17,000	sq. cm	U.S. EPA (1996)
Scuba Diver Scenario					
Adult Scuba Diving Frequency	2	12	24	event/yr	U.S. EPA (1988); Best Professional Judgement
Adult Scuba Diving Event Time	0.17	1	2.6	hr/event	U.S. EPA (1988); Best Professional Judgement
Adult Skin Surface Area Exposed to Water	4,900	19,400	21,800	sq. cm	U.S. EPA (1996)
Wind Surfer Scenario					
Adult Wind Surfing Frequency	2	12	24	event/yr	U.S. EPA (1988); Best Professional Judgement

Table 3-17. Human Health Exposure Assumptions for the Direct Pathways (continued)

Exposure Parameter	Exposure Level			Units	Source
	Low Value	Medium Value	High Value		
Adult Wind Surfing Event Time	0.17	1	2.6	hr/event	U.S. EPA (1988); Best Professional Judgement
Adult Skin Surface Area Exposed to Water	4,900	19,400	21,800	sq. cm	U.S. EPA (1996)
Swimming Scenario					
Adult Swimming Frequency	2	12	24	event/yr	U.S. EPA (1996); Best Professional Judgement
Child Swimming Frequency	2	12	24	event/yr	U.S. EPA (1996); Best Professional Judgement
Adult Swimming Event Time	0.17	1	2.6	hr/event	U.S. EPA (1988); Best Professional Judgement
Child Swimming Event Time	0.25	1	2.6	hr/event	U.S. EPA (1988); Best Professional Judgement
Adult Skin Surface Area Exposed to Water	4,900	19,400	21,800	sq. cm	U.S. EPA (1991b)
Child Age 3 to 6 Skin Surface Area Exposed to Water	6,200	7,200	8,400	sq. cm	U.S. EPA (1991b)
Child Age 7 to 12 Skin Surface Area Exposed to Water	9,000	10,400	12,500	sq. cm	U.S. EPA (1991b)
Child Age 13 to 18 Skin Surface Area Exposed to Water	13,800	15,800	18,400	sq. cm	U.S. EPA (1991b)
Adult Skin Surface Area Exposed to Sediment	4,900	9,300	17,000	sq. cm	U.S. EPA (1991b)
Child Age 3 to 6 Skin Surface Area Exposed to Sediment	6,200	7,200	8,400	sq. cm	U.S. EPA (1991b)
Child Age 7 to 12 Skin Surface Area Exposed to Sediment	9,000	10,400	12,500	sq. cm	U.S. EPA (1991b)
Child Age 13 to 18 Skin Surface Area Exposed to Sediment	12,700	14,500	16,900	sq. cm	U.S. EPA (1991b)

Estimates of meal size of fish and shellfish were used to estimate consumption rates on days that seafood was consumed. Meal sizes were derived from the published literature (U.S. EPA 1996).

The consumption rate for hepatopancreas was estimated using the measured mass of crab hepatopancreas (King County unpublished data) and professional judgement. The low, medium, and high consumption rates were estimated as the mass of one small (36 g), one large (55 g) and two average (total 91 g) sized hepatopancreas, respectively.

General Exposure Parameters. The exposure duration was assumed to range from 9 to 75 years for adults. The low and medium estimates of nine and 33 years are the average and 95th percentile estimate of residency time in the U.S. (U.S. EPA 1996). The high exposure estimate is equivalent to the estimate of lifespan for a U.S. resident. The length of the exposure durations in the medium and high exposure scenarios are intended to account for the possibility that an individual may change residences and still continue to fish in Elliott Bay or the Duwamish River. For each of the child age groups evaluated, the exposure duration was assumed equal to the entire duration of childhood (i.e., 6 years for each age group).

Body weights were derived from the literature (U.S. EPA 1996). The low, medium, and high exposure body weights are the reported 75th, average, and 25th percentile estimates of the average combined male and female body weights for U.S. residents aged 18 to 74. The 75th percentile estimate (79 kg) was used to represent the low exposure group, because of the inverse relationship between body weight and dose. That is, the greater an individuals body weight, the lower a chemical dose they would receive on a mass per unit of body weight basis. Similarly, body weight estimates for each of the three (1 to 6, 7 to 12, and 13 to 18) child age groups were derived as the 75th (low), average (medium), and 25th (high) percentile estimate of body weights.

The intakes estimated for non-carcinogens were averaged over the period during which exposure was assumed to occur. Therefore, averaging times are set to be equal to the exposure duration for both adults and children. When calculating the LADI to assess potential carcinogenic effects, the intake is averaged over the lifespan estimate of 75 years.

Direct Exposure Pathway Parameters. The incidental water and sediment ingestion rates were derived from U.S. EPA guidance (U.S. EPA 1996). The medium water ingestion rate (for both adults and children) of 50 ml/hr was taken from the U.S. Superfund Exposure Assessment Manual (SEAMS) (U.S. EPA 1988). The low and high estimates were estimated from this value using professional judgement.

Similarly, the medium estimates of the incidental soil ingestion rate for adults and children were derived using an average value from U.S. EPA guidance and the low and high estimates were derived from this point using professional judgment. The adult medium value is 50 mg/day. However, since children are expected come into closer contact with soils, the medium estimate is 100 mg/d.

Frequency of exposure in the recreational scenarios is based on estimates of the frequency of swimming. An estimate of average swimming frequency of once per month was taken from U.S. EPA Region 10 guidance (1998) and from the U.S. EPA (1988). This was used as the basis for medium estimate of frequency of 12 days per year exposure for swimmers, windsurfers, and SCUBA divers. For each of these scenarios an estimate of 24 days per year and two days per year was used for the high and low estimates, respectively.

For netfishers, two days/year and 12 days/year were used for the low and medium estimates of exposure frequency, respectively. However, there is greater uncertainty as to an estimate of a high number of days of exposure that might occur for netfishing. An estimate of 91 days per year was used based on best professional judgement.

Estimates of the exposure time (duration of each exposure event) were taken from U.S. EPA Region 10 (1998) guidance and from (U.S. EPA 1988). An estimate of the average swimming time of one hour taken from U.S. EPA Region 10 (1998) guidance was used to estimate medium exposures. Estimates of 2.6 hours and 10 minutes were used as the high and low estimates of time spent swimming, respectively. These exposure time estimates were applied to all four direct exposure scenarios.

The dermal exposure pathways require an estimate of the surface area of the skin exposed to either water or sediments, the absorbed fraction of COPCs from sediment and the sediment deposition rate to skin. The absorbed fraction from sediment varies dependent of the properties of the specific chemical, while sediment deposition rates vary dependent of the activity and extent of contact with sediments. In general, sediment deposition rates can be expected to be higher for children. Sediment deposition rates were estimated from the U.S. EPA exposure factors handbook (U.S. EPA 1998). The estimates of chemical fraction absorbed from sediment were estimated using U.S. EPA's dermal exposure assessment methodology (U.S. EPA 1992).

Exposed skin surface areas varied dependent on whether water or sediment exposure was evaluated. Surface area estimates were derived from the U.S. EPA (1996) exposure factors handbook. In the case of water exposure to adults it was assumed that the whole body could be immersed. Therefore, estimates of the whole body surface area were used for the medium (50^{th} percentile) and high (95^{th} percentile) exposure scenarios. The low estimate is a mean estimate of the surface area of the forearms, hands, lower legs and feet. For child exposures, the 10^{th}, 50^{th}, and 90^{th} percentile estimates of a child surface area were used for low, medium, and high exposures, respectively, to both water and sediment. For adult sediment exposures, the low estimate was based on the mean estimate of the surface area of the forearms, hands, lower legs and feet, while the medium estimate was based on the mean area of upper and lower extremities and the high estimate was based on the mean total surface area of the body excluding the head and hips.

3.2.4 Uncertainties in Exposure Assessment

For certain chemicals, (e.g, Aroclors and PAHs) the analytical detection limits for the methods used are greater than the environmental concentrations associated with potential risks (i.e., risk-based concentrations). Therefore, it is uncertain whether the chemical is present at concentrations below the analytical detection limit, but above concentrations that may pose risks to health. For this assessment, the assumption was made that if a chemical was not detected in a sample, it was present at one-half the detection limit, and that concentration estimate was used to estimate an exposure concentration. However, if a COPC was never detected in any sample it was assumed not to be present in that tissue type at that location where the sample was collected.

As described in the Field Sampling Work Plan (Appendix A) each tissue sample consisted of a composite of 3 to 20 individuals, depending on the species. Two to three composite samples were collected for each species, except crab hepatopancreas and prawns, where one composite sample was collected. Composite samples were used to obtain average tissue concentrations within the study area. However, for tissues with only one composite sample available, uncertainty exists regarding the extent to which these data represent average conditions in the study area.

Many assumptions were made in the exposure assessment that introduce uncertainties into the quantification of chemical exposures. For many assumptions, a simple sensitivity analysis was conducted to test their influence on the estimated exposures. This was accomplished by calculating exposures assuming plausible values resulting in high, medium, and low levels of exposure. For the remaining assumptions, conservative estimates intended to overestimate exposures were generally used.

Sediment and water exposure concentrations were obtained from the calibrated water and sediment quality model. Because the model was calibrated to an extensive database, there is a relatively low degree of uncertainty with the output. However, there are three uncertainties associated with the sediment and water exposure concentrations worth noting:

- Sediment concentrations in areas with no sediment data available were estimated using linear interpolation from available data (Appendix B-1). Actual sediment concentrations in these areas remain uncertain. In some areas with no known sources of contamination the linear extrapolation method may have resulted in very conservative estimated concentrations. For example, there are no known sources of PCBs into Elliott Bay sediments near Duwamish Head, yet elevated PCB concentrations are predicted in this location due to the use of the linear extrapolation method. Exposures to PCBs in sediments at this location were predicted to be higher than exposures at Duwamish Park in the Duwamish River. It is possible that the PCB concentrations in sediments at Duwamish Head in Elliott Bay are substantially below the estimated concentration, and that exposures are substantially lower than estimated.

- Conservative estimates of the average exposure concentration (95th percent upper confidence limit of the mean concentration) were used. This approach is consistent of U.S. EPA (1989a) guidance and is intended to ensure that exposures are not underestimated.

- Exposure concentrations were estimated for the areas where exposures are assumed to occur. Because there are uncertainties associated with exposure location, exposure concentrations remain uncertain.

Uncertainties associated with the exposure models are primarily associated with the ability of the models to mimic real world exposures. The models for estimating exposures from ingestion (of seafood, water and sediment) are relatively simple and generally believed to be appropriate. The model for estimating dermal adsorption from water and sediment are highly uncertain and likely overestimate potential exposures. For example, for estimating sediment exposures, conservative default estimates of the fraction of chemical that crosses the skin were used. These estimates do not account for chemical bioavailability, sediment structure, or any other chemical-specific parameters (except sediment concentration). This approach likely substantially overestimates potential exposures from dermal adsorption.

Uncertainties in exposure assumptions were addressed by assessing a range of possible exposure levels and reporting the resulting range of exposures (and risks). For example, the adult exposure durations for both the direct exposure and seafood consumption pathways were assumed to be 9 (low), 33 (medium) or 75 (high) years. The numbers of times per year that exposures were assumed to occur also was varied, along with several other parameters. It is likely that the range of exposures predicted using this approach provide adequately conservative estimates of the range of actual exposures.

The seafood risk assessment was conducted on a tissue-specific basis; that is, chemical concentrations measured in various species of fish and shellfish were used to estimate exposure concentrations. It is unlikely that any individual would eat exclusively perch or crabs or crab hepatopancreas at the estimated ingestion rates over a number of years, although this scenario may be more likely with some species such as salmon. Therefore, the chemical concentrations used can only approximate the actual concentrations to which an individual may be exposed due to the likely variability in seafood consumed.

3.3 Human Health Risk Characterization Methods

The human health risk characterization identified the potential risk posed by COPCs and the types of health risks on which the risk estimates were based. The methods used to numerically estimate cancer and non-carcinogenic health risks are presented below.

3.3.1 Calculation of Health Risks for Non-Carcinogenic Chemicals

Human health risks were evaluated by comparing exposure of estimates (chronic daily intake) with the estimate of the lowest dose (U.S. EPA oral reference dose or RfD) at

which non-carcinogenic effects could occur (see Section 3.1 above). The calculation of the non-carcinogenic "hazard quotient" is shown in Equation 3-16.

$$\text{Hazard Quotient} = \frac{\text{Chronic Daily Intake}}{\text{RfD}} \qquad \text{(Equation 3-16)}$$

Hazard quotients (HQs) greater than one indicated that health effects could occur and the chemical exposure requires further evaluation. HQ values less than one suggest negligible risks. If the chemical-specific HQ exceeds one, it does not explicitly identify a health risk from chronic exposure. Rather, it indicates the potential for health effects and that further evaluation of the chemical exposure may be necessary. This interpretation of the HQs is based on the conservative exposure assumptions used to derive the CDI and in the derivation of the RfD (see Section 3.1 above). RfDs frequently include in their derivation "adjustments", often large, termed uncertainty factors, as well as an implicit assumption that humans are at least as sensitive as the most sensitive animal species tested for that chemical. In fact, where concern for sensitive human subpopulations is an issue for a chemical (this is almost universally the case), then the assumption is that these groups are more sensitive than the most sensitive species tested. Incorporation of these uncertainty factors could result in artificially high-risk predictions. See Issue paper #8 - "*Human Health Toxicology*" (Appendix C) for a further discussion of these issues.

Additionally, HQs do not provide any information about the source of the chemical (i.e., whether the chemical is anthropogenic or naturally occurring). For example, background concentrations of substances in soils and water may result in intake estimates that exceed toxicity reference concentrations.

Humans are frequently exposed to more than one chemical at a time. To assess the exposure to multiple chemicals, potential additive affects of COPCs with similar endpoints were evaluated for both non-carcinogenic and carcinogenic effects. The potential for non-carcinogenic additive effects was evaluated using a hazard index approach (U.S. EPA 1989). The fundamental assumption that underlines this approach is that cumulative effect could result from simultaneous exposures to multiple chemicals. When the hazard index (HI) for a chronic exposure pathway exceeds a value of one, it suggests the possibility of non-carcinogenic adverse health effect from chronic exposure to all chemicals evaluated. The chronic HI is calculated as the sum of the chemical-specific HQs for each exposure pathway, as shown in Equation 3-17 (U.S. EPA 1989a):

$$\text{Hazard Index} = CDI_1 / RfD_1 + \ldots + CDI_n / RfD_n \qquad \text{(Equation 3-17)}$$

Where:

CDI_n	=	Chronic daily intake for the nth chemical, mg/kg day
RfD_n	=	Chronic reference dose for the nth chemical, mg/kg day

3.3.2 Calculation of Health Risks from Chemical Carcinogens

Cancer potential is estimated by multiplying the cancer slope factor[16] by an estimated lifetime average daily intake (U.S. EPA 1989):

$$\text{Cancer Risk} = \text{Lifetime Average Daily Intake} \times \text{Slope Factor} \qquad \text{(Equation 3-18)}$$

The cancer risk represents the probability of an individual developing cancer over a lifetime of exposure (U.S. EPA 1989a). Interpretation of cancer risk results is similar to the interpretation of non-cancer risk results in that consideration must be given to the magnitude of the risk prediction and the degree of certainty in the exposure assessment. Three risk predictions were estimated for each chemical exposure seafood tissue type corresponding to each of the high, medium, and low exposure estimates. The level of risk considered to be "acceptable" must take these factors into consideration. Health risks for substances causing cancer (e.g., arsenic) are expressed as unit probabilities (e.g., $1x10^{-4}$ or 1 in 10,000)[17] of developing cancer during a 75-year lifetime.

A similar potential exists for exposure to two or more carcinogens resulting in a greater risk of developing an environmentally induced cancer than would occur by exposure to a single carcinogen. The total risk associated with simultaneous exposure to multiple carcinogens is estimated as shown in Equation 3-19:

$$RISK_T = LADI_1 \times SF_1 + \ldots + LADI_n \times SF_n \qquad \text{(Equation 3-19)}$$

Where:

$RISK_T$	=	Total carcinogenic risk
$LADI_n$	=	Lifetime average daily intake for the nth chemical, mg/kg day
SF_n	=	Carcinogenic slope factor for the nth chemical, mg/kg-day

3.3.3 Uncertainties in Risk Characterization

Chemical constituents that affect the same target organ or tissue, by the same or similar mechanisms of toxicity may have additive effects. In the absence of information to suggest that COPCs may have synergistic or antagonistic interactions, additivity was assumed. COPCs were identified in the risk screening based on the individual concentrations and RfDs. Exposure to multiple chemicals at concentrations less than their individual RfDs may create a risk through additive effects from multiple simultaneous exposures. The impact of the assumption of additivity and no synergistic or antagonistic

[16] The slope factor is the value used to estimate the cancer-causing potency.

[17] A 1 $x10^{-4}$ carcinogenic risk prediction is interpreted as a one in 10,000 incremental chance of a person developing cancer in a lifetime from exposure to the lifetime average daily intake. It may also be expressed as one person in 10,000 developing cancer in an exposed population.

interactions is unknown, but some general inferences can be made, and are discussed below.

The potential for additive effects for non-carcinogenic risks was assessed using the hazard index approach and additive carcinogenic risks were assessed by calculating total potential risks as described in Section 3.3. Uncertainties are associated with the methodology used to calculate the potential for additive effects. For non-carcinogenic COPCs the hazard indices were calculated by summing the HQs over each exposure pathway. This approach is consistent with U.S. EPA guidelines (U.S. EPA 1989a). However, summing all HQs is a conservative approach because the hazard indices are most properly applied to chemicals with similar mechanisms of action. Chemicals with unlike mechanisms or dissimilar toxic endpoints (e.g., neurotoxicity and kidney effects) are unlikely to have additive affects because they are acting on differing target organs or tissues. Additionally, the RfDs for each of the COPCs are not necessarily equivalent with respect to confidence in the supporting toxicological data and the "safety factors" used. Therefore, the degree of concern does not increase linearly as HQs are summed. For these reasons, the calculation of a hazard index by summing the COPC HQs provides a conservative indicator of the potential for non-carcinogenic risks.

Total site carcinogenic risks were estimated by summing the individual COPC risks as described in Section 3.3. The assumption of additive effects is likely to result in over predictions of risk because slope factors are an upper 95^{th} percentile estimate of carcinogenic potency, and the upper 95^{th} percentiles of probability distributions are not strictly additive (U.S. EPA 1989a). The summed risk estimate may, therefore, be over predicted.

3.4 Human Health Results

As described above, the human health risk characterization combines the results of the exposure characterization described in Section 3.2 with toxicological information described in Section 3.1 to derive quantitative estimates of potential health risks. This section presents a summary of the results of the risk characterization. Potential risks from pathogens are presented in Section 4. As described in the exposure characterization (Section 3.2), risks were estimated for both direct exposure pathways (i.e., contact with water and sediment) for several recreational activities intended to be representative of the potential for human exposures resulting from recreational uses of the study area. In addition, potential risks resulting from consumption of seafood harvested from the study area were assessed. Potential risks were assessed for both adults and three groups of children aged 1 to 6, 7 to 12, and 13 to 18.

Risks were characterized in several differing ways. Risks to individuals were assessed assuming three differing levels of exposure as described in Section 3.2. In addition, individual risks were assessed on a per event basis. That is, risks from individual events such as a single recreational or fishing activity or consuming a single fish meal were determined. Risks to an entire population were not evaluated quantitatively but are discussed below.

Risks are presented according to the type of potential health effect, the potential for carcinogenic effects are discussed separately from all other (non-carcinogenic) health effects. Comparisons of risks under baseline conditions are compared to those predicted under a potential without CSO scenario. Additionally, risks predicted for either recreational activities occurring in, or seafood collected from, the study area were compared to potential risks in "background" or "reference" locations in Puget Sound outside of the study area. Finally, uncertainties specific to this risk assessment are presented at the end of this section.

3.4.1 Direct Exposure Pathways

Direct exposures to water and sediments were evaluated for four recreational scenarios intended to be representative of all types of recreational exposures that may occur in the WQA study area. The pathways evaluated were swimming at Duwamish Park in the Duwamish River and at Duwamish Head in Elliott Bay, SCUBA diving at Seacrest Park, windsurfing/sailboarding in Elliott Bay, and netfishing in the Duwamish River. Non-carcinogenic and carcinogenic risk results for direct exposure to sediment and water are discussed below.

Non-carcinogenic Risks. No HQs exceeding one were predicted for any of the four exposure scenarios for either adults or children at any exposure level. Therefore, all of the chemical substances evaluated are expected to pose negligible risks for any non-carcinogenic health effects by direct exposure to sediments or water to swimmers, netfishers, scuba divers, or windsurfers, regardless of age during which the exposures may occur.

To assess the potential for cumulative risks associated with multiple chemical exposures, a hazard index was calculated for each exposure pathway as the sum of the chemical-specific HQs. This is a conservative approach, as different chemicals frequently have different mechanisms of effect. All hazard indices were less than 1.0, indicating negligible non-carcinogenic risks by direct exposure to sediment or water to swimmers, netfishers, SCUBA divers or windsurfers, regardless of age during which the exposures may occur and the frequency that exposure occurs.

