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

Appendix A: Problem Formulation, Analysis Plan, and Field Sampling Work Plan
A2: Analysis Plan

Prepared by the

Duwamish River and Elliott Bay

Water Quality Assessment Team

February 1999

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LIST OF ACRONYMS

ACR	Acute-chronic ratio
ADI	Allowable daily intakes
ASTM	American Society of Testing and Materials
BCF	Bioconcentration factor
BSAF	Biota-sediment accumulation factor
COPC	Constituents of potential concern
CSO	Combined sewer overflow
EEC	Expected environmental concentrations
EED	Expected environmental doses
GMAV	Genus mean acute values
HEAST	Health Effect Assessment Summary Tables
HI	Hazard index
HQ	Hazard quotient
IRIS	Integrated Risk Information System
MID	Minimum infective dose
MDL	Method detection limit
NCASI	National Council of the Pulp and Paper Industry for Air and Stream Improvement
NOAA	National Oceanic and Atmospheric Administration
NOAEL	No observed effect level
PER	Pulse exposure ratio
RfD	Reference dose
TDI	Tolerable daily intakes
TRV	Toxicity reference values
TSS	Total suspended solids
U.S. EPA	United States Environmental Protection Agency
WHO	World Health Organization
WQA	Water Quality Assessment
WERF	Water Environment Research Foundation

1. INTRODUCTION TO THE ANALYSIS PLAN

The purpose of the Duwamish River/Elliott Bay Water Quality Assessment (WQA) Analysis Plan is to evaluate how the risk hypotheses set forth in the Problem Formulation and Planning Document (Appendix A1) will be assessed using available and new data (U.S. EPA 1996a). This analysis plan also presents and defines the overall design of the risk assessment, identifies the data required to test the risk hypotheses, and presents the methods that will be used in the analysis phase of the risk assessment (U.S. EPA 1996a). This analysis plan completes the problem formulation and identifies the level of uncertainty in the exposure pathways and relationships identified during problem formulation that will be pursued in the analysis phase.

The analysis phase of the risk assessment presents the technical evaluation of the risk hypotheses and the relationships between the stressors and their effects on the identified receptors. The analysis phase includes an exposure characterization[1] and an effects characterization[2], along with an evaluation of ecosystem and receptor attributes to evaluate the questions and issues identified in problem formulation. The end product of the analysis will be the descriptions of exposure and effects that will form the basis for reaching conclusions concerning risk from combined sewer overflow (CSO) discharges and other sources to the Duwamish River and Elliott Bay (U.S. EPA 1996a).

Section 2 of this analysis plan summarizes the conceptual site models and the risk hypotheses, which were developed in problem formulation. Data needed to evaluate these hypotheses are identified in Section 3. The hydrodynamic model that will be used to develop the chemical and bacteriological concentrations used in the risk assessment is introduced in Section 4. The methodologies used to evaluate each major group of receptors—aquatic life, wildlife, and people—are also reviewed in Section 4. Section 5 discusses the level of uncertainty associated with evaluating different exposure pathways and different receptors. Finally, cited references are listed in Section 6.

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[1] Exposure assessment involves the estimation of the magnitude, frequency, and duration of exposure, and characterization of the exposed individuals and/or populations (U.S. EPA 1996a).

[2] Effects characterization is the portion of the analysis phase that evaluates the ability of a stressor to cause adverse effects under a particular set of circumstances (U.S. EPA 1996a).

2. REVIEW OF CONCEPTUAL SITE MODELS AND RISK HYPOTHESES

2.1 Review of Identified Exposure Pathways

The problem formulation, prepared by the WQA Team, identified a series of pathways through which the biological resources under evaluation could be exposed to human impacts that might result in adverse effects (Appendix A1). The receptors identified are:

- Aquatic life—salmon outmigrants, resident fish, epibenthos, and infaunal community

- Wildlife—spotted sandpipers, great blue herons, bald eagles, and river otters

- Human Health—primary and secondary users of the river and bay, and people who consume seafood collected from the river and bay.

Four principal types of stressors are identified as having potential impact on the study area. They are:

- Toxic chemicals

- Physical disturbance

- Changes in water quality parameters

- Microbial contamination

The problem formulation identified that aquatic life was at potential risk from exposure to toxic chemicals, physical disturbances to the environment, and changes in water quality parameters. Similarly, wildlife was identified as being at potential risk from exposure to toxic chemicals and physical disturbances to the environment. People were considered to be at potential risk from exposure to toxic chemicals and microbial contaminants.

2.2 Review of Risk Hypotheses

Using the conceptual models presented in the problem formulation (Appendix A1), a number of risk hypotheses were developed to address the potential risks resulting from CSO discharges and other sources. These hypotheses were developed to address the potential risks associated with toxic chemicals, physical disturbance, changes in water quality parameters (e.g., salinity, dissolved oxygen (DO), temperature, and pH), and microbial contaminants. These hypotheses distinguished between risks from all sources including CSOs (baseline risk) and the risks associated only with CSO discharges (CSO risk).

The following table (Table 2-1) presents the risk hypotheses developed for ecological receptors, which includes both aquatic life and wildlife receptors, and human health for each type of stressor that was present in the Duwamish River and Elliott Bay system.

Table 2-1.   Risk Hypotheses Identified in Problem Formulation

Ecological Receptor Hypotheses	
Toxic Chemical Hypotheses	Elevated chemical baseline concentrations cause reduced survivorship and reproduction of aquatic life and wildlife resources present in the Duwamish River and Elliott Bay.
	Chemicals present in intermittent CSO discharges cause reduced survivorship and reproduction of aquatic life and wildlife present in the Duwamish River and Elliott Bay.
Physical Disturbance Hypotheses	Changes in the physical structure of receiving waters resulting from seasonal runoff cause reduced survivorship and reproduction of aquatic life and wildlife present in the Duwamish River and Elliott Bay.
	Changes in the physical structure of receiving waters resulting from intermittent CSO discharges cause reduced survivorship and reproduction of aquatic life and wildlife resources present in the Duwamish River and Elliott Bay.
Water Quality Parameter Hypotheses	Changes in water quality parameters in ambient waters resulting from seasonal runoff results in reduced survivorship and reproduction of aquatic life and wildlife resources present in the Duwamish River and Elliott Bay.
	Changes in water quality parameters resulting from intermittent CSO discharges cause reduced survivorship and reproduction of aquatic life and wildlife resources present in the Duwamish River and Elliott Bay.
Human Health Receptor Hypotheses	
Toxic Chemical Hypotheses	People using the Duwamish River/Elliott Bay system for recreation (e.g., swimming, fishing, and boating), commercial fishing and subsistence food gathering are at increased risk of non-cancer and cancer health effects from elevated chemical baseline concentrations in surface waters, sediment, fish, and shellfish.
	People using the Duwamish River/Elliott Bay system for recreation (e.g., swimming fishing, and boating), commercial fishing, and subsistence food gathering are at increased risk of non-cancer and cancer health effects from elevated chemical concentrations in intermittent CSO discharges to surface waters and sediments.
Microbial Contaminant Hypotheses	People using the Duwamish River/Elliott Bay system for recreation (e.g., swimming, fishing, and boating), commercial fishing and subsistence food gathering are at increased risk of infection and symptomatic illness from elevated concentrations of microbial contaminants present at baseline conditions in surface waters, sediment, fish, and shellfish.
	People using the Duwamish River/Elliott Bay system for recreation (e.g., swimming and fishing), commercial fishing and subsistence food gathering are at increased risk of infection and symptomatic illness from elevated concentrations of microbial contaminants present in intermittent CSO discharges to surface waters and sediments.

2.3 Assessment of Risk Hypotheses

Each of these risk hypotheses will be tested using the basic components of risk assessment identified above—exposure assessment, effects characterization, and risk characterization. Data will be gathered to determine the exposure level from baseline conditions, CSO discharges, and other sources for each receptor from each potential stressor (chemical, physical, changes in water quality, and microbial contaminants). These data will be compared to the identified effect thresholds for each stressor. Exposure levels of stressors will be evaluated as point estimates for aquatic life, wildlife, and human health followed by an examination of probabilistic ranges for aquatic life and wildlife. Receptors with exposure levels that exceed effects thresholds in the detailed, probabilistic risk assessment, will result in predictions of significant risk for specific aquatic life receptors. In general, exposures that result in exceedances of pre-established effect thresholds will be identified as posing risks.

3. DATA NECESSARY TO ADDRESS HYPOTHESES

3.1 Chemical Stressors

Chemicals present in CSO discharges, in receiving waters downstream of CSO discharge locations, and in tissues of prey organisms are potential stressors that have been identified in the conceptual site models. The specific data needs in each environmental medium to support the risk assessment are identified in Table 3-1 through Table 3-3.

As chemicals are likely to be present in CSO discharges both dissolved in the water and bound to particulate matter, it will be necessary to collect data for both water and particulates in CSO discharges (Table 3-1). Measurement of the volumetric CSO flow rate ($Q_{cso}(t)$) and the mass of the total suspended solids (TSS) per volume of CSO discharges (M_{TSS}) will be required to calculate the initial chemical contribution of CSO discharges to the sediment (Table 3-1).

Table 3-1. Chemical and Physical Data Needs for CSO Discharges

CSO Chemical Data Needs	Description
$C_{Dissolved, CSO}$	Chemical concentration in filtered discharges samples (i.e., dissolved)
$C_{Total\ CSO}$	Chemical concentration in unfiltered discharge samples (i.e., total)
$C_{TSS,\ CSO}$	Chemical concentration in TSS (i.e., particulates) in CSO discharges
$C_{Settleable,\ CSO}$	Chemical concentration in the settleable solids by size fraction in CSO discharges
$f_{Settleable,\ CSO}$	Mass fraction of TSS by size fraction in CSO discharges
$Q_{CSO}(t)$	Volumetric CSO flow rate
M_{TSS}	Mass of TSS per volume of CSO discharges

Similar chemical and physical data will be required for the receiving waters upstream and downstream of CSO discharge points to determine the baseline conditions of the Duwamish River and Elliott Bay estuary (Table 3-2).

Table 3-2. Chemical and Physical Data Requirements for Receiving Waters Upstream and Downstream of CSO Discharge Points

Receiving Water Chemical Data Needs	Description
$C_{Dissolved, Baseline}$	Chemical concentration in filtered ambient water samples (i.e., dissolved)
$C_{Total, Baseline}$	Chemical concentration in unfiltered ambient water samples (i.e., total)
$C_{TSS, Baseline}$	Chemical concentration in ambient water TSS
$C_{Settleable, Baseline}$	Chemical concentration in the settleable solids by size fraction in ambient water
$f_{Settleable, Baseline}$	Mass fraction of settleable solids in ambient water TSS
$Q_{Baseline}(t)$	Volumetric Flow Rate of the River
$f_{Chemical, CSO}$	Mass fraction of chemicals in the baseline water column and baseline sediments attributable to CSO discharge
M_{TSS}	Mass of TSS per volume of baseline flow of the river in ambient water

To test hypotheses that address the exposure of receptors to chemicals in vertebrates and invertebrates will require data on the chemical concentrations in the tissue of a wide array of organisms living in the study site (Table 3-3). Because different methods of preparing seafood can affect the range of chemical concentrations present in fish and shellfish tissues (Sherer and Price 1993), fish and shell fish will be analyzed both uncooked and cooked.