As described in Section 3.2 the direct exposure scenarios selected were chosen to represent the highest potential exposures when compared to other recreational activities (e.g., sailing, line angling). Therefore, the variety of recreational activities that may occur in the study area that were not specifically evaluated in the risk assessment are not expected to pose any risks of non-carcinogenic health effects.

Risks By An Event. As described above, risks were characterized on an event basis. However, since no non-carcinogenic health risks were predicted at any exposure level, no non-carcinogenic risks are expected from any number of recreational events in the study area. That is, recreational use of the study area could frequently occur for many years with no expectation of any non-carcinogenic adverse effects resulting from exposure to the COPCs in water and sediments.

Comparison of Baseline Risks to the Without CSO Scenario. Risks from the direct exposure pathways were evaluated using modeled chemical concentrations in a baseline scenario and a potential without CSO scenario. In general, when the HQs predicted from the baseline chemical concentrations were compared to those calculated using the without CSO concentrations, the results were very similar. The numerical values of the predicted HQs vary slightly on a chemical-specific basis. For example, the water ingestion HQ for arsenic for an adult swimmer at Duwamish Park in the Duwamish River under the "high" exposure assumptions is 0.00053 in the baseline scenario and 0.00052 in the without CSO scenario. Although the numerical values of the results vary slightly, there is no biologically significant difference between the results. They may be interpreted as meaning that there is no risk of non-carcinogenic health effects in either scenario. This interpretation may be generalized to the overall results of the non-carcinogenic evaluation for all the direct exposure pathways.

In the example shown above, the numerical value of the HQs for arsenic increased slightly in the baseline scenario. However, this was not consistently the case for all COPCs. Rather, the values of the HQs varied in either direction (either increasing or decreasing) depending on the specific chemical evaluated. There was no consistent pattern to this variation in the results other than the variation was chemical-specific and the variation in the results was very small. This variation in the results is dependent on the chemical concentrations in the baseline and without CSO scenarios, which may increase or decrease slightly between scenarios on a chemical-specific basis.

Comparison to Background Risks. Potential risks from the sediment direct exposure pathways were evaluated using chemical concentrations from locations in Puget Sound intended to represent background concentrations in sediments. The data were not directly comparable due to that fact that background data on all of the COPCs were not available. Specifically, no data were available for PCBs. Additionally, no water data were available. However, information was available for all of the metals and many of the PAHs in sediment.

No HQs exceeding one were predicted using the background data for comparison. Therefore, no non-carcinogenic health risks are expected from the direct exposure pathways at background chemical concentrations.

Carcinogenic Risks. When risks are evaluated over all exposure levels and COPCs, cancer risk predictions were less than one in one million ($1x10^{-6}$) for windsurfers and SCUBA divers at all exposure levels and for netfishers and swimmers at medium and low exposure levels. Total incremental carcinogenic risks across all COPCs were also predicted to be less than one in one million for these scenarios. However, risks exceeding one chance in a million were predicted for people netfishing in the Duwamish River and swimming at Duwamish Head in Elliott Bay or in the Duwamish River at Duwamish Park at high exposure levels. These potential risks are discussed separately below.

Predicted Risks to Netfishers. Potential incremental carcinogenic risks to netfishers in the Duwamish River of about one in one hundred thousand ($1x10^{-5}$) were predicted under

high exposure assumptions. Under the high exposure assumptions, net fishers were assumed to engage in the activity 91 days per year for 75 years. The majority of these risks were associated with exposure to arsenic and PCBs in sediment. Predicted cancer risks for all chemicals evaluated other than arsenic and PCBs were less than one in one million under all exposure scenarios. PCBs and arsenic in water also contributed risks of about four in ten million. The predicted risks to netfishers are summarized in Table 3-18.

Table 3-18. Baseline Condition Predicted Adult Incremental Cancer Risks (x10^{-6}) from Arsenic and PCBs to Duwamish Netfishers[a]

	Arsenic					PCBs				
	Water		Sediment			Water		Sediment		
Exposure Level	Ingestion	Dermal	Ingestion	Dermal	Total	Ingestion	Dermal	Ingestion	Dermal	Total
High (91 day/year for 75 years)	1.4	0.4	4.2	6.4	12.4	0.006	3.1	0.1	1.9	5.1
Medium (24 day/year for 33 years)	0.03	0.01	0.28	0.04	0.4	0.0001	0.2	0.01	0.01	0.2
Low (2 days/year for 9 years)	0.0001	<0.0001	0.003	<0.0001	0.003	<0.0001	0.0004	0.0001	<0.0001	0.0004

[a] Values shown are the predicted numbers of excess cancers per 1 million exposed persons.

No chemical-specific incremental carcinogenic risks exceeding one in a million were predicted under the medium and low exposure level assumptions. Under the medium exposure assumptions, net fishers were assumed to engage in the activity 24 days per year for 33 years. Therefore, the predicted risks only apply to the most highly exposed individuals. That is, potential risks above one in a million were predicted only for individuals who engage in netfishing 91 days per year for 75 years and are exposed to sediment and water over most of the body surface.

To estimate cumulative risks from multiple chemical exposures, the total incremental carcinogenic risk was calculated for each exposure pathway. The total incremental carcinogenic risk represents the increased probability of developing cancer because of the chemical exposures, regardless if the chemicals are believed to initiate different types of cancer. For the net fisher pathway, the total carcinogenic risk ranged from $2x10^{-5}$ (2 in 100,000) under high exposure levels, to less than $1x10^{-6}$ (one in one million) for the medium and low exposure levels.

Predicted Risks to Swimmers. Potential incremental carcinogenic risks to swimmers in the Duwamish River and in Elliott Bay exceeding one in a million were predicted under high exposure assumptions (24 events per year) for adults (75 years of exposure) and children (six years of exposure) at all age groups. Risks up to about one in a million were also predicted for children aged 1 to 6 at medium exposure levels. However, risks to all other age groups (children aged 7 to 12, children aged 13 to 18, and adults) at the medium exposure level (12 events per year) were less than one in 1 million. All risks at the low exposure level (2 events per year) were less than one in one million. The majority of the risks to swimmers were predicted from dermal exposure to arsenic and PCBs in sediment. The highest risks resulted from arsenic exposures. No other chemicals evaluated were predicted to pose incremental carcinogenic risks of greater than $1x10^{-7}$ (one in ten million) at any age group and exposure level.

None of the predicted chemical-specific risks attained a one chance in one hundred thousand level. The predicted risks to child swimmers from arsenic and PCBs are summarized below in Table 3-19. The swimming risks to children at high exposure levels are based on the assumptions that children swim 24 days per year for 2.6 hours per day and that the 90^{th} percentile estimate of the child's body surface area is exposed to sediments while swimming.

Total incremental carcinogenic risks were estimated by summing the chemical-specific risks. The highest total incremental risks were about $1x10^{-5}$ (1 in 100,000) for the child aged 1 to 6 under the high exposure levels (swimming 24 times per year).

Risks by Event. As described in Section 3.1, evaluation of carcinogenic risks conservatively assumes that any dose of a carcinogenic substance, regardless how small, has some probability of a carcinogenic response. This assumption allows the prediction of the point at which the cumulative dose (i.e., repeated exposures) would result in a cancer risk prediction reaching any given level. Therefore, it is possible to present the number of recreational events per year that would be required to reach a specified risk level or the risk from an individual event. These risks were calculated based on the

Table 3-19. Baseline Condition Predicted Incremental Cancer Risks ($x10^{-6}$) from Arsenic and PCBs to Highly Exposed Adult and Child Swimmers[a]

	Age Group	Duwamish Park in the Duwamish River					Duwamish Head in Elliott Bay				
		Water		Sediment			Water		Sediment		
		Ingestion	Dermal	Ingestion	Dermal	Total	Ingestion	Dermal	Ingestion	Dermal	Total
Arsenic	1 to 6	0.08	0.009	0.4	4.0	4.4	0.1	0.01	0.7	7.0	7.6
	7 to 12	0.04	0.006	0.2	3.1	3.3	0.006	0.001	0.3	5.4	5.7
	13 to 18	0.02	0.005	0.1	2.3	2.4	0.04	0.008	0.2	4.1	4.2
	Adult	0.2	0.07	0.4	0.7	1.1	0.4	0.1	0.8	1.2	1.9
PCBs	1 to 6	0.0004	0.08	0.01	0.9	0.9	0.0003	0.07	0.02	1.8	1.8
	7 to 12	0.0002	0.006	0.004	0.7	0.7	0.0002	0.05	0.009	1.4	1.4
	13 to 18	0.0001	0.05	0.002	0.5	0.5	<0.0001	0.04	0.005	1.0	1.0
	Adult	0.001	0.7	0.01	0.2	0.2	0.001	0.5	0.02	0.3	0.3

[a] Values shown are the predicted numbers of excess cancers per 1 million exposed persons.

combined risks from both ingestion and sediment exposures and are summarized in Table 3-20 below. To calculate the number of events per year required to obtain an incremental carcinogenic risk of one in one million, the risk and exposure equations were rearranged and solved for exposure frequency. Medium exposure level assumptions were used for all parameters (except exposure frequency, which is being solved for) to calculate the required number of events. The calculated number of events are equal to the number of events that can occur per year for 33 years (for adults) or six years (for children) before reaching the one in one million risk level.

Comparison of Baseline Risks to the Without CSO Scenario. In general, risks predicted in the baseline scenario are very similar to those calculated in the without CSO scenario. As described in the discussion on non-carcinogenic effects, the modeled chemical concentrations are very similar in both scenarios. The absolute numerical concentration value may increase or decrease slightly depending on the chemical evaluated and the effect of these concentration differences on the risk results is minimal. The predicted risks to low (two events per year for nine years), medium (24 events per year for 33 years), and highly (91 events per year for 75 years) exposed netfishers under without CSO conditions are summarized in Table 3-21. The predicted risks to highly exposed (24 events per year) swimmers under without CSO conditions are summarized in Table 3-22.

There are some slight differences in the magnitudes of the predicted risks. The values of risk predictions vary between approximately 1 and 10 percent. However, when interpreted in terms of any potential difference in the likelihood of developing cancer from exposure to chemicals, the results are identical between with- and without CSO scenarios. As an extreme example, when numbers such as one in a million and four in a million are compared, they cannot be interpreted as representing significantly different risks due to the uncertainties in the assessment of carcinogenic potency and the overall uncertainty of the assessment. These toxicological uncertainties are discussed in more detail in Section 3.1 and in issue paper No. 8 - "*Human Health Toxicology*" (Appendix C). The appropriate interpretation of both results is that they both represent approximately one in a million excess (i.e., additional risk over background) risk of developing cancer due to the exposure.

Typically, the calculated differences in risk predictions between the two scenarios are less than the example provided above. Thus, the proper interpretation of the results is that there is no difference in the carcinogenic risk potential regardless of the contribution of CSOs to the chemical load in waters or sediments.

Comparison to Reference Site Risks. The risks predicted using reference site Puget Sound chemical data were similar (i.e., generally on the same order of magnitude) to those predicted using the modeled chemical concentrations in the WQA study area. The majority of risks predicted using reference site chemical concentrations were due to arsenic. Reference site PCB concentration data in water and sediment were not available for comparison.

Table 3-20. Number of Exposure Events per Year for 33 Years (for Adults) or Six Years (for Children) Under Baseline Conditions at Medium Exposure Levels Required to Achieve Incremental Lifetime Carcinogenic Risk of One in One Million for the Direct Exposure Pathways

	Arsenic	PCBs	All Other Chemicals Evaluated	Total Across All Chemicals
Child age 1 to 6				
Swimming D.P.	21	92	>1,000	17
Swimming D.H.	12	51	>1,000	10
Child age 7 to 12				
Swimming D.P.	31	132	>1,000	25
Swimming D.H.	18	73	>1,000	14
Child age 13 to 18				
Swimming D.P.	39	158	>1,000	31
Swimming D.H.	22	88	>1,000	18
Adult				
Swimming D.P.	150	164	>1,000	77
Swimming D.H.	87	192	>1,000	58
Netfishing	65	126	>1,000	41
SCUBA	450	407	>1,000	209
Windsurfing	484	164	>1,000	121

D.P. = Duwamish Park

D.H. = Duwamish Head

Table 3-21. Without CSO Scenario Predicted Adult Incremental Cancer Risks (x10^{-6}) from Arsenic and PCBs to Duwamish Netfishers[a]

	Arsenic					PCBs				
	Water		Sediment			Water		Sediment		
Exposure Level	Ingestion	Dermal	Ingestion	Dermal	Total	Ingestion	Dermal	Ingestion	Dermal	Total
High (91 day/year for 75 years)	1.4	0.4	4.3	6.5	12.6	0.006	3.0	0.5	7.2	10.8
Medium (24 day/year for 33 years)	0.04	0.01	0.3	0.04	0.4	0.0001	0.2	0.03	0.05	0.2
Low (2 days/year for 9 years)	<0.0001	<0.0001	0.003	<0.0001	0.003	<0.0001	0.0004	0.0003	<0.0001	0.0007

[a] Values shown are the predicted numbers of excess cancers per 1 million exposed persons.

Table 3-22. Without CSO Scenario Predicted Incremental Cancer Risks (x10^{-6}) from Arsenic and PCBs to Highly Exposed Adult and Child Swimmers[a]

		Duwamish River					Elliott Bay				
		Water		Sediment			Water		Sediment		
Chemical	Age Group	Ingestion	Dermal	Ingestion	Dermal	Total	Ingestion	Dermal	Ingestion	Dermal	Total
Arsenic	1 to 6	0.08	0.008	0.5	5.3	5.9	0.1	0.01	0.7	7.0	7.8
	7 to 12	0.04	0.007	0.3	4.1	4.4	0.06	0.01	0.3	5.4	5.8
	13 to 18	0.02	0.005	0.1	3.1	3.2	0.04	0.008	0.2	4.0	4.3
	Adult	0.2	0.07	0.6	0.9	1.8	0.4	0.1	0.8	1.2	2.4
PCBs	1 to 6	0.0004	0.08	0.002	0.2	0.3	0.0003	0.07	0.02	2.1	2.1
	7 to 12	0.0002	0.06	0.001	0.2	0.2	0.0002	0.05	0.01	1.6	1.6
	13 to 18	0.0001	0.05	0.0006	0.1	0.2	<0.0001	0.04	0.006	1.2	1.2
	Adult	0.001	0.7	0.003	0.04	0.7	0.001	0.5	0.02	0.3	0.9

[a] Values shown are the predicted numbers of excess cancers per 1 million exposed persons.

As was the case using the WQA study area concentrations, risks above one in a million were predicted in the swimming and netfishing scenarios under the high exposure assumptions only. Risks from these two scenarios under high exposure assumptions to adults and children age one to six are presented in Table 3-23 below for comparison purposes.

Table 3-23. Predicted Sediment Reference Site Arsenic Cancer Risks to Highly Exposed Adults and Children[a]

Adults				Children Age 1 to 6	
Swimming		Netfishing		Swimming	
Ingestion	Dermal	Ingestion	Dermal	Ingestion	Dermal
0.8	1	3	5	0.7	7

[a] Values presented as predicted numbers of excess cancers per one million exposed adults or children

3.4.2 Seafood Consumption Pathway

Potential risks from consumption of seafood harvested from the study area were assessed. Comparisons were made between seafoods collected from the Duwamish River, Elliott Bay, and reference locations outside the study area (usually Port Susan or Hood Canal). Additionally, chemical concentrations in seafood tissues were predicted in a without CSO scenario as described in Section 3.2 and used to estimate risks for comparison.

A variety of seafood tissue types were used in the analysis, and tissues varied between Elliott Bay and the Duwamish River. Tissue types evaluated are presented above in Table 3-8.

Non-carcinogenic Risks. The non-carcinogenic risk results from the seafood consumption pathway vary significantly between exposure levels. Using the high exposure assumptions (365 meals per year for 75 years) a number of COPCs are predicted to have HQs greater than one for both adults and children. However, when risks were evaluated using the medium exposure assumptions (24 meals per year for 33 years) arsenic and PCBs are the only two chemicals identified as having HQs greater than one. Because of the considerable difference in these results by exposure level, the results are discussed separately by exposure level below. As previously discussed, for the vast majority of the potentially exposed population (i.e., people who consume seafood taken from Elliott Bay or the Duwamish River) exposures are expected to occur at the medium level or below. For more discussion of this issue, see Section 3.2 or Issue Paper No. 3 - "*Human Site Uses"* (Appendix C).

Risks Predicted under High Exposure Assumptions. Under high exposure assumptions HQs exceeding one were predicted for adults and all three-child age groups, although the largest number and magnitude of exceedances were predicted for children. The COPCs

with HQs greater than one for one or more tissue types at either the Duwamish River or Elliott Bay include arsenic, cadmium, copper, lead, mercury, zinc, TBT, and PCBs. The range of HQs for different tissue types and age groups are presented in Table 3-24 for the Duwamish River, Elliott Bay, and the reference sites.

Table 3-24. Baseline Condition Range of Hazard Quotients (HQs) for Different Species Under High Exposure Levels for Adults and Children of All Age Groups

Chemical	Duwamish River	Elliott Bay	Reference Sites
Arsenic	1.4 – 98.5	1.9 – 88.2	0.8 – 99.0
Cadmium	ND – 6.2	ND – 12.7	ND – 4.8
Copper	<0.01 – 0.6	<0.01 – 1.2	<0.01 – 0.5
Lead	ND – 1.7	ND – 2.9	ND – 0.2
Mercury	0.2 – 6.4	0.1 – 20.2	0.06 – 25.0
Zinc	0.09 – 2.6	0.1 – 3.2	0.04 – 2.3
TBT	0.09 – 7.8	0.04 – 7.4	<0.01 – 2.16
PCBs	7.2 – 663	ND - 176	ND – 46.5

N/D = Not detected

The magnitudes of the HQs varied by tissue type and source of the tissue (i.e., Duwamish River vs. Elliott Bay). In general the magnitudes of the HQs were similar in each tissue type in either Elliott Bay or the Duwamish River. The notable exception is PCBs. The magnitudes of the HQs in the Duwamish River were generally an order of magnitude greater than those predicted for Elliott Bay. However, more chemicals were predicted to have HQs greater than one in Elliott Bay than in the Duwamish River. These results for a highly exposed (365-meals/year) child (ages 1 to 6) are presented below in Table 3-25 for all chemicals that exceed 1.0 for any tissue type. Only the results for the child aged 1 to 6 are shown because under the high exposure seafood consumption scenario, the child aged 1 to 6 was predicted to have HQs about 2 to 3 times higher than adults, about 1.5 to 2 times higher than children aged 7 to 12, and about 2 to 4 times higher than children aged 13 to 18.

For adults, the magnitudes of the HQs for cadmium, lead, zinc, and TBT were relatively small (<5) and mercury was only somewhat greater (<10 for Duwamish River and Elliott Bay). Copper exceeded one (HQ = 1.2) only for the child aged 1 to 6 under the high exposure scenario and only for squid. Based on the relatively low magnitude of these exceedances and the conservative assumptions inherent in toxicity assessment for cadmium and TBT (i.e., use of uncertainty factors in RfD), these two chemicals are expected to pose negligible risks to adults, even based on the assumption that an

individual could consume seafood from the river or bay every day of the year. Zinc and copper both have an essential role in human nutrition and are required for normal physiological functioning (U.S. EPA 1998; WHO 1996). Thus, based on the small magnitude of these exceedances and the conservative assumptions used in the assessment, these two nutrient metals are not expected to pose any potential health risks to either adults or children.