Measured chemical concentrations in fish and shellfish tissues will be used in both the wildlife (uncooked only) and human health (both uncooked and cooked) risk assessment components. Chemical concentrations will also be measured in background/reference areas for comparison with concentrations present in the Duwamish River/Elliott Bay estuary. Specific sampling locations are identified in the Field Sampling Work Plan. Measurements taken from the reference areas are show in Table 3-4.

Table 3-3.   Chemical Data Needs for Fish and Shellfish Tissues

Tissue Chemical Data Needs	Description
$C_{Invertebrates}$	Chemical concentration in invertebrates (a composite of appropriate size fractions of different species)
$C_{Small\ fish}$	Chemical concentration in small fish (< 9 inches)
$C_{Large\ fish\ fillet}$	Chemical concentration in large fish fillets (cooked and uncooked)
F_{Fillet}	Mass fraction of fish in the fillet
$C_{Large\ fish\ remainder}$	Chemical concentration in large fish remaining after filleting (cooked and uncooked)
$F_{Remainder}$	Mass faction of fish remaining after filleting
$C_{Shellfish}$	Chemical concentration in shellfish (cooked and uncooked)

Table 3-4.   Chemical Data Needs in Background/Reference Areas

Background Chemical Measurements	Description
$C_{Sediment}$	Chemical concentration in bulk sediment
C_{Total}	Chemical concentration in unfiltered water samples (i.e., total)
$C_{Tissues}$	Chemical concentrations in fish and shellfish

3.2 Physical Stressors and Water Quality Parameters

The discharge of effluent from CSOs into the receiving environment can result in the physical changes in water and sediment quality that were identified in the conceptual site models. The specific data needs from CSO discharges and the receiving environment to support the risk assessment are identified in Table 3-5 and Table 3-6, respectively.

Table 3-5.   Water Quality Parameter Data Needed from CSO Discharges

CSO Water Quality Data Needs	Description
Salinity	Salinity of unfiltered CSO discharges samples
Dissolved Oxygen	Dissolved oxygen content of unfiltered CSO discharges samples
Temperature	Temperature of unfiltered CSO discharges samples

Table 3-6.   Water Quality Data Needed from the Receiving Environment Downstream of CSO Discharge Points

Receiving Environment Data Needs	Description
$\Delta_{Salinity}$	Amount and duration of salinity change in the receiving environment during a CSO discharge
$\Delta_{Dissolved Oxygen}$	Amount and duration of DO change in the receiving environment during a CSO discharge
$\Delta_{Temperature}$	Amount and duration of temperature change in the receiving environment during a CSO discharge
Sedimentation Rate	Deposition rate of new sediment during a CSO discharge
Habitat Loss	Loss of sediment surface from CSO footprint[3] after a CSO discharge, in m^2
$\Delta_{Velocity}$	Amount and duration of change in river velocity during a CSO discharge

Δ = Change in this parameter from existing levels

3.3 Microbial Contaminant Stressors

To establish whether CSO discharges represent a source of disease causing (pathogenic) microorganisms to the Duwamish River and Elliott Bay, raw sewage will be screened for the presence of *Salmonella*, *Listeria*, *Giardia*, *Yersinia* and enteric viruses. The ratio of indicator bacteria (fecal coliforms) to specific pathogens will be calculated. This will allow the use of historic and ongoing fecal coliform sampling data to predict the amount of disease causing bacteria and viruses present in CSO discharge and receiving waters

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[3]   The CSO footprint is the area directly impacted by the physical flow of the CSO discharge.

upstream and downstream of CSO discharge points. The specific data needs for microbial contaminants are presented in Tables 3-7, 3-8, and 3-9.

Table 3-7.   Raw Influent to West Point Treatment Plant Data Needs

West Point Treatment Plant Needs	Description
Salmonellae	Number per liter
Yersinia enterocolitica	Number per liter
Listerae	Number per liter
Enteric viruses	Number per liter
Giardia	Number per liter

Table 3-8.   Disease Causing Pathogens Data Needs for CSO Discharges

CSO Disease Causing Pathogens Data Needs	Description
Pathogens	Number of Salmonellae, Yersinae, Listerae, *Giardia*, and Enteric viruses estimated based on number in raw sewage
Fecal Coliforms	Number of fecal coliform bacteria present in CSO discharge
$Q_{baseline}(t)$	Volumetric Flow Rate

Table 3-9.   Microbial Contaminant Data Needs for the Receiving Environment Downstream of CSO Discharge Points

Receiving Environment Microbial Contaminant Data Needs	Description
Fecal coliforms, Salmonellae, Enteric Viruses, *Yersinia enterocolitica*	Number in mussels adjacent to CSO immediately post discharge and one sample per day until levels reach background.
Fecal Coliforms	Number of fecal coliform bacteria present in receiving waters upstream of CSO discharge points.
Fecal Coliforms	Number of fecal coliform bacteria present in receiving waters downstream of CSO discharge points.

4. HYDRODYNAMIC MODELING OF CHEMICAL CONCENTRATIONS

To address the issue of how much risk is associated with CSO discharges in the Duwamish River and Elliott Bay relative to other sources using only field samples is a nearly impossible task. It would require an enormous number of samples, and would depend on our ability to identify a specific chemical that is only characteristic of CSOs. Additionally, the costs associated with such a field monitoring program would be prohibitively expensive. Therefore, this risk assessment needs another method that could act as a surrogate for a field monitoring program to give realistic results for use in the risk assessment.

4.1 Application of the Hydrodynamic Model in the WQA process

The WQA Team has created a model of the Duwamish River and Elliott Bay that will predict where chemicals from various discharges travel in these water bodies. While the model is mathematical, it is constructed to simulate the most important aspects of the physical behavior of the estuary system. The Duwamish River/Elliott Bay model consists of two components. The first is a hydrodynamic model that describes the water flow, the second is the chemical and bacteria fate and transport model that describes the addition, removal, movement, and behavior of chemicals and bacteria in the study area, including the chemicals that reach the sediments.

The model will be run initially with CSO inputs as they are now to provide a representation of current conditions, then run again with zero CSO discharges. The difference between the chemical concentration under these two situation will give an estimate of the contribution of CSOs to the concentration of chemicals and bacteria to the study area. These numbers will then be used by the risk assessment.

The physical area covered by the model includes the Green River from the Interstate 405 bridge to the outer bounds of Elliott Bay near Alki Point. The model divides this area into 512 cells, and divides the depth into 10 layers, providing 5,120 cells (Figure 4-1). Thus, the model can realistically simulate how chemicals from the CSOs and other sources are distributed to the 5,120 locations within the Duwamish River and Elliott Bay estuary ecosystem.

To support the development and verification of the model, field data were collected at 39 points within the study area. The data collected to support water flow portion of the model included information from water level sensors, meters that measure the speed and direction of water movement, and automated meters that record water temperature and salinity.

The chemical and bacterial portions of the model were developed and verified with data taken from 39 water stations on a weekly schedule of non-storm sampling and more

frequent sampling associated with storms. Most of these stations were in adjacent groups.
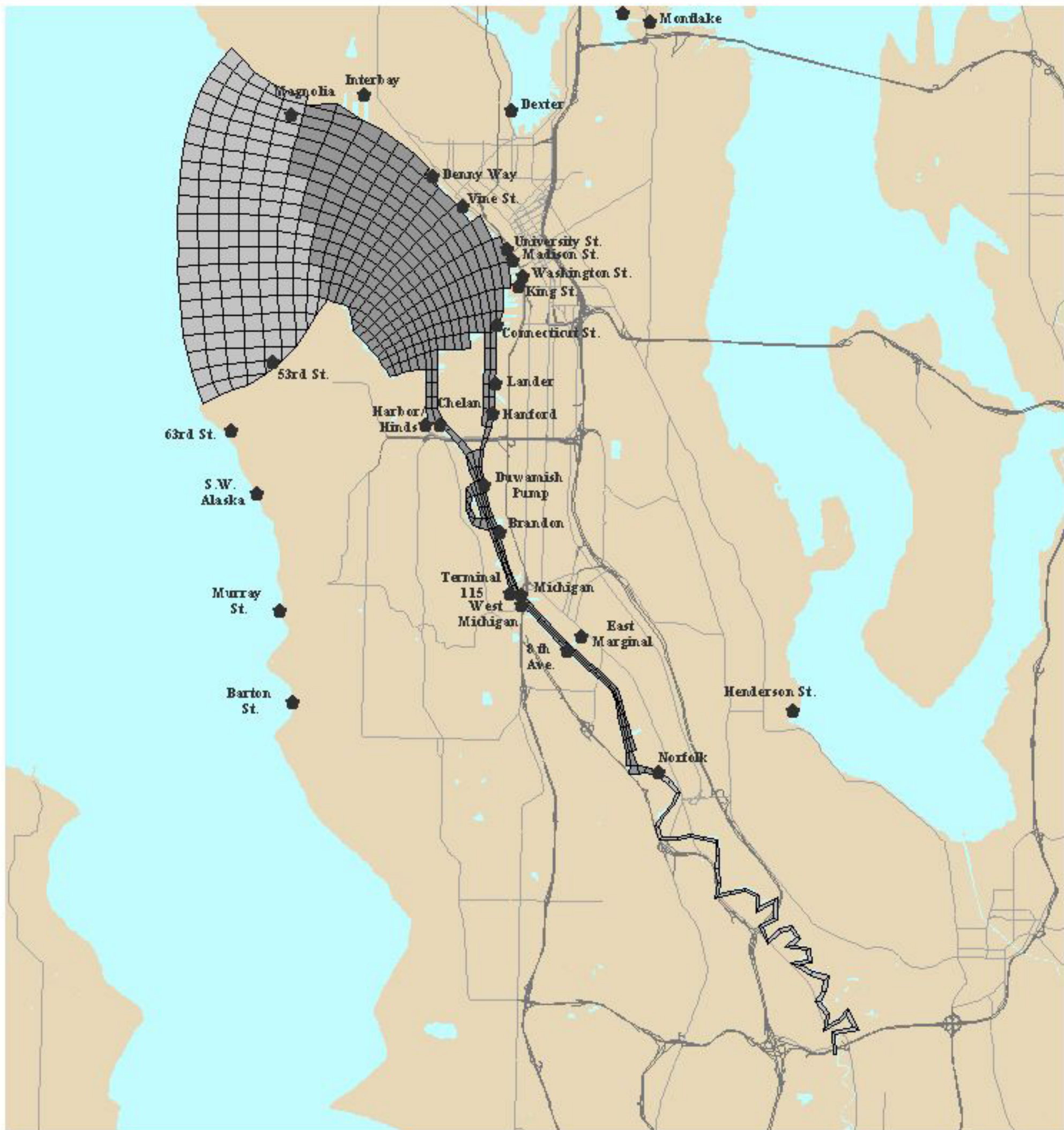
Fig. 4-1. Cell Map

Monlake
Magnolia
Interbay
Dexter
Denny Way
Vine St.
University St.
Madison St.
Washington St.
King St.
Connecticut St.
53rd St.
Lander
Chelan
Hanford
Harbor/Hinds
63rd St.
Duwamish Pump
S.W. Alaska
Brandon
Terminal 115 West Michigan
Michigan
Murray St.
East Marginal
8th Ave.
Barton St.
Henderson St.
Norfolk

Study Area

Cells outside the Study Area

CSO

Water Feature

Freeways

Primary Streets

ixtev/gis/files/asproj/whqutly/water_over.apr

King County
Department of Natural Resources
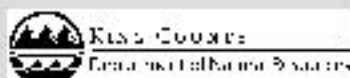
0.3 0 0.3 0.6 0.9 1.2 1.5 Miles

February 26, 1999

to CSOs, with three surface/subsurface pairs of stations located across the river. A station was located upstream of the study area at the Tukwila gauging station to measure inputs to the study area from the river. A corresponding station was also located on the Puget Sound boundary of the model. At these stations, measurements were taken for conventional parameters such as nutrients and oxygen concentration, bacterial numbers, and metals and organic chemicals. The sediment data used for the model were derived from a number of sources including weekly sediment samples at three sites and a large number of sediment samples from recent sediment sampling programs in the area. The measurements from these sites included metal and organic chemical concentrations, and physical characteristics such as sediment particle size. A discussion of how these data were summarized for inclusion in the model is included in Appendix B1: Hydrodynamic and Fate and Transport Numerical Model for the Duwamish River and Elliott Bay.

A crucial piece of information in the building of a fate and transport model of CSO impacts was CSO sample collection during storm events. Automated samplers were installed in five of the most active CSOs in the study area. These samplers collected CSO effluent samples at various times during discharge events. Samples were analyzed for the same chemical and bacteria parameters as the receiving water and sediment samples. These data were used as the CSO inputs to the model.

To ensure the model provides accurate information to the risk assessment, it is calibrated to accurately predict field data concentrations. Once the calibration is completed, the model is further validated with a separate set of field data to determine how well the model works under different sets of conditions. If the model is correctly calibrated, the predicted concentrations should closely match observed concentrations. After the model has been validated, the model can realistically predict how chemicals from various sources are transported through the Duwamish River and Elliott Bay. The computer-based model will then be used to replace a large-scale field monitoring program for the Duwamish River and Elliott Bay, providing chemical concentrations at particular sites of interest for evaluation in the risk assessment.

4.2 Interfacing Model Output with the Risk Assessment

Once the model has been successfully calibrated and validated, it will be used to estimate chemical and bacterial concentrations in the water column and sediments throughout the study site both under baseline and without CSO conditions. The risk assessment will use summary concentrations over one or more model cells (e.g., average, median, 75 percent, and 95 percent). The value for those cells will be used as the input for the risk assessment. It will not be used to estimate tissue concentrations in fish, shellfish, and invertebrates that could be eaten by aquatic life, wildlife, or people. These will instead be measured directly in samples collected from the study site for use in the risk assessment. However, the portion of the total chemical concentration that is derived from CSOs in the water or sediment, to which the animals are exposed, can be used to estimate the proportion of a chemical found in tissue that is of CSO origin. Water and sediment concentrations provided by the hydrodynamic model will be used in the risk assessment

to generate exposure concentrations for use in estimating risks from each constituent of potential concern (COPC) to specific receptors as discussed in the following sections.

4.3 Apportioning Source Contributions to Tissue Chemical Concentrations

The contribution of CSO discharges versus other sources to each environmental compartment will be determined primarily by using the hydrodynamic model. For chemicals that do not bioaccumulate[4], the measured tissue concentrations will be partitioned between CSO and other sources using the percent contribution of each source to water and sediments at specific locations within the study area. These relative contributions will be determined by comparing the results of the two modeling runs— baseline conditions and zero CSO discharge. For chemicals that can bioaccumulate, it may be necessary to determine the contribution of short-term CSO discharges to fish and shellfish tissue concentrations using an unsteady-state bioaccumulation model (NCASI 1996; Abbott et al. 1995). Unsteady-state bioaccumulation modeling allows exposure to vary over time, while depuration[5] and metabolic rates remain constant. Thus, fish and shellfish tissue concentrations will change over time following a CSO discharge until they reach some equilibrium with long-term sources[6]. It is important for assessing risks of CSO events because ignoring the time variation in exposure overestimates bioconcentration factors (BCFs) and biota-sediment accumulation factors (BSAFs). Application of the unsteady-state bioaccumulation model will be decided on a chemical-by-chemical basis based on their potential for bioaccumulation and the magnitude of their concentrations in CSO discharges, as well as the availability of information concerning uptake, metabolism, and depuration for each chemical.

While the hydrodynamic model will not be used to estimate fish and shellfish tissue concentrations, it will be useful in attributing tissue concentrations to either CSO discharges or other sources. In the wildlife risk assessment, for chemicals that do not bioaccumulate, the measured tissue concentrations will be partitioned between CSO and other sources using the percent contribution of each to water and sediments in specific locations within the study area.

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[4] Bioaccumulation is the accumulation of chemicals in the body tissues of fish and shellfish.

[5] Depuration is the loss of chemicals by a variety of excretion mechanisms to the environment from fish and shellfish tissues.

[6] Long-term sources would be sediments, which contain chemicals from both CSO and other sources as well as CSO and other sources in the water column.

4.4 Risk Assessment Methodologies

Potential risks in the Duwamish River or Elliott Bay posed to the three main receptors—aquatic life, wildlife, and people—will be determined using a common set of tools. For example, exposure concentrations will be expressed as either estimated exposure concentrations (EECs) for aquatic life, or estimated exposure doses (EEDs) for wildlife and human health. Both EECs and EEDs will be determined from the results of a statistical evaluation of water and sediment data from the hydrodynamic modeling and biota tissue concentrations (Figure 4-2). Additionally, the contribution of CSO discharges and other sources to EECs and EEDs will be determined and the proportion of risk will be attributed to each source.

Effects thresholds will be determined for each set of receptors from either Washington State standards, U.S. EPA water quality criteria, or existing toxicity bases for use in evaluating the receptor-specific exposure concentrations. Exposure concentrations will be compared to effects thresholds in the risk characterization of the risk assessment to determine total risks to aquatic life and wildlife and to predict risks to human health (both non-cancer and cancer risks and risks of infection). A series of corroborative evaluations will be conducted (e.g., effluent bioassays, benthic surveys) to validate the risk predictions for specific ecological receptors. Also, site-specific investigations will be used to determine patterns of human seafood consumption and fish and shellfish tissue concentrations for use in the risk assessment.

In the following sections, the general methodology that will be used to identify candidate chemical stressors and to determine risks to aquatic life, wildlife, and human health are presented. Additionally, the specific assumptions that must be made in order to complete the risk evaluations are reviewed and the potential impact of these assumptions is addressed.

4.5 Identification of Candidate Chemical Stressors

Chemicals will be screened for potential risks in the WQA using the procedure outlined in Figure 4-3. Chemicals will be initially screened for their ability to cause human cancer. These chemicals will be identified as COPCs. Non-cancer-causing chemicals will then be screened for frequency of detection. Infrequently detected chemicals are then further evaluated to determine if the method detection limit (MDL) is less than the

detection goal[7]. Infrequently detected chemicals will be identified as posing uncertain risks[8]. Those with MDLs less than the detection goal will not be evaluated further.

Frequently detected chemicals are then compared against water and sediment criteria after calculating the appropriate 95th percentile (the population 95th percentile for the water column and the 95th percentile on the mean for sediments). Chemicals with percentiles exceeding the criteria will be identified as being COPCs requiring further evaluation in the detailed risk assessment. Those with percentiles less than criteria will not be selected for further evaluation.

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[7] The detection goal is the lowest concentration for a particular chemical which would produce a hazard quotient of 1.0 for the most sensitive receptor, based on the identified exposure pathway for that receptor.

[8] Risk is uncertain because the high MDLs automatically generate hazard quotients greater than one, without regard to the actual concentrations of this chemical in the environment.

EDRP = East Division Reclamation Plant
EEC = Expected Environmental Concentration
EED = Expected Environmental Dose
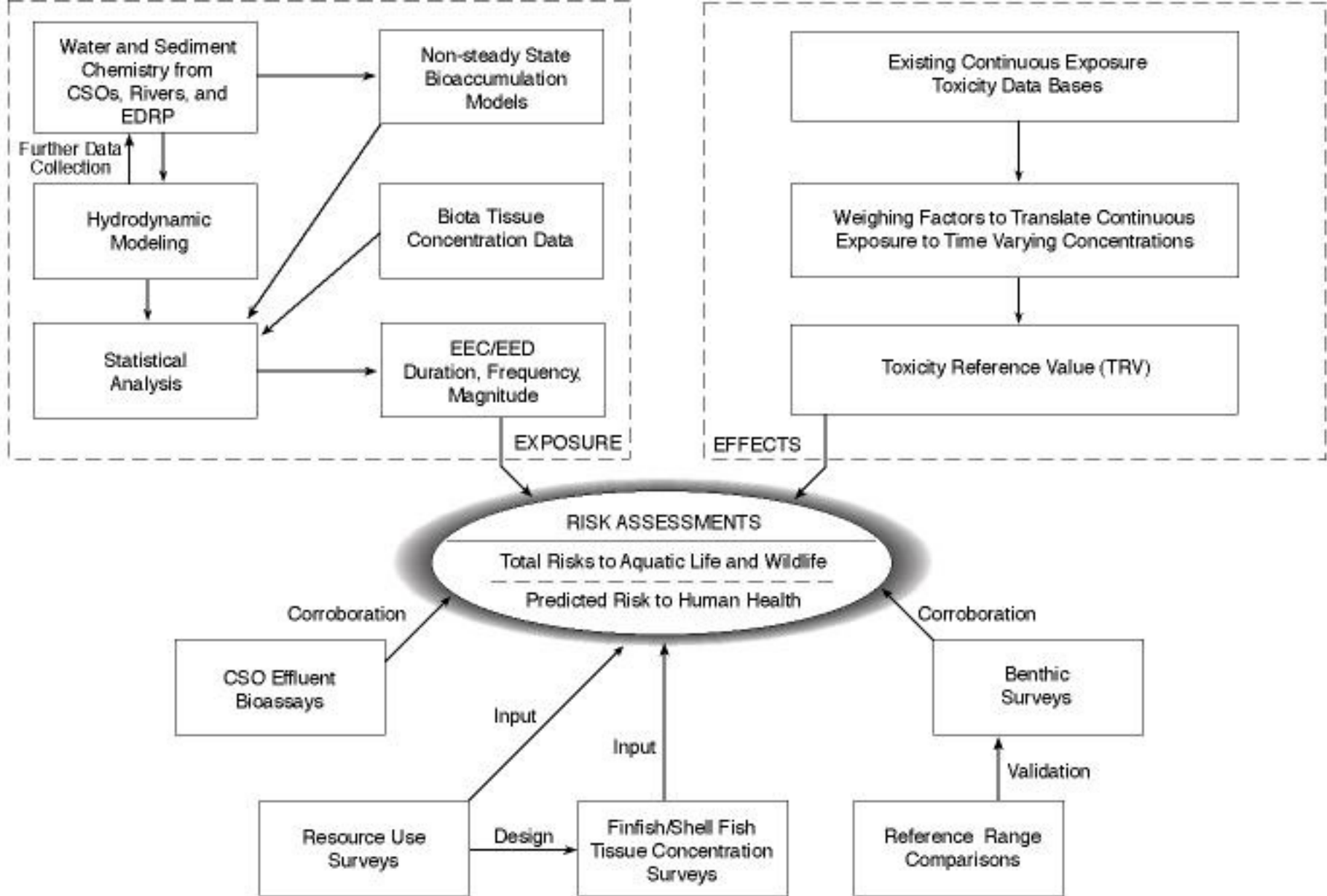
Figure 4-2.
Technical Approach to
Performing the Risk Assessment