Table 3-25. Baseline Condition Seafood Consumption Hazard Quotients for a Highly Exposed Child Aged 1 to 6 for Chemicals with Any HQs Greater than One by Tissue Type[a]

Tissue	Chemical								
	Arsenic	Cadmium	Copper	Lead	Mercury	Zinc	TBT	PCBs	Hazard Index for all chemicals
Duwamish River									
Raw Sole	60.9	ND	<0.1	ND	2.4	0.3	0.2	159.8	224
Cooked Sole	86.5	ND	<0.1	ND	3.9	0.3	0.6	299.1	391
Sole-Whole Body	34.1	ND	<0.1	0.6	2.3	0.9	0.6	663.1	702
Perch-Whole Body	6.1	0.3	0.1	0.6	2.6	0.7	7.8	246.5	265
Salmon	3.7	N/AP	<0.1	0.1	2.3	N/AP	N/AP	18.6	25
Crab Hepatopancreas	8.5	0.4	0.3	0.2	0.6	0.3	0.7	198.0	209
Raw Crab	98.5	0.6	0.5	0.8	4.8	2.6	6.6	115.4	230
Cooked Crab	34.6	0.9	0.6	0.2	6.4	2.2	3.4	66.9	114
Mussel	3.6	6.2	<0.1	1.7	0.4	1.8	2.8	19.8	37
Elliott Bay									
Raw Sole	33.7	ND	<0.1	ND	3.0	0.3	0.1	8.6	46
Cooked Sole	43.8	ND	<0.1	ND	4.1	0.3	0.2	40.4	89
Sole-Whole Body	27.7	ND	<0.1	0.4	2.3	1.0	0.5	74.6	107
Perch-Whole Body	4.5	0.4	<0.1	0.5	0.9	0.7	7.4	106.8	121
Raw Rockfish	4.9	ND	<0.1	ND	20.2	0.1	2.0	38.4	66
Crab Hepatopancreas	9.7	6.1	0.2	0.1	0.5	0.6	0.7	175.6	194
Cooked Crab Hepatopancreas	6.6	12.7	0.3	0.3	0.5	0.7	1.0	126.9	149

Table 3-25. Baseline Condition Seafood Consumption Hazard Quotients for a Highly Exposed Child Aged 1 to 6 for Chemicals with Any HQs Greater than One by Tissue Typea (continued)

Tissue	Chemical								
	Arsenic	Cadmium	Copper	Lead	Mercury	Zinc	TBT	PCBs	Hazard Index for all chemicals
Raw Crab	78.4	5.9	0.4	1.1	3.6	2.5	3.3	82.8	178
Cooked Crab	67.2	2.5	0.6	0.3	12.3	3.2	4.7	29.1	120
Mussel	4.6	8.0	<0.1	2.9	0.4	2.0	2.2	ND	21
Prawn	88.2	0.1	0.1	ND	1.5	0.4	2.6	ND	93
Whole Squid	21.2	2.0	1.2	ND	1.5	0.6	1.4	17.5	46
Reference Sites									
Raw Sole	48.0	ND	<0.1	ND	2.2	0.3	<0.1	ND	51
Cooked Sole	67.8	ND	<0.1	ND	3.7	0.2	<0.1	11.3	83
Sole-Whole Body	33.6	ND	<0.1	0.2	2.2	0.7	<0.1	10.5	47
Perch-whole body	4.3	0.4	<0.1	0.2	3.6	0.8	2.2	46.5	58
Salmon	31.7	ND	<0.1	<0.1	2.1	ND	ND	16.1	50
Raw Rockfish	10.0	ND	<0.1	ND	25.0	0.1	0.4	ND	36
Crab Hepatopancreas	2.9	3.7	0.4	0.0	0.5	0.2	<0.1	18.0	26
Cooked Crab Hepatopancreas	14.4	3.7	0.4	0.0	0.8	0.5	0.1	31.0	51
Raw Crab	15.9	1.7	0.3	0.1	2.2	2.1	0.1	ND	23
Cooked Crab	29.4	3.9	0.5	ND	5.6	2.3	0.2	9.7	52
Mussel	3.3	4.8	<0.1	0.1	0.2	0.3	0.1	ND	9.4
Prawn	99.0	1.2	0.2	ND	8.3	0.4	0.6	ND	110

[a] The highly exposed 1 to 6 year-old child was assumed to have consumed seafood 365 days per year.

ND = Not detected

For children, the HQs were slightly larger. For cadmium, lead, TBT, and mercury the highest HQs were 12.7, 2.9, 7.7, and 20.2, respectively, under the high exposure scenario for 1 to 6 year olds. However, the HQs for most tissues were less than ten for cadmium and mercury and less than two for lead. As was the case for adults, based on the magnitude of the exceedances, the conservative assumptions in the exposure and toxicity assessments, cadmium and TBT are not expected to pose significant risks to children.

The lead and mercury toxicity data are based on epidemiology studies evaluating neurological endpoints (see Section 3.1). U.S. EPA considers the neurological effects of lead to be so sensitive such that it is essentially without a threshold. Therefore, these results suggest some limited potential for adverse neurological effects to highly exposed children consuming seafoods from the study area or reference locations. However, these metals do not represent the greatest source of potential risks of non-carcinogenic health effects (i.e., they are not "drivers").

The highest HQs were predicted for arsenic and PCBs ranging from the single digits for some tissues to the hundreds for others (see Table 3-25). HQs in the 10 to 100 range occurred consistently across tissue type in both the Elliott Bay, the Duwamish River, and at reference locations, although the magnitude of the HQs for PCBs tended to be lower in the reference tissues indicating lower concentrations of PCBs at reference sites. These results suggest the potential for non-carcinogenic health risks from arsenic and PCBs are present for high consumers (daily consumption for years) of seafoods from the study area and other locations in Puget Sound.

Evaluation of possible risks posed by the combination of chemicals was assessed by calculating the hazard index. Because arsenic and PCB HQs were consistently substantially higher than the HQs for other chemicals, the hazard index is similar to the sum of the arsenic and PCB HQs.

Risks Predicted Under Medium and Low Exposure Assumptions. Similar to the non-carcinogenic risks predicted under high exposure conditions, HQs exceeding one were predicted under medium and low exposure assumptions for adults and all three child age groups. However, only arsenic and PCBs predicted HQs greater than one and the number of tissue types in which these exceedances occurred were significantly fewer. HQs were lower under the low exposure assumptions than under the medium exposure assumptions.

The magnitudes of the HQs varied by tissue type although for arsenic the magnitude of the HQs was similar between the Duwamish River, Elliott Bay, and the reference locations. For PCBs, no exceedances were predicted at the reference locations and the magnitude of the HQs was greatest in tissues from the Duwamish River. These results are summarized below in Table 3-26 for the medium exposure assumptions for adults and Table 3-27 for the low exposure assumptions for children ages 1 to 6.

Table 3-26. Baseline Condition Seafood Consumption Hazard Quotients for a Medium Exposure Level Child Aged 1 to 6 for Chemicals with HQs Greater than One by Tissue Type[a]

Tissue	Chemical		Hazard Index for all Chemicals
	Arsenic	PCBs	
Duwamish River			
Raw Sole	1.8	4.6	6.5
Cooked Sole	2.5	8.7	11.3
Sole - Whole Body	1.0	19.2	20.4
Perch – Whole Body	0.2	7.2	7.7
Salmon	0.1	0.5	0.7
Crab Hepatopancreas	0.3	7.5	7.9
Raw Crab	2.9	3.4	6.7
Cooked Crab	1.0	1.9	3.3
Mussel	0.1	0.6	1.1
Elliott Bay			
Raw Sole	1.0	0.2	1.3
Cooked Sole	1.3	1.2	2.6
Sole - Whole Body	0.8	2.2	3.1
Perch – Whole Body	0.1	3.1	3.5
Raw Rockfish	0.1	1.1	1.9
Crab Hepatopancreas	0.4	6.7	7.4
Cooked Crab Hepatopancreas	0.2	4.8	5.7
Raw Crab	2.3	2.4	5.2
Cooked Crab	2.0	0.8	3.5
Mussel	0.1	ND	0.6
Prawn	2.6	ND	2.7
Whole Squid	0.6	0.5	1.3
Reference Sites			
Raw Sole	1.4	ND	1.5
Cooked Sole	2.0	0.3	2.4
Sole - Whole Body	1.0	0.3	1.4
Perch – Whole Body	0.1	1.4	1.7
Salmon	0.9	0.5	1.4
Raw Rockfish	0.3	ND	1.0
Crab Hepatopancreas	0.1	0.7	1.0
Cooked Crab Hepatopancreas	0.6	1.2	1.9
Raw Crab	0.5	ND	0.7
Cooked Crab	0.8	0.3	1.5
Mussel	0.1	ND	0.3
Prawn	2.9	ND	3.2

[a] The medium exposed 1 to 6 year old child was assumed to consumed seafood 24 days per year.

ND = Not detected

Table 3-27. Baseline Condition Seafood Consumption Hazard Quotients for a Low Exposure Level Child Aged 1 to 6 for Chemicals with HQs Greater than One by Tissue Type[a]

Tissue	Chemical		Hazard Index for all Chemicals
	Arsenic	PCBs	
Duwamish River			
Raw Sole	0.4	1.0	1.4
Cooked Sole	0.5	1.8	2.4
Sole - Whole Body	0.2	4.1	4.3
Perch – Whole Body	0.04	1.5	1.6
Salmon	0.02	0.1	0.2
Crab Hepatopancreas	0.05	1.2	1.3
Raw Crab	0.6	0.7	1.4
Cooked Crab	0.2	0.4	0.7
Mussel	0.02	0.1	0.2
Elliott Bay			
Raw Sole	0.2	0.05	0.3
Cooked Sole	0.3	0.2	0.5
Sole - Whole Body	0.2	0.5	0.7
Perch – Whole Body	0.03	0.6	0.7
Raw Rockfish	0.03	0.2	0.4
Crab Hepatopancreas	0.06	1.0	1.2
Cooked Crab Hepatopancreas	0.04	0.8	0.9
Raw Crab	0.5	0.5	1.1
Cooked Crab	0.4	0.2	0.7
Mussel	0.03	ND	0.1
Prawn	0.5	ND	0.6
Whole Squid	0.1	0.1	0.3
Reference Sites			
Raw Sole	0.3	ND	0.3
Cooked Sole	0.4	0.07	0.5
Sole – Whole Body	0.2	0.06	0.3
Perch – Whole Body	0.03	0.3	0.4
Salmon	0.2	0.1	0.3
Raw Rockfish	0.06	ND	0.2
Crab Hepatopancreas	0.02	0.1	0.2
Cooked Crab Hepatopancreas	0.09	0.2	0.3
Raw Crab	0.1	ND	0.1
Cooked Crab	0.2	0.06	0.3
Mussel	0.02	ND	0.1
Prawn	0.6	ND	0.7

[a] The medium exposed 1 to 6 year old child was assumed to consumed seafood 24 days per year.

ND = Not detected

At medium exposure levels, the HQs that exceeded one for arsenic for both adults and children were all less than four and most were between one and two. HQs greater than one were predicted for three types of tissues: crabs, sole and prawns. In the case of PCBs, HQs were greatest in Duwamish River tissues and for children. The HQs ranged from 1.3 (perch - adults-Elliott Bay) to 19 (sole - whole body - child 1 to 6-Duwamish River). The highest HQ for any other chemical under the medium exposure level for a child aged 1 to 6 was for mercury in rockfish from the reference site (HQ = 0.7).

Risks Per Meal. As described in Section 3.1, evaluation of non-carcinogenic risks assumes that a threshold dose must be exceeded for effects to occur. This assumption allows the prediction of the point at which the cumulative dose (i.e., repeated exposures) would result in a exceedance of the threshold dose. Therefore, it is possible to present the number of meals per year that would be required to reach a HQ (or hazard index) equal to 1.0 (i.e., the threshold dose is exceeded). These risks were calculated for each tissue type for each age group evaluated and is summarized in Table 3-28 below. To calculate the number of meals per year required to exceed the threshold dose, the risk and exposure equations were rearranged and solved for exposure frequency. Medium exposure level assumptions were used for all parameters (except exposure frequency, which is being solved for) to calculate the required number of meals.

Comparison of Baseline Risks to the Without CSO Scenario. As was the case for the direct exposure pathways, the baseline scenario predicted HQs of a similar magnitude to the without CSO scenario. The tissue concentrations "without CSOs" were estimated as described in Section 3.2 by assuming that the tissue concentrations would change proportionally to the changes in the sediment and water concentrations.

There are some slight differences in the magnitudes of the HQs. However, the slight differences in the magnitudes of the HQs that were observed in some cases are not toxicologically significant. Overall, adjusting the estimates of the tissue concentrations made no difference in the predicted non-carcinogenic health risks from the seafood consumption pathway. Predicted non-carcinogenic risks for children aged one to six under the without CSO conditions at high exposure levels are presented in Table 3-29.

Comparison of Reference Locations. Overall, the HQs predicted at the reference locations were similar to those predicted for the Duwamish River and Elliott Bay. For some specific chemicals (e.g., PCBs) differences were noted. For arsenic, the magnitudes of the HQs were similar between all three locations when direct comparisons between tissue types were made. The largest difference between the study area and reference locations was noted for PCBs.

The general trend in the magnitude of the PCB HQs was: Duwamish River > Elliott Bay > Reference locations. These differences were most pronounced for fish, specifically sole, where the ratio of the HQs was approximately (100 > 10 >1). For shellfish the differences were less apparent, but both the Duwamish River and Elliott Bay PCB HQs appeared elevated over those predicted for the reference locations. TBT and lead also had generally higher HQs in tissues from the Duwamish River and Elliott Bay than in tissues from the reference sites.

Table 3-28. Number of Meals of Seafood that Must be Consumed Per Year Under Medium Exposure Assumptions to Obtain an HQ=1 Under Baseline Conditions

	Raw Sole Fillet	Sole - Whole Body	Perch – Whole Body	Salmon	Crab Hepatopancreas	Raw Crab	Mussel	Cooked Sole	Cooked Crab	Cooked Crab Hepatopancreas	Rockfish	Prawn	Squid
Duwamish River													
Child age 1 to 6													
Arsenic	14	24	136	225	74	8	232	10	24	N/AP	N/AP	N/AP	N/AP
PCBs	5	1	3	44	3	7	42	3	12	N/AP	N/AP	N/AP	N/AP
All Chemicals	4	1	3	33	3	4	23	2	7	N/AP	N/AP	N/AP	N/AP
Child age 7 to 12													
Arsenic	21	38	212	352	153	13	363	15	37	N/AP	N/AP	N/AP	N/AP
PCBs	8	2	5	69	7	11	65	4	19	N/AP	N/AP	N/AP	N/AP
All Chemicals	6	2	5	52	6	6	35	3	11	N/AP	N/AP	N/AP	N/AP
Child age 13 to 18													
Arsenic	32	58	323	535	257	20	552	23	57	N/AP	N/AP	N/AP	N/AP
PCBs	12	3	8	106	11	17	99	7	29	N/AP	N/AP	N/AP	N/AP
All Chemicals	9	3	7	78	10	9	54	5	17	N/AP	N/AP	N/AP	N/AP
Adult													
Arsenic	31	56	312	518	200	19	535	22	55	N/AP	N/AP	N/AP	N/AP
PCBs	12	3	8	102	9	17	96	6	28	N/AP	N/AP	N/AP	N/AP
All Chemicals	9	3	7	76	8	8	52	5	17	N/AP	N/AP	N/AP	N/AP

Table 3-28. Number of Meals of Seafood that Must be Consumed Per Year Under Medium Exposure Assumptions to Obtain an HQ=1 Under Baseline Conditions (continued)

	Raw Sole Fillet	Sole - Whole Body	Perch – Whole Body	Salmon	Crab Hepatopancreas	Raw Crab	Mussel	Cooked Sole	Cooked Crab	Cooked Crab Hepatopancreas	Rockfish	Prawn	Squid
Elliott Bay													
Child age 1 to 6													
Arsenic	25	30	183	N/AP	65	11	180	19	12	95	169	9	39
PCBs	97	11	8	N/AP	4	10	ND	20	28	5	22	ND	47
All Chemicals	18	8	7	N/AP	3	5	40	9	7	4	13	9	18
Child age 7 to 12													
Arsenic	38	47	286	N/AP	134	16	281	30	19	196	264	15	61
PCBs	151	17	12	N/AP	7	16	ND	32	44	10	34	ND	74
All Chemicals	28	12	11	N/AP	7	7	62	15	11	9	20	14	28
Child age 13 to 18													
Arsenic	58	71	436	N/AP	226	25	428	45	29	330	402	22	93
PCBs	230	26	18	N/AP	12	24	ND	49	68	17	51	ND	112
All Chemicals	43	18	16	N/AP	11	11	95	22	16	15	30	21	43
Adult													
Arsenic	57	69	422	N/AP	175	24	414	44	28	257	389	22	90
PCBs	222	26	18	N/AP	10	23	ND	47	65	13	50	ND	109
All Chemicals	42	18	29	N/AP	9	11	92	21	16	11	29	20	42

Table 3-28. Number of Meals of Seafood that Must be Consumed Per Year Under Medium Exposure Assumptions to Obtain an HQ=1 Under Baseline Conditions (continued)

	Raw Sole Fillet	Sole - Whole Body	Perch – Whole Body	Salmon	Crab Hepatopancreas	Raw Crab	Mussel	Cooked Sole	Cooked Crab	Cooked Crab Hepatopancreas	Rockfish	Prawn	Squid
Reference													
Child age 1 to 6													
Arsenic	17	25	193	27	215	52	249	12	28	44	83	8	N/AP
PCBs	ND	79	18	51	35	ND	ND	73	85	20	ND	ND	N/AP
All Chemicals	16	17	14	17	24	36	88	10	16	12	23	8	N/AP
Child age 7 to 12													
Arsenic	27	38	301	41	442	81	389	19	44	90	129	13	N/AP
PCBs	ND	124	28	80	72	ND	ND	115	133	42	ND	ND	N/AP
All Chemicals	26	27	22	26	50	57	138	16	25	26	36	12	N/AP
Child age 13 to 18													
Arsenic	41	59	458	63	745	124	593	29	67	152	196	20	N/AP
PCBs	ND	188	42	122	122	ND	ND	175	203	71	ND	ND	N/AP
All Chemicals	39	42	34	40	85	87	210	24	38	43	55	18	N/AP
Adult													
Arsenic	40	57	444	61	578	120	574	28	65	118	190	19	N/AP
PCBs	ND	182	41	118	94	ND	ND	169	197	55	ND	ND	N/AP
All Chemicals	38	40	33	38	66	84	204	23	37	33	54	17	N/AP

[a] Order high exposures scenario for adults

ND – Chemical not detected in this seafood.

N/AP – Not applicable seafood not collected and analyzed from this location.

Table 3-29. Without CSO Condition Seafood Consumption Hazard Quotients for a Highly Exposed Child Aged 1 to 6 for Chemicals with HQs Greater than One by Tissue Type[a]

Tissue	Chemical								
	Arsenic	Cadmium	Copper	Lead	Mercury	Zinc	TBT	PCBs	Hazard Index for all chemicals
Duwamish River									
Raw Sole	61.5	ND	<0.1	ND	2.4	0.3	0.2	159.7	224
Cooked Sole	87.4	ND	<0.1	ND	3.9	0.3	0.6	299.0	391
Sole-Whole Body	34.4	ND	<0.1	0.6	2.3	0.9	0.6	662.7	702
Perch-Whole Body	6.1	0.1	<0.1	0.6	2.7	0.7	8.0	244.2	263
Salmon	3.7	N/AP	<0.1	<0.1	2.3	N/AP	N/AP	18.6	25
Crab Hepatopancreas	8.6	0.2	0.3	0.2	0.6	0.3	0.8	196.2	207
Raw Crab	99.0	0.2	0.5	0.8	4.8	2.6	6.9	114.3	229
Cooked Crab	34.8	0.3	0.6	0.2	6.5	2.2	3.5	66.3	114
Mussel	3.6	2.3	<0.1	1.6	0.4	1.7	2.8	19.6	33
Elliott Bay									
Raw Sole	34.0	ND	<0.1	ND	3.0	0.3	0.1	8.6	46
Cooked Sole	44.2	ND	<0.1	ND	4.1	0.3	0.2	40.4	89
Sole-Whole Body	27.9	ND	<0.1	0.4	2.3	0.9	0.5	74.6	107
Perch-Whole Body	4.5	0.1	<0.1	0.5	0.9	0.7	7.7	105.8	121
Raw Rockfish	4.9	ND	<0.1	ND	20.4	0.1	2.1	38.1	66
Crab Hepatopancreas	9.8	2.3	0.2	0.1	0.5	0.6	0.8	174.0	188
Cooked Crab Hepatopancreas	6.7	4.8	0.3	0.3	0.5	0.7	1.1	125.7	140
Raw Crab	78.9	2.2	0.4	1.1	3.7	2.5	3.5	82.0	174
Cooked Crab	67.6	1.0	0.6	0.2	12.5	3.1	4.9	28.9	119
Mussel	4.6	3.0	<0.1	2.8	0.4	1.9	2.7	ND	16
Prawn	88.7	<0.1	0.1	ND	1.5	0.4	2.7	ND	94
Whole Squid	21.3	0.8	1.2	ND	1.5	0.6	1.5	17.4	45

[a] The medium exposed 1 to 6 year old child was assumed to consumed seafood 24 days per year.

Comparison of Tissue Types. Overall, results between tissue types were generally similar, but some exceptions were apparent. In general, the highest PCB HQs were predicted for raw sole and cooked sole - whole body, and crab hepatopancreas raw and cooked. The highest metal concentrations tended to occur in shellfish tissues, particularly the crab and mussels, followed by prawns and squid. An exception to this is mercury, resulting in higher HQs in fish tissues in Elliott Bay and reference locations. This result is likely due to the fact that the majority of mercury in fish tissues occurs in the organic form, particularly methylmercury, the primary form of organic mercury in seafoods (Bloom 1992). Due to the accumulation of methylmercury over the lifetime of the fish, the larger fish may be expected to have higher concentrations of measured total mercury.

Carcinogenic Risks. The carcinogenic risk results from the seafood consumption pathway vary significantly between exposure levels. Using the high exposure assumptions (365 meals per year for 75 years), a number of COPCs are predicted to present incremental cancer risks greater than one in one million (10^{-6}) for both adults and children. However, when risks were evaluated using the low exposure assumptions (eight meals per year for nine years), arsenic and PCBs are the only two chemicals identified as presenting incremental cancer risks greater than one in one million. Because of the considerable difference in these results by exposure level, the results are discussed separately by exposure level below. As previously discussed, for the vast majority of the potentially exposed population (i.e., people who consume seafood taken from Elliott Bay or the Duwamish River) exposures are expected to occur at the medium level or below. For more discussion of this issue, see Section 3.2 or Issue Paper No. 3 "*Human Site Uses*" (Appendix C).