Is the
Chemical a
Carcinogen? — Yes

No

Is the
Chemical
Detected
in more than
5% of the
samples? — No → Is the
Detection
Limit <
the Detection
Limit Goal — No → Risks from this
Chemical are
Uncertain

Yes

Yes

Calculate the 95th
Percentile

Is
95th Percentile
> Criterion? — No

Yes
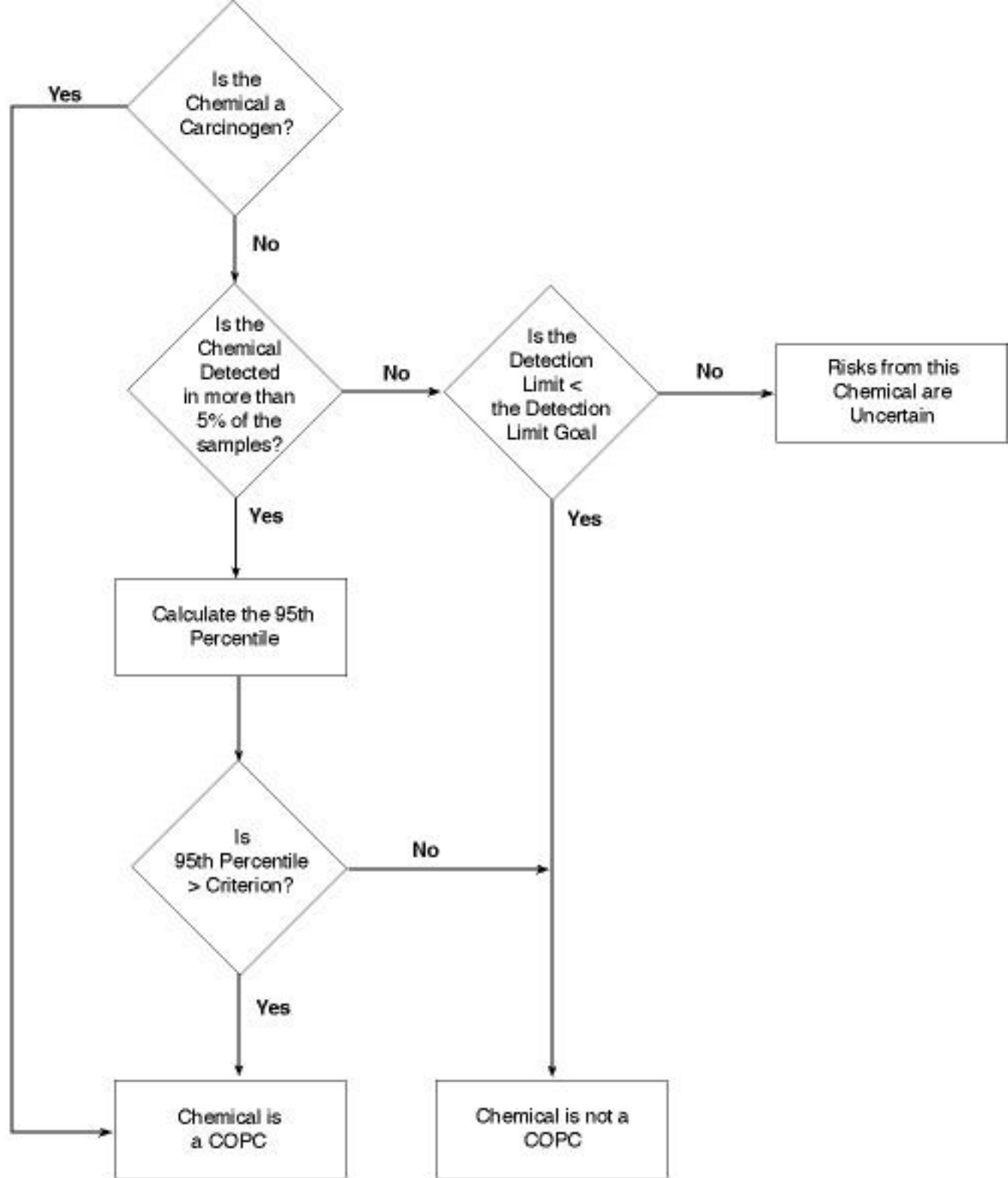
Chemical is
a COPC

Chemical is not a
COPC

Figure 4-3.
Decision Flowchart for Identifying
Constituents of Potential Concern

Water quality and sediment criteria will be selected following the hierarchy presented in Table 4-1 and Table 4-2, respectively. Criteria will be evaluated in the order listed, with the first authority in the specified order to identify water quality or sediment criteria being used as the risk screening value. For example, a review of the U.S. EPA ambient water quality criteria revealed no criterion for manganese. Consequently, the criteria developed by Parametrix following the U.S. National Guidelines (Stephan et al. 1985) will be used in the risk screening. If no U.S. EPA ambient water quality criterion is available (excepting manganese and cobalt), then the lowest literature value available will be used after applying a safety factor of 20.

Table 4-1. **Water Column Selection Hierarchy**

Freshwater Criteria	Washington State Surface Water Quality Standards (Title 173-201A WAC), **or** U.S. EPA Ambient Water Quality Criteria (AWQC) (U.S. EPA 1994), **or** Parametrix Criteria for Manganese and Cobalt (Parametrix 1993), **or** Lowest Literature Value Divided by 20.
Saltwater Criteria	Washington State Surface Water Quality Standards (Title 173-201A WAC), **or** U.S. EPA Ambient Water Quality Criteria (U.S. EPA 1994), **or** Parametrix Criteria for Manganese and Cobalt (Parametrix 1993), **or** Lowest Literature Value Divided by 20, **or** Freshwater Criterion when no saltwater criterion/literature values are available.

Table 4-2. **Sediment Criteria Selection Hierarchy**

Freshwater Criteria	U.S. EPA Sediment Quality Criteria[a], **or** Ecotox Threshold (U.S. EPA 1996b), or Long and Morgan (1990), **or** Ingersoll et al. (1996), **or** Ontario Freshwater Sediment Guidance (Persaud et al. 1993), **or** Application Equilibrium Partitioning (EqP) to Acute Water Quality Criteria after applying the 20 safety factor
Saltwater Criteria	Washington State Sediment Management Standards (Title 173-204 WAC), or Long et al. (1995), **or** Ecotox Threshold (U.S. EPA 1996b), **or** Application Equilibrium Partitioning (EqP) to Acute Water Quality Criteria after applying the 20 safety factor

[a] U.S. EPA Sediment Quality Criteria are found in U.S. EPA (1993a, b).

A safety factor of 20 is derived from the Tier II process developed in the Final Water Quality Guidance for the Great Lakes System (U.S. EPA 1995). This guidance document established a procedure for extrapolating from the National Guideline requirements of eight genus mean acute values (GMAV), which represents specific taxa needed to establish a criterion to data sets containing less information. This process applies increasing safety factors moving from data sets containing seven GMAVs to those with only one. An adjustment factor of 21.9 was recommended by U.S. EPA for use with only one study. This is modified in this approach to a safety factor of 20.

Sediment criteria are selected following the same hierarchical process using the criteria identified in Table 4-2. For some sediment chemicals, the U.S. EPA has published criteria developed using the Tier II process termed Ecotox Thresholds (U.S. EPA 1996b). Finally, if no sediment criteria are available for a nonionic organic chemical, then one will be calculated using the equilibrium partitioning approach (DiToro et al. 1991) to compare with the acute water quality criteria.

4.6 Aquatic Life Risk Assessment

The aquatic life risk assessment will be conducted to estimate potential risks from exposure to toxic chemicals, physical disturbances to the environment, and changes in water quality parameters through the following:

- Exposure to chemical concentrations in water and sediments

- Exposure to changed water quality parameters (e.g., DO, temperature)

- Reduction in available habitat from sedimentation and scouring related to CSO discharges

The approach that will be used to assess risks to aquatic life will follow available U.S. EPA ecological risk guidance, specifically the *EPA Proposed Guidelines for Ecological Risk Assessment* (U.S. EPA 1996a), *WERF Ecological Risk Assessment Protocol* (Parkhurst et al. 1994), and U.S. EPA's *Final Water Quality Guidance for the Great Lakes System* (U.S. EPA 1995). Salmon outmigrants, resident fish, epibenthos, and infaunal community structure were selected as representative of aquatic life because of their potential exposure to Duwamish River and Elliott Bay water, sediments, and habitat/water quality changes.

4.6.1 Measures and Methods for Conducting the Analysis Phase of the Aquatic Life Risk Assessment

Chemical data will be used to assess risks to aquatic life from CSOs discharging to the Duwamish River and Elliott Bay system, as well as from existing baseline conditions in these water bodies. For aquatic life, mean and maximum chemical concentrations in the water column and sediments will be compared with effects thresholds[9] to determine whether specific chemicals pose potential risks to benthos, epibenthos, shellfish, and fish. Chemicals posing potential risks to aquatic life will be further evaluated using the Water Environment Research Foundation (WERF) probabilistic risk approach to determine the total risk to aquatic life.

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[9] Effects thresholds are the concentrations where a specific effect (e.g. mortality of 50 percent of the exposed organisms) is observed.

To assess how changes in water quality parameters might affect aquatic life, changes in receiving water salinity, DO, and water temperature from CSO discharges will be compared to appropriate effects thresholds. Risks to benthos from increased sedimentation (i.e., smothering) and loss of habitat will also be compared to effects levels to determine whether potential risks exist. Risks from changes in water velocity will be qualitatively assessed for epibenthos and fish. The following sections present further details of how these assessments will be made.

Assessment of Chemical Stressors on Aquatic Life. Aquatic life risks from chemical stressors will be evaluated on two levels—a Tier 1 assessment of chemicals as single point estimates and a Tier 3 probabilistic assessment for both CSO discharges and other sources for the Duwamish River and Elliott Bay. The exposure areas will be identified based on the use of the study site by specific types of aquatic life. The chemical concentrations assessed in both the Tier 1 and Tier 3 assessments will be derived from the hydrodynamic model for both the water column and sediments.

The purpose of the aquatic life effects characterization will be to identify aquatic life toxicity reference values (TRVs). A TRV is an estimate of the lowest dose of a chemical that may cause toxic effects to the aquatic life receptors. Using the modeled data, point estimates of the mean and the maximum concentration will be calculated for specified sub-regions of the study area for the different receptor types. These estimates will be compared to the TRVs developed during the COPC selection process in the Tier 1 assessment. If either of the mean or the maximum exceed their respective criteria for any of the model configurations considered, the chemicals will be identified as requiring further evaluation in the Tier 3 probabilistic assessment. Chemicals which do not exceed criteria for any of the model configurations will be identified as not posing significant risk to aquatic life and will not be considered further. Use of a probabilistic range of concentrations in the Tier 3 assessment provides a quantitative expression of the uncertainty involved in determining exposure.

The contribution of CSO discharges versus other sources to each medium (water, sediment and tissues) will be determined primarily by using the hydrodynamic model. As discussed in Section 4.3, for chemicals that do not bioaccumulate, the measured tissue concentrations will be partitioned between CSO and other sources using the percent contribution of each source to water and sediments at specific locations within the study area. For chemicals that can bioaccumulate, it may be necessary to determine the contribution of short-term CSO discharges to fish and shellfish tissue concentrations using an unsteady-state bioaccumulation model.

Water Quality Parameters Stressors. Changes in water quality parameters can exert adverse effects on aquatic life through decreases in DO concentrations, increases in DO, increases in temperature, and decreases in salinity. Each of these types of changes

(except for DO[10]) will be estimated using the hydrodynamic model applied to the chemical stressors, with the contribution of CSO discharges and other sources identified for each parameter. The modeled exposure concentrations associated with each source will be compared to effect thresholds. Those parameters with hazard quotients (HQs) greater than 1.0 will be identified as posing risks to aquatic life receptors.

Physical Stressors. Risks from physical disturbance (such as sedimentation and scouring) will be expressed as the amount of habitat disturbed in the areas directly impacted by CSO discharges. The potential adverse effects to benthic communities will be qualitatively addressed (i.e., whether or not an adverse impact occurs after a CSO discharge). Any resuspension of sediments during erosion events leading to the re-release of chemicals into the water column will be addressed in the characterization of risks from chemical stressors.

4.6.2 Pulse Exposure Ratio Bioassays

A series of Pulse Exposure Ratio (PER) laboratory bioassays will be conducted to extrapolate from the continuous exposure of standard toxicity tests to the short-term episodic events characterizing CSO overflows. Bioassays will be conducted to derive weighting factors for chemicals with peak CSO discharge concentrations that exceed toxicity effects levels. The ratio of the effect level between continuous exposure and pulse exposure will be referred to as the pulse exposure ratio (PER). This is similar to the use of acute-chronic ratio (ACR), a common technique for estimating chronic toxicity from acute toxicity when insufficient data are available (e.g., Stephan et al. 1985). For example, most of the U.S. EPA's chronic water quality criteria are calculated using an ACR.

Pulse exposure ratios will be important to the risk assessment when developing the effects data to assess the acute exposure of aquatic life to CSO discharges. Pulse exposure ratios will be used to extrapolate from acute toxicity data to predicted pulse exposure toxicity data in the aquatic life risk assessment.

4.6.3 Benthic Assessment

The objective of the benthic bioassessment is to support the aquatic life risk assessment through an independent measure of potential risk. These data will serve to compare observed versus expected health in terms of the:

1. Risk assessment based on chemical toxicity data
2. Bioassays of site sediments

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[10]   Any potential changes in dissolved oxygen will be assessed using historical information in CSO discharges and receiving waters during wet weather events.

3. Assessments of the benthos

Together these three will provide three lines of evidence concerning risks. Additionally, comparison of the three lines provides a weight of evidence concerning overall risk to aquatic life. Having independent lines of evidence and field validation increases the reliability of risk assessment predictions, and is an established technique (Mount et al. 1986a,b; Norberg-King and Mount 1986).

Proposed benthic invertebrate field validation sites are the Connecticut Street, Brandon Street, Kellogg Island, and Denny Way CSOs. Final site selection will depend on the results of initial field verification. Several subsamples will be collected from (1) the CSO footprint and (2) the model cell[11] as well as from a reference area adjacent to the CSO discharge point. These subsamples will be split, with one half composited for immediate analysis and the other half suitably preserved for possible analysis in the future. The target organisms to be sieved from sediment for analysis will be large enough (>1 mm) and abundant enough to form the food base for the receptors and assessment endpoints.

Data from the benthic assessment will be compared to reference areas to determine if a significant reduction in the number of species present had occurred in areas potentially impacted by CSO discharges. Any statistically significant reduction in the number of benthic species present in the study area will be used to validate aquatic life risk assessment predictions.

4.6.4 Bioassay Program

The aquatic toxicity bioassay program is designed to address two elements of the risk assessment:

1. Corroboration (validation) of aquatic risk estimates

2. Development of a method to extrapolate from continuous to pulse exposure toxicity

The latter will be used in developing some of the aquatic life risk estimates. To corroborate risk estimates, water column and sediment bioassay results will be compared with risk predictions as a check on validity, which will provide one of the weights of evidence for the risk predictions.

The pulse exposure testing will be limited to samples of effluent and surface waters because these are expected to vary significantly after CSO discharges. Such testing will not be conducted with sediments because sediments integrate long-term exposure, hence

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[11]   The model cell is the smallest area that will be modeled by the hydrodynamic, water, and sediment quality model.

toxicity represents the mean exposure in the oxygen-bearing or toxic layer (Karickhoff and Morris 1985).

Recent work by Herricks et al. (1995) provides support to the working hypothesis that episodic, pulse exposures can elicit different effects from continuous exposure to these same concentrations over days. Pulse chemical concentrations can be an order of magnitude or more above background for periods of minutes to hours to days. However, the standard approach for aquatic bioassays has involved continuous and constant exposure of test organisms to chemical concentrations for periods of 24 to 96 hours (acute tests) to 7 to 28 or more days (chronic tests). These durations are unrepresentative of the short-term episodic events which characterize sewer overflows (Jaworski and Mount 1985, U.S. EPA 1991). Therefore, the bioassay program is designed to allow extrapolation of the extensive continuous exposure bioassay database to the pulse exposures conditions characteristic of CSO events.

4.6.5 Assumptions of the Aquatic Life Risk Assessment

The aquatic life risk assessment procedures used in the WQA require us to make certain assumptions in the exposure assessment and the effects characterization. Specific assumptions that will be made in the aquatic life risk assessment concern:

- Concentrations of chemicals in water and sediments
- Changes in water quality parameters
- Toxicity of chemical concentrations and changing water quality parameters
- Reduction in habitat availability

The hydrodynamic model used to represent chemical concentrations of water and sediment in the Duwamish River and Elliott Bay study site assumes that the values used to calibrate and validate the model are representative of the chemical concentrations found in the entire estuary. Thus, this assumes that the probabilistic range calculated for each environmental media (food, water, and sediments) accurately and realistically represents the complete range of concentrations possible for each chemical. Additionally, collection of environmental samples over the past year for model calibration assumes that these concentrations do not change significantly over time.

Identifying the toxicity of the chemicals found in water and sediment also requires specific assumptions. First, this assessment will rely on Washington State Water Quality Standards, Washington State Sediment Management Standards, U.S. EPA Ambient Water Quality Criteria, or literature values. The uncertainties in toxicity data and the assumptions made to account for these uncertainties (such as safety factors) are related to lack of data and the extrapolation of laboratory test data to aquatic life receptors. The net effect of these assumptions is to impart a conservative bias to the risk estimates.

A key assumption in calculating total risk is that the toxicological sensitivities of a wide range of taxa are represented on the species curve. Because chronic toxicity data are

often limited to a small number of species for a given chemical, the chronic species curve is usually estimated from the acute species curve using an ACR. For example, most of the U.S. EPA chronic water quality criteria are calculated using an ACR.

4.7 Wildlife Risk Assessment

Potential risks to wildlife from exposure to toxic chemicals and physical disturbances will be assessed by evaluating:

- Ingestion of contaminated vertebrate and invertebrate prey

- Ingestion of contaminated water

- Changes in the physical environment that adversely affects wildlife use of the site

- Changes in water quality parameters that adversely effects wildlife use of the site

The approach that will be used to assess risks to wildlife will follow available U.S. EPA ecological risk guidance and WERF protocols for ecological risk, specifically the *EPA Proposed Guidelines for Ecological Risk Assessment* (U.S. EPA 1996a), U.S. EPA's *Wildlife Exposure Factors Handbook* (U.S. EPA 1993f), and U.S. EPA's *Final Water Quality Guidance for the Great Lakes System* (U.S. EPA 1995). The spotted sandpiper, great blue heron, bald eagle, and river otter were selected as representative avian and mammalian wildlife species because of their potential exposure to Duwamish River and Elliott Bay water, sediments, and prey.

4.7.1 Measures and Methods for Conducting the Analysis Phase of the Wildlife Risk Assessment

Risks to wildlife will be assessed for the exposure pathways of CSO effluent and baseline conditions identified in the *Problem Formulation* (Appendix A1) for each avian and mammalian receptor. Exposure concentrations resulting from eating fish and shellfish prey, drinking water, and dermal exposure to water and sediments in the Duwamish River and Elliott Bay will be estimated using the following:

1. Analytical measurements of fish and shellfish tissues collected from the study site

2. Modeled concentrations in the water column and sediments

3. U.S. EPA identified food, water, and sediment consumption rates for each receptor (U.S. EPA 1993f)

Only tissue concentrations appropriate to the type of prey consumed by each receptor will be used in determining exposure.

Chemical doses or EEDs will be calculated using the measured and/or modeled concentrations in food, water, and sediments weighted by the consumption rates for each. These doses will be estimated probabilistically to quantify the uncertainty about the actual dose received by each receptor. Concentrations in prey, water, and sediment, as well as consumption values, will be expressed as probability distributions to express the range of exposure values for each wildlife receptor.

The toxic effects of concern in the wildlife risk assessment are those that can affect the survival and reproduction of individual sandpipers and bald eagles and the survival and reproduction of populations of great blue herons and river otters. As no U.S. EPA or Washington wildlife criteria or standards currently exist, the data that will be used to define TRVs will be obtained from the scientific literature (such as the toxicity benchmark values presented in Sample et al. 1996). Mammalian toxicity data will be scaled using the body weight of river otters to extrapolate the effects of laboratory animals to these receptors (Chappell 1992, Watanabe et al. 1992, Sample et al. 1996). In contrast, avian toxicity data will be used directly to estimate risks to sandpipers, great blue herons, and bald eagles regardless of the test organism because toxicity in different birds scales weight to the 1st power (Sample et al. 1996).