Risks Predicted Under High Exposure Assumptions. Under high exposure assumptions incremental cancer risks exceeding one in one million (10^{-6}) were predicted for adults and all three child age groups, although slightly higher risks were predicted for adults due to their long exposure time (75 years) relative to that for children (6 years). The COPCs predicted to present incremental cancer risks greater than one in one million for one or more tissue types at either the Duwamish River or Elliott Bay include: arsenic, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, bis(2-ethylhexyl)phthalate, chyrsene, and PCBs.

Two of the above chemicals present the majority of carcinogenic risk potential: arsenic and PCBs. These two chemicals are clearly the risk "drivers" in all scenarios evaluated. In general, the risks predicted by these two COPCs range from 10 to 1,000 times greater than potential risks predicted by other COPCs. However, risks above one in a million are predicted for other COPCs. The risks predicted for these other chemicals are relatively lower than the risks from arsenic and PCBs.

The range of predicted incremental cancer risks for different tissue types and age groups are presented in Table 3-30 for the Duwamish River, Elliott Bay, and the reference sites.

Table 3-30. Baseline Condition Range of Predicted Cancer Risks for Different Species Under High Exposure Levels for Adults and Children of All Age Groups

Chemical	Duwamish River	Elliott Bay	Reference Sites
Arsenic	$5x10^{-5}$ – $2x10^{-2}$	$6x10^{-5}$ – $2x10^{-2}$	$3x10^{-3}$ – $2x10^{-2}$
Benzo(a)anthracene	ND – $7x10^{-5}$	ND – $1x10^{-4}$	ND
Benzo(a)pyrene	ND	ND – $1x10^{-3}$	ND
Benzo(b)fluoranthene	ND – $9x10^{-5}$	ND	ND
Bis(2-ethylhexyl)phthalate	ND – $5x10^{-5}$	ND – $8x10^{-6}$	ND – $1x10^{-5}$
Chyrsene	ND – $1x10^{-6}$	ND – $1x10^{-6}$	ND
PCBs	$2x10^{-5}$ – $2x10^{-2}$	ND – $6x10^{-3}$	ND – $1x10^{-3}$

ND = Not detected in at least one tissue type

Predicted risks from PCBs were somewhat lower than those for arsenic but were also consistently elevated above one in a million. Similar to arsenic the predicted risks ranged widely over several orders of magnitude and were consistent across tissue type and location. However, similar to the pattern observed in the non-carcinogenic PCB HQs, the carcinogenic risk predictions for PCBs were higher in Duwamish River tissues than in Elliott Bay or reference tissues. The predicted incremental risks to adults from daily seafood consumption for 75 years are summarized by tissue type in Table 3-31. Incremental risks to adults were about 5 to 14 times higher than the predicted risks to children. These differences were largely associated with differences in the exposure duration between adults (75 years) and children (6 years) at the high exposure level.

As described above, potential carcinogenic risks were predicted for several PAHs and bis(2-ethylhexyl)phthalate. The magnitudes of the carcinogenic risk predictions ranged from one in one hundred million to one in ten thousand. Although PAH risks above one in a million are predicted for a few tissues in Duwamish River and Elliott Bay, the magnitudes of these risks are relatively small when compared to risks predicted from exposure to arsenic and PCBs in the same tissues and exposure scenarios. Additionally, the PAHs were detected infrequently compared to arsenic and PCBs.

Table 3-31. Baseline Condition Predicted Incremental Carcinogenic Risks to Adults at High Exposure Levels (365 meals/yr) from Consumption of Seafood from the Duwamish River, Elliott Bay, and Reference Sites

	Chemical										
Tissue	**Arsenic**	**Aroclor 1248**	**Aroclor 1254**	**Aroclor 1260**	**Total PCBs**	**Chrysene**	**Benzo(a)anthracene**	**Benzo(a)pyrene**	**Benzo(b)fluoranthene**	**Bis(2-ethylhexyl)phthalate**	**Chemical Total**
Duwamish River											
Sole	$1x10^{-2}$	ND	$3x10^{-3}$	$2x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$2x10^{-2}$
Sole – Whole Body	$7x10^{-3}$	$1x10^{-3}$	$1x10^{-2}$	$9x10^{-3}$	N/AP	N/AP	N/AP	N/AP	N/AP	N/AP	$3x10^{-2}$
Perch – Whole Body	$1x10^{-3}$	ND	$4x10^{-3}$	$3x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$9x10^{-3}$
Salmon	$7x10^{-4}$	ND	$3x10^{-4}$	$2x10^{-4}$	$6x10^{-4}$	N/AP	N/AP	N/AP	N/AP	$5x10^{-5}$	$2x10^{-3}$
Crab Hepatopancreas	$2x10^{-3}$	$4x10^{-4}$	$3x10^{-3}$	$1x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$7x10^{-3}$
Crab	$2x10^{-2}$	$3x10^{-4}$	$2x10^{-3}$	$5x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$2x10^{-2}$
Mussel	$7x10^{-4}$	ND	$4x10^{-4}$	ND	N/AP	$1x10^{-6}$	$7x10^{-5}$	ND	$9x10^{-5}$	$3x10^{-6}$	$1x10^{-3}$
Cooked Sole	$2x10^{-2}$	$3x10^{-4}$	$5x10^{-3}$	$3x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$3x10^{-2}$
Cooked Crab	$7x10^{-3}$	ND	$1x10^{-3}$	$7x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$9x10^{-3}$
Elliott Bay											
Sole	$7x10^{-3}$	ND	$2x10^{-4}$	$1x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$7x10^{-3}$
Sole – Whole Body	$6x10^{-3}$	ND	$1x10^{-3}$	$2x10^{-3}$	N/AP	N/AP	N/AP	N/AP	N/AP	N/AP	$9x10^{-3}$
Raw Rock Fish	$1x10^{-3}$	ND	$7x10^{-4}$	$1x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$3x10^{-3}$
Perch – Whole Body	$9x10^{-4}$	ND	$2x10^{-3}$	$1x10^{-3}$	N/AP	ND	ND	$2x10^{-3}$	ND	ND	$6x10^{-3}$
Crab Hepatopancreas	$2x10^{-3}$	ND	$3x10^{-3}$	$3x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$8x10^{-3}$
Crab	$2x10^{-2}$	ND	$1x10^{-3}$	$1x10^{-3}$	N/AP	$1x10^{-6}$	$1x10^{-4}$	$1x10^{-3}$	ND	ND	$2x10^{-2}$
Mussel	$9x10^{-4}$	ND	ND	ND	N/AP	$1x10^{-6}$	$1x10^{-4}$	ND	ND	ND	$1x10^{-3}$
Prawn	$2x10^{-2}$	ND	ND	$1x10^{-4}$	N/AP	ND	ND	ND	ND	4x10-6	$2x10^{-2}$
Squid	$4x10^{-3}$	ND	$3x10^{-4}$	ND	N/AP	ND	ND	ND	ND	8x10-6	$5x10^{-3}$
Cooked Sole	$9x10^{-3}$	ND	$7x10^{-4}$	$7x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$1x10^{-2}$

Table 3-31. Baseline Condition Predicted Incremental Carcinogenic Risks to Adults at High Exposure Levels (365 meals/yr) from Consumption of Seafood from the Duwamish River, Elliott Bay, and Reference Sites (continued)

	Chemical										
Tissue	**Arsenic**	**Aroclor 1248**	**Aroclor 1254**	**Aroclor 1260**	**Total PCBs**	**Chrysene**	**Benzo(a)anthracene**	**Benzo(a)pyrene**	**Benzo(b)fluoranthene**	**Bis(2-ethylhexyl)phthalate**	**Chemical Total**
Reference Site											
Cooked Crab	$1x10^{-2}$	ND	$5x10^{-4}$	$4x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$1x10^{-2}$
Cooked Crab Hepatopancreas	$1x10^{-3}$	ND	$2x10^{-3}$	$2x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$5x10^{-3}$
Raw Rock Fish	$2x10^{-3}$	ND	ND	ND	N/AP	ND	ND	ND	ND	ND	$2x10^{-3}$
Perch – Whole Body	$9x10^{-4}$	ND	$8x10^{-4}$	$3x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$2x10^{-3}$
Salmon	$6x10^{-3}$	$1x10^{-5}$	$3x10^{-4}$	$2x10^{-4}$	$4x10^{-4}$	ND	ND	ND	ND	$1x10^{-5}$	$7x10^{-3}$
Crab Hepatopancreas	$5x10^{-4}$	ND	$3x10^{-4}$	$2x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$1x10^{-3}$
Crab	$3x10^{-3}$	ND	ND	ND	N/AP	ND	ND	ND	ND	ND	$3x10^{-3}$
Mussel	$7x10^{-4}$	ND	ND	ND	N/AP	ND	ND	ND	ND	ND	$7x10^{-4}$
Prawn	$2x10^{-2}$	ND	ND	ND	N/AP	ND	ND	ND	ND	$4x10^{-6}$	$2x10^{-2}$
Cooked Sole	$1x10^{-2}$	ND	$2x10^{-4}$	ND	N/AP	ND	ND	ND	ND	ND	$1x10^{-2}$
Cooked Crab	$6x10^{-3}$	ND	$2x10^{-4}$	ND	N/AP	ND	ND	ND	ND	ND	$6x10^{-3}$
Cooked Crab Hepatopancreas	$3x10^{-3}$	ND	$5x10^{-4}$	$3x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$4x10^{-3}$

ND = Not detected

N/AP = Not applicable because chemical was not analyzed

Risks Predicted Under Medium and Low Exposure Assumptions. Similar to the carcinogenic risks predicted under high exposure conditions, carcinogenic risks exceeding one in one million were predicted under medium and low exposure assumptions for adults and all three-child age groups. Carcinogenic risks were lower under the low exposure assumptions than under the medium exposure assumptions. Fewer chemicals exceed one in one million for at least one tissue type under the medium and low exposure levels than the high exposure level. Chemicals that exceed one in one million under the medium exposure assumptions in at least one tissue type include arsenic, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, and PCBs.

Chemicals that exceed one in one million under the low exposure assumptions in at least one tissue type include: arsenic, benzo(a)pyrene, and PCBs.

The magnitudes of the cancer risks varied by tissue type although for arsenic the magnitude of the cancer risks were similar between the Duwamish River, Elliott Bay, and the reference locations. For PCBs, the magnitude of the cancer risks was greatest in tissues from the Duwamish River. These results are summarized below in Table 3-32 for the medium exposure assumptions (for adults) and Table 3-33 for the low exposure assumptions (for children ages 1 to 6).

Under medium exposure conditions all of the predicted risks to adults from bis(2-ethylhexyl)phthalate are less than one in a million, while benzo(a)anthrancene risks in Elliott Bay crab and mussels and benzo(b)fluoranthene in Duwamish mussels are approximately at this level at 1.7, 1.4, and 1.1 in a million , respectively. Chrysene also predicted risks at approximately the one in a million level in shellfish tissues in the Duwamish River and Elliott Bay. Benzo(a)pyrene predicted risks remain the highest at this exposure with the predicted risks for adults at 2 in 100,000 in crab and perch in Elliott Bay. All other PAH risks are well below the one in a million level.

These results suggest that for people (both adults and children) who consume seafood from the Duwamish River, Elliott Bay, and other areas of Puget Sound over many years, potential carcinogenic risks from arsenic and PCBs exist. Potential risks from several PAHs are also predicted, but these risks are lower that those occurring from the two risk drivers and are infrequently detected in seafood tissues. Arsenic risks are of similar magnitude at all locations evaluated while risks from PCBs are elevated in the Duwamish River, relative to Elliott Bay and reference sites.

Risks Associated with Individual Meals of Specific Seafoods. Carcinogenic risk potential was calculated on a meal-specific basis. Carcinogenic risks are assumed to be cumulative. Therefore the incremental dose (i.e., number of meals) required to reach a specific risk level may be calculated. Table 3-34 summarizes relative risks (by meal) for each tissue type.

Comparison of Baseline Risks to the Without CSO Scenario. When potential risks estimated in the baseline and without CSO scenarios were compared, the risk estimates were nearly identical. In some cases, the chemical-specific risk estimates did vary slightly between scenarios. However, as described above, the risk estimates did not differ enough to change the interpretation of the risk estimates between scenarios. Thus, the carcinogenic risk potential both with and without CSOs are predicted to be the same for adults and all three-child age groups. Predicted incremental carcinogenic risks to adults at high exposure levels under without CSO conditions are presented in Table 3-35 for consumption of seafood from the Duwamish River and Elliott Bay.

Comparison of Reference Locations. In general, arsenic risks were elevated at all locations evaluated. These results suggest that arsenic concentrations in seafood tissues in Puget Sound are not significantly influenced by any factors specific to the WQA study area. In contrast, PCB risks were elevated above those predicted at reference locations.

The general trend of PCB risk potential by location was Duwamish River > Elliott Bay > reference locations.

Table 3-32. Baseline Condition Predicted Incremental Carcinogenic Risks to Adults at Medium Exposure Levels (24 Meals/Year) from Consumption of Seafood from the Duwamish River, Elliott Bay, and Reference Sites

Tissue	Arsenic	Aroclor 1248	Aroclor 1254	Aroclor 1260	Total PCBs	Benzo(a)anthracene	Benzo(a)pyrene	Benzo(b)fluoranthene	Chemical Total
Duwamish River									
Sole	$2x10^{-4}$	ND	$4x10^{-5}$	$2x10^{-5}$	N/AP	ND	ND	ND	$2x10^{-4}$
Sole – Whole Body	$8x10^{-5}$	$1x10^{-5}$	$1x10^{-4}$	$1x10^{-4}$	N/AP	N/AP	N/AP	N/AP	$4x10^{-4}$
Perch – Whole Body	$2x10^{-5}$	ND	$5x10^{-5}$	$4x10^{-5}$	N/AP	ND	ND	ND	$1x10^{-4}$
Salmon	$9x10^{-6}$	ND	$4x10^{-6}$	$3x10^{-6}$	$7x10^{-6}$	N/AP	N/AP	N/AP	$2x10^{-5}$
Crab Hepatopancreas	$2x10^{-5}$	$6x10^{-6}$	$5x10^{-5}$	$2x10^{-5}$	N/AP	ND	ND	ND	$1x10^{-4}$
Crab	$2x10^{-4}$	$3x10^{-6}$	$3x10^{-5}$	$6x10^{-6}$	N/AP	ND	ND	ND	$3x10^{-4}$
Mussel	$9x10^{-6}$	ND	$4x10^{-6}$	ND	N/AP	$9x10^{-7}$	ND	$1x10^{-6}$	$2x10^{-5}$
Cooked Sole	$2x10^{-4}$	$4x10^{-6}$	$7x10^{-5}$	$4x10^{-5}$	N/AP	ND	ND	ND	$3x10^{-4}$
Cooked Crab	$9x10^{-5}$	ND	$1x10^{-5}$	$8x10^{-6}$	N/AP	ND	ND	ND	$1x10^{-4}$
Elliott Bay									
Sole	$8x10^{-5}$	ND	$2x10^{-6}$	$2x10^{-6}$	N/AP	ND	ND	ND	$9x10^{-5}$
Sole – Whole Body	$7x10^{-5}$	ND	$2x10^{-5}$	$2x10^{-5}$	N/AP	N/AP	N/AP	N/AP	$1x10^{-4}$
Raw Rock Fish	$1x10^{-5}$	ND	$9x10^{-6}$	$2x10^{-5}$	N/AP	ND	ND	ND	$4x10^{-5}$
Perch – Whole Body	$1x10^{-5}$	ND	$2x10^{-5}$	$1x10^{-5}$	N/AP	ND	$2x10^{-5}$	ND	$7x10^{-5}$
Crab Hepatopancreas	$3x10^{-5}$	ND	$4x10^{-5}$	$5x10^{-5}$	N/AP	ND	ND	ND	$1x10^{-4}$
Crab	$2x10^{-4}$	ND	$2x10^{-5}$	$1x10^{-5}$	N/AP	$2x10^{-6}$	$2x10^{-5}$	ND	$2x10^{-4}$
Mussel	$1x10^{-5}$	ND	ND	ND	N/AP	$1x10^{-6}$	ND	ND	$1x10^{-5}$
Prawn	$2x10^{-4}$	ND	ND	$2x10^{-6}$	N/AP	ND	ND	ND	$2x10^{-4}$
Squid	$5x10^{-5}$	ND	$4x10^{-6}$	ND	N/AP	ND	ND	ND	$6x10^{-5}$
Cooked Sole	$1x10^{-4}$	ND	$9x10^{-6}$	$8x10^{-6}$	N/AP	ND	ND	ND	$1x10^{-4}$

Table 3-32. Baseline Condition Predicted Incremental Carcinogenic Risks to Adults at Medium Exposure Levels (24 Meals/Year) from Consumption of Seafood from the Duwamish River, Elliott Bay, and Reference Sites (continued)

Tissue	Arsenic	Aroclor 1248	Aroclor 1254	Aroclor 1260	Total PCBs	Benzo(a)anthracene	Benzo(a)pyrene	Benzo(b)fluoranthene	Chemical Total
Cooked Crab	$2x10^{-4}$	ND	$6x10^{-6}$	$5x10^{-6}$	N/AP	ND	ND	ND	$2x10^{-4}$
Cooked Crab Hepatopancreas	$2x10^{-5}$	ND	$3x10^{-5}$	$3x10^{-5}$	N/AP	ND	ND	ND	$8x10^{-5}$
Reference Site									
Sole	$1x10^{-4}$	ND	ND	ND	N/AP	ND	ND	ND	$1x10^{-4}$
Sole – Whole Body	$8x10^{-5}$	ND	$2x10^{-6}$	ND	N/AP	N/AP	N/AP	N/AP	$9x10^{-5}$
Raw Rock Fish	$2x10^{-5}$	ND	ND	ND	N/AP	ND	ND	ND	$2x10^{-5}$
Perch – Whole Body	$1x10^{-5}$	ND	$1x10^{-5}$	$4x10^{-6}$	N/AP	ND	ND	ND	$2x10^{-5}$
Salmon	$8x10^{-5}$	$2x10^{-7}$	$4x10^{-6}$	$2x10^{-6}$	$6x10^{-6}$	ND	ND	ND	$9x10^{-5}$
Crab Hepatopancreas	$8x10^{-6}$	ND	$4x10^{-6}$	$3x10^{-6}$	N/AP	ND	ND	ND	$2x10^{-5}$
Crab	$4x10^{-5}$	ND	ND	ND	N/AP	ND	ND	ND	$4x10^{-5}$
Mussel	$9x10^{-6}$	ND	ND	ND	N/AP	ND	ND	ND	$9x10^{-6}$
Prawn	$2x10^{-4}$	ND	ND	ND	N/AP	ND	ND	ND	$2x10^{-4}$
Cooked Sole	$2x10^{-4}$	ND	$2x10^{-6}$	ND	N/AP	ND	ND	ND	$2x10^{-4}$
Cooked Crab	$7x10^{-5}$	ND	$2x10^{-6}$	ND	N/AP	ND	ND	ND	$8x10^{-5}$
Cooked Crab Hepatopancreas	$4x10^{-5}$	ND	$8x10^{-6}$	$5x10^{-6}$	N/AP	ND	ND	ND	$5x10^{-5}$

ND = Not detected

N/AP = Not applicable because chemical was not analyzed

Table 3-33. Baseline Condition Predicted Incremental Carcinogenic Risks to Children Aged 1 to 6 at Low Exposure Levels (8 Meals/Year) from Consumption of Seafood from the Duwamish River, Elliott Bay, and Reference Sites