Wildlife risks will be determined by combining the results of the exposure assessment (EEDs) with the effects characterization (TRVs) to calculate probabilistic HQs (U.S. EPA 1998). Chemicals with HQ values greater than 1.0 will be identified as presenting potential risks to that wildlife receptor.

4.7.2 Assumptions of the Wildlife Risk Assessment

The wildlife risk assessment procedures that will be used in the WQA require assumptions in the exposure assessment and the effects characterization. Specific assumptions that will be made in the wildlife risk assessment will concern:

- Concentrations of chemicals in fish and shellfish prey and sediments
- Wildlife consumption rates of fish/shellfish prey and sediments
- Home range for each wildlife receptor
- Extrapolation of toxicity data from laboratory test animals to wildlife receptors

The techniques used to determine chemical concentrations in fish, shellfish, water, and sediment ingested by wildlife in the Duwamish River and Elliott Bay study area assumes that the samples collected are representative of the chemical concentrations found in the entire study area. Thus, this assumes that the probable range calculated for each environmental media accurately and realistically represents the complete range of concentrations possible for each chemical. Additionally, collection of environmental samples in the past year assumes that these concentrations do not change significantly over time.

Wildlife consumption rates for water, food, and sediment, as well as mean body weights, taken from U.S. EPA's *Wildlife Exposure Factors Handbook* (U.S. EPA 1993f) are based on measurements throughout the range of each species and do not necessarily represent the specific populations present in the study area. Similarly, literature information used to establish home ranges of each wildlife receptor will reflect the complete species and not necessarily populations using the Duwamish River and Elliott Bay.

Identifying the toxicity of the chemicals found in these environmental media also requires assumptions. First, this assessment will rely on either Washington State Water Quality Standards, Washington State Sediment Standards, U.S. EPA Ambient Water Quality Criteria, or literature values. The uncertainties in toxicity data and the assumption made to account for these uncertainties are related to lack of data and the extrapolation of laboratory animal test data to wildlife receptors. The net effect of these assumptions is to impart a conservative bias to the risk estimates in the wildlife risk characterization.

4.8 Human Health Risk Assessment

The human health risk assessment will estimate potential health risks from chemicals and pathogens under baseline or current conditions, as well as estimate the fraction of health risks attributable to CSO discharges. The human health risk assessment will generally follow the methodology formulated by the U.S. EPA (1989). This methodology consists of three primary steps: exposure assessment, toxicity assessment, and risk characterization. The exposure assessment evaluates potentially exposed populations and exposure pathways, and quantitates a range of potential chemical and pathogen exposures. The toxicity assessment reviews toxicological data for each COPC and provides estimates of their toxicities. The results of the exposure and toxicity assessments are then combined in the risk characterization, where risk estimates are formulated.

The U.S. EPA (1989) methodology will be expanded upon in several ways. First, an explicit problem formulation and analysis plans are prepared to accurately scope the risk assessment. Second, the range of potential toxic effects will be assessed for any chemical identified as being of concern. Third, as much site-specific data will be used as possible, including the results of a fishing (creel) survey of the study area. Finally, the risk assessment will address potential health effects from pathogens that may be present in the CSO discharges.

The methodologies to be used in the exposure assessment, toxicity assessment, and risk characterization, along with assumptions to be made in the evaluation, are discussed in the sections that follow.

4.8.1 Exposure Assessment

In the exposure assessment stage of the human health risk assessment, potentially exposed populations are assessed, exposure pathways are determined, and the range of

chemical and pathogen exposures are calculated. This approach allows for numerical estimates of potential exposure for each COPC evaluated. The exposed populations, exposure pathways, and the quantification of exposure are discussed below.

Exposed Populations and Exposure Pathways. People may be exposed to chemicals in the Duwamish River and Elliott Bay through a variety of activities. As described in the Problem Formulation (Appendix A1), these include a wide variety of direct exposure activities, such as sailing, wind surfing, swimming, SCUBA diving, wading, line fishing, net fishing, and gathering shellfish and other organisms (e.g., sea cucumbers, seaweed). Indirect activities that may result in exposure may include eating seafood (i.e., fish, shellfish and other organisms) harvested from the Duwamish River and Elliott Bay.

Exposure pathways to be evaluated in the human health risk assessment are detailed in the problem formulation and are summarized in Table 4-3. 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 will be quantitatively assessed.

Table 4-3. Exposure Pathways to be Evaluated in Human Health Risk Assessment

Route of Exposure	Media	COPCs Evaluated	Activities Potentially Resulting in Exposure
Incidental Ingestion	Water	Chemicals, pathogens	Swimming, sailing, wind surfing, net fishing, line fishing, gathering shellfish and other organisms, playing
Dermal Contact	Water	Chemicals	Swimming, sailing, wind surfing, net fishing, line fishing, gathering shellfish and other organisms, playing
Incidental Ingestion	Sediment	Chemicals	Swimming, sailing, wind surfing, net fishing, line fishing, gathering shellfish and other organisms, playing
Dermal Contact	Sediment	Chemicals	Swimming, sailing, wind surfing, net fishing, line fishing, gathering shellfish and other organisms, playing
Ingestion	Fish	Chemicals	Eating recreationally-caught fish, eating commercially-caught fish
Ingestion	Shellfish	Chemicals, pathogens	Eating recreationally-harvested shellfish, eating commercially-caught shellfish

Quantification of Exposure. Two chemical exposure scenarios and one pathogen exposure scenario will be quantified for each exposure pathway evaluated. Exposures will be estimated by combining measured and modeled chemical and pathogen concentrations in the environment and estimates of the types and frequencies of human

activities that may result in exposures. When possible, site-specific information, such as that derived by the fishing survey for the Duwamish River and Elliott Bay, will be used.

Direct contact human exposures will be calculated using modeled chemical and pathogen concentrations in Duwamish River and Elliott Bay waters and sediments. Chemical and pathogen exposures from seafood consumption will be estimated using measured concentration data in several species for the baseline conditions. Chemical concentrations in seafood will be modeled to estimate the fraction attributable to CSO discharges.

Exposures will be evaluated separately for children and adults. Evaluation of children is often a worst-case scenario, because children typically receive larger chemical doses based on their smaller body weights than adults do. However, because adults may potentially be exposed for the majority of a lifetime, they will also be evaluated.

A range of exposure assumptions will be used to evaluate the variability of possible human exposures. This range will include low, medium, and high estimates for each COPC. For example, low, medium, and high estimates of the number of times a person may come into direct contact with the waters and sediments will be estimated. Such direct contact may occur while wading, fishing, wind surfing, SCUBA diving, etc.

Estimates for the amount of seafood (i.e., fish, shellfish, and other organisms) that people consume from the Duwamish River and Elliott Bay will be derived from the ongoing fishing survey, from other available site-specific information (e.g., phone interviews), and from previous fishing surveys. The fishing survey was designed to estimate the frequency and amount that people catch and consume seafood from the study area, the species of seafood, and the number of people that eat seafood from the study area.

4.8.2 Toxicity Assessment

The toxicity assessment will present numerical estimates of chemical toxicity and pathogen infectivity for each of the COPCs evaluated. These estimates of chemical toxicity and pathogen infectivity will be combined with the exposure estimates in the risk characterization.

It is acknowledged that a wide range of potential effects, such as developmental effects, systemic effects, reproductive effects, or cancer, may occur from chemical exposure. However, numerical estimates of chemical toxicity are only available for systemic effects and carcinogenicity. Therefore, while other potential effects will be discussed, only numerical estimates of systemic toxicity and carcinogenicity will be presented.

The risk assessment will use the U.S. EPA-derived reference dose (RfD) as an estimate of chemical dose (milligram of chemical per kilogram body weight per day) to which an individual may be exposed without adverse systemic effects. The RfD is calculated based on the theory that there is a threshold dose below which no systemic effects will occur. When data are available, the RfD is based on epidemiological studies in exposed

people. When no study results for humans are available, the RfD is derived by combining results from studies using laboratory animals (ideally a no observed adverse effect level [NOAEL]) with various uncertainty factors. The uncertainty factors are generally designed to account for uncertainties in the differences in chemical sensitivity between laboratory animals and humans, the differences in chemical sensitivity between healthy and sensitive individuals in the human population, and the differences between less than lifetime and lifetime exposures. An RfD is usually based on changes in organ (e.g., liver) weight, function, and/or appearance.

To evaluate potential chemical carcinogenicity, the risk assessment will use the U.S. EPA-derived carcinogenic slope factor. The slope factor is calculated based on the current toxicological assumption that for carcinogenic chemicals there is no dose, no matter how small, that does not pose a finite risk of cancer. The cancer slope factor for each chemical is a conservative estimate of the slope of the line that relates the frequency of tumors (the response) to the level of chemical exposure (the dose). Therefore, combining an estimate of an environmental dose (e.g., from eating fish) with the slope factor, the frequency of tumors can be predicted. The slope factor is typically based on results (adjusted for body weight differences) from studies on laboratory animals. When available, results of epidemiological studies in exposed people are used.

Pathogen infectivity will be assessed using either an estimate of the minimum infective dose (MID) derived from the scientific literature or a distribution of infectivity. The MID for each pathogen (e.g., *Salmonella* sp.) is the minimum number of organisms that a person must be exposed to for infection to occur. The MID that is expected to result in infection in less than 25 percent of the population will conservatively be used.

Chemical toxicity data will be taken from the U.S. EPA IRIS data base (U.S. EPA 1996c). If a chemical does not have a toxicity value entered into the IRIS database, U.S. EPA's Health Effect Assessment Summary Tables (HEAST) will be used. For chemicals with no toxicity values in either of these databases, World Health Organization (WHO) databases on Allowable Daily Intakes (ADI) and Tolerable Daily Intakes (TDI) will be searched. Chemicals with no available toxicity information will be identified as posing uncertain risks in the discussion of human health risk.

4.8.3 Risk Characterization

The potential for human health effects from baseline conditions, along with those attributable to CSO discharges, will be evaluated in the risk characterization stage of the human health risk assessment. Risk characterization takes the results of the exposure and toxicity assessments and derives numerical estimates of potential health risks. Potential health risks will be quantitatively assessed for systemic effects, carcinogenic effects, and

pathogenic effects. Other types of effects, such as developmental effects in children and fetuses, will be discussed as appropriate.

Systemic Effects. The potential for non-carcinogenic, systemic[12] health effects from chronic (long-term) exposure to a chemical will be expressed as an HQ, in a similar manner to those that will be calculated for aquatic life and wildlife. The HQ represents the ratio of the estimated chemical dose to the chemical RfD. An HQ value that is less than 1.0 implies that non-carcinogenic health effects are unlikely to occur due to the chemical exposure (i.e., the estimated dose is less than the reference dose). An HQ greater than one indicates that the RfD is exceeded. An exceedance of the RfD does not necessarily imply that health effects will occur but instead that there is an increased potential for effects.