Tissue	Arsenic	Aroclor 1248	Aroclor 1254	Aroclor 1260	Benzo(a) pyrene	Chemical Total
Duwamish River						
Sole	$1x10^{-5}$	ND	$3x10^{-6}$	$2x10^{-6}$	ND	$2x10^{-5}$
Sole – Whole Body	$8x10^{-6}$	$1x10^{-6}$	$1x10^{-5}$	$9x10^{-6}$	N/AP	$3x10^{-5}$
Perch – Whole Body	$1x10^{-6}$	ND	$5x10^{-6}$	$3x10^{-6}$	ND	$9x10^{-6}$
Salmon	$8x10^{-7}$	ND	$4x10^{-7}$	$3x10^{-7}$	N/AP	$2x10^{-6}$
Crab Hepatopancreas	$2x10^{-6}$	$4x10^{-7}$	$4x10^{-6}$	$2x10^{-6}$	ND	$8x10^{-6}$
Crab	$2x10^{-5}$	$3x10^{-7}$	$2x10^{-6}$	$6x10^{-7}$	ND	$2x10^{-5}$
Mussel	$8x10^{-7}$	ND	$4x10^{-7}$	ND	ND	$1x10^{-6}$
Cooked Sole	$2x10^{-5}$	$4x10^{-7}$	$6x10^{-6}$	$3x10^{-6}$	ND	$3x10^{-5}$
Cooked Crab	$8x10^{-6}$	ND	$1x10^{-6}$	$7x10^{-7}$	ND	$1x10^{-5}$
Elliott Bay						
Sole	$7x10^{-6}$	ND	$2x10^{-7}$	$1x10^{-7}$	ND	$8x10^{-6}$
Sole – Whole Body	$6x10^{-6}$	ND	$1x10^{-6}$	$2x10^{-6}$	N/AP	$1x10^{-5}$
Raw Rock Fish	$1x10^{-6}$	ND	$8x10^{-7}$	$1x10^{-6}$	ND	$3x10^{-6}$
Perch – Whole Body	$1x10^{-6}$	ND	$2x10^{-6}$	$1x10^{-6}$	$2x10^{-6}$	$6x10^{-6}$
Crab Hepatopancreas	$2x10^{-6}$	ND	$3x10^{-6}$	$4x10^{-6}$	ND	$9x10^{-6}$
Crab	$2x10^{-5}$	ND	$2x10^{-6}$	$1x10^{-6}$	$2x10^{-6}$	$2x10^{-5}$
Mussel	$1x10^{-6}$	ND	ND	ND	ND	$1x10^{-6}$
Prawn	$2x10^{-5}$	ND	ND	$2x10^{-7}$	ND	$2x10^{-5}$
Squid	$5x10^{-6}$	ND	$3x10^{-7}$	ND	ND	$5x10^{-6}$
Cooked Sole	$1x10^{-5}$	ND	$8x10^{-7}$	$7x10^{-7}$	ND	$1x10^{-5}$
Cooked Crab	$1x10^{-5}$	ND	$6x10^{-7}$	$4x10^{-7}$	ND	$2x10^{-5}$
Cooked Crab Hepatopancreas	$1x10^{-6}$	ND	$2x10^{-6}$	$2x10^{-6}$	ND	$6x10^{-6}$
Reference Site						
Sole	$1x10^{-5}$	ND	ND	ND	ND	$1x10^{-5}$
Sole – Whole Body	$7x10^{-6}$	ND	$2x10^{-7}$	ND	N/AP	$8x10^{-6}$
Raw Rock Fish	$2x10^{-6}$	ND	ND	ND	ND	$2x10^{-6}$
Perch – Whole Body	$9x10^{-7}$	ND	$9x10^{-7}$	$3x10^{-7}$	ND	$2x10^{-6}$
Salmon	$7x10^{6}$	$2x10^{8}$	$3x10^{7}$	$2x10^{7}$	ND	$8x10^{6}$
Crab Hepatopancreas	$6x10^{-7}$	ND	$3x10^{-7}$	$2x10^{-7}$	ND	$1x10^{-6}$
Crab	$4x10^{-6}$	ND	ND	ND	ND	$4x10^{-6}$
Mussel	$8x10^{-7}$	ND	ND	ND	ND	$8x10^{-7}$
Prawn	$2x10^{-5}$	ND	ND	ND	ND	$2x10^{-5}$
Cooked Sole	$1x10^{-5}$	ND	$2x10^{-7}$	ND	ND	$2x10^{-5}$
Cooked Crab	$6x10^{-6}$	ND	$2x10^{-7}$	ND	ND	$7x10^{-6}$
Cooked Crab Hepatopancreas	$3x10^{-6}$	ND	$6x10^{-7}$	$4x10^{-7}$	ND	$4x10^{-6}$

ND = Not detected

N/AP = Not applicable because chemical was not analyzed

Table 3-34. Human Health: Number of Meals Required Per Year to Achieve Lifetime Carcinogenic Risk of One in a Million[a]

	Raw Sole Fillet	Sole - Whole Body	Perch	Salmon	Crab Hepatopancreas	Raw Crab	Mussel	Cooked Sole	Cooked Crab	Cooked Crab Hepatopancreas	Rockfish	Prawn	Squid
Duwamish River													
Child age 1 to 6													
Arsenic	0.4	0.7	3.8	6.3	2.1	0.2	6.4	0.3	0.7	N/AP	N/AP	N/AP	N/AP
PCBs	1.6	0.4	1.0	7.7	1.0	2.2	13	0.9	3.9	N/AP	N/AP	N/AP	N/AP
All Chemicals	0.3	0.2	0.5	2.4	0.5	0.2	3.8	0.2	0.5	N/AP	N/AP	N/AP	N/AP
Child age 7 to 12													
Arsenic	0.6	1.1	5.9	9.8	4.2	0.4	10.1	0.4	1.0	N/AP	N/AP	N/AP	N/AP
PCBs	2.5	0.6	1.6	12.0	2.1	3.5	20.4	1.4	6.0	N/AP	N/AP	N/AP	N/AP
All Chemicals	0.4	0.3	0.8	3.7	1.0	0.3	5.9	0.3	0.8	ND	N/AP	N/AP	N/AP
Child age 13 to 18													
Arsenic	0.9	1.6	9.0	14.9	7.1	0.6	15.3	0.6	1.6	N/AP	N/AP	N/AP	N/AP
PCBs	3.8	0.9	2.5	18.3	3.5	5.3	31	2.1	9.2	N/AP	N/AP	N/AP	N/AP
All Chemicals	0.7	0.4	1.3	5.6	1.7	0.5	8.9	0.4	1.2	N/AP	N/AP	N/AP	N/AP
Adult													
Arsenic	0.2	0.3	1.6	2.6	1	0.1	2.7	0.1	0.3	N/AP	N/AP	N/AP	N/AP
PCBs	0.7	0.2	0.4	3.2	0.5	0.9	5.5	0.4	1.6	N/AP	N/AP	N/AP	N/AP
All Chemicals	0.1	0.1	0.2	1.0	0.2	0.1	1.6	0.1	0.2	N/AP	N/AP	N/AP	N/AP

[a] Calculated using medium exposure assumptions. Exposures assumed to occur for 33 years for adults and 6 years for children.

Table 3-34. Human Health: Number of Meals Required to Achieve Lifetime Carcinogenic Risk of One in a Million (continued)

	Raw Sole Fillet	Sole - Whole Body	Perch	Salmon	Crab Hepatopancreas	Raw Crab	Mussel	Cooked Sole	Cooked Crab	Cooked Crab Hepatopancreas	Rockfish	Prawn	Squid
Elliott Bay													
Child age 1 to 6													
Arsenic	0.7	0.8	5.1	N/AP	1.8	0.3	5.0	0.5	0.3	2.6	4.7	0.3	1.1
PCBs	30.2	2.3	2.4	N/AP	1.1	3.1	ND	6.4	8.9	1.6	3.6	32.6	14.8
All Chemicals	0.7	0.5	0.8	N/AP	0.4	0.2	4.4	0.5	0.3	0.6	1.6	0.3	1.0
Child age 7 to 12													
Arsenic	1.1	1.3	8.0	N/AP	3.7	0.5	7.8	0.8	0.5	5.4	7.3	0.4	1.7
PCBs	47.1	3.6	3.8	N/AP	2.2	4.9	ND	10.0	13.9	3.2	5.6	50.9	23.1
All Chemicals	1.0	0.8	1.3	N/AP	0.9	0.4	6.9	0.7	0.5	1.2	2.4	0.4	1.6
Child age 13 to 18													
Arsenic	1.6	2.0	12.1	N/AP	6.3	0.7	11.9	1.2	0.8	9.2	11.2	0.6	2.6
PCBs	71.7	5.5	5.8	N/AP	3.7	7.4	ND	15.2	21.1	5.4	8.5	77.5	35.1
All Chemicals	1.6	1.2	1.9	N/AP	1.5	0.6	10.5	1.1	0.8	2.1	3.7	0.6	2.4
Adult													
Arsenic	0.3	0.3	2.1	N/AP	0.9	0.1	2.1	0.2	0.1	1.3	2.0	0.1	0.5
PCBs	12.6	1.0	1.0	N/AP	0.5	1.3	ND	2.7	3.7	0.8	1.5	13.6	6.2
All Chemicals	0.3	0.2	0.3	N/AP	0.2	0.1	1.9	0.2	0.1	0.3	0.7	0.1	0.4

Table 3-34. Human Health: Number of Meals Required to Achieve Lifetime Carcinogenic Risk of One in a Million (continued)

	Raw Sole Fillet	Sole - Whole Body	Perch	Salmon	Crab Hepatopancreas	Raw Crab	Mussel	Cooked Sole	Cooked Crab	Cooked Crab Hepatopancreas	Rockfish	Prawn	Squid
Reference													
Child age 1 to 6													
Arsenic	0.5	0.7	5.4	0.7	6.0	1.4	6.4	0.3	0.8	1.2	23	0.2	N/AP
PCBs	ND	24.7	5.6	10.2	10.9	ND	ND	23.0	26.7	6.4	ND	ND	N/AP
All Chemicals	0.5	0.7	2.3	0.6	3.1	1.4	6.4	0.3	0.8	0.9	2.3	0.2	N/AP
Child age 7 to 12													
Arsenic	0.7	1.1	8.4	1.2	12.3	2.3	10.1	0.5	1.2	2.5	3.6	0.4	N/AP
PCBs	ND	38.6	8.7	15.9	22.5	ND	ND	35.9	41.7	13.1	ND	ND	N/AP
All Chemicals	0.7	1.0	3.6	1.0	6.4	2.3	10.1	0.5	1.2	1.9	3.6	0.4	N/AP
Child age 13 to 18													
Arsenic	1.1	1.6	12.7	1.8	26.7	3.4	15.3	0.8	1.9	4.2	5.5	0.6	N/AP
PCBs	ND	58.8	13.2	24.3	38	ND	ND	54.6	63.5	22.1	ND	ND	N/AP
All Chemicals	1.1	1.6	5.5	1.5	10.7	3.4	15.3	0.8	1.8	3.2	5.5	0.6	N/AP
Adult													
Arsenic	0.2	0.3	2.2	0.3	2.9	0.6	2.7	0.1	0.3	0.6	1.0	0.1	N/AP
PCBs	ND	10.4	2.3	4.3	5.4	ND	ND	9.6	11.2	3.1	ND	ND	N/AP
All Chemicals	0.2	0.3	1.0	0.3	1.5	0.6	2.7	0.1	0.3	0.5	1.0	0.1	N/AP

ND – Chemical not detected in this seafood.

N/AP – Not applicable seafood not collected and analyzed from this location.

Table 3-35. Without CSO Conditions Predicted Incremental Carcinogenic Risks to Adults at High Exposure Levels (365 meals/yr) from Consumption of Seafood from the Duwamish River and Elliott Bay

	Chemical										
Tissue	**Arsenic**	**Aroclor 1248**	**Aroclor 1254**	**Aroclor 1260**	**Total PCBs**	**Chrysene**	**Benzo(a)anthracene**	**Benzo(a)pyrene**	**Benzo(b)fluoranthene**	**Bis(2-ethylhexyl) phthalate**	**Chemical Total**
Duwamish River											
Sole	$1x10^{-2}$	ND	$3x10^{-3}$	$2x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$2x10^{-2}$
Sole – Whole Body	$7x10^{-3}$	$1x10^{-3}$	$1x10^{-2}$	$9x10^{-3}$	N/AP	N/AP	N/AP	N/AP	N/AP	N/AP	$3x10^{-2}$
Perch – Whole Body	$1x10^{-3}$	ND	$4x10^{-3}$	$3x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$8x10^{-3}$
Salmon	$7x10^{-4}$	ND	$3x10^{-4}$	$2x10^{-4}$	$6x10^{-4}$	N/AP	N/AP	N/AP	N/AP	$5x10^{-5}$	$2x10^{-3}$
Crab Hepatopancreas	$2x10^{-3}$	$4x10^{-4}$	$3x10^{-3}$	$1x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$7x10^{-3}$
Crab	$2x10^{-2}$	$3x10^{-4}$	$2x10^{-3}$	$5x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$2x10^{-2}$
Mussel	$7x10^{-4}$	ND	$4x10^{-4}$	ND	N/AP	$1x10^{-6}$	$7x10^{-5}$	ND	$9x10^{-5}$	$2x10^{-5}$	$1x10^{-3}$
Cooked Sole	$2x10^{-2}$	$3x10^{-4}$	$5x10^{-3}$	$3x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$3x10^{-2}$
Cooked Crab	$7x10^{-3}$	ND	$1x10^{-3}$	$7x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$9x10^{-3}$
Elliott Bay											
Sole	$7x10^{-3}$	ND	$2x10^{-4}$	$1x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$7x10^{-3}$
Sole – Whole Body	$6x10^{-3}$	ND	$1x10^{-3}$	$2x10^{-3}$	N/AP	N/AP	N/AP	N/AP	N/AP	N/AP	$9x10^{-3}$
Raw Rock Fish	$1x10^{-3}$	ND	$7x10^{-4}$	$1x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$3x10^{-3}$
Perch – Whole Body	$9x10^{-4}$	ND	$2x10^{-3}$	$1x10^{-3}$	N/AP	ND	ND	$2x10^{-3}$	ND	ND	$6x10^{-3}$
Crab Hepatopancreas	$2x10^{-3}$	ND	$3x10^{-3}$	$3x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$8x10^{-3}$
Crab	$2x10^{-2}$	ND	$1x10^{-3}$	$1x10^{-3}$	N/AP	$1x10^{-6}$	$1x10^{-4}$	$1x10^{-3}$	ND	ND	$2x10^{-2}$
Mussel	$9x10^{-4}$	ND	ND	ND	N/AP	$1x10^{-6}$	$1x10^{-4}$	ND	ND	ND	$1x10^{-3}$
Prawn	$2x10^{-2}$	ND	ND	$1x10^{-4}$	N/AP	ND	ND	ND	ND	$3x10^{-5}$	$2x10^{-2}$
Squid	$4x10^{-3}$	ND	$3x10^{-4}$	ND	N/AP	ND	ND	ND	ND	$6x10^{-5}$	$5x10^{-3}$
Cooked Sole	$9x10^{-3}$	ND	$7x10^{-4}$	$7x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$1x10^{-2}$
Cooked Crab	$1x10^{-2}$	ND	$5x10^{-4}$	$4x10^{-4}$	N/AP	ND	ND	ND	ND	ND	$1x10^{-2}$
Cooked Crab Hepatopancreas	$1x10^{-3}$	ND	$2x10^{-3}$	$2x10^{-3}$	N/AP	ND	ND	ND	ND	ND	$5x10^{-3}$

ND = Not detected
N/AP = Not applicable because chemical was not analyzed

This result is comparable to that observed for PCB HQs. These differences were most pronounced for fish, specifically sole where the ratio of the risks was approximately (100 > 10 >1). The PAH risks were predicted exclusively in the Duwamish River and Elliott Bay.

Comparison of Tissue Types. Overall, results between tissue types were similar. However, some differences were apparent. For arsenic the highest risks were predicted in crab, prawns followed by sole and crab hepatopancreas.

As was the case with the PCB HQs, the highest PCB cancer risks were predicted for sole fillets (raw and cooked), whole body sole, and crab hepatopancreas (raw and cooked). No apparent pattern in PAH risks by tissue was observed, possibly due to the relative infrequency with which these COPCs were detected.

Population Risk Results. As described in Section 3.3 above, risks to exposed populations may be estimated by combining estimates of the probability of a individual developing cancer resulting from a chemical exposure (i.e., the individual risk estimate) and information about the size of the exposed population. This calculation results in estimates of the increased incidence of cancer resulting from the estimated exposures. To put a one in a million risk level into perspective, if a population of one million people were exposed at the estimated exposure level, then one incident of cancer above the background rate would be expected. However, even if it were possible to verify the exposure level of a large population, the predicted cancer risk would likely not be detectable given the background rate of cancer which has been estimated at between 20 to 25 percent or approximately two to three of every ten people (American Cancer Society 1993).

A quantitative assessment of population risks was not done in this assessment due to the difficulties in obtaining an accurate assessment of the size of the exposed population. The numbers of individuals who engage in netfishing or recreational activities in the study area is unknown. Similarly, it was not possible to derive reliable estimates of the number of people consuming seafood at the estimated exposure levels. However, it is expected that the number of people who consume seafood at the high level of daily consumption is very small due to the small number of people (seven) who reported this frequency in the fishing survey (see Section 2).

4. METHODS AND RESULTS OF THE HUMAN HEALTH PATHOGEN RISK ASSESSMENT

The human health risks being addressed as part of the CSO WQA are those associated with pathogens and chemicals that may occur in CSO discharges, surface water, sediments, and edible aquatic organisms. This section presents the methods and results of the pathogen risk assessment. Pathogens as the stressors may affect anglers through a shellfish consumption exposure pathway or effect net fishers, swimmers, scuba divers and windsurfers through direct contact and/or ingestion of the water. The assessment endpoint for pathogens is infection and disease. The problem formulation for the pathogen risk assessment is presented in Appendix A.

The pathogen/CSO assessment involves the development of a scientific risk assessment that can be integrated with the overall approach in the study of the watershed and water quality goals. The human health pathogen risk assessment consisted of the following components:

- In the human health pathogen exposure characterization, the exposure concentrations and/or doses (i.e., intakes) of pathogens to which people may be exposed were calculated. Pathogen exposures were quantitatively evaluated for direct water contact activities and qualitatively assessed for shellfish consumption. Exposures were assessed for indicators of fecal contamination (i.e., fecal coliforms) and for specific pathogens.

- In the human health pathogen effects characterization, the data and models used to assess the potential effects are identified. Indicator organism concentrations protective of public health and the distribution of doses associated with different risk levels from specific pathogenic organisms were researched.

- In the human health pathogen risk characterization, the results of the exposure and effects characterizations were combined to obtain numerical estimates of risk. The potential for effects based on indicator organisms and the potential for infection and illness based on specific pathogens were calculated separately.

- In the uncertainty assessment, the uncertainties associated with the human health pathogen risk assessment are discussed, along with their potential for influencing or affecting the results of the assessment.

Each of the components of the human health pathogen risk assessment are discussed in the following sections.

4.1 Exposure Characterization

The exposure characterization presents:

- Identification of potentially exposed populations and exposure pathways
- Estimation of microorganism exposure concentrations
- Quantification of exposures to microorganisms
- Summary

4.1.1 Identification of Potentially Exposed Populations and Exposure Pathways

People may be exposed to microorganisms in the Duwamish River and Elliott Bay through a variety of activities. As described in the problem formulation in Appendix A, populations potentially exposed to microorganisms in the river and bay include those persons who use the river and bay for recreational activities (e.g., swimming, SCUBA diving, etc.), and those that collect and consume seafood, primarily shellfish.

The exposure pathway is the course that the microorganism takes from its source to a given receptor (U.S. EPA 1989a). Each exposure pathway includes a source, an exposure point (point of contact) and an exposure route (route of entry into the body, e.g., ingestion). It is recognized that microorganisms may enter the body through a variety of exposure routes, such as ingestion, skin contact, inhalation, or through an open cut or wound. Ingestion will be the only route of exposure assessed in this risk assessment because (1) the available epidemiology data suggest that ingestion is the most significant exposure route, and (2) no effects characterization data are available for exposure routes other than ingestion (see Section 4.2 below).

4.1.2 Estimation of Microorganism Exposure Concentrations

Microorganism exposure concentrations were calculated by combining the results of the sampling and analysis program with the computer model of the Duwamish River and Elliott Bay. The methods and results of combining the sampling and analysis program results with the computer model are discussed below for fecal coliforms and for pathogenic microorganisms.

Fecal Coliforms in Surface Water. Fecal coliform concentrations in the Duwamish River and Elliott Bay were estimated from the results of the sampling and analysis program and the computer model output. Standard methods were utilized for enumeration of fecal coliform bacteria. During a six-month time period approximately 1,300 samples were collected from the Duwamish River and Elliott Bay. Discharges from five different CSOs were sampled, with eight to 33 samples per site. Wastewater treatment plant influent was sampled seven times. Fecal coliform sampling data are presented on the WQA web page at: ***http://splash.metrokc.gov/wlr/waterress/wqa/wqapage.htm***. Fecal

coliform concentrations in CSO discharges and in the Duwamish River and Elliott Bay were then used to calibrate the computer model for existing conditions (Appendix B1).

Fecal coliform concentrations in the CSO discharges used in the computer model are presented in Table 4-1 for each of the CSOs that discharge into the river and bay. As shown, the Denny Way CSO is predicted to discharge CSO effluent at the highest fecal coliform concentration.

Table 4-1. Fecal Coliform Input Data for Each of the CSOs in the Duwamish River and Elliott Bay

Location	Fecal Coliform Concentration (count/100mL)
8th Ave	$1.4x10^5$
S.W. Michigan St.	$1.5x10^4$
Harbor Ave.	$1.4x10^5$
Chelan Ave.	$1.4x10^5$
Norfolk St.	$1.4x10^5$
Brandon St.	$3.6x10^4$
Hanford	$3.8x10^4$
Lander	$3.8x10^4$
Connecticut St.	$1.4x10^5$
King St.	$3.8x10^5$
Denny Way	$2.8x10^6$
S. Magnolia	$1.4x10^5$

Survival of microorganisms of fecal origin is a factor that needs to be addressed because these organisms have a finite life in marine waters and undergo some level of inactivation (decrease in viable numbers/total population per unit of time). For microorganisms the time for 90 percent reductions (one ten-fold reduction in viable concentrations) is used to estimate inactivation (die-off).

Once enteric microorganisms enter the environment some natural inactivation begins. In water, many factors influence the rate of inactivation, including the amount of solids, oxygen, salinity, UV light, and in particular the temperature. Temperature plays the most significant role in the survival of these microorganisms and most of the data available deal with the effect of varying temperatures.

The survival of pathogenic bacteria is closely related to survival of the coliforms (Feachem et al. 1983, McFeters, and Terzieva 1991, McFeters 1990, Korhonen, and Martikainen 1991). The inactivation rate for 90 percent (1.0 $\log_{10}$) reductions may range from 1 to 6 days, depending on the temperature. At temperatures between 5 and 10^{o}C, it could take 9 to 24 days to see a 99.9 percent reduction in the bacterial levels. Salinity also effects survival of the microorganisms associated with wastewater inputs, however, temperature appears to play the major role. For this project, the time for a 90 percent reduction in fecal coliform concentrations (T-90) was conservatively assumed to be six days.

Fecal coliform concentrations in each Duwamish River and Elliott Bay model cell were calculated during a modeled simulation for each hour during a one year period under baseline conditions, and again for each hour during a one year period after removal of CSOs (the without CSO scenario). Concentrations of fecal coliforms attributable to CSOs only were also calculated as the difference in baseline concentrations and the without CSO concentrations. To model the without CSO conditions, fecal coliform concentrations in the CSO inputs were set to zero. The hourly fecal coliform concentrations were then calculated for each cell in the river and bay based on inputs from other sources. Modeling methods and calibration are presented in Appendix B-1.

The range of fecal coliform concentrations in the Duwamish River and Elliott Bay during January (a high-flow month) and August (a dry month with no CSO discharges) are summarized in Table 4-2 for baseline conditions and Table 4-3 for without CSO conditions.

Table 4-2. Summary of Fecal Coliform Concentrations (count/100mL) in the Surface Layer at Select Locations Under Baseline Conditions

Location	Cell #	January			August		
		Geometric mean	90th percentile	Maximum	Geometric mean	90th percentile	Maximum
Upstream of Norfolk	10	144	694	20,370	60	181	11,670
Kellogg Island @ Duwamish Diagonal CSO	129	137	566	1,582	61	160	724
East Waterway	161	123	499	1,234	40	96	228
West Waterway	158	117	444	10,060	30	68	1,205
Seattle waterfront	253	52	207	3,918	5	8	136
Seacrest Park	220	44	166	2,258	5	9	118
Middle of Bay	346	7	31	374	1	2	24
Next to Denny Way CSO	312	121	9,336	39,120	3	4	263

Table 4-3. Summary of Fecal Coliform Concentrations (count/100mL) in the Surface Layer at Select Locations Under Without CSO Conditions

Location	Cell #	January			August		
		Geometric Mean	90th Percentile	Maximum	Geometric Mean	90th Percentile	Maximum
Upstream of Norfolk	10	143	694	20,370	60	177	12,030
Kellogg Island @ Duwamish Diagonal CSO	129	115	521	1,680	54	132	293
East Waterway	161	96	405	1,216	33	76	170
West Waterway	158	91	352	8,655	24	50	910
Seattle waterfront	253	33	73	1,281	5	7	137
Seacrest Park	220	26	81	1,709	4	7	114
Middle of Bay	346	3	7	164	1	1	25
Next to Denny Way CSO	312	21	58	133	2	4	6

Pathogenic Microorganism Concentrations in Surface Water. Human pathogens were measured in wastewater treatment plant influent and in the tissues of mussels from the Duwamish River. Human pathogens were not monitored in CSO discharges or in surface waters of the Duwamish River or Elliott Bay.

Pathogen Sampling. Seven wastewater treatment plant influent samples were monitored for enteric viruses and enteric protozoa. The virus levels in the untreated sewage based on monitoring was found to be between 2 and 15 pfu/L[18]. The *Giardia* cyst levels in untreated sewage based on monitoring data were found to be between 460 to 8,800 cysts per liter, depending on the testing method used and the amount of internal structure within the actual cyst.

The bacteria, *Salmonella*, *Listeria,* and *Yersiniae* were also monitored for in wastewater treatment plant influent samples and shellfish tissue samples. *Yersiniae* was not detected in two shellfish samples, with concentrations less than 2 mpn/100 ml[19], but were detected in four out of six influent samples at concentrations of 4 mpn/100ml. *Listeria* was not analyzed in shellfish samples, and was not detected (less than 3 mpn/100 ml) in six out of six influent samples. *Salmonellae* was not detected (less than 3 mpn/100ml) in four out

[18] pfu = plaque forming units

[19] mpu = most probable number

of five shellfish samples and was detected (4 mpn/100ml) in one shellfish sample. *Salmonellae* was detected in four out of six influent samples at concentrations ranging from 2 to 190 mpn/100ml and was not detected (less than 2 mpn/100ml) in two out of six influent samples.

Enteric viruses (those which can be measured in water samples) and *Giardia* were chosen for assessment because they are known to be found in wastewater. Although it is acknowledged that many other enteric pathogens may be present in CSO discharges, it is believed that these two groups represent the greatest risk and serve as surrogates for the other risks (due to levels and prevalence and infectivity).

Virus and *Giardia* Survival in Marine Waters. Virus survival has been evaluated by other investigators, who found that most of the variation in inactivation was a result of the water temperature differences (Feachem et al. 1983, Kutz and Gerba 1988). Feachem et al. (1983) summarized available data (Table 4-4), showing that a 90 percent inactivation (T-90) of viruses in marine waters occurs within 0.67 to 1.0 day at a temperature of 20^{o} C. In polluted surface waters, the viruses survived longer than in tap water (Kutz and Gerba 1988). Viruses also survive longer at colder temperatures than at warmer temperatures (Feachem et al. 1983, Kutz and Gerba 1988). Between six and 10 days are required for 99.9 percent inactivation at ambient temperatures of between 15 and 25^{o} C (Kutz and Gerba 1988). T-90 may be as long as 30 days at 4^{o} C (Kutz and Gerba 1988).

Table 4-4. Survival of Enteric Pathogens and Indicator Bacteria in Marine Waters (Feachem et al. 1983)

Microorganism	Temperature ^{o}C	Time in Days for 90% Reductions in Marine Water
Coliforms	10-20	0.025-0.33 avg. 0.083
E. coli	0 30	1.6 0.58
Salmonella	4 37	0.96 0.7
Yersinia	4-37	0.6
Giardia	2- 5	14-143
Enteric Viruses	20 18-20 4-15	0.67 to 1.0 6.0 (in sediment) 14 (in sediment)

Viruses may also accumulate in sediments and shellfish, where survival time is greatly enhanced relative to survival in water (Volterra et al. 1985). Greater than 99 percent of

the enteric viruses were found to adsorb to marine sediments and suspension in sewage effluents did not alter this pattern (LaBelle and Gerba 1979). The T-90 reduction rates in sediments were reportedly 6 days at 18 to 21^{o} C and 14 days at 4 to 15^{o} C (Feachem et al. 1983). Viruses were also found to remain infectious for 9 days in water with high suspended solids concentrations and 19 days in the underlying sediment (Rao et al. 1984). There is no doubt that suspended solids and sedimentation are important considerations in determining the fate of microorganisms in marine waters.

For the modeling efforts, a T-90 of 1 day was used for viruses as the estimate of pathogen die-off over time. This is the high end of the range of estimated T-90s for viruses at 20^{o} C (Feachem et al. 1983). This value was used because the temperature at the Duwamish River and Elliott Bay is typically above 5° C.

There are very little data on survival in water for the protozoa. Feachem et al. (1983) summarized available survival data (see Table 4-4) and showed T-90 for *Giardia* to range from 14 to 143 days in marine waters at temperatures of 2 to 5^{o} C. DeRegnier et al. (1989) found that mice could no longer be infected with *Giardia* cysts after 56 days in river and lake water at 5^{o} C. However, the level of cysts was below the infectivity level for mice and viability inclusion dyes suggested a -0.01 to 0.05 $\log_{10}$ per day inactivation rate at low temperatures. Therefore, after 60 days 75 percent to 99.9 percent reduction in cyst viability may be observed. Robert et al. (1992) have demonstrated that only 55 percent of the *Cryptosporidium* oocysts are dead in river water after 47 days and 99 percent are dead after 176 days at temperatures between 5 and 10^{o}C. Both have been reported to survive in marine waters (Johnson et al. 1997).

For the modeling efforts, a T-90 of 14 days was used for *Giardia* as the estimate of pathogen die-off over time. This is the lower end of the range of T-90s reported for 2 to 5^{o} C (Feachem et al. 1983). This value was used because the temperature of the Duwamish River and Elliott Bay is typically above 5° C.

Virus and *Giardia* Concentrations in the Duwamish River and Elliott Bay. Hourly concentrations of viruses and *Giardia* from CSO discharges were modeled in each model cell of the river and bay. In other words, we assumed CSOs were the only source of viruses and *Giardia.* The modeled virus and *Giardia* concentrations attributable to CSO discharges can be interpreted as being equal to the amount of reduction that will be observed in concentrations in the river and bay after removal of the CSOs. The modeled concentrations also represent the pathogen concentrations attributable to CSO discharges over and above the concentrations resulting from other possible sources of pathogens to the Duwamish River and Elliott Bay. Other possible sources of pathogens besides CSO discharges include stormwater runoff, domestic and wild animals, agricultural runoff, and leaky septic systems.

Because only CSO contributions of viruses and *Giardia* were modeled, baseline conditions and without CSO conditions were not modeled, as was done for other parameters evaluated. In addition, no calibration of the virus and *Giardia* results occurred, due to insufficient data on their concentrations in other sources and the receiving waters.

Virus concentrations in the CSO discharges were estimated using a combination of site-specific and national data. Virus concentrations of 15 pfu/L were assumed for all CSO discharges. This was the highest concentration observed in West Point Treatment Plant influent. This concentration is also within the range of concentrations reported as influent averages for different systems throughout the United States (see the *Problem Formulation* in Appendix A).

Giardia concentrations of 490 cysts per liter were assumed for all CSO discharges. This is the average *Giardia* concentration reported in untreated wastewater in Occoquan, Virginia (Rose et al. 1996a). Lower average *Giardia* concentrations were reported in untreated wastewater from St. Petersburg, Tampa, San Diego and Denver (NRC 1998).

Modeled pathogen concentrations at selected locations in the Duwamish River and Elliott Bay resulting from CSO discharges (i.e., assuming no other sources of viruses into the river and bay) are summarized in Table 4-5 (viruses) and Table 4-6 (*Giardia*). Concentrations are shown for January, a high-flow month with multiple CSO discharges, and August, a low-flow month with no CSO discharges.

Table 4-5. Summary of Virus Concentrations in the Surface Layer at Select Locations Resulting from CSO Discharges (count/100ml)

Location	Cell #	January			August		
		Geometric Mean	90th Percentile	Maximum	Geometric Mean	90th Percentile	Maximum
Upstream of Norfolk	10	0	0	9.5×10^{-8}	1.31×10^{-6}	2.78×10^{-6}	0.000024
Kellogg Island @ Duwamish Diagonal CSO	129	0.000202	0.000269	0.0072	3.82×10^{-6}	0.0000125	0.000029
East Waterway	161	0.000461	0.000636	0.013	3.82×10^{-6}	0.0000123	0.000027
West Waterway	158	0.000263	0.000460	0.0057	3.58×10^{-6}	0.0000112	0.000028
Seattle waterfront	253	0.000564	0.00192	0.0062	3.10×10^{-6}	0.0000101	0.000023
Seacrest Park	220	0.000164	0.000471	0.0019	1.43×10^{-6}	5.49×10^{-6}	0.000013
Middle of Bay	346	0.0000774	0.000308	0.00045	7.15×10^{-7}	2.34×10^{-6}	5.8×10^{-6}
Next to Denny Way CSO	312	0.000938	0.00420	0.015	2.38×10^{-6}	7.35×10^{-6}	0.000017

Table 4-6. Summary of *Giardia* Concentrations in the Surface Layer at Select Locations Resulting from CSO Discharges (cysts/100ml)

Location	Cell #	January			August		
		Geometric Mean	90th Percentile	Maximum	Geometric Mean	90th Percentile	Maximum
Upstream of Norfolk	10	0	0	3.1×10^{-6}	0.0000713	0.000148	0.0011
Kellogg Island @ Duwamish Diagonal CSO	129	0.00959	0.0135	0.36	0.000193	0.000598	0.0014
East Waterway	161	0.0206	0.0318	0.66	0.000201	0.000647	0.0014
West Waterway	158	0.0125	0.0227	0.28	0.000200	0.000624	0.0014
Seattle waterfront	253	0.0267	0.107	0.31	0.000177	0.000555	0.0012
Seacrest Park	220	0.00811	0.0237	0.094	0.0000905	0.000319	0.00070
Middle of Bay	346	0.00383	0.0154	0.022	0.0000488	0.000165	0.00038
Next to Denny Way CSO	312	0.0428	0.210	0.75	0.000140	0.000405	0.00088

Exposures to pathogens in discharges from the Denny Way CSO were examined in more detail because the Denny Way CSO discharges the largest volume of combined sewage and stormwater of any of King County's CSOs. Table 4-7 shows the hours at this site that can be logged into several types of categories associated with the timing of the discharges from all CSOs during the year. Most of the rain events and subsequent discharges occurred during 634 hours over the year (about 7.2 percent of the time) with the greatest numbers of hours logged in December, January, and March.

The virus and *Giardia* concentrations in the cell next to the Denny Way CSO during CSO discharges and at various time periods after discharges have ended are shown in Figure 4-1 and Figure 4-2. The figures give the concentrations per 100mL. At the 90th percentile during discharge the virus levels were approximately 0.01/100mL (or 1/10L, detection limit associated with monitoring the marine environment is usually around 1/100L). *Giardia* levels were greater than the virus levels with almost 1 cyst/100mL at the 90th percentile. The contamination dropped quickly to 100 fold less than during the discharge after 6 to 48 hours. After 7 to 14 days baseline conditions were achieved through pathogen die-off and dilution. There was only a slight increase 7 to 14 day post discharge at the 70th percentile.

Table 4-7. Number of Hours During the Modeled Year which the Denny Way CSO Discharged for at Least Five Minutes, and the Classification of Remaining Hours by the Time Since Cessation of the Discharge

Month	During Discharge	Time Since Denny Way CSO Stopped Discharging					
		0 to 6 Hrs	6 to 24 Hrs	24 to 48 Hrs	48 Hrs to 7 Days	7 to 14 Days	>14 Days
Annual	634	516	1,246	1,327	3,035	1,097	905
September	17	26	65	96	337	179	0
October	56	56	151	141	276	64	0
November	56	78	166	140	280	0	0
December	112	53	142	137	254	46	0
January	140	52	113	105	298	36	0
February	8	12	36	48	240	254	74
March	116	64	138	142	253	25	6
April	44	40	105	144	261	126	0
May	7	28	90	120	373	126	0
June	52	69	159	165	222	53	0
July	26	38	81	89	241	188	81
August	0	0	0	0	0	0	744

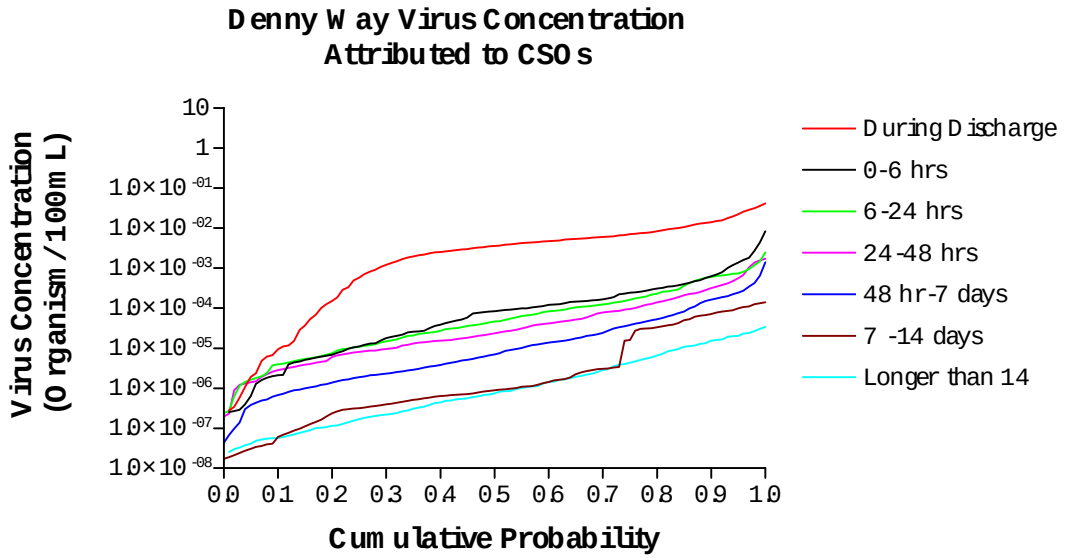

Figure 4-1. Virus Concentration Associated with CSO Discharges in the Surface Layer Cell Next to the Denny Way CSO

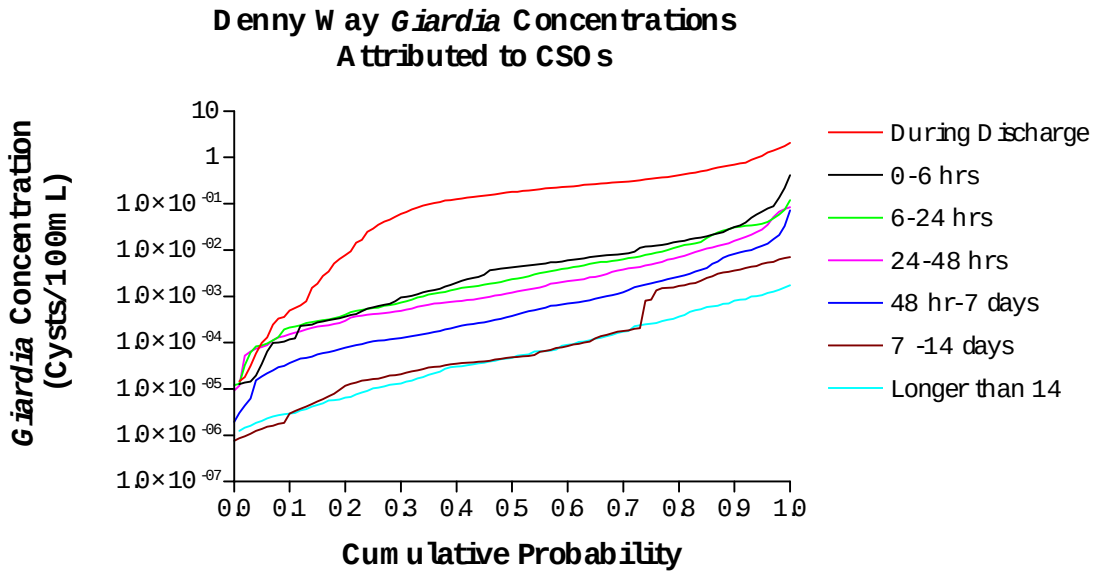

Figure 4-2. *Giardia* Concentration Associated with CSO Discharges in the Surface Layer Cell Next to the Denny Way CSO

Virus Concentrations in Shellfish. It is well known that infectious hepatitis and viral gastroenteritis are caused by consumption of contaminated raw or, in some cases, cooked clams and oysters. The percentage of samples contaminated with viruses ranged from 9 to 40 percent in surveys in the east coast of the U.S. (Rose and Sobsey 1993). The levels of viruses ranged from 0.3 to 200/100 g. Enteric viruses were not detected (less than 2 pfu/100g) in two mussel samples from the Duwamish River during the winter of 1997. Attempts to calculate concentrations of viruses in shellfish based on concentrations in surface water were unsuccessful because water/shellfish bioconcentration factors for viruses are not currently available.

4.1.3 Quantification of Exposure to Microorganisms

Human exposures to microorganisms were quantified based on the estimated environmental concentrations and assumptions about human activities. Exposures for fecal coliforms in surface water, pathogens in surface water, and pathogens in shellfish are discussed below.

Exposure to Fecal Coliforms. Fecal coliforms are an indicator of pathogenic microorganisms and are not themselves pathogenic to humans. The concentrations of fecal coliforms predicted in the Duwamish River and Elliott Bay (rather than estimated doses) were used to estimate the potential for exposure to pathogenic organisms because fecal coliform standards are written in terms of concentrations rather than doses.

Exposure to Pathogens During Recreational Activities. Many people use the waters of the Duwamish River and Elliott Bay for recreational and commercial activities. Examples of some activities that people engage in are swimming, SCUBA diving, windsurfing, net fishing, recreational seafood collection, boating, kayaking, and parasailing. Most of these activities result in some degree of exposure to surface water.