Carcinogenic Effects. Potential cancer risks will be calculated for each chemical that has a carcinogenic slope factor as the product of the estimated lifetime average daily intake and the cancer slope factor (U.S. EPA 1989). The cancer risk is interpreted as a probability between zero and one that an individual will develop cancer as a result of the chemical exposure, in addition to the background risk of cancer. Therefore, a 0.000001 cancer risk is interpreted as a 1 in 1,000,000 risk of developing cancer due to the estimated chemical exposure. A 0.000001 cancer risk may alternatively be interpreted as a prediction that one person in 1,000,000 exposed people will develop cancer.

Chemical-specific cancer risks will be assumed to be additive, consistent with U.S. EPA risk assessment guidance (U.S. EPA 1989). Total cancer risks in excess of the background cancer risk will be calculated by summing the chemical-specific excess cancer risks (U.S. EPA 1989).

Pathogenic Effects. The potential for infection due to microbial contaminants will be expressed as an HQ, similar to that used for system chemical effects. The pathogenic HQ is the ratio of the estimated dose of each pathogen with it's respective MID. An HQ less than one indicates that the dose is less than the MID for 25 percent of the population. An HQ greater than one indicates that the dose is larger than the MID for 25 percent of the population.

Population Risks. If sufficient data are available describing the number of people that engage in different activities (e.g., SCUBA diving) and the frequency that they do so, then an estimate of the population risks will be described. The population risk estimates will combine the risk estimates for all exposed people to describe the number of people that may experience different risk levels. It is understood that data describing the number

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[12]   A systemic health effect is any effect that does not result in immediate mortality, but represents an adverse impact on an organ system (e.g., the nervous system, the blood system) that can present a important burden on a person's quality of life.

of people that engage in different activities and the frequency that they do so may not be available. In such instances, the population risks will not be estimated.

4.8.4 Assumptions and Uncertainties

A series of assumptions will be made in the exposure assessment, toxicity assessment, and risk characterization sections of the human health risk assessment. These assumptions and the resulting uncertainties are briefly summarized below.

Assumptions in the Exposure Assessment. Major assumptions in the exposure assessment will include:

- Use of a combination of measured and modeled COPC concentrations in surface water, sediment, and seafood

- Use of a combination of national average and site-specific data to estimate a range of human exposure rates to surface waters, sediments, and seafood

- Use of fecal coliforms as a tracer for other pathogens

The measured and modeled COPC concentrations in surface water, sediment, and seafood assumes that the samples collected are representative of the entire study area. Additionally, collection of samples in a relatively short time period assumes that concentrations do not change significantly over time. Use of national average exposure rates is based on the assumption that these data are representative of the exposed populations. Use of site-specific data (i.e., the current and historical fishing surveys) assumes that these surveys adequately represent the patterns of people eating fish and shellfish in the Duwamish River and Elliott Bay. Finally, use of fecal coliforms as a tracer for other pathogens assumes that the ratio of concentrations of fecal coliform and the other pathogens remain constant after release into the environment. This assumption is uncertain because many pathogens exhibit different die off rates and rates of inactivation.

Assumptions in the Toxicity Assessment. Major assumptions in the toxicity assessment include use of chemical toxicity data for laboratory animals to estimate toxicity to people and use of infectivity data from the scientific literature.

The use of chemical toxicity data for laboratory animals to estimate toxicity to people is a generally accepted method for evaluating chemical toxicity in people. However, differences in chemical sensitivities and biological differences between organisms introduce large uncertainties into this method. Use of infectivity data from the scientific literature assumes that sufficient studies have been completed for the pathogens evaluated to assess their infectivity. There typically are few data available concerning the infectivity of specific organisms.

Assumptions in the Risk Characterization. Major assumptions in the risk characterization include:

- Assumed additivity of chemical systemic toxicity when effects are for the same organ or system

- Assumed additivity of chemical carcinogenicity regardless of the location of effect

- Additivity does not account for possible synergystic or antagonistic effects that may occur; the magnitude this uncertainty has not been quantified. It may increase or decrease any risk estimates made.

5. LEVEL OF UNCERTAINTY IN EXPOSURE PATHWAYS

While a number of exposure pathways have been identified in the Problem Formulation (Appendix A1), not all pathways can be equally evaluated because of limited information. Additionally, each pathway (or stressor) will not be equally important in posing risks to receptors. Consequently, it is important to identify relationships that have the greatest potential to cause the greatest impact and the uncertainty in our ability to assess that impact to help guide and prioritize the risk assessment efforts (U.S. EPA 1996a). U.S. EPA's guidance on the conduct of ecological risk assessment identifies the following criteria for identifying critical relationships:

- Availability of information
- Strength of information and relationships between stressors and effects
- The assessment endpoints and their relationship to ecosystem function
- Relative importance or influence and mode of action of stressors
- Completeness of known exposure pathways

U.S. EPA advises that where data are insufficient or cannot be gathered, pathways that cannot be assessed are a source of uncertainty and should be described in the analysis plan (U.S. EPA 1996a). Table 5-1 summarizes the uncertainty about the relationships between sources, stressors and receptors presented in the problem formulation.

Information gathered to date indicates that chemical concentrations in water, sediment, and tissues have a potential to pose risks to exposed receptors either directly through consumption (aquatic life, wildlife, and people) or indirectly through reducing prey availability to aquatic life and wildlife. Changes in water quality parameters (e.g., DO, salinity, TSS, and temperature) also have the potential to adversely impact aquatic life and wildlife.[13] Data concerning the threshold levels for adverse impact for each of these stressors are available. Additionally, data will be available from either historical studies conducted in the Duwamish River and Elliott Bay, the current field sampling program, or the hydrodynamic model. The model will allow the assessment of any risks posed by these stressors from either CSO or other sources. Thus, we have a low level of uncertainty about the importance of these stressors and exposure pathways and our ability to assess any potential risks posed to receptors from these stressors.

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[13] However, historical data suggests that these changes infrequently approach the thresholds required to elicit adverse impacts.

Table 5-1.   Level of Uncertainty in Exposure Pathways between Stressors and Receptors Identified in the Problem Formulation

Exposure Pathway	Reason for Level of Uncertainty
Low Level of Uncertainty Relationships	
Exposure of all receptors to toxic chemicals in water, tissue, and sediments	Data available on exposure and effects
Exposure of aquatic and wildlife receptors to changes in water quality parameters	Data available on exposure and effects
High Level of Uncertainty Relationships	
Habitat destruction and its impact on benthos	Limited data available on effects (also a natural phenomenon)
Dermal exposure of humans to water and sediments	Limited data available on skin transmissivity of many chemicals
Sediment consumption by bald eagles	Limited data available on consumption rates
Exposure of humans to microbial contaminants	Limited data available on exposure and effects

In contrast, data on the effects of habitat destruction, the importance of human dermal exposure to water and sediments, and the consumption of sediments by bald eagles are much more limited. For habitat destruction, the biological community using the sediment (benthos) has a ready source of colonists to replace individuals lost in a population from scouring/habitat destruction. Thus, habitat destruction is related to a natural component of this system, and thus it is not clear that enhanced scouring is likely to be a significant stressor in this system.

Dermal exposure is a difficult pathway to evaluate due to the large number of assumptions required for such an evaluation. U.S. EPA has published a decision matrix on evaluating the importance of dermal exposure (U.S. EPA 1992), which will be used in the human health risk assessment. Sediment consumption by bald eagles is likely to be an extremely limited contributor to the total chemical exposure of this receptor[14]. Finally, any efforts to assess risks from exposure to microbial contaminants must also be considered relatively uncertain.

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[14]   For example, dabbling ducks, which have a much closer association with sediment, only consume 2 percent of their diet as sediment (U.S. EPA 1993f).

6. ADDENDUM—DEVIATIONS FROM THE ANALYSIS PLAN

As with all large scientific studies, some of the tasks described in the analysis plan were implemented differently than described, while some others were not implemented at all. These changes to the plans were implemented, as new information became available or better understanding of the study area developed. We believe that the changes improved the quality of the final product and, in some instances, focused the study on issues that clearly need to be addressed. These deviations to the analysis plan occurred after the analysis plan was completed in June 1997. This addendum, added in February 1999, points out the deviations, and briefly explains we did some things differently than we originally planned.

Proposed changes in plans were routinely discussed at weekly project team meetings. If such changes could potentially affect the WQA results, they were discussed at monthly meetings with the stakeholder committee. Significant changes were also brought to the WERF peer review committee. An example was the decision to use all the field data to calibrate the Duwamish Estuary model, rather than withholding half the data for model validation. The modelers proposed this change to the project team, which discussed and agreed with the change. The stakeholder committee was told of the proposed change at one of its monthly meetings and given an opportunity to question or comment on it. The modeler on the peer review committee was asked to provide his opinion on the proposed change. A consensus developed that the proposed change was a good idea, and it was implemented.

This section identifies the deviations, and briefly explains the rationale for each deviation.

6.1 Changes in Terminology

The analysis plan does not always use the terminology we adopted in writing the Overview and Interpretation Report (Volume 1) and the Methods and Results (Appendix B). For example, during the course of the project we adopted the terms "baseline" and "without CSOs" for describing the two scenarios evaluated in the exposure analysis, and "other sources" for sources (of stressors) other than CSOs. In the Problem Formulation and the Analysis Plan, we use different terms, for example, "with CSO" for baseline, "no CSO" and "zero CSO" for without CSO, and "non-CSO sources" for other sources.

6.2 Chemical Measurements in Background/Reference Areas

We collected more reference site chemistry data than called for in the analysis plan. Table 3-4 presented the types of reference site samples we planned to collect for chemical analysis. The types of samples we did collect included:

- Sole, crab, perch, and prawns from Port Susan

- Invertebrates (*Coropheum and Eogammarus)* from the Nisqually Delta

- Sediment samples from the Kellogg Island benthic survey reference site

- Mussel data from in-river reference sites and a "clean" marine baseline site (Totten Inlet)

We used sediment concentration data from WSDOE's Puget Sound reference sediment sites rather than collecting new reference sediment samples. We did not collect reference water samples.

6.3 Habitat Loss

The analysis plan called for estimating the size of any areas scoured by CSO discharges (Table 3-6). We did not estimate areas scoured, instead we simply reported that CSOs do cause scouring when they discharge to the intertidal zone at low tide.

6.4 Pathogen Monitoring

The analysis plan called for analyzing raw sewage for the bacteria *Salmonella, Listeria* and Yersiniae, as well as enteric viruses. We did this, and additionally we analyzed for *Giardia* cysts. We did monitor for the bacteria *Salmonella*, *Listeria* and Yersiniae in mussels as well, but we did not collect daily mussel samples after a CSO event to determine the time required for concentrations to return to background.

6.5 Model Validation

As noted previously, we did not validate the model, choosing instead to use all the data for calibration. This is discussed in greater depth in Volume 1, at the end of Section 5.3, *Precautions for Future Investigations*. The purpose of using all the data for calibration was to get the best model calibration possible. Also, the field sampling program took place over a single six-month period, so we did not believe the calibration and validation data sets would be sufficiently independent to really consider the model validated.

6.6 Interfacing the Model with the Risk Assessment

The analysis plan called for evaluating concentration for the average, median, 75th percentile and 95th percentile model cells. We evaluated concentrations (and risks) for all model cells comprising the Duwamish River and Elliott Bay study area.

6.7 Bioaccumulative COPCs

The analysis plan indicated we would, if necessary, use an unsteady state bioaccumulation model to estimate fish tissue concentrations under the without CSOs

scenario. This was unnecessary because CSOs were found to contributed minimally to fish PCB exposures.

6.8 Identification of Candidate Chemical Stressors

We followed the procedure for identifying candidate chemical stressors as described in (Figure 4-3, and Tables 4-1 and 4-2), but in addition we also looked at other data, including mussel tissue concentrations and historical records to identify other candidate chemical stressors, as described in Appendix B-1.

6.9 Risks from Changes in Water Velocity (Displacement)

The analysis plan called for evaluating risks from this stressor qualitatively, but we were able to develop numerical thresholds and evaluate the risk quantitatively.

6.10 Aquatic Life Risk Assessment – Water Quality Parameters

The analysis plan called for modeling temperature in the estuary; we instead based the risk assessment on actual measured temperature data.

6.11 Aquatic Life Risk Assessment – Physical Stressors

The analysis plan called for evaluating potential adverse effects to benthic communities qualitatively; we conducted this analysis quantitatively as described in Appendix B-4.

6.12 Pulse Exposure Ratio Bioassays

The analysis plan called for conducting a set of Pulse Exposure Ratio (PER) laboratory bioassays to extrapolate from the continuous exposure of standard toxicity tests to the short-term episodic events we expected to characterize CSO events. We found, though, that exposure durations associated with CSOs in the Duwamish Estuary were sufficiently long to obviate the need for PER bioassays, so they were not conducted.

6.13 Benthic Assessment

The objectives and design of the benthic survey as described in the analysis plan were modified. Rather than a farfield investigation to corroborate risk estimates, the benthic survey was conducted as a nearfield investigation, to quantify the potential risks to benthos from CSOs in the immediate vicinity of the CSO discharge. The benthic survey methods and results are presented in Appendix B-4.

6.14 Water Column and Sediment Bioassays

It was decided to redirect the effort originally budgeted for water column and sediment bioassays to other needs, including additional tissue chemistry data. We did do one CSO bioassay (Brandon Street). Pulse exposure bioassays were deemed unnecessary because we found exposures were of acute duration or longer.

6.15 Wildlife Risk Assessment – Changes in Habitat and Water Quality Parameters

The analysis plan called for evaluating changes in habitat and water quality parameters that adversely affect wildlife use of the estuary. We did not explicitly assess these stressors for risks to wildlife, based on a subsequent determination that CSO-caused changes in habitat and water quality parameters were insignificant from a wildlife risk perspective.

6.16 Wildlife Risk Assessment – Hazard Indices

The analysis plan called for computing hazard indices by summing hazard quotients for chemicals with similar mode of toxicity. We did not explicitly compute hazard indices because the wildlife hazard quotients were too low for additive risk to affect the findings of risk only from lead and (for river otter only) arsenic.

6.17 Minimum Infective Dose for Pathogen Risk Assessment

The analysis plan called for using minimum infective doses (MIDs) as effects thresholds for the pathogen risk assessment. We were able to improve on the proposed methodology by obtaining dose-response relationships from the scientific literature for *Giardia* and enteric viruses. We were unable to determine MIDs or dose-response relationships for the bacteria *Salmonella*, *Listeria* and Yersiniae, so we assessed risks only for *Giardia* and enteric viruses.

6.18 Benthic Survey

The problem formulation called for conducting benthic community surveys near the mouth of several CSOs, at a reference site outside the study area, and at a reference site outside the study area. We only did one CSO and did not do reference sites outside the study area, instead using Washington State's reference ranges.

7. REFERENCES

Abbott, J.D., S.W. Hinton, and D.L. Borton. 1995. Pilot-scale validation of the RIVER/FISH bioaccumulation modeling program for nonpolar hydrophobic organic compounds using the model compounds 2,3,7,8-TCDD and 2,3,7,8-TCDF. Environmental Toxicology and Chemistry. 14(11):1999-2012.

Chappell, W.R. 1992. Scaling toxicity data across species. Environmental Geochemistry and Health. 14(3):71-80.

DiToro, D.M., C.S. Zarba, D.J. Hansen, W.J. Berry, R.C. Swartz, C.E. Cowan, S.P. Pavlou, H.E. Allen, N.A. Thomas, and P.R. Paquin. 1991. Technical basis for establishing sediment quality criteria for nonionic organic chemicals by using equilibrium partitioning. Environmental Toxicology and Chemistry. 10:1541-1583.

Herricks, E.E., I. Milne, and I. Johnson. 1995. Region specific timescale toxicity in aquatic ecosystems. Project 92-BAR-1. Annual Report 1995. Water Environmental Research Foundation. Alexandria, Virginia.

Ingersoll, C.G., P.S. Haverland, E.L. Brunson, T.J. Canfield, E.J. Dwyer, C.E. Henke, N.E. Kemble, and D.R. Mount. 1996. Calculation and evaluation of sediment effects concentrations for the amphipod, *Hyallela azteca*, and the midge, *Chironomus riparius*. J. Great Lakes Res. 22:602-623.

Jaworski, N.A. and D.I. Mount. 1985. Use of statistical information to improve compatibility between various components of the water quality based approach. *In*: R.D. Cardwell, R. Purdy, and R.C. Bahner, eds. Aquatic Toxicology and Hazard Assessments: Seventh Symposium. ASTM STP 854. American Society of Testing and Materials. Philadelphia, Pennsylvania. pp. 565-573.

Karickhoff, S.W. and K.R. Morris. 1985. Sorption dynamics of hydrophobic pollutants in sediment suspensions. Environmental Toxicology and Chemistry, 4:469-479.

Long, E.R. and L.G. Morgan. 1990. The potential for biological effects of sediment-sorbed contaminants testes in the National Status and Trends Program. NOAA Technical Memorandum NOS OMA 52. National Oceanic and Atmospheric Administration, National Ocean Service, Office of Oceanography and Marine Assessment. Seattle, Washington.

Long, E.R., D.D. MacDonald, S.L. Smith, and F.D. Calder. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. Environmental Management, 19:81-97.

Mount, D.I., A.E. Steen, and T. Norberg-King [Editors]. 1986a. Validity of effluent and ambient toxicity tests for predicting biological impact, Back River, Baltimore Harbor, Maryland. U.S. Environmental Protection Agency. EPA 600/8-86/001.

Mount, D.I., T. Norberg-King, and A.E. Steen [editors]. 1986b. Validity of effluent and ambient toxicity tests for predicting biological impact, Naugatuck River, Waterbury, Connecticut. U.S. Environmental Protection Agency. EPA 600/8-86/005.

National Council of the Pulp and Paper Industry for Air and Stream Improvement (NCASI). 1996. A procedure for estimating TCDD/TCDF concentration in river water and fish. Technical Bulletin. New York, New York. (In Press).

Norberg-King, T.J. and D.I. Mount [Editors]. 1986. Validity of effluent and ambient toxicity tests for predicting biological impact, Skeleton Creek, Enid, Oklahoma. U.S. Environmental Protection Agency. EPA 600/8-86/002.

Parametrix, Inc. 1993. Ecological risk assessment for Pinal Creek WQARF site, Gila County, Arizona. Prepared for HydroGeo Chem, Inc. Kirkland, Washington. 120p+appendices.

Parkhurst, B.R., W. Warren-Hicks, R.D. Cardwell, J.S. Volosin, T. Etchinson, J. Butcher, and S. Covington. 1996. Methodology for aquatic ecological risk assessment. Prepared for Water Environment Research Foundation. Alexandria, Virginia.

Persaud, D., R. Jaagumagi, and A. Hayton. 1993. Guidelines for the protection and management of aquatic sediment quality in Ontario. Water Resources Branch, Ontario Ministry of the Environment and Energy. Queen's Printer for Ontario. ISBN 0-7729-9248-7.

Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. Toxicological benchmarks for wildlife: 1996 Revision. Oak Ridge National Laboratory, under contract DE-AC05-84OR21400 for the U.S. Department of Energy.

Sherer, R.A. and P.S. Price. 1993. The effect of cooking processes on PCB levels in edible fish tissue. Quality Assurance: Good Practice, Regulation and Law, 2(4):396-407.

Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W.A. Brungs. 1985. Guidelines for deriving numerical national water quality criteria for the protection of aquatic life and its uses. U.S. EPA, Environmental Research laboratory-Duluth. Duluth, Minnesota. NTIS No. PB85-227049. 98 pp.

U.S. EPA. 1989. Risk assessment guidance for Superfund: Volume 1: Human health evaluation manual (Part A), Interim Final. U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. EPA/540/1-89/002.

U.S. EPA. 1991. Technical support document for water quality-based toxics control. U.S. Environmental Protection Agency, Office of Water. EPA/505/2-90-01.

U.S. EPA. 1992. Dermal exposure assessment: principles and applications. U.S. Environmental Protection Agency, Office of Research and Development. EPA/600/8-91/011B. Washington, D.C.

U.S. EPA. 1993a. Sediment quality criteria for the protection of benthic organisms: fluoranthene. United States Environmental Protection Agency, Office of Water and Office of Research and Development, Office of Science and Technology, Health and Ecological Criteria Division. EPA-822-R-93-012. Washington D.C.

U.S. EPA. 1993b. Sediment quality criteria for the protection of benthic organisms: phenanthrene. United States Environmental Protection Agency, Office of Water and Office of Research and Development, Office of Science and Technology, Health and Ecological Criteria Division. EPA-822-R-93-014. Washington D.C.

U.S. EPA. 1993c. Wildlife exposure factors handbook. Office of Research and Development, U.S. Environmental Protection Agency. EPA/600/R-93/187a. Washington, D.C.

U.S. EPA. 1994. Water quality standards handbook: Second Edition. United States Environmental Protection Agency, Office of Water. EPA-823-B-94-005a. Washington, D.C.

U.S. EPA. 1995. Final water quality guidance for the Great Lakes System; Final Rule. 40 CFR 9, 122, 123, 131, and 132.

U.S. EPA. 1996a. Proposed guidelines for ecological risk assessment; Notice. Federal Register, Vol. 61, No. 175.

U.S. EPA. 1996b. Ecotox thresholds. United States Environmental Protection Agency, Office of Solid Waste and Emergency Response. Publication 9345.0-12FSI, EPA 540/F-95/038.

U.S. EPA. 1998. IRIS (integrated risk information system) online database. Information system updated regularly by U.S. Environmental Protection Agency. Washington, D.C. Available at: http://www.epa.gov/iris.

Watanabe, K., F.Y. Bois, and L. Zeise. 1992. Interspecies extrapolation: reexamination of acute toxicity data. Risk Analysis, 12(2):301-310.