The dose of viruses and Giardia from incidental ingestion of surface water during recreational or commercial activities was calculated. This dose was combined with effects information during the risk characterization to estimate the risk of infection. In general, the larger the dose, the greater the risk of infection. Therefore, assuming all else equal, the larger the amount of contaminated water swallowed, the greater the risk of infection. In this risk assessment it was assumed that a recreational or commercial user of the river or bay would incidentally ingest 50 mL of water (U.S. EPA 1991b).

Exposure was calculated using the following equation:

$$\text{Dose} = C_w \times IR_w \qquad \text{(Equation 4-1)}$$

Where:

Dose	=	The dose of virus or *Giardia* from incidental ingestion of water (viral agents per exposure or cysts per exposure)
C_w	=	The virus or *Giardia* concentration in surface water (viral agents per liter or cysts per liter)

IR_w = The incidental ingestion rate of water (liters per exposure)

We do not know how many people use the estuary in a way that could result in incidental ingestion during or after storms that trigger CSOs, although we suspect the numbers are small.

Exposure to Viruses Through Consumption of Shellfish. Exposures to viruses through the consumption of shellfish was not quantitatively assessed because of the lack of data available on virus concentrations in shellfish and the limited number of people that currently collect shellfish from the Duwamish River and Elliott Bay. The results of the fishing survey conducted along the shores of the river and bay during the summer of 1997 indicated that only 1 out of about 1,200 different people had collected clams from the river or bay, and none had collected mussels or oysters.

4.1.4 Summary of the Exposure Characterization

1. Recreational and commercial users of the river and bay, along with people that consume shellfish from the river and bay, may be exposed to pathogens originating in CSO discharges.

2. Fecal coliform data were used to calibrate the fate and transport model and reflect the potential for virus and protozoan movement as well.

3. Virus and *Giardia* levels predicted in the bay were developed from sewage monitoring data and the transport model. Average virus levels in sewage were 15 pfu/L with a T-90 of 1 day and average *Giardia* cyst levels in sewage were 490/L with a T-90 of 14 days.

4. A 50 mL volume was assumed to represent the average incidental ingestion exposure for use of the bay and rivers.

5. Exposure to viruses and *Giardia* through consumption of shellfish was not calculated because of the limited data available.

4.1.5 Uncertainties in the Exposure Characterization

Several notable uncertainties exist in the exposure characterization that may result in either over- or under-estimates of exposures. Major uncertainties that we believe may have the largest impact on the exposure estimates include:

- The model calibration for fecal coliforms included no data points for the summer months. It was therefore not possible to calibrate the fecal coliform model output for these months.

- The model was not calibrated for virus and *Giardia* concentrations. Without appropriate calibration, it is unclear whether the model accurately predicts virus and *Giardia* concentrations. Also, lack of virus and *Giardia* concentration data in the receiving water and from other sources only allowed an estimate of pathogen concentrations attributable to CSOs. It is not possible to state the significance of the predicted decrease in pathogen concentrations without estimates of baseline conditions (i.e., without knowing the pathogen concentrations from other sources).

- Concentrations of virus and *Giardia* in CSO discharges were estimated from a limited data set using a combination of King County-specific and national average data.

- Virus concentrations in shellfish were not available for this assessment and corresponding health risks from shellfish consumption were therefore not calculated.

Other uncertainties in the exposure characterization include:

- Exposure was estimated based on an assumed incidental ingestion of 50 mL of water per event. Although ingestion may be greater or less than 50 mL depending on the activity (SCUBA diving versus boating), this volume was used as a best-estimate of average exposure (U.S. EPA 1991b) and was used as an estimate of average ingestion rate in the chemical risk assessment. This assumption may over- or under-estimate exposures.

- CSO concentrations of fecal coliforms were assumed to remain constant for each CSO. Fecal coliform concentrations likely vary both within a single discharge and between discharges.

- Die-off rates for fecal coliforms, virus, and *Giardia* used in the model may either over- or under-estimate actual die off rates.

4.2 Effects Characterization

This section summarizes the water quality standards for the indicator fecal coliforms and the dose-response information for viruses and *Giardia* and presents the relationships used to estimate risks to human health. These dose-response relationships are combined with estimates of exposure in the risk characterization to estimate risk of infection. The effect characterization is summarized at the end of this section.

4.2.1 Water Quality Standards for Fecal Coliforms

Washington State water quality standards have been developed for fecal coliforms for the Duwamish River and Elliott Bay (Chapter 173-201A WAC Water Quality Standards for Surface Waters of the State of Washington). These standards are intended to be

protective of the designated uses of the waterbodies. The Duwamish River is classified as a class B (good) freshwater waterbody, with characteristic uses including, but not limited to:

1. Water supply (industrial and agricultural)
2. Stock watering
3. Fish and shellfish: salmonid migration, rearing, and harvesting. Other fish migration, rearing, spawning, and harvesting. Clam, oyster, and mussel rearing and spawning. Crustaceans and other shellfish (crabs, shrimp, crayfish, scallops, etc.) rearing, spawning and harvesting
4. Wildlife habitat
5. Recreation (secondary contact recreation, sport fishing, boating, and aesthetic enjoyment)
6. Commerce and navigation

The fecal coliform water quality standard for class B freshwater waterbodies is a geometric mean of 200 organisms per 100 milliliters and a concentration of 400 organisms per 100 milliliters not to be exceeded by greater than 10 percent of the samples. These standards are to be applied to the 30 most recent samples available.

Elliott Bay is classified as a Class A (excellent) marine waterbody, with characteristic uses including, but not limited to:

1. Water supply (domestic, industrial and agricultural)
2. Stock watering
3. Fish and shellfish: salmonid migration, rearing, spawning and harvesting. Other fish migration, rearing, spawning, and harvesting. Clam, oyster, and mussel rearing, spawning, and spawning. Crustaceans and other shellfish (crabs, shrimp, crayfish, scallops, etc.) rearing, spawning and harvesting
4. Wildlife habitat
5. Recreation (primary contact recreation, sport fishing, boating, and aesthetic enjoyment)
6. Commerce and navigation

Some of the characteristic uses listed do not occur for Elliott Bay, such as water supply and stock watering. The fecal coliform water quality standard for Class A marine waterbodies is a geometric mean of 14 organisms per 100 milliliters and a concentration

of 43 organisms per 100 milliliters not to be exceeded by greater than 10 percent of the samples. These standards are to be applied to the 30 most recent samples available.

Several studies are available that test the appropriateness of various indicator organisms for marine waters. In Santa Monica, an epidemiological study of illnesses among swimmers at beaches indicated that the best indicator of illness was the ratio of total to fecal coliforms (Haile et al. 1996). Other indicators studied included fecal coliforms, total coliforms, enterococcus, and *E. coli*.

An epidemiological investigation was undertaken of water quality and health effects at marine beaches during the 1970s (Cabelli et al. 1979, 1981). This study was done as a part of a study by the U.S. EPA for reassessment of ambient water quality standards. Water samples were analyzed for coliforms, enterococci, *Pseudomonas*, and *Clostridium* bacteria as possible indicators. A correlation was demonstrated, showing an association between the enterococci bacterial levels in water and excess gastrointestinal illness by the swimmers using the water. Recommendations by the U.S. EPA in the 1986 Ambient Bacteriological Water Quality report suggested that 35 enterococci/100mL related to a risk of 19 illnesses/1,000 swimmer-days (between 2/100 and 1/100).

4.2.2 Dose Response for Pathogens

Human feeding studies, with ingestion at various doses of microorganisms followed by evaluation of infection in the exposed groups, have been used to develop dose response models for pathogens. The probability of infection is described by exponential and beta-poission equations. These have been developed for viruses and *Giardia*. These models have been used to evaluate health risks by the U.S. EPA under the *Safe Drinking Water Act* for the development of proper controls (U.S. EPA 1989b).

Giardia. The risk equation for Giardia may be described as by an exponential model, in terms of the probability of infection π (P_i):

$$\pi(P_i) = 1\ e^{(-rN)} \qquad \text{(Equation 4-2)}$$

where r is the fraction of microorganisms that are ingested which survive to initiate infection (which is organism specific) and N is the daily exposure. The *Giardia* risk assessment model was previously published (Rose et al. 1991b) and the parameter for *Giardia* was $r = 0.0198$ (0.009798-0.03582, 95 percent conf. limits). The exponential model was the best-fit model for the *Giardia* infectivity data. For comparison, the parameter for *Cryptosporidium* was r = 0.00467 (95 percent C.I., 0.00195-0.0097) (Haas and Rose 1994).

Figure 4-3 compares these models for *Giardia* and *Cryptosporidium* and shows the 95 percent confidence intervals surrounding the model. Although the confidence intervals surrounding the *Giardia* model are wider, a greater risk is estimated as compared to *Cryptosporidium*. This may be a result of limited volunteers, doses, and/or study design in the *Giardia* experiments (Rendtorff 1954a). In the *Giardia* study, Rendtorff performed a series of feeding studies with one to three people per dose of *Giardia* cysts. The

Cryptosporidium study had eight doses, 29 volunteers and was designed to examine dose-response modeling (DuPont et al. 1995).

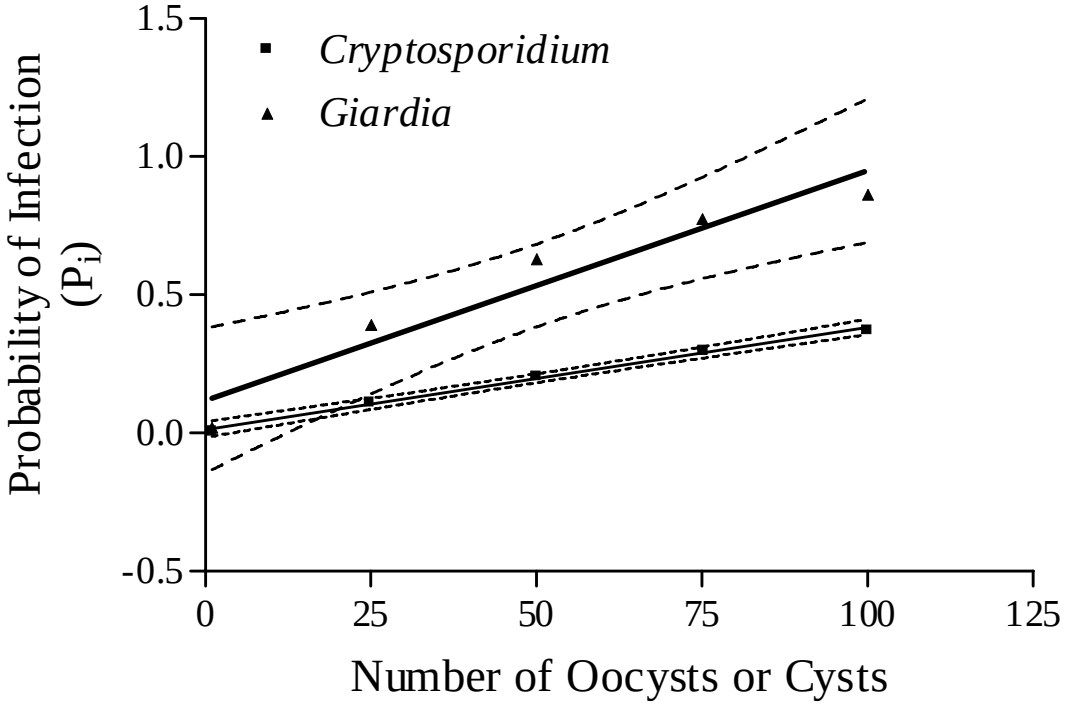

Figure 4-3. Comparison of Cryptosporidium and Giardia Dose-Response

More recently repeats of the dose-response experiments were undertaken for *Cryptosporidium* (Okhuysen et al. 1998). This study investigated whether *C. parvum* exposure could produce resistance to re-exposure. Nineteen healthy immunocompetent adults were rechallenged, one year after primary exposure (30 to 10^6 oocysts) with a second dose of 500-C. *parvum* oocysts. The results show comparable rates of diarrhea between the primary and secondary exposures, fewer oocysts were shed after secondary exposure, and clinical severity (measured by number of unformed stools passed) was lower after reexposure. Prior to conducting the study, the author and his colleagues determined by interim analysis that the ID_{100} (the dose that would infect 100 percent of those exposed) would be 500 oocysts. However, after primary challenges were completed, 500 oocysts represented an ID_{86}, which is in close agreement with the study results of the first infectivity investigation (DuPont et al. 1995) where 500 oocysts represented an ID_{83}. Okhuysen et al. (1998) concluded that initial exposure was not sufficient to protect against reexposure and/or clinical illness one year later.

Viruses. Dose-response for rotaviruses, Hepatitis A virus (HAV), Coxsackie viruses, echo viruses have all been developed in human feeding studies (Haas, et al. 1998). These models can provide the basis for comparative risk. The dose-response models used in

these studies arise from the best-fit beta-Poisson dose-response model[20]. This model depicts the probability of infection π (P_i) following a single dose (d) of viruses to be given by:

$$\pi(P_i) = 1 - \left[1 + \frac{d}{N_{50}}\left(2^{1/\alpha} - 1\right)\right]^{-\alpha} \qquad \text{(Equation 4-3)}$$

Where N_{50} is the number of viral agents necessary to infect 50 percent of the exposed population and α is an empirical parameter that describes the distribution.

Regli et al. (1991) reviewed available data for various viruses and used the method of maximum likelihood to fit dose-response models to the experimental data. This review showed that at low doses the rotavirus is the most infectious waterborne virus for which dose-response information is available. If rotavirus is assumed to represent the most infective virus likely to be present in CSO discharges, then a plausible upper-limit risk assessment can be based on the dose-response properties of this organism. Rotavirus infectivity data were developed by Ward et al. (1986) in a study during which 62 adult volunteers ingested doses of rotavirus ranging from 0.009 to 90,000 pfu. The infectivity model parameters used in this risk assessment are: N_{50} = 6.17 pfu and α = 0.253 (Haas et al. 1993).

4.2.3 Summary of the Effects Characterization

1. The Washington State standards for fecal coliforms were used as an indicator of potential effects from pathogens associated with fecal contamination.

2. The *Giardia* dose-response model is an exponential distribution that best-fits available *Giardia* infectivity data developed from human exposure studies conducted during the 1950s.

3. The rotavirus dose-response model conservatively estimates effects for enteric viruses. The rotavirus dose-response model is a beta-Poisson distribution that best-fits available infectivity data developed from human exposure studies conducted during the 1980s.

4.2.4 Uncertainties in the Effects Characterization

There are a number of uncertainties associated with the effects characterization. For example, the Washington State water quality standards for fecal coliforms were used as an indicator of potential effects from pathogens associated with fecal contamination.

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[20] The beta-Poisson distribution is a generalized version of the Poisson distribution where the dose is described as a beta distribution (Regli et al. 1991).

These standards may either over- or under-estimate levels that would be protective against pathogens. An additional certainty is the *Giardia* dose-response relationship that was derived from data developed during the 1950s. It is unclear whether the population exposed during the studies was representative of the population using the Duwamish River and Elliott Bay. It is also unclear whether the accuracy of measurement techniques used by Rendtorff (1954a,b) reflect current standards. Also, the virus and *Giardia* dose-response data were developed using healthy adult volunteers and no attempt was made to extrapolate the results to more sensitive individuals. This may potentially result in an under-estimation of infectivity for some individuals because it is unclear whether these data are reflective of the dose-response relationship for individuals who may be more susceptible to infection or have compromised immune systems (e.g., children, elderly, people infected with HIV, people on immunosuppression drugs). Finally, the dose-response for rotavirus was assumed appropriate to characterize the dose-response for all enteric viruses. This is believed to be a conservative assumption and likely results in an over-estimation of risk.

4.3 Risk Characterization

The risk characterization presents the risk characterization methods, the risk characterization results for indicator organisms, and the risk characterization results for viruses and *Giardia*.

4.3.1 Risk Characterization Methods

The microbiological risk characterization consisted of several components. For fecal coliforms in surface water, the following comparisons were made for baseline conditions, the without CSO scenario, and for fecal coliforms attributable to CSO discharges only:

1. Modeled fecal coliform concentrations for each cell were compared to standards on a monthly basis to ascertain compliance.

2. Modeled fecal coliform concentrations for each cell were compared to the various numerical standards to identify the percent of time during the year that the numerical standard was exceeded.

Risk characterization for viruses and *Giardia* attributable to CSO discharges in surface water included:

1. Calculation of the percent of time during the year that the risk of infection from ingestion of 50 mL of water exceeds 1 in 1,000 and 1 in 10,000 risk levels for each cell. To calculate this percentage, the dose associated with ingestion of 50 mL of water was calculated for every hour of the year. These doses were then compared to the dose required to obtain a 1 in 100, 1 in 1,000 and 1 in 10,000 risk of infection and illness to estimate the percent of time the risk levels were exceeded.

2. For specific cells where exposures are most likely to occur, a Monte Carlo21 computation was run (using Crystal Ball software) to compare the pathogen risks from using the recreational site during a CSO discharge and at various time intervals after the discharge. Two different distributions were used: (a) the temporal distribution at the exposure location (the surface cell next to the Denny Way CSO) of the concentrations of viruses/Giardia attributable to CSO discharges in the water, and (b) the conditional distribution on the probability of infection as described by the dose-response model.

Risk from exposure to pathogens accumulated by shellfish were not quantitatively estimated because of the lack of exposure data.

4.3.2 Indication of Potential Risks as Predicted by Fecal Coliform Concentrations

Fecal coliform concentrations were assessed to test compliance with state standards, and also to identify the percent of time that the concentrations exceed concentrations believed to be protective of public health.

Compliance with State Standards. Monthly geometric mean and 90^{th} percentile fecal coliform concentrations were calculated for each of the cells in the study area for baseline conditions (all sources including CSOs), without CSO conditions (all sources except CSOs), and CSOs only (no other sources). A cell was considered to be in compliance with the state standard for any given month if both of the geometric mean and 90^{th} percentile concentrations were below the appropriate state standards. The frequencies (expressed as number of months per year) that modeled fecal coliform concentrations in the surface water layer exceed the state standards are presented in Figure 4-19 and Figure 4-20 of Volume 1- *Overview and Interpretation Report* for baseline conditions and without CSO conditions, respectively. Fecal coliform concentrations in the surface layer were above the state standards more frequently than in the deeper layers. This difference in results at different depths likely occurs because the less dense fresh water of the discharges rises to the surface of the river and bay. Fecal coliform concentrations in the surface layers for most of the Duwamish River are above the state standards for over nine months of the year, both with- and without CSO discharges. This result implies that fecal coliforms from other sources are of such magnitude that the complete removal of CSO discharges would not allow the Duwamish River to meet the fecal coliform standards.

Figure 4-19 and Figure 4-20 of Volume 1- *Overview and Interpretation Report* also shows that fecal coliform concentrations in the surface layer of Elliott Bay under baseline conditions frequently exceed state standards along the shoreline, with only occasional

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[21] A Monte Carlo computation is a simulation technique that calculates a distribution of results based on the defined probability distributions that describe each of the input parameters. For this project, the distribution of results was estimated using 10,000 iterations.

exceedances in the middle of the bay. Under the without CSO scenario, state standards are exceeded less frequently along the shoreline north and west of the Denny Way CSO in Myrtle Edwards Park. In other areas of the bay, removal of CSOs is not predicted to substantially alter the frequency that state standards are exceeded.

Fecal coliform concentrations attributable to CSO discharges in surface waters were also compared to state standards to identify whether the CSO discharges would result in exceedances without considering other sources. Figure 4-21 of Volume 1- *Overview and Interpretation Report* presents the frequencies that modeled fecal coliform concentrations attributable to CSOs in the surface layer were below state standards. On a monthly basis, fecal coliform concentrations attributable to CSO discharges frequently (at least 10 months per year) meet the state standards throughout the Duwamish River and most of Elliott Bay. Fecal coliform concentrations attributable to CSO discharges frequently exceed standards along the shoreline north and west of the Denny Way CSO in Myrtle Edwards Park.

Potential seasonal effects on fecal coliform concentrations were investigated by calculating the percentage of cells that meet the state water quality standards each month (Table 4-8). The percentage of cells with fecal coliform concentrations that meet the state standards was calculated under baseline conditions (all sources and CSOs), without CSO conditions (all other sources), and CSOs only (no other sources). Obvious differences in the percentage of cells that meet the standards were observed based on cell depth and season. In general, surface cells were less likely to meet state standards that deeper cell. State standards were also generally less likely to be met during the winter months and more likely to be met during the summer months.

Percent of Time Fecal Coliform Concentrations Exceed Numerical Standards. The percent of time during the year that fecal coliform concentrations in each cell would exceed 43 organisms/100 mL and 400 organisms/100mL was calculated for baseline conditions, without CSO conditions, and for CSO discharges only. These comparisons were conducted because the 43 organism per 100 mL standard is intended to be protective of shellfish harvesting, and the 400 organism per 100 mL standard is intended to be protective of recreational use.

Figure 4-4 and Figure 4-5 present the percent of time that each of the surface cells would exceed the selected standards under baseline conditions. Figure 4-6 and Figure 4-7 present the percent of time that each of the surface cells would exceed the selected standards under without CSO conditions. Figure 4-8 and Figure 4-9 present the percent of time that each of the surface cells would exceed the selected standards due to CSO contributions only.

Table 4-9 summarizes the result of these comparisons by presenting the percent of time that the surface layer cells would exceed the selected standards under baseline conditions, without CSO conditions, and from CSOs only. As shown, there would be few differences between the baseline conditions and the without CSO conditions except in Elliott Bay along the shoreline north and west of the Denny Way CSO. The Elliott Bay

shoreline north and west of the Denny Way CSO is also the area that most frequently exceeds these standards due to fecal coliforms that originate from CSO discharges only.

Table 4-8. Percent of Cells that Meet the Fecal Coliform Water Quality Standards by Month for the Duwamish River and Elliott Bay

	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug
Duwamish River												
Baseline												
All Cells	100%	89%	72%	72%	64%	54%	64%	75%	80%	74%	75%	100%
Surface Only	100%	54%	7%	6%	0%	0%	0%	21%	32%	0%	1%	100%
Without CSO												
All Cells	100%	90%	71%	81%	71%	52%	64%	74%	79%	74%	76%	100%
Surface Only	100%	57%	6%	23%	17%	0%	0%	20%	33%	8%	9%	100%
Elliott Bay												
Baseline												
All Cells	97%	89%	91%	87%	47%	87%	74%	90%	96%	89%	90%	100%
Surface Only	90%	66%	46%	40%	6%	20%	19%	44%	68%	38%	49%	96%
Without CSO												
All Cells	100%	98%	94%	96%	93%	88%	91%	94%	97%	95%	97%	100%
Surface Only	100%	86%	49%	63%	43%	21%	36%	49%	70%	59%	69%	99%

Table 4-9. Percent of Time that Surface Cells Exceed Selected Standards Under Different Scenarios

Scenario Evaluated	Standard Compared (organisms /100mL)	Duwamish River (%)	Elliott Bay Except Shoreline North and West of Denny Way CSO (%)	Elliott Bay Along Shoreline North and West of Denny Way CSO (%)
Baseline Conditions	400	10 – 25%	< 5%	5 – 25%
	43	50 – 100%	0 – 50%	25 50%
Without CSO Conditions	400	10 – 25%	< 5%	< 1%
	43	50 – 100%	0 – 50%	5 – 25%

CSOs Only	400	0 – 5%	0 – 5%	5 – 25%
	43	1 – 10%	0 – 10%	10 – 25%

Figure 4-4. Percent of Time that Surface Layer Fecal Coliform Concentrations Exceed 400 Organisms/100 mL under Baseline Conditions

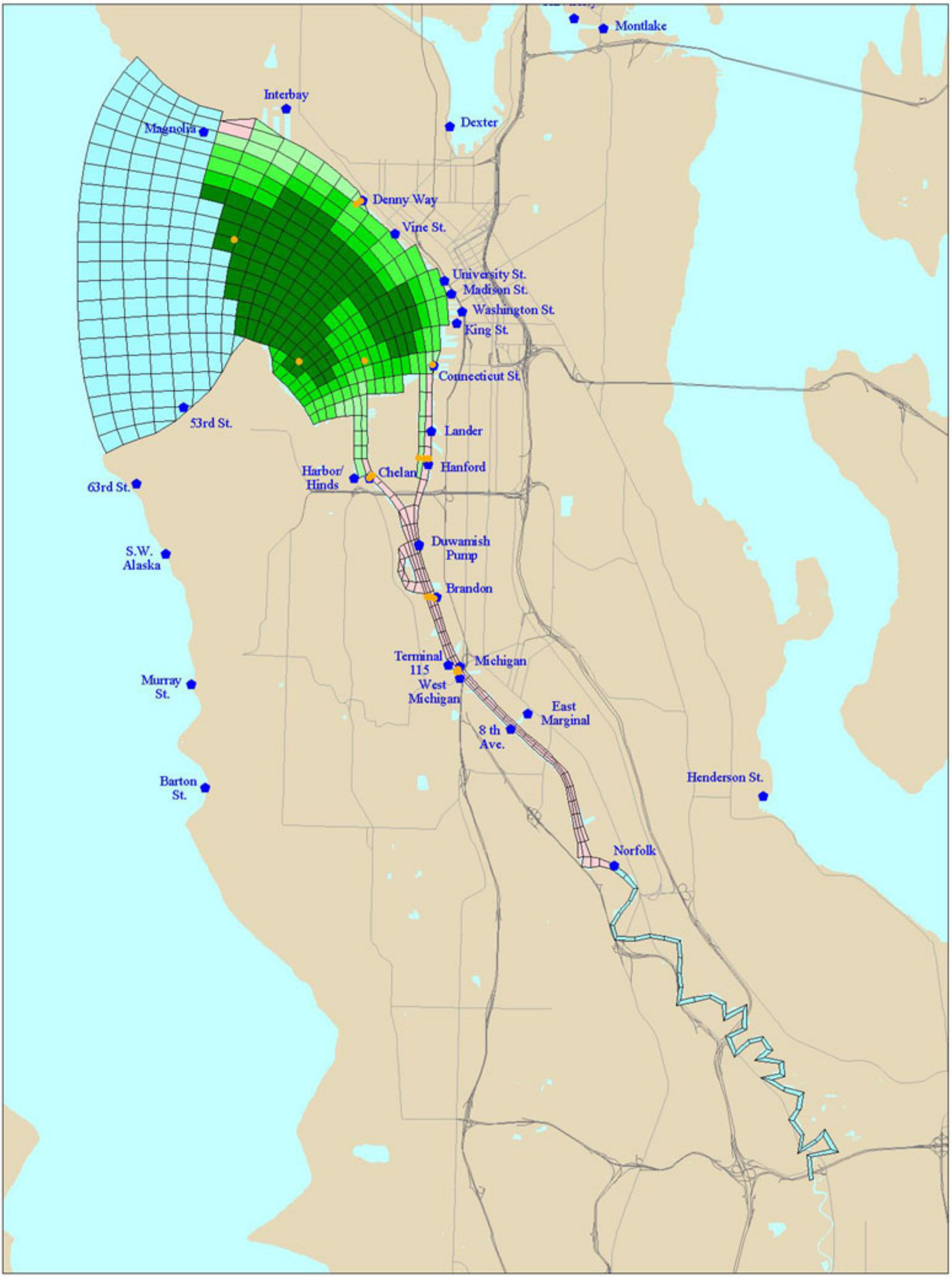

Figure 4-4. Percent of Time that Surface Layer Fecal Coliform Concentrations Exceed 400 Organisms/100 ml under Baseline Conditions

Concentration of the indicator bacteria would frequently exceed Washington State standards with- or without CSOs. These maps allowed for the identification of hot spots or areas where there may be a need to prioritize CSO and other source remediation efforts based on contamination levels. Higher fecal coliform bacteria would be seen with CSOs than without, particularly near the Denny Way CSO and along the north shore of Elliott Bay.

Cells adjacent to CSOs were investigated to identify the percent of time during the year that the fecal coliform concentrations attributable to CSO discharges would be below the geometric mean and 90th percentile state standards (Table 4-10). This analysis was conducted to assess fecal coliform concentrations if CSOs were the only sources of fecal coliforms in the river and bay (i.e., no other sources of fecal coliforms were assumed). Again, fecal coliform concentrations associated with CSO discharges in the Duwamish River near the CSO discharges would be below the state standards more frequently than concentrations in Elliott Bay near the CSO discharges into the bay.

Table 4-10. Percent of Time that the Fecal Coliform Concentrations in Surface Layer Cells Adjacent to CSO Discharges, Based on CSO Discharges Only, are Below their Geometric Mean and 90th Percentile Standards

CSO	Percent of time Below the Geometric Mean Standard[a]	Percent of Time Below the 90th Percentile Standard[b]
Duwamish River		
Norfolk	96	98
8th Ave	95	97
West Michigan	93	97
South Michigan	91	94
Brandon	90	93
Hanford/Rainier	88	92
Chelan	90	94
Harbor	89	94
Hanford	87	92
Lander	87	92
Connecticut	88	93
Elliott Bay		
Denny Way	51	63
King	52	68
South Magnolia	47	59

[a] Geometric mean standard of 200 colonies per 100 mL for the Duwamish River and 14 colonies per 100 mL for Elliott Bay.

[b] 90th percentile standard of 400 colonies per 100 mL for the Duwamish River and 43 colonies per 100 mL for Elliott Bay.

4.3.3 Risk from *Giardia* and Viruses Attributable to CSO Discharges in Surface Water

Two types of risk characterizations were undertaken for *Giardia* and viruses. The first was to compare the river and bay exposures to risk thresholds (based on ingestion of 50 mL of water) of 1 infection/100, 1 infection/1,000 and 1 infection/10,000. The second was to examine the Denny Way CSO discharge in greater detail and calculate the risk of infection during discharges and at several time intervals after discharges have ended.

Comparison of Predicted Virus and Giardia Risks to Different Risk Levels. Acceptable risk goals associated with swimming have been established based on epidemiological investigation of water quality and health effects at marine beaches (Cabelli et al. 1979 1981). The U.S. EPA did this as a part of a study for reassessment of ambient water quality standards. Recommendations by the U.S. EPA in the 1986 Ambient Bacteriological Water Quality report suggested that 35 enterococci/100mL related to a risk of 19 illnesses/1,000 swimmer-days (~2/100).

For drinking water the U.S. EPA (1989b) as part of the "*Surface Water Treatment Rule"* had suggested that an acceptable risk level goal would be 1 infection in 10,000 in a years time for 365 days of exposure. The current safety level for microbial risk from drinking water as suggested by the U.S. EPA is 10^{-4} yearly risk, with 2 L of daily exposure for 365 days. Viruses are capable of causing disease at very low concentrations and to meet this level of safety less than 0.00001 pfu/100L should be present in drinking waters (Regli et al. 1991). This was based on the rotavirus model as one of the best-described models, representing the risks for enteric viruses in drinking water.

In this case, 1/100, 1/1,000 and 1/10,000 for a single swimming event were chosen as the target goals. A 50-ml exposure and the dose-response models previously described were used. For comparison purposes, epidemic proportions are normally 1/10 or greater. Table 4-11 shows the virus and *Giardia* levels that would result in the three respective risks.

Table 4-11. Doses of Virus and *Giardia* Associated with Different Risk Levels

Microorganism	1/100 Risk	1/1,000 Risk	1/10,000 Risk
Giardia (cysts)	10	1.0	0.10
Total Enteric Viruses (organisms)	0.017	0.0017	0.00017

Maps were prepared that displayed the cells in the watershed and the percent of time the three risk levels would be exceeded. Figure 4-10 and Figure 4-11 shows the percent of time that each cell exceeds the 1/100 risk level based on incidental ingestion of 50 mL for viruses and *Giardia*, respectively, in CSO discharges. Figures 4-22 and 4-23 of Volume 1—*Overview and Interpretation Report* presents the same information for the 1/1,000 risk level and Figure 4-12 and Figure 4-13 shows similar information for the 1/10,000 risk level.

These show clearly that for the 1/100 risk the exceedances occur less than five percent of the time in any cell, and frequently less than one percent of the time. Also shown is that for the 1/1,000 risk the exceedances would occur only 1 to 5 percent of the time in the nearshore waters and in the river. For the more stringent safety goal (1/10,000 risk), the exceedances would be 5 to 10 percent of the time, except in the lower Duwamish River and along the Elliott Bay shoreline along the Seattle waterfront and Myrtle Edwards Park, where risks from *Giardia* may exceed 1 in 10,000 as frequently as 25 percent of the time. These results are summarized in Table 4-12.

Table 4-12. Percent of Time that Surface Cells Exceed Risk-Based Virus and *Giardia* Concentrations Based on CSO Discharges Only

Microorganism	Risk	Duwamish River (%)	Elliott Bay	Elliott Bay Shoreline
Total Enteric Virus	1 in 100	0 to 1%	0%	0 to 1%
	1 in 1,000	0 to 5%	0 to 1%	1 to 5%
	1 in 10,000	1 to 25%	0 to 5%	1 to 25%
Giardia	1 in 100	0 to 1%	0%	0 to 1%
	1 in 1,000	0 to 5%	0 to 5%	1 to 5%
	1 in 10,000	1 to 25%	0 to 10%	10 to 25%

Calculation of the Risk of Infection at the Denny Way CSO. Risks were calculated for the Denny Way CSO at various times during and after discharge using the Monte Carlo analysis. Figure 4-14 and Figure 4-15 displays the risks from viruses and *Giardia* graphically. The highest risks are seen during discharge. During discharge, the giardiasis risks exceed 1 in 10,000 about 78 percent of the time, 1 in 1,000 about 64 percent of the time, and 1 in 100 about five percent of the time. During discharge, the virus risks were estimated to exceed 1 in 10,000 about 76 percent of the time, 1 in 1,000 about 51 percent of the time, and 1 in 100 about one to two percent of the time. Within 6 to 24 hours after discharge, the risks were reduced by about 10 fold.

4.3.4 Shellfish Associated Risks

The results of the fecal coliform analysis indicates that fecal coliform concentrations in Elliott Bay and the Duwamish River frequently exceed Washington State standards considered to be protective for shellfish harvesting. These results indicate the potential for health risks from the consumption of raw or partially cooked shellfish from the river and bay. Quantitative risk estimates based on exposures to specific pathogens from shellfish consumption were not calculated because of limited exposure data on virus concentrations in shellfish.

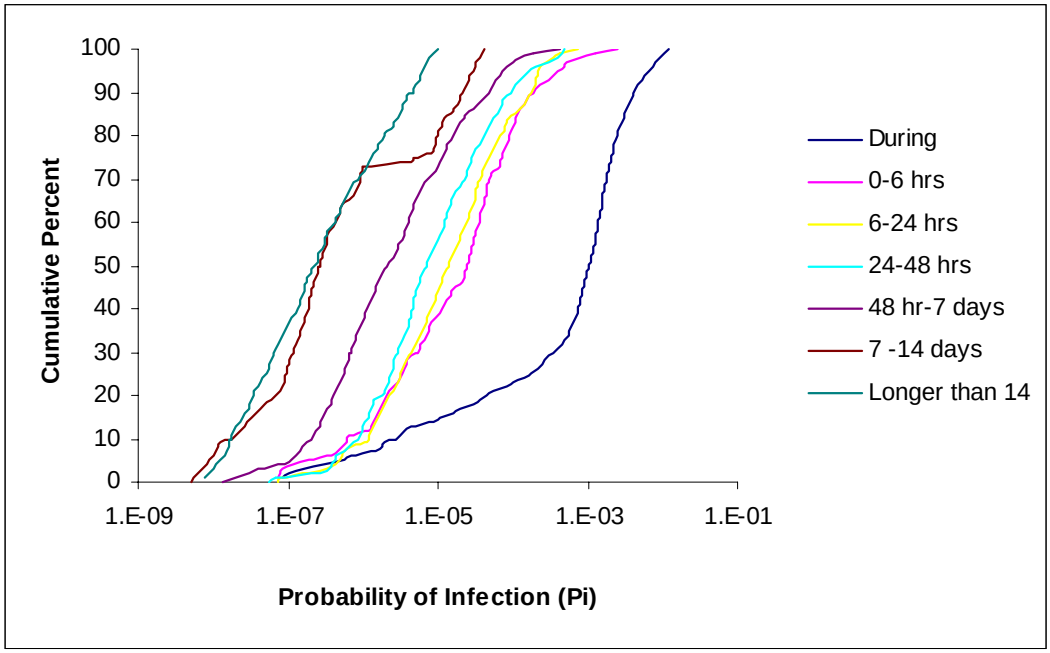

Figure 4-14 Virus Infection Risks at Denny Way

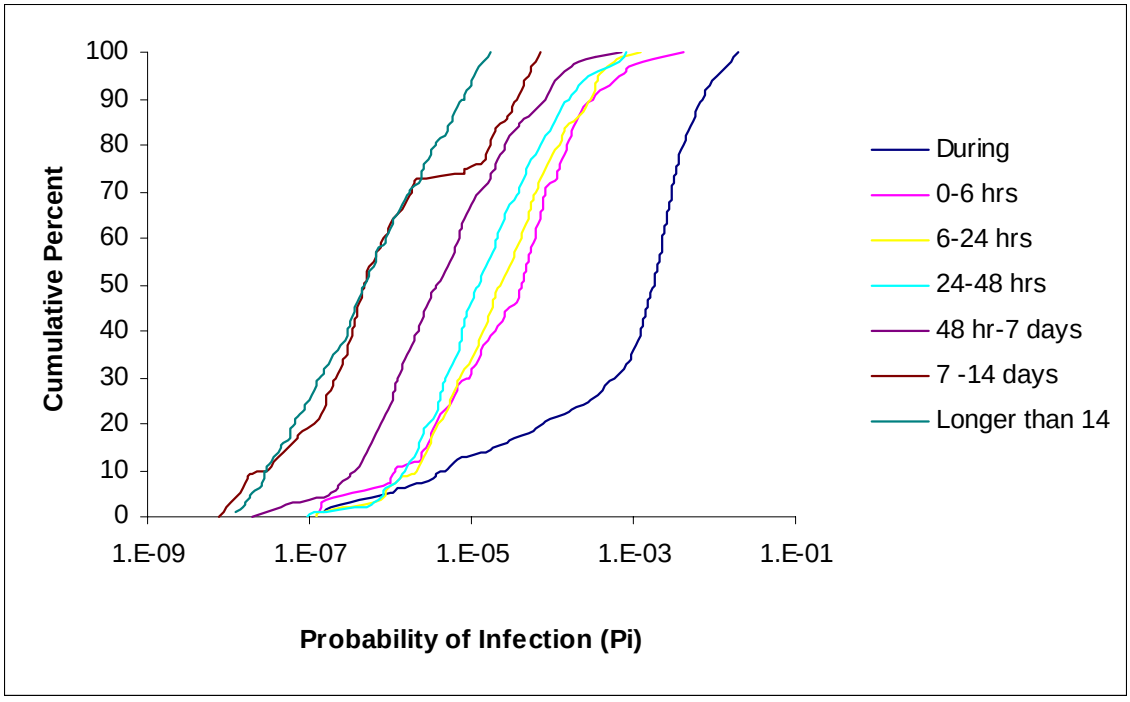

Figure 4-15 Giardia Infection Risks at Denny Way

4.3.5 Summary of the Risk Characterization

1. Fecal coliform concentrations frequently exceed state standards (based on comparison of hourly model output to the standards on a monthly basis), both with and without CSOs.

2. Removal of CSOs will not substantially change fecal coliform concentrations except in Elliott Bay along the shoreline north and west of the Denny Way CSO.

3. Based on the fecal coliform evaluation, the Duwamish River frequently does not have acceptable water quality for primary contact recreational uses (e.g., swimming, SCUBA diving, etc.), both with and without CSOs.

4. Based on the fecal coliform evaluation, Elliott Bay frequently has acceptable water quality for recreational uses, except along the shoreline north and west of the Denny Way CSO.

5. Based on the fecal coliform evaluation, the Duwamish River does not have acceptable water quality for harvesting of shellfish, either with or without CSOs.

6. Based on the fecal coliform evaluation, Elliott Bay frequently does not have acceptable water quality for harvesting shellfish.

7. Risk of infection from viruses (based on ingestion of 50 mL) attributable to CSO discharges exceeds risk thresholds applicable to recreational marine waters (1 in 100 and 1 in 1,000) less than five percent of the time and exceed risk thresholds applicable to drinking water (1 in 10,000) less than 10 percent of the time.

8. Risk of infection from *Giardia* (attributable to CSO discharges exceed risk-based concentrations for recreational marine waters less than five percent of the time and exceed risk based concentrations for drinking water less than 25 percent of the time.

9. Distributions for *Giardia* and viruses were plotted and Monte Carlo analysis was run for the estimate of risks associated with use during and after CSO discharges at the Denny Way CSO. This evaluation indicates a 10-fold reduction in risk is obtained within six hours of the end of the discharge.

10. During discharge, the giardiasis risks were estimated to exceed 1 in 1,000 about 64 percent of the time and exceed 1 in 100 about 5 percent of the time. During discharge, the virus risks were estimated to exceed 1 in 1,000 about 51 percent of the time and exceed 1 in 100 about 1 to 2 percent of the time.

4.3.6 Uncertainties in the Risk Characterization

Uncertainties in the risk characterization typically include the uncertainties associated with the exposure and effects characterizations. Additional uncertainties are associated with:

- Use of fecal coliforms as indicators of risk for direct exposures to surface water and consumption of seafood. The correlation between fecal coliform concentrations and illness may not be appropriate when considering the different sources of fecal coliforms (e.g., CSO discharges, storm water runoff, and agriculture).

- Lack of consideration of the risk of infection and illness from other organisms in CSO discharges. It is possible that the risks of infection have been under-estimated because all organisms were not quantitatively assessed.

- Lack of consideration of the risk of infection from organisms originating from other sources besides CSO discharges. Total risks under baseline and without CSO conditions are likely higher than the calculated risks because other sources of pathogens were not quantitatively assessed.

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